

Case Report

Spontaneous thoracic subdural hematoma associated with warfarin therapy: Case report with serial MRI

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Abstract

Background: Spontaneous acute spinal subdural hematoma (SASSDH) is a rare but serious condition. We present diagnostic challenges and serial magnetic resonance imaging (MRI) findings of a patient who developed warfarin-associated thoracic SASSDH that was managed surgically.

Case Description: A 68-year-old male presented with sudden onset left-sided chest and back pain, left leg weakness, and bilateral loss of sensations below T4 level. His symptoms developed after strenuous physical activity. He was taking warfarin for atrial fibrillation. His admission international normalized ratio was 4.25. Deterioration of neurological status 3 days after admission prompted spinal computed tomography (CT) scan that demonstrated nonhomogenous hyperdense intradural mass lesion in the thoracic spine. MRI demonstrated heterogeneous mass lesion on the left side of the spinal canal and thoracic myelopathy. The patient underwent urgent surgical evacuation of subacute subdural hematoma extending from T3 to T6 levels. MRI scan following the surgery showed no signs of the hematoma and thoracic myelopathy. MRI at 3 months follow-up demonstrated myelopathy extending from T3 to T6 levels with deviation of the spinal cord. The patient's motor strength and sensations improved but he retained left leg weakness with sensory deficit below T8 level.

Conclusions: Spinal subdural hematoma should be suspected in patients presenting with acute onset back pain and myelopathy in the absence of trauma history. Coagulopathy should raise the suspicion for SASSDH. MRI is a valuable imaging modality for initial diagnosis to rule-out other lesions, and to assess postoperative re-bleeding and residual lesions.

Key Words: Magnetic resonance imaging, spinal subdural hematoma, surgery, warfarin

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INTRODUCTION

Spontaneous acute spinal subdural hematoma (SASSDH) is a rare disorder that is often associated with high risk for poor outcomes and disability.^[9] Diagnosis of SSDH can be challenging, resulting in delayed definitive treatment interventions. We present diagnostic challenges with serial magnetic resonance imaging (MRI) of large warfarin-associated thoracic SASSDH that was managed surgically.

We present serial MRI findings of a patient who was diagnosed with large thoracic SASSDH and managed surgically.

CASE PRESENTATION

A 68-year-old male presented to an outside hospital with sudden onset left-sided chest and back pain accompanied by left leg numbness and weakness. His symptoms started suddenly after strenuous physical activity. The patient was taking warfarin by alternating daily doses (1.5 mg and 5 mg) for atrial fibrillation. On admission, he had left leg plegia (motor power 0/5) and reduced sensation to touch from T4 level bilaterally. Coagulation panel showed elevated international normalized ratio (INR) of 4.25 (normal range: 0.9–1.2), prolonged activated partial thromboplastin time of 40.9 (normal range: 28–38), and reduced prothrombin time (Stago Prothrombin Assay) activity 11% (normal range: 70–130%). The patient was admitted for diagnostic work-up and was continued on warfarin. Noncontrast head computed tomography (CT) was within normal limits. Three days after admission, the patient's neurological function deteriorated and he developed right leg paresis and bladder retention. These symptoms prompted noncontrast spinal CT that demonstrated nonhomogenous hyperdense intradural mass lesion extending from T2 to T6 levels [Figure 1a and b]. The patient was transferred to the neurosurgery department for further management.

Upon admission to our department the patient had grade C myelitis based on the American Spinal Injury Association (ASIA) Classification from T4 level. He had left leg plegia (power 0/5) and right leg paresis (power 0–1/5 hip/knee flexors/extensors and 2/5 plantar flexors/extensors). Based on the Autonomic Standards Assessment Form, the patients' general autonomic function assessment was normal, and he had loss of urinary, anal sphincter, and erectile functions. Spinal MRI showed heterogeneous mass lesion extending from T3 to T6 levels on the left side of the spinal canal. The lesion was hyperintense on T2-weighted sequences and heterogeneous intensity on T1-weighted sequences, which was suggestive of acute-to-subacute subdural blood clot, with displacement of the spinal cord by the hematoma and signs of myelopathy [Figure 1c-e]. The

