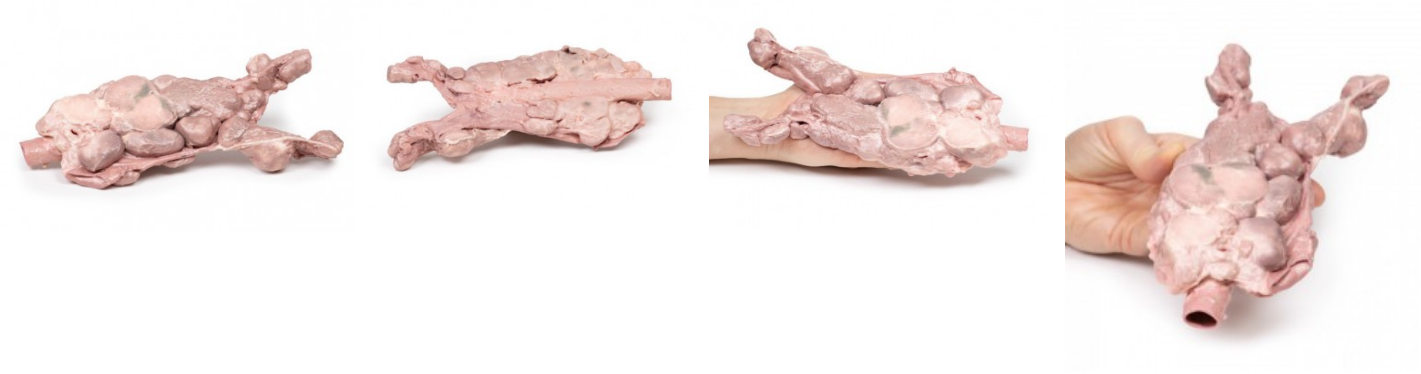
Aorta & para-aortic lymph nodes

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02802



Clinical History

This was a case of a 75-year-old female who presented with symptoms of recurrent disease and confirmed to have chemo-resistant multiple retroperitoneal lymph node metastases five years after the initial therapy for stage IIIc serous adenocarcinoma of the ovary. Positron emission tomography/computed tomography (PET/CT) revealed the involvement of para-aortic nodes and pelvic nodes. She died of liver complications before therapeutic options, such as radical lymphadenectomy, could be considered.

Pathology

The specimen consists of the abdominal aorta and common iliac arteries surrounded by large numbers of extremely enlarged iliac nodes paraaortic lymph nodes. Histopathological examination revealed metastatic high-grade adenocarcinoma in some of the resected lymph nodes.

Further Information

Occasionally, lymph node metastases represent the only component at the time of recurrence of ovarian cancer. In this case, the metastatic nodes predicted by PET/CT completely corresponded to the actual metastatic nodes. Ultrasound (US) could equally have confirmed the presence of such large lymph nodes. PET/CT or US often fails to identify microscopic disease in histopathologically-proven positive nodes. Therefore, it is difficult to reliably exclude lymph node metastases during surveillance following initial surgery for ovarian cancer. In the context of a recurrent ovarian disease, systematic aortic and pelvic node dissection would be considered appropriate in younger women with no other evidence of metastatic disease. This is unlikely to be curative but may produce palliation with symptom control and allow for the trial of novel therapies if available. More commonly, sampling of pelvic and aortic lymph nodes is part of the formal surgical staging of epithelial ovarian cancer at the time of initial surgical treatment. In addition, in women presenting initially with advanced stage ovarian cancer, systematic debulking of enlarged retroperitoneal lymph nodes will be considered if this leads to complete debulking of the tumour.

Hydatid Disease Affecting the Heart and Aorta

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02738

Clinical History

This 11-year-old female had an 18-month history of hydatid disease (see below). In total, 17 cysts were removed from the child's brain at craniotomy on three occasions, and subsequently cysts were found in the kidneys, mesentery, and abdominal aorta at its bifurcation. X-ray of the heart showed a calcified cyst, and the patient was referred to a tertiary hospital for its removal. The patient deteriorated and died following open-heart surgery during which a dead hydatid cyst was found in the left ventricle.

Pathology

The specimens are of the heart, with the left ventricle being laid open, and of the aorta at its common iliac bifurcation. The aorta shows some atheromatous depositions in the upper portion. There is a large mass of antemortem clot at the point of iliac bifurcation with extension down both common iliac arteries.

The heart shows hypertrophy of the left ventricular wall, and an abnormal communication between the left ventricle and atrium running through the posterior cusp of the mitral valve via the papillary muscle into the left ventricular cavity. This channel is surrounded by thickened fibrous-looking tissue. The posterior cusp of the valve has been split. Sutured surgical incisions are visible on the posterior aspect of the specimen and the ventricular wall and in the left atrial appendage. Hydatid cysts occupy the abdominal aorta at its bifurcation, and the channel joining the left ventricle and left atrium.

Histology demonstrated cysts within the aorta wall comprised of 3 layers: an outermost pericyst fibrous layer; a middle ectocyst layer that was laminated, hyaline and acellular; and the inner endocyst in the germinative layer, consisting of daughter cysts and brood capsules with scolices. A focal granulomatous palisading reaction was also present within the aorta wall.

Further Information

Hydatid cyst is a human parasitic disease caused by the larval stage of the cestode tapeworm Echinococcus granulosus, which infests the gut of dogs—its definitive hosts. Human beings may serve as incidental hosts by the ingestion of ova in vegetables or water contaminated with dog faeces. Humans become infected by the ingestion of eggs passed in dog faeces. Oncospheres released from the eggs penetrate the intestinal mucosa and, via the portal system, lodge in the liver, lungs, muscle or other organs, where the hydatid cysts form.

Hydatid disease is endemic in cattle-raising areas of the world, notably in the Mediterranean countries, the Middle East, South America, Australia, and New Zealand. Although no body part can be spared from hydatid cysts, they mostly affect the liver and lungs. Cardiac involvement is much rarer, yet potentially fatal condition and comprises 0.5–2% of all hydatid cases. Cardiac complications and presentation vary depends on the location, size and integrity of the cyst(s). The myocardium of the left ventricle more frequently involved. Pericardial involvement occurs mostly in multifocal cardiac echinococcosis. Growth of the cyst leads them being pushed toward a weaker side of the cardiac

wall, either the epicardium or the endocardium. LV HCs are usually located subepicardially, therefore rarely rupture into the pericardial space. However, if rupture happens, it may be silent or it may cause acute pericardial tamponade, constrictive pericarditis or secondary pericardial cysts[1].

Although E. granulosus is still found in sheep and rural dogs in Australia, the prevalence of transmission is less common than it was. The marked reduction in prevalence in rural domestic dogs, and also sheep, is the result of the highly effective cestocidal drug, praziquantel, being included in readily available, cheap, generic, all-wormers for dogs and the development of inexpensive commercial dry dog food[2].

References: 1. Oraha et al. Ann Med Surg (Lond). 2018 18–21 2. Jenkins et al. Int J Parasitol Parasites Wildl. 2019: 256–259.

Hirschsprung‘s Disease

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02780

Clinical History

A 5-year old male presents with a history of constipation since birth. A barium enema showed a constricted rectum with a dilated sigmoid colon. Surgical resection of constricted section of bowl was attempted but the patient died during the surgery.

Pathology

This postmortem section of sigmoid colon has been opened to display the internal surface shown here. There is large dilation of the proximal section of bowel (sigmoid) with loss of the normal mucosal pattern. The distal section of bowel (rectum) has a normal diameter and a normal mucosal pattern but an absence of ganglion cells in the myenteric plexus. This is an example of Hirschsprung‘s disease, also known as congenital aganglionic megacolon.

Further Information

Hirschsprung‘s disease is characterised by lack of coordinated peristaltic contraction in a segment of bowel, due to a lack of parasympathetic ganglia. It mainly affects the rectum but the length of the aganglionic sections vary. It is caused by defective proximal to distal migration of neural crest cells from the caecum to rectum during embryogenesis, which leads to development of a distal bowel segment lacking both a myenteric and submucosal plexus. This causes an obstruction with hypertrophy and dilation of the proximal normally innervated colon, which can lead to perforation, peritonitis, enterocolitis and electrolyte imbalances. It occurs in 1 in 5000 live births. It is more common in males, those with siblings with Hirschsprung’s disease and those with other developmental disorders, such as Down Syndrome. Mutations in the receptor tyrosine kinase RET, which is necessary for neural crest cell migration, account for a majority of the familiar cases and 15% of the sporadic cases. Patients typically present with failure to pass meconium within 48 hours of birth. Less severe cases present later with chronic constipation, vomiting, abdominal pain and distension. Treatment involves resection of the aganglionic section of bowel with anastomosis of the normal sections of bowel to the rectum.

Chondrosarcoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02771

Clinical History

A 57-year old male attends complaining of recurrent pain in his right thigh. On examination there is no palpable abnormality in the thigh. An x-ray of the limb showed bony absorption associated with expansion and periosteal reaction at the proximal right femur. A CT of the limb showed a mass in the proximal right femur. A biopsy was taken of the lesion. Subsequently he had excision of the right upper femur followed by insertion of a prosthesis.

Pathology

The specimen comprises the head, neck and upper third of the shaft of the right femur, sawn longitudinally to display the cut surface. In the medullary cavity of the upper portion of the shaft is an ovoid tumour that is 6.5 cm in maximum diameter. The tumour is not encapsulated and has a haemorrhagic cut surface with pale hyaline and cystic areas. Histologically, this is a low grade chondrosarcoma.

Further Information

Chondrosarcomas are malignant bone tumours that produce cartilage. These are the third most common primary bone malignancy after myeloma and osteosarcoma. Conventional tumours are the most common subtype of chondrosarcoma, making up 90% of cases. Less frequently diagnosed subtypes include clear cell, dedifferentiated and mesenchymal chondrosarcomas.

Some chondrosarcomas arise from pre-existing benign lesions, such as enchondroma or osteochondroma. Common mutations in chondrosarcomas are point mutations in the IDH1 and IDH2 genes as well as silencing of CDKN2A tumour suppressor gene. Chondrosarcomas that occur in multiple osteochondroma syndrome have mutations in the tumour suppressor EXT genes.

Men are twice as likely to develop chondrosarcoma than women. The axial skeleton is more frequently affected than the appendicular skeleton. Around 20% affect the femur. These are largely slow growing tumours. They usually present with painful and gradually enlarging masses. At the time of diagnosis, most are low grade tumours that rarely metastasize. The lungs are the most common site for distant spread. Grade 1 tumours have an almost 90% 5-year survival rate, whereas with grade 3 chondrosarcoma the 5-year survival rate drops to 43%.

CT scan is the optimal radiological investigation for diagnosis with MRI also frequently used. Biopsies may be taken to assist diagnosis. Treatment depends on the grade and the location of the tumour. Complete surgical resection is the standard treatment. Generally, chondrosarcomas do not respond to chemotherapy or radiotherapy given they very slow growing tumours.

Chondrosarcoma of femur and ilium

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02811

Clinical History

A teenage boy presents with groin pain after horse-riding. Examination revealed a large, deep lump. Following biopsy and imaging, the diagnosis of chondrosarcoma was made and a radical surgical resection of his right leg was performed.

Pathology

The specimen consists of the upper end of the femur and its articulation with the pelvis. Within the neck and head of the femur and replacing most of the ilium there is a lobulated pale grey tumour with areas of cavitation, necrosis and haemorrhage. The tumour is extending out beyond bone into the surrounding soft tissues and appears encapsulated. The presence of infiltration, necrosis and haemorrhage are macroscopic features of malignancy.

Further Information

Chondrosarcoma is a primary malignant bone tumour with cartilaginous differentiation. It is a rare cancer that accounts for about 20% of bone tumours. The only available treatment is excisional surgical resection since the current adjuvant treatments are ineffective. The pelvic location creates specific technical difficulties both for exeresis and reconstruction. The disease usually starts in the bones of the arms, legs or pelvis, but it can be found in any part of the body that contains cartilage. Sometimes chondrosarcoma grows de novo form an otherwise healthy bone; however, sometimes it may arise from a benign bone tumour (an enchondroma or osteochondroma). There are several subtypes of chondrosarcoma, named based on their microscopic and genetic characteristics. These include: conventional chondrosarcoma; Clear cell chondrosarcoma; Myxoid chondrosarcoma; Mesenchymal chondrosarcoma; Dedifferentiated chondrosarcoma.

Chondrosarcoma of scapula

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02773

Clinical History

A 60-year old female presented with a 12-month history of recurrent pain and increasing swelling in her right shoulder. On examination, there was a palpable mass over the superior aspect of her right scapula. There was limitation of abduction and external rotation at the shoulder joint. There was no palpable lymphadenopathy. X-ray of her shoulder showed a mass involving the superior scapula above the spine. The mass was biopsied and the scapula was completely excised.

Pathology

The specimen is the patient’s excised right scapula. An irregular lobulated tumour 11 cm in maximum diameter arises from the spine of the scapula and extends to involve the acromion and coracoid process. The tumour is a mottled pale-yellow brown colour with patchy surface haemorrhage. There is some adherent muscle and fibrous tissue. The mass has infiltrated and replaced the normal bone. Histologically the tumour consisted of pleomorphic rounded and spindle-shaped cells with numerous mitotic figures and cartilage formation. This is chondrosarcoma of the scapula.

Further Information

Chondrosarcomas are malignant bone tumours that produce cartilage. These are the third most common primary bone malignancy after myeloma and osteosarcoma. Conventional tumours are the most common subtype of chondrosarcoma; making up 90% of cases. Less frequently diagnosed subtypes include clear cell, dedifferentiated and mesenchymal chondrosarcomas.

Some chondrosarcomas arise from pre-existing benign lesions, such as enchondroma or osteochondroma. Common mutations in chondrosarcomas are point mutations in the IDH1 and IDH2 genes as well as silencing of CDKN2A tumour suppressor gene. Chondrosarcomas that occur in multiple osteochondroma syndrome have mutations in the tumour suppressor EXT genes. Men are twice as likely to develop chondrosarcoma than women. The axial skeleton is more frequently affected than the appendicular skeleton. Around 5% affect the scapula. These are largely slow growing tumours. They usually present with painful and gradually enlarging masses. At the time of diagnosis most are low grade tumours that rarely metastasize. The lungs are the most common site for distant spread. Grade 1 tumours have an almost 90% 5-year survival rate, whereas with grade 3 chondrosarcomas, the 5-year survival rate drops to 43%.

CT scan is the optimal radiological investigation for diagnosis with MRI also frequently used. Biopsies may be taken to assist diagnosis. Treatment depends on the grade and the location of the tumour. Complete surgical resection is the standard treatment. Generally, chondrosarcomas do not respond to chemotherapy or radiotherapy given they are very slow growing tumours.

Lymphoma of the thyroid

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02799

Clinical History

A 68-year old woman presented with a small hard lump in the thyroid. During the following six weeks, there was progressive rapid enlargement of the mass leading to laryngeal stridor and oesophageal obstruction, which were the terminal features. No lymph nodes were enlarged and there was no splenomegaly.

Pathology

The larynx, thyroid, upper trachea and oesophagus are included in the specimen. The enlarged left lobe and to a lesser degree, the right lobe of the thyroid, are replaced by homogeneous pale tumour tissue. Stretched over the lateral margin of the left lobe is the common carotid artery. Note on the internal aspect how the larynx is compressed and the oesophagus virtually disappears into the bulk of the tumour. The histological appearance of the tumour was consistent with lymphoblastic lymphoma of the thyroid. The rarity of this tumour makes it necessary to exclude the diagnosis of anaplastic carcinoma of the thyroid, and secondary spread from lymphoma elsewhere.

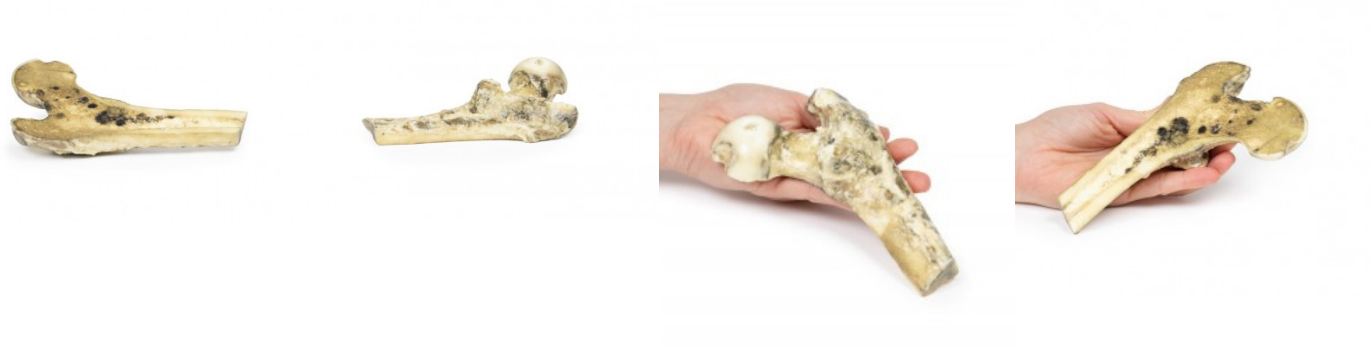
Further Information

Primary lymphoma of the thyroid is rare but should always be considered as a differential diagnosis of any thyroid mass or nodule. Primary thyroid lymphomas are rare, and are usually of the non-Hodgkin type. Lymphoblastic lymphoma is an aggressive form of non-Hodgkin’s lymphoma (NHL), usually seen in children. Currently, the only known risk factor for developing primary thyroid lymphoma is chronic autoimmune (Hashimoto’s) thyroiditis, with a 50% occurrence rate. Thyroid lymphoma should be considered in any individual presenting with a rapidly enlarging ‘goitre’, as over 90% of individuals with this disease present this way. As the goitre enlarges, there can be compression of nearby structures such as the trachea, oesophagus and neck vessels (the common carotid in this case). The resulting symptoms include stridor or hoarseness, dysphagia and neck pain. Other systemic symptoms of lymphoma include so-called B-symptoms, i.e. night sweats, fevers and loss of weight. Diagnosis can be achieved by an ultrasound scan followed by fine needle aspiration or excisional biopsy. As thyroid lymphomas cannot be excluded from Hashimoto’s or other carcinoma, cytology and immunohistochemical stain of the biopsied tissue is required to confirm the diagnosis.

Metastatic Malignant Melanoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02770



Clinical History

A 65-year old male with presents with pain in his left groin. He has a history of skin melanoma on his left foot treated with surgical resection and radiotherapy. On examination, he is cachexic with a hard, enlarged liver and has a discharging sinus in the left groin surrounded by black nodules. He is admitted and dies from a hospital-acquired pneumonia.

Pathology

The specimen is the patient’s proximal right femur sawn longitudinally to display the cut surface. The medullary cavity contains many deposits of tumour tissue varying in colour from a pale brown to black. Cancellous bone has been completely destroyed by the larger deposits, which appear dark and measure up to 3 cm in maximum diameter. Elsewhere pale brown tumour infiltrates the marrow cavity diffusely. Cortical bone has been spared, although at the junction of the shaft and neck, medially the cortical bone is discoloured and thickened. These are metastatic deposits from a melanoma of the skin.

Further Information

Melanoma is a malignant skin cancer associated with exposure to UV radiation in sunlight or tanning beds. Other risk factors for developing melanoma include fair complexion, presence of large number of melanocytic naevi, severe sunburn as a child and immunosuppression. It accounts for around 5% of all skin cancer diagnosis but has the highest mortality rate of all skin cancers. Melanomas typically occur in sun exposed areas as a pigmented lesion with irregular borders, variegated colour, an asymmetrical shape and which evolves of time. There are multiple mutations common in melanoma. Loss of cell cycle control gene from mutation in CDKN2A gene. Mutations in pro-growth signalling pathways such as BRAF and PI3K mutations are seen frequently in melanomas, as well as mutations that activate telomerase such as the TERT gene. Recognition that melanoma antigens activate host immune responses has led to promising immunotherapy, which enhances host T-cell identifying of these antigens.

The most common sites for metastasis of malignant melanoma are the lungs, liver, brain and bone as well as regional lymph nodes. Bone metastases are found in 25-50% of metastatic melanoma. The axial skeleton is more frequently affected by metastatic melanoma spread. These metastatic deposits cause pain and even pathological fractures. The probability of metastatic spread depends on the stage of the primary tumour, which is based on tumour depth, mitotic activity and ulceration of the skin as well as node and solid organ involvement.

