Paraparesis after Primary Percutaneous Coronary Intervention for ST-segment Elevation Myocardial Infarction: Combined Uncommon Complications of Acute Aortic Syndrome in a Patient

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Abstract

Acute aortic syndrome complicated by both ST-segment elevation myocardial infarction (STEMI) and spinal ischemia is exceedingly rare. We herein report the case of a 66-year-old man who presented with paraparesis after primary percutaneous coronary intervention for STEMI. He was found to have an intramural hematoma of the ascending aorta and a severe dissection in the descending aorta, which led to both STEMI and paraparesis.

Key words: aortic dissection, aortic intramural hematoma, myocardial infarction, paraplegia

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Introduction

Acute aortic syndrome (AAS) is a life-threatening medical emergency associated with high rates of morbidity and mortality. The symptoms of AAS can be variable and may mimic those of more common conditions such as myocardial infarction, stroke, shock, and paraplegia (1), which could cause difficulty to diagnose early and treat in a clinical settings. We herein report a patient with an AAS presenting as paraplegia with ST-segment elevation myocardial infarction (STEMI), after receiving treatment with percutaneous coronary intervention (PCI) alone.

Case Report

A 66-year-old man was admitted to our emergency department because of the sudden onset of a compressing, very severe, and lasting chest pain 1 hour before. The electrocardiogram results were compatible with a diagnosis of a STEMI of the inferior wall (Fig. 1). We decided to perform urgent coronary angiography without further evaluation, which revealed a critical diffuse occlusive lesion in the ostium to the middle portion of the right coronary artery (RCA), with a thrombolysis in myocardial infarction (TIMI) flow grade 0 (Fig. 2A). After predilatation with a 2.0×15 mm balloon and aspiration of a piece of red thrombus with Thrombobuster II aspiration catheter (Kaneka Medics, Tokyo, Japan) (Fig. 2B), we performed an intravascular ultrasound (IVUS) examination, which showed a large extensive intramural hematoma with no visible intimal flap (Fig. 2C, D). Although we did not notice the underlying pathophysiology of coronary artery occlusion at first, two drug-eluting stents (size, 4.0×24 mm and 4.0×28 mm) were implanted in the RCA with an optimal angiographic result and TIMI 3 flow. After PCI, his chest pain symptoms completely subsided, and the preexisting ST elevation normalized within the next few minutes. A transthoracic echocardiography showed no definite abnormalities in the ascending aorta, valve, regional wall motion, and global left ventricular function in conventional echocardiographic views (data not shown). He was admitted to the coronary care unit (CCU) where he was given dual antiplatelet drugs with unfractionated heparin and ordered to take absolute bed rest for two

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Figure 1. Initial ECG showing ST elevation in leads II, III, and aVF; reciprocal changes in leads I, aVL, and V2-5; and junctional escape rhythm. ECG: electrocardiogram

Figure 2. Coronary angiography images showing a diffuse occlusive lesion in the proximal to mid segment of the RCA (A) and after predilatation with a balloon (B). (C, D) Intravascular ultrasound images showing an intramural hematoma in the media of the arterial wall, which resulted in the compression (arrows) of the lumen from the RCA ostium (C) to distally (D). H: hematoma
tomographic (CT) angiography confirmed the intramural STEMI was highly suspected. The subsequent computed mural hematoma formation in the RCA ostium site and no angiography and IVUS showed a significant, extensive intra-subacute stage of spinal cord infarction (Fig. 3). Coronary catheter manipulation injury such as an embolism from an atherosclerotic plaque. We performed spinal magnetic resonance imaging (MRI), and reviewed the coronary angiography and IVUS to differentiate the cause of paraparesis. Spinal MRI scans revealed a high signal intensity in the conus medullaris on T2-weighted and diffusion-weighted images. The contrast-enhanced T1-weighted image showed enhancement of the conus medullaris on T2-weighted and diffusion-weighted images. The contrast-enhanced T1-weighted image showed enhancement of the conus medullaris. These findings suggested a subacute stage of infarction.

days. However, he started to complain about difficulty in voiding during his CCU stay, and was thus inserted with a Foley urine catheter. Two days after admission, we encouraged ambulation around the ward. However, he complained of weakness and numbness in the legs, thus making it unable for him to walk. We investigated the possibility of a catheter manipulation injury such as an embolism from an atherosclerotic plaque. We performed spinal magnetic resonance imaging (MRI), and reviewed the coronary angiography and IVUS to differentiate the cause of paraparesis. Spinal MRI scans revealed a high signal intensity in the conus medullaris on T2-weighted and diffusion-weighted images. The contrast-enhanced T1-weighted image showed enhancement of the conus medullaris. These findings suggested a subacute stage of infarction (Fig. 3). Coronary angiography and IVUS showed a significant, extensive intramural hematoma formation in the RCA ostium site and no definite plaque rupture site. AAS complicated with an inferior STEMI was highly suspected. The subsequent computed tomographic (CT) angiography confirmed the intramural hematoma of the ascending aorta, which may be retrograde extended from the subclavian tearing site, below which there was a severe dissection of the external iliac arteries complicated with a pleural hematoma around the intrathoracic aorta (acute Stanford type A aortic dissection, Fig. 4A-C). We therefore carried out the strict control of the patient’s blood pressure and stopped the anticoagulation treatment except for aspirin and clopidogrel. The treatment decision in this situation was very complicated. Although an intramural hematoma involving the ascending aorta and dissection in the descending aorta with hemotherax and malperfusion symptom should be surgically treated in general, the patient repeatedly refused the operation because he was hemodynamically stable without symptoms of myocardial ischemia except for the paraparesis. He considered the possibility that surgery might not provide any neurologic improvement, in addition to the high risk of perioperative mortality and morbidity. Therefore, we decided to perform treatment with medical therapy with frequent imaging follow-ups. Fortunately, a partial improvement of the neurologic complications of AAS was observed after a hospital-based rehabilitation, and the follow-ups have been uneventful to date. After three months, the CT follow-up imaging showed that the intramural hematoma surrounding the ascending aorta and hemotherax had been completely absorbed and the dissection of the descending aorta showed no change in diameter (Fig. 4D-F).

