CASE REPORT

Paralytic ileus as the first presentation in type A acute aortic dissection

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Abstract: A 78-year-old female was referred to our hospital with a diagnosis of type A acute aortic dissection. There was a history of thrombosed aortic dissection six months prior and conservative management has been performed. Enhanced computed tomography showed type A acute aortic dissection with patent false lumen limited to the ascending aorta and ileus of the small intestine. Emergency hemiarch replacement was performed under mild hypothermic circulatory arrest and selective antegrade cerebral perfusion. Due to preoperative paralytic ileus, oral intake was initiated postoperative day four. Postoperative computed tomography revealed improvement of paralytic ileus. J. Med. Invest. 64 : 286-287, August, 2017

Keywords: paralytic ileus, first presentation, type A acute aortic dissection

INTRODUCTION

Type A acute aortic dissection (AAD) is a life-threatening disease which need an emergency operation. The overall mortality rate is up to 27% despite improvement of surgical strategy (1). The process of AAD is dynamic and it can occur anywhere along the entire aorta. Because vascular complications associated with involvement of each aortic branch are reported up to 33% of patients with AAD (2), broad clinical spectrum of presentation such as neurologic deficits, myocardial infarction or extremity ischemia is present (1). In this paper, we described paralytic ileus as an initial presentation in type A AAD.

CASE REPORT

A 78-year-old female was referred to our hospital for the purpose of an operation for type A AAD by practitioner. She had a history of thrombosed aortic dissection limited to the ascending aorta six months prior. At that time, a diameter of dissected ascending aorta was less than 40 mm and no pericardial effusion was recognized. Therefore, conservative management had been performed at another hospital. As she had this history of thrombosed aortic dissection, in this time, re-dissection of ascending aorta was suspected. She has complained about abdominal pain, not chest or back pain at an emergency room. Her consciousness was alert and blood pressure was 128/65 mmHg using intravenous antihypertensive agent. Physical finding revealed abdominal tenderness and distension. Metallic sound was heard on abdomen. Laboratory examination showed the C-reactive peptide of 0.72 mg/dL, elevated white cell count of 13100/µL and lactate acid value of 1.8 mmol/L. Enhanced computed tomography showed type A AAD with patent false lumen limited to the ascending aorta. (Figure 1 A). Type A AAD did not extend to the descending aorta and distal malperfusion syndrome also did not occur. The patency of celiac trunk and superior mesenteric artery was confirmed (Figure 1 B and C). We also recognized bowel ileus without dissection of abdominal aorta (Figure 1 D). No pericardial effusion was detected. The mechanism of ileus was unknown, so a diagnosis of paralytic ileus was made. Transthoracic echocardiography showed that the left ventricular ejection fraction rate greater than 60% and mild aortic regurgitation. No pericardial effusion was recognized. Based on a diagnosis of re-dissection of ascending aorta, an emergency hemiarch replacement using a prosthetic vascular graft was performed under mild hypothermic circulatory arrest and selective antegrade cerebral perfusion. Postoperatively, consciousness was alert and neither permanent or temporary neurologic deficit was present. As to preoperative paralytic ileus, conservative management had been continued and oral intake was initiated postoperative day four with improvement of bowel movement. Computed tomography revealed no residual dissection at the anastomosis site and improvement of paralytic ileus. She was transferred to a private hospital for the purpose of further rehabilitation. Until now, she has been followed up at outpatient clinic without recurrent ileus.

DISCUSSION

Because the false lumen statically and/or dynamically obstructs the origin of the visceral arteries in AAD (3), malperfusion syndrome associated with AAD can occur. They include ischemic stroke, myocardial infarction, mesenteric or extremity ischemia. With respect to mesenteric complications, they occur in only 5.3% of patients with type B AAD. However, mortality rate in patients who suffered from them was high, which was up to 33.3% (4). In type B AAD, mesenteric vessels are involved and mesenteric complications can occur. This is a mechanism of mesenteric malperfusion syndrome in type B AAD.

In the meanwhile, there has been no report describing mesenteric complications, in particular paralytic ileus described here in the setting of type A AAD. In type A AAD limited to the ascending aorta, mesenteric malperfusion syndrome cannot occur.

Retrospectively, present patient was doing well, had something to eat and stool every day before she suffered from present type A AAD. She felt sudden abdominal pain at that time and referred to a
hospital. The relationship between type A AAD and paralytic ileus was unclear.

In thoracic aortic dissection, activation of both overall and regional sympathetic nervous systems including elevation of plasma norepinephrine has been evident (5). The mechanism of paralytic ileus we described in this paper may be possibly explained by this activated sympathetic nervous system in AAD. As a report says that the clinical presentations in AAD are broad (1), we must take into consideration paralytic ileus as one symptom in type A AAD from our unusual experience. In addition, non-occlusive mesenteric ischemia might occur and affect temporary paralytic ileus in our case. This is a well-known complication after cardiovascular surgery. A diagnosis of non-occlusive mesenteric ischemia needs the vasospastic finding (6). Although computed tomography revealed the patency of visceral arteries preoperatively, the vasospasm of visceral arteries might occur before computed tomography, leading to paralytic ileus.

Paralytic ileus as the first symptom in type A AAD was an extremely rare condition. From a present report, we must consider that this condition can be occurred in type A AAD.

CONFLICT OF INTEREST DISCLOSURE

The authors have no financial or institutional interest in this study.

REFERENCES


