

Spontaneous Spinal Epidural Hematoma Causing Paraparesis in a Patient of Mitral Valve Replacement with Anticoagulant Treatment – A Decision Dilemma

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Abstract

Summary and Background: Spontaneous spinal epidural hematoma (SSEH) is a known occurrence in patients on anticoagulant therapy. There is an increased risk of developing hematoma after the spine surgery if anticoagulation therapy is reinstated.

Purpose of Study: The purpose of the study was to find out solution related with perioperative anticoagulant therapy in high-risk cases if patient redevelops hematoma and paraplegia due to continuation of anticoagulant therapy.

Case Report: A 30-year-old male presented to us with history of progressive paraparesis. He had history of mitral valve replacement twice followed by cerebrovascular stroke and on regular oral anticoagulant therapy. Magnetic resonance imaging revealed SSEH from C6-T1 level with cord compression. Initial decision was taken to conservatively treat as his coagulation parameters were altered and he was on high-risk for developing thromboembolism related complications if anticoagulant medicines were stopped. However, urgent laminectomy and evacuation of SSEH had to be performed due to rapid worsening of neurology. Postoperatively, patient had significant neurological recovery and anticoagulant therapy reinstated after 12 hours of surgery. Patient developed acute paraplegia within 2 hours of anticoagulant therapy due to post-operative hematoma, which was drained out by opening the wound bedside. He regained neurological recovery within 5 min. Anticoagulation therapy was withheld for next 36 hours and reinstated with low-dose intravenous heparin followed by low-molecular weight heparin without any complications. His coagulation parameters and 2-D echo were followed up daily to check cardiac conditions. Patient improved clinically and became self-ambulatory.

Conclusion: Post-operative hematoma after spine surgery should be kept in mind in patients who are on anticoagulant treatment. Reinstating anticoagulation treatment in such high-risk patients should be done with lot of caution and initially with low-dose heparin followed by regular anticoagulation therapy. Close observation on neurological status is must to avoid permanent neurological injury.

Keywords: Spontaneous spinal epidural hematoma, Anticoagulant treatment, Decision dilemma

Introduction

Spinal epidural hematoma was first described by Jackson in 1869 [1]. After that, more than 600 cases of spontaneous spinal epidural hematomas (SSEH) treated with surgical decompression have been reported [2]. Frequently, SSEH peaks at the age group 15–20 and 47–75 years with male predominance. Recently, several studies have been published

regarding the conservative management of SSEH [3, 4, 5]. The increase in the reported SSEHs is most likely due to increased use of magnetic resonance imaging (MRI) in establishing the radiological diagnosis [6]. SSEH treatment should be considered a life-saving procedure, where pre-operative neurological condition is a major prognostic factor [7].

There are a few reports available suggesting when to stop anticoagulation therapy if a patient is on anticoagulation therapy requiring spine surgery [8, 9]. There is also a report suggesting when to restart anticoagulation therapy once spine surgery is over depending on type of anticoagulation agent [10]. However, there is no report available regarding perioperative management of anticoagulation treatment for such high-risk patient suggesting when develop SSEH. If we do not start anticoagulation therapy after the surgery, there is



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Submitted: 01/12/2021; Reviewed: 10/01/2022; Accepted: 05/02/2022; Published: 10/04/2022

Back Bone: The Spine Journal (The Official Journal Of "Spine Association of Gujarat") | Available on www.backbonejournal.com | DOI:10.13107/bbj.2022.v03i01.038
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increased risk of developing thromboembolism and related cardiac or neurological complications; and if we reinstate anticoagulation therapy soon after the surgery, there is inherent risk of developing hematoma again.

In this case report, we report a patient who was on regular anticoagulant therapy after the mitral valve replacement, developed spinal epidural hematoma C6-T1 with spinal cord compression with paraparesis. The aim of this case report was to decide in such cases regarding anticoagulant therapy during perioperative period as patient redeveloped hematoma and paraplegia on the 1st post-operative day due to continuation of anticoagulant therapy with a view of mitral valve replacement. We believe that this case will focus on the dilemma of whether to continue or withhold anticoagulation therapy during perioperative period while considering spine surgery for drainage of SSEH.

