CENTRAL NERVOUS SYSTEM

MUSEUM CATALOGUE

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Department of Pathology, University of Adelaide, 2004

INTRODUCTION

At first glance, trying to understand the pathology of the nervous system may seem a bit daunting. After all, it's hard enough to understand the normal structure and function of the CNS. It's true that neuropathology can be an incredibly complex topic, but at an undergraduate level you don't have to get bogged down in detail to pass or even to do well. What you need is:

- an understanding of some basic pathological processes
- a little neuroanatomy
- a realisation of some of the idiosyncratic responses of the CNS

HOW TO USE THIS CATALOGUE

This catalogue is to be used as a tool to consolidate and expand on your current knowledge base, as well as provide an opportunity for revision.

It is divided into:

- Introduction and approach to specimens (pages 2-4).
- Index (pages 5-11). Examples of specific diagnoses can be found via the index.
- Core and classic disease processes (pages 12-29). This gives examples and discussion of some core and/or classic diseases of the central nervous system. These are the specimens that students should focus on being able to identify initially. However, it depends to some extent on what you have covered in lectures and practical classes or resource sessions as to what you should know. Some of the specimens and discussion are directed more towards clinical medical students.
- Main catalogue (pages 30-99). This section covers the specimens in numerical order. Questions and/or comments accompany some of the specimens to help you expand your knowledge. In order to fit more specimens in the museum, not all of the pots are in numerical order on the shelves, and large specimens are often found on the bottom shelves.

You might find it easy to work (quietly) with a few friends.

Just as in clinical practice, you will not become proficient in diagnosing a condition if you have only seen one case. Exposure to a variety of cases (specific diagnoses can be found via the index) to experience the variability in morphology will help your learning greatly. In general the red and blue dots on the pots indicate basic and straightforward pathology, whereas yellow dots tend to indicate less readily diagnosable conditions. This is not a hard and fast rule, and you will find yellow dot specimens turning up in resource sessions/practical classes and even examinations, if they represent classic pathology.

In general when assessing a pot

- read the clinical information given
- look at the entire specimen, not just the front
- identify and orientate the organ or tissue
- from your knowledge of pathology look for relevant features to help you make the diagnosis. Of course to appreciate the abnormal you first need to have an appreciation of normal anatomy to be able to recognize and orientate the organ/tissue and identify the abnormality
- make a diagnosis or differential diagnosis using any clinical information given to you – it is often relevant – sometimes the diagnosis is only made with knowledge of the clinical features. Even when you know the diagnosis, attempt to identify relevant features in the specimen and understand why this is the diagnosis.

- attempt to correlate the pathological features with the clinical features (clinico-pathological correlation) i.e. explain how the pathological features have caused the patients symptoms and signs (when relevant)
- try to answer any questions presented yourself before reading the answers.

You may prefer to look at the specimen 'blind', without reading the clinical information given first.

In all cases a final diagnosis is given, but it is important to remember that sometimes the final diagnosis was made using clinical information and histological examination. In some cases the macroscopic appearance is classic and even without the clinical information and histopathological findings you should be able to make the diagnosis from the appearance, in others, it might only be possible for you to give a list of differential diagnoses or a more general diagnosis.

Whenever there has been any clinical history available it has been given. As some of these specimens are very old (some up to 80 years) some of the investigations may seem a bit strange and treatments out of date. Pneumoencephalography was an investigation where air was inserted into the subarachnoid space to outline the brain on x-ray. Ventriculography was a procedure where a radiopaque dye was introduced into the CSF. These investigations seem pretty crude and almost barbaric - but they serve to remind how far medical technology has progressed. By the time you retire from clinical practice, the investigations and treatments in use at the current time may appear just as crude.

BASIC APPROACH TO INTERPRETATION AND DESCRIPTION OF CENTRAL NERVOUS SYSTEM SPECIMENS

Orientation and identification of the specimen

Starting at the most basic level: always look at the front of the specimen first (the one with the number and the coloured dot on the top). Having said this, it is a well-known fact that you should always look at the back of a pot as well - especially if you are in a viva exam and want a few more minutes to think.
Now, identifying the specimens in this section is generally quite easy: it's either a brain or a spinal cord, possibly a piece of dura, or a bit of bone, an eye, or a dissected out circle of Willis. Working out what the specimen is isn't going to be the main problem you have to face when someone gives you a CNS pot. Working out which side is which may be harder. It is easy with horizontal/transversely cut specimens but not always easy (or possible for anyone less than a neuropathologist) in coronal sections. Remember that the poles of the temporal lobes, the cerebral peduncles and the optic nerves extending out from the optic chiasm, present in many of the coronal sections, point towards the front.
And remember if there is more than one tissue specimen in a pot, it is there for a reason and that reason is to help you with the answer. No one puts extra specimens in just because they were around at the time. Also note that most of the brains in the pots are still covered by arachnoid with the subarachnoid vessels beneath. The dura is stuck to the skull rather than the brain, so is generally not removed when the brain is removed at autopsy.

