Paraplegia after percutaneous iliac stenting: How did that happen?

Perkütan iliyak stent sonrası olan parapleji: Bu nasıl oldu?

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Summary– Paraplegia after percutaneous iliac angioplasty is very rare, and is typically associated with spinal cord ischemia (SCI). Presently described is a case of SCI and paraplegia developing after bilateral iliac stenting. This complication may be caused by a change in spinal blood flow in patients with diffuse atherosclerosis, and should be kept in mind.

Paraplegia after an abdominal aortic or iliac procedure is a rare and unpredictable complication. The general neu-

Abbreviations:

CT	Computed tomography
EMG	Electromyography
MRI	Magnetic resonance imaging
SCI	Spinal cord ischemia

rological risk for endovascular and open abdominal aortic surgery has been reported to be $\leq 1\%$.^[1] Paraplegia is frequently due to spinal cord ischemia (SCI), which has a high rate of morbidity. The main cause of SCI-associated paraplegia is an intervention in the presence of atherosclerosis.^[2] Aortic cross-clamping during endovascular or surgical treatment of an abdominal aortic aneurysm may be a mechanism of SCI. ^[3] Percutaneous intervention to the iliac artery may also occasionally cause SCI.

This is a description of a case of a patient who developed paraplegia after percutaneous iliac stenting and a discussion of potential etiological factors.

CASE REPORT

A 68-year-old male with known coronary artery disease (history of coronary bypass) presented at the cardiology clinic with claudication. Bilateral iliac stenosis was detected on lower extremity Doppler ultrasonography images. Peripheral angiography re**Özet**– Perkütan iliyak anjiyoplasti sonrası spinal kord iskemisi (SKİ) ve parapleji çok nadir bir komplikasyondur. İki taraflı iliyak stent sonrası gelişen SKİ ve paraplejinin olduğu nadir bir olguyu sunarak, yaygın aterosklerozu varlığında spinal kan akımının değişimi ile böyle bir komplikasyonun olabileceğinin akılda olması gerektiğini vurgulamak istedik.

vealed total occlusion of the right main iliac artery and a 90% lesion in the left iliac artery.

An antegrade pass of the total occlusion using the left femoral approach failed. A retrograde pass was successful using puncture through the collateral filling of the right femoral artery with a left femoral access opaque injection and right iliac stenting. The patient was discharged 1 day after the procedure (Fig. 1). One week later, the patient was re-hospitalized for left iliac arterial angioplasty. A retrograde left femoral arterial access was planned, but no pulse could be obtained in the left femoral artery. Following a right femoral artery puncture, peripheral angiography was performed and the right iliac stent was observed to be patent, but the 90% lesion of the left iliac had become a total occlusion. The left iliac lesion could not be passed with an antegrade approach via the right femoral artery, and a retrograde approach with a puncture through the collateral filling of the left femoral artery also failed. An Agilis Steerable Introducer (St. Jude Medical, Inc., St. Paul, MN, USA) was then selected to provide better back-up. Using the right femoral approach, the left main iliac artery was engaged with the introducer and the total occlusion was successfully passed. Superficial femoral artery angioplasty was performed with a

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SeQuent Please OTW peripheral drug-coated balloon (B. Braun Melsungen AG, Hessen, Germany) and the main iliac artery was stented using a 10 mmx90 cm VascuFlex peripheral self-expanding stent (B. Braun Melsungen AG, Hessen, Germany) (Fig. 2).

Twenty minutes after being returned to bed, the patient suffered from pain, motor and sensory deficits in the left leg. No pulse could be obtained in the left femoral, popliteal, or dorsalis pedis arteries. A control peripheral angiography revealed that the left iliac stent was thrombosed. Balloon dilatation was performed and distal arterial blood flow was achieved (Fig. 3). Intravenous heparin and an infusion of iloprost were initiated. Administration of acetylsalicylic acid + clopidogrel + statin from the first stent procedure was continued. During follow-up in the intensive care unit, the patient developed paraplegia with no distal arterial pulse.

After a neurology consultation, cranial computed tomography (CT) and magnetic resonance imaging (MRI) were performed but no cerebral pathology was determined. Hemodialysis treatment was applied 4 times due to contrast-induced nephropathy. Based on MRI and electromyography (EMG) results, methylprednisolone 4 mg was added as a steroid regimen to prevent potential spinal cord and lumbar neural plexus injury. On the fifth day after the event, EMG was performed to evaluate the sacral spinal cord. A bilateral lower limb motor deficit was found that was thought to be a neural injury. As there was no pathology seen in the aortography recorded during the angioplasty, a rare variation in the spinal blood supply originating from the internal iliac artery was considered as an explanation. CT angiography was performed once kidney functions improved. No spinal arterial variation was observed, but diffuse atherosclerosis was determined in the thoraco-lumbar aorta. The bilateral stents were patent (Fig. 4a-d). The angiograms were reviewed, and it was observed that the left internal iliac artery was patent before the procedure, but the right internal iliac artery could not be visualized clearly (Fig. 4e).

After 20 days of physiotherapy, motor function was almost totally restored in the right leg, but monoparesis remained on the left side. On the 30th day, the patient was transferred to the physical therapy and rehabilitation clinic after a cardiology follow-up.