Diagnosis of melanoma is made with excisional biopsy. Investigation for bone metastasis is done using blood test (raised Alkaline phosphatase, calcium and LDH) and radiological investigations most commonly X-ray and CT but MRI and PET scan may also be used. Treatment depends on the stage or the tumour as well as the immune profile of the melanoma. Treatment usually involves surgical resection, chemotherapy, immunotherapy, radiotherapy or more commonly a combination of treatments.

Bicuspid Aortic Valve

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02736

Clinical History

A 64-year old woman presented with a story of chest pain for 5 months, associated with breathlessness and wheezing for 4 months. On examination, she was dyspnoeic, with an expiratory wheeze, left-sided crepitations and signs of a right pleural effusion. The pulse rate and blood pressure were normal. There was a precordial systolic murmur and a heaving apex beat in the 5th left intercostal space 10 cm from the midline. There was no peripheral oedema. The patient died 4 days after admission.

Pathology

The heart has been opened to display the left ventricle and associated valves. The aortic valve has 2 cusps instead of the usual three. The valves are otherwise normal apart from patchy slight thickening. The aortic origins of the left and right coronary arteries are widely patent, as is the left circumflex coronary artery, seen cut transversely in the atrio-ventricular groove at the right hand lower edge of the specimen. There is dense pericardial fibrosis and adhesions on the posterior side of the specimen, suggestive of a constrictive pericarditis. The cause of this is not apparent from the history. At autopsy, there was ascites, a small shrunken cirrhotic liver, bilateral pleural effusions (R>L), and right pulmonary collapse. The cause of death was liver cirrhosis and failure, possibly consequential to the above-described constrictive pericarditis. The bicuspid aortic valve was an incidental finding.

Further Information

Bicuspid aortic valve is a more common congenital anomaly than widely appreciated as it may remain asymptomatic till later in life. The condition predisposes to the development of calcific aortic stenosis, usually in the 5th to 7th decades of life. They may occur alone or as part of a congenital syndrome, such as Tetralogy of Fallot. The latter is a combination of four congenital abnormalities, including a ventricular septal defect, pulmonary valve stenosis, a misplaced aorta and a right ventricular hypertrophy.

Bicuspid aortic valves have unequal cusp sizes usually as a result of two or the three normal cusps being fused together. This can lead to greater valvular dysfunction. Patients with bicuspid aortic valves are at high risk of aortic dilatation and dissection.

Bicuspid aortic valves are more likely to become calcified in older age than tricuspid aortic valves, and this is due to abnormal motion and turbulence caused by the unequal leaflet sizes.

With increasing age, patients can develop aortic stenosis or aortic regurgitation. When these become severe, symptoms associated with dyspnoea and reduced exercise tolerance may develop. This may be the first sign of a

bicuspid aortic valve. Diagnosis of bicuspid aortic valves is confirmed using transthoracic echocardiogram.

Villous adenoma of colon

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02765

Clinical History

A 70-year old man was admitted for investigation of muscular weakness and the passage of large amounts of mucus per rectum. The patient was found to be hypokalaemic. A tumour of sigmoid colon was discovered and later resected.

Pathology

A 15 cm long segment of colon has been opened longitudinally to display a large sessile tumour with a velvety surface. The tumour measures 11 x 7 cm. in diameter and approaches to within 2 cm of the distal resection margin. The mucosa is otherwise normal. The serosal surface is unremarkable. Histological examination confirmed the presence of a villous adenoma.

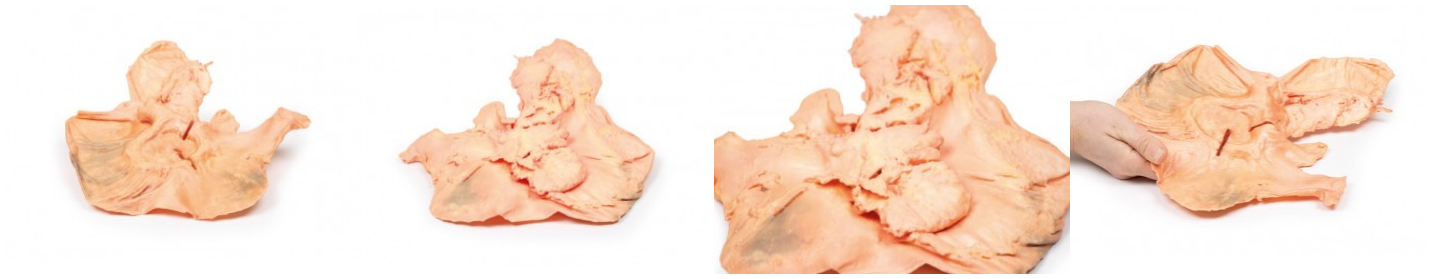
Further Information

Villous adenomas occasionally secrete large amounts of mucoid protein and/or potassium rich fluid, sufficient to result in hypoalbuminaemia or hypokalaemia. Villous adenoma is the least common but most ominous type of adenomatous polyp. Invasive carcinoma is present in up to 30% of cases at the time of resection.

Adenocarcinoma of the stomach

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02781



Clinical History

An 82-year old female presents with an episode of melena (dark tarry faeces). She had a 6-month history of dyspepsia and nausea. Recently she had noted weight loss and early satiety. Soon after admission she had a large melena episode and died.

Pathology

This is a post mortem specimen sliced to include a sagittal view of the oesophagus, stomach, proximal duodenum and pancreas. A large 7x5cm ulcer is evident on the lesser curve of the stomach. The ulcer is shallow and broad with raised rolled edges and necrotic debris at the base. There is loss of gastric rugae radiating along the mucous from the ulcer. Dissection of the ulcer reveals elevation of the edge by pale homogenous tumour tissue. There were two eroded arteries present within the ulcer crater with evidence of recent haemorrhage. The pancreas is adherent to the serosal aspect of the ulcer. Histology taken from the lesion (sites visible as regular 3cm defects) demonstrated an ulcerating, well-differentiated adenocarcinoma of the stomach with direct invasion into the pancreas.

Further Information

Gastric adenocarcinoma is the most common malignancy of the stomach. The incidence varies widely with geography: with a much higher incidence in Japan, Chile, Eastern Europe when compared to North America, Africa, South East Asia and Northern Europe. Risk factors include smoking, high salt diets, H. Pylori infection, Gastro Esophageal Reflux Disease (GERD), atrophic gastritis and intestinal metaplasia of the gastric mucosa. There are two distinct classifications: intestinal and diffuse gastric adenocarcinoma. Intestinal adenocarcinoma resembles glandular tissue similar to colonic or oesophageal adenocarcinoma. Intestinal types tend to be bulky: growing as either an ulcerated or exophytic tumour. Intestinal type occur most frequently in endemic areas, has a male predominance and a mean age of 55 years at presentation. Intestinal type can occur from precursor lesions, such as dysplasia and/or adenomas with dysplasia. Diffuse type gastric cancers have an infiltrative growth pattern and are composed of ‘signet ring’ cells – i.e. cells that have large vacuoles full of mucin leading to displacement of the nucleus to the cell‘s periphery. The cells appear to have lost adhesion between each other, and can therefore be widely distributed within the stomach mucosa. A mass may not be appreciated in this diffuse type as a desmoplastic reaction can occur around the tumour cells, causing a thickened and rigid stomach wall with loss of rugae, creating a “leather bottle” appearance also know of linea plastica. Diffuse type has equal incidence across sexes and countries, and does not have precursor lesions. Germ line mutations in CDH1, which causing loss of function of E-cadherin leading to the loss of cell adhesion, can result in an increased risk of diffuse gastric cancer, which can be familial. Patients with Familial Adenomatous Polyposis (FAP) with germ line mutation in adenomatous polyposis coli (APC) gene have an increased risk in developing intestinal type gastric adenocarcinoma. Early symptoms include dyspepsia, dysphagia and nausea. Later symptoms include weight loss, anorexia, early satiety, fatigue, anaemia and haemorrhage. Treatment depends on tumour stage with surgical resection for early tumours and chemotherapy for later stage cancers.

Fibrocaseous Tuberculosis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02752

Clinical History

A 89-year old male presents with an episode of large haemoptysis. He has a history of diabetes and immunosuppression secondary to steroid treatment for rheumatoid arthritis. Further history reveals a long history of cough, haemoptysis, fevers and weight loss. On examination, he is noted to be cachexic, hypoxic and have crepitations throughout the left lung. Chest x-ray shows multiple cavitation lesions in the left lung. Subsequently, he has another massive haemoptysis and dies.

Pathology

The left lung is cut longitudinally to display the cut surface. The upper lobe is almost entirely replaced by several large irregular cavities lined by necrotic debris and fibrous tissue. Blood vessels are seen in the upper cavity with evidence of haemorrhage. The lower lobe contains several smaller caseous areas, some of which are breaking down. The intervening lung parenchyma is scarred. The pleura is thickened. This is fibrocaseous tuberculosis with cavitation.

Further Information

Tuberculosis (TB) is a chronic pulmonary and systemic infectious disease caused by Mycobacteria tuberculosis. Transmission most commonly occurs via inhalation of aerosolized droplets of M. tuberculosis. Risk factors for contracting TB include being an inhabitant of a developing country where the disease may be endemic, immunosuppression (e.g. HIV, steroid use, anti-TNF use and diabetes), chronic lung disease (e.g. silicosis), alcoholism and malnutrition.

After initial pulmonary infection of M. tuberculosis clinical manifestation varies. In 90% of individuals with an intact immune system they enter an asymptomatic latent infection phase. This latent TB may reactivate at any time in the patient‘s life. In the other 10% of patients, especially in the immunocompromised, they develop primary disease which is immediate active TB infection. Manifestations of primary TB include pulmonary infection symptoms (e.g. consolidation, effusion and hilar adenopathy) and extra pulmonary symptoms including lymphadenopathy, meningitis and disseminated miliary TB.

Secondary tuberculosis occurs when there is reactivation of previous latent TB infection. Around 10% of latent TB will reactivate usually during periods of weakened host immunity. Typical symptoms of reactivation are cough, haemoptysis, low grade fever, night sweats and weight loss.

The immune response against TB is mediated via TH1-cells stimulate alveolar macrophages to attack the mycobacteria. These macrophages surround the infection forming a ‘granuloma’ with central caseous necrosis.

Secondary pulmonary TB may heal with fibrosis or progress as in this case. Progressive pulmonary TB sees erosion and expansion of the infectious lesion into adjacent lung parenchyma. This leads to evacuation of the caseous centre leading to fibrous cavitation. Erosion of blood vessels can occur causing haemoptysis. Post treatment of TB the tissue heals by fibrosis but does not recover the pulmonary architecture.

TB diagnosis is usually made with a clinical history and chest x-ray and multiple sputum cultures. Mantoux skin tuberculin test and serum interferon gamma release assay may also be used to help screen for infection. Biopsies may be taken of suspected infection site for culture to assist diagnosis. Treatment involves prolonged courses of multiple antibiotics, which depend on the antibiotic resistance of the infecting mycobacterium.

Tuberculosis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02810

Clinical History

A 37-year old female presents with increasing thoracic back pain. She has a history of untreated human immunodeficiency virus (HIV) infection and pulmonary tuberculosis. History revealed ongoing low-grade fevers, chills and weight loss. Examination revealed a cachexic patient with tender thoracic vertebrae at multiple levels. Blood test showed an elevated serum calcium and erythrocyte sedimentation rate. X-ray of her spine showed lytic areas in the thoracic vertebrae. During her hospital admission, she developed urosepsis and died.

Pathology

The specimen is a portion of the patient’s thoracic vertebral column that has been sawn longitudinally and mounted to display the cut surface of 7 thoracic vertebrae. In all vertebrae, there are osteolytic areas, varying from 1 to 12 mm in diameter, which contain caseous degenerative material* (mostly now lost) and are surrounded by a thin zone of dense bone. The tuberculous inflammatory process has extended into one of the intervertebral discs, and has also spread outside the vertebral bodies to form collections of caseous material beneath the anterior longitudinal ligament. This is an example of tuberculous mycobacterial osteomyelitis of the vertebral column with paravertebral extension, also known as Pott’s Disease.

Further Information

Tuberculosis (TB) is a chronic pulmonary and systemic infectious disease caused by Mycobacteria tuberculosis. Transmission most commonly occurs via inhalation of aerosolized droplets of M. tuberculosis. Risk factors for contracting TB include being an inhabitant of a ‘developing’ country where the disease may be endemic, immunosuppression (e.g. HIV, steroid use, anti-TNF use and diabetes), chronic lung disease (e.g. silicosis), alcoholism, and generalized malnutrition. After initial pulmonary infection of M. tuberculosis clinical manifestation varies. In 90% of individuals with an intact immune system, they enter an asymptomatic latent infection phase. This latent TB may reactivate at any time in the patient’s life. In the other 10% of patients, especially in the immunocompromised population, they develop primary disease, which is immediate active TB infection. Manifestations of primary TB include pulmonary infection symptoms (e.g. consolidation, effusion and hilar adenopathy) and extra pulmonary symptoms - lymphadenopathy, meningitis and disseminated miliary TB. Secondary tuberculosis occurs when there is reactivation of a previous latent TB infection. Around 10% of latent TB will reactivate usually during periods of weakened host immunity. Typical symptoms of reactivation are cough, haemoptysis, low grade fever, night sweats and weight loss. Osseous infection occurs 1-3% of patients with TB infection. There is a higher incidence of developing bone disease in patients from developing countries and immunocompromised patients. The TB usually spreads haematogenously from the site of active disease. Pott’s disease accounts for 40% of TB bone infections. The infection is destructive eroding vertebral discs and vertebrae

leading to compression fractures, which may cause symptoms of cord or nerve root compression. Symptoms include pain at the site of disease, fevers, chills, weight loss, symptoms of compression and spinal deformities, such as kyphosis and scoliosis. TB diagnosis is usually made with a clinical history and chest x-ray and multiple sputum cultures. Mantoux skin tuberculin test and serum interferon gamma release assay may also be used to help screen for infection. Biopsies may be taken of suspected infection site for culture to assist diagnosis.

Right lung miliary tuberculosis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02758

Clinical History

A 74-year old male presented with increasing shortness of breath and haemoptysis. Further history reveals 20kg weight loss in 6 months, night sweats and a chronic cough. He has recently moved from a country where TB is endemic. On examination, he has hypoxic and tachypnoea and has bilateral crepitations throughout his lung fields and a dull left lung base on percussion. His quantiferon gold blood test is positive. His chest x-ray showed bilateral small nodular deposits and a left basal pneumonia. He died from respiratory failure soon after admission.

Pathology

The right lung has been sliced longitudinally and mounted to display the cut surface. The bronchi and bronchioles are mildly ectatic. Scattered throughout the entire lung parenchyma are large numbers of small, pale yellows nodules less than 1 mm in diameter. Similar tiny subpleural nodules are seen on the surface of the visceral pleura. The nodules are tubercles. This is miliary tuberculosis, so-called due to the resemblance of the nodules to millet seeds.

Further Information

Tuberculosis (TB) is a chronic pulmonary and systemic infectious disease caused by Mycobacterium tuberculosis. Transmission most commonly occurs via inhalation of aerosolized droplets of this pathogenic bacteria, first described by Robert Koch (1882). Risk factors for contracting TB include being an inhabitant of a developing country where the disease is endemic, immunosuppression (e.g. HIV, steroid use, anti-TNF use and diabetes), chronic lung disease (e.g. silicosis), alcoholism and malnutrition.

After initial pulmonary infection of M. tuberculosis clinical manifestation varies. In 90% of individuals with an intact immune system they enter an asymptomatic latent infection phase. This latent TB may reactivate at any time in the patient‘s life. In the other 10% of patients, especially in the immunocompromised, they develop primary disease which is immediate active TB infection. Manifestations of primary TB include pulmonary infection symptoms (e.g. consolidation, effusion and hilar adenopathy) and extra pulmonary symptoms including lymphadenopathy, meningitis and disseminated miliary TB.

Secondary tuberculosis occurs when there is reactivation of previous latent TB infection. Around 10% of latent TB will reactivate usually during periods of weakened host immunity. Typical symptoms of reactivation are cough,

haemoptysis, low grade fever, night sweats and weight loss.

Miliary TB occurs when the mycobacterium erodes into a pulmonary vein and seeds elsewhere. The organism can circulate back to the lung and disseminate thought the lung parenchyma as in this case. Systemic miliary tuberculosis can occur when the mycobacterium is disseminated through the arterial system. The TB can then deposit in any organ but most commonly in the liver, bone marrow, spleen and adrenal glands.

The immune response against TB is mediated via TH1 cells stimulate alveolar macrophages to attack the mycobacteria. These macrophages surround the infection forming a granuloma surrounding a central area of ‘caseous’ (white cheese-like) necrosis. Secondary pulmonary TB may heal with fibrosis or progress as in this case. Progressive pulmonary TB sees erosion and expansion of the infectious lesion into adjacent lung parenchyma. This leads to evacuation of the caseous centre leading to fibrous cavitation. Erosion of blood vessels can occur causing haemoptysis. Post treatment of TB the tissue heals by fibrosis but does not recover the pulmonary architecture. TB diagnosis is usually made with a clinical history and chest x-ray and multiple sputum cultures. Mantoux skin tuberculin test and serum interferon gamma release assay may also be used to help screen for infection. Biopsies may be taken of suspected infection site for culture to assist diagnosis. Treatment involves prolonged courses of multiple antibiotics, which depend on the antibiotic resistance of the infecting mycobacterium.

Hepatic duct calculi and Obstructive Biliary Cirrhosis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02785

Clinical History

An 85-year old male presented with urinary retention due to benign prostatic hypertrophy. On admission it was noted that he was jaundiced with cholestatic derangement of his liver function tests. He underwent a transurethral prostate resection but died from pneumonia 5 days post-operative.

Pathology

The specimen is a slice of liver mounted to display the cut surface. The capsule is slightly thickened and the liver substance has a finely nodular appearance. Intrahepatic bile ducts are dilated. When the posterior or inferior surface is viewed an irregular pigmented calculus, 10 mm in diameter, is seen impacted in a distended hepatic duct. Another smaller calculus 3 mm in diameter has been dislodged. This specimen represents an example of secondary biliary cirrhosis due to large duct obstruction from hepatic calculi.

Further Information

Hepatolithiasis is characterised by the presence of intrahepatic gallstones. These calculi can lead to cholangitis, progressive hepatocyte atrophy and destruction, and an increased risk of cholangiocarcinoma. It is common in East Asia but rare in Western countries. There is no difference in incidence between genders. The stones are most commonly made up of pigmented calcium bilirubinate stones.

These stones cause intrahepatic bile duct obstruction. Proximal to the obstructing stone distension and dilation of the bile ducts is evident. There is also bile duct proliferation at the portalparenchymal interface with stromal oedema and infiltrating neutrophils, indicating an acute-chronic inflammation. If untreated this inflammation leads to periportal fibrosis and eventually obstructive biliary cirrhosis. Microscopic appearance would show feathery degeneration of periportal hepatocytes, cytoplasmic swelling often with Mallory Denk bodies (i.e. an inclusion found in the cytoplasm of liver cells with twisted-rope appearance caused by damaged intermediate filaments within the hepatocytes) and bile infarcts from extravasated bile. Chronic inflammation can lead to biliary dysplasia which may develop into cholangiocarcinoma. Patients may present with repeated cholangitis, intermittent abdominal pain, jaundice or frequently no symptoms. Treatment is usually surgical removal of the calculi.

Cholelithiasis (Gallstones)

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02776

Clinical History

A middle-aged woman was investigated for recurrent bouts of epigastric pain. Endoscopy failed to reveal any peptic ulceration. A cholangiogram demonstrated a non-functioning gallbladder. She died from a myocardial infarction Unfortunately, the patient dies at a later stage after this procedure from a myocardial infarction".

Pathology

The specimen is a portion of liver with attached gallbladder, which has been opened to display six large faceted mixed calculi. This is an example of cholelithiasis (gallstones).