Nature of the lesion

Focal

Focal lesions are probably the easiest. They are single abnormalities to which any one can point to with confidence and say "This is the abnormality". That's the easy bit: the next thing is to describe it in the appropriate degree of detail. To do this you need to know what the important features of a focal lesion are and concentrate on them:

- Colour: What colour is it? Is it all one colour or is it variegated (many colours)? Does it look homogenous (i.e. the same all the way through)? Greyish discolouration in tumours suggests necrosis.
- Size: You can give a measurement, but don't get too obsessive.
- Shape

- Consistency: This can be quite difficult to assess in a specimen in a pot that you can't touch, but even just by looking you can get some idea. Does it look solid? Does it look friable (as if it's falling to pieces), are there holes in it or bits missing? - this suggests that there may have been necrosis.
- Margins
    - the best way to think of margins is are they well defined or demarcated – i.e. is there a clear line that you can trace between normal tissue and the lesion - or are they diffuse or irregular – i.e. the line between normal and abnormal is harder, perhaps impossible to trace.
    - malignant tumours typically have infiltrative, irregular or invasive margins. Benign tumours have well-defined and sometimes encapsulated margins.
      There are of course exceptions to this rule: sometimes malignant tumours, especially secondary or metastatic tumours can have deceptively well-defined margins. Often these are particularly aggressive tumours that grow so quickly they just push tissue out of the way, rather than bother infiltrating it in the normal manner. On the other hand, non-neoplastic lesions such as infarcts may have poorly defined margins.
      It is also important to differentiate between a well-defined/well-demarcated lesion and an encapsulated lesion. To describe something as encapsulated means that there is a definite fibrous band surrounding the lesion and means that the lesion is benign.

Multifocal

This means that there is more than one distinct lesion in the specimen. All the comments regarding the description of focal lesions apply here as well. In addition, it's important to note any variation between lesions.

Diffuse

These are probably the hardest specimens for students to come to terms with. There is nothing worse than the sinking feeling that comes from looking at a pot in an exam, and not being able to identify areas of obvious abnormality. As you get a better understanding of neuropathology, you'll realise that there are only a few things that it's likely to be. The thing to do if you cannot see any gross abnormalities in the brain or spinal cord parenchyma is to turn your attention to areas where more subtle abnormalities may exist:

- The meninges: are they thickened or opaque? - this may indicate meningitis. Check the base of the brain as well as the hemispheres as some forms of meningitis e.g. TB affect the base of the brain preferentially.
- The gyral pattern
    - is there atrophy? Does this affect the entire brain or just parts?
    - are the sulci less obvious, suggesting swelling? Check the rest of the brain for signs of herniation, indicating raised intracranial pressure
    - is there an abnormal gyral pattern? This is sometimes seen in congenital disorders
- The cortex
    - is there any thinning of the cortical ribbon, suggesting past hypoxic/ischaemic injury?
    - is there blurring of the normal grey-white matter demarcation (from swelling) or discolouration (from petechial haemorrhage in haemorrhagic infarction) of the cortical ribbon suggesting early infarction?
- Absence
    - this is probably the most difficult type of specimen when the diagnosis is based on the absence of normal structures - it presumes a good knowledge of neuroanatomy. Examples include loss of pigmentation of the substantia nigra in Parkinson's disease; atrophy of the caudate nucleus in Huntington's disease etc

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CORE AND CLASSIC DISEASE PROCESSES

CEREBRAL INFARCTION

CASE 50080/83

Clinical information
This 59 year old woman presented with a sudden onset of aphasia and right hemiparesis without any prior symptoms. Although initially alert her conscious state deteriorated and she died after 24 hours.
At autopsy there was evidence of hypertensive cardiomegaly with an old myocardial infarct.

Specimen description
The specimen displays two coronal sections through the cerebral hemispheres viewed from the front. The left hemisphere is swollen with loss of grey-white demarcation and cortical petechial haemorrhages in the left middle cerebral artery territory. There is compression of the left lateral ventricle and displacement of the midline structures to the right.

Diagnosis
Haemorrhagic left middle cerebral artery territory infarct

CASE 22153

Clinical information
The patient was a man aged 83 who had a stroke 2 years ago, resulting in transient dysarthria but no permanent muscular weakness. On the morning of his latest admission he developed dysarthria and left hemiplegia. Two days later he had a severe unexpected haematemesis for which massive transfusion was required, but he died on the 4th hospital day. At post-mortem, the haematemesis was found to have resulted from a bleeding duodenal ulcer.

Specimen description
The specimen consists of a coronal slice through the cerebral hemispheres viewed from behind. Portions of both common carotid arteries with their bifurcations are also present. Within the left cerebral hemisphere is an area of loss of tissue measuring 40 x 30mm, which involves both grey and white matter and which is partially covered by a grey membrane. This is in the territory of the left middle cerebral artery. On the right side, also in the middle cerebral artery territory is an ill-defined area of swelling with reduced demarcation between cortex and white matter.
One internal carotid artery (probably left, on the right of the jar) is totally occluded by old atherosclerosis and organised thrombus and the artery above the block is reduced to a shrunken fibrous cord. The other (probably right on the left of the pot) internal carotid artery is grossly stenosed by atherosclerosis just above its origin and there is recent antemortem thrombus above.