Case Presentation

A 30-year-old male patient presented to outpatient department clinic with history of 1 week of severe neck and upper back pain associated with progressive bilateral lower limb radicular pain, loss of sensation, weakness, and imbalance while walking and urinary incontinence. He was unable to walk and was wheelchair bound on presentation. Power in both lower limbs was 3/5 with decreased reflexes in both lower extremities and decreased sensations below T2. Reflexes in both lower extremities were exaggerated with planter extensors. There was no history of trauma or physical exertion. In his medical history, he was diagnosed with severe mitral regurgitation in his childhood which was operated by mitral valve replacement. After 1 month of mitral valve replacement, he had developed thrombosis in replaced valve and therefore, revision surgery was performed. He also had history of cerebrovascular stroke with the left hemiparesis 6 years back which was treated conservatively. He had been on anticoagulant therapy since the mitral valve replacement with regular maintenance of international normalized ratio (INR) between 2 and 3. With a

view of his current disability and history of heart and neurological problems, he was admitted in intensive care unit (ICU) for further evaluation and investigations under care of a team comprised neurologist, cardiologist, and spine surgeon.

On admission, his blood investigations and radiological investigations were performed. His MRI spine showed spinal epidural hematoma at C6-T1 with spinal cord compression (Fig. 1). Hematological investigation showed normal complete blood counts with hemoglobin of 13.3 g/dl; however, coagulation profiles [increased activated partial thromboplastin time (APTT) 52.5 s, increased partial thromboplastin time (PTT) > 90.0 s, and increased INR > 6.7] were abnormal. 2D-echo was done by cardiologist which did not exhibit any significant abnormality in the functioning of heart and replaced valve. MRI brain suggested by neurologist did not indicate any other abnormality. Although surgery was indicated in the form of drainage of SSEH, initial conservative approach was taken collectively due to altered coagulation profile and high INR. However, within 6 hours of this decision, he started complaining of severe radiating pain in both lower extremities with significant reduction in muscle power to 1/5 in both lower extremities with absent reflexes and loss of sensations below T2. Therefore, call for urgent surgical decompression and drainage of hematoma was taken.

The patient underwent posterior laminectomy C6-T1 with drainage of epidural hematoma and decompression of spinal cord (Fig. 2a-c). The surgery was uneventful with 500 ml of blood loss and there were no complications or dural tear intraoperatively. There was significant hematoma found in epidural space compressing the spinal cord (Fig. 2a). There was also a small bleeder found which was coagulated with electrocautery (Fig. 2b). Wound was closed after achieving thorough decompression of the cord with negative suction drain. Patient was shifted to ICU postoperatively and muscle power became 4/5 in both lower extremities within 12 hours after surgery.

