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Type B Aortic Dissection with Visceral Artery Involvement Following Blunt Trauma: A Case Report

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Aortic dissection caused by blunt trauma is a rare injury that can be complicated by malperfusion syndrome resulting from obstruction of branch vessels of the aorta. Here, we present a case of traumatic type B aortic dissection with right renal and small bowel ischemia, successfully managed by endovascular fenestration.

Keywords: Endovascular procedures; Thoracic injuries

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INTRODUCTION

Aortic injury after blunt trauma is associated with high mortality, and the majority of patients die at the scene before treatment can be initiated [1,2]. While intimal tear, intramural hematoma, pseudoaneurysm, and transection of the aorta are well-documented forms of blunt thoracic aortic injury (BTAI), only few reports of traumatic aortic dissection (AD) have been published [3]. Uncomplicated AD can be managed conservatively with antihypertensive medication, particularly in patients with additional severe injuries. However, prompt surgical or endovascular intervention is required when cases of AD are complicated by malperfusion syndrome. Here, we report a case of traumatic type B aortic dissection (TBAD) with renal and superior mesenteric artery (SMA) involvement, managed with endovascular fenestration.

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CASE REPORT

A 44-year-old construction worker presented to our trauma center with severe abdominal and back pain, one hour after falling from a height of 11 meters. Vital signs were assessed on arrival at the emergency department (blood pressure, 151/87 mmHg; pulse, 80 beats per minute; respiratory rate, 20 breaths per minute; and temperature, 36.8℃). The patient had previously been diagnosed with hypertension, although he was not receiving antihypertensive medication. Physical examination showed equivocal tenderness in the lower abdomen. Peripheral pulses were palpable in both extremities, and no neurologic deficits were apparent, with the exception of mild tingling in the right lower leg. Computed tomography (CT) scan taken 30 minutes after arrival at the hospital, revealed an AD extending from the proximal descending aorta to the level of bifurcation of the abdominal aorta (Fig. 1). Starting from the proximal entry point (which was in close proximity to the left subclavian artery) and throughout the dissected aorta, the true lumen was very narrow and crescentic. The primary entry tear was located at 1.5 cm distal to the opening of left subclavian artery, and the re-entry tear was located at the aortic bifurcation. Perfusion to the right kidney was markedly decreased as a result of the false lumen causing almost total occlusion of the renal artery. The SMA was occluded approximately 1 cm distal to its point of origin from the aorta. Coronal view revealed complete occlusion of the proximal 7 cm of the SMA, with reconstitution distally; several hypoattenuating bowel loops were seen in the pelvic cavity. In addition to the aortic injury, the patient had an extra-axial brain hemorrhage in the right frontal region, bilateral lung contusion, and L1 to L3 vertebral compression fractures, as well as fracture of the transverse process of L1 to L3.

Immediately after the CT scan, intravenous infusion of antihypertensive medication was initiated to control blood pressure. After considering the extent of the AD, involvement of multiple visceral arteries, equivocal abdominal tenderness, and presence of multiple additional injuries, surgery was not considered to be an optimal approach and an endovascular procedure was undertaken to

