Craniopharyngioma

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

Product code: AM02719



Clinical History A 62-year-old woman presented with disorientation to time, place and person. Physical examination revealed no localised neurological signs. Radiological investigations revealed a space occupying lesion in the floor of the 3rd ventricle. Tissue was removed at surgery but the lesion could not be completely excised. Histology confirmed the diagnosis of Craniopharyngioma. Post-operatively the patient developed complex metabolic disturbances, probably hypothalamic in origin. She gradually deteriorated, and 10 weeks after admission she died following an episode of gastric aspiration. Pathology The brain has been sectioned in the sagittal plane, displaying the medial surface. A pink-grey, ovoid tumour measuring 2.5 x 1.5 cm on the cut surface is centred in the region of the hypothalamus. It is encapsulated except at its ventral pole where tissue has been removed at previous surgery, and the cut surface reveals a microcystic or spongy appearance. The tumour distorts the 3rd ventricle and extends to obliterate the Foramen of Munro. The optic chiasm is displaced caudally (arrow). Previous ventriculo-atrial shunting has prevented dilatation of the lateral ventricles despite this obstruction. Further Information Craniopharyngiomas constitute 1-3% of all brain tumours, and 5-10% in children, with a bimodal distribution favouring ages 5-14 years, and a second peak between ages 50-75 years. There is a higher incidence in Japan and parts of Africa. Craniopharyngiomas are epithelial tumours generally arising from the pituitary stalk. Other sites of origin include the sella turcica, optic system and third ventricle. There are frequently solid and cystic components, the latter containing cholesterol crystals. Craniopharyngiomas can be divided into two categories - adamantinomatous and papillary types, each with distinct histology and genetic alterations, although the prognostic significance of these types remains unclear. Treatment includes surgical resection and radiation therapy (RT) to treat and post-surgical residual disease. Prognosis depends largely on tumour resection, control and treatment-related complications arising from local and endocrine and local sequelae.

Metastatic melanoma

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

Product code: AM02720



Clinical History In the 1970s, a 31-year-old woman presented with severe headache and diplopia on a background of having a pigmented skin lesion (diagnosed as an invasive skin melanoma) removed from her neck 8 months earlier. Clinical examination revealed no abnormality, and following discharge the patient was later re-admitted with persistent vomiting. Her condition deteriorated and she died. Pathology This specimen demonstrates widespread intracerebral melanoma metastases. The inferior surface is characterised by many elevated dark nodules up to 1.5 cm in diameter. Similar lesions are present on the cut superior surface where it is seen that these secondary melanotic deposits are confined exclusively to the grey matter. The tumour deposits are not encapsulated and are invading the cortex. Some necrosis and haemorrhage is present. Further Information Of all patients who have metastatic disease to the brain, 10% are from skin melanoma. Risk increases with age over 60 years, male gender, disease duration and more advanced tumour/ metastatic stage. BRAF and NRAS mutations, expression of CCR4 receptors on tumour cells, and activation of the PI3K pathway are all risk factors for the development of cerebral metastasis. 80% of melanoma brain metastases are supratentorial. Presentation is often with headache, neurologic deficits and/or seizures. Furthermore, these lesions are at risk of spontaneous haemorrhage. Modern diagnosis is based on neuroimaging and often histology of a stereotactic brain biopsy, if no previous diagnosis has been made. Treatment includes stereotactic radiosurgery (SRS), radiotherapy and/or systemic therapy with “checkpoint inhibitor immunotherapy” or targeted treatments. This has improved median survival upto 11 months in recent years.

Glioma grade 3-4, causing papilloedema

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

Product code: AM02698



Clinical History

The patient was a female aged 24 years, who presented 18 months before death with an abnormal electroencephalogram (EEG) after a single epileptic fit. Six months later she complained of blurred vision and headaches. Bilateral papilloedema was seen on ophthalmoscopy; however, there were no localising signs to explain this. Further investigations showed a space occupying mass, which was biopsied and histologically diagnosed as a Grade III-IV glioma. The patient was treated with radiotherapy. A month after treatment commenced, the patient experienced weakness of the left arm and leg. Soon afterwards she was admitted with drowsiness and vomiting; thereafter, she rapidly and died.

Pathology

The specimen shows a large intracerebral lesion, which has obliterated the lateral ventricles and the inner 2/3 of the internal capsule and basal ganglia on the right side. It is infiltrating across the corpus callosum and distorting the aqueduct. The tumour is fairly well demarcated and vascular with numerous areas of haemorrhage and necrosis, causing its mottled variegated appearance.