Diagnosis
Recent (right side) and old (left side) middle cerebral artery territory infarctions

Correlate the pathological findings with the clinical history in case 22153.
The old left-sided infarct would have occurred 2 years prior to death, being responsible for the dysarthria at this time. It may well have been caused by occlusion of the left internal carotid artery at its origin or from embolism of thrombus or plaque from it occluding the left middle cerebral artery. The very recent right-sided infarct occurred 4 days before death, being responsible for the recent dysarthria and left hemiplegia. There appears to be recent thrombus in the right internal carotid artery that was probably responsible.

Comment

Recent cerebral infarcts may be either haemorrhagic - as in case 50080/83 - or pale - as in case 22153. In both cases they result from occlusion of a major cerebral artery, such as the left middle cerebral artery in these cases. Typically wedge shaped in appearance, with the base on the cortical surface and the apex lying centrally, they lie entirely within the vascular territory of the affected vessel.

The different appearances of cerebral infarcts - pale or haemorrhagic - are thought to be the result of differences in the nature and duration of the occlusion. In pale infarcts the arterial occlusion is typically the result of thrombosis overlying an atherosclerotic plaque. A haemorrhagic infarct is more likely to be the result of occlusion of an artery by an embolus, which occludes the lumen for a sufficient length of time to cause ischaemic necrosis, before being lysed, allowing blood to re-enter the infarcted area. Blood then leaks out of damaged capillaries, causing the haemorrhagic appearance. The bleeding is typically petechial but may rarely be more massive.

Haemorrhagic infarction may also be caused by

- Compression of a cerebral artery e.g. the posterior cerebral in transtentorial herniation (e.g. case 24770a)
- Spasm of a cerebral artery as following subarachnoid haemorrhage (e.g. case 21099)
- Venous occlusion due to thrombosis (e.g. case 50204/85). The distribution of venous infarctions will not of course be in the usual arterial vascular territories.

As with any infarct, macroscopic changes are not apparent for 8-12 hours. Oedema (due to failure of sodium pumps and disturbance of the blood-brain barrier) develops in and around a cerebral infarct over hours – days, leading to the blurring of the normally distinct demarcation between white and grey matter. Raised intracranial pressure and herniation may result. Early areas of infarction are softer than the surrounding tissue, and palpation can help distinguish them.

Infarction induces an acute inflammatory response, however, healing of a cerebral infarct is not by fibrosis. The dead tissue is removed over weeks – months leaving a cavity surrounded by gliosis.

The relationship of an infarct to the site of arterial occlusion can be variable. It depends on

- the anatomy and patency of the circle of Willis and other collaterals
- the presence of atherosclerotic narrowing, thrombosis and embolism
- the systemic blood pressure

E.g. occlusion of an internal carotid artery may result in:

- no disturbance
- a TIA
- infarction of part of its territory (e.g. MCA territory)
- infarction of all of its territory

Lacunes (e.g. case 25174) are small infarcts (1-15mm), typically occurring in the basal ganglia or brainstem. They don't necessarily cause symptoms. They may be multiple. They tend to arise in hypertensive patients due to thickening with severe narrowing of small arteries and arterioles (hyaline arteriolosclerosis). Larger lacunes may be due to atherosclerotic narrowing +/- thrombosis of small intracerebral arteries, more vulnerable to atherosclerosis in hypertensive and diabetic patients.

It is important that you are clear as to the difference between a haemorrhagic infarct and a haemorrhage. A haemorrhagic infarct is an area of ischaemic necrosis (infarct) that has a blood stained appearance from variable amounts of haemorrhage occurring secondarily due to reperfusion, but is the result of occlusion of an artery. A haemorrhage is due to the rupture of a vessel causing massive bleeding into the tissues and does not cause infarction.

REDUCED CEREBRAL BLOOD FLOW

CASE 50116/88 A and B

Clinical information
The patient was a 59 year old man who suffered a cardiac arrest while under anaesthesia during an operation on his lumbar spine. He was resuscitated, but remained in a vegetative state until his death three weeks later.

Specimen description
There are 2 specimens. Both consist of a coronal section of the cerebral hemispheres. A is viewed from the front, B from the back. The two specimens are similar in appearance and show widespread thinning of the cortex throughout the various vascular territories. The cortex also shows patchy discolouration, with the deeper layers appearing darker in colour.

Diagnosis
Diffuse hypoxic damage/ widespread selective neuronal necrosis

CASE 21260

Clinical information
A 41 year old alcoholic presented to the Port Lincoln hospital in delirium tremens and was treated with intravenous vitamin B. Shortly thereafter he had a vascular collapse with acute renal failure complicated by Pseudomonas septicaemia. He was transferred to the QEH where a renal biopsy showed acute tubular necrosis. Peritoneal dialysis was commenced. He then suffered a cardiac arrest and had a minor epileptic fit. He died 9 months later.

Specimen description
The specimen consists of two coronal slices through the cerebral hemispheres and a transverse slice through the brain stem and cerebellum. There are scattered, ill-defined lesions characterised by necrosis of white matter with some thinning of the overlying cortex. These necrotic areas are within the parasagittal regions of the hemispheres, the basal nuclei, the infero-lateral aspects of the temporal lobes and in the cerebellum laterally.

