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Cerebral hypoperfusion due to rapid blood pressure control in a patient with type B aortic dissection: A case report

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Abstract

Type B aortic dissection represents a life-threatening cardiovascular event, necessitating a comprehensive treatment approach that includes anti-impulse therapy for blood pressure and heart rate control, movement restriction, analgesia, sedation, and consideration of subsequent endovascular or open surgery. Management of acute aortic dissection involves stringent blood pressure control to prevent extension of the dissection, which is critical in both surgical preparation and conservative treatment strategies. While the treatment regimen is relatively well-established, and early and long-term follow-up results are promising, there have been reports of adverse events during treatment. In this context, we present a case of a 40-year-old male with acute type B aortic dissection who developed cerebral hypoperfusion as a consequence of rapid blood pressure reduction, underscoring the need for balanced hemodynamic management. Following thoracic endovascular aortic repair surgery and hyperbaric oxygen therapy, the patient fully recovered 6 months later with no lingering sequelae. While current guidelines emphasize specific numerical values for blood pressure control in type B aortic dissection, the case underscores the potential need for a more individualized approach tailored to the unique characteristics of each patient.

Keywords

Type B aortic dissection, rapid preoperative control of blood pressure, cerebral hypoperfusion, endovascular treatment

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Introduction

Over the past 30 years, anti-impulse therapy has emerged as a crucial component in the cardiovascular surgical management of aortic dissection.[1,2] This therapy involves meticulous control of heart rate (<60 beats/min) and blood pressure (<120/80 mmHg) to mitigate shear stress on the aortic wall, thereby reducing the risk of dissection expansion and associated complications, such as aneurysmal degeneration and rupture. This approach lowers the likelihood of a ruptured aortic dissection prior to surgery, creating favorable conditions for subsequent surgical interventions.[3]

Given that patients with aortic dissection often present with elevated systolic blood pressure, a diverse array of antihypertensive drugs (including β-blockers, Angiotensin II receptor blockers (ARBs), and calcium antagonists) is typically employed. Some patients may require hospital admission for blood pressure control due to suboptimal hypertension management or poor compliance. While this strategy helps maintain relatively constant cerebral blood flow, dysfunctional automatic regulation of cerebrovascular resistance in certain pathological conditions renders brain tissue highly sensitive to even minor changes in cerebral perfusion pressure.

Another noteworthy consideration is the altered self-regulatory response in patients with chronic hypertension. In cases of mild to moderate blood pressure, the initial reaction involves arterial and arteriole constriction. This autoregulatory mechanism ensures stable tissue perfusion levels while preventing increased pressure from affecting smaller, more peripheral blood vessels. Consequently, even if the final blood pressure value remains within the normal range, an acute drop in blood pressure can manifest as cerebral ischemia symptoms in individuals with chronic hypertension.[4–11]

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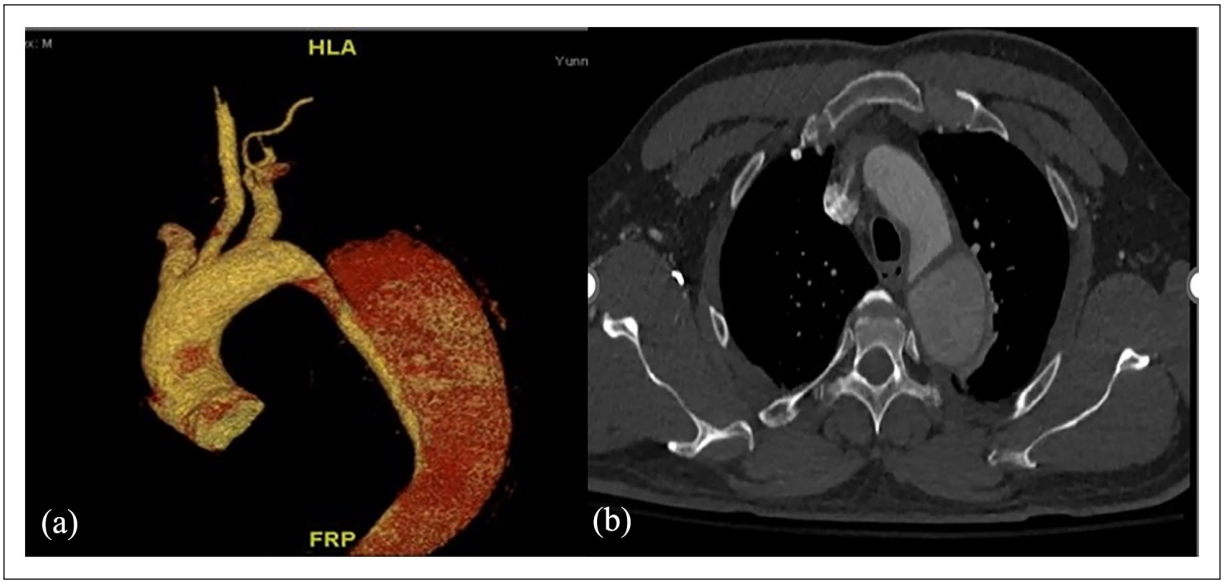

