ABSTRACT

We report 2 cases of spontaneous spinal epidural haematoma after antiplatelet treatment. One patient was on ticlodipine therapy and the other was on aspirin and clopidogrel. Both diagnoses were made using magnetic resonance imaging. One patient underwent multilevel laminectomies and posterior spinal fusion within 24 hours of symptom onset and achieved complete neurological recovery. The other underwent unilateral hemilaminectomies 40 hours after symptom onset and did not show neurological improvement. A high index of suspicion is needed for patients presenting with severe lower back pain and rapid neurological deterioration, especially in those on antiplatelet therapy. Urgent spinal cord decompression and epidural haematoma cord evacuation may provide more favourable outcome.

Key words: aspirin; hematoma, epidural, spinal; platelet aggregation inhibitors; spinal cord compression; ticlopidine

INTRODUCTION

Aspirin and thienopyridines (such as ticlopidine and clopidogrel) are commonly used antiplatelet agents to prevent recurrent strokes and other vascular events. Aspirin permanently acetylates cyclooxygenase 1 (COX-1) and prevents the conversion of arachidonic acid to thromboxane A2. This decreases platelet aggregation at the site of the vascular injury.1,2 Thienopyridines inhibit platelet function by binding irreversibly to the P2Y12-receptor.1,2 Haemorrhagic complications of antiplatelet therapy commonly occur in the gastrointestinal tract, skin, intracranially or at sites related to surgical procedures. In the CHARISMA (Clopidogrel for High Atherothrombotic Risk and Ischemic Stabilization, Management, and Avoidance) trial, 3.1% of 15 603 patients had bleeding complications over a median period of 28 months; 1.5% of whom entailed severe bleeding,1 which includes fatal bleeding, intracranial haemorrhage, or bleeding causing haemodynamic compromise requiring blood or fluid replacement, inotropes, or surgery.1 In a meta-analysis, compared to placebo, aspirin increases the relative risk of major gastrointestinal bleeding 2 fold,
and intracranial bleeding 1.6 fold. Low-dose aspirin increases the risk of major bleeding by approximately 70%. Dual antiplatelet therapy may further increase the risk of bleeding. We report 2 cases of spontaneous spinal epidural haematoma (SSEH) after antiplatelet treatment.

CASE REPORTS

Case 1

In April 2010, a 60-year-old man presented to the accident and emergency department 7 hours after an acute onset of increasing back pain, bilateral anterior thigh numbness, left lower limb weakness, and urinary retention. He had no history of back pain or trauma and was initially treated for back sprain elsewhere. For atrial fibrillation, he had been taking aspirin for 2 years and clopidogrel for 6 months.

According to the Medical Research Council classification, the patient had grade-4 motor power in his left extensor hallucis longus, left flexor hallucis longus, and left ankle dorsiflexion. Sensation in the L3 dermatome was reduced bilaterally. Perianal sensation was intact but voluntary contraction of the anal sphincter appeared diminished. Abdominal examination revealed a palpable bladder, and 1 litre of clear urine was drained upon catheterisation.

Results were normal for platelet count (183 x 10^9/l), prothrombin time (13.8 s), partial thromboplastin time (27.6 s), and bleeding time (5.8 min). 12 hours after the onset of symptoms, an urgent magnetic resonance imaging (MRI) of the spine revealed a posterior epidural haematoma extending from T10 to L2 levels and compressing the conus medullaris (Fig. 1). The patient also recalled that in the past month, razor cuts while shaving took longer than usual to stop bleeding.

22 hours after presentation, emergency thoracolumbar decompression was performed. A large epidural haematoma was found. It extended from T10 to L2, with maximal spinal cord compression at T11 and T12. The patient underwent lower T10 to upper L2 laminectomies with spinal instrumentation and fusion (Fig. 1). A drain was left in situ to drain the epidural haematoma.

On postoperative day 1, the patient had a complete neurological recovery with full sensorimotor function in the lower limbs. There was no urinary dysfunction when the urinary catheter was removed on day 3. The spinal drain was removed on day 3. The volume of spinal drainage was 430, 270, 180, and 120 ml on the operation day and postoperative days 1, 2, and 3, respectively. The patient was ambulant on day 4. A repeat MRI showed complete haematoma evacuation (Fig. 2). Histology confirmed that the haematoma had no findings of vascular malformation, tumour, or infection. At the 9-month follow-up, the patient remained well and had no neurological deficit.

