CASE REPORT

Total occlusive thrombosis of the abdominal aorta following holmium laser lithotripsy and ureteroscopy

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ABSTRACT

Abdominal aortic thrombosis (AAT) is a rare condition with substantial morbidity and mortality risk if not treated promptly. AAT typically occurs in postoperative patients, particularly those with certain risk factors. Here, we present the case of a urology patient who underwent ureteroscopy and laser lithotripsy for ureterolithiasis. During the postoperative period, the patient was re-admitted with a massive intra-abdominal arterial thrombosis involving the iliac and renal arteries and abdominal aorta. Emergency thrombectomy, angioplasty, and catheter-directed thrombolysis were performed in multiple stages. After a complex postoperative course in the intensive care unit, the patient survived and was discharged in stable condition.

KEYWORDS: abdominal aortic thrombosis; occlusive thrombosis; abdominal aorta; laser lithotripsy; ureteroscopy

INTRODUCTION

Abdominal aortic thrombosis (AAT) is a serious and potentially life-threatening condition. While AAT commonly occurs postoperatively, it is rarely observed after urological procedures. While virtually all surgical procedures increase the risk of venous thromboembolism (VTE) in the perioperative period, the potential association between ureteroscopy, laser lithotripsy, and vascular thromboembolism remains unclear. Several surgical and patient-related risk factors for AAT have been identified. Patients with diabetes, hypertension, ischemic heart disease (IHD), peripheral vascular disease, a history of heavy smoking, or malignancy are at higher risk. Meanwhile, prolonged surgical duration (greater than two hours) and extended postoperative bedrest (more than four days) also contribute [1]. Routine, same-day endoscopic urological procedures are rarely associated with VTE, with an incidence below 2% [1,2]. However, the risk in these patients increases with prolonged ureteroscopy duration and the use of the lithotomy position [2,3]. Therefore, thromboprophylaxis with low-molecular-weight heparin (LMWH) and mechanical stockings is strongly recommended for high-risk patients during the perioperative period until complete mobilization is achieved [4].

Among the recognized forms of AAT, aortic mural thrombosis is an extremely rare postoperative complication; it has not been reported previously following ureteroscopy. Prompt diagnosis is necessary to prevent major ischemic complications to the abdominal viscera and lower limbs. If not diagnosed at an early stage, occlusive aortic thrombus can be fatal [5-7].

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CASE PRESENTATION

A 41-year-old male with type-2 diabetes, hypertension, and IHD presented to the emergency department one week after ureteroscopy and laser lithotripsy for a right-sided mid-ureteric stone. The patient had complaints of severe back pain, nausea, and vomiting. The pain was radiating to both lower limbs and was associated with significant weakness in both legs. The patient reported no history of trauma, fever, dysuria, or hematuria. The patient's vital signs were as follows: heart rate 130/min, blood pressure 150/90 mmHg, O_2 saturation 98%, respiratory rate 20/min, and temperature 36° C. Both lower limbs were dusky in color; bilateral peripheral pulses of the femoral, popliteal, and dorsalis pedis arteries were absent. An echocardiogram (ECG) showed sinus tachycardia with evidence of previous anterior ischemic changes. At this time, lower limb ischemia was strongly suspected, and an urgent computed tomography angiography (CTA) of the chest, abdomen, and lower limbs was performed. Laboratory results showed a normal CBC of Hb 15.3 g/dL (ref. value:12–17.5 g/dL), WBC 4.6 x10^9/L (ref. value: 4-10 x10^9/L) and platelet count 287 x10^9/L (ref. value: 150-410 x10^9/L). The chemistry panel showed normal electrolytes with mildly elevated creatinine 109 μmol/L (ref. value: 44-115 μmol/L). Creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) enzymes were markedly

elevated. CPK level was 58,155 IU/L (ref. value: 22-200 IU/L) while LDH level was 1,793 IU/L (ref. value 140-280 IU/L).

CTA showed that the distal part of the abdominal aorta was totally occluded, with a massive intramural thrombus that extended distally to both iliac arteries (Figure 1). Further thrombosis was detected in both the renal and splenic arteries, resulting in a subtotal infarction of the right kidney, a segmental infarction of the left renal lower pole, and a wedge-shaped infarction of the mid-part of the spleen (Figure 2A, 2B). Notably, the scan did not reveal any evidence of dissection in any aortic division.

