

Development of Cervical Subarachnoid Hematoma Following Coronal Artery Stenting for Angina Pectoris

—Case Report—

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Abstract

A 78-year-old man presented with a rare massive subarachnoid hematoma (SAH) in the cervical spine after coronary stenting for angina pectoris. Chest pain and electrocardiographic changes were resolved after administration of coronary dilator and coronary stenting, but shoulder pain persisted. At 6 hours after stenting, left hemiparesis was found with deteriorated shoulder pain. Computed tomography and magnetic resonance imaging revealed massive SAH in the cervical spine. Emergent hematoma evacuation with laminoplasty was performed because of rapid progression of the hemiparesis and pain. The hemiparesis was ameliorated after surgery. Spinal hematoma should be considered in the differential diagnosis in patients receiving anti-platelet or anti-coagulant drugs with rapid progression of pain.

Key words: referred pain, spine, subarachnoid hematoma

Introduction

Subarachnoid hematoma (SAH) in the cervical spine without vascular lesion is rare. We describe a case of massive SAH in the cervical spine after coronary stenting for angina pectoris.

Case Report

A 78-year-old man suffering pain in the left shoulder and chest visited the department of cardiovascular internal medicine of our institute. He had been an outpatient for angina pectoris with coronary artery bypass for 7 years. He was taking oral anti-platelet medication. On admission, cardiac electrography (EKG) demonstrated ST depression. After administration of coronary dilator, his chest pain disappeared with normalization of ST level on EKG, although his shoulder pain persisted. Computed tomography (CT) excluded aorta dissection, and emergent coronary angiography revealed 90% stenosis of the left circumflex artery. Drug eluting stent (Cypher Stent; Cordis Corp., a Johnson & Johnson company, Bridgewater, New Jersey, USA) was inserted with efficient dilation of the lesion, but his shoulder pain gradually exacerbated. Systemic heparinization and additional anti-platelet drug administration was started. His blood pressure was unstable with peak values of 190/110 mmHg. At 6 hours after stenting, left hemiparesis (manual muscle test [MMT] 4/5) and deteriorated shoulder pain occurred, so he was referred to

our department.

On admission, he had no sensory dysfunction with normal level of tendon reflexes. Head CT revealed scattered lesions of SAH (Fig. 1A–C). Head magnetic resonance (MR) imaging revealed SAH and a high signal intensity lesion in the right corona radiata on high b-value diffusion-weighted images (b value = 2000) (Fig. 1D), but no lesion in the right corona radiata was detected by other b-value diffusion-weighted imaging (b value = 0 or 1000) or fluid-attenuated inversion recovery imaging. MR angiography revealed no hemorrhagic vascular lesion. We initially thought of incidental cerebral infarct with SAH due to the medication, because his shoulder pain might have been referred pain from the coronary lesion and he just presented with pure hemiparesis. However, we also considered the possibility of spinal hematoma because the shoulder pain worsened with rapid progression of the left hemiparesis (MMT 2/5). Spinal CT and MR imaging revealed spinal hematoma (Fig. 1E, F). Our diagnosis was subdural spinal hematoma based on the location of the hematoma on MR imaging. Spinal CT with contrast medium and three-dimensional CT (3D-CT) angiography showed no vascular lesion in the spine.

Emergent decompression was performed because of the deterioration of the hemiparesis. Laminotomy (C3–C5) exposed high tension dura without epidural hematoma. After dural incision, thick SAH appeared (Fig. 2A) without hematoma between the dura and arachnoid. We confirmed that the spinal cord was decompressed and pulsative after hematoma removal without vascular lesions. Then GoreTex sheet (0.3 mm thickness; W. L. Gore & As-

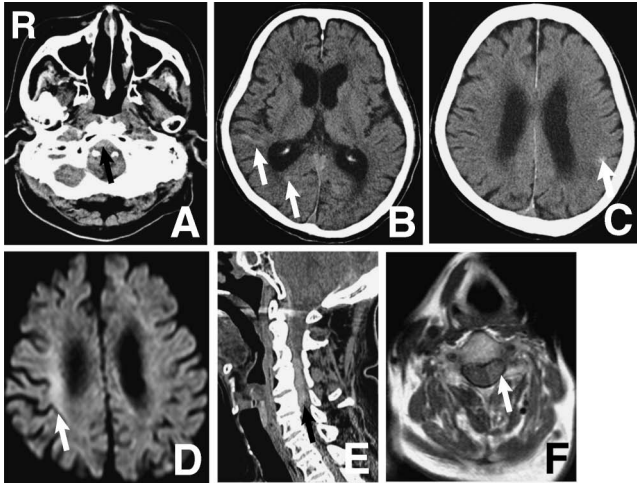


Fig. 1 A-C: Head computed tomography (CT) scans revealing intracranial scattered lesions of subarachnoid hematoma (arrows). D: Head magnetic resonance (MR) image showing a high signal intensity lesion in the right corona radiata (arrow) on high b-value diffusion-weighted imaging (b value = 2000). E, F: Spinal sagittal CT scan (E) and axial T₁-weighted MR image (F) revealing a spinal hematoma (arrow) and the dura as black rim outside the hematoma compressing the spinal cord. The location of the hematoma indicated subdural hematoma.

