

Spontaneous Spinal Epidural Haematoma Mimicking a Heart Attack: A Case Report

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Abstract

Spontaneous spinal epidural hematoma (SSEH) is a rare disease. Typical symptoms develop chest and back pain, numbness, radicular paresthesia, sensory deprivation and progressive paralysis. SSEH sometimes mimics other diseases like stroke, lung pathologies, myocard infarctus (MI). This situation makes it difficult to treat this pathology. We presented a case with epidural hematoma which compressing the spinal cord at the T2-4 level. He had applied to our emergency service with acute severe chest pain and numbness. He was given clopidogrel, acetylsalicylic acid for the medical treatment of MI by the cardiologist. Therefore, progressive paralysis of lower limbs, incontinence and sensory deprivation were developed. Laminectomy and hematoma evacuation were performed. After physical therapy, he had a complete neurological recovery on the postoperative day 14. SSEH should be kept in mind and early surgical treatment should be performed if neurological deterioration develops.

Keywords: Chest pain, myocardial ischemia, spinal epidural hematoma, emergency

Introduction

Spinal hematoma is a rare disease and epidural hematomas are the most common type of this disease. The incidence of SSEH is 0.1 per 100000 patients per year¹. The etiology is unclear in 40% of cases². Neoplasm, trauma, vascular malformation, in prolonged valsalva maneuver, anticoagulation therapy, in patients with internal jugular vein thrombosis, in pregnancy, and thrombolytic therapy are the most known causes³. Typical symptoms develop chest and back pain, numbness, radicular paresthesia, sensory deprivation and progressive paralysis. Delayed diagnosis and treatment cause poor prognosis. As many article reported, the primary treatment option is decompressive surgery. But some articles report that non surgical treatment or delayed surgery can be performed^{4,5}.

SSEH sometimes mimics other diseases like stroke, lung pathologies, myocard infarctus (MI). This situation makes it difficult to treat this pathology. We presented a case which mimics a heart attack.

Case Report

A 49-year-old man presented to our emergency service with acute severe chest pain and numbness. Because of the chest pain, he was referred to cardiology by emergency service. Electrocardiography showed T-wave inversion, but the troponin value was normal. He was admitted to the intensive care unit with a preliminary diagnosis of acute coronary syndrome and was given clopidogrel and acetylsalicylic acid by the cardiologist. Five hours later, the patient developed progressive paralysis of the lower limbs, incontinence and sensory deprivation. Physical examination showed paraplegia and sensory deficit below the T4 level. Urgent spinal magnetic resonance imaging (MRI) revealed a posterior epidural haematoma which compressed the spinal cord at the T2–T4 levels. In our patient, the T2-weighted images were hyperintense (Figures 1A and 1B). As soon as spinal spontaneous epidural haematoma was detected, the patient was operated on immediately. Clopidogrel and acetylsalicylic acid were discontinued, as they may increase

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Figure 1A. T2-W sagittal MR images (Pre-operative)



Figure 2A. T2-W sagittal MR images (Post-operative)



Figure 1B. T2-W sagittal MR images (Pre-operative)

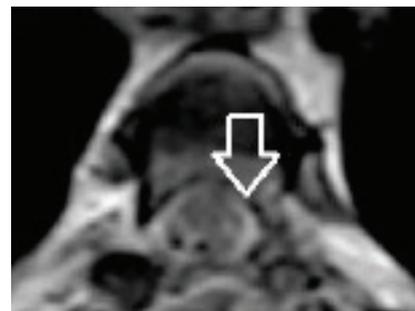


Figure 2B. T2-W axial MR images (Post-operative)

bleeding. Three levels of laminectomy from the lower T2 to the upper T4 and haematoma evacuation were performed. No evidence of vascular malformation or neoplasia was found during the operation. After the haematoma evacuation, the spinal cord returned to its normal shape. Histopathological examination confirmed haematoma. Chest pain healed in the early postoperative period. After physical therapy, the patient had complete neurological recovery on postoperative day 14. No urinary dysfunction occurred. Postoperative MRI showed decompression of the spinal cord (Figures 2A and 2B).

Discussion

Patients with SSEH present with severe and acute back-neck or chest pain. These complaints can be attributed to spinal musculoskeletal strains, heart diseases, degenerative spine

diseases or lung diseases. With the onset of neurological deficits, such as paraplegia, paraparesia, urinary or anal sphincter dysfunction, spinal abscess, spinal mass and disc herniation should be considered. Some patients have nerve root symptoms with or without spinal cord and nerve root compression. Bilateral or unilateral sensorimotor deficits and the Brown-Sequard syndrome are common⁶. This situation causes confusion in diagnosis. Furthermore, a delayed diagnosis leads to poor prognosis. A meta-analysis study reported that the best neurological recovery occurred in patients who were operated on in the first 12 hours after the onset of symptoms⁷. MRI is the gold standard for early diagnosis⁸. T2-weighted MR images show heterogeneously hyperintense lesions as SSEH. In T1-weighted images, SSEH appears isointense or hypointense. There is no contrast enhancement except hyperacute stage haemorrhage.

The appropriate modality of treatment in SSEH is neural decompression and haematoma evacuation. If necessary, posterior spinal instrumentation can be added for fusion. In a review of 330 SSEH patients, the role of early surgery was

emphasised⁹. Surgical treatment was recommended within 36 hours for complete spinal cord dysfunction or within 48 hours for incomplete spinal cord dysfunction.

Some authors advocate the spreading theory, which states that haematomas are spread along the epidural space and neural foramina. In this way, neural compression does not occur^{5,8}. In a review of the literature, the mean length of the haematoma was found to be significantly higher in patients who undergo conservative treatment. Although this finding supports the spreading theory, it is not conclusive evidence. Haematoma length is not a guide for treatment⁸. Kim et al. reported that they administered anticoagulant and antiplatelet therapy in two patients, but this did not result in improved clinical outcomes⁴. In our case, the patient's neurological condition deteriorated after anticoagulant and antiplatelet treatment for acute coronary syndrome. As the spreading theory was not an appropriate reason for treatment delay with acute paraplegia, we operated on the patient immediately when paraplegia and a sensory deficit below the T4 level occurred. In a case report with SSEH, Sakaguchi et al.¹⁰ emphasised that the antiplatelet drugs given to the patient should be discontinued because of the effect of increased bleeding. In our case, clopidogrel and acetyl salicylic acid were given primarily because acute coronary syndrome was considered. However, when spinal spontaneous epidural bleeding was detected in the patient, treatment was discontinued, as the drugs may contribute to increased bleeding. Chest pain healed in the early postoperative period. After physical therapy, the patient had complete neurological recovery on postoperative day 14.

Conclusion

Patients with SSEH may present with back or chest pain without neurological deficits and trauma, like heart attack. Anticoagulant and antiplatelet therapy for acute coronary syndrome can be administered. Although some authors advocate the spreading theory this therapy can lead to an increase in hematoma, and neural compression as in our case. Although it is rare, SSEH should be kept in mind and early surgical treatment should be performed if neurological deterioration develops.

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Conflict of Interest

The authors declared no potential conflict of interest.

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