Diagnosis
Watershed cerebral infarction

Comment
Global impairment of cerebral perfusion can have a variety of affects depending on its completeness and duration. Cardiac arrest with no or minimal cerebral blood flow, depending on duration, may lead to widespread selective neuronal necrosis with selected neurones in the cerebral cortex, hippocampus, basal nuclei and the Purkinje cells of the cerebellum being most affected. If the patient survives, loss of neurones in the cortices leads to thinning and atrophy (case 50116/88) and permanent neurological deficit of variable severity. Glial cells and blood vessels are less susceptible to ischaemia. In the context of a complete circulatory arrest at normal body temperature, complete clinical recovery is unlikely if the period of arrest is more than 10 minutes. Adequate cerebral perfusion does not start immediately the heart starts pumping again, and any period of pre or post- arrest hypoperfusion is also important in determining outcome.

Episodes of prolonged hypotension but with some cerebral blood flow may lead to watershed infarction (case 21260) with death of all tissue elements. The areas at the boundary zones of the major cerebral arteries are the areas that are perfused most poorly leading to infarction.

INTRACEREBRAL HAEMORRHAGE

CASE 24892

Clinical information

This 59 year old woman was admitted to hospital complaining of the sudden onset of slurred speech and pain over the right eye. On examination in hospital she had left upper motor neurone facial weakness and flaccid paralysis of both her left arm and left leg. Sensation was absent from the left leg and to a lesser extent from the left arm. Her eyes were deviated to the right. She died from pneumonia on the 7th hospital day.

Specimen description

The specimen is a coronal slice through the cerebral hemispheres viewed from behind. Within the right thalamus and basal ganglia is a dark brown, round lesion 30mm in diameter with well-demarcated borders and a solid appearance. The cavities of the third and right lateral ventricles are compressed and the lesion has ruptured into the lateral ventricle through the caudate nucleus. The septum pellucidum is deviated slightly to the left.

Diagnosis

Right capsular intracerebral haemorrhage

Comment

This demonstrates the classic appearance of an intracerebral haemorrhage. The site - within the basal ganglia/internal capsular region (deep cerebral or capsular haemorrhage) - is also typical. Such haemorrhages are the consequence of longstanding hypertension. The striate or deep penetrating arteries that supply these regions are very small arteries coming directly off the middle cerebral arteries and in hypertensive patients they are exposed to unusually high pressures for vessels of their calibre. This results in damage (hyaline arteriolosclerosis/lipohyalinosis) and sometimes the formation of microaneurysms of Charcot-Bouchard, but their role in the haemorrhage is unclear. The damaged vessels are prone to rupture, causing an intracerebral haemorrhage.
Other common sites for such hypertension related haemorrhages are the white matter of the hemispheres, pons and cerebellum.

Other causes of intracerebral haemorrhage include intracerebral rupture of a berry/saccular aneurysm (e.g. case 25486), ruptured arteriovenous malformation (e.g. case 25645), rupture of a vessel in congophilic (amyloid) angiopathy, trauma, certain drugs (including cocaine and heroin) and spontaneous haemorrhage in persons with a bleeding diathesis e.g thrombocytopenia in leukaemia (e.g. case 21267) or excessive anticoagulation.

The haemorrhage and the related oedema have a space occupying effect and patients frequently die of the effects of raised intracranial pressure and herniation. In patients who survive, the haematoma is resorbed over weeks-months leaving a cavity with surrounding haemosiderin staining and gliosis.

It is important that you are clear as to the difference between a haemorrhagic infarct and a haemorrhage. A haemorrhagic infarct is an area of ischaemic necrosis (infarct) that has a blood stained appearance due to variable haemorrhage occurring secondarily due to reperfusion, but is the result of occlusion of an artery. A haemorrhage is due to the rupture of a vessel causing massive bleeding into the tissues and does not cause infarction. It is also important that you realise that this type of haemorrhage is not normally caused by rupture of a berry or saccular aneurysm but is normally caused by rupture of small arteries damaged by hypertension.

BERRY/SACCULAR ANEURYSM AND SUBARACHNOID HAEMORRHAGE

CASE 25486

Clinical information

The patient was a woman aged 61 who had been hypertensive for 20 years and had suffered from angina pectoris for 14 years, which had been getting worse during the preceding 6 months. She was brought into hospital collapsed and unconscious. Her BP was 215/115 and her pupils were fixed and dilated. There was paralysis of the left side of the tongue and of the left side of the body. She died two days later.

Specimen description

The specimen consists of the lower half of the brain divided in the transverse plane. There is a large thin-walled aneurysm about 15mm in diameter arising where the left middle cerebral artery divides into its branches in the Sylvian fissure, about 20mm from its origin. There is recent subarachnoid haemorrhage over the orbital surfaces of both frontal lobes, over the lateral aspect of the left frontal and temporal lobes and also around the brain stem and cerebellum. In addition, there is a large elongated haematoma measuring 70 x 30mm within the white matter of the left frontal lobe that communicates with the subarachnoid space near the aneurysm. The haematoma has caused midline shift to the right. The right hemisphere otherwise appears within normal limits. A small unruptured aneurysm about 4mm in diameter is present at the division of the right middle cerebral artery in a position similar to that on the left. The arteries of the circle of Willis show only mild atherosclerosis. The cerebellar tonsils, particularly the left, appear prominent.