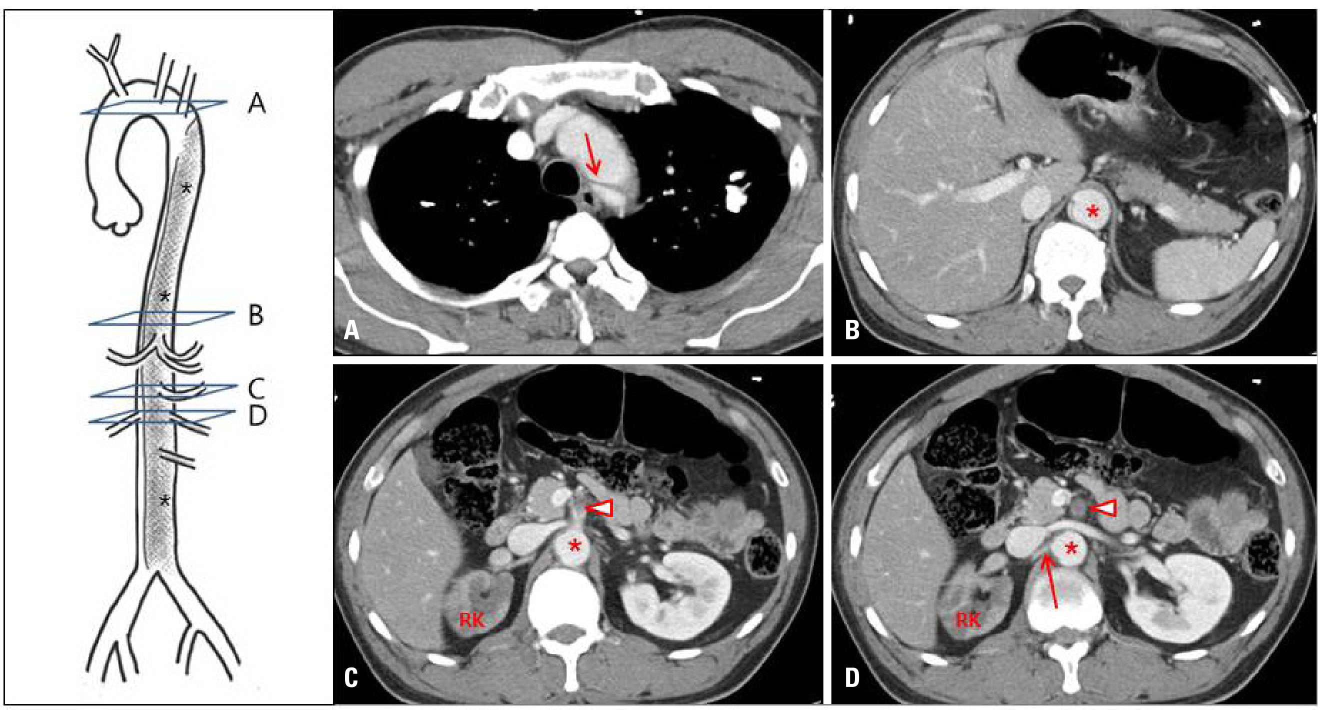

Fig. 1. Illustration and axial contrast-enhanced computed tomography scan of the thoracoabdomen showing type B aortic dissection. (A) Dissection flap (arrow) originating just distal to left subclavian artery. (B) True lumen is collapsed by the large false lumen (asterisk). (C, D) Superior mesenteric artery occluded 1cm distal to its origin (arrowhead). Hypoattenuating right kidney due to dynamic occlusion of right renal artery (arrow) by the dissecting flap. Large false lumen (asterisk) suppressing the true lumen is visualized. RK: right kidney.

relieve renal and bowel ischemia. Under local anesthesia, a 6 French sheath (Terumo, Tokyo, Japan) was placed in the left common femoral artery. Abdominal aortography with the pigtail catheter in the false lumen showed opacity in the celiac, left renal, inferior mesenteric, and multiple lumbar arteries; the right renal artery was not visualized. The SMA was also occluded, but some of the proximal jejunal branches were visualized during celiac arteriogram as they were supplied by the pancreaticoduodenal arcade. Also, some of the distal SMA branches were perfused by the inferior mesenteric artery through collateral pathways.

To relieve malperfusion of the right renal artery, septal fenestration was performed proximal and distal to the renal artery (Fig. 2). A dissection flap was punctured using the re-entry catheter system (Outback LTD; Cordis, Miami Lakes, FL, USA), and the fenestration was widened using a balloon catheter (Mustang 12×60 mm; Boston Scientific, Nattick, MA, USA). Time from initial injury to fenestration was 4 hours. After fenestration had been performed, aortography showed expansion of the true lumen and restoration of blood flow to the right renal artery. However, flow did not appear to be restored through the SMA, despite expansion of the true lumen at the appropriate level, and the obstruction was, therefore, thought to be static. Although further attempts were made to recanalize the SMA, the guidewire could not be passed beyond the occluded region.

After the fenestration procedure, the patient was admitted to the intensive care unit. Blood pressure was strictly controlled to a target systolic pressure of 100-110 mmHg, and the patient's abdomen was examined frequently. Abdominal symptoms showed gradual improvement over the following 2 days. CT scan on hospital day 5 showed improved perfusion of the right kidney with complete restoration of flow in the right renal artery (Fig. 3); the SMA also appeared to be partially recanalized (Fig. 4). The patient was started on liquid diet the day after the CT, and advanced to normal diet. Despite the narrow true lumen of SMA, he tolerated normal diet well, and thus, no further surgical or endovascular intervention was performed. Following successful management of his other injuries, the patient was discharged on hospital day 27. The day before his discharge he had a final thoracoabdominal CT scan which showed stable dissection without antegrade or retrograde progression, and well-perfused right kidney. There also was no change in the narrow recanalized true lumen of SMA. After 4 months of follow-up, the patient remained free from abdominal symptoms and renal dysfunction. We plan to follow this patient with routine CT scan; one at 2 months later, and annually thereafter.

