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Spinal cord transection in utero

Several cases of cervical cord damage caused by birth trauma have been described.¹⁻³ But to our knowledge no case of antenatal cervical cord damage has been reported. We describe such a case.

Case report

A 32-year-old woman (gravida 2) was found to have a breech presentation at 36 weeks' gestation in an otherwise normal pregnancy. One unsuccessful attempt at external cephalic version was made gently at that time. The pregnancy was allowed to go to term, when the baby was delivered by elective caesarian section.

A boy weighing 3.360 kg was delivered easily and in good condition but,

Top: macroscopic view of dorsal aspect of spinal cord showing 25-mm length of softening (between broad arrows). Cervical cord is to left of picture. Bottom: transverse section of upper thoracic spinal cord showing almost complete destruction and necrosis of cord tissue with macrophage invasion (n). dr=Dorsal roots. a=Thickened arachnoid. s=Anterior spinal artery. (Luxol fast blue Nissl. $\times 15$.)

apart from some facial grimacing, made no respiratory effort. Intermittent positive pressure ventilation was started immediately and presented no difficulties. Subsequent attempts to discontinue this were, however, associated only with further facial movements; the infant made no respiratory effort despite his deteriorating condition. This persisted for the remaining 80 hours of his life, after which assisted ventilation was discontinued.

On examination at birth the baby was noticed to have normal movements of the face, palate, and sternomastoids. There was a flaccid paralysis of his arms with absent reflexes, the abdominal reflexes were absent, and there was increased tone and brisk reflexes in his lower limbs with spontaneous clonus at the hips and knees. Plantar stimulation and pin-prick on the feet produced a strong withdrawal reflex.

Sensory examination showed grimace to pin-prick on the face down to the angle of the jaw, but no response to stimulation of the arms or trunk. These neurological findings, which remained unchanged throughout the infant's life, suggested a cord lesion extending down from the level of the third and fourth cervical segments.

Necropsy showed no bony injury and no abnormality on macroscopic examination of the brain. There was gross softening and shrinkage of a 25-mm length of the lower cervical and upper thoracic cord (see figure). Microscopic examination of transverse sections of the softened areas of cord showed almost total destruction of cord tissue, with cystic spaces and invasion by macrophages. The anterior spinal roots were atrophic but the posterior roots showed no abnormality. There was thickening of the arachnoid mater (figure). The anterior spinal artery was normal. At the level of the lesion the cord showed degeneration of the ascending tracts, while below the lesion there was degeneration of the lateral corticospinal tracts with normal anterior horn cells.

Comment

The clinical and pathological findings described in this report support a diagnosis of antenatal cord trauma. At birth there was clinical evidence of a lesion at the third cervical segment, with a sensory level and presumptive affection of the phrenic nerve and intercostal muscles; the child also had well-developed spasticity of the legs. The pathological evidence of shrinkage and macrophage invasion, with degeneration of ascending and descending tracts is compatible with a lesion three to four weeks old.⁴

Although no bony injury was found, the thickening of the arachnoid mater suggests trauma as the cause, and Hadley⁵ has shown that in the normal newborn compression and shearing forces affecting the spinal cord can be produced easily without causing any bony injury.

The attempted external cephalic version, which perhaps inadvertently extended the fetal neck, occurred at a time compatible with the age of the injury and should be considered the likely cause.

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Rhabdomyosarcoma of the right ventricle associated with polyarthritis

We report a patient with a rhabdomyosarcoma of the right ventricle who presented first with a seronegative polyarthritis and later developed congestive heart failure and signs of pulmonary stenosis.

Case history

A 53-year-old woman had had mild, symmetrical, seronegative polyarthritis for four years affecting her hands and feet. In August 1975 her