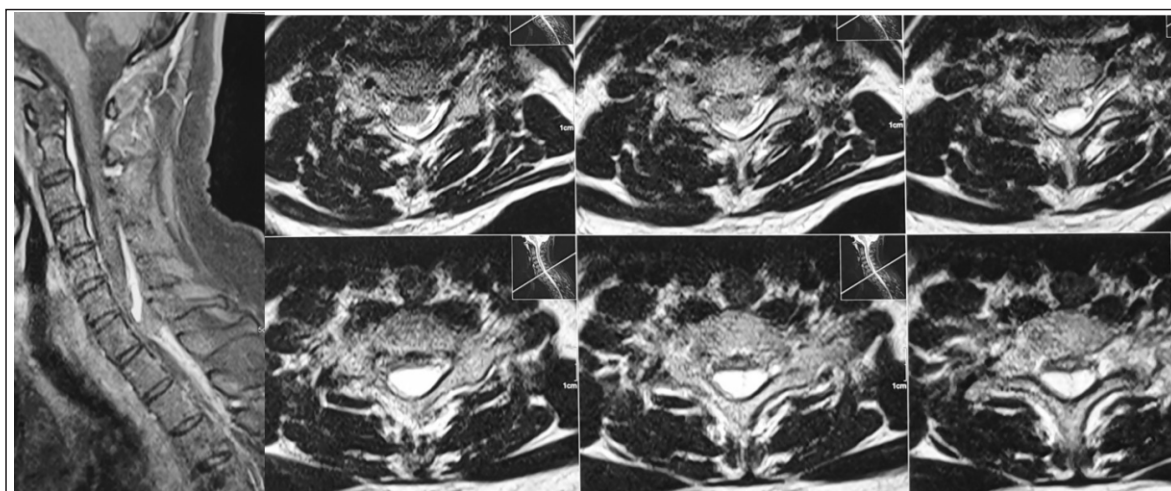


Figure 1: T2 sagittal and axial images of MRI of cervico-dorsal spine shows epidural hematoma formation from C6-T1 level causing severe compression of spinal cord.

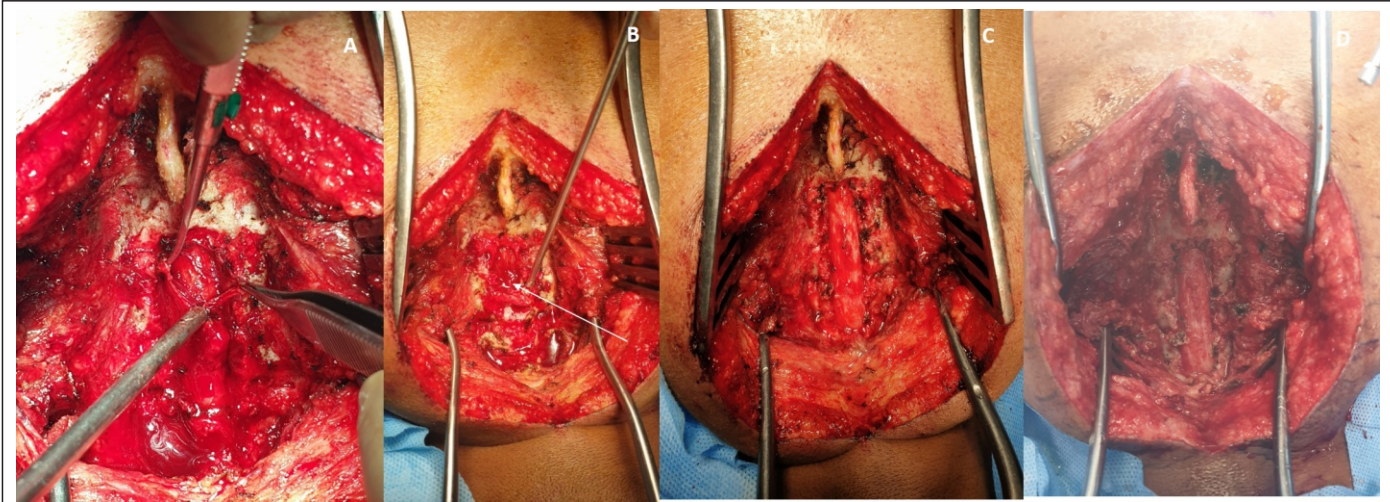


Figure 2: Intraoperative picture of cervico-dorsal spine A) shows significant hematoma formation in the epidural space, B) arrow suggesting a small bleeder causing hematoma formation and C) after the thorough decompression of the cord at the 1st surgery; and D) after decompression of repeat hematoma formation that caused acute paraplegia.