Further Information

Gallstones contain a mixture of cholesterol, calcium salts, bilirubin, proteins, and mucin. There is a high prevalence in fair-skinned populations. Risk factors are age (greater than 50) and female sex, along with genetic factors1, pregnancy, diabetes mellitus and dyslipidaemia. Lifestyle factors, such as rapid weight loss and certain medications (e.g. erythromcyin, ampicillin, octreotide, cephalosporin), can also promote gallstone formation. Gallstones may be asymptomatic or may present with a spectrum of disease ranging from uncomplicated biliary colic through to infection, cholecystitis, pancreatitis or gallstone ileus. The typical symptoms include bouts of epigastric or right upper quadrant pain, sometimes associated with eating, and often with sweating, nausea and vomiting. Pain is usually caused by the gallbladder or biliary tract contracting forcefully against a stone, thereby causing increased pressure in the gallbladder, and pain. The risk of developing complications of gallstones is approximately 2-3 percent per year once biliary colic develops. Diagnosis is generally via transabdominal ultrasound, which has largely replaced oral cholecystography studies. Cholescintigraphy (HIDA Scan) can be used distinguish biliary colic from acute cholecystitis. Treatment of attacks are initially with simple analgesia, and subsequently definitive management usually includes elective laparoscopic cholecystectomy. Very severe cases can be life threatening, but deaths from gallstone disease are rare. It should be noted that epigastric pain can also be caused by myocardial ischemia, particularly in females, who do not necessarily present with the classic 'left shoulder tip pain' of an acute myocardial infarct. Therefore, should gallbladder stones be excluded in a patient presenting with epigastric pain, an ECG should always be performed, to help rule out cardiac disease.

Tertiary Syphilis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02774

Clinical History

A 66-year old male presents with postprandial epigastric pain. Of note, he is deaf and non-verbal. On examination, he has a tender epigastrium and several nodular tender lesions over his forehead and scalp. Blood tests show a low haemoglobin, impaired hepatic function and are positive for anti-treponemal antibodies. After admission, he has a large gastrointestinal bleed, and dies despite intervention.

Pathology

This specimen is the vault of the patient‘s skull. On the external surface, there are multiple circumscribed necrotic lesions in the parasagittal area to the left of the midline. The lesions are brown in colour and measure up to 3-4 cm in maximum diameter. The lesions have eroded the outer table of the skull and the adjacent periosteum is thickened with a fibrinous inflammation.

Further Information

Syphilis is a chronic infection caused by the spirochete bacterium Treponema pallidum. Sexually transmitted infection is most common, but it may also be congenitally-acquired by transplacental transmission of the bacteria. Those who have the higher risk of infection, include those of a sexually active age, intravenous drug users, HIV-infected patients and homosexual males. Syphilis infection rates decreased significantly with the introduction of penicillin in 1943, which remains the main treatment today. However, the infection rate has been increasing since the early 2000’s.

Syphilis is divided into three stages with distinct clinical and pathological features with characteristic proliferative endarteritis affecting small vessels. Primary syphilis occurs usually 3 weeks after initial infection. This manifests typically as a single, painless and erythematous lesion called a chancre at the site of inoculation. The syphilis spreads throughout the body from this chancre, which then heals spontaneously after 3 to 6 weeks.

Secondary syphilis occurs weeks to a few months after the primary chancre resolves in 75% of untreated patients. During this stage patients commonly have generalised symptoms, such as malaise, lymphadenopathy and skin rashes. Palmar/plantar rashes are the most frequent site but rashes can also be diffuse. These rashes can be maculopapular, scaly or pustular. Condylomata lata are elevated gray plaques that arise on the moist mucous

membranes, such as oral or genital regions. Other less common manifestations include hepatitis, gastrointestinal invasion or ulceration and neurosyphilis - discussed below.

Tertiary syphilis has three main characteristics: cardiovascular syphilis, neurosyphilis and gummatous syphilis. These occur after a latent period of 5 years or more in one third of untreated patients.

Cardiovascular syphilis involves an aortitis for which the exact pathophysiology is unclear. The vasculitis involves the ascending thoracic aorta leading to progressive dilation of the aortic root which can cause aortic valve insufficiency and aneurysms. Clinical manifestation usually happens 15-30 years post initial infection.

Neurosyphilis can be symptomatic or asymptomatic. It occurs in 10% of untreated patients. Early clinical manifestations include headaches, meningitis, hearing loss and ocular involvement, most commonly uveitis, causing vision loss. Late manifestations can occur up to 25 years post initial infection. Main features are meningovascular neurosyphilis, paretic neurosyphilis and tabes dorsalis. Meningovascular involvement involves chronic meningitis and endarteritis which can lead to strokes. Tabes dorsalis is caused from degeneration of the posterior columns within the spinal cord. This causes loss of proprioception, ataxia, loss of pain sensation, and loss of reflexes. Paretic neurosyphilis is caused by invasion and damage of the brain parenchyma, most commonly the frontal lobes. This leads to progressive cognitive impairment and mood disturbance.

Gummatous syphilis is characterised by the formation of nodular lesions most commonly bone, skin and mucosa of the upper airway and mouth called gummas. Gummas can occur anywhere including viscera. The formation of gummas is rare but occurs more frequently in HIV-infected patients. Skeletal involvement causes pain and pathological fractures.

Osteosarcoma of femur

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02772

Clinical History

On examination, there was a palpable tender swelling above the right knee. Blood test showed a raised Alkaline Phosphatase level. A knee x-ray showed periosteal reactive changes in the distal femur suspicious for a bone malignancy. The patient then underwent staging CT and MRI evaluation of the right leg. He underwent adjuvant chemotherapy prior to resection of his right femur. He made a full recovery.

Pathology

The specimen is the patient’s excised distal femur. On the cut surface, there is a large pale infiltrating tumour, 10 cm in greatest diameter, extending through the periosteum near the articular surface. This is an osteosarcoma of the femur.

Further Information

Osteosarcomas are a malignant tumour of bones that are characterised by the production of osteoid matrix or immature bone. It is the most common primary malignancy of bone. Most occur in the distal femur with the tibia and humerus being the most frequent sites affected. Men are more commonly affected than women. They occur in a bimodal age distribution, with most occurring in children and adolescents under 20 years of age and the second peak occurring in older adults over 60.

Secondary osteosarcomas are more common in older patients. Secondary osteosarcomas occur in patient’s bones with predisposing conditions such as Paget‘s disease, bone infarcts and previous irradiation. Mutations in tumour suppressors and oncogenes, such as RB, TP53 and INK4a have been shown in osteosarcomas.

Osteosarcomas usually present with painful, enlarging masses. Pathological fractures can also be the first presenting complaint. Constitutional symptoms are usually not present. Alkaline phosphatase and lactate dehydrogenase may be elevated on blood tests. X-rays can show features of bone destruction, a mass or signs of a periosteal reaction, such as a sunburst appearance or triangular shells of reactive bone (Codman‘s Triangle). MRI of the affected bone is used to evaluate local staging of the tumour while CT of the body is used to evaluate for distant spread. The tumour may be biopsied in some cases.

The lungs are the most common site for distant metastases followed by the bones and brain. Treatment involves neoadjuvant chemotherapy followed by surgery. 5-year survival rate for localised osteosarcoma is 60-70% but this drops to <20% in patients with distant metastases.

Adrenal haemorrhage / WaterhouseFriderichsen Syndrome

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02787

Clinical History

A 77-year old male presented with a 3-day history of abdominal and flank pain with fevers and rigors. He was 2 weeks post-operative from a duodenal ulcer repair surgery. He was hypotensive, hyperkalemic and hyponatremic with a purpuric rash. His blood cultures grew Escherichia coli. He failed to respond to treatment, and died soon after admission due to septic shock.

Pathology

The combined kidney and adrenal gland have been mounted, in order to display the cut surfaces. Extensive haemorrhage has occurred into the adrenal medulla, and there is some extravasation of blood into the periadrenal fat. This is an example of adrenal haemorrhage in the setting of severe septic shock also known as ‘Waterhouse-Friderichsen’ syndrome.

Further Information

Waterhouse-Friderichsen syndrome is characterised by adrenal haemorrhage cause by overwhelming sepsis leading to hypotensive shock, disseminated intravascular coagulation (DIC) and adrenocortical insufficiency. It most commonly occurs in children and rarely in adults. Neisseria meningitis causes over 80% of cases of adrenal haemorrhage. Other organisms that may cause it include Streptococcus pneumoniae, pseudomonas aeruginosa, Escherichia coli, Klebsiella pneumoniae, Haemophilus influenzae and staphylococci. The exact cause of the haemorrhage is unclear. It may be due to bacterial seeding of the adrenal vessels, to the DIC or to endothelial dysfunction from inflammatory mediators or bacterial toxins. Adrenal haemorrhages can occur bilaterally. The haemorrhage begins in the medulla and extends outwards to the cortex, and may extend into the periadrenal fat. This leads to adrenal gland failure. Patients present with rapidly progressive septic shock, diffuse purpuric skin rash and adrenal insufficiency crisis. Treatment includes supportive therapy, intravenous antibiotics directed against the cultured organisms, and steroid therapy. The mortality rate is over 50%.

Uterus Bicornuate Unicollis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02803

Clinical History

A 36-year old female has a large postpartum hemorrhage after the birth of her 4th child by breech delivery. Her previous 3 children have all been breech deliveries, and no miscarriages. She has a history of intermittent mild abdominal pain. The obstetricians were unable to stop the bleeding and performed an emergency radical hysterectomy and bilateral salpingo-oophorectomy. The patient and baby made a full recovery.

Pathology

This hysterectomy specimen is of a bicornuate uterus, fallopian tubes and ovaries; sliced coronally and mounted to display cut and external surfaces. Both uterine bodies are equal in size and share a common cervical canal. A few small cysts are present in the cervix.

Further Information

A bicornuate uterus is a congenital uterine malformation where the uterus fundus has an indentation of more than 1 cm. The vagina and cervix are usually normal. There is generally one cervix. There are usually two moderately separate endometrial cavities. Bicornuate uterus develop during embryogenesis when the Mullerian ducts only partially fuse instead of completely fusing. The risk of developing these malformation increases if exposed to diethylstilbestrol (DHS) in-utero, a synthetic estrogen previously used to prevent pregnancy loss. The karyotype of most women with uterine malformations is 46,XX. These malformations are present in around 0.5% of women, although the actual number may be greater as not all women are symptomatic. Symptoms may include pelvic pain (cyclic and non-cyclic), abnormal uterine bleeding and discharge and urinary tract infection (UTI). In pregnancy bicornuate uterus may lead to recurrent miscarriage, pre-term labour, fetal growth restriction, fetal malpresentation and placenta previa. Malpresentation of the fetus leads to increased need for caesarean section. There is increased risk postpartum or placental retention and postpartum hemorrhage. Bicornuate uterus is usually diagnosed with pelvic ultrasound scan. MRI scanning is rarely used to consolidate the diagnosis. Most cases do not require any treatment.

Liver cirrhosis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02767

Clinical History

This 63-year old man had several previous admissions to hospital for bleeding from oesophageal varices. On admission he was noted to be mildly febrile, jaundiced with severe flapping tremor of his hands. He had a constellation of other findings included ascites, prominent periumbilical veins and haemorrhoids. He had a massive haematemesis and died.

Pathology

A slice of liver has been mounted to display the cut surface, which shows multiple well demarcated nodules varying in size from 1 to 7 mm in diameter. The external surface of the liver is also nodular and irregular. This is an example of cirrhosis of the liver, with a mixed micro- and macro- nodular pattern and marked fatty change.

Further Information

The commonest cause of cirrhosis and fatty change is chronic alcoholism.

Uterine Leiomyoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02806

Clinical History

A 30-year old female presents with inability to conceive. She also reports a history of intermittent pelvic discomfort, menorrhagia and painful periods. On examination a pelvic mass was palpable. All of her blood tests were within normal ranges. A pelvic ultrasound showed a hypoechoic mass within the myometrium of her uterus. She went for hysteroscopic myomectomy but unfortunately complications meant her surgery was converted to an emergency hysterectomy. She made a full recovery post-op.

Pathology

The specimen includes the cervix, body and fundus of the uterus. The uterus, which is of normal size, has been cut in the sagittal plane. A large ovoid mass approximately 4cm x 2cm protrudes into the uterine cavity and extends as far inferiorly as the opening of the cervix. It originates from the posterior aspect of the uterus. The cervical canal is clearly visible.

Further Information

Uterine leiomyomas , also called fibroids, are the most common pelvic tumours in females. They are present in almost 25% of reproductive females. They are benign tumours arising from smooth muscle and fibroblasts of the myometrium. They usually involve the myometrium of the uterine body. Rarely they can affect the lower uterus or cervix. Leiomyomas can occur as single lesions or multiple and can grow very large. There are rare variants, which can extend and spread distally but are still considered benign: e.g. the benign metastasizing leiomyoma, which commonly spread to the lining; or disseminated peritoneal leiomyomatosis, which appears on the peritoneum covering the uterus Risk factors for developing fibroid include being of reproductive age, being a black woman and early menarche. Higher parity has been found to be protective. Most leiomyomas have normal karyotypes but there are some which show mutation in the HMG genes. Transformation into malignant leiomyosarcoma is very rare. Common symptoms of uterine fibroids include abnormal vaginal bleeding, pelvic pain, dyspareunia, dysmenorrhea and symptoms of pelvic structure compression such as urinary symptoms or venous compression symptoms. Leiomyomas can decrease fertility and in pregnant females increase the rate of early pregnancy loss, fetal malpresentation and postpartum hemorrhage. Pelvic ultrasound is usually used to diagnose leiomyomas. CT and MRI scans are rarely used to diagnose. Leiomyomas can grow but can also regress. Treatment is reserved for persistently or severely symptomatic fibroids. Hormonal treatment may be used to regulate irregular menstrual bleeding symptoms. Surgical treatments include myomectomy (removal of fibroids from myomectomy), hysterectomy, myolysis (thermal ablation of leiomyoma) and uterine artery ablation/embolisation.

Horseshoe Kidney

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02790

Clinical History

This specimen was found during a routine post-mortem of a 56-year-old male who died of rheumatic heart disease.

Pathology

The kidney is 12 cm in length and the two parts are fused at the lower pole forming this horseshoe-like shape. The ureters can be seen emerging from the hilum on the anterior aspect of the 3D print. The kidney is bisected in the horizontal plane which is evident on the posterior aspect. There is persistent foetal lobulation. The renal pelvis is characteristically antero-medial positioned with the ureters travelling anterior to the fused lower poles or isthmus of the kidney.

Further Information

Horseshoe kidneys are the commonest renal developmental abnormality. This anomaly is twice as common in males as in females. They are found in about 1 in 500 to 1000 post-mortem examinations. Most cases are sporadic but may be associated with some chromosomal anomalies, such as those resulting in Downs and Edwards Syndromes as well as non-aneuploidic anomalies, such as VACTERL* association. In 90% of cases an isthmus of renal tissue connects the lower poles of the kidneys across the midline, forming a horseshoe shape. Fusion of the upper lobes is rare. The renal pelves that drain the two halves of the horseshoe kidney are directed more anteriorly than normal and there is some angulation of ureters as they cross the isthmus. This malformation is usually asymptomatic and picked up incidentally on routine ultrasound or CT scans. These kidneys usually function normally. There is an increased incidence of urinary calculi, possibly due to angulation of the ureters and to the resulting stasis. There is an increased risk of hydronephrosis usually from pelvi-ureteric junction obstruction. There is a higher incidence of urinary tract infections mainly due to vesico-ureteric reflux. A higher incidence in some forms of renal malignancies (e.g. transitional cell carcinoma and Wilms tumours) has also been described.

Gall Stone Ileus

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02779

Clinical History

A 54-year-old man presented to hospital with 12 hours of severe colicky pain, nausea and vomiting. On history, he was noted to have had a 3-year history of intermittent right subcostal pain for which he had not seen a doctor. He was diagnosed as having an acute bowel obstruction and a laparotomy was performed.

Pathology

This segment of small bowel has been opened to display a large pigmented, ovoid gall stone with a roughened surface. This is an example of gall stone ileus.

Further Information

Gallstone disease is an uncommon cause of bowel obstruction - accounting for only 0.5% of cases with a preponderance for older and female patients. It most commonly secondary to biliary-enteric fistulae (can be to proximal or distal portions of bowel) but can also occur after sphincterotomy. Stones are usually over 2-2.5cm and 70% impact in the ileum, while others obstruct at sites of stricture/narrowing. History may include episodic obstructive symptoms. Diagnosis is confirmed either radiologically (often on CT scan) or at time of removal. Rigler’s triad is typical for gallstone ileus and consists of: (1) small bowel obstruction, (2) a gallstone outside the gallbladder, and (3) air in the bile ducts (pneumobilia) seen on imaging and gallstone presence on plane XR. Treatment usually surgical with removal of the obstructing stone, closure of the fistula and cholecystectomy to stop recurrence. These procedures may need to be staged.

Carcinoma of Larynx

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02750

Clinical History

A 47-year-old male presents with a 13-month history of dysphonia and odynophagia at the level of his thyroid cartilage. He has a significant smoking history. Investigations revealed a laryngeal tumour. He received radiotherapy to the tumour followed by a laryngectomy. Six months later pulmonary metastases were discovered, and he subsequently died.

Pathology

This is the patient’s laryngectomy specimen. The larynx has been sliced open and is viewed from the posterior aspect. There is significant right vocal cord distortion by an irregular ulcerating tumour. Mucosal congestion is also noted. Histologically this was a well differentiated squamous cell carcinoma (SCC).

Further Information

Over 95% of laryngeal cancers are SCC. The tumour usually develops on the vocal cords but may occur above or below the cords, on the epiglottis, aryepiglottic folds or in the pyriform sinuses. The cancer usually begins as carcinoma in situ, progressing to ulcerated and fungating carcinoma with continued exposure to carcinogens.

The greatest risk factors for developing cancer of the larynx are tobacco smoke and alcohol consumption. Human Papilloma Virus (HPV) infection, asbestos exposure and irradiation have also shown increased incidence. Males are affected more than females. It most frequently presents in the 6th decade of life.

Laryngeal cancer may spread by invading into surrounding structures, via lymphatics usually to local cervical nodes or haematogenous metastasis most commonly to the lungs. Common symptoms on presentation include dysphonia, dysphagia, odynophagia, globus and cough. Less commonly haemoptysis, stridor, dyspnoea and halitosis may be described. Treatment varies on the stage of the disease. Smoking and alcohol cessation are important for all disease stages. In early disease laryngeal preservation treatments may include laser therapy, microsurgery and radiotherapy. Later stage disease treatments may involve a combination of laryngectomy, radiotherapy and chemotherapy

Carcinoma of Larynx

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02749

Clinical History

A 74-year old male presented with a 2-months history of dysphagia, dysphonia and weight loss. He had a history of heavy alcohol consumption and smoked 40 cigarettes per day for 40 years. Investigation discovered a laryngeal tumour. He received radiotherapy but his tumour reoccurred. He died 9 months after his initial presentation.

Pathology

The specimen consists of tongue, pharynx, larynx, oesophagus and trachea and has been mounted in the coronal plane. The oesophagus and trachea have been opened from the posterior aspect. There is a 5 x 4 x 2 cm fungating carcinoma evident extending into both pyriform fossae. The surface of the tumour is irregular with shaggy areas of necrosis. The tumour has arisen from the larynx and involves both vocal cords, the left aryepiglottic fold and both pyriform fossae.

Further Information

Over 95% of laryngeal cancers are squamous cell carcinomas. The tumour usually develops on the vocal cords but may occur above or below the cords, on the epiglottis, aryepiglottic folds or in the pyriform fossae. The cancer usually begins as squamous cell carcinoma in situ, progressing to ulcerated and fungating invasive carcinoma with continued exposure to carcinogens. The greatest risk factors for developing cancer of the larynx are tobacco smoke and alcohol consumption. Human Papilloma Virus (HPV) infection, asbestos exposure and irradiation have also been shown to be associated with increased incidence of head-and-neck squamous cell carcinoma (HNSCC). Males are affected more than females. It most frequently presents in the 6th decade of life. Laryngeal cancer may spread by invading into surrounding structures, via lymphatics usually to local cervical nodes, or haematogenous metastasis most commonly to the lungs. Common symptoms of HNSCC on presentation include dysphonia, dysphagia, odynophagia, globus and cough. Less commonly haemoptysis, stridor, dyspnoea and halitosis may be described.

Treatment varies on the stage of the disease. Smoking and alcohol cessation are important for all disease stages. In early disease laryngeal preservation treatments may include laser therapy, microsurgery and radiotherapy. Later stage disease treatments may involve a combination of laryngectomy, radiotherapy and chemotherapy.