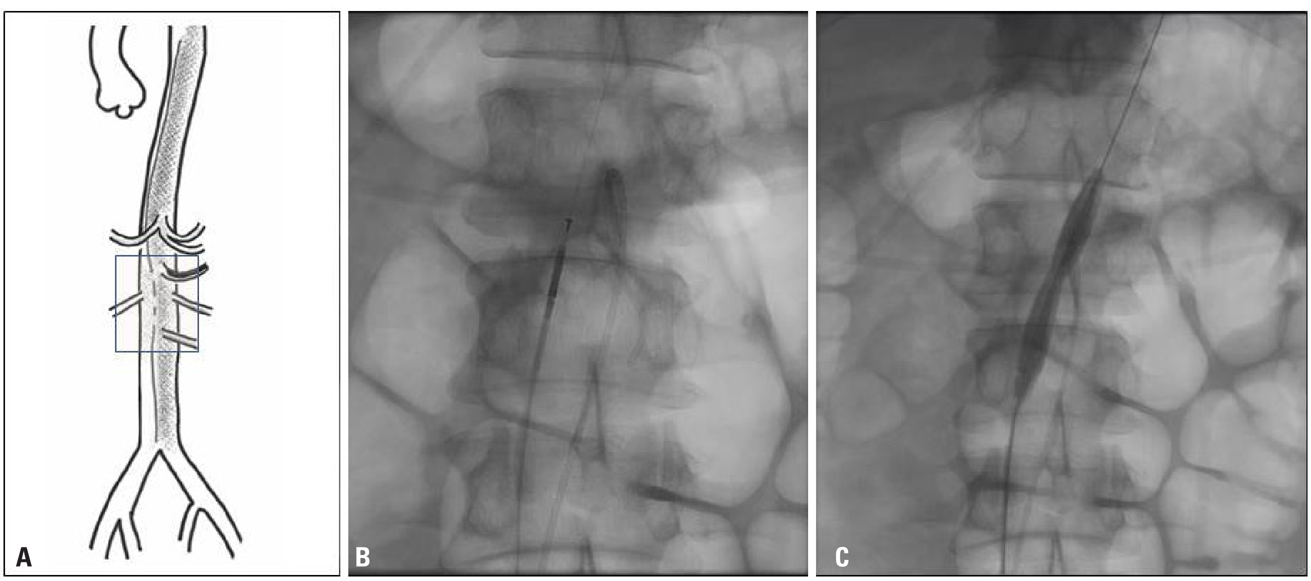

Fig. 2. Septal fenestration to relieve malperfusion of the right kidney. (A) Septal fenestration was performed at 3 levels, close to the level of origin of right renal artery. (B) Outback re-entry system was used for fenestration. (C) The fenestration was subsequently enlarged using noncompliant balloon catheter.

