A Rare but Serious Complication of Percutaneous Coronary Intervention: Spinal Cord Embolism

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Abstract

Background/Objective: Many atherothrombotic complications are associated with coronary angiography. Spinal cord embolism with high morbidity and mortality is one of these complications.

Methods: Case report.

Results: A 65-year-old woman was admitted to the hospital with acute myocardial infarction. Immediately after coronary angiography, she complained of paresthesia and paraparesis of her legs. Magnetic resonance imaging (MRI) detected hyperintensity at the level of the conus medullaris. Antiaggregant therapy and a physiotherapy program continued. After 2 months, clinical and MRI findings had improved.

Conclusions: Invasive procedures such as coronary angiography can lead to serious atherothrombotic complications.

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Key Words: Spinal cord injuries; Spinal embolism; Myocardial infarction; Coronary angiography, complications; Thromboembolism; Paraparesis; Paraplegia

INTRODUCTION

Acute spinal cord ischemia is a rare pathology with high morbidity and mortality, accounting for 1.2% of all apoplexy cases (1,2). It has various clinical presentations because of many etiologic factors (3). A few studies can be found in the literature conducted with small patient samples investigating the etiologies, long-term therapies, and outcomes of patients with spinal cord infarction (1,3). Although aortic surgery and anesthesia methods have been frequently reported as iatrogenic causes, a few cases occurring after coronary angiography have been reported (4–6). In these, the authors discuss spinal cord embolism in patients undergoing workup for acute myocardial infarction and describe potential problems during management of these patients.

Case Report

A 65-year-old woman was admitted to the emergency department for ongoing chest pain that had started 18 hours earlier. The patient had no history of hypertension or diabetes mellitus. The patient was referred to the

catheterization laboratory because of ongoing chest pain and ST-segment elevation on precordial leads of the electrocardiogram. No neurologic disorder had been previously reported. Premedication included the following: unfractionated heparin, 7,500 IU; acetylsalicylic acid (ASA), 300 mg; clopidogrel, 600 mg. Coronary angiography was attempted from a right femoral artery approach with an 8F guiding catheter; however, the catheter turned to the right femoral artery at the iliac artery bifurcation. The procedure was again attempted with a left femoral arterial approach, but this time, the guiding catheter did not pass from the iliac artery to the abdominal aorta. The catheter was successfully inserted from the bifurcation to the abdominal aorta by gentle manipulation, and the procedure was continued through use of an exchange catheter. The patient was transferred to the coronary intensive care unit after unsuccessful percutaneous coronary intervention.

The patient complained of paresthesia and of being unable to move her lower extremities immediately after the procedure. Blood pressure was 120/70 mmHg, heart rate was 90 beats/min, and peripheral pulses were normal. Neurology consultation was ordered because of ongoing complaints and new onset of urinary incontinence. During neurologic examination, the patient was alert and cooperative, and upper extremity reflexes and muscle strength were normal. In the lower extremities, deep tendon reflexes were absent, and muscle strength was bilaterally 0/5 in all key muscles of

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Table 1. Manual Testing of Muscle Strength of Key Muscles Before and After Rehabilitation

Key Muscles	Initial		After Rehabilitation	
	Right	Left	Right	Left
Upper extremity				
Elbow flexors (C5)	5/5	5/5	5/5	5/5
Wrist extensors (C6)	5/5	5/5	5/5	5/5
Elbow extensors (C7)	5/5	5/5	5/5	5/5
Finger flexors (C8)	5/5	5/5	5/5	5/5
Finger abductors (T1)	5/5	5/5	5/5	5/5
Lower extremity				
Hip flexors (L2)	0/5	0/5	3/5	3/5
Knee extensors (L3)	0/5	0/5	3/5	3/5
Ankle dorsiflexors (L4)	0/5	0/5	2/5	2/5
Long toe extensors (L5)	0/5	0/5	1/5	1/5
Ankle plantarflexors (S1)	0/5	0/5	2/5	1/5

the lower extremities by manual muscle testing (Table 1). Patient had loss of light touch and pin prick sensation at and below the L2 level, but vibratory and positional senses were preserved throughout. Anal superficial touch examination (S4–S5), light touch, and pin-prick revealed hypoesthesia (Table 2). The patient had no voluntary anal contraction or bulbocavernous reflex. It was presumed that the symptoms were caused by a spinal cord embolism at the level of the medulla spinalis as a complication of the cardiac intervention study.

