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Case Report

Hepatic portal venous gas complication associated with the thoracic endovascular aortic repair for aortic dissection: a case report and literature review

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Abstract: Aortic dissection (AD) is a serious disease with a higher mortality. The thoracic endovascular aortic repair (TEVAR) is a first line regimen for aortic dissection. Hepatic portal venous gas (HPVG) is a rare disease, and its definite mechanism is unknown. This is a rare association between the aortic and HPVG. In the present report, we present a case of thoracic aortic dissection, which was the type of Standford B by the computer tomography (CT) angiography, which implicated acute abdominal pain and abdominal distention after TEVAR and immediate abdominal CT shown hepatic portal venous gas (HPVG). The patient, who was treated with conservative treatment of gastrointestinal decompressing, fluid resuscitation, electrolyte replacement, anti-infection, anti-inflammation and anticoagulation, was recovered and discharged without abnormalities. This patient has been followed up for 5 years and has not experienced any physical discomfort related to HPVG. This is the first report that the aortic dissection patient implication with HPVG after thoracic endovascular aortic repair.

Keywords: Aortic dissection, thoracic endovascular aortic repair (TEVAR), hepatic portal venous gas (HPVG), ischemia reperfusion injury

Introduction

HPVG, a pessimistic radiologic sign, was first described by Wolfe and Evans about six infants with enterocolitis [1]. The Hepatic portal venous gas (HPVG) is a rare condition, which clearly pathological mechanism is unknown [2]. There were many risk factors maybe result the HPVG such as gastric ulcer, gastric emphysema, acute hemorrhagic pancreatitis, bowel infraction, closed loop obstruction, necrotizing enterocolitis, and granulomatous enterovenous fistula [3]. In some cases, HPVG is associated with the severe abdominal disease which needs further operative intervention. At present, with the use of CT and ultrasound, the underlying severe diseases and HPVG are also diagnosed early, which can reduce the progression of the disease in time. The severity of the outcome and prognosis is related to the disease itself, not the HPVG. The conservative treatment for the patients with HPVG always got the satisfactory outcomes, however, a surgical intervention should be taken for the obvious signs that indicate the intestinal perforation or peritonitis. Without early diagnosis and treatment, thoracic aortic dissection is an event with high mortality [4]. For the treatment of thoracic aortic diseases such as type B aortic dissection, thoracic endovascular aortic repair is an alternative treatment in contrast to open surgery [5]. In the present report, we present a case of thoracic aortic dissection, who was the type of Standford B diagnosed by the computer tomography (CT) angiography. This patient, with the symptom of acute abdominal pain and abdominal distention after TEVAR, was diagnosed as HPVG by abdominal CT. With the conservative treatment, he was recovered and discharged without abnormalities. This patient has been followed up for 5 years and has not experienced any physical discomfort related to HPVG. This is the first report that the aortic dissection patient implication with HPVG after thoracic endovascular aortic repair.

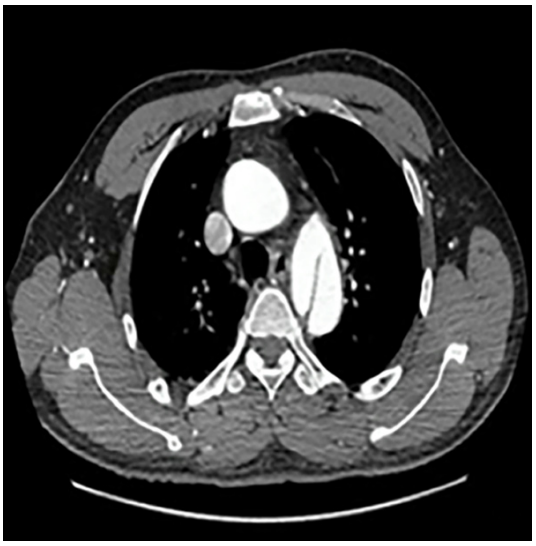

Figure 1. A Standford B aortic dissection.

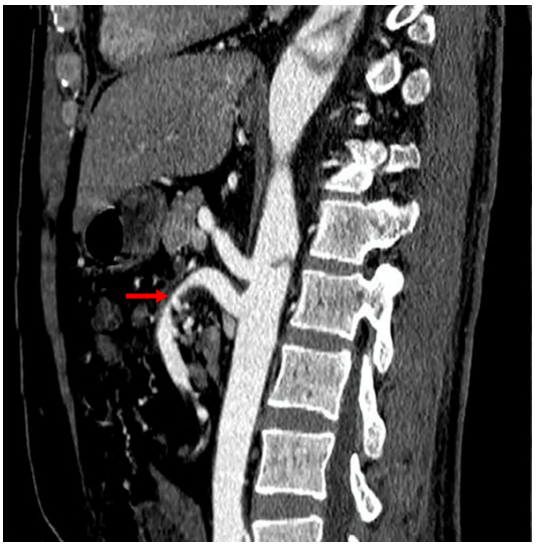

Figure 2. The diameter of the superior mesenteric artery has decreased due to the involvement of the dissection as shown by the arrow.