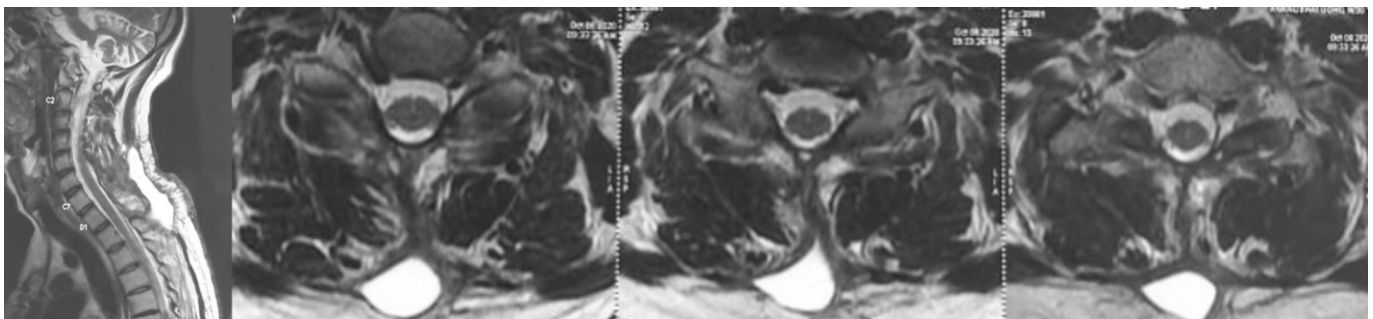


Figure 3: T2 sagittal and axial images of MRI of cervico-dorsal spine at 3-month postoperatively showed resolution of epidural hematoma with adequate decompression of spinal cord except small collection found in posterior myofascial plane.

After the discussion with the team, anticoagulant treatment was restarted after 12 hours of surgery in the form of injection heparin 5000 IU intravenously every 6-hourly. The aim was to prevent any complications due to his cardiac condition. However, on the 1st post-operative day, patient developed sudden onset of loss of sensations below T2 with complete loss of power in both lower extremities to 0/5. There was no reflexes elicitable in both the lower limbs. Emergency call was sent to spine team regarding his condition. Confirming acute paraplegia with a suspicion of post-operative hematoma, all the sutures were removed and operative wound was opened in ICU with all antiseptic and aseptic precautions. There was drainage of 150 ml of blood from the wound which was covered with sterile dressing. Patient regained his power in both lower extremities to the level of 2–3/5 and started feeling sensations below T2 within 5 min of drainage. The patient was then shifted to OR for evacuation and drainage of remaining hematoma to achieve and reconfirm cord decompression followed by suturing of the wound (Fig. 2d). This time, the decision was taken not to start his anticoagulation therapy for next 36–48 hours depending on his condition. 2D-echo was performed twice daily to check on his cardiac conditions along with observation of his coagulation parameters. After the

second surgery, the patient showed significant improvement in the lower limb power. His anticoagulation therapy was reinstated with heparin initially 2500 IU intravenously 8-hourly for 2 days followed by Enoxaparin 0.4 mg subcutaneously twice a day and 0.6 mg twice a day later. His surgical wound was dry, and the surgical drain was removed after 5 days. During this time, there was a significant improvement in his neurology with power in both lower extremities that were 4/5 with regained sensations in all four limbs. He was made stand on 4th post-operative day and take few steps on 5th post-operative day. Pre-discharge, patient's clinical condition improved, and coagulation profile were within normal limits (APTT- 25.5 s, PTT- 14.2 s, and INR- 1.6). He was discharged with anticoagulation therapy of injection Enoxaparin 0.6 mg subcutaneously once a day for 2-weeks followed by oral anticoagulation therapy. He remained on regular follow-up initially at every 2-week interval with cardiologist and spine team for 1 month. His sutures were removed after 15 days of surgery. He became independent walker within 6-weeks of surgery. His post-operative MRI also confirmed resolution of his spinal epidural hematoma at 3 months (Fig. 3). At post-operative follow-up of 1 year, he is back to his work without any sequel.

