Retroperitoneal haemorrhage as a differential diagnosis of spinal haematoma post spinal anaesthesia in a patient on prophylactic anticoagulant

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Abstract
Among the haemorrhagic complications of warfarin therapy presenting with neurological symptoms, spinal epidural haematoma and retroperitoneal bleeding into the psoas and iliac muscles are two of the important diagnoses to consider. Spinal epidural haematoma (traumatic or spontaneous) is an uncommon, but recognised, clinical entity that needs emergency management. The association of spinal epidural hematomas with warfarin therapy has been described and, in 1956, Alderman stated that this diagnosis should be entertained in any patient receiving anticoagulants presenting with lower back pain or sciatic pain. Retroperitoneal bleeds on the other hand can be particularly difficult to diagnose and manage. Both are serious conditions, especially if there is a delay in diagnosis, as early treatment confers a marked prognostic advantage. Hence awareness, a high index of suspicion and a willingness to seek the prompt help of the imaging department, are crucial to successful management before the opportunity to treat is lost.

A case report follows, the purpose of which is to increase the awareness among medical personnel and to stress the urgency of management.

A 75 year old woman with a history of prosthetic mitral valve replacement, atrial fibrillation & TIA on warfarin was scheduled for TURBT to be done under spinal anaesthetic. Warfarin was stopped one day prior to admission and heparin infusion commenced on admission, with target APTT 2.5 times the normal. Heparin was stopped 4 hours prior to the spinal anaesthetic, which was difficult due to ankylosing spondylitis and needed four attempts. After an atraumatic tap and good sensory motor block, surgery was commenced without incident. Post-operatively, the patient developed a lower respiratory tract infection for which co-amoxyclav was commenced. On the fourth day post-op, the patient developed sudden onset, right leg weakness and paraesthesia, with right lower limb power 3/5, decreased tone and absent reflexes, leading to the diagnosis of a spinal haematoma post spinal anaesthesia. On further examination, she was also noted to be anaemic with a drop in haemoglobin to 6g/dl, with an INR of 3.4 and an acute renal impairment with a serum creatinine of 120. In addition, bruising in the right flank, abdominal pain and a right iliac fossa mass were also noted. An urgent MRI was booked, but as the patient was haemodynamically unstable, a CT scan was deemed more appropriate, which showed a retroperitoneal bleed into the right ilio-psoas. This was confirmed with a spinal MRI done subsequently, which also ruled out any spinal haematoma. The patient was treated conservatively with 5units PCV and 3units FFP. Her clotting profile gradually normalised as did her renal function and her right sensory-motor deficit continues to improve.

Discussion:
Retroperitoneal bleed: The predilection for bleeding into the retroperitoneal space has not been fully explained but a unique weakness of the vascular and connective tissue has been suggested. It is also most commonly seen in association with patients on anticoagulation therapy or haemodialysis, or with bleeding abnormalities, and may represent one of the most serious and potentially lethal complications of anticoagulation therapy. The incidence of retroperitoneal haematoma has been reported at 0.6-6.6% of patients undergoing therapeutic anticoagulation. Warfarin, unfractionated and low-molecular weight heparin have all been implicated. However, it is nonetheless important to note that the therapeutic index of warfarin is narrow and anticoagulant control is easily deranged by drugs (such as antibiotics) and co-morbid factors such as renal or hepatic dysfunction. Frequent INR measurement is the best way to avoid haemorrhagic complications.

Patients report lower abdominal or hip pain radiating to the
groin or anterior thigh. Bleeding into the psoas muscle causes spasm and hip flexion and, as it extends, flank or thigh bruising may appear. Femoral nerve compression reduces quadriceps power and causes loss of knee jerk and paraesthesia in the area of cutaneous supply.

CT scan is the investigation of choice but ultrasound is also sensitive and is more rapidly available. Delay in diagnosis is potentially fatal because severe haemorrhage can supervene. Locally the haematoma may cause ureretic obstruction and acute renal failure, or femoral nerve compression. Treatment options are surgery and conservative management consisting of treating the anaemia associated with the bleed and correcting the coagulopathy. Options to treat the coagulopathy would mainly depend on how quickly correction is required, to what range and how long normal clotting indices would be safe in a patient on therapeutic or treatment anticoagulation. Fresh frozen plasma (FFP at a dose 15ml/kg) is given for rapid but short-lived correction with the usual risks of transfusion of blood products. Vitamin K (2.5mg) is given for a slower but more prolonged correction (leaving patients with artificial valves at risk of thromboembolic events and valve failure). Over-anticoagulation due to warfarin can be reversed completely and immediately by infusion of a complex concentrate of factors 2, 7, 9 and 10.

**Spinal haematoma:** The true incidence of spinal haematoma is unknown and due to its rarity it is very difficult to evaluate risk factors prospectively and any properly powered study would require many thousands of patients to investigate this. Therefore, data on the incidence of spinal haematoma following neuraxial blockade are mainly based on audit studies and case reports. Tryba reported that the incidence of spinal haematoma after epidural and spinal anaesthesia is 1 in 150,000 and in 1 in 220,000, respectively. The insertion and removal of an epidural catheter appeared to be of far greater importance in the genesis of a spinal haematoma. The incidence of spontaneous spinal haematomas rarer still and is estimated at 1 patient per 1,000,000 patients per year. Central neuraxial blockade has a low incidence of major complications, many of which resolve within 6 months.

The symptoms of an acute spinal hematoma include a sharp irradiating back pain of radicular character, and sensory and motor deficits which outlast the expected duration of the anaesthetic. Not all of these symptoms have to be present at the same time. The clinical suspicion can only be confirmed by means of an emergency CT-scan (with myelography) or magnetic resonance imaging.

The only treatment of a compressing spinal hematoma is an emergency decompressive laminectomy with evacuation of the hematoma. Final neurologic outcome depends on the speed with which the hematoma develops; the severity of the preoperative neurologic deficit; the size of the hematoma; and most importantly, the time span between hematoma formation and surgical decompression. Complete recovery of neurologic function is possible if surgery is performed within 8 hours of the onset of the paraplegia.

**Conclusion**

The aim of this report is in no way to undermine the importance of Alderman’s advice to suspect the spine as an area of bleeding in patients on anticoagulant therapy. The above case is a reminder to consider retroperitoneal bleeding as one of the differential diagnoses of spinal haematoma in an anticoagulated patient who develops sudden onset spinal pain, with or without neurological deficit post spinal anaesthetic. The presenting symptoms are similar and early management is equally important in terms of associated morbidity when management is delayed.

**COMPETING INTERESTS**

None Declared

**AUTHOR DETAILS**

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