Figure 1. Illustrates the admission CTA, revealing aortic dissection of Stanford type B. (a) The rupture was localized at the distal end of the left subclavian artery, with no involvement of the superior arch branch (b).
CTA: Computed tomography angiography.

While current guidelines emphasize specific blood pressure control values for type B aortic dissection, adopting a more personalized approach to blood pressure reduction tailored to the unique characteristics of each patient may contribute to improved prognostic outcomes.[12]

Case report

We present a case of a 40-year-old male with acute type B aortic dissection and a high cardiovascular risk profile, including a history of sleep apnea, grade 3 hypertension, irregular use of antihypertensive medication, who developed cerebral hypoperfusion as a consequence of rapid blood pressure reduction, highlighting the need for balanced hemodynamic management. The patient presented with a chief complaint of "severe chest and back pain for 2 hours" and was transported to our hospital's emergency department by ambulance. Symptoms included severe and persistent tearing chest and back pain radiating to the interscapular region, accompanied by restlessness, without signs of coma, abdominal pain, limb paralysis, or oliguria. Additional symptoms included a systolic blood pressure as high as 200 mmHg. Upon admission, routine examinations including vital sign monitoring and emergency imaging (CTA and head computed tomography (CT)) were conducted. Due to the suspicion of aortic pathology, an emergent computed tomography angiography (CTA) was performed, revealing an acute type B aortic dissection extending from the distal arch to the abdominal aorta without the involvement of the ascending aorta. No advanced diagnostic interventions, such as transesophageal echocardiography, were performed initially due to the urgency of the patient's condition. Patients diagnosed with aortic dissection are admitted to the emergency intensive care unit. Continuous intra-arterial blood pressure monitoring is employed, along with a protocol of strict heart rate and blood pressure control using β-blockers, calcium channel blockers, and intravenous antihypertensives as required. Thoracic endovascular aortic repair (TEVAR) is considered for patients with complications or impending rupture. Upon entering the emergency department, we implemented strict blood pressure control (not exceeding 120/80 mmHg) and heart rate control (not exceeding 80 beats/min). The patient was instructed to remain in bed, vital signs were monitored, analgesia was administered, sedation was provided if necessary, and measures were taken to address constipation.

The selected antihypertensive regimen included oral metoprolol sustained-release tablets (100 mg/day) and intravenous nicardipine infusion (initial dose 5 mg/h, increased by 2.5 mg/h every 15–30 min until the target blood pressure was achieved) (Figure 1). Figure 2 illustrates the changes in blood pressure over time.