Discussion

AAS complicated by both STEMI and spinal ischemia is exceedingly rare. The International Registry of Acute Aortic Dissection reported the incidences of new myocardial infarction and spinal cord ischemia to be 3.2% and 2.7%, respectively (1, 2). Our literature search did not yield any report describing a concurrence of STEMI and neurologic complication of AAS in a patient. In patients with aortic dissection presenting with STEMI, because of clinical symptoms and signs such as typical chest pain with electrocardiographic findings suggestive of myocardial infarction alone, the correct diagnosis may be missed. This clinical scenario may lead to inappropriate thrombolytic or antiagulant therapy, resulting in hemorrhagic complications and mortality (3, 4). Nevertheless, both in cases of a missed or correct diagnosis, the Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease, 2010, recommend to perform PCI with the aim of stabilizing the myocardial ischemia (5). A careful interpretation of coronary angiography and IVUS imaging should have been made for an earlier correct diagnosis. Coronary angiography showed a diffuse long, smooth-margined stenotic lesion from the ostium to the proximal portion of the RCA, which might implicate another pathophysiology rather than a typical atherosclerotic plaque rupture lesion. Furthermore, the IVUS finding of a
After 3 months, the intramural hematoma and hemothorax became completely absorbed (D, E) and the dissection of the descending aorta showed no change in diameter (D-F).

Figure 4. Initial and follow-up contrast-enhanced computed tomography angiography images. Initially, an intramural hematoma was detected in the ascending aorta (arrowhead, A), which may be retrograde extended from the subclavian tearing site, below which a dissection of the external iliac arteries (B, C) complicated with a pleural hematoma (arrow, B) was observed. After three months, the intramural hematoma and hemothorax became completely absorbed (D, E) and the dissection of the descending aorta showed no change in diameter (D-F).

Long intramural hematoma along the whole length of the lesion was suspected to be a complication of AAS, although we did not find an intimal flap or tearing site. In this case, the STEMI could have occurred secondary to the compression of the RCA by the expanding intramural hematoma of the ascending aorta, which extended from the tearing site near the branching portion of left subclavian artery.

Paraplegia or paraparesis after primary PCI also is a rare manifestation. If a patient complains of neurologic symptoms after PCI, clinicians should thus consider many conditions in the differential diagnosis, such as embolism of the spinal artery, acute stroke, spinal hematoma due to bleeding complications, and concurrent neurologic diseases such as Guillain-Barré syndrome. One study series in 44 patients with spinal ischemia or infarction reported that AAS occurred in two patients (4.4%), which was not extremely rare (6). Therefore, evaluation for the cause of nontraumatic paraplegia or paraparesis must be included in aortic imaging studies such as CT. In some cases, only an imaging study can find AAS as a cause of paraplegia or paraparesis, especially in patients with painless aortic dissection with paraplegia (7).

The definitive surgical treatment of an intramural hematoma in the ascending aorta still remains controversial. The guidelines recommend, based on expert consensus, that surgical treatment of an intramural hematoma in the ascending aorta is similar to an aortic dissection (class II and evidence C) (5). However, Asian investigators have another recommendation with a more selective indication for surgical treatment, especially in the case of an intramural hematoma with a thickness of <10 mm. Because emergency surgical intervention did not provide any survival benefit to Asian patients with type A intramural hematoma, aggressive medical treatment therefore normally proposed, except in patients with complications including progression to aortic dissection (8). Furthermore, the neurologic symptoms of the present patient were steadily improving, and an incomplete surgical correction without saving the greater radicular artery of
Adamkiewicz was considered to potentially result in false lumen thrombosis, which could aggravate the spinal circulatory disturbance. Although the dissection of the descending aorta was approximately 50 mm size and it was accompanied by a hemothorax in this patient, the perioperative mortality or morbidity of a reconstruction surgery of the descending aorta is still high. In this situation, the clinical decision was very therefore difficult for both the physicians and the patient. Initially, we preferred to perform a surgical correction due to concerns about a potential rupture of the descending aorta. However, retrospectively, considering the small size of the intramural hematoma in the ascending aorta without coronary ischemia due to coronary intervention and the stable dissection of the descending aorta during 48 hours, although pleural hematoma was initially observed, intensive medical therapy and frequent regular CT follow-up might have been a better choice for treatment in terms of benefits and risks, compared with surgery, which has a high mortality and morbidity. In conclusion, the correct diagnosis of AAS has many potential pitfalls because AAS may mimic myocardial ischemia, stroke, heart failure, acute abdomen, and shock. Careful interpretation of the imaging results and a high index of suspicion in patients with STEMI is therefore critical. To make the right decision about the surgical treatment of AAS with multiple complications, the benefits and risks should therefore be considered in individual patients. We herein described a rare case of type A AAS presenting as STEMI and paraparesis.

The authors state that they have no Conflict of Interest (COI).

References