The pathology of traumatic brain injury (TBI): a practical approach

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Abstract

TBI is one of the most common causes of hospital admission and a leading cause of death, it is therefore frequently encountered by pathologists in the coronial and medicolegal services. It is not a single entity but a heterogeneous group of pathologies which are caused by different mechanisms and have different survival outcomes and medicolegal implications. TBI is caused by physical contact or acceleration and deceleration mechanisms or a combination of these. It is classified into focal and diffuse damage; both of which should be correlated with the circumstances of death and clinical history before a conclusion can be made regarding their contribution to the cause of death. Focal damage in the brain such as intracranial haemorrhages and contusions can be easily recognised by naked eye examination, but they require detailed description of their anatomical distribution and extent. However the diffuse traumatic brain damage, particularly those related to axonal injury, requires histological and immunohistochemical (IHC) tests to be established.

The understanding of the medicolegal implications of focal and diffuse axonal injury has evolved in the last decade with the introduction of better IHC and neuro-radiological methods.

Keywords BAPP; diffuse axonal injury; head injury; intra cranial haemorrhage

Introduction

Head injury is a substantial cause of morbidity and mortality. It leads to approximately one million accident and emergency department visits annually; of these cases 6% suffer permanent disability and 3% die.[1] Indeed, head injury and consequent traumatic brain injury (TBI), is a leading cause of death in Westernised society and accounts for one quarter to one third of all traumatic deaths.[1]

TBI is a heterogeneous group of pathologies with different mechanisms (Box 1) and prognoses. The severity of injury (mild, moderate, or severe) depends upon the amount of energy transferred to the brain by direct, rotational, and inertial forces, the site of impact, and the duration of injury. There are several classification systems available to describe different types of TBI. These include primary injury versus secondary injury. Primary injury occurs at the moment of impact (e.g. bleeding, contusion) whilst secondary injury occurs as a consequence of the primary injury (e.g. swelling, raised intracranial pressure). Focal versus diffuse injury is another helpful classification which has better clinical-pathological correlation (Figure 1).

Mechanisms of TBI

Mechanism of TBI

- Contact
- Acceleration and deceleration
- Combination

Box 1

The relative of head injury and TBI means pathologists undertaking medicolegal autopsies are likely to encounter such cases. In some instances a naked eye examination by an experienced pathologist will be sufficient to provide a cause of death, such as in cases with severe contusions, lacerations, and intracranial haemorrhages in the context of a clear and undisputed witnessed accident such as road traffic collision (RTC). However, a more thorough examination is both prudent and necessary to garner more detail in less clear cut cases and referral for specialist neuropathological opinion may be required (Boxes 2 and 3). It is therefore important for pathologists performing medicolegal autopsies to be familiar with the classification and mechanism of head injury, the types of TBI likely to be encountered, the necessary approaches to examination and investigation, and potential pitfalls.

Contusions and lacerations

Contusions represent localised damage to the brain with preserved pia, whilst with lacerations there is additional damage to the pia and meninges. These lesions are described as direct (coup) or indirect (counter coup) and can be differentiated based upon their relation to the site of impact. The direct lesions (Figure 2e) can be associated with severe scalp bruises and sometimes fracture sites such as those occurring on the dorsal surfaces of the cerebral hemispheres and cerebellum. The indirect lesions (Figure 2d) are often diametrically opposite to the site of impact and are classically seen in the anterior and inferior surfaces of the frontal and temporal lobe following impact at the back of the head such as those resulted from accelerated and unprotected backward falls.

These lesions are more severe on the crest of the gyri and macroscopically present as punctate haemorrhage or confluent haemorrhagic lesions. Microscopically, they appear as small and streak haemorrhages oriented perpendicular to the surface. More extensive haemorrhage may involve the underlying superficial or, in severe cases, the deep white matter. Associated haemorrhage, including subarachnoid haemorrhage (SAH) can be exacerbated by the poor coagulation status of the patient, such as in hepatic cirrhosis or anticoagulation therapy. After several days to weeks duration the contusions may appear orange-brown due to haemosiderin pigment deposition, and are associated with fragmentation and irregularity of the cortical tissue.

The main pathological differential diagnosis is haemorrhagic infarction.