On admission day 1, the patient's level of consciousness was assessed by the physician, yielding a Glasgow Coma Scale score of 7, necessitating prompt intubation and mechanical ventilation to secure the airway and prevent further complications from reduced consciousness. By the first day of admission, the patient remained drowsy with no response to stimuli. Physical examination revealed bilateral dilated and reactive pupils, intact light reflex, and no facial asymmetry. Emergency head CT and bedside color ultrasound were conducted to rule out aortic dissection and reverse tear, revealing scattered small, low-density shadows in both basal ganglia (Figure 3). Neurological consultation recommended osmotherapy and adjustments to antihypertensive medications to optimize cerebral perfusion. Following the TEVAR

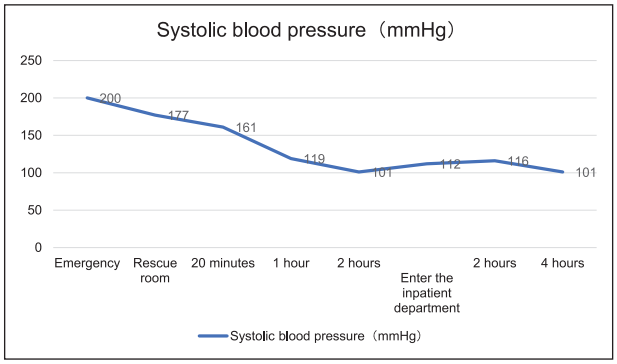

Figure 2. Depicts the variations in early systolic blood pressure following admission.

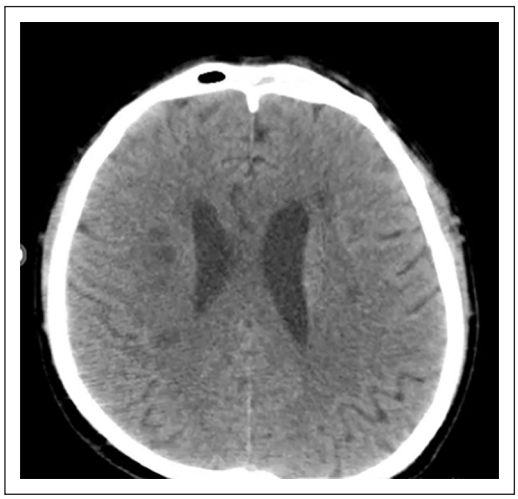

Figure 3. Displays scattered small, flaky, low-density shadows in the bilateral basal ganglia.

procedure, Post-stroke treatment at our institution includes butylphthalide to enhance microcirculation, citicoline to promote neural repair, and hyperbaric oxygen therapy to improve oxygen delivery to hypoxic brain tissues. These interventions are complemented by tailored blood pressure management and physiotherapy.

On the third day of admission, the patient experienced a decrease in heart rate (68 beats/min), reduced blood pressure (105/55 mmHg), and lowered oxygen saturation (88%) in the early morning. Ultrasound imaging showed no signs of bleeding. Despite discontinuing intravenous medications, there was no significant improvement. Consequently, the patient was transferred to the surgical intensive care unit for tracheal intubation, mechanical ventilation, central venous line insertion was performed for accurate hemodynamic monitoring and medication administration.

By the fourth day of admission, the patient's circulation stabilized after tracheal intubation and adjustments to the drug regimen. TEVAR was performed to address the high risk of aortic rupture, which outweighed the risks posed by the patient's existing neurological deficits. Given the patient's young age and minimal underlying conditions, a multidisciplinary team concluded that the watershed cerebral infarction was caused by hypoperfusion. Following active rehabilitation therapy, the prognosis was favorable. However, the risk of aortic dissection rupture hindered the potential for rehabilitation training. Under the strong request of the family, TEVAR surgery was performed (see Figure 4).

During the intraoperative procedure, angiography was utilized to identify the true and false lumens, and a Lifetech Ankura stent measuring 28-24-160 mm was successfully inserted to isolate the aortic rupture (Figure 4).