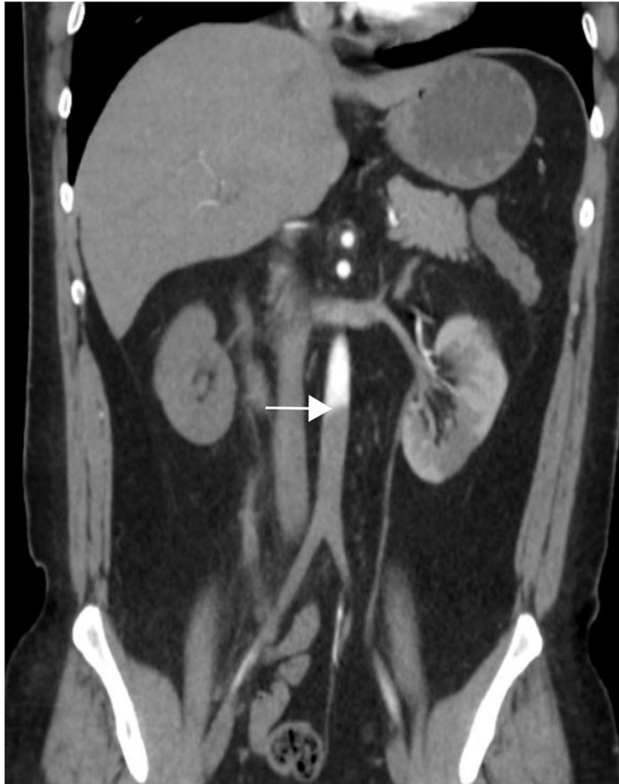

Fig. 1. Abdominal computed tomography angiography. The coronal view shows total occlusion of the abdominal aorta with massive intraluminal mural thrombus (white arrow) extending to both iliac arteries.

A vascular surgeon and endovascular interventionist then assessed the patient to determine the need for emergency vascular intervention. The physicians met with the family to discuss the patient's critical condition and appropriate treatment. The patient subsequently underwent an emergency bilateral lower limb angioplasty that consisted of a thrombectomy, balloon angioplasty, and catheter-directed thrombolysis (CDT) of the aortic thrombus, with a bolus dose of 5 mg. This was followed by an infusion with tissue plasmin activator (0.5 mg/hour). Systemic intravenous infusion of unfractionated heparin was initiated at 500 IU/hour, alongside oral antiplatelet therapy of aspirin and clopidogrel. The patient was then transferred to the intensive care unit (ICU) for monitoring under a multidisciplinary team comprising a vascular surgeon, an endovascular interventionist, a nephrologist, and a hematologist.

On the third day, the patient was moved to a catheterization lab for stage II pharmaco-mechanical thrombolysis. An abdominal angiogram showed patency of the distal aorta and iliac vessels, confirming their successful recanalization after CDT (Figure 3).

During the ICU stay, the patient's kidney function deteriorated secondary to the infarction and was followed closely by the nephrology team. Creatinine increased to 409 mmol/L but improved with intravenous hydration. One week later, the patient's general condition was stable enough to be downgraded to the regular surgical floor. The patients' oral anticoagulant therapy began with 5 mg warfarin daily. The patient subsequently showed dramatic improvement, and he was discharged in stable condition.

DISCUSSION

Etiology of AAT

Aortic thrombosis can occur in any part of the aorta, including the ascending and descending sections and the

