

Painless aortic dissection presenting as pseudo ileus: A case report

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Abstract. Aortic dissection is a serious acute cardiovascular disease with rapid onset, progression and a high mortality rate. Due to the range of different branching vessels involved, the clinical symptoms are complex and diverse. The typical clinical symptom is a severe tearing pain in the chest, back or abdomen, but some patients also have atypical symptoms, which are easily missed or misdiagnosed and can be life-threatening. The present study reports a case of painless type B aortic dissection, initially diagnosed as ileus. The objective of this study is to enhance the clinical understanding of painless aortic dissection so that the disease can be quickly and accurately detected, and treated in a timely manner, thereby improving patient outcomes.

Introduction

Aortic dissection (AD) is a life-threatening disease that requires early diagnosis and treatment to prevent death. The estimated incidence is 2.5 to 3.5 per 100,000 person-years (1). The mortality rate for acute AD increases by 1% per hour during the first 24 h (2), and the overall mortality rate for patients with acute AD complicated by adverse perfusion syndrome may be as high as 45% (3). The high mortality rate of AD underscores the need for rapid identification and treatment.

The Stanford classification has been used in clinical practice for a number of years. Type A dissection involves the

ascending aorta and type B dissection begins in the descending aorta. The most typical presentation is a sudden onset of tearing pain in the chest or back (4). Type A is considered to be a surgical emergency, while type B is more often treated with medication. Painless AD is relatively rare, but is more likely to be missed or misdiagnosed due to the atypical presentation. Numerous patients die before they reach a hospital or obtain a definitive diagnosis (1). For such cases, it is necessary to maintain a high degree of vigilance to avoid misdiagnosis and mistreatment. The present study report a case of painless AD in a patient who was admitted to hospital with symptoms of flatulence and a reduced defecation frequency. The preliminary diagnosis was ileus, but the patient was eventually diagnosed with painless type B AD.

Case report

A 45-year-old man was admitted to Bethune International Peace Hospital (Shijiazhuang, China) in October 2015 due to flatulence accompanied by reduced defecation for 8 days. At 8 days prior to admission, the patient felt continuous abdominal fullness without obvious inducement and the defecation frequency was reduced, but there was no abdominal pain, nausea or vomiting. Abdominal distension could be relieved after flatus. Food intake was significantly reduced, with no aversion to greasy food, abdominal enlargement was not observed and the urine was normal. At 5 days prior to admission, the patient came to the Outpatient Department of the Bethune International Peace Hospital, and at that time, blood routine examination, electrolyte levels, liver function, renal function and blood lipid levels were normal. Double adrenal color ultrasound indicated no obvious abnormalities in both renal adrenal areas and double renal arteries. An abdominal vertical X-ray showed gas accumulation in the intestine without an air-fluid level. Laxatives were not very effective. After oral administration of 250 ml mannitol 3 days before admission, yellow sparse defecation occurred 4-5 times. However, the abdominal distention became significantly worse, and the patient stopped defecating and passing gas 2 days before admission. Due to these symptoms, the patient attended the Department of Gastroenterology in the Bethune International Peace Hospital. Since the onset of the

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Abbreviations: AD, aortic dissection; CTA, computed tomography angiography

Key words: AD, type B aortic dissection, painless, hypoperfusion syndrome, ileus



Figure 1. Computed tomography showing type B AD, (a) starting from the descending aorta and (b) extending to the superior mesenteric artery. (c) Aortic angiography showing type B AD. (d) Aortic angiography showing that the type B AD has an S-shaped course, extending to the superior mesenteric artery. (e) Three-dimensional digital subtraction angiography of the aorta after thoracic aortic coated stent implantation. AD, aortic dissection.

disease, the patient had consumed a poor diet and had lost 6 kg of body weight. The patient had a history of hypertension for >10 years, with levels up to 180/100 mmHg. Intermittent oral administration of indapamide had resulted in poor blood pressure control, fluctuating at ~160/100 mmHg. At the physical examination on admission, the patient had a body temperature of 36.6°C, a pulse rate of 104 beats/min, a respiration rate of 18 breaths/min and a blood pressure of 156/102 mmHg. There was no obvious abnormality of the heart and lungs, and no obvious tenderness and rebound pain in the whole abdomen. No mass was palpable and auscultation showed hypoactive bowel sounds.

After admission, laboratory tests showed no abnormalities in the blood, urine and stool routine examinations, or in liver and kidney function, electrolyte levels, coagulation and tumor markers, blood gas analysis and lactic acid levels. No abnormality was found upon cardiac, abdominal or pelvic ultrasound. After clean enema and catharsis treatment, there was still no flatus or defecation. An AD was incidentally found on abdominal computed tomography angiography (CTA). To further delineate the extent of the dissection, CTA of the aorta was performed, which confirmed a type B AD where the AD extended from the arch of the aorta to the superior mesenteric artery (Fig. 1a and b). The diagnosis of type B AD was definitive, and the patient was treated using a micropump to continuously administer intravenous urapidil hydrochloride (50 mg) and sodium nitroprusside (50 mg) for blood pressure and heart rate control. Aortic angiography and a thoracic aorta coated stent implantation were performed (Fig. 1c-e). At 11 days post-surgery, the patient had recovered and was discharged. Follow-up was performed every 3-6 months, and after 5 years of follow-up, the general condition of the patient was good, and the blood pressure was well controlled by oral antihypertensive drugs.