Glioblastoma multiforme

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

Product code: AM02718



Clinical History A 56-year old male presented with a generalised seizure. He remained unconscious after this seizure and later died. A collateral history revealed 6 months of progressive confusion, short-term memory loss and personality change. Pathology Coronal sections of the brain post mortem show a 4cm necrotic and haemorrhagic tumour. Tumour invasion from the inferior frontal lobe into the lateral ventricle is apparent. Meningeal spread is evident on examination of the posterior aspect of the specimen. Further Information Gliomas are the second most common cancer of the central nervous system after meningiomas. The term “glioma” refers to tumours that are histologically similar to normal glial cells i.e. astrocytes, oligodendrocytes and ependymal cells. They arise from a progenitor cell that differentiates down one of the cell lines. Glioblastoma multiforme (GBM; also called glioblastoma) develop from the astrocyte lineage. GBMs can arise in the brain “de novo” or evolve from lower-grade astrocytomas or oligodendrogliomas. GBM is often referred to as a grade IV astrocytoma. They are differentiated histologically from anaplastic astrocytomas by necrotising tissue surrounded by anaplastic cells as well as by the presence of hyperplastic blood vessels. GBMs are more common in males. It is most commonly diagnosed in the 6th decade of life. Genetic risk factors include neurofibromatosis type 1 and Li-Fraumeni syndrome. Previous brain radiotherapy is also associated with increased risk of GBM. Symptoms vary depending on the location of the GBM, but may include any of the following: • Persistent headaches • Double or blurred vision • Vomiting • Loss of appetite • Changes in mood and personality • Changes in ability to think and learn • New onset of seizures • Speech difficulty of gradual onset. Diagnostic tools include computed tomography (CT scan) and magnetic resonance imaging (MRI). Around 50% of these tumours occupy more than one cerebral hemisphere. GBMs commonly extend into the ventricular walls or meninges, and thus into the central spinal fluid (CSF). Spinal cord spread is uncommon. Metastasis beyond the central nervous system is rare. Tumour growth causes cerebral oedema leading to increased intra-cranial pressure. These are biologically aggressive tumours, and if left untreated survival is typically 3 months. The mainstay of treatment for GBMs is surgery, followed by radiation and chemotherapy.

Glioblastoma multiforme

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

Product code: AM02717



Clinical History

Over a 3-year period, a 57-year-old woman had intermittent frontal headache and memory disturbance with progression to psychiatric disturbance, and ultimately vomiting and meningeal signs. Localising neurological signs only developed late in the course of the disease.

Pathology Coronal section through the cerebral hemisphere demonstrates a round, haemorrhagic, variegated tumour in the left temporal lobe. Less welldefined tumour tissue extends across the mid-line replacing the corpus callosum. The ventricular system has been almost totally obliterated. Further sections through the cerebral hemisphere confirmed that these apparently separate lesions are extensions of one massive tumour. Further Information Gliomas are the second most common cancer of the central nervous system after meningiomas. The term “glioma” refers to tumours that are histologically similar to normal glial (macroglial) cells* i.e. astrocytes, oligodendrocytes and ependymal cells. They arise from a progenitor cell that differentiates down one of the cell lines. GBMs can arise in the brain “de novo” or evolve from lower-grade astrocytomas or oligodendrogliomas. GBM is often referred to as a grade IV astrocytoma. They are differentiated histologically from anaplastic astrocytomas by necrotising tissue surrounded by anaplastic cells as well as by the presence of hyperplastic blood vessels. GBMs are more common in males. It is most commonly diagnosed in the 6th decade of life. Genetic risk factors include neurofibromatosis type 1 and Li-Fraumeni syndrome. Previous brain radiotherapy is also associated with increased risk of GBM. Symptoms vary depending on the location of the GBM, but may include any of the following: • Persistent headaches • Double or blurred vision • Vomiting • Loss of appetite • Changes in mood and personality • Changes in ability to think and learn • New onset of seizures • Speech difficulty of gradual onset Diagnostic tools include computed tomography (CT scan) and magnetic resonance imaging (MRI). Around 50% of these tumours occupy more than one cerebral hemisphere. GBMs commonly extend into the ventricular walls or meninges, and thus into the central spinal fluid (CSF). Spinal cord spread is uncommon. Metastasis beyond the central nervous system is rare. Tumour growth causes cerebral oedema leading to increased intra-cranial pressure. These are biologically aggressive tumours, and if left untreated survival is typically 3 months. The mainstay of treatment for GBMs is surgery, followed by radiation and chemotherapy. *Microglia are a different lineage from macroglia. The former are related to the macrophage lineage, and arise initially from the yolk sac and later in development from the bone marrow.

Pituitary Adenoma

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Product code: AM02708



Clinical History

A 29-year old male presented with a 22 month history of headaches and blurred vision. Examination revealed a bi-temporal hemianopia and a left VIth nerve palsy. Skull X-ray showed erosion of most of the sphenoid body with some dorsum sellae and anterior clinoid process intact. Carotid angiography showed upward and lateral displacement of the anterior and middle cerebral arteries. Pneumoencephalography (a common imaging procedure used until the 1970s in which CSF is drained and replaced by air, oxygen or helium that acted as a contrast medium in X-ray examinations) showed upward displacement of the lateral and third ventricles from below. A craniotomy was performed but the patient died immediately afterwards

Pathology

The brain specimen is sliced in the sagittal plane to the right of the falx cerebri, which remains in-situ. The pituitary gland has been completely replaced by a round tumour 4cm in maximum diameter. The tumour cut surface is pale brown and homogenous (except for an area of haemorrhage superiorly, likely caused by surgical trauma). The tumour has resulted in upward displacement of the midbrain. Tumour erosion has destroyed the sphenoid bone; thus, the sella turcica is enlarged (arrow). The optic chiasma is compressed by the tumour. Histologically, this tumour was a chromophobe adenoma arising from the anterior pituitary.