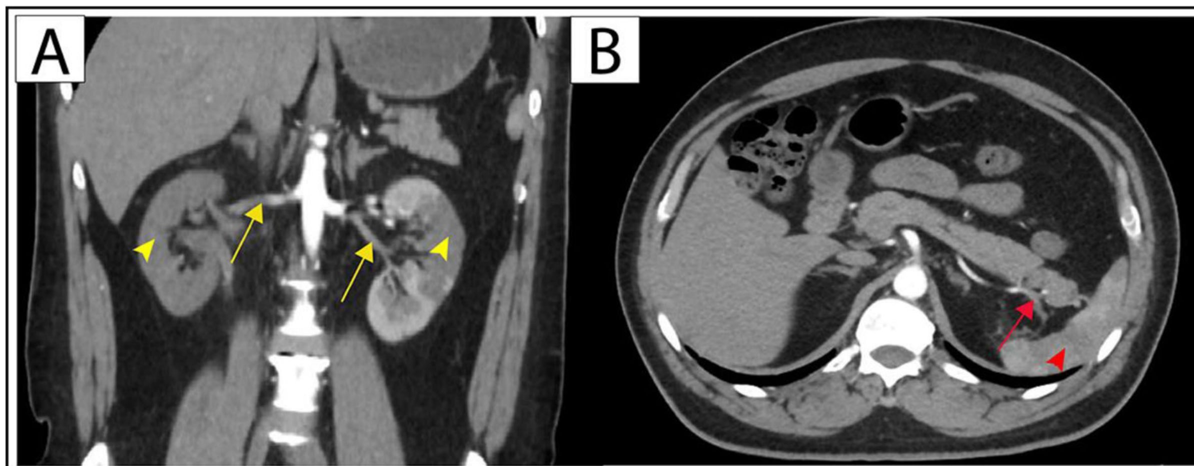

Fig. 2. (A): Extensive thrombosis of the renal arteries on both sides (yellow arrows) that led to subtotal infarction of the right kidney and segmental infarction of the left renal lower pole (yellow arrowheads). **(B)**: Thrombosis of the splenic vein (red arrow), causing a wedge-shaped infarction of the spleen (red arrowhead).

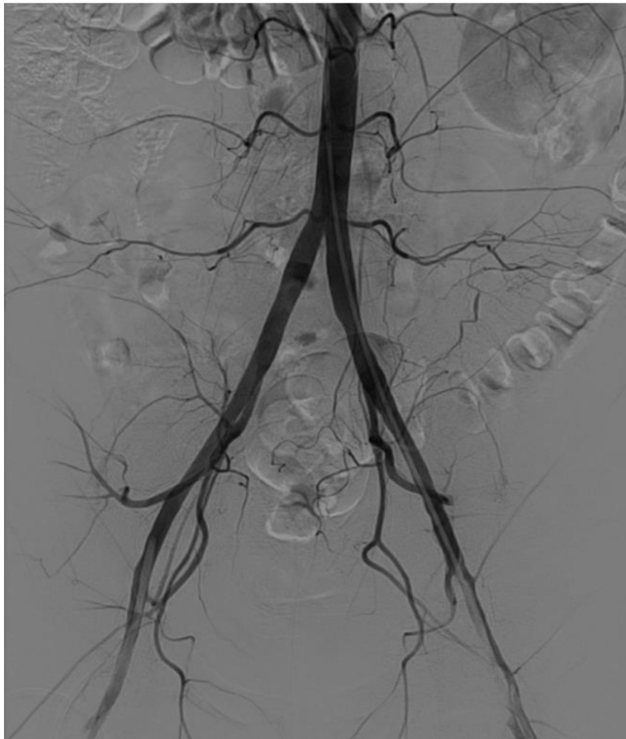

Fig. 3. Post-pharmacomechanical thrombolysis and angioplasty. The abdominal angiogram showed patency of the distal aorta and iliac vessels, confirming successful recanalization of the aorto-iliac vessels after catheter-directed thrombolysis.

aortic arch [8]. Structural abnormalities of the aortic lumen, such as dissection, aneurysm, or severe atherosclerosis, may predispose patients to thrombosis [9]. In general, the risk of venous and arterial thrombus formation is higher in patients with diabetes, renal impairment, IHD, and those with a history of heavy smoking. Arterial thrombi are also associated with several conditions, such as vasculitis, trauma, malignancy, and thrombophilic disease [10].

Hypercoagulable hematological states, such as polycythemia or thrombocytosis, can potentiate aortic thrombosis. Thrombocytosis is associated with small and medium blood vessels, but has been rarely reported in relation to large vessel thrombosis.

Malignant disease remains a major risk factor for VTE. Complex mechanisms of cancer-associated thrombosis are not well understood, but are believed to result from multiple factors. These include secretion of tissue factors, mucins, and cysteine proteinases from carcinoma cells that enhance platelet aggregation and fibrin formation [11,12].

