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SPONTANEOUS AORTIC SEALING WITH TRANEXAMIC ACID IN TYPE A DISSECTION: A NOVEL LIFE-SAVING FINDING

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ABSTRACT

Aortic dissection is a life-threatening cardiovascular emergency, particularly Stanford type A, which typically necessitates urgent surgical intervention. Despite advances in surgical techniques and perioperative care, preoperative bleeding and coagulopathy remain significant challenges. Tranexamic acid, an antifibrinolytic agent, is widely used to minimize perioperative bleeding in cardiovascular surgeries; however, its role in the non-surgical, preoperative stabilization of aortic dissection has not been well established. We present the case of a 56-year-old woman with a history of hypertension and dyslipidaemia who presented with sudden-onset, severe interscapular chest pain. Computed tomography angiography revealed an ascending aortic dissection. Initial management included intravenous labetalol for blood pressure and heart rate control, along with the administration of 1 gram of tranexamic acid.

She was subsequently transferred for emergent surgical repair. Intraoperatively, however, no active aortic bleeding was identified. Instead, a clot was found sealing the dissected segment of the ascending aorta, effectively halting further propagation and haemorrhage. This unexpected finding led to the cancellation of surgical repair. We hypothesize that tranexamic acid contributed significantly to the formation of this stabilizing clot through its antifibrinolytic effects. The patient's clinical outcome was favourable, with no complications observed. This case highlights a novel potential application of tranexamic acid in the emergency management of aortic dissection. To our knowledge, this is the first reported case in which tranexamic acid contributed to spontaneous thrombus formation within a dissected aortic segment, preventing catastrophic haemorrhage and obviating the need for surgery.

KEYWORDS

Tranexamic acid, aortic dissection, antifibrinolytic therapy, surgical repair, chest pain

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LEARNING POINTS

- This case highlights a potentially important role for tranexamic acid (TXA) in the management of aortic dissection. To our knowledge, this is the first reported case of its kind.
- While its established use in reducing postoperative bleeding and transfusion requirements is well documented, our case demonstrates a preoperative benefit, with TXA contributing to the formation of a stabilizing thrombus within the dissected aortic segment.
- This life-saving clot prevented further dissection and haemorrhage, ultimately deferring the need for surgical repair and reducing the risk of perioperative complications.
- We aim to underscore the need for further investigation into the potential role of tranexamic acid in the acute management of aortic dissection.

INTRODUCTION

Aortic dissection, the most common acute aortic syndrome (AAS), occurs when an intimal tear allows blood to enter the aortic media, creating a dissection flap that separates the true lumen from a false lumen. Aortic dissection is classified using two primary anatomic systems: the DeBakey and Stanford classifications. Stanford type A dissection, involving the ascending aorta, is a surgical emergency requiring urgent operative intervention, as the mortality risk is high without prompt surgical repair. Hyperacute dissection is defined as occurring within less than 24 hours of symptom onset[1]. Multiple risk factors have been associated with aortic dissection, primarily hypertension, prior aortic surgery, and aortic aneurysm[2]. Coagulopathy can develop in the preoperative setting of aortic dissection repair due to consumption of clotting factors, increasing the risk of bleeding complications[3]. Management involves rapid blood pressure control to a target systolic blood pressure (SBP) of less than 120 mmHg and a heart rate below 60 bpm[2], along with the use of blood transfusions and blood products to maintain hemodynamic stability and manage bleeding. Tranexamic acid (TXA), an antifibrinolytic agent, has demonstrated efficacy in reducing bleeding in cardiovascular surgeries; however, its role in the preoperative management of aortic dissection has not yet been established in clinical guidelines[4]. We present the case of a 56-year-old woman with a history of hypertension who presented with an ascending aortic dissection and hypertensive emergency. The patient received antihypertensive treatment and TXA and was subsequently transferred for urgent surgical repair. Intraoperatively, the surgical team noted an incidental finding of a clot occupying the dissected segment of the aorta, effectively preventing further bleeding. This case highlights the potential life-saving role of TXA, through its antifibrinolytic properties, in preventing life-threatening haemorrhage. To our knowledge, this is the first reported case of TXA-induced clot formation within a dissected aortic segment.