Further Information This specimen is from an old case and the investigations used would now be considered antiquated. Modern investigation would include an initial brain CT followed by an MRI of the brain to further visualise the pituitary lesion prior to any surgical intervention. Pituitary adenomas are the most common pituitary tumour and are most commonly found in adults with peak incidence between 35-60 years. Primary carcinoma of the pituitary is very rare, and the pituitary is an uncommon site for metastases. Clinical manifestations of pituitary adenomas are related to local mass effect and tumour function. Local effects include increasing intracranial pressure (headache, nausea and vomiting), sellar expansion, bony erosion and compression of decussating nerve fibres in the optic chiasma, causing bitemporal hemianopia. Pituitary adenomas can be functioning (i.e. associated with hormone excess) or non-functioning (i.e. without clinical symptoms of hormone excess). About 75% of adenomas are functional: usually secreting prolactin, growth hormone or ACTH. Secretion of TSH, LH and FSH from pituitary adenomas is very rare. Some adenomas can secrete two hormones: growth hormone and prolactin being the most common combination. Non-functional pituitary adenomas come to clinical attention later than those associated with endocrine abnormalities, and they may lead to hypopituitarism due to compression atrophy of the surrounding normal gland.

Metastatic Adenocarcinoma in the Brain

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

Product code: AM02709



Clinical History

A 56-year old male underwent a total gastrectomy and splenectomy for gastric adenocarcinoma. Over a period of two months he developed a progressively unsteady gait, increasing weakness of his left hand and frontal headaches associated with nausea and vomiting. Imaging revealed a lesion in the right frontal lobe. He underwent a craniotomy with resection of the lesion, which was confirmed metastatic gastric adenocarcinoma. He experienced gradual increasing symptoms as well as jaundice, deteriorating consciousness and papilloedema from increased intracranial pressure. Repeat imaging revealed recurrence of the right frontal metastatic lesion as well as liver metastases. The patient died 9 months after his initial gastrectomy surgery.

Pathology This brain specimen is cut in the coronal plane. A circumscribed, variegated, pink-grey tumour is evident in the right frontal lobe. The tumour is involving the grey and white matter. Compression of the right lateral ventricle by the lesion is apparent with shift of the midline structures also seen. Further Information Stomach cancer is one of the most common causes of cancerrelated death worldwide. Risk factors include male gender, diet, smoking and chronic Helicobacter pylori infection. The most common sites for metastases of gastric adenocarcinoma are the liver, peritoneum, lungs and bones. Brain metastases are rare, occurring in <1% of cases. Isolated brain metastases are very uncommon with them being more commonly seen in disseminated disease and associated with a poor prognosis. Palliative treatment may include surgery, radiotherapy, steroid, chemotherapy or a combination thereof

Astrocytoma

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

Product code: AM02716



Clinical History A 73-year-old female was admitted with new left-sided hemiplegia. On further questioning she revealed a 3-month history of headaches, nausea and deteriorating balance. CT brain revealed an inoperable brain tumour. She died 1 week after being admitted. Pathology This brain specimen is a coronal section. In the right temporal lobe, a poorly demarcated tumour is present. There is enlargement of the hemispheres and flattening of the gyral pattern. From the posterior aspect of the specimen subfalcine herniation* is appreciated and the tumour appear less well differentiated with haemorrhagic and necrotic foci. Histology of this tumour showed an astrocytoma, Grade III/IV. *In subfalcine (or cingulate) herniation, the most common type of brain herniation, the innermost part of the frontal lobe is pushed under part of the falx cerebri, between the two hemispheres of the brain. Further Information Gliomas are the second most common cancer of the central nervous system after meningiomas. The term “glioma” refers to tumours that are histologically similar to normal glial cells i.e. astrocytes, oligodendrocytes and ependymal cells. They arise from a progenitor cell that differentiates down one of the cell lines. Astrocytomas develop from the astrocyte lineage of glial cells. Tumours are staged according to histological differentiation and range from diffuse astrocytoma (Grade II/IV) to anaplastic astrocytoma (Grade III/IV) to glioblastoma (Grade IV). Histological features include the prominent eosinophilic cytoplasm in some astrocytic tumour cells (gemistocytes) as well as a fibrillary background. Astrocytomas occur most commonly between the fourth and sixth decades of life. Tumours usually occur in the cerebral hemispheres but may also occur in the cerebellum, brainstem or spinal cord. They most commonly present with seizures, headaches, nausea and focal neurological deficits depending on area involved. Without treatment Grade III median survival is 18 months. Treatment includes surgical resection, radiotherapy, chemotherapy or a combination thereof, depending on the clinical context.