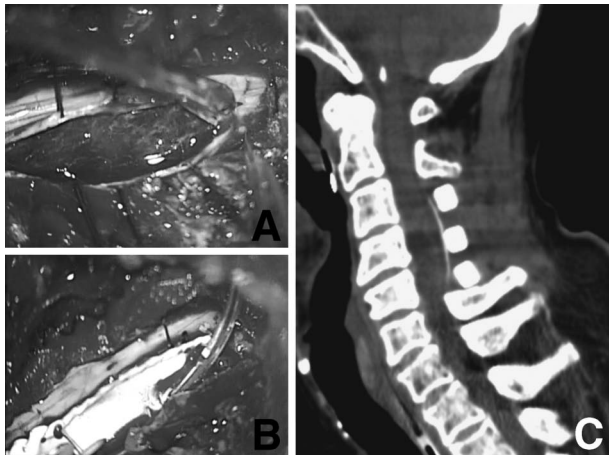


Fig. 2 A, B: Intraoperative photographs after dural incision showing thick subarachnoid hematoma (A), and dural plasty with GoreTex sheet with drainage tube below the dura (B). C: Postoperative sagittal computed tomography scan showing that the hematoma was removed with decompressed spinal cord.

sociates, Inc., Medical Products Division, Flagstaff, Arizona, USA) was used for decompressive dural plasty (Fig. 2B). A drainage tube was placed inside the dura with laminoplasty using Apaceram spacers (HOYA Corp., PENTAX New Ceramics Division, Tokyo) (Fig. 2C).

Immediately after surgery, his weakness ameliorated (MMT 3/5). At 2 months after surgery, he underwent walking training with a walker and transferred to another hospital for rehabilitation (MMT 5-/5).

Discussion

The present case of SAH in the cervical spine occurred after coronary dilation stenting for angina pectoris, with subsequent resolution of chest pain and abnormal EKG findings. After the treatment for angina pectoris, left hemiparesis appeared and deteriorated with consequent surgical treatment. Symptoms of hemiparesis with severe pain might be essential diagnostic indicators for spinal hematoma, although in this case the initial shoulder pain may have been related to the preceding spinal hematoma. In this case, intracranial scattered SAH and high signal intensity lesion on diffusion-weighted imaging (b value = 2000) were also confusing for the correct diagnosis. Clearly, spinal hematoma is an important differential diagnosis in patients with strong anti-platelet/anti-coagulant therapy.

Spinal hematoma is well known after administration of anti-coagulant or anti-platelet drugs.^{1,2,11} In our case, the medication was probably responsible for the deteriorated symptoms due to the increased size of the hematoma. Before admission, the patient had taken oral anti-platelet agent for 7 years. In addition, systemic heparinization for coronary stenting might have promoted SAH in the cervical spine and cranium. SAH in the cervical spine sometimes occurs from vascular lesions in the spine.¹⁰ Spinal angiography, 3D-CT angiography, and MR angiography should be considered for the hemorrhagic vascular lesion, although in our case, intraoperative findings and repeated 3D-CT angiography and MR angiography did not reveal any hemorrhagic lesions. Spontaneous spinal subdural hematoma is sometimes treated conservatively with subsequent recovery of the symptoms.^{5,8} However, hematoma evacuation and rehabilitation are required for many cases with rapid progression of symptoms.⁶ High b-value diffusion-weighted imaging provides better identification of extension of cerebral ischemia, although imaging artifact might increase.¹² In our case, the high signal intensity lesion in the right corona radiata on high b-value diffusion-weighted imaging (b value = 2000) might have been an artifact, which made the correct diagnosis difficult.

Review of 106 cases of non-traumatic acute spinal subdural hematoma found that 70% of patients presented with lumbar or thoracolumbar lesions.³ Over half of the patients were related to the spinal puncture and/or hemostatic disability. Fourteen of 59 cases receiving surgical treatment presented with SAH with relatively poor prognosis. The onset and location of our case was atypical, although anti-platelet/anti-coagulant agents and coronary intervention were risk factors for spinal hematoma. Neuroradiological findings of spinal subdural hematoma were clarified in several reports.⁷ Because of the semicircular distribution of the subdural hematoma, the imaging finding is less conspicuous with caudocranial extension than epidural hematoma.⁹ Subdural hematoma is usually located ventrally,⁴ which might be due to the tight attachment of the dura to the posterior longitudinal ligament. In our case, CT revealed spinal hematoma on the ventral side in the canal with relatively long caudocranial extension.

MR imaging, especially axial T₁-weighted imaging, demonstrated the dura as a rim of low signal intensity and the hematoma extending to the left inside the dura.

The present rare case of SAH after coronary stenting for angina pectoris shows that we should always think of the possibility of spinal hematoma in patients with risk factors and likely symptoms.

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