Case report

A 46-year-old male presented with two hours history of acute chest pain, who was diagnosed of aortic dissection (**Figure 1**) by the CT angiography (CTA), and hypertension. The CTA showed that the tear is at the descending aorta with 2 cm from the left subclavian artery and another tear is at the abdominal aorta. The blood of the superior mesenteric artery and other visceral arteries are from the true lumen. The patient was admitted to hospital, with mild abdominal pain and without abdominal distension and constipation. The CT shown that the diameter of the superior mesenteric artery has decreased due to the involvement of the dissection (**Figure 2**). There were no abnormalities about blood routine test, liver function and renal function. The patient, with conservative treat as lowing blood pressure and control heart rate for two weeks, was executed an operation of TEVAR under general anesthesia. In this operation, two tear entries were blocked by covered stent without endoleak. The visceral arteries were imaging clearly by angiography. Everything was normal, however, the patient complained an acute abdominal pain and abdominal distension at the first day after operation. Physical examination shown that abdominal distension, abdominal tenderness and weakened borborygmus. The CT scans shown that there was amount of gas in hepatic portal venous and intestinal tract (**Figure 3**). Laboratory tests shown that leukocyte counts $18.15*10^9$/L, hs-CRP 1.33 mg/dl, LDH 129 U/L and without any other abnormalities. The patient was treated with gastrointestinal decompression, fluid resuscitation, electrolyte replacement, anti-infection (Cefotiam 1 g add to NS 100 ml ivgtt. Bid) and anti-inflammation (Ulinastatin 20 WU add into NS 100 ml ivgtt) and anti-coagulation (Low molecular weight heparin calcium 4100 U, hypodermic injection, Qid). The abdominal pain and abdominal distension were alleviated next day. The physical examination revealed active borborygmus. Laboratory examination showed that leukocyte counts $17.88*10^9$/L, hs-CRP 20.61 mg/dl, LDH 154 U/L, BUN 13.58 mmol/L, CRE 337 umol/L and without any other abnormalities, next day. We choose the conservative treatment as mentioned above. The day after next day, the symptom was disappeared generally, and the laboratory test shown that leukocyte counts $17.88*10^9$/L, LDH 151 U/L, hs-CRP 19.55 mg/dl, BUN 13.5 mmol/L, CRE 222 umol/L and without any abnormalities, and the CT scans that hepatic portal venous gas was disappeared with the intestinal canal mild expansion (**Figure 4**). The patient continues to be treated with conservative as anti-infection, gastrointestinal decompression, volume resuscitation and maintaining electrolyte imbalances. The laboratory examination showed that leukocyte counts $9.08*10^9$/L, LDH 105 U/L, hs-CRP 9.67 mg/dl, BUN 8.2 mmol/L, CRE 99 umol/L and

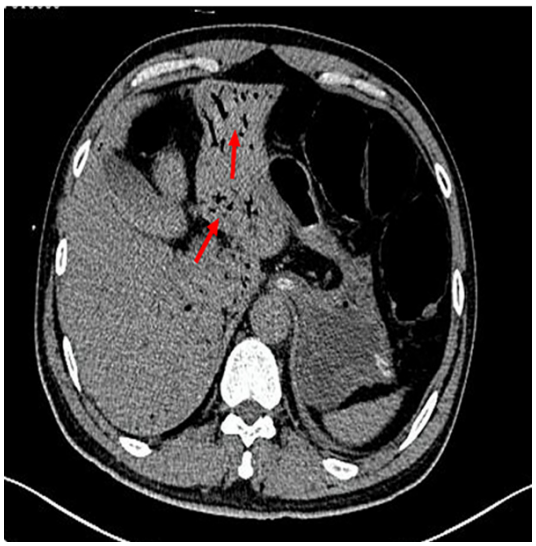

Figure 3. There was amount of gas in the intestinal lumen and hepatic portal venous as the arrows showed.