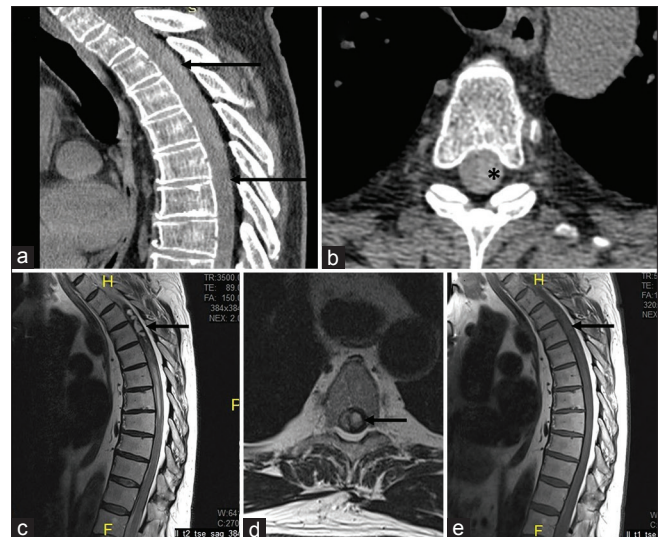


Figure 1: Preoperative noncontrast spine CT (a and b) scan showing nonhomogenous hyperdense lesion extending from T2 to T6 levels (a; solid arrows) on the left side of the spinal canal (b; asterisk), and MRI (c-e) showing hyperintense lesion (solid arrow) on T2-weighted sagittal (a) and axial (b) images and hypointense lesion on T1-weighted (c) images from extending T3 to T6 levels

patient was administered 2000 International Units of human prothrombin complex concentrate (PCC) for coagulopathy and underwent emergent surgery that was performed 3 days after his initial symptoms and within 24 hours of neurological deterioration. Left side hemilaminectomy extending from T3 to T6 levels was performed. The dural sac was tense and nonpulsatile with bluish discoloration. The dura was opened longitudinally. The clot was 5–6 mm thick and was located in the left posterolateral portion of the dural sac, partially organized, gently adhesive to the spinal cord, and encasing thoracic nerve roots. The clot was gently dissected from the spinal cord and nerve rootlets using dissector, suction, and bipolar cautery. Clot remnants were rinsed from the subdural space with isotonic saline. Pulsation of the spinal cord was evident after the clot was removed. Bleeding source was not identified. After complete hemostasis, the dura was closed in water-tight fashion. Drainage was left in the epidural space and the wound was approximated in layers.

Postoperative period was uneventful. Warfarin was replaced with fraxiparin and reinstated on postoperative day 7. MRI at day 1 after the surgery demonstrated completely removed hematoma. There was T2-weighted hyperintense signal in the central portion of the spinal cord suggestive of myelopathy and edema extending from T1 to T8 levels, as well as nonhomogenous hypointense areas on T2-weighted sequences extending from T1 to T6 levels [Figure 2a-c]. Epidural drainage was removed on day 2. At discharge to the inpatient rehabilitation unit 2 weeks after the surgery, the patient reported improved sensations and right leg strength (power

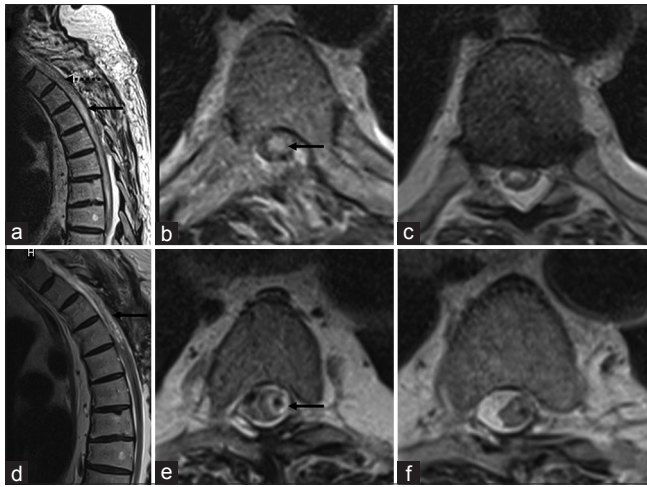


Figure 2: MRI the next days after the surgery (a-c) showing completely removed hematoma with hyperintense signal (a and b; solid arrow) and nonhomogenous hypointense (a; dashed arrows) suggestive of myelopathy. T2-weighted MRI at 3 months after the surgery (d-f) showing myelopathy extending from T3 to T6 levels (d) on the left side (e) of the spinal cord (solid arrow) with CSF collection on the right side of the dural sac causing medullary dislocation to the left (f)

3/5 hip/knee flexors/extensors and 1/5 plantar flexors/extensors) with remaining left leg plegia and altered urinary and bowel functions.

Three months after the surgery, leg motor strength (power: left leg 2/5 and right leg 5/5 plantar flexors/extensors) and bladder function were improved. The patient was able to stand with support and was ambulatory with wheelchair. He had reduced sensation to touch below the T8 level. MRI was suggestive of myelopathy extending from T3 to T6 levels with deviation of the spinal cord to the left [Figure 2d-f].