CASE DESCRIPTION

A 56-year-old woman, known to have hypertension and dyslipidemia, presented to the emergency department with a five-hour history of sudden-onset, severe, interscapular, oppressive chest pain at rest, associated with diaphoresis. The pain occurred in recurrent episodes, each lasting approximately 30 minutes. There was no history of loss of consciousness, focal weakness, fever, chills, cough, vomiting, or hematemesis. On presentation, vital signs revealed a significant difference in blood pressure between the arms: 200/100 mmHg in the left arm and 140/80 mmHg in the right arm. On physical examination, the patient was alert, awake, and oriented. Cardiopulmonary auscultation was unremarkable, with no significant findings. Electrocardiography (ECG) demonstrated a normal sinus rhythm without ST-segment changes *(Fig. 1)*. Laboratory investigations showed a normal white blood cell count and a hemoglobin level of 12.6 g/dl. Cardiac enzymes and D-dimer levels were within normal limits. Computed tomography angiography (CTA) of the chest revealed an ascending aortic dissection *(Fig. 2)*. The patient was started on intravenous labetalol via syringe pump to maintain a target SBP below 120 mmHg and a heart rate under 70 bpm. Additionally, 1 gram of TXA was administered to help control potential internal bleeding. She was then transferred to another hospital for urgent surgical intervention. Intraoperatively, the cardiothoracic surgeon unexpectedly found no active aortic bleeding. Instead, a clot was identified at the site of the dissected aortic segment. This incidental finding played a critical role in the patient's survival, as it prevented internal hemorrhage and ultimately obviated the need for surgical

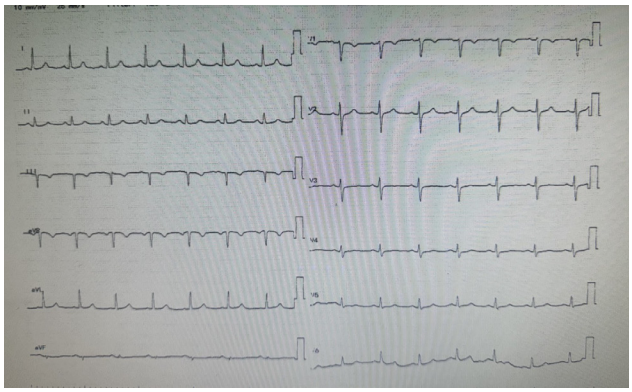


Figure 1. Electrocardiogram showing a normal sinus rhythm with no significant ST segment changes.

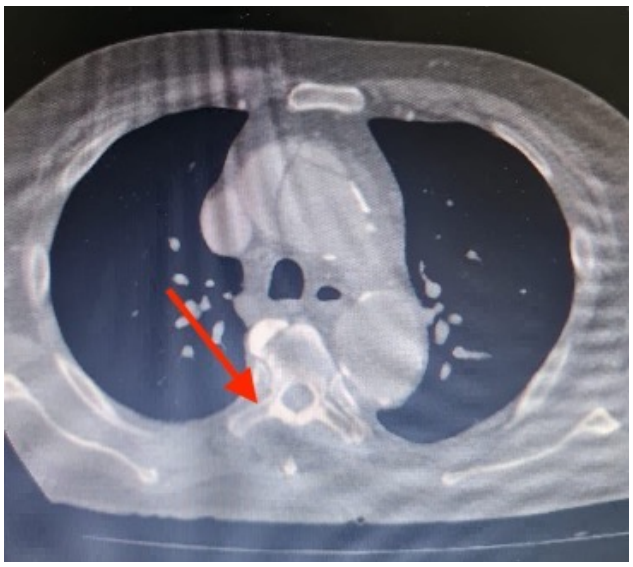


Figure 2. Computed tomography angiography of the chest showing ascending aortic dissection (arrow).

repair. This outcome was attributed to the antifibrinolytic effect of tranexamic acid, which not only reduced the risk of bleeding but also facilitated the formation of a stabilizing clot.

DISCUSSION

Aortic dissection, the most common form of AAS, occurs when an intimal tear allows blood to enter the aortic media, creating a dissection flap that separates the true lumen from a false lumen. This flap can propagate in either direction and lead to life-threatening complications, including acute aortic regurgitation, myocardial ischemia, cardiac tamponade, stroke, or malperfusion syndromes. Malperfusion syndrome results from end-organ ischemia due to inadequate perfusion of the aortic branch vessels. If blood ruptures through the outer wall of the aorta, catastrophic aortic rupture may occur. Dissections are classified by duration into hyperacute (<24 hours), acute (2–7 days), subacute (8–30 days), and chronic (>30 days) phases to guide management strategies. Notably, acute ascending aortic dissection carries an early mortality of 1–2% per hour if left untreated[1]. Two primary anatomic classification systems are used: the DeBakey and Stanford classifications. The DeBakey system categorizes dissections based on the location of the intimal tear and the extent of dissection: type I involves the ascending aorta and extends beyond the arch and descending aorta, type II is confined to the ascending aorta, and type III originates in the descending thoracic aorta, with IIIa limited to above the diaphragm and IIIb extending below it. The Stanford classification simplifies this approach, dividing dissections into type A (involving the ascending aorta, irrespective of the tear's origin) and type B (involving only the descending aorta). Type A dissections are surgical emergencies due to their high risk of complications, while type B dissections are generally managed medically unless complications occur[1].

