Spontaneous spinal epidural haemorrhage: good results after early treatment

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SYNOPSIS Extravasation of blood in the spinal epidural space is an uncommon but often disastrous problem. Severe trauma, anticoagulants, bleeding diatheses, and intraspinal vascular malformations have been associated with such haemorrhage, but occasionally it occurs without apparent cause. It may then be confused with transverse myelopathy or vascular occlusion. Early diagnosis by myelography and treatment by surgery can result in good recovery, as illustrated by two cases.

CASE 1

M.T., a 15 year old student, awoke from sleep with pain in both shoulders, radiating down the ulnar aspect of the right arm and increasing with neck movement. The pain worsened progressively and extended into the entire back. Within eight hours he was unable to walk, demonstrating generalized spasticity with paraparesis. Hyperaesthesia of the C8 dermatome and weakness of the right hand were present. Moderate tenderness was noted over the cervicothoracic spine.

Lumbar puncture, including manometrics, was normal. Cervical spine radiographs were unrevealing. Myelography demonstrated a dorsal block from C6 to T1 vertebral level with spinal cord displacement to the left.

Laminectomy (C6–T2) was performed 12 hours after the onset of symptoms. A large haematoma in the right dorsal epidural space was evacuated. Neither infection nor vascular anomaly was found. Dural pulsations appeared after removal of the clot. Pathological examination showed epidural connective tissue without vascular malformation. A post-operative survey for bleeding diathesis was unrevealing.

Complete resolution of paraparesis and spasticity was evident by the second day after operation. Myelography was repeated and showed that the block had cleared. The profound right hand deficit slowly improved over the next several months. No neurological sequelae were present one year after operation.

CASE 2

R.D., a 29 year old salesman, was well until he experienced the sudden, spontaneous onset of intense interscapular and left arm pain while crossing a street. Within 15 minutes progressive weakness of both legs appeared. An hour after the onset of symptoms he was unable to move either leg; severe arm pain persisted. Physical examination in the hospital showed complete paraplegia with a sensory level at T10 dermatome. The lower extremities were areflexic, the anal sphincter was flaccid, and priapism was present.

Cisternal and lumbar myelography were performed. The cerebrospinal fluid was normal. A complete myelographic block was identified at T1 vertebral level from below, at C6 vertebral level from above. The sensory level ascended to T2 dermatome during the radiographic study.

Laminectomy (C7, T1) was performed within six hours of the onset of symptoms. An epidural haematoma, composed of both solid and liquid clot, was encountered posteriorly, compressing the dural sac forward and to the left. Removal of the haematoma resulted in prompt return of dural pulsations and complete expansion of the dural sac. Pathological examination of epidural tissue from the haematoma site showed haemorrhagic connective tissues. A definite vascular lesion could not be seen, and a bleeding diathesis could not be identified during the patient’s convalescence.

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On the first day after operation, the myelographic block was no longer present. By the third day the sensory level had descended and voluntary toe movement returned bilaterally. Neurological improvement progressed steadily thereafter. At discharge six weeks after operation the patient was fully ambulatory with normal bowel and bladder control and without sensory deficit.

**DISCUSSION**

Since its original description by Bain in 1897, spinal epidural haemorrhage has been reported sporadically (Kaplan et al., 1949; Sadka, 1953; Schultz et al., 1953; Lowrey, 1959; Lougheed et al., 1960; Cube, 1962; Gold, 1963). Its aetiology remains obscure. Coagulation defects and bleeding diatheses have been associated with some of the reported cases (Cube, 1962). Additional cases are associated with trauma or extreme exertion by the patient. A few more patients harbour underlying venous malformations or angiomas which rupture as blood pressure abruptly increases (Kaplan et al., 1949; Gold, 1963). Finally, a number of patients have no identifiable cause for their neurological catastrophe, presumably having bled 'spontaneously' (Schultz et al., 1953; Lougheed et al., 1960; Gold, 1963).

Symptoms are usually acute and demonstrate localized pain over the spinal area that is involved. Radicular pain, prominent in both of our patients, may precede the abrupt onset of paraparesis by days or weeks (Lougheed et al., 1960). Sensory and motor neurological deficits usually occur simultaneously with pain, but may be delayed and slowly progressive.

The differential diagnosis of sudden, non-traumatic paraplegia may be assisted by early myelography. Transverse myelopathy and spinal artery thrombosis rarely show abnormalities on a myelogram. Arteriovenous malformations are commonly accompanied by abnormal blood vessels. Haematomyelia is associated with enlargement of the spinal cord, although intraspinal epidural haemorrhage resembling an intramedullary lesion has been described (Lougheed et al., 1960). Usually acute epidural infection can be ruled out by the absence of systemic signs. As a rule, cord compression from neoplasm is associated with bony abnormalities and a less precipitous course. Only myelography will make the diagnosis possible by showing a subarachnoid block, usually with asymmetrical compression of the cord.

Our two patients with spontaneous spinal epidural haemorrhage recovered from profound neurological deficits after prompt surgical decompression. Such a recovery cannot be expected if there is any delay in diagnosis and treatment.

**REFERENCES**


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