

Case Report

Spinal epidural hematoma mimicking subarachnoid hemorrhage: A case study

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Abstract

Background: Spontaneous spinal epidural hematoma (SSEH) is a rare spinal cord disorder requiring urgent diagnosis and prompt treatment to prevent irreversible neurological damage. Subarachnoid hemorrhage (SAH), usually presenting with headache and neck stiffness, is also a worldwide neurosurgical emergency. In this case study, a patient on clopidogrel presented with an SSEH mimicking a spontaneous SAH.

Case Description: A 74-year-old female presented with severe headache and neck pain which ultimately attributed to a cervicothoracic SSEH resulting in cord compression. Notably, there was a long delay in establishing the diagnosis of SSEH as her symptoms mimicked a SAH. Although the patient ultimately underwent spinal surgery followed by intense rehabilitation, the diagnostic delay resulted in a poor neurological outcome.

Conclusions: Patients with spontaneous cervicothoracic epidural hematomas may occasionally present with symptoms of severe headache and neck pain/stiffness mimicking an SAH. Long diagnostic delays in establishing SSEH may result in irreversible cord damage.

Key Words: Clopidogrel, magnetic resonance imaging, spinal epidural hematoma, subarachnoid hemorrhage

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INTRODUCTION

Subarachnoid hemorrhage (SAH), usually presenting with headache and neck pain/stiffness, is a worldwide neurosurgical emergency and must be differentiated from spontaneous spinal epidural hematoma (SSEH). SSEH, however, is a rare entity that must be differentiated from SAH early on in the clinical course to avoid irreversible spinal cord injury (0.1 patients per 100,000 individuals per year).^[2,3,6]

Here, we present a 74-year-old female on clopidogrel whose headache/neck stiffness was originally misdiagnosed as a SAH. This led to delayed treatment of a cervicothoracic SSEH that resulted in an irreversible quadriplegia.

CASE REPORT

An obese 74-year-old female patient presented with a 3-day history of spontaneous sudden-onset headache and neck pain, accompanied by nausea, and a worsening

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occipital headache unresponsive to analgesics. Notably, she was taking clopidogrel 75 mg/d for coronary artery disease.

Neurological examination revealed marked nuchal rigidity, positive Kernig's sign, normal fundoscopic examination (e.g., no papilledema), and no other focal neurological deficits. The initial clinical diagnosis was a spontaneous SAH.

Diagnostic studies

Routine laboratory and coagulation tests were within normal limits. Chest X-ray showed no abnormalities. Noncontrast cranial computed tomography (CT) was negative for intracranial hemorrhage. Lumbar puncture (LP) yielded clear cerebrospinal fluid (CSF); laboratory analysis of the CSF was normal. Doppler ultrasonography of the carotid revealed no significant stenosis or arterial dissection.

Progressive neurological deterioration

Thirty hours postadmission, the patient developed a rapidly progressive right hemiparesis accompanied by severe pain in the right shoulder. On examination, she exhibited marked hypesthesia with a severe flaccid paresis in both the right upper and right lower extremities (2/5 motor function). On the right side, deep tendon reflexes were decreased and there was no Babinski response. The second emergent brain CT scan was negative.

The patient was transferred to another institution 8 hours after the onset of hemiparesis at which point she was nearly quadriplegic (e.g., severe bilateral motor and sensory deficits below C5 level, labored respiration, and decreased chest expansion). Following cardiopulmonary resuscitation, magnetic resonance imaging (MRI) scan

of the cervical spine demonstrated a large extradural mass, dorsally to the spinal cord extending from C3 through mid-T3. The mass had well-defined contours and contributed to multilevel posterior to anterior spinal cord compression. It was hyperintense on T1 [Figure 1a] and heterogeneously hyperintense on T2-weighted studies [Figure 1b]. It was more right-sided in location and was accompanied by an increased cord signal from C3 all the way down to the T2 level, which was suggestive of severe cord edema/infarction [Figure 1b]. Following gadolinium contrast administration, no enhancement was observed. These radiological findings were compatible with a cervicothoracic spinal epidural hematoma.

Operational procedure

The patient underwent a C3 to T1 laminectomy with lateral masses screws – posterior fixation to prevent instability; the C3–T3 epidural hematoma was completely evacuated. Below the ligamentum flavum, large hard, dark clots were found, but no discrete bleeding points or abnormal vessels could be identified [Figures 2a and b]. At the end of the procedure, routine hemostasis was achieved and dural pulsations were evident. Histopathological examination was consistent with a hematoma; there was no evidence of a neoplasm or vascular malformation. The patient failed to improve following surgery and expired 18 months later.

