Case Report

Spinal subarachnoid haemorrhage complicating oral anticoagulation therapy

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Introduction

We report a rare case of spinal subarachnoid haemorrhage (SSH) complicating oral anticoagulation therapy in an elderly patient with minimally elevated PT/INR. MRI of spine plays a major role in the diagnosis of SSH. SSH with minimal neurological symptoms, without rapid deterioration, can be managed medically without surgical intervention.

Case report

A 76-year-old man was admitted to hospital with severe lower backache of one-week duration and difficulty in walking for three days. He did not have sphincter disturbances, headache or mental state changes. His back pain was severe and radiated along both lower limbs. He had a history of hypertension, chronic kidney disease and peripheral neuropathy and had undergone a mitral valve replacement four months previously. He was on warfarin following the mitral valve replacement. Examination revealed normal bilateral hip joint power with impaired bilateral knee (4/5 power) and ankle power (4/5 power). Bilateral patellar reflexes were intact with absent Achilles tendon reflexes. Pinprick sensation was impaired bilaterally up to mid-calf level with intact sacral sensation. Anal tone was normal. There was no neck stiffness or papilloedema.

Laboratory investigations revealed a prothrombin time (PT) of 43.2 seconds with an international normalization ration (INR) of 3.63. Full blood count showed a white cell count of 4.3 x 10^3/µL, hemoglobin of 11.3 g/dl and platelet count of 240 x 10^3/µL. Renal and liver function tests were within normal limits. Magnetic resonance imaging (MRI) of the lumbosacral spine showed a T1 and T2 weighted high signal lesion in the subarachnoid space extending from the L3 vertebral to the upper border of the S2 vertebra. There was a small area of T2 weighted hypointense and T1 weighted isointense in the upper end of the lesion. There were no features suggestive of vascular malformation. MRI imaging findings were highly suggestive of subacute subarachnoid hematoma (SSH). It was presumed that SSH developed as a result of anticoagulation therapy as PT/INR was elevated in our patient. The patient was managed medically...
without proceeding to surgery as he had minimal neurological disability without further deterioration. He was able to walk without any disability at one month of follow up.

Figure 1A: T1-weighted sagittal magnetic resonance imaging (MRI) reveals a hyper-intense lesion extending from L3 vertebra (upper white arrow) to upper border of S2 vertebra (lower white arrow)

Figure 1B: T2-weighted sagittal MRI reveals a hyper-intense lesion extending from L3 vertebra (upper white arrow) to upper border of S2 vertebra (lower white arrow), suggestive of a subacute haematoma. T2 weighted hypointense and T1 weighted small isointense area in the upper end of the haematoma is suggestive of acute stage blood (red arrow).

Figure 1C: T2-weighted axial MRI reveals peripherally displaced sacral nerve roots due a subarachnoid haematoma (blue arrows).

Discussion

SSH is defined as a blood collection forming a clot in the subarachnoid space with an intact arachnoid membrane [1]. SSH may compress and damage the spinal cord and cauda equina or nerve roots. SSH is rare and accounts for 0.05-1.5% of cases of subarachnoid haemorrhage [2,3]. SSH is commonly caused by tumours, arteriovenous malformations (AVM) and aneurysms [4,5]. Other causes associated with SSH are bleeding disorders, anticoagulation therapy, vasculitis (systemic lupus erythematosus, Behçet's disease, polyarteritis nodosa), coarctation of the aorta and hypertension [5,6,7]. Anticoagulation associated spinal cord bleeding is more common among the elderly population [8]. The most common location for the intraspinal haemorrhage is the dorsal epidural space and haemorrhages in the subdural and subarachnoid spaces are rare [5,8].

Anticoagulation associated spinal haemorrhage is also commonly located in the dorsal epidural space than the subarachnoid space. Anticoagulation associated spinal haemorrhages can occur without trauma and even when the prothrombin time (PT), clotting time or activated partial thromboplastin time (aPPT) are within therapeutic
ranges [9]. Anticoagulation related SSH is commonly reported in association with lumbar puncture [1,5,9].

The clinical presentation of SSH is characterized by a combination of back pain, meningism and progressive compression of the spinal cord or cauda equina. Sudden onset severe back pain is the most common presentation [10,11]. Radicular pain, meningism, headache and altered mental state are other symptoms reported in SSH [8,10]. Neurological deficits that can occur secondary to SSH include paraparesis, paraplegia, sphincter disturbances and sensory loss [10,12]. CT, MRI and CSF analysis are the main investigations used in suspected SSH. MRI is useful to define the level of SSH and to rule out trauma and tumours. MRI can be used to monitor the progression of SSH as the MR signal intensity characteristics of haemorrhage are time-dependent. Acute SSH appears hyperintense or isointense on T1 and hyperintense or hypointense on T2 MRI. Subacute SSH becomes hyperintense or isointense on T1 and hyperintense on T2 weighted MRI [13]. These signal changes are secondary to the effect of strongly paramagnetic methaemoglobin [13].

Lumbar puncture may produce blood stained CSF or a “dry tap” [5]. Spinal angiogram may help in detecting spinal vascular malformation. MRI may also provide initial radiological signs of AVM such as serpentine signal void vessels and intramedullary nidus [14,15]. MRI of our patient did not show any evidence of spinal AVM and we did not proceed with a spinal angiogram.

The treatment for SSH depends on presence or absence of neurological deficits [16]. Surgical laminectomy to remove the haemorrhage and haematoma is only indicated in the presence of a mass effect with spinal cord compression, severe neurological dysfunction or deteriorating neurological condition [16,17]. Conservative medical management is preferred in the absence of significant neurological disability [18]. Prognosis following surgical intervention depends on the preoperative neurological status, duration between onset of symptom and surgical intervention and rapidity of deterioration of neurological symptoms [19]. When surgery is indicated, early surgical intervention is advisable.

**Conclusion**

We report a case of SSH in a 76-year-old patient with minimally elevated PT/INR in the setting of oral anticoagulation. Oral anticoagulation associated SSH can occur with a minimally elevated PT/INR. MRI of the spine plays a major role in the diagnosis of SSH. SSH with minimal neurological symptoms without rapid deterioration can be managed medically without surgical intervention.

**References**