Diagnosis

Ruptured berry/saccular aneurysm of the left middle cerebral artery causing subarachnoid and intracerebral haemorrhage

Comment

Saccular or berry aneurysms arise in arteries of the Circle of Willis or its major branches at the base of the brain. Although referred to as congenital, they are not identifiable at birth. They generally arise at branch points in the vessels that represent areas of weakness, with gaps in the tunica media, under the influence of haemodynamic stress. The role of hypertension in their development is unclear. Genetic factors appear to be important in causing early onset saccular aneurysms/subarachnoid haemorrhage and patients with certain inherited diseases such as autosomal dominant polycystic kidney disease, Marfan syndrome and Ehlers-Danlos syndrome have an increased incidence. Smoking and alcohol consumption are independent risk factors for subarachnoid haemorrhage.

Rupture of a saccular or berry aneurysm typically leads to subarachnoid haemorrhage because of their location, but depending on the specific site of rupture in the aneurysm there may be a greater or lesser degree of intracerebral haemorrhage. If a saccular aneurysm is closely adherent to the overlying brain when it ruptures, blood may jet into the brain, destroying tissue and resulting in an intracerebral haemorrhage. If the point of rupture of the aneurysm is on the side away from the brain, bleeding will be limited to the subarachnoid space.

In this case, the side of the patient's paralysis does not seem to fit with the side of the lesion. It is possible that the contralateral cerebral peduncle was compressed as a result of transtentorial herniation. The parahippocampal gyri are not readily seen to assess its presence, though it is likely as there is probable tonsillar herniation. Also the brainstem and cerebellum have not been examined.

Vasospasm leading to cerebral ischaemia and infarction (e.g. case 21099) is an important cause of morbidity and mortality among patients with subarachnoid haemorrhage. It typically happens between 4-14 days after the initial haemorrhage.

SUBDURAL HAEMATOMA

CASE 16136

Clinical information

The patient was a man aged 68 whose mental condition had been deteriorating over the preceding 6 months until he was no longer able to look after himself. He was found in a confused state and admitted to a regional hospital where he became stuporose.
He was transferred to the RAH and on admission there he was found to respond to painful stimuli and to verbal commands but was mute. His left pupil was larger than the right but both reacted to light. There was right facial paresis and weakness of the right arm.

Specimen description

The specimen consists of the upper half of the cerebral hemispheres together with the overlying dura mater. The dura mater has been reflected back from the left hemisphere to expose an extensive flattened dark brown-black, solid but friable mass adherent to its undersurface. It is about 0.5cm deep and in areas is covered by a delicate wispy membrane. In addition there is some flattening of the surface of the left hemisphere especially in the parietal region. The reverse of the specimen shows some compression of the left lateral ventricle with slight midline shift to the right.

Diagnosis

Chronic subdural haematoma

Comment

The subdural haematoma was diagnosed during life. The patient underwent an operation and the haematoma was removed. He improved for a few hours but then relapsed into mutism and died suddenly on the 7th hospital day. A massive pulmonary embolism was found at post-mortem, originating from thrombi in the femoral and iliac veins.

Subdural haematomas are typically the result of trauma, when bridging veins extending from the surface of the cerebral hemispheres to the superior sagittal sinus are torn. This is often the result of excessive movement of the brain within the cranial cavity. The elderly are particularly prone to this condition because of the possible presence of cerebral atrophy. The bleeding may be very slow, with a gradual accumulation of blood in the subdural space. The patient may thus have a very slow deterioration in their mental function, and this may mimic the onset of dementia. The compression of the underlying brain may give rise to focal symptoms - such as right facial and arm weakness as in this case – and the blood may act as a space-occupying lesion. The patient's final death from pulmonary embolism is not surprising in view of a prolonged hospital stay.

Acute subdural haematomas are often accompanied by oedema of the underlying brain, contributing to the space occupying effect.

EXTRADURAL HAEMATOMA

CASE 20523

Clinical information
The patient was a man aged 21. He was alleged to have fallen from a horse and suffered a stellate fracture of the right occipital bone.

Specimen description
The specimen consists of the lower half of the brain divided in the transverse plane, with a portion of dura overlying the cerebellum. There is a collection of blood on the external surface of the dura and it can be seen to compress the postero-inferior aspect of the right occipital lobe and the adjacent right cerebellar hemisphere. There is bilateral patchy subarachnoid haemorrhage on the undersurfaces of both frontal lobes and on the cut surface of the specimen, a small area of petechial haemorrhage is seen in the left frontal lobe anteriorly. The right cerebellar tonsil appears prominent.

Diagnosis
Extradural haematoma in posterior fossa with contre-coup contusion and tonsillar herniation

How are the pathological findings related to the clinical history?
Extradural haemorrhage is typically the result of tearing of a meningeal artery, often as a complication of skull fracture. The fact that the fracture was in the occipital bone indicates that the man hit the back of his head, thus the frontal lobe contusion represents a contre-coup lesion. The extradural haemorrhage would act as a space-occupying lesion in the posterior fossa leading to raised pressure and tonsillar herniation and death from respiratory arrest.