HPV-related HNSCC have better outcomes than those non-HPV positive tumours. HPV-vaccination programmes have been introduced in several countries, including Australia and the UK, for both boys and girls, in order to reduce their risk for HNSCC.

Carcinoma of Pyriform Fossa

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02748

Clinical History

A 60-year old male presents with a 6-week history of globus (i.e. the feeling of a lump in his throat) and dysphonia. On examination, he had enlarged cervical lymph nodes. Investigations discovered a laryngeal tumour. He underwent a laryngectomy and a cervical lymph node dissection. He made a full recovery

Pathology

The specimen is the amputated larynx viewed from behind. It shows an irregular fungating tumour arising from the left pyriform fossa. There is distortion and oedema of the laryngeal tissues. Histologically, this was a squamous cell carcinoma.

Further Information

Over 95% of laryngeal cancers are squamous cell carcinomas (SCC). The tumour usually develops on the vocal cords but may occur above or below the cords, on the epiglottis, aryepiglottic folds or in the pyriform sinuses. The cancer usually begins as carcinoma in situ, progressing to ulcerated, fungating invasive carcinoma with continued exposure to carcinogens.

The greatest risk factors for developing cancer of the larynx are tobacco smoke and alcohol consumption. Human Papilloma Virus (HPV) infection, asbestos exposure and irradiation have also shown increased incidence. Males are affected more than females. It most frequently presents in the 6th decade of life. Laryngeal cancer may spread by invading into surrounding structures, via lymphatics usually to local cervical nodes or haematogenous metastasis most commonly to the lungs. Common symptoms on presentation include dysphonia, dysphagia, odynophagia, globus and cough. Less commonly haemoptysis, stridor, dyspnoea and halitosis may be described. Treatment varies on the stage of the disease. Smoking and alcohol cessation are important for all disease stages. In early disease laryngeal preservation treatments may include laser therapy, microsurgery and radiotherapy. Later stage disease treatments may involve a combination of laryngectomy, radiotherapy and chemotherapy. HPV-related HNSCC have better outcomes than those non-HPV positive tumours. HPV-vaccination programmes have been introduced in several countries, including Australia and the UK, for both boys and girls, in order to reduce their risk for HNSCC.

Bronchopneumonia

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02756

Pathology

The specimen is a parasagittal section of the left lung. There are patchy regions of focal consolidations and discolorations caused by congested and hyperaemic lung tissue distributed within both lobes; however, the upper lobe is more severely affected. The consolidation appears to be concentrated around the bronchioles, which are ectatic. The costal (pleural) surface of the upper lobe is especially discoloured.

Further Information

Bronchopneumonia is a form of pneumonia characterized by inflammatory exudate within the intra-alveolar space resulting in consolidation that affects a large and continuous area of the lobe of a lung. It is one of the two anatomic classifications of pneumonia (the other being lobar pneumonia). The affected regions in this case shows classical focal red hepatization or consolidation in focal regions, which is due to vascular congestion with extravasation of red cells into alveolar spaces, along with increased numbers of neutrophils and fibrin. The filling of airspaces by the exudate leads to a gross appearance of solidification, or consolidation, of the alveolar parenchyma.

Bronchopneumonia is a subtype of pneumonia. It is the acute inflammation of the bronchi, accompanied by inflamed patches in the peribronchial and peribronchiolar lobules of the lungs.

It is often contrasted with lobar pneumonia but in clinical practice the types are difficult to apply, as the patterns usually overlap. Bronchopneumonia (sometimes called lobular) often leads to lobar pneumonia as the infection progresses to affect an entire lobe. The same organism may cause one type of pneumonia in one patient, and another in a different patient.

Bronchopneumonia is usually a bacterial pneumonia rather than being caused by viral disease and is more commonly a hospital-acquired pneumonia than a community-acquired pneumonia, in contrast to lobar pneumonia.

Acute Bacterial Endocarditis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02744

Clinical History

A 15-year old boy with cough and sputum developed a hectic (spiking) fever and chest pain a few days before being admitted in a comatosed condition. Examination revealed an early diastolic murmur at the aortic area, which radiated down the left sternal edge. He deteriorated very quickly and died, despite antibiotic chemotherapy. Blood cultures grew Staphylococcus aureus.

Pathology

This small heart displays the left ventricle and associated valves. The non-coronary cusp of the aortic valve is ulcerated and perforated and has friable vegetations attached. Immediately below this cusp a perforation extends into the right atrium just above the tricuspid valve (see back of specimen. The other aortic cusp is also thickened. This is an acute bacterial endocarditis with aortic cusp and atrioventricular perforations.

Further Information

Acute bacterial endocarditis is a form of infective endocarditis. Endocarditis due to fungal infections can also occur although they are rare.

In normal circumstances, the endothelial lining of the heart and valves is relatively resistant to infection by most bacteria or fungi. Therefore, in order for infective endocarditis to occur, there needs to be initial damage or injury to the endocardial tissue. This often results in aggregation of platelets and fibrin, which then become infected, resulting in vegetation formation (i.e. an infective nidus). Staphylococcus aureus, however, is highly virulent and can sometimes infect normal heart valves.

After the initial platelet-fibrin aggregation, there is further activation of the coagulating system via the extrinsic clotting pathway and initiation of the inflammatory response via monocytes, resulting in further growth of the vegetation/thrombus. The microbial growth tends to occur within the fibrin matrix, which makes it difficult for the immune responses to eradicate the infection. An additional problem is that these infected thombi can also embolise causing distant sites of infection in smaller capillaries (e.g. in the kidney).

Risk factors for developing an infective endocarditis include valvular heart disease, such as previous rheumatic heart disease, congenital heart disease (e.g. ventricular septal defect or bicuspid aortic valve), prosthetic heart valves or any previous invasive cardiac procedures. Anti-thrombotic medications – e.g. heparin or aspirin – may have to be administered in patients at risk. Diagnosis is initially made by clinical examination followed by pathology (blood cultures) and diagnostic imaging. Transthoracic echocardiogram is often first line, followed by transoesophageal echocardiogram. Treatment includes antimicrobial therapy, anti-coagulants, and in some complicated cases, surgical intervention such as valve surgery

Ruptured Thoracic Aortic Aneurysm

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02746

Pathology

The heart displays both ventricles from the posterior aspect. There is a prominent saccular dilatation of the thoracic ascending aorta, which shows several atherosclerotic plaques and posteriorly is seen to be ruptured (identified by the dark staining). Both ventricles are hypertrophied. The coronary arteries together with the aortic and tricuspid valves are normal. This is an example of a ruptured aneurysm of the ascending aorta.

Further Information

The dilation of the ascending aorta is a common incidental finding on transthoracic echocardiography performed for unrelated indications.

The thoracic aorta is divided into 3 parts: ascending, arch and descending. The ascending aorta originates beyond the aortic valve and ends right before the innominate artery (brachiocephalic trunk). It is approximately 5 cm long and is composed of two distinct segments. The lower segment, known as the aortic root, encompasses the coronary sinuses and sinotubular junction (STJ). The upper segment, known as the tubular ascending aorta, begins at the STJ and extends to the aortic arch (innominate artery). More than 50% of thoracic aortic aneurysms are localized to the ascending aorta, which may affect either the aortic root or tubular aortic segment.

An aneurysm is defined as a localized dilation of the aorta that is more than 50% of predicted (ratio of observed to expected diameter ≥ 1.5). Aneurysm should be distinguished from ectasia, which represents a diffuse dilation of the aorta less than 50% of normal aorta diameter. The incidence of ascending thoracic aortic aneurysms is estimated to be around 10 per 100,000 person-years[1].

Reference: 1. Saliba et al. (2015). Int J Cardiol Heart Vasc. 6: 91–100.

Hypertrophic Subaortic stenosis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02739

Clinical History

A thin 42-year-old American tourist was found dead in his hotel bedroom. A coroner’s autopsy was performed.

Pathology

This is a longitudinal section through the heart displaying the left and right ventricles and interventricular septum. The outstanding abnormality is a grossly thickened interventricular septum and left ventricular hypertrophy. The aortic cusps that are visible appear unremarkable, as does the mitral valve. The ventricular septum is so large that it encroaches on the lumen of the left ventricle.

Diagnosis: Idiopathic hypertrophic subaortic stenosis, also known as hypertrophic cardiomyopathy.

Further Information

Subaortic stenosis is considered to be acquired rather than congenital and is suggested to result from an underlying defect in the architecture of the left ventricular outflow tract (LVOT). The defect may be such that the resulting turbulent blood flow leads to progressive thickening and fibrosis of the LVOT and the aortic valve. Progression of the disease will often lead to a hypertrophic cardiomyopathy secondary to the increased aortic pressure needed to be overcome by the left ventricle. Mild or moderate stenosis is often asymptomatic. As disease progresses and the stenosis becomes severe, symptoms such as exertional dyspnoea and syncope may become apparent. Investigation and subsequent diagnosis are often prompted by the presence of an ejection systolic murmur on physical examination. Echocardiography is used to confirm diagnosis. These days, definitive treatment of subaortic stenosis consists of surgical correction of the obstruction.

Multiple Polyposis Coli

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02764

Pathology

The specimens from this case consists of two segments of sigmoid colon. The mucosa of the bowel is studded with numerous sessile and pedunculated partially pigmented polyps up to 1.5 cm in maximum diameter. There is no macroscopic evidence of malignant change.

Note

Microscopically, the polyps are most commonly tubular adenomas (>75% have a tubular structure; also called adenomatous polyps). Less frequently, they are villous adenomas (>75% have villous morphology) or tubulovillous adenomas (25-75% villous). They can have varying grades of dysplasia. The histological appearances are identical to sporadic colonic adenomas.

Patients with familial adenomatous polyposis (FAP; a form of hereditary colon cancer syndrome involving the APC gene located on chromosome 5q21) are offered prophylactic colectomy because invasive adenocarcinoma is almost certain to develop in one or more of the polyps, usually about 15 years after the onset of the adenomatosis. This condition is transmitted as an autosomal dominant trait.

Right Ventricular Hypertrophy (RVH)

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02734

Clinical History

This 56-year old female suffered from emphysema and gave a 2-year history of increasing shortness of breath on exertion associated with recurrent attacks of bronchitis. On examination, she had a BP 160/90 mm Hg, pulse rate of 96 beats/min, and 6 cm of jugular venous congestion. The apex beat was impalpable, bilateral crepitations were heard and pitting oedema was present peripherally. Special investigations: ECG showed right heart strain pattern. Arterial blood examination showed respiratory acidosis. Despite treatment there was steady deterioration and death.

Pathology The specimen is of the external surface of the heart viewed from the anterior aspect. The right ventricle is greatly enlarged and hypertrophied. All appears to be normal otherwise. This is an example of right ventricular hypertrophy (RVH) in a patient with emphysema. **Further Information** RVH usually occurs due to chronic lung disease or structural defects in the heart. One of the most common causes of RVH is pulmonary hypertension (PH), which leads to increased pulmonary artery pressure. As the right ventricle tries to compensate for this increased pressure it changes its shape and size causing hypertrophy and right ventricular wall thickness. The global incidence of PH is 4 per 1M people: RVH occurs in approximately 30% of these cases. Common causes of PH include chronic obstructive pulmonary disease (COPD), pulmonary embolism, and other restrictive lung diseases. RVH also occurs in response to structural defects in the heart, such as tricuspid insufficiency, which allows the backward flow of blood into the ventricle. Other structural defects that lead to RVH include tetralogy of Fallot, ventricular septal defects, pulmonary valve stenosis, and atrial septal defects. RVH is also associated with abdominal obesity and high systolic blood pressure.

Metastatic carcinoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02754

Clinical History

This 47 year old woman was admitted with terminal carcinomatosis. On examination a hard liver and a right pelvic mass were palpable. She had been ill with constitutional symptoms for months and she finally sought medical attention. She was admitted for palliative care and died shortly afterwards.

Pathology

The left lung has been sliced longitudinally to display the cut surface. Multiple pale tumour nodules of varying size are scattered throughout the lung substance. Near the hilum several nodules are confluent. The hilar lymph nodes contain pale tumour tissue. Small tumour nodules can be seen beneath the thickened pleura. Histologically these were metastatic deposits of adenocarcinoma. At post-mortem there was an adeno-carcinoma of ovary, with metastases in lungs, heart, liver and pericardium.

Further Information

Pulmonary metastases are more common than primary lung cancer. Malignant disease arising anywhere in the body may spread to the lungs. Sarcomas usually metastasize by the bloodstream, and carcinomas spread either via the bloodstream or the lymphatic system or both.

Mesenteric Metastases from Cutaneous Malignant Melanoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02784

Clinical History

A 44-year-old man had a skin lesion on his back that grew slowly. At presentation at A&E several years later, he complained of bone pain, and had hepatomegaly and a pleural effusion. He died shortly afterwards.

Pathology

The specimen is a loop of small intestine mounted to display the mesentery, which contains numerous small dark brown, circumscribed nodules varying from pin head size to approximately 1 cm in diameter. Histology confirmed the diagnosis of metastatic melanoma.

Further Information

The most common form of melanoma is cutaneous melanoma, which develops from the pigment-producing cells known as melanocytes. In women, they most commonly occur on the legs, while in men they most commonly occur on the back. About 25% of melanomas develop from moles. Changes in a mole that can indicate melanoma include an increase in size, irregular edges, change in colour, itchiness or skin ulceration. Skin melanoma is associated with exposure to UV radiation in sunlight or tanning beds. Other risk factors for developing melanoma include fair complexion, presence of large number of melanocytic naevi (moles), severe sunburn as a child, and immunosuppression. It accounts for around 5% of all skin cancer diagnosis but has the highest mortality rate of all skin cancers. Melanomas typically occur in sun exposed areas as a pigmented lesion with irregular borders, variegated colour, an asymmetrical shape and which evolves with time. There are multiple mutations common in melanoma. Loss of cell cycle control gene from mutation in CDKN2A gene. Mutations in pro-growth signalling pathways, such as BRAF and PI3K mutations, are seen frequently in melanomas as well as mutations that activate telomerase, such as the TERT gene. Recognition that melanoma antigens activate host immune responses has led to promising immunotherapy, which enhances host T-cell identifying of these antigens. The most common sites for metastasis of melanoma are the lungs, liver, brain and bone as well as regional lymph nodes, and is highly dependent on the site of the primary tumour. Metastatic melanoma involving the gastrointestinal tract may present with anaemia, overt bleeding, pain, obstruction, or intussusception. The jejunum and ileum are the most commonly involved sites, followed by the colon, rectum, and stomach. Surgery has usually been reserved for patients with the above complications. The probability of metastatic spread from skin melanoma depends on the stage of the primary tumour, which is based on tumour depth, mitotic activity and ulceration of the skin as well as node and solid organ involvement. Diagnosis of melanoma is made with excisional biopsy. Investigation for bone metastasis is done using blood test (raised Alkaline phosphatase, calcium and LDH), and radiological investigations most commonly X-ray and CT but MRI and PET scans may also be used. Treatment depends on the stage or the tumour as well as the genetic and immune profiles of the melanoma. Treatment usually involves surgical resection, chemotherapy, targeted

therapies (e.g. BRAF inhibitors), immunotherapy , radiotherapy or more commonly a combination of treatments.

Metastatic Tumour in Lung from Primary Testicular Cancer

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02753

Clinical History

A 37-year old male patient presents with a 1-month history of lethargy, cough and weight loss. He had a history of an orchiectomy 18 months previous for a testicular tumour. Then 12 months post-op he underwent neck radiotherapy to treat metastasis. On admission, he became acutely dyspnoeic and hypoxic and died.

Pathology

This right lung specimen (and portions of 4 ribs) has been sliced longitudinally. There are numerous rounded tumour nodules evident in the lung parenchyma ranging from 5 to 30mm in diameter. The tumours are variegated in appearance with pale yellow and dark brown cut surfaces. One tumour is extending along the bronchus of the lower lobe forming a cast. Several nodules project from the pleural surface and some show central umbilication from necrosis and haemorrhage. This is an example of pulmonary metastases from a mixed germ cell testicular tumour, most likely choriocarcinoma arising in a malignant teratoma.

Further Information

Germ cell testicular tumours (GCT) are the most common tumours found in men. Average age of diagnosis is 30 year of age and are rarely diagnosed pre-puberty. Risk factors for development include cryptorchidism and a positive family history of GCT. Familial GCT increased risk can be linked to genes encoding for kinases, e.g. KIT and BAK.

They can be divided into two groups: seminomatous (resemble primordial germ cells) and non-seminomatous (resemble embryonic stem cells). Over one third of GCT are mixed GCT, with two or more GCT types in one mass. Many possible combinations of seminoma, teratoma, embryonal carcinoma, yolk sac tumor, and choriocarcinoma can be seen. Teratomas components are found in one third of mixed GCT. Elevated serum Alpha Fetoprotein and beta-hCG are produced by choriocarcinoma. Lymphatic spread involves the retroperitoneal para aortic nodes initially. Mediastinal and supraclavicular nodes may later become involved. The lungs are the most common site for haematogenous spread but the liver, brain or bones may also be affected.

Symptoms may include a painless testicular mass and haematospermia. Later symptoms of distant metastases may occur. Common symptoms of lung metastases include cough, dyspnoea, haemoptysis, recurrent infection.

Treatment depends on clinical stage but usually involves radical orchiectomy, chemotherapy and sometimes radiotherapy. More than 95% on early stage GCT can be cured.

Tracheoesophageal Fistula and Oesophagus Atresia

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02757

Clinical History

A 32-year-old female G3P0 (gravida 3, para 0‘ – i.e., has had two pregnancies, with neither of the embyros surviving to a gestational age of 24 weeks) presents in preterm labour at 25 weeks gestation. The GP had noted an increased fundal height at 30cm one week prior, but the mother had refused prenatal testing or ultrasound, and was lost to follow up. She delivered a live born male baby. Examination of the baby noted polydactyly, imperforate anus, excessive drooling, and a loud pan-systolic murmur. A single umbilical artery was noted in the umbilical cord. The baby had difficulty feeding with increasing respiratory distress. The baby died 2 days later from aspiration pneumonia.

Pathology

The specimen comprises the tongue, larynx, trachea, bronchi, both lungs and oesophagus of the foetus. The trachea and bronchi have been divided in the midline. A fistula is present just above the bifurcation at a communicating fistula can be seen connecting the distal oesophagus to the trachea (arrow). This is an example of a Type C Tracheoesophageal Fistula (oesophageal atresia with distal tracheoesophageal fistula). It is difficult to discern if the oesophagus ends as a blind pouch at the lower extent of the specimen.

Further Information

Tracheoesophageal Fistula (TEF) is a common congenital abnormality occurring in about 1 in 4000 live births. TEF usually occurs with oesophageal atresia (sometimes abbreviated to EA, reflecting the US spelling of ‘esophagus’). TEF are classified according to their anatomical configuration. Type C is the most common configuration; as described above, in which oesophageal atresia with distal tracheoesophageal fistula making up 86% of cases. TEF occurs without oesophageal atresia in only 4% of cases, Type E.

TEF and oesophageal atresia are caused by defective lateral septation of the foregut into the oesophagus and trachea. It is believed that a defect in epithelial-mesenchymal interactions causes failed branching of a lung bud branch which becomes the fistula tract. It is associated with VACTERL (vertebral defects, anal atresia, cardiac defects, TEF, renal anomalies, and limb abnormalities) or CHARGE syndrome (Coloboma, Heart defects, Atresia choanae, Growth retardation, Genital abnormalities, and Ear abnormalities).

Oesophageal atresia can be seen on prenatal ultrasound as polyhydramnios, absent/collapsed stomach, and proximal oesophageal pouch dilation. EA with TEF can be more difficult to see on ultrasound as fistula allows fluid flow into the stomach. Polyhydramnios occurs in one third of cases of EA with distal TEF. Postnatal symptoms vary on the configuration of the fistula. These include excessive drooling, respiratory distress, difficulty feeding and choking. Reflux of gastric contents can lead to aspiration pneumonia as in this case.

Diagnosis can be made by failing to pass a nasogastric tube into the stomach along with X-ray imaging. Fluoroscopy with contrast can be used for more indeterminate cases. For milder cases diagnosis may be made later with endoscopic investigation. Treatment involves surgical correction of the defects. Prognosis is usually good. However, cases with associated chromosomal, prematurity and cardiac defects are at increased risk of death.

