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Paraplegia due to spinal epidural haematoma

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Epidural haematoma may form at any spinal level and no age is exempt. The rate of development of the signs and symptoms of spinal cord and radicular compression due to epidural haematoma varies from case to case. In some patients there are no warning symptoms, and the case presents straight away after a minor injury with a paraplegia of sudden onset and rapid development. In other patients there is a long retrospective history of intermittent minor attacks of pain in the neck or in the back accompanied by transient symptoms and signs of root compression with pain radiating into the limbs or around the sides of the trunk, the site of the pain in the back and of its accompanying symptoms of root compression depending upon the level at which the bleeding starts. These minor prodromal attacks may occur for years until there is ultimately a severe attack in which the spinal compression results in paraplegia.

Spinal epidural haematoma is still not usually recognized as a possible cause of paraplegia early enough for effective surgical treatment to be undertaken, and undiagnosed and untreated cases may be more frequent than usually supposed. Since a decompressive laminectomy performed soon after the onset of the paraplegia can relieve the spinal paralysis, prompt diagnosis of the condition is imperative.

In previous communications the results of pathological examination of epidural tissue taken from the site of the haematoma are seldom reported. Discussing the pathogenesis of spinal epidural haematoma, Kaplan and Denker (1949) suggested on theoretical grounds the possibility of a pre-existent venous anomaly and that intermittent and slow leakages of blood from this abnormality were provoked by repeated minor injuries or straining efforts which raised intra-abdominal venous tension. After pathological examination of tissue they had removed from the site of epidural haematomata they felt, as did Schultz, Johnson, Brown, and Mosberg (1953), that repeated small haemorrhages had occurred into granulation tissue organizing previous haematomata. Maxwell and Puletti (1957) observed at laminectomy an epidural haematoma apparently arising from a ruptured epidural varicose vein. In both our own cases a small venous angioma was seen and removed from the site of the epidural haematoma at the time of operation. We presume that the haematoma formed after straining as a result of a wave of increased pressure passing from the abdominal and pelvic veins into the spinal epidural venous plexus, and that blood leaking from the weak veins of the venous angioma formed a spinal epidural haematoma and compressed the spinal cord.

The first reported case of spinal epidural haemorrhage was found at necropsy in the upper cervical region of a housemaid who developed quadraplegia while straining at stool and died two hours later Bain (1897). Successful surgical relief of paraplegia due to spinal epidural haematoma was first reported by Jonas (1911). In his patient, a farmer who had fallen from a hay stack the day before, the haematoma had collected in the mid-thoracic epidural space. Lowrey (1959), reviewing the literature, tabulated the clinical features of the 24 previously reported cases: five died, 11 recovered after laminectomy, and in eight the symptoms of spinal compression were only partially improved by laminectomy. In a large proportion of the patients a minor injury or straining effort precipitated the bleeding into the spinal epidural space and the onset of symptoms of spinal compression.

Hopkins (1899) described a patient who developed symptoms of spinal epidural compression after suddenly lunging forward while shovelling coal, and Shenkin, Horn, and Grant (1945) recorded a patient in whom the trouble appeared after a sudden twist in bed. In three patients described by Lowrey (1959) haematomas collected following a fall from a chair in one patient, in another after lifting a heavy weight, and in the third patient while he was pulling a conveyor chain. One of Ver Brugghen's (1946) patients developed the spinal epidural haematoma after falling a distance of 4 ft. onto the buttocks.

CASE REPORTS

CASE 1 At 10 years of age this girl experienced attacks of stabbing pain between the shoulder blades, the pain radiating forwards around the chest causing her momentarily to catch her breath. Examination revealed no

physical abnormality, and she continued to take part in school games without hindrance. At 15 years of age she started work in an office. From time to time aching pains were felt between the shoulders, and she thought that the pains were due to her badly shaped office chair. At 17 years of age she had a slight cough accompanied by stabbing pains around the chest, and examination again revealed no abnormal physical signs; sputum tests and chest radiographs were negative. During the next two years she would often awake in the morning and complain of aching pain between the shoulders which her mother relieved by local applications of heat and massage.

On three occasions the pain was sufficiently severe to prevent her going to work, at this time even slight movement provoking severe cramps around the chest and upper abdomen. Intervals of freedom lasted three months or more. Flexion of the neck during the attacks sent pains around the chest and one particularly severe attack was precipitated when she split a cup of tea down the front of her dress causing her to bend her neck sharply to inspect the damage. At 19 years of age, and now engaged to be married, she amused her friends and relations with remarks concerning the painful cramps in the back and around the ribs which, to her, were a consequence of the hugs of courtship. On several occasions she remarked in confidence to her mother that the hugs of her prospective husband sometimes left her weak at the knees. She awoke on the morning of 7 April 1959 with quite severe pain between the shoulders; she got out of bed and went into her mother's room complaining of tingling sensations in both the calves. She wriggled her legs to try to rid herself of this abnormal sensation, but found that the legs did not move as well as they should. Her parents helped her out of bed and encouraged her to walk around the room. With difficulty she struggled to the bathroom, and found herself walking with a high stepping gait. While sitting on the W.C. passing water, she lost urethral sensation, later stating that she heard herself passing water, but had none of the usual accompanying sensations. It was very difficult for her to get back into bed, but once there she noticed a loss of sensation moving up her body. As the sensory loss travelled to the mid-thoracic region her legs shook in a rhythmic beat, and she had severe headache accompanied by vomiting. She was admitted as an emergency to Hope Hospital, Salford, under the care of Mr. T. G. Barlow.