Magnetic resonance (MR) and diffusion MR imaging performed 1 day after the procedure showed an acute ischemic lesion in the region of the conus medullaris exhibiting hyperintensity and lack of diffusion in T2A (Figure 1). Widespread atherosclerotic structure and plaque were also seen in MR angiography (Figure 2). The patient was diagnosed with L2 ASIA B anterior cord syndrome secondary to angiographic intervention (7). The level of neurologic impairment was determined to be the Adamkiewicz artery. Cortisone was added to anti-aggregant therapy (ASA, 100 mg/d; enoxaparin, 60 mg/d; clopidogrel, 75 mg/d). Early rehabilitation (passive

range of motion exercise for lower extremities, positioning) was initiated in addition to medical treatment. Cortisone therapy was gradually decreased and stopped within 5 days.

The patient, whose feeling of sensation improved in 5 days but without improvement in motor strength, was transferred to the rehabilitation unit. A physiotherapy program was administered, including strengthening of the upper extremities, passive range of motion of the lower extremities, and seating balance exercises before and after active assistive range of motion, standing balance in parallel bars, and progressive ambulation for 2 months. She was monitored for fatigue, blood pressure, and heart rate during rehabilitation. The patient showed progressive neurologic improvement and was discharged with neurologic level L2 ASIA C and was capable of short length ambulation with the aid of a walker and bilateral ankle foot orthosis after 2 months of treatment. She had improvement in light touch and pin prick sensation. Superficial touch examination, light touch, and pin-prick tests were normal to the L2 dermatomal level; below L2, dermatomal level hypoesthesia was found and anal superficial touch was normal (Table 2). Muscle strength of key muscles was 3/5 at the hip flexors and knee extensors, but this value was 1 to 2/5 at the other muscles (Table 1). The patient had voluntary anal contraction/bulbocavernous reflex. We also noted diminution of the infarction area in an MR image administered at 2-month follow-up (Figure 3).

DISCUSSION

One anterior and two posterior spinal arteries originate from the vertebral artery and supply the spinal cord vertebrae; however, infarction frequently occurs in the region of the anterior spinal artery (8). Although the anterior spinal artery originates from the vertebral artery in the region of the upper cervix, it originates from radicular branches of the cervical artery and intercostal and lumbar arteries at lower levels (9). The major anterior radicular artery originating from any level between T5 and L2 is named the Adamkiewicz artery (10).

Neurovascular syndromes have various clinical presentations depending on the level of infarct area at the spinal cord. Anterior spinal artery syndrome in particular

Table 2. Sensory Evaluations Before and After Rehabilitation

Dermatomal Level	Initial				After Rehabilitation			
	Light Touch (R)	Light Touch (L)	Pin Prick (R)	Pin Prick (L)	Light Touch (R)	Light Touch (L)	Pin Prick (R)	Pin Prick (L)
C1–L2	2/2	2/2	2/2	2/2	2/2	2/2	2/2	2/2
L3–S1	1/2	1/2	1/2	1/2	1/2	1/2	1/2	1/2
S2–S3	0/2	0/2	1/2	1/2	1/2	1/2	1/2	1/2
S4–S5	1/2	1/2	1/2	1/2	2/2	2/2	2/2	2/2

R, right; L, left.

Figure 1. Spinal MRI showing hyperintensity at the level of conus medullaris, 1 day after atheroembolism.

may present with weakness, backache, areflexia, loss of sensation, and autonomic dysfunction (11). This patient had loss of light touch and pin prick sensation, loss of lower extremity muscle strength, and urinary incontinence, with no problems in the upper extremities, respiratory system, or with blood pressure. Vibratory and position senses were also preserved. Therefore, it was predicted that the infarction was at the level of the Adamkiewicz artery or below. MR imaging had already detected that the infarction was at the level of the conus medullaris.