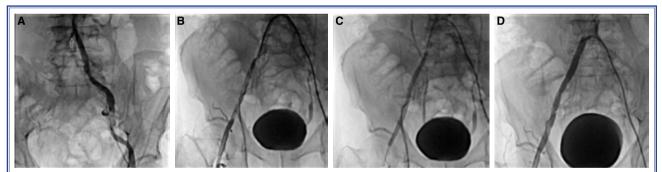


Figure 1. (A) The white arrow shows the right main iliac segment with total occlusion; (B) The occlusion was passed with an 0.035inch guidewire; (C) After balloon angioplasty of the occluded segment, (D) a stent was implanted and blood flow was restored.

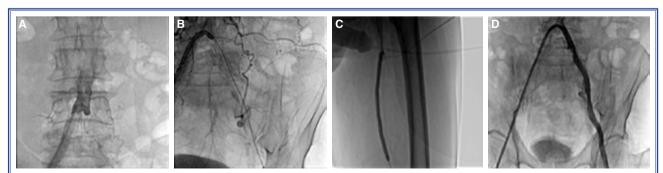


Figure 2. (A) Total occlusion seen in the left main iliac artery; (B) The occlusion was passed with a 0.035-inch guidewire with a steerable introducer; (C) After balloon angioplasty of the occluded segment, (D) a stent was implanted and blood flow was provided with non-critical residue.

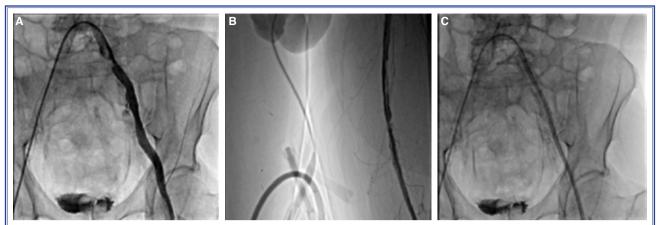


Figure 3. (A) The proximal stent thrombosis that separated after passage of the wire; (B) Thrombosis in the distal femoral artery; (C) Repetition of balloon angioplasty.

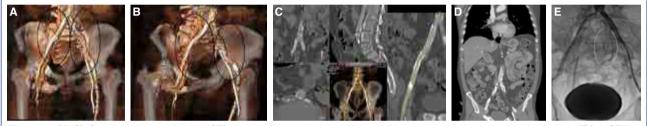


Figure 4. (A-C) Computed tomography (CT) images illustrating the bilateral iliac stents (marked with black circles); (D) A CT image revealing diffuse atherosclerosis in the lumbar aorta; (E) The left internal iliac artery can be seen to be open on an angiogram image recorded before the procedure.

DISCUSSION

Neurological events after abdominal aortic and iliac artery interventions are not frequent. This catastrophic event was a sequela of SCI. SCI is unpredictable, may be permanent, and may be a cause of major morbidity. The total neurological risk of abdominal aortic and iliac artery interventions ranges between 0% and 1%,^[3] and the risk associated with percutaneous procedures is so small that it is not known.^[4]

The unpredictability of SCI is due to the complexity and variability of the blood supply of the spinal cord.^[5] The main blood supply to the distal spinal cord and nerve roots comes from the lumbar and sacral arteries. The main branch of those is the artery of Adamkiewicz, which usually originates from the suprarenal intercostal arteries or the infrarenal lumbar artery.^[6] If there is atherosclerotic disease in the artery of Adamkiewicz, the lumbar arteries and branches from the internal iliac arteries become the main blood supply to the distal spinal cord and nerve roots. If the internal iliac arteries are damaged, the blood supply to the lower part of the spinal cord is halted, which can cause SCI.^[7]

The MRI and EMG results showed bilateral lower extremity motor and sensory deficits associated with spinal cord and lumbar plexus injury, with more observed on the left side.

This patient had undergone coronary bypass, which is associated with atherosclerotic disease in the artery of Adamkiewicz, indicating that the main blood supply was likely provided by the internal iliac arteries. The peripheral angioplasty of the right iliac arteries may have led to decreased blood flow in the lower spinal cord and interruption of the main blood flow in the lower spinal cord may have been caused by the left iliac artery angioplasty. This could explain the paraplegia after iliac stenting.

Acute thrombosis of the left iliac stent also increased the damage to the left femoral nerve, which could explain the better clinical improvement of the right leg in comparison with the inadequate healing of the left leg during the follow-up period. There is a lack of clinical data related to specific therapeutic regimens for patients with SCI. Anticoagulant or antiplatelet medications can be useful for atherosclerotic vascular pathologies. Although hypothermia and steroids can be effective, the treatment must begin within a very short time after the event. Steroids may provide a benefit due to the protective effect on cell function and reduction of the free radicals that occur after SCI. Naloxone may reduce neurological sequelae by increasing spinal blood flow and preventing calcium from entering nerve cells.^[8]

In the current case, a heparin infusion and a dual antiplatelet regimen were initiated after the event, as well as steroid therapy. These treatments may have contributed to the healing observed in this patient.

Conclusion

Paraplegia is not generally expected as a complication after percutaneous iliac angioplasty. Nevertheless, it must not be forgotten that such a significant complication may be caused by the change in spinal blood flow in patients with diffuse atherosclerosis. In the post-procedural period, any sensory or motor impairment or unexpected incontinence should bring SCI to mind.

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