Discussion

SSEH is a rare surgical emergency and accounts for <1% of all spinal epidural lesions, with an estimated annual incidence of only 0.1/1,00,000 [11]. Precipitating factors associated with SSEH include traumatic spinal injuries, blood dyscrasia, anticoagulation therapy, spinal and epidural anesthesia, lumbar puncture, Paget's disease, tumor genesis, and arteriovenous malformation [12, 13, 14]. Therefore, etiology of SSEH in our case may be anticoagulant therapy due to mitral valve replacement. There are few guidelines available when to restart anticoagulation therapy after the spine surgery to reduce the risk of thromboembolism related complications [10, 15, 16]. However, no literature suggests how to manage perioperatively when a patient with a high-risk of developing thromboembolism needs to undergo spine surgery. We believe that this is the first case report presenting a dilemma regarding use of perioperative anticoagulation therapy in a patient of mitral valve replacement and cerebrovascular stroke developing SSEH and requires spine surgery.

Antiplatelet, anticoagulant, or thrombolytic medications, such as aspirin, warfarin, heparin, tissue plasminogen activator, and streptokinase, have been reported to be associated with SSEH [11, 17]. Bakker et al. reported that the percentage of patients (22.8%) with SSEH using oral anticoagulants is much higher than the percentage of patients using platelet inhibitors (10.1% of all patients) [11]. In our case, the patient was taking warfarin for mitral valve replacement and CVA which fits into the most likely risk factor for developing SSEH. Beatty et al. concluded that the source of bleeding was the "free" anastomotic arteries in the epidural space that connects with radicular arteries and which causes rapid spinal cord compression [18]. However, Zuo et al. concluded that reasons for SSEH are local pooling within valve-less, thin-walled epidural veins, and brief increases in intravenous pressure leading to epidural vein rupture [7]. In our case, we found a small bleeder during the surgery which was the resultant factor for hematoma. The initial symptoms of SSEH can mimic disc prolapse and even rarely be misdiagnosed as transient ischemic attack or stroke. Approximately 37% of patients present with complete loss of sensory-motor functions, while the others have some residual sensory or motor function. This has prognostic significance, as those with some residual sensory-motor functions are more likely to show complete recovery than those with no motor function [18, 19, 20, 21, 22]. A possible cause of cerebrovascular event was thought at the first in our patient initially due to history of stroke which eventually confirmed with SSEH [14].

SSEH can be treated conservatively and surgically depending on size of hematoma and neurology of the patient. Laminectomy and decompression with hematoma evacuation

are traditionally a choice of the treatment for SSEH [20, 23, 24, 25]. Some authors concluded that immediate replacement therapy in patients with a coagulopathy prevents progression of the hematoma, allowing for relief of symptoms and regression of neurological signs [2, 20, 26]. On the other hand, Connelly et al. concluded that coagulopathy related SSEH can be treated conservatively because hematoma remains in liquid form for longer period of time and allows spread of hematoma in the epidural space [27]. On the other hand, prolong bleeding may cause a larger hematoma causing more compression on the spinal cord. Kim et al. concluded that among 15 patients, 10 underwent decompressive surgery, and remaining five were treated with conservative management [21]. Conservative management is feasible in selective patients who present with neurologic status as American Spinal Injury Association (ASIA) Scale E or in whom early recovery of function has initiated with ASIA Scale C or D [17]. Therefore, conservative group patients were treated with the low-dose methylprednisolone as the equivalent dose of dexamethasone. Duffill et al. concluded that all four patients with SSEH were managed conservatively with steroids followed by repeat MRI after 1 week and mobilization was allowed after improvement in neurological symptoms [28]. Therefore, conservative management for SSEH can be done in whom recovery has started with observation and steroids. Other studies have reported conservative treatment in patients with severe neurological involvement after SSEH because of the coexistence of serious coagulopathy, anticipated risks of operation and/or refusal for surgery [20, 29]. In our case, initial conservative trial was thought with a view of his coagulation parameters and previous history of mitral valve replacement despite power in both limbs 3/5. However, it changed to surgical decision on emergency basis once his neurological condition worsened with power becoming 1/5. We feel that such patients need continuous neurological monitoring to make decisions about conservative versus surgery.