On the first day post-surgery, the patient's recovery showed a delay, and improvement was observed in the head

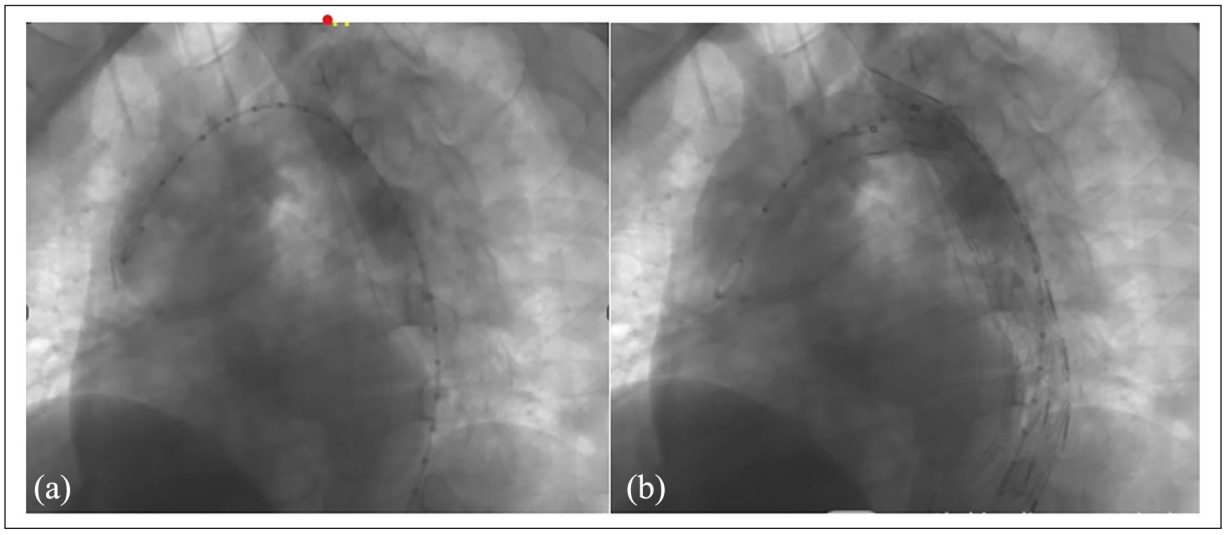

Figure 4. Illustrates intraoperative (a) and post-stent imaging (b), showcasing the successful isolation of the aortic rupture.