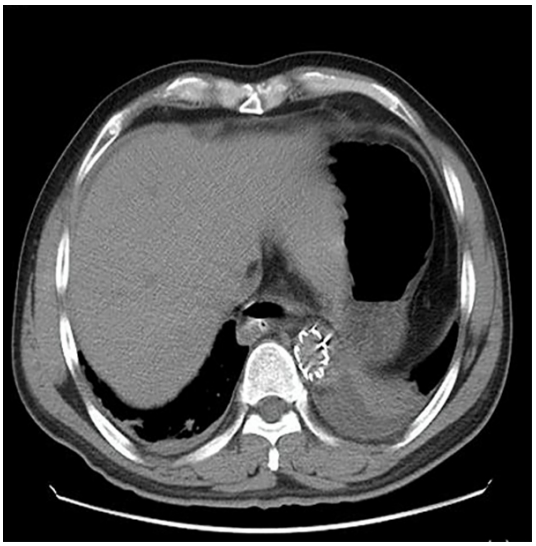

Figure 4. The hepatic portal venous gas disappeared with the intestinal canal mild expansion.

without any abnormalities on the eighth day and discharged at the tenth day with any abnormal symptoms. The patient was followed five years and without abnormality.

Discussion

Hepatic portal venous gas (HPVG) is a rare condition, with air in the hepatic and portal vein. Although the definite etiology was unknown, the abdominal disease was considered, and the surgical intervention would be made for the HPVG associated with the lethal disease [2].

What is the reason for the HPVG in this patient? Although the contrast agent (iohexol) as the cause and has been reported in the literature [6], the patient was safe from the CT angiography before the operation. So the contrast agent should be excluded in this patient. Although the digital subtraction angiography (DSA) shows that the visceral arteries of the patient are all being supplied by the true lumen, the diameter of the visceral arteries has decreased due to the involvement of the dissection, which is the reason of mild abdominal pain at the time of hospital. We believe that reperfusion injury following visceral ischemia could be the main cause of this phenomenon. After the operation of TEVAR, the true lumen diameter of the aorta increases, resulting in increased blood supply to the superior mesenteric artery and leading to reperfusion injury. Of course, post-implantation syndrome (PIS) is also worth considering and may be an independent or additional factor contributing to the occurrence of HPVG in this patient. The PIS may be result in the fever, inflammation, and coagulation dysfunction, which could lead the visceral organs failure and even to death.

We speculated two major etiologies: first, due to the aortic true lumen and visceral blood flow volume increase, the ischemia reperfusion injury was happened, which the inflammation reaction and intestinal mucosa damaged as followed; second, much of the inflammation factors, resulted from the PIS, was released into the blood and aggravated the extent of ischemia reperfusion injury of the intestinal mucosa, which results the dilatation of intestinal, enteroparalysis. The laboratory test such as leukocyte counts, CRP and CRE was increasing at the time of disease onset and gradually decreased as the patient recovered.

There is no consensus on the treatment of HPVG. There was no difference between the conservative and surgery interventions for the common case, and the prognosis was based on the extent of intestinal necrosis [7]. Koami H et al. [8], with analysis of the 33 cases of HPVG, found that the abdominal symptom, peritoneal irritation sign, systolic pressure, Aspartic acid dehydrogenase, Lactate dehydrogenase (LDH), dilatation of intestinal, intestinal pneumatosis

were significant correlation with the intestinal necrosis. In this case, although with the abdominal pain and dilatation of the intestinal, we could speculate there was no serious intestinal necrosis from the manifestation such as stable vital signs, no peritoneal irritation, borborygmus and no black stool. In addition to physical examination, the laboratory test showed that the level of LDH was in the normal spectrum. The conservative treatment was chosen for this patient which is based on the speculation mentioned above. We should mention that the slow blood flow of portal vein is a risk for portal vein thrombus, which is why we use the anti-coagulation medicine.

The TEVAR is the first line regimen for the treatment of aortic dissection, especially the type of Standford B, however, HPVG as the postoperative complication had not been reported ago. Although this is the first time present the clinical phenomenon, it gave us some clinical experience. From this case, we think that the ischemia reperfusion injury may be a complication of TEVAR. We should monitor patient closely for abdominal signs and symptoms. The presence of intestinal ischemic necrosis was determined by physical examination and blood index monitoring. It is very necessary to reduce the pressure in the gastrointestinal tract in combination with anticoagulant and anti-infection. Although this kind of situation is not common, it can give some guiding value to the clinical work of our center or other centers. This patient has been followed up for 5 years and has not experienced any physical discomfort related to HPVG.

Disclosure of conflict of interest

None.

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