Cranial Arterial Circulation

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

Product code: AM02887



This 3D print presents an expanded version of the same dataset that underlies our circle of Willis 3D print derived from careful segmentation of angiographic data. Like our circle of Willis print, this model demonstrates the internal carotid and vertebral arteries entering the skull, branching into the intracranial arteries that supply the brain. This more expanded 3D print of the internal carotid and vertebral artery anastomoses and branches, inclusive of the circle of Willis, displays the full branching pattern of the cerebral and cerebellar arteries. This includes the pericallosal arteries (from the anterior cerebrals) with its named branches, the superior and inferior divisions of the middle cerebral (including sulcal, temporal, and parietal arteries), and the posterior cerebral artery branches.

Cranial Arterial and Venous Circulation

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

Product code: AM02885



Cranial Arterial and Venous Circulation: This 3D print integrates segmented angiographic data of both the cranial arterial and venous circulation into a single model. Further description of the visible structures can be found under the ‘Circle of Willis’, ‘Cranial Arterial Circulation’ and ‘Cranial Venous Circulation’ prints.

Cranial Venous Circulation

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

Product code: AM02886



This 3D print presents the same dataset that underlies our circle of Willis and cranial arterial circulation 3D prints and is derived from careful segmentation of angiographic data. Here, the dural venous sinus network has been segmented based on structures visible from the circulation of contrast medium. As a result, while most of the sinuses are present, the lack of contrast in the anterior portions of the venous system means that some structures (cavernous sinus, petrosal sinuses) are not included in the model. The extensive network of dural veins and venous lacunae are visible, joined in the midline to the superior sagittal sinus. Deep to this network of sinus veins are the great cerebral vein, the inferior sagittal sinus and the straight sinus to its convergence with the superior sagittal at the confluence of sinuses. Several dural veins drain into the left and right transverse sinuses as they pass anterior towards the petrous portion of the temporal bone. The sigmoid sinuses can be seen in the posterior cranial fossa prior to exiting the skull at the jugular foramen and forming the internal jugular vein (visible on the inferior surface of the skull).

Cerebral Haemorrhage

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

Product code: AM02697



Clinical History

A woman of 56-years was admitted following 2 episodes of severe headache with loss of consciousness. Clinical examination revealed systemic hypertension with cardiac enlargement, and a right hemiparesis. Angiography showed bilateral middle cerebral aneurysms. The patient‘s condition deteriorated, and she died soon after admission.

Pathology

The left hemisphere of the brain has been sliced in the parasagittal plane, and the cut surface displays a large cerebral haemorrhage in the parietal and frontal lobe. The haemorrhage and associated clot are causing extensive distortion of the left external capsule and lateral ventricle. The source of the bleeding was a ruptured aneurysm of the left middle cerebral artery

Intracerebral Haemorrhage

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

Product code: AM02714



Clinical History The patient was an 80-year old man who suddenly lost consciousness. On examination there was a right gaze palsy, a left hemiplegia and right hemiparesis. Pathology The specimens are coronal sections of the brain at the level of the mammillary bodies (specimen in which the cut surface of the brainstem where the cerebral peduncles and sustantia nigra are also visible), and more anteriorly where part of both temporal lobes are included. A massive blood clot has replaced the cerebral tissue in the region of the left basal ganglia and internal capsule. The haemorrhage has originated in this area and has ruptured into the left lateral ventricle, and its temporal horn, destroying the walls of the left lateral ventricle and extending into adjacent brain tissue. The right lateral ventricle is also filled with blood, but its walls are intact. The blood clot forms a space-occupying lesion, which has expanded the left cerebral hemisphere, causing a shift of the mid-line structures to the right. There is subfalcine herniation of the left cingulate gyrus under the falx cerebri (the latter has been removed). Further Information Intra-cerebral haemorrhages of this type are usually a complication of systemic hypertension. The haemorrhage occurs due to rupture of a micro-aneurism of a branch of the striate arteries, which arise from the middle cerebral artery and supply the basal ganglia.

Cerebral Haemorrhage, secondary to Acute Myeloid Leukaemia (AML)