DISCUSSION

The most common clinical presentation of SASSDH is acute onset back pain radiating to the arms or legs with or without neurological deficits. Our patient had typical presentation and was admitted for diagnostic workup; however, spinal CT was performed when the patient's neurological status deteriorated 3 days after admission. Diagnosis of spinal cord compression in the absence of trauma history can be challenging. An audit from the United Kingdom of patients admitted with spinal cord compression to the neurosurgery unit showed that for 62% of patients it took more than a week to reach the referring hospital after initial consultation and nearly half of the patients waited a week or more before referral to neurosurgeon.^[8] During the referral process, neurological status deteriorated in half of the patients. Therefore, spinal hematoma should be included in the differential diagnosis of patients presenting with acute onset back pain and symptoms of myelopathy even in the absence

of trauma history. Coagulation deficits or therapeutic anticoagulation should raise suspicion for SASSDH.

Hyperdense mass lesion was noted on noncontrast spinal CT, and subsequent MRI demonstrated partially organized spinal subdural hematoma causing spinal cord compression with myelopathy. Postoperative MRI showed complete removal of the hematoma and myelopathy. Follow-up MRI at 3 months demonstrated residual intramedullary lesions. MRI is a reliable imaging modality for initial diagnosis of spinal subdural hematoma to rule-out postoperative bleeding and to identify residual changes. Kulkarni *et al.* presented serial MRI findings of spontaneously resolving spinal subdural hematoma in a 61-year-old female that developed after a lumbar puncture.^[6] MRI scans at 4, 7, 13, and 25 days after the injury documented similar progression of the spinal hematoma as seen in intracranial hematomas.^[6] MRI is the most reliable imaging modality for the lesion of peridural space and neural structures, and can demonstrate underlying bleeding sources and associated medullary lesions.^[10] However, MRI is less widely available than CT, which is commonly employed for initial diagnostic work-up. Langmayr *et al.* showed that CT was inconclusive in 4 out of 5 spontaneous extramedullary spinal hematoma cases due to isointensity of the hematoma to the spinal cord and insufficient image resolution of a CT scan in the thoracic region.^[7] In the present case, however, noncontrast spine CT scan demonstrated hyperdense lesion suggestive of blood clot and prompted subsequent MRI. Therefore, noncontrast CT scan can be a valuable diagnostic modality for thoracic hematoma in the acute phase and can be used to rule out other potential causes of myelopathy such as disc herniation, degenerative conditions, and metastatic destruction of vertebral bodies. CT with intrathecal contrast injection can also be considered for assessment of spinal stenosis if MRI is contraindicated.

MRI at 3 months after the surgery demonstrated myelopathy with the spinal cord deviation to the left side with cerebrospinal fluid (CSF) collection on the right side of the spinal canal, suggestive of subdural extra-arachnoid CSF collection. Two cases of postoperative lumbar subdural extra-arachnoid hygroma attributed to inadvertent durotomy during lumbar decompression surgery and microdiscectomy and causing cauda equinae syndrome have been previously described.^[11] In both cases, there was symptom resolution after surgical decompression of the cyst through wide opening of the arachnoid membrane. In our patient, motor and sensory functions were improved, therefore, surgical exploration of the observed CSF collection was not attempted.

Three months after the surgery the patient reported improved sensations; however, he still had severe left leg paresis and intermittent bowel and bladder

incontinence. A recent review by Perreira *et al.* reported that 70 out of 150 SASSDH patients had full recovery or mild deficits.^[9] In regression analysis, established deficit and coagulopathy were significant predictors of poor outcome. Our patient had both established poor prognostic indicators. Furthermore, surgical removal of the hematoma was delayed and performed 3 days after an index event. Nevertheless, substantial neurological recovery of our patient dictates that delayed surgical evacuation of the hematoma causing incomplete spinal cord lesion can improve neurological functioning.

There are a handful of reports linking warfarin use to SASSDH.^[1,2,5] Patients taking warfarin require close monitoring of the clotting system functioning to maintain therapeutic concentration and prevent overdose. Warfarin is associated with a three-fold increased risk for intracranial subdural hematoma^[4] as well as worse outcomes of patients with intracranial hemorrhage.^[3] Warfarin overdose was reversed with PCC that was previously documented as safe and effective in patients with intracranial bleeding.^[3]

CONCLUSIONS

SSDH should be suspected in patients presenting with acute onset back pain accompanied by myelopathy. Coagulopathy should raise suspicion for SASSDH. CT can be used to rule out other organic causes of myelopathy and to suspect spinal subdural hematoma. MRI is a valuable diagnostic modality for initial diagnosis, assessment for immediate postoperative re-bleeding, and follow-up. Surgical removal of the hematoma causing spinal cord compression is associated with improvement of neurological functioning and should be attempted.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported

in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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