Comment
Extradural haemorrhage most often results from tearing of the middle meningeal artery and thus usually occurs in the temporoparietal region.

TRAUMATIC BRAIN INJURY

CASE 20961

Clinical information
The patient was a male pedestrian aged 16 years struck by a car. He lived for 10 days following the accident but remained deeply unconscious. The right pupil reacted to light but the left pupil was fixed and dilated.

Specimen description
The specimen consists of two coronal slices through the cerebral hemispheres and a transverse slice through the upper pons. Both hemispheres are swollen and the cavities of the lateral ventricles are markedly compressed. There are several small patches of sub-arachnoid haemorrhage. Scattered petechial haemorrhages are present within the corpus callosum, which appears to have ruptured focally. Minor contusions are present in the right cingulate gyrus and in the superior frontal gyri bilaterally. In addition there is an area of haemorrhage within the right dorsolateral quadrant of the pons and some grooving with tiny haemorrhages in both parahippocampal gyri.

Diagnosis
Traumatic brain injury with cerebral oedema, transtentorial herniation and vascular markers of diffuse axonal injury

Comment
These changes in the corpus callosum and haemorrhage in the dorsolateral quadrant of the pons are commonly seen in association with severe diffuse axonal injury. Acceleration/deceleration injuries cause shear and tensile stresses that lead to widespread disruption of axons. Impact of the head against a hard object is not necessary for this damage to occur thus there is frequently no associated skull fracture. There is no lucid interval. Diffuse axonal injury is the commonest cause of severe permanent disability after head injury.

Other CNS sequelae of head injury include

- Diffuse cerebral oedema
- Diffuse vascular injury
- Cerebral contusions and lacerations (e.g. case 16183)
- Intracerebral and intraventricular haemorrhage
- Subarachnoid haemorrhage (usually not clinically significant)
- Extradural haemorrhage (e.g. case 20523)
- Subdural haemorrhage
- Infections following skull fracture and breaching of the meninges
- Ischaemic damage related to hypotension
- Damage related to raised intracranial pressure and herniation
- Fat embolism (e.g. case 16846) related to peripheral fractures

BACTERIAL MENINGITIS

CASE 23243

Clinical information

The patient was a 50-year old man who had been treated at home for pneumonia for 4 days before being found unconscious and incontinent of urine.
On examination in hospital he was drowsy but well orientated. His eyes were deviated to the right, he had a left accessory nerve palsy and was incapable of moving his left arm or leg.
Over the next four days his left-sided paralysis remained unchanged.
Five days after admission he became semicomatose and barely responsive to painful stimuli. He died three hours later.

Specimen description

The specimen consists of the upper part of the brain sectioned transversely through the cerebral hemispheres.
There is a thick purulent exudate in the subarachnoid space over the convexities of both hemispheres. It is thinner over the occipital pole.
The cut surface shows mild swelling of both hemispheres, particularly on the right, with slight compression of the lateral ventricles. In addition there are some small petechial haemorrhages in the white matter of both hemispheres, with one larger haemorrhage 4mm in diameter present in the white matter of the right inferior frontal gyrus.

Diagnosis

Suppurative meningitis

Comment

At post-mortem Streptococcus pneumoniae was grown from the CSF: apparently no lumbar puncture had been performed during his hospital stay. Other autopsy findings included extensive bilateral bronchopneumonia and cirrhosis of the liver.

This specimen has the classical appearance of suppurative meningitis with clouding of the meninges due to the presence of purulent exudate within the subarachnoid space. The history, however, is a bit unusual. Bacterial meningitis often presents with a short history of general malaise, fever, headache and photophobia (abnormal visual intolerance of bright light). A vasculitic rash is a characteristic feature of meningococcal septicaemia occurring in association with meningitis and is a bad prognostic sign. The common causative organisms are Streptococcus pneumoniae, Neisseria meningitidis (aka meningococcus) and Haemophilus influenzae. These organisms tend to affect different age groups. If untreated, death or severe disability is likely.

Tuberculous meningitis (e.g. case 2586) macroscopically may look similar, although the exudate (microscopically necrotising granulomatous, not purulent) tends to be worst around the base of the brain and the clinical history won't be so acute. There is no visible exudate in viral meningitis, though excessive lymphocytes will be seen in the CSF.

BRAIN ABSCESS

CASE 22081

Clinical information

The patient was a 19 year old female with Trisomy 21 (Down's syndrome) who lived in residential care. She was observed to suddenly lose consciousness and was admitted to hospital.
On admission she remained unconscious and was noted to have irregular breathing (Cheyne-Stokes respirations). The right pupil was dilated. A lumbar puncture was performed and the CSF was turbid with increased numbers of leucocytes. She died the following day.

Specimen description

The specimen consists of two coronal slices through the cerebral hemispheres viewed from the front. Within the left temporal lobe is an irregular 30 x 20mm cavitating lesion lined by pale necrotic slough. The surrounding tissue is oedematous. The abscess has ruptured into the cavity of the left lateral ventricle and both ventricles contain pus. The left parahippocampal gyrus is notched.