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

Product code: AM02711



Clinical History A 22-year-old male presented with a 2-week history of generalised malaise, weight loss and bruised skin without any trauma. He recently developed 5 days of productive cough and fevers. He was admitted to hospital for further investigations but had a sudden loss of consciousness and died a few minutes after admission to hospital. Pathology The specimen is a horizontal slice of brain displaying the superior cut surface. In the right frontal and parietal regions are two large intraparenchymal haemorrhages each 5 cm in maximum diameter. Several smaller haemorrhages are present in the white matter of both hemispheres. This is an example of multiple intraparenchymal cerebral haemorrhages in a patient with acute myeloid leukaemia (AML).of vascular lumen and involvement of the blood vessel walls. The inflammation extended into the cerebral parenchyma causing haemorrhage and necrosis. Further Information Intraparenchymal brain haemorrhages are a result of small, ruptured vessels within the brain causing haemorrhage. They are usually associated with sudden onset neurological symptoms. The most common causes for spontaneous (no-traumatic) intraparenchymal hemorrhages are hypertension and cerebral amyloid angiopathy with a peak incidence in the 6th decade of life. AML is a cancer of haematopoietic progenitors causing bone marrow failure, due to immature blast cell accumulation in the marrow. The immature blasts affect the production of other bone marrow-derived cells leading to bone marrow failure causing anaemia (decreased erythrocytes), thrombocytopaenia (decreased platelet production and a haemorrhage tendency) and neutropenia (decreased neutrophil production). These in turn lead to a wide spectrum of disease complications, which have to be anticipated and managed. Infection is the leading cause of death in AML as a result of neutropenia. Intracranial haemorrhage is the second most common cause of death in AML, due to low or dysfunctional platelets caused by the thrombocytopaenia. A severe form of haemorrhage is termed disseminated intravascular coagulation (DIC) is common in AML. DIC is characterised by small blood clots developing throughout the bloodstream, blocking capillaries. The increased clotting depletes the platelets and clotting factors needed to control bleeding, causing excessive bleeding. Symptoms depend on the location and severity of the bleed. Interventions include surgical evacuation of the haematoma, reversal of the coagulopathy, treatment of any seizures, and regulation of the intracranial pressure. Interventions are dependent on the severity of the bleed and clinical context. The 30-day mortality for intracerebral haemorrhage ranges from 35-50% with most deaths occurring in the first 48 hours.as obstructive hydrocephalus, secondary to resulting aqueductal obstruction. Diagnosis is dependent on laboratory CSF testing and imaging, in particular using CT scans and MRI. Prolonged intravenous antibiotic therapy is a mainstay of treatment with consideration to achieving effective concentrations in CSF and brain tissue.

Brain (Cerebrum)

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Product code: AM02684



This 3D model provides a unique perspective on the anatomy of the cerebrum relative to the meninges. The cerebrum has been separated from the brainstem and cerebellum, with only parts of the midbrain and cerebral peduncles visible on the inferior surface. Adjacent to the cut section the olfactory tracts and bulbs can be seen extending along the inferior margin of the frontal lobes of the cerebrum. Varying dissection between the left and right cerebral hemispheres allows an appreciation for the organisation of the brain and meninges as it would normally appear within the cranial cavity. In the midline, the dura mater has been preserved from anterior (rostral) to posterior. The central portion of the true (endosteal) dura opened to expose the superior sagittal sinus (between endosteal and meningeal layers of dura mater). Numerous arachnoid granulations (clusters of arachnoid villi) are visible within the opened superior sagittal sinus – as well as across the margins of the preserved dura. On the right cerebral hemisphere, the dura mater has been completely removed to expose the underlying arachnoid mater, which obscures the appearance of the underlying cerebral gyri and sulci as well as the terminal branches of cerebral arteries. In contrast, the arachnoid mater has dissected across most of the hemisphere (excepting a margin for reference) to expose the gyri and sulci covered in pia mater. This allows a clear view of the lateral sulcus and the central sulcus, with the latter defining the boundaries of the frontal and parietal lobes - and separating the primary sensory and motor cortical areas on the gyri on either side of the sulcus.

Brain (Hemisection)

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

Product code: AM02683



This 3D model is a midsagittal hemisection through a whole brain, preserving the right side anatomy and deep brain structures and spaces visible in the midline. In lateral view, the right cerebral and cerebellar hemispheres are covered in the arachnoid mater. In the midline view, the brain regions from the cerebrum to the medulla oblongata are preserved. Centrally, the third ventricle is opened, with an intact septum pellucidum superiorly positioned and obscuring the lateral ventricles within the cerebral hemisphere. On the inferior margin of the third ventricle both the right mamillary body and right optic tract can be observed, whereas posteriorly the cerebral aqueduct can be observed extending across the midbrain between the tectum and tegmentum towards the fourth ventricle (between the cerebellum and pons). The cerebellum is separated from the occipital lobe by a preserved portion of the tentorium cerebelli, and in cross-section the cerebellar cortex helps form the prominent arbor vitae. A series of arterial branches have been false coloured to contrast their course across the preserved brain structures. In the midsagittal view the anterior cerebral artery courses from around the corpus callosum to supply the cingulate gyrus and other midline cortical regions. The base of the middle cerebral artery can be seen passing deep between the temporal and frontal lobes, with the posterior communicating artery connecting it to a small remnant of the posterior cerebral artery. Adjacent to the posterior cerebral is the superior cerebellar artery, extending laterally to pass between the temporal lobe and the cerebellum before passing deep into the transverse fissure.

