Spontaneous spinal epidural hematoma developing after percutaneous coronary intervention: early diagnosis, early intervention, and good outcome

Perkütan koroner girişim sonrası gelişen spontan spinal epidural hematom: Erken tanı, erken girişim ve iyi sonuç

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Summary– A 56-year-old female patient hospitalized with diagnosis of acute coronary syndrome underwent early coronary intervention. Anticoagulant and antithrombotic treatment was administered, including acetylsalicylic acid, clopidogrel, and heparin in periprocedural period. Severe back pain and rapidly progressing paraplegia developed in early period of follow-up. The patient underwent surgery immediately after diagnosis of spontaneous spinal epidural hematoma (SSEH) causing pressure, and decompression was performed. The patient rapidly improved without recurrence through early diagnosis and early surgical intervention. A common problem encountered by interventional cardiologists is back pain in patients who have undergone interventions in the femoral region and have lain in the same position for an extended period. Clinical onset of SSEH includes similar complaints, a fact of which cardiologists should be aware. Early diagnosis and early intervention may provide a good outcome, as is reported in the present case.

Özet– Kliniğimize akut koroner sendrom tanısı ile yatırılan 56 yaşında kadın hastaya erken koroner girişim yapıldı. İşlem çevresi dönemde asetilsalisilik asit, klopidogrel ve heparini içeren antikoagülan ve antitrombotik tedavi verildi. İzlemin erken döneminde şiddetli sırt ağrısı ve ardından hızlı ilerleme gösteren parapleji gelişti. Basıya neden olan spinal epidural hematom tanısı konduktan sonra hızlı bir şekilde ameliyata alındı ve dekompresyon yapıldı. Erken tanı ve erken cerrahi müdahale ile hasta hızlı bir şekilde sekelsiz olarak olarak iyileşti. Femoral bölgeden girişim yapılan hastalarda uzun süre aynı pozisyonda yatmaya bağlı gelişen sırt ağrıları girişimsel kardiyologların sık karşılaştığı bir durumdur. Spontan spinal epidural hematom'un klinik başlangıcı da benzer şikayetler şeklindedir. Bu sebeple kardiyologların bu konuda dikkatli olmaları gerekir. Sunulan hastada olduğu gibi erken tanı ve erken müdahale oldukça iyi sonuçlar almamızı sağlayabilir.

Spontaneous spinal epidural hematoma (SSEH) is a rare condition.[1] It is rarely seen due to acute coronary syndrome and antithrombotic therapy given in the course of treatment,[2,3] and may lead to severe morbidity and mortality unless diagnosed and treated early.

In the present report, the case of a patient who fully recovered in a short period of time through early diagnosis and early treatment of SSEH that developed during treatment of myocardial infarction without ST elevation is described.

CASE REPORT

A 54-year-old female patient was admitted to emergency service with complaint of typical chest pain. Hypertension was a risk factor. Electrocardiography revealed sinus rhythm and precordial 1 mm ST depression. Troponin value was 0.4 ng/mL and the patient was hospitalized with diagnosis of myocardial infarction without ST elevation. Initial treatment included 300 mg of acetylsalicylic acid, 600 mg of clopidogrel, nitroglycerin and 50 mg of metoprolol. Angiography

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was performed, and sequential severe stenosis was detected in the left anterior descending artery (Figure 1a). Unfractionated heparin in the amount of 7000 IU was administered during procedure. Pre-dilation of lesions of the left anterior descending artery was achieved with 2.5x20 mm balloon, and 2.75x18 mm drug eluting stent and 3.0x9 mm of bare metal stents were then implanted. Procedure was completed successfully without complications (Figure 1b). Three hours after the operation, sheat was taken. Because leison's thrombus load was too much, aiming to start 6 hours after the operation, enoxaparin 60 mg bid sc was added to her treatment. Severe back pain and paraplegia developed in the 12th hour following the procedure. Neurology and neurosurgery clinicians were consulted. A fusiform mass lesion measuring 5x1 cm and placing significant pressure on the spinal cord at levels T5–7, suggesting hematoma, was observed on dorsal magnetic resonance imaging (Figure 2). No abnormalities were detected in coagulation parameters, including coagulation tests and platelet count, and bleeding diathesis did not occur. The patient underwent urgent surgery due to SSEH causing neurologic deficit, and laminectomy and decompression intervention was performed. Extra treatments for the hematoma were not performed due to the urgency of the surgery. Vascular anomaly was not observed. After post-operative bleeding stabilization, dual antiplatelet treatment was initiated. No pathologic findings were obtained in factor deficiency testing. The patient improved progressively after surgery, and paraplegia resolved by day 6 of follow up. Treatment was arranged, and the patient was discharged.

