Cranial subdural haematoma with concomitant spinal epidural and spinal subarachnoid haematomas: a case report

Thomas Kishen, Greg Etherington, Ashish Diwan
Spine Service, Department of Orthopaedic Surgery, St George Clinical School, University of New South Wales, Sydney, Australia

ABSTRACT

A 76-year-old man presented with a 4-day history of bilateral leg pain. Magnetic resonance imaging (MRI) of the lumbosacral spine revealed a spinal subarachnoid and spinal epidural haematomas. MRI of the brain revealed a chronic intracranial subdural haematoma with a midline shift. On further questioning, the patient reported a history of a fall 6 weeks earlier and had no evidence of coagulopathy. He underwent a burr-hole decompression of the intracranial subdural haematoma. At the one-year follow-up, the patient was symptom free with no leg pain or headache. The concomitant occurrence of an intracranial subdural haematoma with spinal epidural and spinal subarachnoid haematomas is rare. MRI of the brain and the entire spine is essential in the presence of a spontaneous spinal haematoma.

Key words: hematoma, epidural, spinal; hematoma, subdural, chronic; subarachnoid hemorrhage

INTRODUCTION

Chronic intracranial subdural haematomas are more common in the elderly following trauma, particularly those on anticoagulants. Spinal epidural and spinal subarachnoid haemorrhage are uncommon and coagulopathy is a predisposing factor. The concomitant occurrence of a chronic intracranial subdural haematoma with spinal epidural and spinal subarachnoid haemorrhages is rare, especially in the absence of coagulopathy.

CASE REPORT

On 11 December 2006, a 76-year-old man presented with a 4-day history of pain extending from the buttocks to the knees, along the back of his thighs. The pain was aggravated by standing and walking and partially relieved by sitting. The patient had no back pain, paraesthesia and weakness in his lower limbs or bladder or bowel dysfunction. He was alert and well oriented and his spine was aligned normally in the coronal plane with mild loss of lumbar lordosis.
His lumbar spine movements in the sagittal plane were restricted by pain. The straight leg-raising test was 60° on lying down with no neurological deficits in the limbs. The cervical spine was normal. There was no evidence of cranial nerve palsy or lateralising neurological deficits.

Magnetic resonance imaging (MRI) of his lumbosacral spine showed an acute epidural haematoma opposite the L4-L5 disc (without marked neural compression), a fluid-fluid level in the distal sacral subarachnoid compartment (a subarachnoid haematoma), and a grade-1 anterolisthesis at L4-L5 with disc bulges at the L2-L3, L3-L4 and L5-S1 levels (Fig. 1). Four days after presentation, MRI of the brain and the rest of the spine showed a chronic left subdural haematoma measuring 1.6 cm at its widest transverse diameter causing gyral swelling, sulcal effacement, and a midline shift of 4 mm (Fig. 2). A small amount of fresh blood was noted within the haematoma. The remaining intracranial structures and cervicothoracic spine were normal. His clotting profile was normal.

The patient recalled that he had sustained an injury to his forehead following a fall 6 weeks earlier resulting in periorbital swelling, bruising and a headache that lasted one day. He had not undergone a lumbar puncture nor did he take any anticoagulants. In addition to half a tablet of aspirin a day and Lamotrigine (anti-epileptic), the patient was on medications for gastro-oesophageal reflux disease and hypercholesterolaemia.

Nine days after presentation, a burr hole was opened and the intracranial subdural haematoma was drained. At the one-year follow-up, the patient was free of leg pain and had no recurrence of his symptoms.

**DISCUSSION**

Head injuries are the leading cause of chronic intracranial subdural haematomas. Owing to the insidious nature of the venous bleed, the presentation can be delayed, especially in the elderly and those with bleeding diatheses. Electron microscopic studies have shown that the subdural space does not exist and that subdural collections occur when dural border cells are torn off from the arachnoid layer by trauma. The intracranial subdural blood can occasionally track down, assisted by gravity, into the spinal subdural space.

Spinal subarachnoid haemorrhages are rare and are usually secondary to coagulopathies, lumbar punctures, and trauma. The cerebrospinal fluid dilutes and washes away the blood preventing the formation of a clot. Rarely, copious bleeding and/or a diminished flow of cerebrospinal fluid can lead to a subarachnoid haematoma and neural compression. Spinal epidural haematomas may be spontaneous or secondary to lumbar punctures or epidural anaesthesia. Spontaneous epidural haematomas may be associated with coagulopathies, neoplasms or aneurysms, and are usually secondary to a venous bleed from the epidural venous plexus. The dorsal aspect of the thoracic and lumbar regions is most commonly involved, with expansion limited to a few vertebral levels.

The outer periosteal and inner meningeal layers of the cranial dura enclose the dural venous sinuses.
The periosteal layer ceases at the foramen magnum and the spinal dura represents only the meningeal layer of the cranial dura. The spinal arachnoid membrane is continuous with the cranial arachnoid membrane, contains the cerebrospinal fluid, and ends at the lower border of the second sacral vertebra. There is no anatomic communication between the various meningeal compartments.

Our patient had a haematoma in 3 separate non-communicating compartments of the neural-covering layers. The cranial subdural haematoma was probably caused by the head injury, but the cranial subdural compartment does not have anatomic continuity with the spinal subarachnoid compartment or the spinal epidural compartment. The spinal epidural and subarachnoid haematomas may have been caused by the same trauma, but simultaneous haematomas in different sites are rare in the absence of any coagulopathy, and this does not explain the delayed onset of leg pain. The presence of bilateral leg pain in our patient can be explained by the meningeal irritation caused by blood in the subarachnoid space. A combined subarachnoid, subdural, and epidural haematoma of the thoracolumbar spine was reported in a patient on dicoumarol therapy. 20% of spontaneous spinal subarachnoid haematomas are associated with a concomitant spinal subdural haematoma.

There have been reports of bleeding originating in the subarachnoid space dissecting into the spinal subdural space. In one case report, a patient with a ruptured left internal carotid artery aneurysm that projected posterolaterally and was located beneath the left petroclinoideal ligament presented with an intracranial subarachnoid and subdural haematoma and later with a spinal subdural haematoma. The extensive subarachnoid haematoma was speculated to have dissected into the intracranial subdural space and then migrated into the spinal subdural space. Another possible explanation was that the subdural haematoma ruptured into the subarachnoid compartment, but this did not explain the presence of the epidural haematoma.

The concomitant occurrence of an intracranial subdural haematoma with spinal epidural and spinal subarachnoid haematomas is rare. MRI of the brain and the entire spine is essential in the presence of a spontaneous spinal haematoma.

ACKNOWLEDGEMENT
We thank Helen Houridis for proofreading the manuscript.

REFERENCES