Diagnosis

Temporal lobe abscess with rupture into the ventricular system and transtentoiral herniation

Comment

Brain abscesses are most frequently caused by spread of infection from a chronic suppurative otitis media, thus the temporal lobe is a common site, or a chronic mastoiditis. Other causes include haematogenous spread of infection and penetrating head injuries. In the case of otitis media and mastoiditis it is thought that the adjacent bone becomes infected (chronic osteitis) and is eroded. Infection then extends to the extra- and sub-dural space and thus the adjacent brain. It is proposed that the reason that patients do not develop meningitis is that local obliterations of these spaces occurs from chronic inflammation and fibrosis.

By the time most abscesses are diagnosed they are subacute or chronic. The abscess becomes surrounded by a capsule containing collagen and inflammatory cells with surrounding reactive astrocytes and oedema. Abscess formation is the only time when there will be a degree of fibrosis or scarring (i.e. deposition of collagen) within the brain. Normally the brain responds by gliosis.

A well as causing meningitis, tuberculosis may cause localised areas of infection in the brain known as tuberculomas (e.g. case 6718)

PRIMARY BRAIN TUMOUR: ASTROCYTOMA

CASE 25280

Clinical information

The patient was a man aged 46 who first came to medical attention when he was knocked unconscious in a brawl. While waiting to be seen in the Emergency Department he had a grand mal seizure. A skull x-ray showed a linear fracture in the left temporo-parietal area and he was admitted to hospital for observation. During this time he was noted to be dysphasic and disorientated with a very poor memory. More careful examination found mild right lower facial weakness and weakness of grip in the right hand.
Further imaging suggested an intracerebral haematoma and exploratory surgery was performed. A biopsy was taken at that time.
Post-operatively the patient became aggressive and hostile and had to be detained to a mental hospital under certification. There his condition deteriorated with dyspraxia, right homonymous hemianopia and right facial weakness.
Terminally he was transferred back to the RAH where he died, a year after his first presentation.

Specimen description

The specimen consists of the cerebral hemispheres sectioned in the transverse plane. There is a poorly defined mass with infiltrating margins within the white matter of the left temporo-parietal region. It is about 60mm in diameter with areas of haemorrhage and extensive necrosis. There is surrounding oedema with compression of the left lateral ventricle and midline deviation to the right. The tumour extends through the splenium of the corpus callosum into the septum pellucidum and into the left uncus and hippocampus that appear nodular and are markedly notched from herniation. The pons is compressed and there is haemorrhage and necrosis within it posteriorly. There is old brown stained superficial cortical damage affecting the inferior and lateral surfaces of the right temporal pole and the right orbital gyri.

Diagnosis

Astrocytoma (confirmed histologically) with transtentorial herniation

Comment

There are a wide range of primary brain tumours. One of the largest groups is the gliomas - tumours arising from the glial or supporting cells of the CNS. Gliomas include astrocytomas (the most common), oligodendrogliomas and ependymomas. Patients with brain tumours may present in a number of ways. This patient demonstrates some of the common symptoms: seizures and loss or disturbance of neurological function - in this case personality change; dysphasia; disorientation; poor memory; dyspraxia etc. Patients may also have symptoms of slowly rising intracranial pressure: headache, typically worse in the morning, and nausea.
The different primary brain tumours tend to affect different locations in the brain and the type can be suggested from its location. Astrocytomas tend to arise in the cerebral hemispheres but also occur elsewhere, including the deep grey matter and spinal cord. Glioblastoma multiforme is a highly aggressive astrocytoma characterised by the presence of necrosis. You will find that due to changes in classification, some of the astrocytomas showing necrosis in the older specimens are only diagnosed histologically as astrocytoma, but they could be glioblastomas. If you see a large single tumour in a cerebral hemisphere showing necrosis, chances are it's a glioblastoma. Ependymomas arise in relation to the ventricular system and are also relatively frequent in the spinal cord.
In children, cerebellar tumours tend to be cystic astrocytomas or medulloblastomas.

Note that most primary brain tumours do not metastasise outside the CNS though some may spread within the meninges.

MENINGIOMA

CASE 21117

Clinical information

The patient was a woman aged 83 who had apparently been in good health until one week before admission when she began to have difficulty with swallowing and talking. Her condition rapidly deteriorated. On admission she was almost mute. She died a week later from bronchopneumonia.

Specimen description

The specimen comprises the upper portion of the cerebral hemispheres cut in the transverse plane. The dura has been reflected back from the surface of the right hemisphere. A large lesion 40mm in diameter arises from the under surface of the dura over the right fronto-parietal region and compresses the underlying brain leaving a smooth depression. The tumour has a nodular pale appearance and although it has compressed the cortex, it has not invaded it.

Diagnosis

Meningioma

Comment

This specimen has the classic appearance of a meningioma, the essentially benign tumours that grow from arachnoidal cells of the meninges and compress but do not invade the underlying brain. Surgical excision is usually curative. Uncommonly the tumour may recur, especially if there has been incomplete removal.

SECONDARY BRAIN TUMOUR: MULTIPLE METASTASES FROM A PRIMARY LUNG TUMOUR

CASE 21463

Clinical information
A man aged 53 was diagnosed with carcinoma of the lung that was treated with radiotherapy. Two months later he was admitted to hospital because of personality changes and finally he was admitted to hospital following a seizure. On examination the only significant finding was a left hemiplegia. Palliative care was commenced and he died 2 weeks later.