Discussion

AD, also known as an aortic dissecting aneurysm, refers to a serious aortic disease in which the blood in the aortic intima passes through the intima tear and enters the middle layer

of the arterial wall to form a dissecting hematoma, which expands along the long axis of blood vessels and forms pathological changes in the true and false lumens of the artery (5). The typical clinical presentation of AD is a tearing pain in the chest, abdomen or back, but 4.5-6% of AD is painless, and these cases have a very high rate of missed diagnosis (6). The preponderant use of medication for type B AD does not mean that these patients do not have a complex dissection, as they may still experience poor perfusion and aortic rupture. Tolenaar *et al* (7) found an increased trend in in-hospital mortality among patients with type B AD, which may be the result of the delayed diagnosis and treatment due to a lack of typical symptoms. Park *et al* (8) found that patients with painless type B AD had significantly higher rates of mortality and aortic rupture than patients with type A AD with pain. Since painless type B AD is associated with increased mortality, timely identification of these patients is important.

Hypertension is the most important risk factor for AD. In total, 65-75% of patients with AD have hypertension, and their blood pressure is poorly controlled. In addition to the absolute increase in blood pressure, the increase in blood pressure change rate is also a factor causing AD. In addition atherosclerosis and aging are also important risk factors for AD (6). In the present case, the patient had a long history of hypertension, which was poorly controlled, and this was considered as the main cause of the AD.

Retrospectively, although the patient had no obvious abdominal pain, the clinical manifestations of type B AD were more like ileus; however, x-rays did not support this diagnosis. Until CT found an AD involving the superior mesenteric artery, AD-related hypoperfusion was considered to be the primary cause of the ileus symptoms. At 2 days prior to admission, the patient took mannitol orally to relieve the constipation, and after discharging loose stools several times, defecation and flatus stopped completely. It was considered that the aggravation of the condition, perhaps related to the mannitol stimulating gastrointestinal peristalsis, lead to the expansion and progression of the dissection, which increased the risk of intestinal ischemia, intestinal necrosis and dissection rupture. Although mesenteric complications occur in only

5% of patients with type B AD, the mortality rate is higher at $\leq 33.3\%$ (9).

The reasons for the absence of pain in patients with AD are unknown, but according to reports, there are several possible reasons: i) The hematoma dilates into the aortic lumen, lateral pressure is low for the outer membrane with plexus distribution and there is no pain caused by the outer membrane protruding; ii) the dissection progresses slowly, and chronic stretch stimulation may increase the pain threshold (7); iii) the severe spinal cord ischemia that has occurred makes the viscera, spinal cord and thalamus lose their ability to sense pain, thus the ability of the patient to sense pain is reduced (10); iv) the gap between the false cavity and the true cavity is large and numerous, the pressure in the false cavity is low and no obvious pain is caused. A previous study has also shown that patients with AD who have a history of Marfan syndrome may also present with painless symptoms (11).

There were a number of deficiencies in the diagnosis and treatment process of this case, such as the pre-existing diagnosis of ileus, ignoring hypertension as the main cause, failing to compare the blood pressure of both upper limbs and failing to pay attention to vascular murmurs due to insufficient physical examination. Painless AD is rare in the clinic. The present case report suggested that there was an insufficient blood supply, mainly due to the tear of the intima and media of the vessel, and blood flowing into the interlayer from its own tubular channels, resulting in hemodynamic changes. An insufficient distal blood supply may lead to reduced or absent arterial pulsation, ischemia of the head may lead to lethargy, apathy and syncope, and ischemia of the spinal cord may lead to limb weakness and paraplegia (12,13). When dissection occurs in the aortic arch, there is a significant difference in the blood supply between the brachiocephalic trunk and the left subclavian artery, which may result in a significant difference in blood pressure between the left and right upper limbs. The same thing can happen in other parts of the body to cause huge differences between the upper and lower limbs (14). As the torn intima forms a 'reservoir sac', this portion of blood may fall back during diastole to cause aortic valve murmurs (12). Therefore, a new aortic murmur is also a high-risk sign for AD; however, it has been reported that 32% of patients with painless AD have no vascular murmur (15). Clinical attention to the physical examination can lead to a more timely diagnosis of painless AD. Fortunately, in the present study, the AD was incidentally found during abdominal CTA, which resulted in the correct diagnosis and treatment, and avoided adverse consequences. Therefore, physicians should be aware of this relatively rare presentation of painless type B AD.

In conclusion, the clinical manifestations of typical AD are easy to recognize, but the hidden and complex clinical manifestations are easy to ignore or misdiagnose. The rate of missed diagnosis and misdiagnosis in patients with first painless AD is higher than that in patients with pain, and the mortality rate is higher. Therefore, clinicians should be alert to painless symptoms and subtle signs related to AD, and should pay enough attention to them.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Authors' contributions

CW was responsible for the clinical management of the patient, and the processing and scientific interpretation of the data. BW, JZ and YH were responsible for evaluating the data, revising the manuscript, analyzing the specialized literature and writing the manuscript. ZL and JY were responsible for data processing and revising the manuscript. CW was responsible for analyzing the specialized literature, interpreting and evaluating the data, and revising the manuscript. JY, JZ and YH confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Written informed consent was obtained from the patient.

Competing interests

The authors declare that they have no competing interests.

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