Nedeltchev et al (3) identified different etiologies for spinal cord syndrome. Atherosclerosis has been defined as the main cause, accounting for 33.8% of cases. Risk for atheromatous embolism has been shown to be increased by atherosclerosis of the thoracic and abdominal aorta, by coronary and peripheral arterial disease, and by advanced age (>60 years) (12). Many factors trigger atheromatous embolism, including spontaneous occurrences, endovascular procedures, surgical procedures, anticoagulation, thrombolytic treatment, and trauma (12). We hypothesized that mechanical trauma secondary to catheter manipulation at the level of the iliac artery had caused plaque to rupture, leading to embolism. This speculation was also supported by the fact that the patient experienced neurologic symptoms after the procedure in the catheter laboratory and by the finding

Figure 2. Thoracoabdominal aorta MR angiography showing diffuse atherosclerosis.

of severe atherosclerosis in the thoracic and abdominal aorta demonstrated on MR angiography.

Spinal cord embolism is diagnosed by MR imaging; however, in the study by de la Barrera et al (1) of 36 patients with acute spinal cord ischemia syndrome, MR imaging conducted in the acute phase of the disease showed typical cord enlargement in T1 and increased linear signal intensity in T2 imaging in 73% of patients. MR imaging was administered 1 month later in the remaining patients, and one half still had normal spinal cord images. However, in MR images and diffusion MR images taken in the acute phase of this patient, increased signal intensity was detected at the conus medullaris level in T2 imaging; this finding finally confirmed the diagnosis of spinal cord infarction.

Data are lacking in the literature about management of these patients; however, avoidance of hypotension, amelioration of other risk factors, and administration of antiaggregants (ASA and clopidogrel) and steroids are recommended for conservative treatment. Besides these routine recommendations, the potential side effects of steroid treatment during acute myocardial infarction should be taken into consideration. Steroids can increase the risk of myocardial rupture by retarding the recovery period. Retention of fluid and sodium retention may result in congestive symptoms, especially in patients with heart failure. As for this patient, the extent of myocardial

Figure 3. Spinal MRI showing improved hyperintensity at the level of conus medullaris 2 months after atheroembolism.

damage was assessed as moderate according to echocardiographic examination, and no congestive symptoms caused by left ventricular dysfunction were observed. For these reasons, after consultation with both a neurologist and a cardiologist, steroid therapy was initiated and continued for 5 days. In addition, optimal antiaggregant treatment was also continued because of acute myocardial infarction.

Another important issue is the rehabilitation program. It is generally accepted that a patient should also be transferred to a rehabilitation unit along with other therapies. In studies published by Cheshire et al (10) and Iseli et al (13) following 44 and 28 patients, respectively, who engaged in rehabilitation for 1.2 ± 2 years and 6 months, it was emphasized that significant clinical improvement resulted and that rates of walking independently or with support were 38% and 25%. In the series by de la Barrera et al (1) and Nedeltchev et al (3), with 36 and 57 patients at follow-up of 19 ± 30 months and 4.5 ± 4 years, respectively, these rates were 43% and 71%, respectively (on return). Also, Iseli et al (13) found similar recovery rates between traumatic and ischemic spinal cord injuries. However, in these studies, they evaluated all patients with ischemia without considering the level of spinal cord injury. Therefore, comparing the

outcome of our case with these outcome results is difficult.

Although rehabilitation has been recommended for patients with spinal cord embolism, risk of myocardial ischemia was a major problem for this patient as a result of acute myocardial infarction and failed coronary intervention. Because of the risk for myocardial ischemia, doses of anti-ischemic medications such as β blockers, statins, nitrates, and ASA were optimized before rehabilitation. Once angina pectoris was controlled with medical therapy, early rehabilitation was started in the coronary intensive care unit, with passive range of motion exercise preferred in this phase. Nevertheless, no anginal episodes were seen in early rehabilitation, and the patient went to the second phase in the rehabilitation department. The cardiologist advised shorter physical exercise time than normal in the second phase and to continue anti-ischemic therapies throughout the rehabilitation. Finally, the patient showed significant improvement in motor and sensorial potencies. She had functional ambulation with the aid of a walker and bilateral ankle-foot orthoses after 2 months.

CONCLUSION

Acute spinal cord ischemia syndrome is a potentially fatal condition that can cause permanent disability. Therefore, invasive procedures should be performed with caution in patients of advanced age with probable atherosclerosis. Patients need multidisciplinary management by a cardiologist, a neurologist, and a physiatrist. Early rehabilitation carefully monitored and with appropriate medical treatment is important for patients with spinal cord ischemia.

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