Cerebral Arterio-Venous Malformation

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Product code: AM02712



Clinical History This patient died at the age of 58 years from post-operative complications following transurethral resection of the prostate. At the ages of 28 and 35, he had suffered two episodes of transient neurological deficit. However, at 50 years, he developed permanent hemiparesis of the left leg chiefly affecting his ankle. Pathology The specimen is a coronal slice of the brain that passes through the parietal lobes. Cortex and white matter on the medial aspect of the right cerebral hemisphere have been replaced by a mass of abnormal tissue 4 cm in greatest diameter. This lesion extends from the superior surface down to the roof of the lateral ventricle. A closer inspection reveals the tissue to be a network of tortuous vascular channels and intervening tissue. Histological examination of this arterio-venous malformation showed glial tissue surrounding dilated vessels. All vessels had a typical endothelial lining, some showed thick muscular walls and others thin walls, thus identifying themselves as arteries and veins, respectively. Further Information The most frequently observed problems, related to cerebral arteriovenous malformations (AVM), are headaches, seizures, cranial nerve deficits and back pain, and nausea may follow the occurrence of coagulated blood escaping into the CSF in the vertebral column. Some patients with AVM have no symptoms at all. Progressive weakness and numbness and vision changes as well as debilitating, excruciating pain may also occur depending on the location of the AVMs. In serious cases, the vessels may rupture and cause intracranial haemorrhage. In patients with AVM haemorrhage, symptoms caused by bleeding include loss of consciousness, sudden and severe headache, nausea, vomiting, incontinence, and blurred vision, amongst others. Local damage on the bleed site are also possible and can cause seizure, one-sided weakness (hemiparesis, as in this patient), a loss of touch sensation on one side of the body, and deficits in language processing (aphasia). Ruptured AVMs are responsible for considerable mortality and morbidity

Metastatic carcinoma in the brain

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

Product code: AM02731



Clinical History (pre access to CT and MRI imaging)

This 51-year old woman had surgery for breast carcinoma 2 years before presentation. Her main complaint was left-sided ataxia for the 2 weeks prior, and this had been preceded by a fainting attack followed by left-sided weakness. Examination revealed a left spastic paresis. There was doubt as to the diagnosis because the rapidity of onset suggested a vascular lesion. She was discharged from hospital but six weeks after her initial presentation she was readmitted with left-sided fitting. Lumber puncture and re-examination were not informative. EEG showed a right anterior temporal abnormality. Angiography confirmed the presence of a large spaceoccupying lesion in the right cerebrum. On the ward, there was a steady deterioration of the patient’s condition, and ultimately death.

Pathology:

The specimen is the cerebrum sliced horizontally. On the superior view, the right hemisphere is clearly enlarged, particularly in the parietal region where the gyrae are widened and 3 cystic tumours are evident. The largest, 5 cm in diameter, is in the right parietal region. A smaller tumour, 2 x 1.5 cm in diameter, is seen close to the posterior margin of the largest tumour. A third one, 1.5 cm in diameter, is present in the left parietal region. The tumours have mainly involved white matter. The wall of each lesion is composed of shaggy friable greyish tissue. At necropsy, there was ulceration of the largest tumour into the right lateral ventricle (seen more clearly when the inferior surface is examined). Sub-falcine herniation was also seen, as is displacement of the basal ganglia and internal capsule. Histological examination revealed metastatic carcinoma in the viable areas. Other metastases were found in the liver and bone. Histology of a liver metastasis was consistent with origin from a primary carcinoma of breast.

Meningioma

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

Product code: AM02699



Clinical History

A 68-year-old female presented with recent onset of seizures and was diagnosed with epilepsy. Collateral history revealed a gradual change in the patient’s personality. She subsequently died several months later from a myocardial infarction.

Pathology

between the two frontal lobes. The tumour is compressing the frontal lobes. It has a pinkish cut surface with some yellow areas indicating necrosis. It was attached to the dura mater anteriorly. This is an example of a meningioma.

Further Information

Meningiomas are often said to be the most common tumours of the central nervous system (CNS); however, in fact they arise in the meninges (dura, arachnoid and pia), which are strictly speaking not part of the CNS per se. They arise from arachnoid cells closely associated with the dura; hence, these tumours can be associated with the dura or dural folds (falx cerebri and tentorium cerebelli). Meningiomas are predominantly slow growing benign tumours. Symptoms are determined by the tumour location and the speed of growth. Symptoms include seizures, change of mental state, vision, hearing- or smell alterations, and symptoms of increased intracranial pressure. Meningiomas are frequently asymptomatic. Treatment includes observation, surgery or radiotherapy, depending on the clinical context and tumour morphology. Meningiomas are rare in children with a median age of 65 years at diagnosis. There is a 3:2 female predominance. Exposure to ionising radiation, including cranial radiotherapy, increases the risk of development meningiomas. The greatest genetic predisposition for development is seen in patients with neurofibromatosis type 2 (NF2). NF2 is an autosomal dominant disease caused by mutations in the NF2 gene on Chromosome 22 leading to multiple tumours associated with the nervous system.