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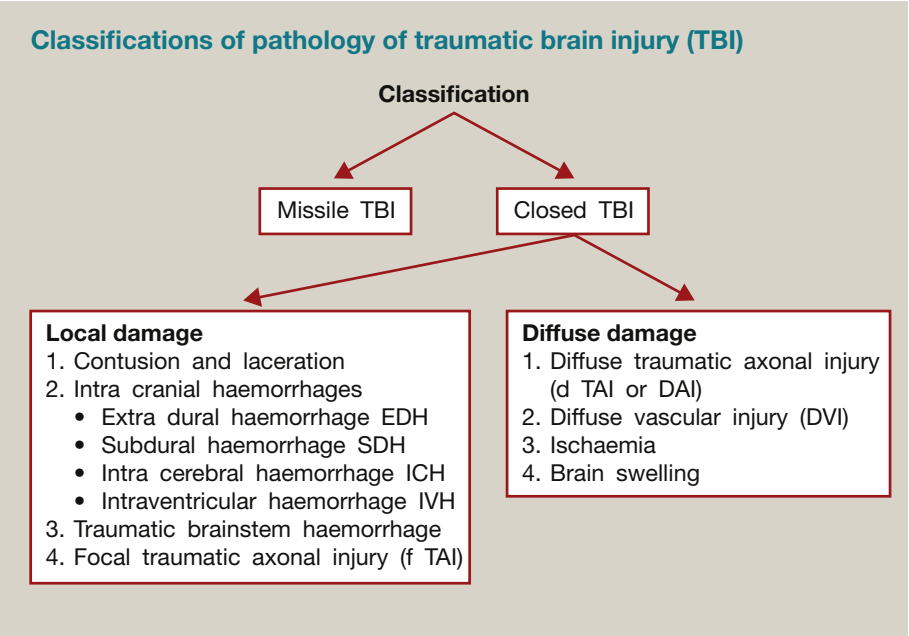

Figure 1

These lesions are not usually the cause of immediate fatality, and full recovery may be expected in the absence of more severe injuries. Identification of the pattern, site and severity of the contusions and lacerations may be useful indicators for the mechanism and the magnitude of the force applied. However, careful correlation with other injuries and natural disease is required.

A special type of contusion is the gliding contusion (Figure 3c). This describes focal vascular damage in the parasagittal cortex and adjacent white matter (sometimes restricted to the white matter only) of the cerebral hemispheres. They are commonly related to more diffuse TBI and result from angular and rotational movement of the brain leading to tensile strain on blood vessels in the parasagittal region (please see below Section 3). For this reason they are often associated with a worse outcome. They are also difficult to identify without good fixation of the brain and close examination (Box 4).

Rationale for brain examination in TBI in medicolegal autopsy

- Establish cause of death in relation to other systemic injury
- Provide details of the nature and extent of intracranial and intracerebral pathologies
- Provide information regarding the mechanism of injury
- Provide information related to the timing of injuries
- Correlation of pathological findings with pre-mortem imaging and clinical management
- Support research into traumatic brain injury and its prevention and management
- Provision of national statistics

Box 2

Intracranial haemorrhages

Extradural haemorrhage (EDH): EDH occurs in about 39% of referred cases of traumatic brain injury to King's College Hospital and is frequently associated with skull fracture (96%) and scalp bruises (98%). However, EDH can also occur without skull fracture, particularly in children. This type of haemorrhage is usually caused by damage to meningeal blood vessels causing enlarging haematoma that strip the dura from the skull to form a circumscribed ovoid mass (Figure 2a). It is classically seen after fracture to the squamous temporal bone resulting in tearing of the middle meningeal artery. Although the majority present immediately after the impact, in a few cases the haematoma may

Circumstances in which a comprehensive brain examination may be required for suspected traumatic brain injury

- Likely criminal or legal proceedings
- Minimal pathology on macroscopic examination despite a history of head injury
- Vague account of circumstances leading to death such as an elderly person found at the bottom of a flight of stairs (TBI vs. haemorrhagic stroke)
- History of old head injury (acute vs. acute-on-chronic vs. chronic)
- Contribution of pre-existing pathology to brain injury e.g. amyloid angiopathy or coagulopathy
- Delayed presentation e.g. delayed intracranial bleed
- Dispute regarding the nature of the incident leading to head injury and the mechanism of injury
- Death following neurosurgical intervention
- Questions regarding the clinical management of the head injury

Box 3