Spinal subarachnoid haemorrhage presenting as spinal block without meningism

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Summary: A case of spinal subarachnoid haemorrhage with progressive spinal cord compression and without any evidence of meningism is described. Spinal block was demonstrated by myelography and computerized tomography and surgical decompression of the subarachnoid blood clot resulted in almost complete recovery. A diagnosis of spinal subarachnoid haemorrhage should be considered in any patient who presents with acute back pain and slowly or rapidly progressive neurological signs in the limbs, even when meningism is absent.

Introduction

Spinal subarachnoid haemorrhage is a well-recognized but seldom discussed clinical entity which frequently presents diagnostic difficulties. Henson & Croft (1956), in their major review of this condition, noted that the symptoms and signs of meningism were usually so marked that a mistaken diagnosis of intracranial subarachnoid haemorrhage was often made, and surprisingly minimal spinal cord signs were found. Most of their cases were treated conservatively and surgical intervention was not deemed necessary in their single case in which a complete spinal block was demonstrated on myelography.

In contrast, we describe here a patient with a spinal subarachnoid haemorrhage without any symptoms or signs of meningism presenting with prominent and progressive spinal cord signs, and in whom a total spinal block was demonstrated on myelography and computerized tomography (CT). Surgical decompression of the block resulted in resolution of most of the signs.

Case report

A 64 year old hypertensive woman awoke one morning with severe pain in her lower back and then difficulty walking. The next day the pain radiated down the front and back of her right leg, and over the next 10 days she developed increasing weakness of her legs and increasing difficulty with micturition. At the end of this time she went into urinary retention with overflow incontinence, and bowel function became severely impaired. At no stage did she complain of headache, photophobia or sensory symptoms and neck stiffness was never detected.

On transfer to The National Hospital she was noted to be slightly mentally obtunded, being vague and slow to answer questions. There was no neck stiffness or photophobia and Kernig's sign was negative. Examination of the cranial nerves and upper limbs was entirely normal. Tone was increased bilaterally in the legs with marked pyramidal weakness in the right leg and moderate proximal weakness in the left leg. The knee and ankle jerks were absent and both plantar responses were extensor. Vibration sense was diminished in both legs and proprioception was mildly impaired in the toes bilaterally, but other sensory modalities were intact. On general examination she was obese and spine was tender locally at the L1/L2 level. The blood pressure was 150/70 mmHg, and there was no peripheral oedema. The abdomen was markedly distended with infrequent bowel sounds.

Investigations revealed normal routine haematology including clotting screen. A biochemical screen was normal apart from a plasma sodium of 106 mmol/l with a plasma osmolality of 230 mmol/kg and a urine osmolality of 583 mmol/kg. The plasma urea was 5.1 mmol/l and the potassium was 3.8 mmol/l. Two weeks previously, prior to starting a thiazide diuretic, the plasma sodium was 135 mmol/l. Plain X-rays of the chest, thoracic and lumbar spine were normal. Heavily blood stained lumbar cerebrospinal fluid (CSF) was obtained but analysis of the specimen was impossible. Cervical puncture revealed CSF containing $3.4 \times 10^9$ red blood cells/l, $0.07 \times 10^9$ white blood...
cells/l which were mainly polymorphonucleocytes with a few histiocytes. The protein was 570 mg/dl, the CSF sugar was 2.0 mmol/l (blood sugar 4.9 mmol/l) and no organisms were seen. Myelography was performed at this cervical level and a complete spinal block was demonstrated at the T10/T11 level with evidence of cord compression anteriorly; 9800 CT scanning of the spine was consistent with a spinal haematoma from T6 to T11. A CT cranial scan was entirely normal with no evidence of intracranial subarachnoid haemorrhage.

A decompressive laminectomy and exploration of T10 to L1 was performed. Evacuation of a large subarachnoid blood clot was performed over the segments T10 to L1 although it was noted that there was still extensive blood clot extending above the level of the exploration. No extradural blood was seen. The patient made a good functional recovery post-operatively, regaining control of bladder and bowel function within 72 h of operation, and regaining power in the legs over the next 2 weeks such that she could walk independently. Ten days post-operatively repeat myelography suggested extensive thoracic filling defects but without pathological vessels and CT scanning confirmed the persistence of a column of subarachnoid clot over the segments previously described. Her abnormal mental state resolved completely within a few days of operation in parallel with the correction of her plasma sodium and osmolality through water restriction and withdrawal of diuretics. At follow-up, 2 months post-operatively, there was only mild weakness proximally in the right leg and left leg power was completely normal. All of the sensory signs had resolved. In view of her excellent functional recovery and the patient’s reluctance to undergo further investigations, it was decided not to perform spinal angiography in an attempt to exclude definitively an arteriovenous malformation as the underlying lesion.

Discussion

The initial presentation of spinal subarachnoid haemorrhage with sudden severe back pain was described by Henson & Croft (1956) in several of their cases. In our case the initial and incorrect diagnosis was of a prolapsed intervertebral disc, which is a diagnostic difficulty also well described previously (Henson & Croft, 1956). In spinal subarachnoid haemorrhage absence of neurological signs in the early stages and urinary retention are typical (Jellinger 1972), and both occurred in our patient.

Our initial interpretation of the clinical signs was of a progressive and extensive cord lesion with involvement of lumbar and sacral segments, and possibly also of the cauda equina, but this was not completely confirmed at operation where the lower limit of haemorrhage and clot was found to be at L1. It is possible, however, that some compression of the lower end of the cord and/or spinal roots may have been produced from the extensive clot superiorly.

A common feature in the patients described by Henson & Croft (1956) was the prominence of symptoms and signs of meningism which occurred invariably. They found that meningism developed rapidly when spinal subarachnoid haemorrhage occurred in the cervical region, whereas, when it occurred at a low level, meningism appeared at an interval of 1 h to several days. In addition, in the majority of their cases the meningism was so severe that it gave rise to diagnostic confusion with primary intracranial subarachnoid haemorrhage. The remarkable feature of the present case was the complete absence of signs of meningism throughout her entire 14 d pre-operative period despite the presence of subarachnoid blood clot and red cells in the CSF, detected by cervical puncture.

Surgery was clearly effective in relieving the block as evidenced by the remarkable clinical improvement following her operation. However, there was very little difference in the myelographic and CT scan appearances on the 10th post-operative day compared with those seen on pre-operative studies. The presence of large amounts of subarachnoid blood was frequently observed by Henson & Croft (1956) who managed such cases conservatively. It would seem therefore that the presence of large amounts of subarachnoid blood is not an absolute indication for operative intervention per se but signs of progressive spinal cord compression makes surgical exploration imperative, as in the present case.

There was no evidence in our patient that the spinal subarachnoid haemorrhage was due to either a spinal tumour or an arterio-venous malformation, and the most likely cause was hypertension which has been previously reported to be a precipitating factor for this condition (Jellinger, 1972). Although mental slowing and confusion can be features of spinal subarachnoid haemorrhage when it has extended intracranially (Henson & Croft, 1956), we believe that our patient’s abnormal mental state was a consequence of the severe hyponatraemia. Moreover, there was no evidence whatsoever that intracranial extension of the haemorrhage had occurred in this case. Although hyponatraemia may occur in intracranial subarachnoid haemorrhage via many different mechanisms (Affi et al., 1965; Doczi et al., 1981; Joynt et al., 1965), diuretic therapy was probably the major cause of her hyponatremia. The rapid development of the hyponatraemia after diuretic therapy was started and the well-documented ability of diuretics to cause severe hyponatraemia via a number of different mechanisms (Kennedy et al., 1978) is consistent with this view.
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References


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