Chronic hydrocoele

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02809

Clinical History

An 80-year old male presented with haematemesis. He has a known history of alcoholic liver cirrhosis with oesophageal varices. On examination, he is noted to have multiple spider naevi, large volume abdominal ascites and a scrotal swelling. Transillumination of the swelling transmitted red light. He experienced another large volume haematemesis and died shortly after admission.

Pathology

The specimen consists of a testis, tunica vaginalis and distal end of the spermatic cord. The testis and its surrounding layers have been bisected to display the cut surface. The tunica vaginalis is thickened and the enclosed cavity is distended. The testis is normal. This is an example of a chronic secondary communicated hydrocoele.

Further Information

A hydrocele is an accumulation of serous fluid between the parietal and visceral layers of the tunica vaginalis around the testes. Hydroceles can be described as communicating with the peritoneal cavity or noncommunicating with the peritoneal cavity. Communicating hydroceles develop due to failure of the processus vaginalis to close after the descent of the testes into the scrotum. These may present after birth as a congenital hydrocele or may present later in life due to increase in intra-abdominal pressure such a cardiac failure in this case. Non-communicating hydroceles are caused by imbalances in fluid secretion and reabsorption (e.g. orchitis, epididymitis), testicular tumour, physical trauma (e.g. hernia, testicular torsion) or defective lymphatic drainage (e.g. filariasis, elephantiasis). Patients present with a scrotal mass. The mass may be uni- or bilateral. Communicating hydroceles may be reducible and increase in size with raised intra-abdominal pressure. Non-communicating are usually nonreducible swellings. The swelling is usually non tender unless there is an underlying infection or torsion causing the hydrocele. Larger hydroceles may be cumbersome and cause erosion and skin infections on the scrotum. Diagnosis can be made on physical examination. Serous fluid allows the passage of light shined through the scrotum when examined: this is called transillumination. Ultrasound may be used to consolidate diagnosis and exclude other testicular pathology. Testicular cancer serum markers, such as alpha fetoprotein and B-HCG, may be taken to exclude testicular cancer. Many congenital hydroceles resolve spontaneously before the age of 2. If communicating hydroceles persist beyond 2 year they are surgically repaired due to the risk of developing incarcerated hernias. Surgical repair of communicating hydroceles in older patients may be offered if they are symptomatic. Treatment of the underlying aetiology of reactive hydrocele may cause them to resolve.

Chronic Gastric Ulcer

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02777

Clinical History

This elderly patient had a long history of „indigestion“. He collapsed and died after a massive stoke.

Pathology

The specimen is a 2cm coronal slice of tissue, which incorporates a portion of stomach diaphragm, liver and pancreas. The specimen has been opened to display a large ulcer at the upper end of the lesser curvature near the gastro-oesophageal junction. Macroscopically, the loss of substance at the site of the ulcer is oval, has 5-6cm in diameter and slightly elevated borders. The base is clean and smooth with no evidence of haemorrhage. The gastric wall surrounding the ulcer is indurated, because of the fibrosis that involves the base of the ulcer and spreads beneath the surrounding mucosa. Being retractile, the fibrosis manages to „pull“ the gastric mucosa towards the base of the ulcer, so that gastric mucosal folds converge radially around the loss of substance (this feature is not seen in ulcerated malignant gastric tumours). This is evident on the inferior aspect of the ulcer and less so superiorly.

Further Information Patients with a gastric ulcer may experience pain worsening with eating, often described as a burning or dull ache. Other symptoms include belching, vomiting, weight loss, or poor appetite. Complications may include bleeding, perforation, and blockage of the stomach. Common causes include the bacteria Helicobacter pylori and non-steroidal anti-inflammatory drugs (NSAIDs). H. pylori was first identified as causing peptic ulcers by Barry Marshall and Robin Warren of the University of Western Australia in the late 20th century, a discovery for which they were awarded the Nobel Prize in 2005. Other, less common causes include tobacco smoking, stress due to serious illness, Beh et’s disease, Zollinger-Ellison syndrome, Crohn’s disease, and liver cirrhosis[1]. Older people are more sensitive to the ulcer-causing effects of NSAIDs[1]. The diagnosis is typically suspected due to the presenting symptoms with confirmation by either endoscopy or barium swallow[1]. H. pylori can be diagnosed by testing the blood for antibodies, a urea breath test, testing the stool for signs of the bacteria, or a biopsy of the stomach[1]. Other conditions that produce similar symptoms, include stomach cancer, coronary heart disease, and inflammation of the stomach lining (gastritis) or gallbladder inflammation (cystitis) [1]. Treatment includes stopping smoking, stopping use of NSAIDs, reducing or preferably stopping alcohol consumption, and taking medications to decrease stomach acid[1]. Ulcers due to H. pylori are treated with a combination of medications, such as amoxicillin, clarithromycin, and a proton pump inhibitor (PPI). The medication used to decrease acid is usually either a PPI or an H2 blocker (histamine H2-receptor antagonists). Bleeding ulcers may be treated by endoscopy, with open surgery typically only used in cases in which it is not successful. Peptic ulcers are present in around 4% of the population.

Lobar pneumonia

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02755

Pathology

The specimen is a parasagittal section of the right lung and the boundaries between the three lobes are visible. The entire upper and middle lobes are congested and hyperaemic* causing the darker appearance. There are smaller foci in the left lung.

Further Information

Lobar pneumonia is a form of pneumonia characterized by inflammatory exudate within the intra-alveolar space resulting in consolidation that affects a large and continuous area of the lobe of a lung. It is one of the two anatomic classifications of pneumonia (the other being bronchopneumonia). The affected lobe in this case shows classical red ‘hepatization’ or consolidation of the lung parenchyma, which is due to vascular congestion with extravasation of red cells into alveolar spaces, along with increased numbers of neutrophils and fibrin. The filling of the airspaces by the exudate leads to a gross appearance of solidification, or consolidation, of the alveolar parenchyma. This reddish appearance has been likened to that of cut surface of the liver, hence the term „hepatization“.

The most common organisms that cause lobar pneumonia are Streptococcus pneumoniae, also called pneumococcus, Haemophilus influenzae and Moraxella catarrhalis. Mycobacterium tuberculosis, the tubercle bacillus, may also cause lobar pneumonia if pulmonary tuberculosis is not treated promptly. Other organisms that lead to lobar pneumonia are Legionella pneumophila and Klebsiella pneumoniae.

Like other types of pneumonia, lobar pneumonia can present as a community-acquired infection, in immune suppressed patients or as nosocomial infection. However, most causative organisms are of the community-acquired type.

On a posteroanterior and lateral chest radiograph, an entire lobe will be radiopaque with no evidence of air within it, indicative of lobar pneumonia.

*Hyperaemia = active engorgement of vascular beds with a normal or decreased outflow of blood.

Papillary Transitional Cell Carcinoma of the Renal Pelvis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02798

Clinical History

A 60 year old man who had worked in a paint factory for 40 years developed painless haematuria for one month. CT scan showed a suspected tumour in the left renal pelvis. He underwent a nephrectomy.

Pathology

This is the post-nephrectomy kidney. Of note the kidney maintains its foetal lobulation. There is a friable papillary tumour of 35mm in diameter projecting in the renal pelvis. The renal pelvis is visibly dilated due to this obstructing tumor. Histological examination revealed this is papillary transitional cell carcinoma arising in the renal pelvis.

Further Information

Between 5-10% of primary renal cancers arise in the urothelium lining the renal pelvis and calyces. These are similar to tumours which may arise in the ureter and urinary bladder. These tumours range from benign papillomas (rare) to well differentiated papillary carcinomas , which are common, and poorly differentiated tumours which can be either papillary, or flat and infiltrating. Symptoms of these renal pelvis tumors tend to occur early. Due to the friable nature of the tumours haematuria is common. As they tumours grow obstructive symptoms such as palpable hydronephrosis and flank pain can be noticed. The tumours can sometimes be multiple; involving the pelvis, ureter and bladder. There is an increased risk in developing urothelial tumours in individuals with Lynch syndrome and analgesic nephropathy. Smoking significantly increases the risk of developing urothelial tumours. Industrial chemicals called aromatic amines, such as benzidine and beta-naphthylamine, which are sometimes used in the dye industry, can lead to urothelial cancers. Infiltration of the wall of the pelvis and calyces is common in theses tumour. The prognosis with infiltration is not good. 5 year survival rates vary from 50-100% for low grade and non-invasive lesions to 10% for high grade infiltrating tumours.

Renal Cell Carcinoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02796

Clinical History

A 64-year old male presents with a 5-month history of generalised malaise, weight loss and dull right flank pain. On examination, there is a palpable right sided abdominal mass. He is noted to be hypertensive. Urinalysis reveals microscopic haematuria. The patient underwent right nephrectomy.

Pathology

The specimen is a kidney, which has been incompletely dissected in the coronal plane, and mounted to display the cut surface. The lower pole of the kidney has been replaced by a rounded ill-defined irregular mass 5cm in diameter, which has compressed and distorted the overlying renal parenchyma. The cut surface of the tumour has a variegated appearance caused by areas of haemorrhage and necrosis. Several small pale-yellow tumour nodules are present in the cortex and medulla above and separate from the lower pole tumour. These are intrarenal metastases. The renal pelvis appears slightly dilated with some blunting of the renal papillae, suggesting a degree of hydronephrosis. The capsular surface is finely nodular with a few coarse scars and contains several small simple cysts (see rear of specimen). Histologically, the tumour was diagnosed as a renal cell carcinoma.

Further Information

Renal cell carcinoma (RCC) comprise 85% of the primary renal malignancies. They originate within the renal cortex. The risk of developing RCC is doubled in males. It most commonly occurs in the 6th decade of life. Other risk factors for RCC include smoking, obesity, hypertension, unopposed estrogen therapy, as well as exposure to asbestos, petroleum and heavy metals. Most RCC are sporadic but around 5% are due to autosomal dominant familial cancers, such as Von Hippel Lindau syndrome, hereditary leiomyomatosis and Birt-Hogg-Dub syndrome. There are several major primary renal tumour types according to genetic and histological tumour characteristics: clear cell carcinoma (70-80%), papillary carcinoma (10-15%), chromophobe carcinoma (5-10%), oncytic carcinoma (3-7%) and collecting (Bellini) Duct carcinoma (<1%). Clear cell carcinoma typically have a deletion of chromosome 3p and arise from the proximal tubule. They may be solid or less commonly cystic. They occur in association with Von Hippel Lindau as well as sporadically. Papillary carcinomas arise from the proximal tubule. They are associated with trisomies 7 and 17; loss of Y in male patients; and MET kinase domain mutations. They are frequently multifocal in origin. Chromophobe carcinoma originate from intercalated cells of the collecting ducts. They are associated with multiple chromosome losses and hypodiploidy. They have a low risk of disease progression. Renal oncocytic carcinomas are typically comprised of well-differentiated cells with prominently eosinophilic granular cytoplasm; they are associated with a good prognosis. In contrast, collecting (Bellini) duct carcinoma of the kidney is a highly aggressive tumour with an extremely poor prognosis as it does not respond well to chemotherapy drugs used for renal cell carcinoma, and progresses and spreads more quickly. It is a variety of renal cell carcinoma (RCC) arising from the distal segment of

the collecting ducts of Bellini in the renal medulla. The typical clinical features of RCC are costovertebral pain, palpable mass and haematuria. RCC is the great mimic in medicine producing many manifestations including: polycythemia, hypercalcaemia, hypertension, pyrexia, Cushing‘s syndrome, eosinophilia and amyloidosis. RCC tend to metastasize before producing may local symptoms. The most common sites of distal spread are the lungs (50%) and bones (33%) followed by lymph nodes, adrenal glands and brain. RCC has a tendency to invade the renal vein and extend up it as a tumour thrombus, growing as a solid column extending upto the inferior vena cava. Ultrasound and CT are the most common investigations used to assess renal lesions and diagnose RCC. A tissue biopsy may be required in some patients. An increasing number of patients are being diagnosed with RCC because of incidental kidney lesions being detected on abdominal CT requested for other medical reasons. The average 5-year survival rate for RCC is 70%. Treatment depends on the stage of the tumour. Radical nephrectomy is the usual surgical option. Medical treatment includes chemotherapeutic drugs as well as Vascular endothelial growth factor (VEGF) inhibitors and tyrosine kinases inhibitors in patients with metastatic disease.

Carcinoma of Breast

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02804

Clinical History

A 76-year old female presented to the emergency department sudden loss of consciousness. She had signs of a left sided cerebrovascular accident. She was intubated and her stroke was treated. During her admission in ICU she was noted to have a fixed mass in her left breast with palpable lymphadenopathy in her left axilla. She died from a respirator associated pneumonia.

Pathology

The specimen is the patient’s left breast mounted to display the cut surface. Immediately beneath and attached to the skin is a large oval tumour mass 11cm in maximum diameter. The tumour is adherent to the underlying muscle. The tumour is not encapsulated and has a variegated cut surface with areas of necrosis, haemorrhage and cyst formation. This is a breast adenocarcinoma, which involved the regional lymph nodes.

Further Information

Breast carcinoma is the second most commonly diagnosed cancer in women worldwide. It is rare in women under 30 years of age but the incidence increases significantly after 30 with the peak occurring at 70 to 80 years. Incidence has been decreasing since the introduction of breast cancer screening programmes, which offer mammography to women at risk and public awareness and education in self-examination of the breast. However, breast cancer remains one of the leading causes of cancer-related death in women. Major risk factors for developing breast carcinoma include being of female gender (men account for 1% of breast cancer diagnosis), exposure to estrogen (early menarche, late menopause, exogenous estrogen), family history of breast cancer, being nulliparous, not breastfeeding, radiation exposure and obesity. Germline mutations in tumour suppressor genes, such as BRCA1, BRCA2, TP53, ATM, CDH1 and CHEK2 are linked with some hereditary cases of breast cancer. Most breast neoplasms are adenocarcinomas that begin in the duct/lobular system as carcinoma in situ (DCIS). These malignancies are further subdivided according to their expression of estrogen receptors (ER) and Human epidermal growth factor 2 (HER2), which guides treatment. The most frequent sites if distant metastasis occur are bone, liver, lung and brain. In developed countries with screening programmes most patients present following an abnormal mammogram. Symptomatic patients present with a breast mass which is classically hard, irregular, immovable. Other clinical symptoms are axillary lymphadenopathy, overlying skin changes (erythematous or thickened skin, dimpled skin (peau d‘orange) and nipple retraction. Symptoms of distant spread of disease may also cause patient presentations. Treatment depends on the stage of the disease and the ER and HER2 status of the tumour. Surgical treatments include uni- or bilateral mastectomy or breast conserving lumpectomy. Surgical axillary node clearance is performed in cases with positive nodal disease. Radiotherapy is given to patients with high risk of local recurrence. Patients with HER2 positive cancers are treated with targeted drugs, such as trastuzumab (Herceptin). Patients with ER positive tumors can be treated using anti-estrogen therapy, such as tamoxifen. Systemic chemotherapy is also used to treat some patients with breast cancer.

Endometrial Carcinoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02805

Clinical History

A 63-year old woman presented with a history of dull lower abdominal pain for 2 months and heavy persistent vaginal bleeding for 1 week. The menopause had occurred 13 years previously. Radical abdominal hysterectomy and bilateral salpingo-oophorectomy were commonly performed for the treatment of endometrial cancer following confirmation of endometrial carcinoma in biopsy.

Pathology

The specimen consists of uterus, fallopian tubes and ovaries. The endometrial cavity and endocervical canal have been opened on the anterior aspect. The endometrial lining is grossly abnormal especially on the right side and a brown polypoid tumour has invaded the myometrium and extends inferiorly into the cervical canal. Histologically this was a welldifferentiated adenocarcinoma of the endometrium. The left ovary, which has been sectioned in the coronal plane, is enlarged and has several large follicular cysts/cavities.

Further Information

Endometrial carcinoma is the most common gynecological malignancy in developed countries and the second most common in developing countries after cervical cancer. There are two major types of endometrial carcinoma. Endometroid carcinoma account for almost 80% of endometrial carcinoma. They usually present early and so have a more favourable outcome. These tumours may arise from atypical endometrial hyperplasia. Common genetic abnormalities seen in endometrioid tumours are mutations in the PTEN, PIK3Ca and ARID1A genes. Serous carcinoma are a less common form of endometrial carcinoma. These tumours are associated with mutations in TP53 gene and carry a poorer prognosis. Endometrioid tumours tend to affect women aged 55 to 65 years. Risk factors for developing endometrioid endometrial cancer include obesity, impaired glucose tolerance, infertility, unopposed estrogen therapy (e.g. early menarche, late menopause or exogenous sources). Serous neoplasms affect older women aged 65 to 75 years with other risk factors for development include having a lower BMI and an atrophic uterus. Women with Hereditary Nonpolyposis Colorectal Cancer (Lynch Syndrome) have a significantly higher risk of developing endometrial cancer. The most common symptom of endometrial cancer is abnormal vaginal bleeding. Most frequently it presents as post-menopausal bleeding, which often allows early presentation. Others may be asymptomatic or an incidental finding of an abnormal endometrium on abdominopelvic imaging. The main radiological sign of endometrial cancer is abnormally thickened endometrium on pelvic ultrasound or CT scan. Diagnosis is made on endometrial biopsy, endometrial curettage or hysterectomy. Treatment depends on the stage of the cancer and includes local radiotherapy, systemic chemotherapy and surgical hysterectomy +/- salpingo-oophorectomy.

Hepatocellular Carcinoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02786

Clinical History

A 60-year old male is admitted with jaundice, melena and abdominal distension. He has a past medical history of untreated Hepatitis C infection from previous intravenous drug use. Further questioning reveals a 9-month history of significant fatigue, weight loss, nausea and intermittent dull right upper quadrant pain. Liver ultrasound demonstrated two large lesions within the liver. Soon after admission the patient dies from a suspected oesophageal variceal haemorrhage.

Pathology

This is the liver specimen of the patient on postmortem examination. The cut surface of the liver has a multinodular appearance consistent with macronodular cirrhosis. These multiple nodules are of varying size up to 2cm in diameter, and are separated by narrow bands of fibrous tissue. There are two large round tumours also visible. These are 8cm and 6cm in diameter with a variegated cut surface due to focal necrosis, haemorrhage and bile staining. This is an example of hepatocellular carcinoma that has developed on the background of a cirrhotic liver.

Further Information

Hepatocellular carcinoma is the most common primary malignant liver cancer. HCC arises from hepatocytes in the liver. Risk factors for developing HCC include viral infections (Hepatitis B and Hepatitis C), liver cirrhosis, aflatoxin exposure, Non Alcoholic Fatty Liver Disease (NAFLD), haemochromatosis and Wilson‘s Disease. The latter is an inherited disorder in which excessive amounts of copper accumulate in the body, particularly in the liver, brain, and eyes. HCC incidence is highest in Asia and sub Saharan Africa. There is a higher risk of developing HCC in males. HCC is associated with acquired driver mutation in oncogenes and tumour suppressor genes. The two most common driver mutations that can lead to HCC are gain-offunction mutations in beta-catenin and loss-of-function mutations in p53. Clinically HCC can present with abdominal pain, fatigue, weight loss, abdominal fullness and less commonly jaundice, gastrointestinal or variceal bleeding. HCC metastatic spread is haematogenous with lung, abdominal lymph nodes and bones being the most common extrahepatic sites. Death usually occurs from cachexia, haemorrhage or liver failure. Treatment varies on the stage of the tumour and the patient’s underlying general status and co-morbidities. Treatment can include surgical resection of ablation of the tumour, chemotherapy and liver transplantation can be curative.

Rheumatic endocarditis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02742

Clinical History

The patient was a 52-year old female with increasing dyspnoea. She gave a past history of fever with flitting joint pains in childhood following a sore throat. On examination: cyanotic, pulse showed atrial fibrillation, jugular venous pulse elevated, pan-systolic murmur at apex, hepatomegaly, and dependent oedema. She was being treated with digoxin, lasix (furosemide) and penicillin but died after cardiac arrest.

Pathology

The specimen is that of a heart opened to show the left atrium and left ventricle. The mitral valve has been cut, but those visible parts show significant thickening. The left atrial wall shows deposition of blood and fibrin. The left auricular appendage is filled with blood clot, caused by atrial fibrillation. The mural thrombus on the atrial wall is in the typical site:- the deep layers of the endocardium forming irregular thickenings, called MacCallum’s plaques (arrows).