Abbreviation:

SSEH *Spontaneous spinal epidural hematoma*

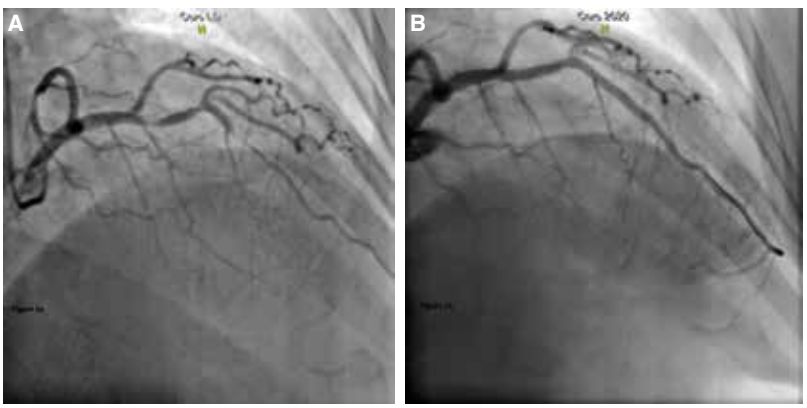

Figure 1. **(A)** Severe stenosis in the left anterior descending artery in coronary angiography; **(B)** improvement of stenosis in left anterior descending coronary artery after percutaneous coronary intervention.

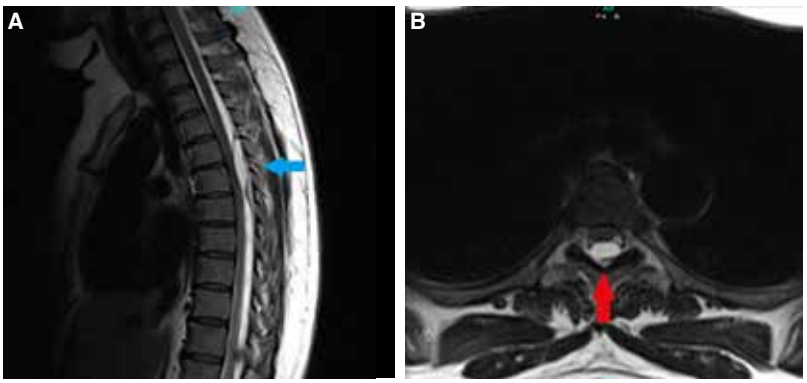

Figure 2. **(A)** T2-weighted sagittal dorsal magnetic resonance images. A posterior epidural mass lesion at levels T5–7 (arrow); **(B)** T2-weighted axial dorsal magnetic resonance images. A posterior epidural mass lesion at T5 level (arrow).

DISCUSSION

Antithrombin agents are the mainstay in management of acute coronary syndrome. The most common side effect of antithrombin agent use is bleeding, the most threatening of which is surely intracranial hemorrhage. Another complication is SSEH, which, while very rare, should not be overlooked. SSEH results from antithrombin agent use and leads to permanent neurologic injury which may lead to mortality. SSEH is a hematoma that develops without underlying disease or an underlying traumatic cause such as trauma, lumbar puncture, or epidural intervention. It is an urgent clinical condition which may lead to permanent neurologic deficit due to spinal cord compression. SSEH usually develops due to hemorrhagic diathesis, anticoagulant use, disc herniation, hypertension, Paget's Disease, tumor, or vascular abnormalities,[4–6] and the incidence of SSEH in the general population is estimated to be 1:1 million.[1] Hemorrhages in the spinal epidural space are believed to arise from epidural veins, and a silent clinical interval may be seen between the onset of symptoms like neck or back pain and neurologic deficit.[7]

The most common clinical presentation of SSEH is severe pain with abrupt onset in the neck or back region, followed by progressive neurologic deficit developing due to spinal cord compression.[8] The clinician should consider spinal hematoma with potential spinal cord compression in the presence of acute back or neck pain and neurologic deficit. Magnetic resonance is the most useful diagnostic tool, as demonstrated in many cases.[6,8] Standard approach is surgical evacuation of the hematoma following rapid diagnosis, providing detailed information about underlying cause, extension of the hematoma, and the compression on spinal cord. Early surgical decompression is recommended treatment method in these cases. The duration between onset of symptoms and surgical decompression is an important determinant for neurologic outcome.[8,9] Surgical decompression is recommended within 36 hours in patients with complete neurologic deficit and 48 hours in patients with incomplete neurological deficit.[10] Treatment may be managed by non-operative conservative approach in selected cases, a decision influenced by factors including the volume of the hematoma, the control of active hemorrhage, spinal cord flexibility, spinal canal width, and width of the hematoma along the epidural space.[11]

Though frequently experienced, pain in the back and lower back must not be overlooked in these cases. Presence of neurologic deficit may be determined with questions and physical examination, which are neither time-consuming nor expensive. In the present case, neurologic deficit accompanied pain at the 12th hour following the procedure, and the administration of medication preceded complete neurologic improvement following early diagnosis and quick surgical decompression. Clinical suspicion, and early diagnosis and treatment ensure complete recovery without permanent injury.

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