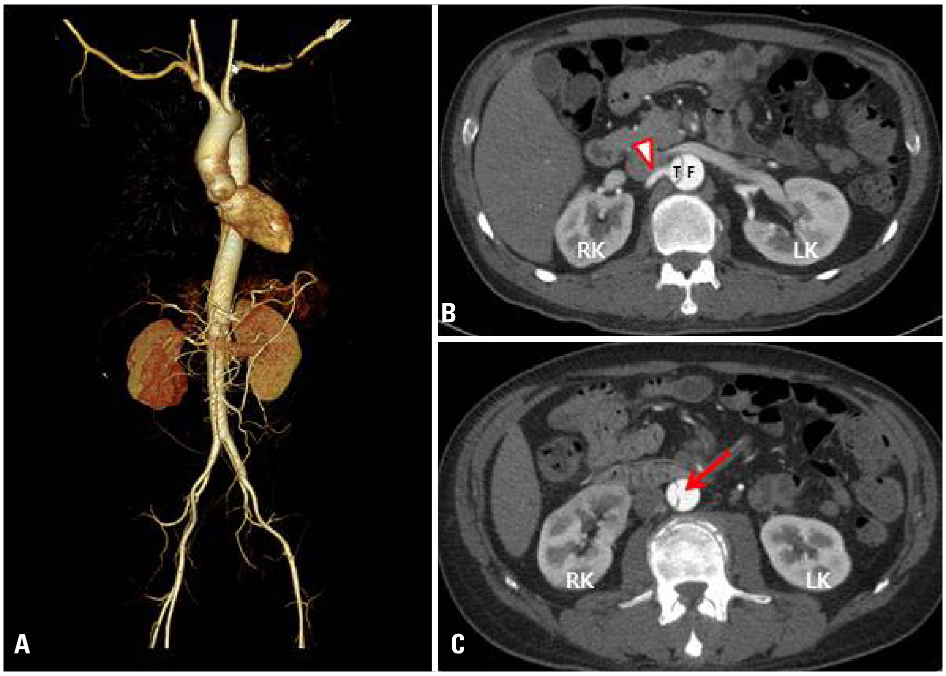

Fig. 3. Follow-up CT at day 5 in (A) three dimensional reformations, and (B, C) axial views show widened true lumen with restored flow to the RK (arrowhead). Fenestrated septum is visualized (C, arrow). CT: computed tomography, RK: right kidney, LK: left kidney, T: true lumen, F: false lumen.

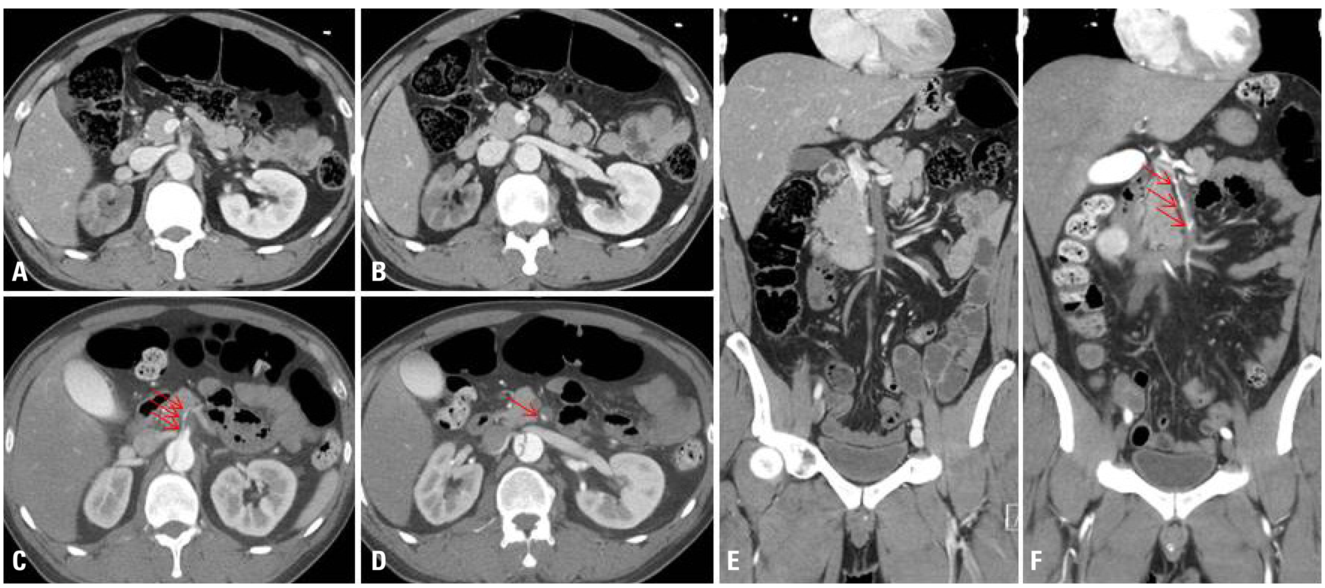

Fig. 4. Change in superior mesenteric artery flow before and after the fenestration. (A, B, E) Initial CT before fenestration showing occluded superior mesenteric artery occluded 1 cm distal to its origin. (C, D, F) Follow up CT after fenestration, at day 5 shows narrow recanalized true lumen of superior mesenteric artery (arrows; images about the same level as A, B, E, respectively). CT: computed tomography.

