# Spontaneous Spinal Epidural Hematoma: A rare neurological complication in a patient following routine Coronary Angiography

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### **Abstract**

Spinal epidural hematoma is an uncommon but potentially devastating complication associated with various medical interventions. We present a case report of this rare neurological complication following a routine coronary angiography. A 69-year-old male with Aortic Stenosis presented for a scheduled coronary angiography to assess his cardiac status before valve replacement. The procedure was uneventful but approximately 15 minutes after the angiography, he developed sudden-onset of rapidly progressing weakness and numbness in bilateral lower extremities. An urgent neurologic evaluation revealed signs of spinal cord compression, prompting a magnetic resonance imaging (MRI) scan which showed an extensive spinal epidural hematoma spanning multiple levels within the cervicothoracic spine. This case highlights the importance of recognizing this rare but a potential complication following routine coronary angiography even in the absence of significant procedural complications. Timely diagnosis and prompt surgical intervention are crucial for achieving favorable outcomes and minimizing morbidity associated with this condition.

Keywords: Coronary angiography, Neurological complication, Spinal epidural hematoma

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## Introduction

Coronary angiography (CAG) is the gold standard procedure for various diagnostic and therapeutic purposes. Although CAG is considered relatively safe and operator friendly, various complications may be seen. In this case report, we present a rare neurological complication following the routine coronary angiography.

## **Clinical History**

A 69 Y/M presented to Out Patient Department with history of exertional shortness of breath and chest discomfort. There were no background medical comorbidities. On clinical cardiovascular examination, his blood pressure was 140/70 mmHg with low volume, slow and delayed rising pulse of 70 bpm without radio-radial and radio-femoral delay. On precordial auscultation, there was soft S2 and late peaking harsh crescendo-decrescendo systolic ejection murmur of grade III intensity which was heard best at 2nd right intercostal space with radiation to carotid artery. Moreover, an early diastolic murmur of grade III intensity was heard at 3rd and 4th left intercostal space heard best in sitting and leaning forward with breath held at end expiration. The twelve lead electrocardiography showed normal sinus rhythm with left ventricular hypertrophy (according to Sokolow-lyon voltage criteria). Transthoracic echocardiography demonstrated degenerative severe aortic stenosis with moderate aortic regurgitation with normal left ventricular ejection fraction.

Blood investigations were within normal limits. Pre-operative CAG was planned for possible need of aortic valve replacement (AVR). Bleeding disorder and coagulopathies were ruled out. The CAG was done through right radial artery with 5F radial sheath. As a part of routine procedure, intravenous unfractionated heparin 2500U was given through right radial artery to prevent thromboembolism. Intravenous verapamil 2.5mg and glyceryl trinitrate (GTN) 50 mcg were given through same radial artery to prevent radial artery spasm before the catheterization. Coronary angiography was done with 5F Tiger catheter where about 40 ml contrast was used. The CAG revealed normal coronaries.

After 15 minutes of completion of the procedure, patient developed paraparesis with loss of sensation over bilateral lower limbs. On clinical examination, he was conscious, well oriented to time, place and person, with Glassgow coma scale (GCS) of 15/15. The power of upper limbs was 4/5 and lower limbs 0/5. However, the spinal level was not well demarcated. Bilateral plantar reflex was mute and cranial nerve examination was within normal limit. Subsequently, plain CT head was done for suspected cerebrovascular accident but revealed normal findings except for age related cerebral atrophy (fig.1). Then MRI spine was done which revealed extradural content suggestive of hematoma in anterior aspect of spinal cord at cervicothoracic region (C7-T1 level) measuring 19cm in length with maximum width of 6.2mm causing cord compression with signal intensity changes at same level (myelopathy). Neurosurgery consultation was done and he was advised for urgent hematoma



evacuation. But the patient underwent Laminectomy (C5-D7) and drainage of the extradural hematoma after 2 days due to delayed consent. Per operative finding included extradural clots at anterior aspect of C5-D7 spine with cord pushed posteriorly. Vascular malformations were absent. Then patient was kept for observation and physiotherapy was initiated. He was discharged on 10th post-operative day without immediate neurological improvement at the time of discharge. Rehabilitation support was continued and advised for follow up.