Ruptured Berry Aneurysm

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Product code: AM02715



Clinical History Five days before admission this 38-year old female experienced the sudden onset of pain behind the right eye, associated with a slow development of weakness of the left leg. Examination disclosed lefthemiparesis in a confused hypertensive female. There was also a right homonymous hemianopia and a right VIth cranial nerve weakness. Clonus was present at ankle and knee on the left, and the left plantar reflex was upgoing. Lumbar puncture showed a raised pressure and the fluid was blood-stained. Angiography revealed an intracerebral aneurysm. This was clipped at operation but the day following operation the patient died suddenly. Pathology The specimen shows the basal surface of the brain. There is a saccular aneurism 5 mm in diameter at the junction of the right internal carotid and the posterior communicating artery, which has ruptured. There is subarachnoid blood in the immediate area in the cisterna magna and on the inferior surface of the right frontal lobe. There is a similar unruptured aneurysm on the left side. The right frontal lobe appears softer and more friable anteriorly. Further Information Aneurysms of the posterior communicating artery are the third most common Circle of Willis aneurysms, and can lead to compression and palsy of closely-located cranial nerves, such as the VIth in this case. The proximity of the ophthalmic division of the trigeminal nerve to the ruptured aneurysm may also in this patient explain the sudden onset of pain ‘behind the eye’. The visual field defect is most likely due to compression of the right optic tract. The clinical manifestations of stroke are a consequence of the territory of cerebral cortex whose vascular supply is compromised due to the ruptured aneurysm.

Brain Stem anatomical model

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

Product code: AM02680



This 3D **brain stem anatomical model** preserves the several deep cerebral and diencephalic structures through to the proximal medulla oblongata and compliment the other isolated brainstem (BRW10) in our series. Superiorly, on the right side of the 3D model, the lentiform (lenticular) nucleus is in place and the corona radiata of the internal capsule is seen emerging around it. On the left, the lentiform nucleus is absent, but the caudate nucleus head and body are present medially on both sides, wrapping medial to the preserved internal capsule margins and leading to the amygdaloid bodies on each side. The thalami are present bilaterally, and the third ventricle is opened slightly in the midline inferior to the epithalamus (pineal gland). Anteriorly, the cerebral peduncles are present, with the optic nerves extending from the preserved chiasm and tracts. The interpeduncular region is exposed with both the mammillary bodies and the sectioned infundibulum visible. Caudal to the interpeduncular region is the pons preserving the origins of the middle cerebellar peduncles as well as the origins of cranial nerves V, VII, and VIII. The portion of the medulla oblongata preserved possesses prominent pyramids and olives. Posteriorly, the superior and inferior colliculi sit just superior to the sectioned superior cerebellar peduncles, and the fourth ventricle is opened to expose the rhomboid fossa and features of the floor: the medial eminence, facial colliculus, hypoglossal triangle, the vestibular triangle and the vagal triangle.

Brain Stem

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

Product code: AM02682



This 3D model provides a view of the isolated brainstem anatomy from the midbrain to the medulla oblongata, and compliments the other diencephalon/brainstem 3D model (BR 10) in our series. Rostrally, the 3D model has been sectioned at an angle from the overlying diencephalon while retaining the mamillary bodies of the hypothalamus between the cerebral peduncles (anteriorly) and the pineal gland/epithalamus (posteriorly). Posteriorly, the corpora quadrigemina (the collective superior and inferior colliculi) of the midbrain are prominent adjacent to the superior cerebellar peduncles. The cerebellum itself has been removed, leaving the cross-section of the middle and inferior cerebellar peduncles on each side. Inferior to the sectioned peduncles is the partially opened fourth ventricle and remnants of the posterior inferior cerebellar arteries. On the ventral aspect of the 3D model the pons is preserved with the origin of the trigeminal nerve (CN V) preserved (particularly on the left side). Inferior to the pons on the medulla oblongata, both the pyramids and olives are visible on both sides (particularly clear on the right).

Berry Aneurism of Basilar Artery

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

Product code: AM02696



Clinical History

A 37-year-old patient presented to hospital after falling and striking his head, with subsequent symptoms of headache, vomiting and disorientation. CT scan showed dilatation of the lateral ventricles associated with a large mass projecting into the third ventricle posteriorly. One week later a shunt was performed for hydrocephalus. An angiogram revealed a partially thrombosed aneurysm, measuring 1 x 1 cm, arising from the basilar artery. At 3-months post-op the shunt was revised due to obstruction, with repeat cerebral angiogram revealing interval enlargement of the aneurysm. Attempted ligation of the aneurysm was unsuccessful. The patient remained unconscious despite several attempts to revise the shunt and he died.

Pathology

This brain has been sliced in the mid-sagittal plane. It comprises a whole hemi-section of the brain about 1cm thick. On the medial surface a large darkly-coloured ovoid berry aneurysm measuring 5 x 2 cm in diameter, arising from the basilar artery is clearly visible. It has eroded up into the midbrain, compressing the third ventricle from below, and inferiorly into the substance of the pons. The wall of the aneurysm appears intact although blood clot is seen in the third ventricle and appears to be leaking through the lateral wall of that ventricle. The aneurysm is filled with a laminated thrombus. A small area of mucoid degeneration measuring 0.4 cm in diameter is seen posterior to the aneurysm within the pons. Examination of the lateral aspect of the sagittal section shows dilatation of the lateral ventricle, blood staining of the ventricular wall and patchy haemorrhagic infarction of the caudate nucleus. There was some discolouration of the meninges overlying the tip of the left temporal lobe and the cerebellum (not included in 3D print), consistent with sub-arachnoid haemorrhage.