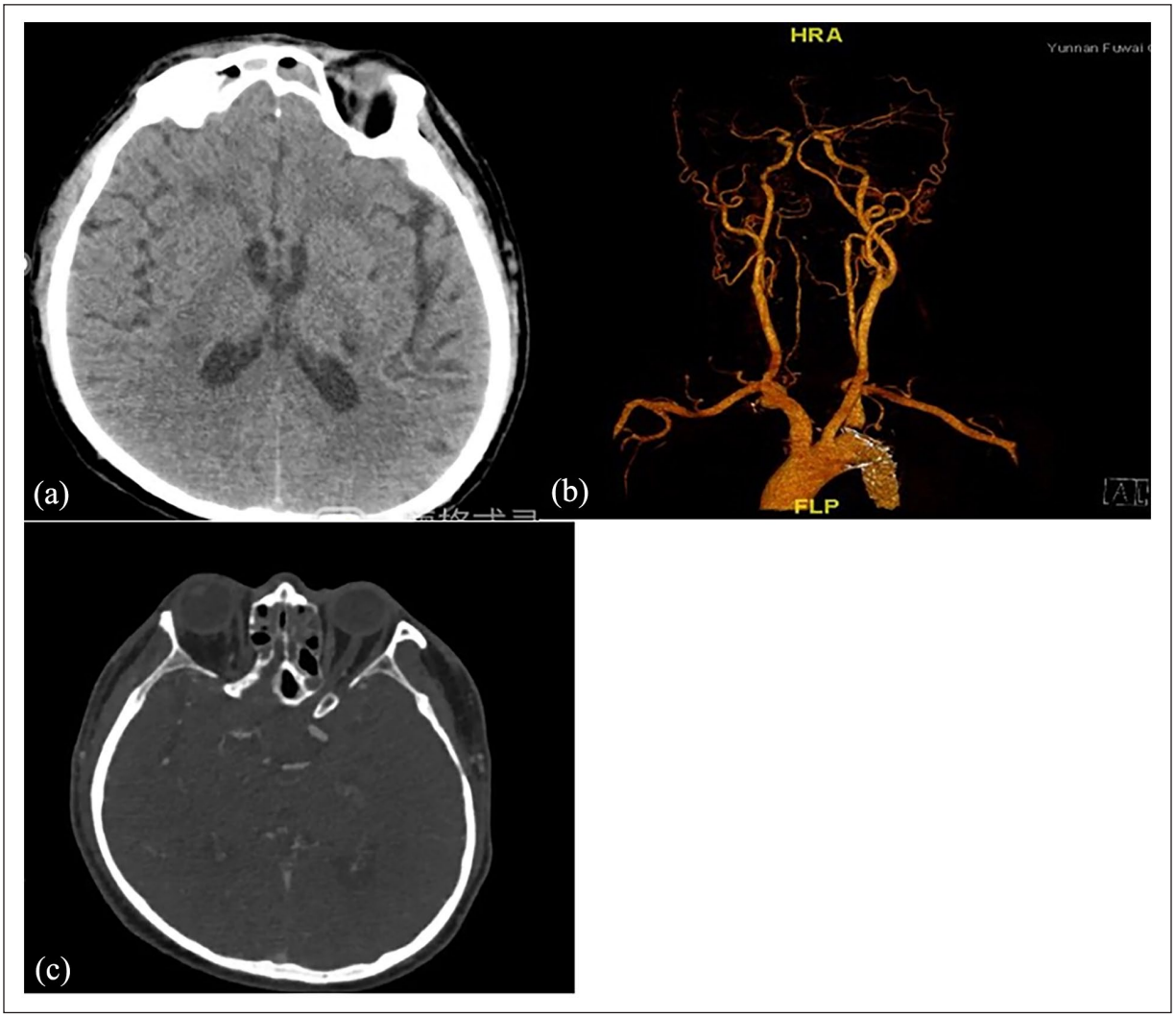

Figure 5. Presents a review of head CT (a) and computed tomography angiography (b and c).
CT: computed tomography.

plain scan. Subsequent reexamination of the head CT revealed the emergence of scattered small spots and flaky low-density shadows in the brain (see Figure 5(a)). CTA results indicated narrowing of the right middle cerebral artery M segment and poor collateral circulation, accompanied by bilateral multiple cerebral infarctions and right middle cerebral artery stenosis (see Figure 5(b) and (c)). Following consultation with neurology experts, it was recommended to limit the treatment course of Argatroban to 7–10 days. In addition, butylphthalide and citicoline were introduced, along with vitamin B1 and mecobalamin tablets. Blood pressure management and rehabilitation efforts were also emphasized.

The patient underwent TEVAR successfully. Postoperatively, he developed neurological deficits, including decreased consciousness and hemiplegia. MRI confirmed cerebral hypoperfusion infarcts. Over 6 months of rehabilitation, the patient gradually recovered neurological function. One year later, the patient returned to the outpatient department of our hospital for a follow-up examination. The comparative CTA revealed the accurate deployment of the stent, with no signs of recurrence, internal leakage, or stent displacement (Figure 6).

Discussion

In this case, blood pressure was not consistently monitored and controlled over an extended period. Cerebral hypoperfusion occurred due to cerebral ischemia and hypoxia following rapid hypotension in the short term, attributed to the patient's substantial body weight and concomitant sleep apnea. This resulted in cerebral infarction, manifested as lethargy, and delayed recovery after general anesthesia. Upon consultation with neurology experts, watershed cerebral infarction caused by cerebral hypoperfusion due to rapid hypotension was considered.[13,14] Similar cases of watershed cerebral infarctions