Multiple risk factors often coexist in patients with aortic dissection including hypertension, genetic and connective tissue disorder. Bicuspid aortic valve, prior aortic surgery, and larger aortic dimensions are more frequently seen in younger patients under 40 years of age[2].

Prompt imaging is crucial for diagnosis, with CTA preferred for its speed and accuracy. In hemodynamically unstable patients, transoesophageal echocardiography is a valuable bedside alternative. While biomarkers such as D-dimer may assist in the diagnostic process, they are not definitive[5]. According to the European Society of Cardiology 2024 guidelines, initial management includes intravenous beta-blockers (such as labetalol or esmolol) as first-line agents. If additional blood pressure control is required, intravenous vasodilators like dihydropyridine calcium channel blockers or nitrates can be added. The target systolic blood pressure is below 120 mmHg, with a heart rate not exceeding 60 beats per minute. Continuous ECG monitoring, arterial line placement, and intensive care unit admission are recommended. Adequate pain management is also essential for achieving hemodynamic goals[2]. Open surgery remains the standard of care for acute Stanford type A aortic dissection and is typically recommended for most patients. While thoracic endovascular aortic repair (TEVAR) has gained favour for suitable lesions in distal aortic dissections, its application in type A dissections remains investigational. A notable complication of endovascular repair is proximal type I endoleak. The primary aims of endovascular repair are to reshape the dissected ascending aorta, seal the entry tear, and reduce false lumen pressure. Complete false lumen thrombosis has been associated with improved long-term outcomes following TEVAR[6].

In patients undergoing thoracic aortic repair for Stanford type A aortic dissection, coagulopathy arises due to factors like preoperative antithrombotic use, consumption of clotting factors, hypothermia, and cardiopulmonary bypass[4]. These patients often develop disseminated intravascular coagulation of the enhanced fibrinolytic type, characterized by severe bleeding, elevated thrombin-antithrombin complex, plasmin-α2 plasmin inhibitor complex, fibrin degradation products (FDP), D-dimer (with a high FDP/D-dimer ratio), and reduced fibrinogen, platelet count, and α2-plasmin inhibitor activity[3]. Targeted transfusion strategies with products like fibrinogen concentrates, platelets, or prothrombin complex concentrates is recommended. Additionally, TXA is recommended to control bleeding by inhibiting fibrinolysis[4]. TXA has demonstrated efficacy in minimizing perioperative bleeding and transfusion requirements during thoracic aortic surgery without increasing the risk of thrombotic events. In a double-blind, randomized controlled trial of patients undergoing thoracic aortic surgery, those who received TXA had significantly lower postoperative bleeding volumes and reduced transfusion needs compared to placebo, both within the first 4 hours and overall. Notably, there was no observed increase in thrombotic complications in the TXA group[7]. These findings were corroborated by a meta-analysis conducted by Zhang et al.[8], and in a separate study conducted by Ahn

et al.[9]. Regarding dosing, a retrospective study by Guo et al. demonstrated that a high-dose TXA regimen (>50 mg/kg) significantly reduced postoperative bleeding and transfusion requirements without increasing adverse events, compared to lower doses[10].

In our case, the patient presented with an ascending aortic dissection, a recognized surgical emergency. We initiated guideline-directed medical management, including strict blood pressure and heart rate control. Notably, the administration of TXA did not only reduce the risk of perioperative bleeding and minimized the need for blood transfusions but also appeared to promote thrombus formation at the site of the dissection. This spontaneous clot effectively sealed the dissection, prevented internal haemorrhage, and obviated the need for surgical intervention. To our knowledge, this is the first reported case describing such an outcome. Although currently not addressed in existing management guidelines, this case highlights a potentially valuable role for TXA in the management of aortic dissections, warranting further investigation.

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