Case 2

In April 2010, a 70-year-old woman presented to the accident and emergency department 7 hours after an acute onset of increasing back pain, bilateral anterior thigh numbness, left lower limb weakness, and urinary retention. She had no history of back pain or trauma and was initially treated for low back pain elsewhere. For atrial fibrillation, she had been taking aspirin for 2 years and clopidogrel for 6 months.

According to the Medical Research Council classification, the patient had grade-4 motor power in her left extensor hallucis longus, left flexor hallucis longus, and left ankle dorsiflexion. Sensation in the L3 dermatome was reduced bilaterally. Perianal sensation was intact but voluntary contraction of the anal sphincter appeared diminished. Abdominal examination revealed a palpable bladder, and 1 litre of clear urine was drained upon catheterisation.

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the accident and emergency department 12 hours
after onset of severe lower back pain, bilateral lower
limb paraesthesia, paralysis, and subsequent urinary
retention. She had been taking ticlopidine for 3 years
following a cerebral vascular accident.

Clinical examination revealed grade 0 motor
power in both lower limbs with a sensory level at
L1. Anal tone was lost, and 850 ml of clear urine was
drained upon catheterisation. Her platelet count
was raised to 492 x10^9/l (reference range, 132–372
x10^9/l), but the coagulation profile (prothrombin
time, 12.8 s; partial thromboplastin time, 24.3 s) was
within normal limits. The MRI of the spine revealed
an epidural haematoma in the thoracolumbar region
extending from T10 to L1 and compressing the cauda
equine (Fig. 3).

40 hours after presentation, posterior spinal
decompression entailing T10 to L1 unilateral
hemilaminectomies was performed. Postoperative
drainage yielded 120 ml on the operation day and
decreased to 0 ml on postoperative day 1. The drain
was removed on day 2.

No neurological improvement ensued in the
peri-operative period. At the 9-month follow-up, the
patient remained catheter dependent and wheelchair
bound with grade 0 motor power in both the lower
limbs.

**DISCUSSION**

SSEH is an emergent condition. Patients may present
with severe back pain followed by rapidly evolving
nerve root symptoms with or without spinal cord
compression. Bilateral sensorimotor deficits are
common. Unilateral neurological deficits and Brown-
Sequard syndromes have also been reported.4,5
SSEH is the presence of blood in the epidural space
in the absence of any underlying haematological
disorder, vascular malformation, direct trauma or
iatrogenically caused by an invasive procedure.6–9
SSEH has been extended to include cases associated
with physical exertion and indirect minor trauma.8

SSEH is more common in older adults and in
males, but also occurs in children.9 Its incidence was
0.1 per 100 000 persons per year, and it accounts
for <1% of space-occupying lesions in the spinal
epidural space.10,11 Epidural haemorrhage is thought
to be caused by rupture of spinal epidural veins in
the venous plexus surrounding the spinal dura.12
This venous system is valveless and permits back
flow of blood with raised intra-abdominal and/or
intrathoracic pressure during straining. The resultant
raised pressure may predispose to epidural vein
rupture. Other causes include vascular malformations
(such as haemangiomas, cryptic, or spinal angiomas)9
and arterial bleeding secondary to the rapidly
evolving clinical symptoms.12 Commonly, SSEH is
found dorsal to the spinal cord between the 2 layers
of spinal dura where they are loosely opposed. Both
the ventral dura layers are often tightly apposed and
thus afford little space for haematoma formation.13

**Figure 2** Patient 1: axial T2-weighted images show
a posterior epidural space lesion resulting in marked
central canal narrowing worst at (a) T12 and (b) L1 with
severe compression of the conus medullaris. The lesion
is hyperintense to the cord and muscle but hypointense to
cerebrospinal fluid. Postoperative images at the same levels
(c) T12 and (d) L1 show a decompressed spinal cord.