Further Information

In this patient, the history of fever and joint pains following a sore throat is very suggestive of a history of rheumatic fever. Rheumatic fever is an inflammatory disease that can involve the heart, joints, skin, and brain. Typical symptoms include fever, multiple painful joints, involuntary muscle movements (chorea), and occasionally a characteristic non-itchy rash known as ‘erythema marginatum’.

Rheumatic fever may occur 2-4 weeks following an infection of the throat by the bacterium Streptococcus pyogenes. If the infection is left untreated (with pencillin), rheumatic fever occurs in up to three percent of people. The underlying mechanism is believed to involve the production of antibodies against a person‘s own tissues (autoimmune disease). Due to their genetics, some people are more likely to get the disease when exposed to the bacteria than others. Other risk factors include malnutrition and poverty, occurring more commonly in low to middle income countries and particularly in Indigenous communities.

The heart is involved in about half of the cases. Damage to the heart valves, known as rheumatic heart disease (RHD), usually occurs after repeated attacks (carditis) but can sometimes occur after one. Carditis can progress to chronic rheumatic heart disease, usually affecting cardiac valves. The mitral valve is the most commonly affected

valve, with fibrosis leading to mitral valve stenosis and this specimen highlights the thickening of the mitral valve. Stenosis is thought to occur due to Aschoff nodules, which are granulomatous lesions with a central area of fibrinoid necrosis and surrounded by an infiltration of autoreactive T cells. The Aschoff nodules also contain ‘giant cells’, which are thought to be some type of degenerative connective or endothelial tissue.

Stenosis may progress through the years and as it worsens, the left atrium will become increasingly dilated. Consequentially, atrial fibrillation may develop and mural thrombi can be formed. Further, tight mitral stenosis can result in severe cardiac failure.

Suppurative arthritis of the knee

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02768

Clinical History

A man aged 82 years had a history of tuberculosis of the left knee in childhood. He developed a painful swollen knee 10 days prior to admission to hospital. Examination revealed an inflamed knee, painful to move, which improved slightly with antibiotic therapy and rest. X-ray showed a disorganized knee joint and mid-thigh amputation was performed 3 days after admission. Post-operative recovery was satisfactory.

Pathology

The specimen displays the articular surfaces of a femur and tibia. The articular surfaces have been severely eroded. They are brown in colour, very irregular, and there are shaggy adhesions and plaques of yellow necrotic material. No normal articular cartilage is present. Some irregular varying in size bony projections (up to 1 cm in diameter) are present on the femoral condyles. Staphylococcus aureus was cultured from the joint. This is an example of suppurative arthritis in a joint previously damaged by tuberculosis.

Further Information

Suppurative arthritis is typically caused by a bacterial infection in the joint. Diagnosis is made by analysis of synovial fluid including microscopic examination and culture. Suppurative arthritis is also referred to as pyarthrosis, and bacterial or septic arthritis. Tuberculous septic arthritis should be considered in patients who present with acute or chronic monoarthritis, and who have an abnormal chest radiograph or eosinophilia or a previous history of TB.

Pulmonary tuberculosis accounts for around 52% of tubercular infection but musculoskeletal involvement is seen in up to 19% of cases.1 It is more common in children than in adults, probably owing to the greater amount of bone marrow present in immature bone. In adults, TB shows a preponderence to the spine (40%), followed by the hip (25%), and then the knee (8%).While extrapulmonary manifestations of TB are common, accounting for around 15–20% of cases in immunocompetent patients, the first presentation of the disease as a joint infection is rare[1].

Reference: 1. Carrol ED, Clarke JE, Cant AJ. Non-pulmonary tuberculosis. Paediatr Respir Rev. 2001;2:113–9.

Pyonephrosis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02795

Clinical History

A 38-year old female presents with severe nausea, vomiting, fevers and rigors. She has a history of recurrent urinary tract infection over the past 6 months. She has required several courses of oral antibiotics, and one admission for IV antibiotics. Blood tests show raised inflammatory markers. Urinalysis is positive for white blood cells (WBC). A CT scan shows unilateral left hydronephrosis and pyelonephritis. She fails to respond to conservative treatment, and undergoes a nephrectomy. She makes a complete recovery.

Pathology

This is the patient’s left nephrectomy specimen. The kidney has been sliced to display the cut surface. The pelvis and calyces are greatly dilated, and contain remnants of yellow pus. There is considerable fibrosis of the renal parenchyma. In the mid-zone near the lateral border, there is a hemorrhagic necrotic area 35 x 12 mm in diameter containing pus. There are two similar small hemorrhagic necrotic areas visible on the capsular surface. These lesions are probably continuous with the lesion seen on the cut surface, likely to be caused by haemorrhage into an abscess cavity. This lesion would have resulted in a perinephric abscess.

Further Information

Pyonephrosis occurs when there is an obstruction within the upper urinary tract and pyelonephritis. Debris of infection, WBC and bacteria collect in the obstructed kidney, resulting in a hydronephrotic kidney that is filled with pus. A staghorn calculus usually forms in association with chronic or recurrent infection as a consequence of the more alkaline urinary pH caused by the bacterial infection. Pyonephrosis is a rare condition. Risk factors for development include immunosuppression, diabetes and anatomical urinary tract obstructions e.g. urinary tract strictures, horseshoe kidneys, tumours, urinary calculi. Clinical presentation can consist of vague symptoms but may include constitutional symptoms of sepsis, flank pain, haematuria, dysuria and pyuria. A grossly nephrotic kidney may be palpable on palpation of the abdomen. Pyuria will be present on urinalysis. Radiological diagnosis can be made using CT investigations usually but also Ultrasound or MRI looking for evidence of urinary tract obstruction and pyelonephritis. Treatment will depend on the cause of the obstructing lesion. Emergent treatment involves drainage of the purulent build up within the kidney. This is performed by urology or interventional radiologists via percutaneous or retrograde ureteral stents to relieve the obstruction and drain the pus. Further surgical treatment will depend on the cause of obstruction. Antibiotic therapy is required for treatment of underlying infection or sepsis. If left untreated complications such as florid sepsis, xanthogranulomatous pyelonephritis, renal or perinephric abscess formation or fistula to pleura, colon or duodenum may occur.

Nodular hyperplasia of the Prostate

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02807

Clinical History

A 63-year old male presented to the emergency departed with acute abdominal pain. He has been unable to pass urine for 5 days. Further questioning revealed a 2-year history of urinary frequency, double void, urinary hesitancy, nocturia and a poor urine stream. Abdominal examination showed a tender distended bladder and an enlarged palpable prostate on digital rectal examination. A bedside bladder scan demonstrated a volume of >1L in the bladder. Blood tests shows a severe acute kidney injury. He is diagnosed with acute renal failure due to acute urinary retention. There were multiple failed attempts at catheterising the patient via the urethra and suprapublically. A total prostatectomy was performed and he made a good recovery.

Pathology

The specimen is an enlarged prostate gland sliced transversely to display the external and cut surfaces, On the cut surface there are numerous nodules varying in size from 2-10mm in diameter. This is an example of benign nodular hyperplasia (BPH) of the prostate gland.

Further Information

Benign prostatic hyperplasia (BPH) or nodular hyperplasia of the prostate is a common disease in older men. BPH is caused by nodular hyperplasia of prostatic stromal and glandular epithelial cells primarily in the periurethral prostate. Hyperplasia is a result of accumulation of senescent cells due to impaired cell death and cell proliferation driven by androgens, mainly dihydrotesterone. Disproportionate enlargement of the median lobe is a common feature of nodular hyperplasia of the prostate. The projecting median lobe may occlude the internal urethral orifice on contraction of the bladder. Prevalence of BPH increases significantly with age. BPH is present in 20% of males at 40 years of age, 70% of males of 60 year of age and almost 90% of males by the age of 80. There is an increased risk of BPH in men with a positive family history of BPH, in obese males and exposure to exogenous androgenicanabolic steroids. Clinical presentation of BPH results from urinary obstructive symptoms. Patients complain of urinary frequency, nocturia, urinary hesitancy, double voiding, poor urinary stream and overflow dribbling. Acute urinary retention may result from complete urinary tract obstruction as in the case discussed above. Post void residual urine results from the obstructing prostate which leads to an increased risk of urinary tract infections. Diagnosis can be made on clinical history and physical examination of the prostate with a digital rectal exam. Prostate specific antigen may be used to screen for prostate cancer. Ultrasound scan or CT can be used to evaluate the volume of the prostate. Treatment of BPH can be treated medically with Alpha-blockers to relax the prostate smooth muscle tone or 5-alpha-reductase inhibitors, which inhibit synthesis of dihydrotesterone. The main surgical treatment for severe cases of BPH is transurethral resection of the prostate (TURP). Total prostatectomy is no longer used due to risk of disabling complications.

Tetralogy of Fallot

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02740

Clinical History

A 21-month old boy was admitted with a history of exhaustion and exertional dyspnoea for the previous 2 to 3 months. During this time there had been several attacks of acute dyspnoea each lasting up to two minutes. Examination revealed central cyanosis, mild finger clubbing, and a harsh systolic bruit maximal at the left sternal edge. Cardiac catheterisation led to a diagnosis of Fallot's tetralogy and severe pulmonary oedema. A surgical correction was performed (Willis-Potts anastomosis between the aorta and the origin of the left pulmonary artery). The child developed acute dyspnoea and left lobar consolidation 12 hours post-operatively and died despite treatment.

Pathology

The child's heart is viewed from the anterior aspect. The anterior wall of the right ventricle has been excised to reveal prominent right ventricular hypertrophy and a narrowed pulmonary outflow tract. The pulmonary valve ring is also small, with a bicuspid stenosed valve. There is a patch of endocardial fibrosis in the outflow tract below the pulmonary valve. The origin of the aorta overlies a high ventricular septal defect. A probe could be passes into the aorta from the hypertrophied right ventricle. The further probe was able to be passed from the narrowed pulmonary trunk into a dilated, thin-walled left pulmonary artery and through the surgical anastomosis into the descending aorta. Examination of the posterior aspect of the specimen reveals an opened right atrium and left ventricle. When viewed from the right side of the heart, there is a large atrial septal defect (ASD), 8 mm in diameter at the site of the foramen ovale (large arrow). Another tiny ASD (small arrow) 3 mm in diameter is present posterior to the upper border of the large ASD. Note that the wall of the left ventricle is slightly thinner than the wall of the right ventricle.

Further Information

The four features of tetralogy of Fallot are: 1. Ventricular septal defect (VSD); 2. An aorta that straddles the VSD with the latter communicating with both ventricles (over-riding aorta) instead of just the left ventricle; 3. Pulmonary stenosis or obstruction of the right ventricular overflow tract; 4. Right ventricular hypertrophy. This condition usually causes cyanosis early in life. Its severity depends on the degree of pulmonary outflow obstruction, which determines whether there is a left-to-right, or right-toleft shunt. In some patients, pulmonary blood flow is increased due to the presence of a patent ductus arteriosus. Patients with this condition may survive untreated into adult life, and a few may reach middle age. However, surgical correction is now possible and is desirable, as the disorder is ultimately fatal. Sometimes additional cardiac abnormalities may be present. (e.g. atrial septal defect, as was found in this case).

In most cases of tetralogy of Fallot, the cause is not known although in some patients, genetic factors play a role. For

example, the condition is more common in patients with Down syndrome (Trisomy 21; in association with AV canal defects) or DiGeorge syndrome (22q11 deletion).

Fatty Liver

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02766

Pathology

A slice of liver reveals the characteristic yellow/grey and greasy appearance on one side. On the other side the appearance is restricted to the outer margin whilst the central area displays darker colouration possibly due to cirrhosis. This is an example of fatty change in the liver.

Further Information

Causes of fatty change (steatosis) or accumulations of triglycerides in the liver include obesity, diabetes, alcohol abuse, starvation, Kwashiorkor, drugs and toxins. Alcoholism is the commonest cause in most communities.

Trachea — Hodgkin Lymphoma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02760

Clinical History

A 45-year old male presented with a lump in his left supraclavicular area. The swelling had been increasing in size over 6 months. Excision biopsy of the lump showed Hodgkin lymphoma (HL). Ten months later he was readmitted with left shoulder pain and swelling of his left arm. Examination revealed generalised lymphadenopathy with significant swelling in his left supraclavicular and axillary regions. He was treated with radiotherapy and Thiotepa chemotherapy. He developed vomiting. A subsequent barium meal showed duodenal obstruction from extrinsic lymph node compression. He continued to deteriorate and died 2 weeks after readmission.

Pathology

The 3D print shows the tracheal bifurcation with adjacent paratracheal and peri-bronchial lymph nodes. The trachea has been opened longitudinally and is viewed from behind. The para-tracheal lymph nodes are pale and matted (fused) together. Similar abnormal tissue is seen as a confluent pale mass on the left side of the trachea, above the aortic arch, which is seen cut in cross-section as a void space with branches arising. The peri-bronchial lymph nodes are also enlarged, and contain carbon pigment. The circumscribed small paler areas in the lymph nodes and extra-nodal tumour are foci of necrosis. There is an atheroma in the wall of the aorta but it is difficult to see in the 3D print.

Further Information

Hodgkin Lymphoma (HL) is a malignancy of lymphocytes. It is characterised by the presence of neoplastic giant cells called Reed Sternberg cells. There are 5 main subtypes according to the WHO Lymphoma Classification, based on the morphology, immunophenotyping and genetics. Activation of the transcription factor NF-kB is a common pathway of tumorigenesis among the subtypes. This promotes proliferation, reduces apoptosis, and induces expression of cytokines that recruit the immune cells that surround Reed Sternberg cells in HL.

There is a bimodal age distribution with a peak in late adolescence/early adulthood and a second peak in older adults. HL accounts for just under 1% of all cancers worldwide. Infection with Epstein Barr Virus (EBV) contributes to the pathogenesis of the main subtypes of HL.The viral genome causes genetic alterations that lead to aberrant signal pathways, although the precise mechanism is not known. Immunosuppression (e.g. HIV infection or post- organ transplant) and positive HL family history are also risk factors. HL commonly presents as painless lymphadenopathy, pruritus, weight loss, fevers and night sweats. Later disease sees organ spread to the spleen, liver and bone marrow. Compressive symptoms can arise from enlargement of lymph nodes and infiltrated organs. HL is diagnosed with staging CT scan, excision biopsy of involved nodes and bone marrow biopsy. Treatment involves radiotherapy and chemotherapy. Although previously incurable, the overall survival of HL has improved significantly over the last 5 decades as a result of modern therapies: diagnosed at an early stage, it is almost 90%, and even later stage disease has a favourable prognosis.

Abdominal Aortic Aneurysm (AAA)

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02733

Clinical History

This 70-year-old man with a past history of mild gastro-oesophageal reflux presented to the Alfred Hospital with a sudden onset of severe upper abdominal pain, which radiated to the left shoulder tip. On examination, he was distressed and hyperventilating, pulse rate 87/ min, and a blood pressure 140/90 mm Hg. Abdominal examination revealed board-like rigidity and diminished bowel sounds. At emergency laparotomy no evidence of a ruptured viscus was found; the pancreas appeared normal and an unruptured abdominal aortic aneurism was noted. Endoscopy on the following day showed a ruptured oesophageal ulcer and a Celestin tube was inserted. The patient developed localised infective complications, pulmonary oedema and congestion, and died 19 days after admission.

Pathology

The specimen consists of lower abdominal segment of aorta together with common iliac vessels and proximal portions of the internal and external iliac arteries. A large 10 x 7 cm aneurysm is situated below the origin of the renal arteries extending to the aortic bifurcation. The aneurysm with its severe thinning of the wall of the abdominal aorta is partly lined by a laminated thrombus, indicating the chronicity of the process. There is evidence of a recent thrombus on the luminal surface. There also appears to be some aneurysmal dilatation of the common iliac and (opened) proximal left external iliac artery. The abdominal aorta at the upper end of the specimen shows multiple focally ulcerated atheromatous plaques. There is no evidence of rupture.

Further Information

Abdominal aortic aneurysm (AAA or triple A) represents a localized enlargement of the abdominal aorta (diameter >3 cm or more than 50% larger than normal)[1]. They are usually asymptomatic, except during rupture[1]. Large aneurysms may be palpable on abdominal examination. Occasionally, abdominal-, back-, or leg pain may occur depending on location and size. Rupture may result in pain in the abdomen or back, sudden low blood pressure with loss of consciousness, and often results in death[1]. AAA’s occur most commonly in those over 50 years of age, in men, and amongst those with a family history of this disease. Additional risk factors include smoking, high blood pressure, and other heart or blood vessel diseases. They are also found in genetic abnormalities, including Marfan’s syndrome and Ehlers-Danlos syndrome. AAAs are the most common form of aortic aneurysm, and about 85% occur below the kidneys[1].

Reference: 1. Kent KC (27 November 2014). „Clinical practice. Abdominal aortic aneurysms“. The New England Journal of Medicine. 371 (22): 2101–8.

Syphilitic Aneurysm

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02745

Clinical History

A 61-year old male presents with exertional anginal chest pain and dyspnoea. He has had these symptoms for 6 years with increasing severity. On examination, he is cyanotic and tachycardic with a collapsing pulse. A swelling was noted on the right side of his neck. There was a thrill in his carotid artery. The apex beat was displaced inferolaterally. A loud systolic and diastolic murmur was auscultated in the aortic area. Chest X-rays showed cardiomegaly with a large rounded lesion in the right upper mediastinum continuous with the heart shadow with radiographic evidence of cardiac failure. Blood tests were positive for anti-treponemal antibodies. The patient‘s condition deteriorated and he died of cardiac failure.

Pathology

This specimen is the patient‘s enlarged heart, including the aortic arch and descending aorta. The ascending aorta is dilated up to 7 cm in diameter, and is expanded superiorly by a large aneurysmal bulge 11 x 13 cm in diameter. This has been opened to display the wrinkled scarred intimal surface. There is also marked atheroma of the intima. The innominate, left common carotid and subclavian arteries have been displaced towards the patients left by the aneurysm. On the internal surface of the aneurysm there is a ridge-like thickening 5 mm high. This is the site of attachment of the pericardial sac externally. There is marked congestion of small blood vessels in the adventitia of the aorta. This is a syphilitic aneurysm of the arch of the aorta.

Further Information

Syphilis is a chronic infection caused by the spirochete Treponema pallidum. Sexually transmitted infection is most common but it may also be congenitally acquired by transplacental transmission of the bacteria. Those who have the higher risk of syphilis infection include those of a sexually active age, intravenous drug user, HIV-infected patients and male same sex relationships. Syphilis infection rates decreased significantly with the introduction of penicillin in 1943; it remains the main treatment today. However, the infection rate has been increasing since the early 2000s.

Syphilis is divided into three clinical stages with distinct clinical and pathological features with characteristic proliferative endarteritis affecting small vessels.

Primary syphilis occurs usually 3 weeks after initial infection. This manifests typically as a single, painless and erythematous lesion called a chancre at the site of inoculation. The syphilis spreads throughout the body from this chancre which then heals spontaneously after 3 to 6 weeks.

Secondary syphilis occurs weeks to a few months after the primary chancre resolves in 75% of untreated patients. During this stage patients commonly have generalised symptoms, such as malaise and lymphadenopathy and skin rashes. Palmar/ plantar rashes are the most frequent site but rashes can be diffuse. These rashes can be maculopapular, scaly or putular.

Condylomata lata are elevated gray plaques that arise on the moist mucous membranes such as oral or genital regions. Other less common manifestations include hepatitis, gastrointestinal invasion or ulceration and neurosyphilis - discussed below.

Tertiary syphilis has three main characteristics: cardiovascular syphilis, neurosyphilis and gummatous syphilis. These occur after a latent period of 5 years or more in ? of untreated patients. Cardiovascular syphilis involves an aortitis for which the exact pathophysiology is unclear. The vasculitis involves the ascending thoracic aorta leading to progressive dilation of the aortic root. This can lead to aortic valve insufficiency from dilation of the aortic valve ring. Endarteritis of the vasa vasorum leads to scarring of the media with loss of muscle and elastic tissue leading to the formation of aneurysms. Clinical manifestation usually happens 15-30 years post initial infection.