Specimen description
The specimen consists of two coronal slices through the hemispheres viewed from behind and a transverse slice through the brain stem and cerebellum. Throughout the specimens are multiple discrete lesions up to 25mm in diameter. They are well-demarcated, pale brown-grey in colour and most show central necrosis. There is one slightly haemorrhagic lesion in the right putamen. There is generalised oedema of the white matter and the ventricular cavities are compressed.

Diagnosis
Metastatic carcinoma

Comment
These lesions have the classic appearance of metastatic tumours in the CNS. They are deceptively well-demarcated but the tissue has a quite difference appearance and texture from normal brain substance.

At post-mortem the primary carcinoma was present in the upper lobe of the right lung and there was involvement of mediastinal nodes and multiple metastases in the viscera.

HERNIATION

CASE 22308

Clinical information
The patient was a man aged 58 employed as an orderly at the RAH. He collapsed at work one morning and fell to the left side. On examination he was unable to use the left arm and leg. He was drowsy but responded to questions, stating that he had felt "funny' for 30 minutes before the collapse, but had no headache. He died on the 2nd day.

Specimen description
The specimen is a transverse section through the cerebral hemispheres, sparing the right cingulate gyrus and parasagittal cortex. Much of the right cerebral hemisphere is markedly swollen with blurring of the normally clear demarcation line between cerebral cortex and underlying white matter. This is in the territory of supply of the right middle cerebral artery. Within this area focally towards its anterior end is an area of dark brown haemorrhagic discolouration of the cortex. There is midline shift with displacement of the right lateral ventricle and the right cingulate gyrus has herniated under the falx, causing a notch on its upper surface. A second area of haemorrhagic discolouration is present in the right posterior cerebral artery territory about 30mm anterior to the occipital pole. The base of the brain (see back of pot) shows marked uncal and parahippocampal gyral notching bilaterally, right worse than left.

Diagnosis
Right middle cerebral artery infarction with right subfalcine herniation, bilateral uncal/transtentorial herniation and probable small right occipital haemorrhagic infarct secondary to compression of the right posterior cerebral artery

CASE 24145

Clinical information
The patient was a 49-year old man with a long history of tuberculosis that was being treated with streptomycin, para-aminosalicyclic acid and isoniazid before his final admission. On that admission he had pancytopenia and he died suddenly during blood transfusion.

Specimen description
The specimen is a transverse section of the cerebellum and brainstem. There is a large, well-demarcated dark brown lesion within the right cerebellar hemisphere that measures 50 x 35mm. The right dentate nucleus is displaced to the left and there is compression of the 4th ventricle. There is a small amount of subarachnoid blood on the surface of the cerebellum and marked bilateral cerebellar tonsillar grooving, right worse than left.

Diagnosis
Cerebellar haemorrhage, probably related to thrombocytopenia, with tonsillar herniation

Comment
Tentorial or uncal herniation is a complication of raised pressure within the supratentorial compartment. As a lesion increases in size, initially the sulci become narrowed and the overlying gyri are compressed against the dura, the lateral and third ventricles are compressed, midline structures are deviated towards the opposite side and the contralateral foramen of Monro may be compressed $\rightarrow$ dilation of the lateral ventricle.

As the lesion continues to expand, the ipsilateral uncus and medial part of the parahippocampal gyrus (on the infero-medial aspect of the temporal lobe) herniate through the tentorial incisura cerebelli.
The effects of tentorial herniation result from the compression (and eventual pressure necrosis) of various structures:

- the ipsilateral III cranial nerve → pupillary dilation (earliest sign and may occur before consciousness is lost)
- the ipsilateral (occasionally contralateral) posterior cerebral artery → infarction
- the ipsilateral cerebral peduncle is compressed against the free edge of the tentorium → contralateral hemiparesis. Occasionally the contralateral cerebral peduncle is compressed against the free edge of the opposite tentorium → hemiparesis on the same side as the expanding lesion.
- the reticular formation in the brainstem→ loss of consciousness
- displacement of the brainstem → distortion and compression of vessels within the midbrain and pons → haemorrhage (Duret's haemorrhages) and infarction → death
- other

Occasionally transtentorial herniation may be bilateral.

Patients with raised ICP also frequently develop elevated blood pressure and decreased heart rate associated with brainstem shift and increased sympathetic activity.

Tonsillar herniation usually develops secondary to an expanding lesion in the posterior cranial fossa but it may occur in association with supratentorial expanding lesions. The cerebellar tonsils become pushed through the foramen magnum with apnoea resulting from distortion of respiratory centres in the medulla.

Subfalcine herniation results from an expanding mass in the frontal or parietal lobe causing the ipsilateral cingulate gyrus to be pushed under the free edge of the falx. This may compromise the circulation in the pericallosal arteries resulting in infarction of parasagittal cortex.

Areas of haemorrhage and necrosis frequently develop in the tissue compressed in herniations.

Diffuse brain swelling and hydrocephalus are also causes of raised intracranial pressure in addition to focal lesions such as haemorrhages and tumours. Oedema around a focal lesion (e.g. haemorrhage, tumour) may contribute to the raised pressure and is the main cause of raised ICP following infarction.