**Figure 3** Patient 2: (a) sagittal T2-weighted images show an
epidural fluid collection posterior to the spinal cord from T10
to L1. Relative to the spinal cord, the lesion exhibits mildly
hyperintense signal characteristics. Axial T2-weighted images
show a right posterior lateral epidural space lesion resulting
in severe cord compression at (b) T12 and (c) L1.
Coagulopathies and anticoagulant therapies,14,15 hypertension,16,17 coronary thrombolysis,18 ankylosing spondylitis,19 insignificant efforts in straining, increased venous pressure, chiropractic manipulation,20 and pregnancy21 have been reported to be risk factors of atraumatic SSEH.

Aspirin and clopidogrel (but not ticlopidine) have been reported as one of the risk factors.6,22–27 Diagnosing SSEHs is challenging, as patients initially present with intense localised pain only. This could be due to spinal musculoskeletal strains and degenerative disc disease. With the onset of neurological symptoms (radiculopathy and myelopathy), exclusion of disc herniations, epidural abscesses, and tumours is necessary. Once urinary or anal sphincter dysfunction occurs, an urgent MRI becomes necessary to arrive at a diagnosis. With the advent of the MRI, the number of new SSEH cases has increased from 2.2 to 6.4 per year.28 The gold standard for diagnosing SSEHs and spinal cord compression is MRI. The use of gadolinium contrast helps differentiate enhancing spinal tumours from abscesses and haematomas. Epidural haematoma signals are often homogenous and isointense to the spinal cord within the first day of symptom onset, and the haematoma turns hyperintense after 36 hours on T1-weighted images.27 In T2-weighted images, the haematoma appears isointense or hyperintense with respect to the spinal cord and may be homogenous or heterogenous. In our patients, T2-weighted images were hyperintense, whereas T1-weighted images were isointense to hypointense.

Delayed diagnosis and treatment lead to poor outcomes. A high index of suspicion is needed for patients presenting with an acute onset of severe back pain with or without a neurological deficit, particularly in those on antiplatelet therapy. In a patient with atraumatic SSEH secondary to clopidogrel therapy for atrial fibrillation,6 severe upper back pain and acute urinary retention were present but not sensorimotor deficit. The haematoma extending from T1 to T12 was surgically drained via a posterior laminectomy 78 hours after symptom onset. Partial sphincter function was regained at the 6-month follow-up. In another patient on clopidogrel for recurrent transient ischaemic attacks presenting with acute paraplegia,26 there was an epidural haematoma extending from T6 to T9. It was drained via a posterior laminectomy 9 hours after symptom onset. The patient had made a full neurological recovery at the 3-month follow-up. In a patient on dual antiplatelet therapy of aspirin and clopidogrel presenting with spontaneous severe back pain and left sciatica with foot drop,27 a left L1 to L3 hemi-laminectomy was performed for a haematoma extending from L1 to L4. The time from symptom onset to surgical decompression was 9 hours, and the patient made a complete neurological recovery 7 days after surgery. In a geriatric patient on aspirin presenting with back pain, urinary incontinence, and bilateral lower limb paresis,31 a T7 to L5 decompressive laminectomy was performed within 24 hours of presentation, and the patient was able to mobilise independently at 4 months. Although spontaneous recovery with conservative management has been reported,29,30 this strategy is usually reserved for patients unfit for surgery. For optimal recovery of neurological deficit, prompt surgical decompression is almost always necessary to achieve complete evacuation of the haematoma.

Preoperative neurological status was also a factor determining neurological outcome. In a meta-analysis of 613 patients with spinal haematomas caused by multiple aetiologies,7 patients operated within the first 12 hours had the best prognosis of neurological recovery. The worst outcome was in patients with complete sensorimotor deficits and associated urinary dysfunction. In a review of 330 patients with spinal haematomas,31 recovery was significantly better when decompression was performed within 36 hours in the presence of complete sensory motor loss and within 48 hours in the presence of incomplete sensory motor loss.

We recommend laminectomy over hemilaminectomy to aid evacuation of the epidural haematoma. Complete laminectomy avoids potential incomplete haematoma evacuation and allows adequate decompression in case of haematoma recurrence. This may have occurred in patient 1, in whom there was a persistent and large amount of posterior spine drainage postoperatively. Posterior instrumentation and fusion may provide added stability to the multilevel laminectomised spine and reduce micro-motion and trauma to the injured spinal cord.

REFERENCES