DISCUSSION

SSEH rarely causes spinal cord compression. It requires early diagnosis and prompt surgical treatment to prevent irreversible neurological damage or even death if the hematoma extends into the cephalad cervical region.^[2,3,6] Presenting symptoms typically include sudden onset of pain, local and/or radiculopathy, followed by progressive myelopathy (e.g., weakness, sensory loss, and sphincter disturbances) developing within minutes to hours or days later.^[1,2,5,6] Somewhat confounding, is the overlap of



Figure 1: An extradural more right -sided mass located dorsal to the spinal cord, extending from C3 through the mid-T3 vertebral level, resulting in marked spinal cord compression and edema/myelomalacia (high intrinsic cord signal). It was hyperintense on T1-weighted images (a), and heterogeneously hyperintense on T2-weighted images (b)

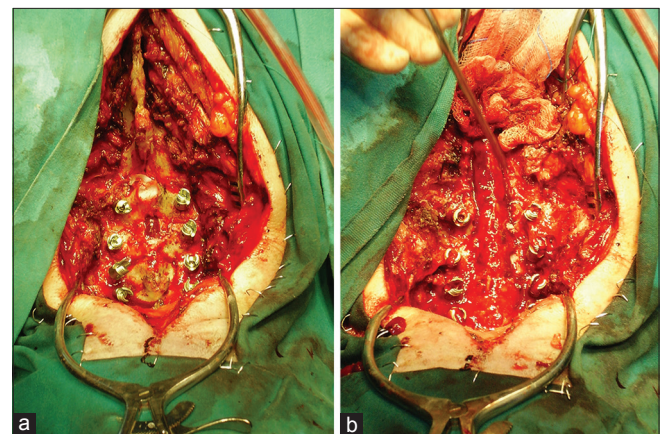


Figure 2: Following multilevel C3-T1 laminectomy with posterior instrumentation, the intraoperative photographs revealed a large epidural hematoma (a and b)

Table 1: Summary of previously reported cases of spontaneous spinal epidural hematomas mimicking subarachnoid hemorrhage

Author (reference number)	Age/Sex	Site	Time from symptoms onset to neurological deficit	Treatment	Neurological outcome
Fukui <i>et al.</i> ^[1]	31/F	C1–C3	No deficit	Conservative	Full recovery
Fukui <i>et al.</i> ^[1]	67/M	C3–C5	10 h	Laminectomy	Minimal residual deficit
Labeodan <i>et al.</i> ^[5]	36/F	T5–T7	2 days	Costotransversectomy	Full recovery
Law <i>et al.</i> ^[6]	71/M	C1–C3	6 days	Conservative	Full recovery
Lucchesi <i>et al.</i> ^[7]	48/F	T1–T7	No deficit	Conservative	Full recovery
Ninomiya <i>et al.</i> ^[8]	68/F	C2–T4	No deficit	Conservative	Full recovery
Sathirapanya <i>et al.</i> ^[9]	62/M	C7–T4	2 h	Conservative	Full recovery
Schwartz <i>et al.</i> ^[10]	80/F	C2–T2	No deficit	Conservative	Full recovery

symptoms/presentation between patients with SSEH vs. SAH [Table 1].^[1,5-10]

Risk factors for SSEH include anticoagulant therapy, aspirin, vascular malformation, venous epidural plexus defects, hypertension, pregnancy, thrombolysis, physical exertion, chiropractic spinal manipulation, and hematological disorders.^[4,6,9] Our patient was receiving clopidogrel that likely contributed to the progression of her hematoma.

Delays in diagnosing SSEH increase the risks of permanent neurological deficits.^[1,4] In the present patient, the first and secondary brain CT scans and LP were negative for SAH. She should have undergone earlier emergent cervical CT scan or MR studies of the cervicothoracic spine to diagnose SSEH.

SSEH requires emergent surgery to avoid rapid neurological deterioration. In our patient, the MR findings documented a subacute C3–T3 epidural hematoma, but the surgical delay of 36 hours resulted in an irreversible neurological injury/quadriplegia.

CONCLUSIONS

Patients with SSEH rarely present with severe headache and neck pain/stiffness. SSEH must rapidly be differentiated from a SAH, and MR studies of the neuraxis must be performed to localize and then surgically treat the SSEH to avoid permanent neurological injury.

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Conflicts of interest

There are no conflicts of interest.

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