DISCUSSION

AD after blunt trauma is a rare injury [3,4]. Although predisposing factors, such as pregnancy or atherosclerosis, have been linked to the occurrence of AD after trauma [5,6], traumatic AD in otherwise healthy young patients has also been reported [7,8]. In the present case, untreated hypertension may have resulted in degenerative changes in the medial layer of the aorta, potentially contributing to the extensive medial dissection of the aorta after traumatic intimal tear. While it is possible that the AD had been present prior to the trauma, we consider this to be unlikely due to the absence of any previous symptoms. As the incidence of traumatic AD is very low, differences in the pathophysiology, optimal management, and outcome versus other types of AD have not been well documented. It is of note that the treatment strategies discussed below are based on the current guidelines for AD, which are largely derived from experience with nontraumatic AD. However, we considered it appropriate to adhere to the existing guidelines until more evidence in traumatic AD can be obtained.

The management of TBAD in general has changed greatly in the past two decades [9]. Antihypertensive medication is now considered as first-line treatment for patients with uncomplicated type B dissections, and endovascular approaches have replaced surgical repair for the management of complicated type B dissections. TBADs with malperfusion syndrome, which constitute 10% of complicated TBAD cases [10], present a particularly challenging scenario. A report from the international registry of acute aortic dissection (IRAD) showed that 19% of in-hospital deaths in patients with TBAD were caused by visceral ischemia and that TBAD with branch vessel involvement was associated with 2.9 times higher mortality than that without [11]. Manifestations of the local and systemic inflammatory cascade caused by end organ ischemic and reperfusion injury are believed to contribute to this poor outcome [12].

The treatment goal in all patients with AD is to prevent rupture. However, in patients with malperfusion syndrome, urgent restoration of flow to the end organs is required [13]. In patients with TBAD, whose risk of rupture is lower than that of patients with type A dissections, reperfusion-directed therapy should be the highest priority. The optimal method of revascularization differs according to the nature and extent of the branch vessel obstruction. In dynamic obstruction, where the intimal flap obstructs the orifice of the branch artery, depressurization of the false lumen is required [14]. This can be achieved either by central aortic repair or fenestration of the dissecting flap. However, in the case of static obstruction, which is caused by extension of the dissection into the branches, endovascular stent grafting or surgical revascularization may be required [14]. The extent of the obstruction and the presence of collateral vessels are also important considerations when deciding whether or not to treat the obstruction. In the present case, although the SMA appeared to be completely occluded for a long segment, blood flow to peripheral branches of the SMA was preserved by collateral vessels from the celiac and inferior mesenteric arteries. In retrospect, we believe that the SMA obstruction had been of a mixed type as it was partly reversed by fenestration.

The dynamic obstruction of the right renal artery in our patient was managed with percutaneous fenestration, rather than by thoracic endovascular aortic repair (TEVAR). Although both methods are used to relieve dynamic obstruction, there are important differences. In the present case, fenestration was selected over TEVAR because most of the intercostal arteries (as well as the celiac and left renal arteries) were supplied by the false lumen, and this approach is in line with recent recommendations from the Michigan group [15]. After TEVAR, there is a risk of occlusion of the aortic branch vessels supplied by the false lumen, whereas flow to the vessels is preserved with the fenestration procedure. Fenestration, therefore, eliminates the risk of spinal cord ischemia, which is a serious complication seen in 2-10% of patients following TEVAR [16]. Other limitations of TEVAR include a high risk of early complications and failures that necessitate reintervention. In addition, follow-up data beyond 20 years are not available. However, an important advantage of TEVAR is that it seals the primary entry point and promotes false lumen thrombosis and favorable aortic remodeling, thereby decreasing the risk of late aortic rupture [17,18]. Fenestration alone does not treat aortic rupture risk, and aneurysmal changes have been seen in up to 50% of patients within 5 years [19]. Indeed, long-term follow-up of our patient would be of paramount importance, particularly considering his young age.

In summary, we present a rare case of TBAD, complicated by malperfusion syndrome. Immediate anti-impulse therapy and urgent percutaneous fenestration were performed to relieve the dynamic obstruction causing malperfusion of the right kidney. TBAD with malperfusion syndrome requires immediate surgical or radiologic intervention. Therapeutic strategies in patients with TBAD and malperfusion should be tailored to the individual, after consideration of concomitant injuries and the anatomic characteristics of AD and branch vessel obstruction.

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