In hospital an hour or so later, examination demonstrated a loss of sensation to the sixth dorsal dermatome. A persistent tingling sensation in the legs and trunk made it difficult for her to cooperate in sensory testing. Vibration and postural senses were absent. All the reflexes were exaggerated. There was knee and ankle clonus with bilateral extensor plantars. One hour later the limbs were completely flaccid: there was complete loss of pain sensation up to the mid-thoracic level with a band of hyperaesthesia to about T.3. Sacral dermatome sensation to pin prick was preserved. Radiographs of the chest and of the thoracic spine were normal. Lumbar puncture showed a complete manometric block, with 95 mg. % protein. A lumbar myelogram with 6 ml. of ethiadone (Dr. Laing) revealed an irregularly shaped block in the column of contrast opposite T.4.

At laminectomy (T.2 to T.6), about 12 hours after the onset of the paraplegia, the epidural space was occupied by a carpet of solid black blood clot 5 cm. long and 1 cm. thick. Several distended venous channels transversed the epidural space, and at the upper margins of the haematoma a small vascular malformation was seen. The haematoma was scraped away and the malformation removed for histology. Haemostasis was difficult. Subsequent microscopic examination of the vascular nodule proved it to be sclerosing fibrous tissue containing an excess of vascular channels in the pattern of a venous angioma. Within 24 hours of laminectomy the level of the sensory loss had retreated down the trunk three or four segments. In 48 hours sensation to light touch had returned to the trunk and legs. There was voluntary movement of the right toes and of the right knee and right ankle. A flicker of voluntary movement was apparent in the left leg but the bladder remained paralysed. Three days post-operatively there was good movement in the right leg and extensor muscles in the left leg had recovered. After a week good power developed in both the legs. Sensation was present in the bladder, though the patient could not voluntarily empty the bladder. Two weeks post-operatively the patient began walking and by the third week she was walking well and bladder control was normal. She was married four months after the operation. At a recent follow-up clinic (three years post-operatively) she reported herself quite well in all respects.

CASE 2 A previously healthy boy of 15 years while playing in the garden strained his back as he lurched backwards and tripped over a 2 foot high wire. Soon after the fall he felt quite severe pain and stiffness in the lumbar region, also pain radiating into both flanks. The lumbar curve was flattened but no local spinal tenderness was found. Radiographs taken in the Casualty Department showed no abnormality. Bed rest at home for 12 days did not help, and with any attempt to sit up sharp pains went into the flanks and down the thighs. Because of the persistent pain he was admitted to Park Hospital, Davyhulme (Dr. H. J. Wade). The patient could not now lift his legs from the bed because of weakness in the hip flexors, although active knee and ankle movement was still possible. Passive straight leg raising to 35° sent pain down the thighs. The temperature was 100·6° and white cell count 20,900. Two days later (14 days after the accident) the right leg became very weak and the knee jerk absent. Flexion of the lumbar spine was limited by painful spasm. No local spinal tenderness was found. Pressure was 140 mm. at lumbar puncture and spinal manometric studies were normal. The fluid contained 72 mg.% protein and four lymphocytes. Later that day the leg weakness was more marked and both knee jerks absent; there was sensory loss as far as the mid thigh. An emergency preoperative lumbar myelogram (6 ml. of ethiodone) showed that the contrast column in the spinal theca was very thin from opposite the body of L.3 to the body of L.4 and it appeared to be pushed back from the vertebral bodies. An extradural lesion with posterior and anterolateral compression of the theca was suggested, and it was decided to operate immediately.

The lumbar laminectomy (L.2 to L.5) disclosed an infected epidural haematoma, and seen embedded in the thickened and congested epidural fat was a small mass of tangled blood vessels. (Subsequent microscopic examination of the vascular nodule, measuring 0·5 cm., proved it to be fibro-connective tissue and fat in which numerous well-formed vascular channels were identified. These appearances were consistent with the impression gained at operation that the haematoma came from a small leaking epidural angioma.) In the anterolateral aspect of the spinal canal opposite the body of L.3 we found a loculus of pus containing *Staphylococcus pyogenes*. The blood clot and purulent material was sucked away then the muscle layers closed. Antibiotics were given postoperatively. Within a week the boy was walking and the knee jerks returned. A little weakness persisted in the extensors of the right foot but two weeks after laminectomy he could walk quite well without assistance and all the abnormal neurological signs had cleared. Pyrexia continued for a further week, but it settled with antibiotics and the wound healed soundly.

SUMMARY

A description is given of the clinical, operative, and pathological findings in two patients with paraplegia due to spinal epidural haemorrhage. The source of bleeding in both cases was a small venous angiomatous malformation in the epidural space. In the first patient there was a long prodromal history before the development of an upper thoracic epidural haematoma and paraplegia; the second patient was free from symptoms until the precipitating injury which resulted in an upper lumbar epidural haematoma and paraplegia. Both patients completely recovered following laminectomy and removal of the haematoma.

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