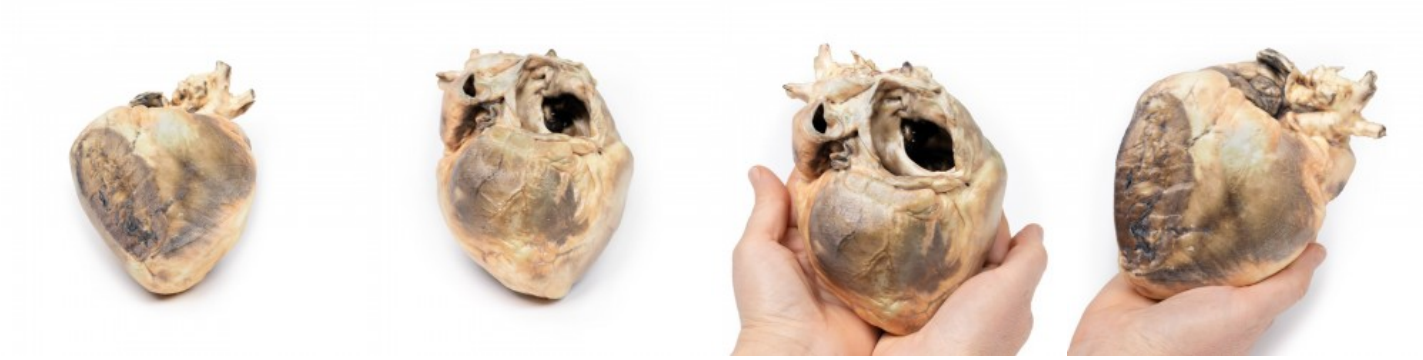
Neurosyphilis can be symptomatic or asymptomatic. It occurs in 10% of untreated patients. Early clinical manifestations include headaches, meningitis, hearing loss and ocular involvement, most commonly uveitis, causing vision loss. Late manifestations can occur up to 25 years post initial infection. Main features are meningovascular neurosyphilis, paretic neurosyphilis and tabes dorsalis. Meningovascular involvement involves chronic meningitis and endarteritis which can lead to strokes. Tabes dorsalis is caused from degeneration of the posterior columns within the spinal cord. This causes loss of proprioception, ataxia, loss of pain sensation, and loss of reflexes. Paretic neurosyphilis is caused by invasion and damage of the brain parenchyma, most commonly the frontal lobes. This leads to progressive cognitive impairment and mood disturbance.

Gummatous syphilis is characterised by the formation of nodular lesions most commonly bone, skin and mucosa of the upper airway and mouth called gummas. These can occur anywhere including viscera. The formation of gummas is rare but occurs more frequently in HIV-infected patients. Skeletal involvement causes pain and pathological fractures.

Atrial septal defect

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02735



Clinical History

A 10-year-old girl with a known congenital heart was admitted for surgical repair because of the recent onset of cyanosis and cardiac failure. On examination, she was breathless with a blood pressure of 105/60mm/Hg and a pulse rate of 140/min. There was a loud heart murmur in the fourth left intercostal space adjacent to the sternum. The jugular venous pressure was elevated, and there were bilateral pulmonary basal crepitations but no peripheral oedema. At operation, the defect was repaired; however, death followed a sudden post-operative deterioration of unknown cause.

Pathology

The heart is viewed from the left side. The left atrium has been opened to display a large ovoid defect 3.5 cm in greatest diameter in the inter-atrial septum. Only a small postero-inferior crescentic rim of septum remains. The left ventricle is small, and the right ventricle is hypertrophied (see posterior aspect of specimen where part of the right postero-lateral wall of the right ventricle has been cut away to demonstrate the thickened wall). The pulmonary artery, seen to the left of the atrial cavities, is greatly enlarged. The smaller vessel seen lying above the cut end of the pulmonary artery is the aortic arch. The cut edge of a lumen 8 mm in diameter immediately below the cut end of the pulmonary artery is the left auricular appendage.

Further Information

Atrial septal defect is usually asymptomatic early in life, even when large. Symptoms may not develop until adult life. The onset of symptoms is due to reversal of the initial left-to-right shunt as a result of increasing right ventricular hypertrophy and pulmonary hypertension. The ensuing rightto-left shunt is associated with cyanosis and dyspnoea, and ultimately leads to congestive cardiac failure. There are several types of atrial septal defects, including:

Secundum - This is the most common type of ASD and occurs in the middle of the wall between the atria (atrial septum).

Primum - This defect occurs in the lower part of the atrial septum and might occur with other congenital heart problems.

Sinus venosus - This rare defect usually occurs in the upper part of the atrial septum and is often associated with other congenital heart problems.

Coronary sinus - In this rare defect, part of the wall between the coronary sinus — which is part of the vein system

of the heart — and the left atrium is missing.

It is not known why all atrial septal defects occur, but some congenital heart defects appear to be familial and sometimes occur with other genetic problems, such as Trisomy 21 (Down’s syndrome). Some conditions during pregnancy can increase the risk of having a baby with a heart defect, including acute infections such as Rubella infection; drug, tobacco or alcohol use, or exposure to certain substances (such as cocaine) during the first trimester of pregnancy; and underlying systemic conditions, such as diabetes or systemic lupus erythematosus.

Traumatic Oesophageal-aortic fistula

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02743

Clinical History

A woman who swallowed a chop bone during lunch collapsed later in the afternoon and suffered a massive haematemesis. At laparotomy, the stomach was filled with fresh blood but the cause was not identified. She died one day later and necropsy revealed a communication between aorta and oesophagus. The stomach was distended with blood and contained a few fragments of bone.

Pathology

The specimen is a block dissection of distal trachea (posterolateral on right margin), aortic arch (opened in coronal plane and viewed from anterior aspect) and oesophagus (posteriorly and opened longitudinally). The oesophageal mucosa is ulcerated and haemorrhagic. A small blue probe identifies a fistula between the oesophagus and posterior wall of the thoracic descending aorta.

Note

While this scenario was a traumatic cause of oesophageal-aortic fistula, it should be noted that there are non-traumatic causes of the same. In fact, these fistulae can be caused by compression of the aorta from an aneurysm, advanced gastrointestinal malignancies or erosion of an aortic graft into adjacent gastrointestinal tract and can occur anywhere along the length of the aorta.

Aorto-enteric fistulas are life-threatening. The most common presentation is gastrointestinal bleeding and can present as either minor bleeding or a large life-threatening bleed that results in haemodynamic compromise. Patients can present with melaena (dark sticky faeces containing partly digested blood) or frank bleeding in stools. In smaller fistulas with slow, minor bleeds, patients can present with malaise or ischaemia of lower limbs due to less blood flow from the aortic bleed. Other presentations include haematemesis as occurred in this case.

Diagnoses of these fistulas can be difficult, depending on the cause, size and location of the fistula. In a stable patient, endoscopic exploration or CT angiography may be first line options for diagnosis. However, diagnosis in hemodynamically unstable patients is more time critical and may require laparotomy as well stabilisation with blood transfusions.

Pedunculated Adenoma of the Colon

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02782

Clinical History

A 50-year old male underwent a colonoscopy after testing positive for faecal occult blood during a screening test. Colonoscopy revealed a pedunculated tumour in the descending colon, which was later resected.

Pathology

This specimen is the resected segment of descending colon. There is a single dark lobulated mass visible arising from the mucosal surface. It is attached to a stalk which is 4cm in length. Histologically, the mass comprises a core of connective tissue covered with hyperplastic glandular epithelium of colonic type, with focal nuclear atypia. This is an example of a tubular colonic adenoma.

Further Information

Colorectal adenomas are intraepithelial neoplasms that characteristically display epithelial dysplasia. They are benign but are precursors to adenocarcinoma. Not all adenomas evolve into adenocarcinoma. They produce polyps (sometimes pedunculated) or sessile lesions or variable size. They occur predominantly in males and are more common in Western countries due to diet and lifestyle. They are present in about 30% of people over the age of 60 years in the West. There is an increased risk in patients with a positive family history of colorectal adenocarcinoma. Regular surveillance colonoscopy in at risk groups with polyp removal reduces incidence of adenocarcinoma. There are three classifications of colonic adenomas based on their architecture: tubular (>75% have a tubular morphology), tubulovillous (25-75% villous morphology) and villous (>75% have villous morphology). Histologically, they may have epithelial dysplasia characterized by nuclear hyperchromasia, elongation and stratification. Tubular adenomas tend to be small, pedunculated polys composed of rounded or tubular glands. Pedunculated adenomas have a slender fibromuscular stalk with blood vessels derived from the submucosa. The stalk is usually non-neoplastic epithelium. The size of the adenoma is the biggest predictor of progression to adenocarcinoma. Progression is rare in adenomas <1cm in diameter. However, up to 40% of lesions larger than 4cm in diameter progress to adenocarcinoma. Most adenomas are asymptomatic and slow growing. Large polyps may present with symptoms of anaemia from occult bleeding. Villous adenomas occasionally secrete large amounts of mucoid protein and/or potassium rich fluid, leading possibly to hypokalemia.

Intussusception of small bowel due to metastatic tumour

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02778

Clinical History

A 66-year-old woman suffered sudden onset of severe colicky central abdominal pain, somewhat relieved by drawing up her knees. She passed a stool containing mucus and blood („like redcurrant jelly“). On examination, there was a mass in the left hypochondrium, which hardened with each spasm of pain. The specimen was resected at laparotomy.

Pathology

The specimen is a segment of small bowel, approximately 20 cm in length, with attached mesentery up to 2 cm in width (more evident on the uncut aspect of the specimen). About 5 cm from the proximal surgical resection margin (which is at the left hand of the specimen), a polypoid tumour 3 cm in diameter has become invaginated into the lumen of the bowel, and has been propelled distally, forming an intussusception 13 cm in length. The tumour is seen at the apex of the intussusception (near the right hand side of the specimen). The congestion and exudate seen on the mucosal surface of the intussusception (invaginated portion) are features considered with early ischaemic necrosis. The histological diagnosis is not recorded in this case; however, the macroscopic appearance is consistent with a metastatic malignant tumour, although the possibility of a primary tumour cannot definitely be excluded.

Further Information

Intussusception of the small bowel is most common in children, in whom it is usually due to invagination of swollen lymphoid tissue (Peyer‘s patches) in the wall of the distal ileum. In adults, it is rare, causing only between 1 - 5 percent of cases of bowel obstruction. The usual cause a polypoid tumour, as seen in this specimen, acting as a pathological lead point being pulled forward by peristalsis, and thereby causing telescoping of the affected portion of bowel distally. Presentation may be of intermittent symptoms of bowel obstruction and in some cases excruciating pain. Classification of intussusception can be by causal pathology or by location. Abdominal CT scan will typically demonstrate a typical “target sign” with alternating hyper/hypodense layers.

Adult polycystic kidney disease

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02789

Clinical History

A 40-year old male attends his GP complaining of 2 weeks of haematuria and new onset of headache with blurred vision. His GP notes a blood pressure of 260/110 and refers the patient to hospital. The patient collapses on arrival to hospital. A CT brain shows a large subarachnoid haemorrhage from a ruptured ‘berry’ aneurysm. The patient dies shortly after admission.

Pathology

The specimen is an enlarged kidney. The renal parenchyma has been almost completely replaced by numerous dilated cysts varying in size, up to 3cm in diameter. The cysts have thin translucent walls, and some cysts contain material of varying colours, giving a ‘marble-like’ appearance to the cut surface of the kidney. The varying colours are caused by the secretions within the cysts, which may be admixed with haemorrhage. The external surface appears lobulated as a result of multiple projecting cysts. Any remaining renal parenchyma is severely atrophic caused by the pressure of the numerous cysts. This is an example of adult polycystic kidney disease.

Further Information

Adult polycystic kidney disease (APKD) is an autosomal dominant disorder characterised by the presence of multiple cysts within the renal parenchyma. The cysts develop from altered renal tubule epithelium. The cysts expand destroying the glomeruli, causing ischaemia, pressure atrophy, and eventually leading to renal failure. APKD occurs in 1 in 40 to 1000 live births. Mutations in the PKD1 gene on chromosome 16p13.3 and PKD2 gene on chromosome 4q21 have been described as causal mutations. These code for membrane proteins polycystin 1 and 2, respectively. Patients with PKD1 mutation are more common and have a more severe phenotype. End stage renal disease (ESRD) occurs at a mean age of 74.0 in PKD2 versus 54.3 years in PKD1. Common symptoms of APKD include haematuria from haemorrhage into cysts and pain or a sensation of dragging from the expansion of cysts and kidney enlargement. Many patients remain asymptomatic until features of renal failure occur such as proteinuria, polyuria, hypertension and uraemia. Extrarenal manifestations of the disease include intracranial ‘berry’ aneurysms, hepatic and pancreatic cysts, as well as mitral valve prolapse and other types of cardiac valve disease. Renal ultrasound is the most common investigation used to diagnose APKD. CT- and MRI scans may also be used as diagnostic tools. Patients with a positive family history of APKD can be offered screening renal US scans and genetic testing in some cases. Treatment involves renal replacement therapy for ESRD and renal transplant (if a donor can be found). Ultimately, over one-third of patients die from renal failure and one-third from coronary or hypertensive heart disease. Approximately 1% of patients die from subarachnoid haemorrhage, because of berry aneurysm rupture (as in this case). Remaining deaths are due to unrelated causes.

Multiple Renal Calculi

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02794

Clinical History

A 68-year old male presented with fevers and rigors. Further questioning reveals a 6-month history of intermittent bilateral flank pain and haematuria. Biochemical investigations reveal significantly impaired renal function with a normal serum calcium. A CT abdomen showed bilateral hydronephrosis with multiple renal calculi as well as perinephric and subphrenic abscesses. He later died from progressive renal failure.

Pathology

The specimen is patient’s kidney, which is grossly and partially bisected. There is gross dilatation of the pelvi-calyceal system visible. Significant atrophy of renal tissue can be seen, in some places being reduced to a mere rim. A large mottled brown-white calculus lies in the pelvis, and a smaller calculus occludes the ureter lumen. The ureter is dilated proximal to the impacted calculus. There are multiple calculi visible within the calyces of the specimen.

Further Information

Urolithiasis (renal calculi) is a very common disease affecting up to 1 in 10 individuals during their lifetime. Formation of the stones can occur anywhere along the urinary tract but most commonly occurs within the kidneys. Risk factors for stone formation include male gender; any condition that affects the composition of the urine, such as hypercalciuria or high urine oxalate; systemic metabolic disorders, such as cystinuria and gout; dietary factors, such as high oxalate and animal protein intake, low fluid intake; and environmental factors, such as high dry temperatures. 80% of renal calculi are unilateral. Symptoms of urolithiasis include excruciating pain, haematuria, nausea, vomiting, fainting, dysuria and urgency. Symptoms depend on the size and the site of the calculus. Urolithiasis can be asymptomatic especially if the stones are formed and remain within the renal pelvis or bladder. Symptoms occur when the stones move into the ureter. Pain from calculi is usually colicky and typically severe in nature; occurring in paroxysms. The flank is the most common site for pain but pain can occur anywhere along the urinary tract and into the genitals. Pain resolves on passage of the stone. Haematuria can be gross or microscopic. There are four main types of renal calculi: Calcium stones are the most common, comprising 70% of all stones. They are made up of calcium oxalate or a mixture with calcium phosphate. Hypercalciuria, hypercalcemia and hyperoxaluria are common causes of these stones. Struvite stones make up 5-10% of stones. They are comprised of magnesium ammonium phosphate. These are commonly formed as a result of proteus infections and lead to the formation of very large “staghorn” calculi. Uric acid stones make up 5-10% of calculi. These occur in patients with hyperuricemia, such as gout and chronic leukaemias. The remainder are made up of cysteine, which is due to impaired renal reabsorption of amino acids such as cystine.

Hydrocoele

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02808

Clinical History

With a history of diabetes and previous myocardial infarctions. On examination bilateral pleural effusion, peripheral pitting oedema and a swollen scrotum were noted. Transillumination of the swelling transmitted red light. Chest x-ray showed radiological features of congestive cardiac failure. He was commenced on appropriate treatment for heart failure but later died during this admission.

Pathology

The specimen is a testis and its coverings, sliced to display the cut surface. The cavity bounded by the visceral and parietal layers of the tunica vaginalis is distended due to the accumulation of serous fluid. This is an example of a hydrocoele, secondary to generalised oedema due to congestive cardiac failure.

Further Information

A hydrocele is an accumulation of serous fluid between the parietal and visceral layers of the tunica vaginalis around the testes. Hydroceles can be described as communicating with the peritoneal cavity or noncommunicating with the peritoneal cavity. Communicating hydroceles develop as a result of failure of the processus vaginalis to close after the descent of the testes into the scrotum. These may present after birth as a congenital hydrocele or may present later in life due to increase in intra-abdominal pressure, such a cardiac failure in this case. Non-communicating hydroceles are caused by imbalances in fluid secretion and reabsorption e.g. orchitis, epididymitis, testicular tumour, physical trauma (e.g. hernia, testicular torsion) or defective lymphatic drainage (e.g. filariasis, elephantiasis). Patients present with a scrotal mass. The mass may be uni- or bilateral. Communicating hydroceles may be reducible and increase in size with raised intra-abdominal pressure. Non-communicating are usually nonreducible swellings. The swelling is usually non tender unless there is an underlying infection or torsion causing the hydrocele. Larger hydroceles may be cumbersome and cause erosion and skin infections on the scrotum. Diagnosis can be made on physical examination. Serous fluid allows the passage of light shined through the scrotum when examined: this is called transillumination. Ultrasound may be used to consolidate diagnosis and exclude other testicular pathology. Testicular cancer serum markers, such as alpha fetoprotein and B-HCG, may be taken to exclude testicular cancer. Many congenital hydroceles resolve spontaneously before the age of 2. If communicating hydroceles persist beyond 2 year they are surgically repaired in order to reduce the risk of developing incarcerated hernias. Surgical repair of communicating hydroceles in older patients may be offered if they are symptomatic. Treatment of the underlying aetiology of reactive hydrocele may cause them to resolve.

Hydronephrosis Hydroureter

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02791

Clinical History

A 49-year old male presents with a 6-week history of malaise, urinary frequency and haematuria for 6 weeks. Further questioning revealed intermittent left flank pain. Abdominal ultrasound showed severe hydro-nephrosis and hydroureter, secondary to multiple obstructing ureteric calculi at the uretero-vesical junction. He underwent a left nephrectomy and ureterectomy, and made a successful recovery.

Pathology

This is the patient’s left nephrectomy and ureterectomy specimen. The kidney has been bisected and the cut surface of both halves is displayed, mounted in continuity with the ureter, which has been opened. The kidney is grossly hydronephrotic, and there is considerable atrophic thinning and loss of renal parenchymal tissue. The ureter is extremely dilated and distally contains a number of small brown-black calculi with irregular sharp surface projections. These are calcium oxalate stones. This is an example of hydronephrosis and hydroureter due to calculi obstructing the lower end of the ureter.

Further Information

Hydronephrosis, or obstructive uropathy, is the dilation of the renal pelvis and calyces caused by an obstruction in the urine outflow. Obstruction can occur at any point in the urinary tract. Any lesion-, intrinsic (within the outflow system) or extrinsic (outwith the ureter)-, that impedes the flow of urine can lead to hydronephrosis. Common causes include: congenital anomalies, urinary calculi, urinary tract tumours, urinary tract inflammation, prostatic hypertrophy, and prostate tumours. Symptoms of the hydronephrosis relate to the pathology causing Pathology This is the patient’s left nephrectomy and ureterectomy specimen. The kidney has been bisected and the cut surface of both halves is displayed, mounted in continuity with the ureter, which has been opened. The kidney is grossly hydronephrotic, and there is considerable atrophic thinning and loss of renal parenchymal tissue. The ureter is extremely dilated and distally contains a number of small brown-black calculi with irregular sharp surface projections. These are calcium oxalate stones. This is an example of hydronephrosis and hydroureter due to calculi obstructing the lower end of the ureter. Clinical History A 49-year old male presents with a 6-week history of malaise, urinary frequency and haematuria for 6 weeks. Further questioning revealed intermittent left flank pain. Abdominal ultrasound showed severe hydro-nephrosis and hydroureter, secondary to multiple obstructing ureteric calculi at the uretero-vesical junction. He underwent a left nephrectomy and ureterectomy, and made a successful recovery. Hydronephrosis Hydroureter the obstruction (e.g. renal colic pain with calculi), the time period of the obstruction (acute or chronic), the site (unilateral or bilateral) and whether it is complete or partial. If the obstruction is not relieved it will ultimately cause pressure to build up proximal to the obstruction. This pressure is transmitted in a retrograde manner through the collecting ducts to the cortex causing progressive atrophy of the kidney with

dilatation of the renal calyces and pelvis. The pressure also compresses the vasculature in the medulla leading to ischaemic medullary damage. Glomerular filtration persists in the affected kidney until late in the disease process when the filtration gradually diminishes or ceases. Obstruction triggers an interstitial inflammatory process leading to fibrosis. Ultrasound is the key diagnostic tool for diagnosis followed by CT or urogram. Most obstructing lesions require surgical intervention to relieve the blockage. Surgical interventions depend on each individual cause, but include nephrostomy or stenting for upper urinary tract obstruction and urinary catheter or suprapubic catheter insertions for lower urinary tract obstructions.