Further Information

Prevalence of aneurysms is approximately 3.2% in the population, while rupture is much less common, occurring only 7.9 per 100,000 person-years. A minority of intracranial aneurysms arise from the posterior circulation, and are mostly situated at junctional points about the basilar, vertebral and cerebellar arteries. Symptoms are either secondary to subarachnoid haemorrhage or a mass effect with associated compression of the adjacent brain parenchyma and cranial nerves. Rupture causes complications due to bleeding and raised intracranial pressure. Hydrocephalus, re-bleed and vasospasm may also occur. Management is via surgical means; in recent years, novel therapies include endovascular intervention with coils and subsequent monitoring.

Ventriculitis, Secondary to Septicaemia

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

Product code: AM02710



Clinical History A 50-year-old alcoholic was admitted with a 2-week history of weakness and shortness of breath. At the onset of the illness he reported a productive cough, chest pain and blood-stained sputum. Examination revealed a febrile, cyanosed, drowsy man with grunting respiration. A friction rub was present over the right lower lobe. The remainder of the examination was unremarkable. The patient steadily deteriorated and on the morning of his death, a lumbar puncture was performed. Green opalescent fluid was obtained. A blood culture grew Streptococcus pneumoniae. Pathology This specimen is an example of ventriculitis, with pneumococcal meningitis and right basal pneumonia also being found at autopsy. The horizontal slice through both cerebral hemispheres displays both of the lateral ventricles. The ventricles show a thickened, rough ependymal lining with cellular debris accumulation around the choroid plexus and also in the anterior horn. The lower surface shows similar changes and also displays the normal arrangement of the caudate nucleus, lentiform nucleus and internal capsule. Histology demonstrated extensive infiltration of neutrophils in the sub-arachnoid space as well as multifocal severe (sub)endothelial infiltration with obstruction of vascular lumen and involvement of the blood vessel walls. The inflammation extended into the cerebral parenchyma causing haemorrhage and necrosis. Further Information Ventriculitis is an uncommon complication of intracranial infection. In adults, it more commonly occurs as a secondary complication of surgical intervention / instrumentation or trauma, rather than from primary community-acquired meningitis. In these cases, causative organisms are similar to other nosocomial (hospital-acquired) infections, in particular staphylococci or resistant Gram-negative bacilli. Neonates aged less than 6 months have a higher incidence of ventricular infection. Presentation may be more subtle than in bacterial meningitis or may be as obstructive hydrocephalus, secondary to resulting aqueductal obstruction. Diagnosis is dependent on laboratory CSF testing and imaging, in particular using CT scans and MRI. Prolonged intravenous antibiotic therapy is a mainstay of treatment with consideration to achieving effective concentrations in CSF and brain tissue.

Left cerebral infarct

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

Product code: AM02707



Clinical History The patient was a 51-year old woman who had a cerebrovascular accident resulting in a left hemiplegia 2 years prior to death. At necropsy, she had severe generalized atherosclerosis and an old left ventricular myocardial infarct with an overlying mural thrombus. Pathology A coronal section of the cerebral hemispheres shows irregular cystic cavities in the territory of distribution of the right middle cerebral artery. The cavities of the infarct have irregular, yellow walls and show partial collapse. There is compensatory dilatation of the left lateral ventricle. On the posterior aspect, the arteries below the mammillary bodies were moderately atheromatous, although this is difficult to visualise macroscopically. Further Information Because of the underlying history of myocardial disease with the presence of the mural thrombus, it is assumed that her cerebral infarct was probably caused by a thromboembolus.

Intracranial space-occupying lesion

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

Product code: AM02713



Clinical History A 56-year-old woman with 6 months of intermittent headache and vomiting was admitted to hospital comatose after a grand mal seizure, and failed to regain consciousness. Pathology The specimen is a coronal section of a brain. It is evident that the brain has been compressed laterally and downwards by a right-sided expanding intracranial mass, probably a meningioma. The original mass is not present. The anterior face shows shift of midline structures with subfalcine herniation* of the cingulate gyrus. The posterior face (see photo) shows haemorrhage of varying ages within the temporal lobe and the pons, typical of supratentorial mass lesions. There is also ventricular asymmetry. *In subfalcine (or cingulate) herniation, the most common type of brain herniation, the innermost part of the frontal lobe is pushed under part of the falx cerebri, between the two hemispheres of the brain. Further Information Symptoms of a space occupying meningioma in the cranial cavity can be caused by the tumour mass pressing on the brain, which can lead to atrophy and displacement of brain parenchyma, leading to symptoms arising from interruptions to cranial nerve functions, blood flow and normal cerebral functions. General symptoms may include: Muscle seizures: e.g. Myoclonic (single or multiple muscle twitches, jerks, and/or spasms) or Tonic-clonic (grand mal: loss of consciousness and body tone, followed by twitching and relaxing muscle contractions, loss of control of body functions, short period of no breathing and the person may turn a shade of blue, a person may be sleepy and experience a headache, confusion, weakness, numbness, and sore muscles) Sensory changes – alterations in vision, smell, and/or hearing without losing consciousness. Symptoms and signs may vary with the location of the tumor.