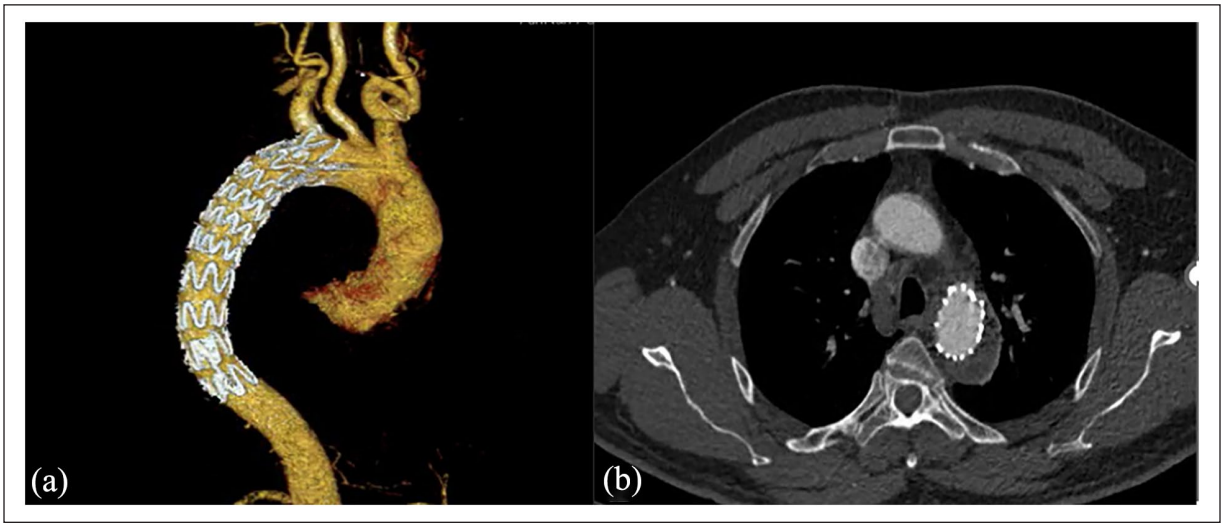

Figure 6. Illustrates the results of a 1-year follow-up (a and b), demonstrating no internal leakage or stent displacement.

due to rapid blood pressure lowering have been reported, emphasizing the need for cautious antihypertensive therapy.[15–19] This case underscores the delicate balance required in managing blood pressure in aortic dissection patients to prevent iatrogenic cerebral hypoperfusion. Imaging, symptoms, and signs supported the diagnosis of watershed cerebral infarction, named primarily based on the site of its onset. It involves two or three cerebral arteries at the end of the transitional zone between brain areas, with the main clinical causes being decreased cerebral blood flow, internal carotid artery stenosis, and collateral circulation dysplasia. Contributing factors include low blood pressure or microemboli resulting from an abnormal clotting state.

Pathophysiologically, cerebral blood flow is generally reduced, leading to diminished perfusion pressure in distal small blood vessels and resulting in infarction in the junction area of cerebral vascular distribution. Similar to other cerebral infarctions, CT reveals low-density areas with clear boundaries. Initial changes in mean arterial pressure are typically compensated by appropriate alterations in arteriolar resistance to maintain cerebral blood flow. However, patients experiencing more pronounced blood pressure fluctuations may eventually lose the ability to self-regulate, resulting in reduced cerebral blood flow (with hypotension) or elevated cerebral blood flow (with pronounced hypertension). These changes are more likely to occur at higher pressure levels in hypertensive patients, presumed to be due to the thickening of small arteries. Consequently, aggressive antihypertensive therapy in potentially hypertensive patients at higher mean arterial pressure can lead to cerebral ischemia.

Conclusion

Individualized assessments are crucial in tailoring patients with type B aortic dissection to tailor antihypertensive regimens. Clinicians should be vigilant for signs of neurological deficits following aggressive antihypertensive therapy.

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Author contributions

Y.Z. Conceptualization, Methodology, Software, Investigation, Formal analysis, Writing—Original draft. H.L. Made important contribution to the revision. Y.G. Conceptualization, Resources, Supervision, Writing—Review and editing.

Data availability statement

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request. Some data may be subject to third-party restrictions. Interested researchers can contact the corresponding author to gain access to the data under the conditions outlined by the data owners.

Declaration of conflicting interests

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Ethical approval and consent to participate

Written informed consent was obtained from the patient for their anonymized information to be published in this article. The study adhered to ethical guidelines and was approved by the institutional ethics committee (Approval Number: 2024-088-01).

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