The most common form of the treatment for SSEH is laminectomy and evacuation of hematoma [14, 29]. Studies have concluded significantly better outcome in patients with complete neurological deficit that underwent surgical decompression within 36 hours of symptom onset [25] and within 48 hours in patients with incomplete deficit [26]. Kim et al. concluded early diagnosis based on MRI findings and hematoma evacuation within 24 hours of symptoms onset can lead to full neurologic recovery in patients with SSEH [21]. On the other hand, Liu et al. concluded that patients with complete neurological deficits got disappointing prognosis, even after getting surgical treatment within 24 hours of onset of symptoms [30]. We have done wide laminectomy with evacuation of hematoma with additional search for bleeder to

decompress the spinal cord in our patient. Postoperatively, his power improved significantly.

Perioperative management of anticoagulation therapy is critical in patients who are at high risk such as cardiac valve replacement, atrial fibrillation, venous thromboembolism, cerebrovascular stroke, and post-myocardial infarction [10]. The literature mentioned to stop or withhold anticoagulation therapy before considering a major surgery depending on which type of anticoagulation treatment is going on [8, 31]. Some have emphasized on controlling INR below 2 before any major spine surgery. We initially tried to control coagulation parameters by treating conservatively with observation; however, we had to go ahead with surgical decompression as patient started deteriorating in neurology. Therefore, it is important to note that if it is a spine surgery with deteriorating neurology, surgery can be considered despite of continuation of anticoagulation therapy or altered coagulation parameters in emergency. In addition, the literature mentioned to reinstate intravenous heparin after 12 hours to prevent thromboembolic complications in high-risk patients like ours [32]. If we want to start low-molecular weight heparin (LMWH) or other types of oral anticoagulation treatment after the spine surgery, 36–48 hours time is also recommended to prevent post-operative hematoma [16]. As our patient was a very high-risk, cardiologist insisted on starting intravenous heparin 5000 IU 8-hourly to avoid any complications; and therefore, we collectively took a call to start heparin after 12 hours of surgery after confirming improvement in the neurological status of patient. However, patient developed acute onset paraplegia in 2–3 hours of anticoagulation treatment for which post-operative hematoma was thought. Therefore, immediately, all sutures were removed and wound was opened to let the hematoma drain which resulted in recovery of neurology

within 5 min. We agree that MRI imaging would have proved occurrence of hematoma before the drainage; however, MRI would have taken longer time and we did not want to miss the crucial time of achieving decompression, we just went ahead with draining hematoma inside the ICU. From this incidence, we believe that if a patient develops worsening of neurology after spine surgery and reinstating anticoagulation therapy, post-operative hematoma should be thought, and actions need to be taken urgently. However, once he developed post-operative hematoma, anticoagulation therapy was completely ceased for next 36 hours, and intravenous heparin was started with 2500IU intravenously 8-hourly with caution for 48 hours which was switched over to LMWH. The decisions regarding anticoagulation therapy and surgery were taken as a team approach along with patient and his relatives concerns. In a recent article by Louie et al. noted significant differences between anticoagulation initiation and cessation methodology among surgeons [9]. We believe that this type of dilemma can happen in patient of mitral valve replacement with regular anticoagulation therapy. Our experience would provide an important guide regarding perioperative anticoagulation therapy in such cases in the future.

Conclusion

We would like to suggest that post-operative hematoma after the spine surgery for SSEH or decompression should be kept in mind in patients who are on anticoagulation therapy. Reinstating anticoagulation treatment in such high-risk patients should be done with lot of cautions and initially with low-dose heparin followed by regular anticoagulation therapy. Vigilant watch on neurological status during the entire process is must to avoid any undulant complication due to thromboembolism or neurology worsening.

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Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the Journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Conflict of Interest: NIL
Source of Support: NIL

How to Cite this Article

Modi HN, Patel UD | Spontaneous Spinal Epidural Hematoma Causing Paraparesis in a Patient of Mitral Valve Replacement with Anticoagulant Treatment – A Decision Dilemma | Back Bone: The Spine Journal | April-September 2022; 3(1): 36-41.