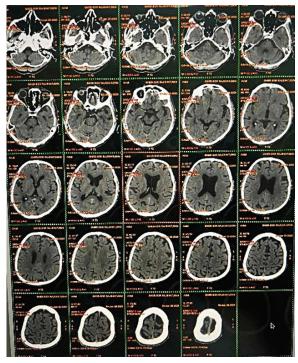


Figure 1: Normal Plain CT Head

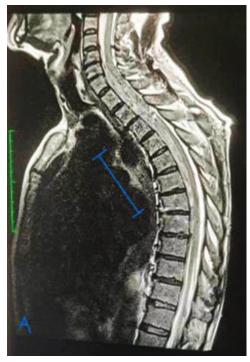


Figure 2(A)

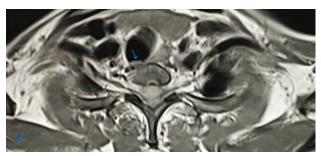


Figure 2(B)

Figure 2 (A, B): MRI spine showing extradural collection in cervicothoracic region.

### **Discussion**

As per the recommended guidelines, for a patient with valvular heart disease undergoing valve replacement surgery, it is necessary to evaluate for concomitant coronary artery disease via CAG prior to surgery. Even though CAG is regarded as a safe procedure with mortality of less than 0.08%, various complications may arise.1 Complications of invasive CAG can be divided into 3 main groups: cardiac related, vascular related and others (thromboembolism, contrast induced allergy and nephropathy, heparin induced thrombocytopenia). Arterial thromboembolism can occur to any vascular bed manifesting as stroke, transient ischemic attack (TIA), intestinal ischemia, or peripheral emboli. Among all complications, local vascular injury with hematoma at the access site is the most common one with the incidence between 0.7-11.7%.(1) However, bleeding at unusual place like spontaneous spinal epidural hematoma (SSEH), retroperitoneal and retropleural hematomas has been recorded.2-4

SSEH is a rare neurosurgical emergency with the incidence of 0.1 out of 100,000 and accounts 40-50% of all spinal epidural hematomas.5 It is considered that SSEH mostly arise from the rupture of valveless low pressure epidural venous plexus system associated with rise of intra-abdominal or intra-thoracic pressure. However, for the acutely developing hematomas, arterial source is proposed.<sup>2,5,6</sup> Depending upon the site and extent of hematoma and degree of spinal cord compression, clinical presentation may vary from neck and/or back pain to neurological deficits such as limb paraplegia/paresis, bowel and bladder incompetence or even death. Direct damage to spinal cord and disturbed vascular supply is the proposed mechanism for the clinical symptoms. Even though most SSEH are idiopathic, there are few reports on predisposing factors such as vascular malformation, coagulation or platelet disorders, thrombolytic therapy, anticoagulation, leukemia, hemophilia, hypertension, pregnancy and minor trauma.6-11 As pertaining to our case, the development of cervicothoracic SSEH is probably related to use of anticoagulant (unfractionated heparin) which was used prior to the procedure.

For diagnosing SSEH, MRI is considered the investigation of choice.<sup>5</sup> It can identify the site, extent and size of hematoma along with degree of spinal cord compression. Angiography is done only for suspected vascular malformations. For those with significant spinal cord compression, urgent hemilaminectomy with evacuation of hematoma is the preferred treatment. However, hematomas with mild or non-progressive neurological deficits and those related to coagulopathy can be treated conservatively.<sup>5,6</sup> There are multiple factors related to prognosis of SSEH, among which pre-operative neurological condition, site and extent of hematoma and spinal cord edema are believed to be the major ones. Moreover, interval

between the development of hematoma and surgical interventions also determine the neurological outcome. Delay in the surgical intervention in our case is the probable cause of poor neurological outcome. Therefore, it is recommended to intervene within 36 hours for patients with complete neurological deficit and within 48 hours for patients with incomplete neurological deficit for better outcome.<sup>5</sup>

## **Conclusion**

This case report highlights that although very rare, bleeding at unusual places such as SSEH may occur after routine coronary angiography procedure even when no predisposing factors exist. Also, timely recognition of the symptoms for early diagnosis and treatment is crucial for favorable outcome.

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