Hydronephrosis and Hydroureter Caused by Obstruction by a Renal Calculus

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02793

Clinical History

A 72-year old female presented with colicky flank pain and increasing malaise. Intermittent haematuria was noted. Biochemical investigations reveal significantly impaired renal function. CT abdomen showed congenital renal agenesis of the left kidney and a right-sided hydronephrosis and hydroureter, due to obstruction by a smaller calculus. Percutaneous lithotomy was attempted to relieve the obstruction, but the patient died of a cardiac event during the procedure.

Pathology

The specimen is patient‘s right kidney, which is grossly and partially bisected. There is gross dilatation of the pelvi-calyceal system visible and significant atrophy of renal tissue particularly in the cortex. There is a large brown calculus visible in the renal pelvis at the ureteropelvic junction.

Further Information

Urolithiasis (renal calculi) is a very common disease affecting up to 1 in 10 individuals during their lifetime. Formation of the stones can occur anywhere along the urinary tract but most commonly occurs within the kidneys. Risk factors for stone formation include male gender; any condition that affects the composition of the urine, such as hypercalciuria or high urine oxalate; systemic metabolic disorders, such as cystinuria and gout; dietary factors, such as high oxalate and animal protein intake, low fluid intake; and environmental factors, such as high dry temperatures. 80% of renal calculi are unilateral. Symptoms of urolithiasis include excruciating pain, haematuria, nausea, vomiting, fainting, dysuria and urgency. Symptoms depend on the size and the site of the calculus. Urolithiasis can be asymptomatic especially if the stones are formed and remain within the renal pelvis or bladder. Symptoms occur when the stones move into the ureter. Pain from calculi is usually colicky and typically severe in nature; occurring in paroxysms. The flank is the most common site for pain but pain can occur anywhere along the urinary tract and into the genitals. Pain resolves on passage of the stone. Haematuria can be gross or microscopic. Diagnosis can be made based on the medical history and examination. Radiological tools frequently used to assist diagnosis include non-contrast CT or ultrasound of the kidneys and bladder. Less commonly used imaging methods include abdominal X-ray, intravenous pyelogram and magnetic resonance imaging. If left untreated renal damage and ultimately renal failure from progressive obstruction and hydronephrosis will occur. If the obstructing calculus is not relieved it will cause pressure to build up proximal to the obstruction. This pressure is transmitted back through the collecting ducts to the cortex causing progressive atrophy of the renal parenchyma with dilatation of the renal calyces and pelvis. The pressure also compresses vasculature in the medulla leading to ischaemic medullary

damage. Glomerular filtration persists in the affected kidney until late in the disease process when it will gradually diminish. Obstruction triggers an interstitial inflammatory process leading to fibrosis. Renal calculi also predispose patients to infection secondary to obstruction and the trauma that they cause to the urothelium. Treatment in acute patients include supportive treatment to allow the passage of the stone. Medical treatment used includes analgesia, commonly NSAIDs and opiates, and agents to aid passage of the stone, such as alpha blockers, calcium channel blockers and antispasmodics. Surgical intervention may be required if there are severe complications due to calculi or if the stone is large and unable to be expelled with conservative treatment. Surgical interventions include lithotripsy (using lasers or electricity), laparoscopic stone removal or percutaneous stone removal. Open surgery is rarely required.

Multinodular goitre

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02800

Clinical History

A 53-year-old female presented with an abnormal swelling in the neck and a persistent cough. She complained of lethargy and weight gain over the previous few years. Whilst being investigated she died of unrelated cardiovascular disease several months later.

Pathology

The specimen, removed at post-mortem, includes the base of the tongue, larynx and trachea. It has been cut in the coronal plane to allow a view of the internal laryngeal and tracheal anatomy. The thyroid gland is grossly enlarged particularly the right lobe, which extends superiorly and inferiorly, well beyond its normal margins when viewed from the anterior aspect. The cut posterior surfaces display many hyper- and hypopigmented nodules as well as cystic areas in both lobes. The tongue base, larynx and trachea appear relatively normal.

Further Information

Nodular goitre is most often detected simply as a mass or swelling in the neck but depending on size and location of growth may produce pressure symptoms on the trachea and the oesophagus. There may be difficulty in breathing, dysphagia, cough, and hoarseness. Paralysis of the recurrent laryngeal nerve may occur by an expanding goitre, but this is rare. Symptoms suggesting obstruction of the trachea including cough, stridor and shortness of breath may occur. Occasionally tenderness and a sudden increase in goitre size arise due to cystic expansion or haemorrhage into a nodule[1]. Causes of goitre include autoimmune disease (Hashimoto’s thyroiditis, Grave’s disease), the formation of one or more thyroid nodules and iodine deficiency. Goitre occurs when there is reduced thyroid hormone synthesis secondary to biosynthetic defects and/or iodine deficiency, leading to increased thyroid stimulating hormone (TSH). This stimulates thyroid growth as a compensatory mechanism to overcome the decreased hormone synthesis. Elevated TSH is also thought to contribute to an enlarged thyroid in the goitrous form of Hashimoto thyroiditis in combination with fibrosis secondary to the autoimmune process in this condition. In Grave’s disease, the goitre results mainly from stimulation by the TSH receptor antibody[1].

Reference: 1. Hughes et al. (2012) Goitre: Causes, investigation and management. Aust Family Physician, 41, 572-576.

Retrosternal Goiter

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02801

Clinical History

A 60-year-old female presented with an abnormal swelling in the neck, persistent cough and difficulty swallowing. She gained weight over the previous few years. She died of unrelated cardiovascular disease and the specimen was obtained at post-mortem.

Pathology

The specimen, removed at post-mortem, includes the larynx, trachea and large multilobular thyroid gland. The thyroid gland is grossly enlarged particularly the right lobe, which has two large lobes extending superiorly and inferiorly for a range of 7-8mm, well beyond its normal margins when viewed on anterior aspect. Posteriorly, the oesophagus has been opened to expose the posterior wall of the trachea. The right lobe presents as larger than from the anterior perspective, and the abnormal growth appears to be mainly the inferior pole of the right lobe. The surfaces do not display major pigmentary changes. Prominent veins are visible on the surface of the right lobe.

Further Information

Goitre is most often detected simply as a mass or swelling in the neck but depending on the size and location of growth it may produce pressure symptoms on the trachea and the oesophagus. There may be difficulty in breathing, dysphagia, cough, and hoarseness. Paralysis of the recurrent laryngeal nerve may occur by an expanding goitre, but this is rare. Symptoms suggesting obstruction of the trachea including cough, stridor and shortness of breath may occur. Occasionally tenderness and a sudden increase in goitre size arise due to cystic expansion and haemorrhage into a nodule [1]. Causes of goitre include autoimmune disease (Hashimoto’s thyroiditis, Grave’s disease), the formation of one or more thyroid nodules and iodine deficiency. Goitre occurs when there is reduced thyroid hormone synthesis secondary to biosynthetic defects and/or iodine deficiency, leading to increased thyroid stimulating hormone (TSH). This stimulates thyroid growth as a compensatory mechanism to overcome the decreased hormone synthesis. Elevated TSH is also thought to contribute to an enlarged thyroid in the goitrous form of Hashimoto thyroiditis in combination with fibrosis secondary to the autoimmune process in this condition. In Grave’s disease, the goitre results mainly from stimulation by the TSH receptor antibody[2].

Reference: 1. Hughes et al. (2012) Goitre: Causes, investigation and management. 2. Aust Family Physician, 41, 572-576.

Congenital Pulmonary Stenosis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02737

Clinical History

This male child had a cardiac murmur discovered at birth. He remained well until the age of 10 months. Two weeks before admission to hospital, he became languid, developed a slight cough, and suddenly gained a lot of weight. Ten days before admission he developed swelling of the hands, feet and face. On examination, he was a chubby child with a blood pressure of 90/59 mmHg. There was a fine thrill over the whole precordium, and a harsh systolic murmur maximal in the pulmonary area. There was oedema of the face and legs. After admission the child gained further weight, and there was no response to therapy. Death was due to congestive cardiac failure.

Pathology

The specimen is the child‘s heart. View from the left side and note the pulmonary artery has been opened to display the upper surface of the pulmonary valve. This abnormal valve consists of a thickened conical diaphragm, with an opening 2 mm in diameter at the apex. The opened pulmonary artery has a large post-stenotic dilatation. There is rightsided cardiac enlargement due to marked dilatation of the right atrium and right auricle (opened), and right ventricular hypertrophy (the wall has been cut in two places [one penetrating, one one not] to expose the hypertrophied myocardium). This is a pure pulmonary valve stenosis.

Further Information

Pulmonary stenosis represents about 7% of congenital heart disease. However, it can also occur later in life secondary to increased pulmonary pressures. In neonates, it may occur as an isolated defect or as part of a more complex lesion, such as the Tetralogy of Fallot. The latter is a combination of four congenital abnormalities, including a ventricular septal defect, pulmonary valve stenosis, a misplaced aorta and a right ventricular hypertrophy. Abnormalities similar to this specimen can now be corrected surgically.

The natural course of congenital pulmonary stenosis varies with age and severity at age of development. Pulmonary stenosis can be thought of on a scale of mild, moderate and severe. While mild pulmonary stenosis can remain asymptomatic and indolent without diagnosis for several years, moderate pulmonary stenosis can rapidly progress to severe stenosis. Severe pulmonary stenosis has significant effects on the heart due to increased pressures leading to right ventricular hypertrophy. Severe pulmonary stenosis is often associated with right ventricular outflow obstruction and subsequent cardiac failure. Thus, moderate to severe pulmonary stenosis may manifest as dyspnoea, chest pain or syncope.

Other complications of severe pulmonary stenosis, include infective endocarditis and arrhythmias caused by remodelling and scarring of the ventricular and atrial walls. Diagnosis of pulmonary stenosis is often achieved through transthoracic echocardiography. Other imaging modalities include MRI and CT.

Ulcerative Colitis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02775

Clinical History A 36-year old female was admitted to hospital with a 3-week history of bloody diarrhoea and lower abdominal pain. Further history taking revealed 4 similar episodes of diarrhoea and abdominal pain of over the past 7 years. Sigmoidoscopy showed a erythematous, ulcerated and oedematous rectal mucosa. She was commenced on steroid treatment but her symptoms failed to improve. She underwent a total colectomy. Pathology The resected colon has been sliced open longitudinally to show the mucosal surface. There is extensive confluent ulceration separated by oedematous islands of residual mucosa. The ulcers have necrotic bases with overhanging edges some of which form ‘pseudo’-polyps. Histology of the bowel mucosa showed acute inflammatory changes with crypt abscesses, focal necrosis and ulceration. This is an example of acute ulcerative colitis (UC). **Further Information** Ulcerative colitis is a chronic ulcero-inflammatory disease that usually involves the rectum and can extend proximally in a continuous pattern to involve other parts to the colon. The inflammatory process is diffuse but is generally limited to the mucosa and superficial submucosa. The triggering factor for UC in unknown. It most commonly begins between the ages of 15-25 years and is slightly more common in females. UC patients can present with diarrhoea, which may contain blood or mucus, faecal urgency, faecal frequency, tenesmus, colicky abdominal pain, as well as weight loss, anaemia and fatigue. Colonic inflammation can lead to toxic megacolon, colonic perforation or colon cancer. Extraintestinal manifestations include anterior uveitis, migrating polyarthritis, sacroileitis, ankylosing spondylitis, erythema nodosum, pyoderma gangrenosum and primary sclerosing cholangitis. Treatments of UC include use of anti-inflammatory drugs such as steroids, disease modifying rheumatoid drug and TNF (tumor necrosis factor) inhibitors. Colectomy essentially cures intestinal symptoms of UC but extraintestinal manifestations of the disease may persist.

Osteochondroma

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02769

Clinical History

A 61-year old male with prostate cancer attends pre-assessment clinic prior to a prostatectomy. Overall, he feels well with no major complaints. On review of symptoms, it is noted he has chronic pain in his right knee, which his GP called osteoarthritis. To exclude boney metastases of the prostate carcinoma, a knee x-ray is ordered, which shows a pedunculated lesion projecting from the medial aspect of the diaphysis of the right femur. His prostatectomy goes ahead but he subsequently dies from a postoperative pulmonary embolism.

Pathology

The specimen is the lower end of the patient’s right femur, which has been cut in the coronal plane and mounted to display the external surfaces. A pedunculated bony protuberance 2 cm in length projects from the medial aspect of the femoral shaft 7 cm above the medial condyle. The projection is composed of normal bone with a thin cap of hyaline cartilage at the tip. This is an example of an osteochondroma.

Further Information

An osteochondroma (or an exostosis) is a benign cartilaginous tumour. They are comprised of a cartilaginous capped bony protrusion from the external surface of the bone from which they arise. They are the most common benign bone tumours. Most osteochondromas occur spontaneously but they may also occur as part of multiple hereditary exostosis syndrome or post radiotherapy. They usually develop from or near the growth plate. They most commonly arise from the appendicular skeleton, especially in the lower limb around the knee or the upper limb at the proximal humerus. Men are more commonly affected than women.

Symptoms vary on the site and size of the growth. Many osteochondroma remain asymptomatic. Osteochondroma lead to symptoms from the compression of surrounding neurovascular structures. They may also cause a pain from myositis or a fracture of the bony spur. They usually present in the second decade of life. They can be diagnosed with plain x-ray but MRI is the gold standard to ensure that there is no malignancy present within the growth.

Hereditary exostoses are associated with mutations in the EXT1 and EXT2 genes. Reduced expression of these genes has also been seen in sporadic osteochondromas. Osteochondromas stop forming as fusion of the growth plate occurs. Treatment of excision is only if symptoms are severe. Malignant transformation to chondrosarcoma is rare in sporadic cases but more common in hereditary exostosis (5-20%).

Cholecystitis and Cholelithiasis

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02783

Clinical History

A 60-year-old man head a history of four episodes of severe gripping abdominal pain during the previous year, each lasting two hours and associated with meals. He presented with a similar attack associated with vomiting and fever. This last attack did not resolve spontaneously, and he underwent cholecystectomy.

Pathology

A thick-walled gallbladder has been opened to display a thickened haemorrhagic mucosa and many irregular faceted calculi. A large calculus is impacted in the neck of the gallbladder. The serosal surface of the gall bladder is congested and has lost its normal sheen. This is an example of cholecystitis complicating cholelithiasis (gallstones).

Further Information

Gallstones account for the vast majority of acute cholecystitis, with only 5 - 10% of cases being due to other pathology. Chronic cholecystitis may occur, resulting from recurrent attacks and causing fibrosis and thickening of the gallbladder wall. 6-11% of patients with symptomatic gallstones will go on to develop acute cholecystitis. Serum biochemistry will demonstrate leucocytosis with or without obstructive liver function tests. Ultrasound will demonstrate gallstones in the gallbladder, along with wall thickening and a sonographic Murphy’s sign (tenderness from the pressure of the ultrasound probe). Other imaging modalities include nuclear medicine cholescintography scans, MRCP (magnetic cholangiopancreatography) and CT. Endoscopic Retrograde Cholangiopancreatography (ERCP) will provide diagnostic information regarding biliary obstruction and may also be therapeutic. Causative organisms (if present) will be from the gut flora, commonly E coli, Enterococcus, Klebsiella and Enterobacter. Complications include gangrenous cholecystitis, perforation, cholecystoenteric fistula or gallstone ileus. Definitive treatment is surgical cholecystectomy.

Septic Renal Infarct

Price inquiry: +48 605999769, kontakt@openmedis.pl

Product code: AM02797

Clinical History

A 54 year old male patient presents with flank pain. He is an active intravenous drug user. Further questioning reveals a history of intermittent haematuria, fevers, malaise and vomiting. On examination he is hypertensive and pyrexic. Inspection of his limbs reveals Janeway lesions on his extremities and track marks from recent IV drug use. A systolic murmur is found on auscultation of his chest. Blood tests reveal elevated inflammatory markers, impaired renal function, elevated LDH and multiple bacteraemic blood cultures. Echocardiogram shows a large mobile tricuspid vegetation. He was commenced on treatment for infective endocarditis but later died from a sudden cardiac arrest.

Pathology

The specimen is the patient’s kidney from post mortem examination. The kidney has been bisected with a cut half surface on display. There are multiple well demarcated wedge shaped pale yellow-white areas evident within the cortex. The base of these pyramids lies against the cortical surface and extend along the renal columns with the apex pointing toward the medulla. The largest is evident lateral upper pole of the kidney. Theses pale areas are infarcted renal tissue. There are dark irregular shaped areas which represent areas of hemorrhage.

Further Information

Renal infarction results from an interruption in the blood flow to the kidney. The kidneys receive almost a quarter of the cardiac output but have limited collateral circulation. The cortex is the most susceptible area to infarction given blood supply is from proximal to distal. The main causes of interruption of this circulation are cardioembolic disease, renal artery damage, hypercoagulable states or idiopathic. Cardioembolic causes are the most common. These include postmyocardial infarction mural thrombi, septic emboli from infective endocarditis and emboli from mechanical valves. Idiopathic renal infarction is the second most common cause. Damage to the renal artery is the third most frequent cause and includes renal artery dissection, acute vasculitis of polyarteritis nodosa, trauma or post endovascular intervention. Hypercoagulable states are the rarest cause of renal infarcts such as hereditary thrombophilia and antiphospholipid syndrome. Infarction is bilateral in ~15% of cases. Presentation of renal infarction depends on the underlying etiology. It can be clinically silent. Common manifestations include costovertebral angle pain, haematuria, hypertension due to increased renin release, nausea, vomiting and sometimes fever. Laboratory test used to aid diagnosis include urinalysis for hematuria and serum creatinine levels which may be elevated, especially in bilateral disease. CT abdomen with contrast is the first choice radiological investigation. A wedge-shaped perfusion defect is the classic finding. Treatment varies depending on the cause of the infarction but generally involves supportive therapy and treatment of the underlying pathology

Calcified Aortic Valvular Stenosis Bicuspid Aortic Valve

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Product code: AM02741

Pathology

The specimen is partial horizontal 1.5cm slice through the plane of the left atrium whose smooth internal lining together with the left auricular appendage and part of the left ventricle are visible on the inferior aspect. On the superior aspect the pulmonary trunk (and part of the pulmonary tricuspid valve) and aorta, including the affected abnormal bicuspid valve, are clearly discernible. Calcified aggregations or thickenings on the opposing margins of the valve can be seen from this upper perspective. There is also a region of calcification on one of the cusps of the pulmonary valves.

Further Information

Bicuspid aortic valve is a common congenital abnormality that is often not detected into adulthood. Indeed, it is the most common congenital anomaly of the heart. Aortic valve stenosis can range from mild to severe, and signs and symptoms generally develop when narrowing of the valve is severe. Some people with aortic valve stenosis may not experience symptoms for many years. Signs and symptoms may include abnormal heart sound (heart murmur), radiating chest pain (angina), shortness of breath and chest pain, especially during times of increased activity, heart palpitations — sensations of a rapid, fluttering heartbeat. The heart-weakening effects of aortic valve stenosis may lead to heart failure. Heart failure signs and symptoms include fatigue, shortness of breath, and swollen ankles and feet.

When the aortic valve is narrowed, the left ventricle has to work harder to pump a sufficient amount of blood into the aorta and onward to the rest of the body. This can cause the left ventricle to thicken and enlarge. Eventually the extra work of the heart can weaken the left ventricle and the heart overall, with it ultimately not being able to function properly (heart failure), causing other downstream problems.

Calcium deposits build up on the valve particularly those with a congenitally abnormal aortic valve, such as a bicuspid aortic valve resulting in stiffening of the valve cusps. This stiffening narrows the aortic valve lumen and thereby increases the blood flow.