BRAIN - EXAMINATION

The fixed brain weighs 1588 g. On external examination there is no subdural or subarachnoid haemorrhage and there is no meningitis. No surface contusions are seen. There is no asymmetrical swelling and no evidence of uncal or tonsillar herniation or necrosis. The vessels of the Circle of Willis show no evidence of atheromatous degeneration or aneurysm formation.

On coronal sectioning there is no evidence of cortical atrophy. Focal haemorrhages are present within the deep white matter of both frontal lobes in a parasagittal location. They are also present in the left thalamus and internal capsule. In addition there is diffuse vascular congestion throughout the white matter. No focal haemorrhage is present in the corpus callosum although it appears congested. There is no evidence of midline shift or internal herniation but there is evidence of a mild degree of ventricular compression. There is no caudal descent of the mammillary bodies.

On sectioning of the brain stem there is diffuse punctate haemorrhage. The cerebellum shows no macroscopic abnormality.

The spinal cord was not examined.

HISTOLOGY

Sections were taken from the right and left frontal lobes, corpus callosum, left and right hippocampi, hypothalamus, cerebellum, midbrain and pons.

Frontal Lobe Examination of the sections from the right and left frontal lobes shows the presence of white matter contusions. This is associated with tissue necrosis and a macrophage reaction. Surrounding this there is extenstive diffuse axonal damage. There is no evidence of hypoxic/ischaemic necrosis of the adjacent cortex. There is no meningoencephalitis.

Corpus Callosum Examination shows diffuse axonal damage.

Left Internal Capsule Examination shows extensive diffuse axonal damage.

Hypothalamus Examination shows no evidence of haemorrhagic necrosis. There is however axonal damage in the hypothalamic region.

Midbrain Examination shows diffuse axonal damage. There is no secondary brain stem haemorrhage. The substantia nigra shows no evidence of degenerative change.

Pons Examination shows the features of severe diffuse axonal damage.

Cerebellum Examination shows no evidence of cortical necrosis. The Purkinje cells are preserved. A small gliotic scar is present in the white matter.

Immunohistochemistry for neurofilament protein and for the myelin stain LFB/H & E confirms the distribution and severity of diffuse axonal damage.

COMMENT:

In summary, the predominant finding in this case is the severe diffuse axonal damage. Diffuse axonal damage is thought to be due to interruption of movement of cytoplasm through axons with subsequent accumulation of axonal material seen on H & E examination as small eosinophilic bulbs. In this case it was distributed throughout the deep white matter, corpus callosum and brain stem. This pattern of damage is consistent with being caused by trauma. It is indicative of a severe head injury.

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