Other systemic diseases associated with aortic thrombosis include autoimmune diseases, such as Systemic Lupus Erythematosus and Takayasu's syndrome [13].

Stimulation of thrombogenesis has also been observed in multiple cases of nephrotic syndrome, due to hypoalbuminemia and urinary loss of plasminogen, protein C, Protein S, and antithrombin-III. This metabolic disruption leads to increased hepatic synthesis of thrombogenic proteins, including fibrinogen, factor V, von Willebrand factor, factor VII, and alpha-1 macroglobulin, increasing the risk of thromboembolism [14].

The current case study patient had multiple risk factors, including diabetes, hypertension, IHD, and smoking. Nevertheless, surgery could have been the precipitating factor that caused the formation of the aortic thrombus. The patient had a prolonged course of intractable renal colic due to an impacted ureteric calculus; this was managed surgically in an extensive ureteroscopy procedure.

Laser Lithotripsy and vascular injury

Laser lithotripsy for stone ablation has previously been associated with aortic thrombosis [15]. In a case similar to that presented here, Kerenick et al. reported a challenging case of a 42-year-old female who developed an aortic arch thrombosis after laser lithotripsy for stone ablation. Flexible ureteroscopy with laser lithotripsy is considered one of the most minimally invasive and safe surgical methods for the treatment of renal calculi. Nevertheless, to date, there has been no clear explanation of how this procedure can cause vascular injury or thrombosis in large vessels. Renal artery pseudoaneurysm has also been reported in the postoperative period following laser lithotripsy [16].

These reports suggest that the procedure itself can induce vascular injury, beginning at the renal parenchyma and propagating to other blood vessels. Thermal waves associated with vibration may contribute to this vascular injury, but the exact mechanism remains to be investigated.

Clinical Presentation and Management of AAT

Aortic thrombosis can be asymptomatic and may only be discovered incidentally on an angiographic aortic scan. However, AAT may present with cerebrovascular, visceral, or peripheral embolism [7,17]. Interestingly, once a thrombus forms, it can propagate in either a descending (caudal) or an ascending (cephalad) direction.

Management of occlusive and non-occlusive aortic thrombosis is challenging and requires a multidisciplinary team, primarily including vascular surgeons, endovascular interventionists, hematologists, and intensivists. Patients usually require admission to the ICU and must be monitored closely for complications in the perioperative period. Therapeutic options include anticoagulation, intra-arterial thrombolytics, endovascular stenting, and vascular reconstructive surgery of the aorta or thrombectomy, with or without embolectomy.

These various options are controversial, and treatment decisions vary according to surgical and institutional preference. Alhan et al. reported a similar case of occlusive aortic thrombosis in the distal aorta in a 53-year-old female patient with a history of heavy smoking but a negative thrombophilia workup. The patient was successfully managed using an endovascular stent and three months of postoperative anticoagulation [18]. Endovascular treatment has emerged as the preferred option over the last decade; advances in such interventions are associated with a high technical success rate, low morbidity and mortality, and satisfying short-term patency [19-20].

Patients with recurrent non-occlusive aortic thrombosis should be readmitted and receive complete vascular imaging. If they are not candidates for endovascular intervention, aggressive anticoagulation should be initiated promptly. This usually begins with systemic heparinization, followed by oral anticoagulants, such as warfarin, for an extended period. This process must be closely monitored and

followed up by vascular and hematology services to achieve complete resolution of the thrombus.

CONCLUSION

Aortic thrombosis is a challenging and potentially life-threatening condition with a rapid, progressive clinical course. Treatment options including medical, surgical, and interventional therapeutics should be individualized depending on the patient's general condition and the status of the thrombotic stenosis. All options should be discussed thoroughly with the patient prior to the final treatment decision.

Conflict of Interest

The author declares that there is no conflict of interest regarding the publication of this article.

Consent for publication

Written informed consent was obtained from the patient. No personal information, names, initials or any form of identification are included in the study.

Ethical Approval

Exemption Letter for Research Project No. **E-25-9953**

AI Tools

No AI generative tools had been used in this study.

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