Case – Thrombophlebitis of the inferior mesenteric vein secondary to compression of an adjacent ureteral stone

Josiah Low, Jonathan Berger
Naval Medical Center San Diego, San Diego, CA, United States

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Corresponding author: Dr. Josiah Low, Naval Medical Center San Diego, San Diego, CA, United States; josiah.low39@gmail.com

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INTRODUCTION

KEY MESSAGES

- Deep vein thrombosis presumed secondary to urolithiasis is rare.
- Establishment of drainage with simultaneous anticoagulation is a safe management strategy.
- Attribution of thrombus formation to urolith should not precede formal hematologic workup.

Urolithiasis affects nearly 10% of Americans and can cause pain, severe infection, and renal compromise.[1] Deep venous thrombosis (DVT) is a rarer pathology and is precipitated by the combination of venous stasis, endothelial injury and hypercoagulability (known also as Virchow's Triad).

While extrinsic vascular compression is an established contributor to DVT formation (e.g. secondary to adjacent lymphocele, vessel, tumor,[2–4] etc.), urolithiasis as a primary cause of compression has been described only once.[5] We present the second case of DVT secondary to compression from urolithiasis. The location of this thrombosis and potential gastrointestinal sequelae as well as the difference in drainage method distinguish this case from that which was previously reported. This case report has been prepared according to the Surgical CAse REport (SCARE) Guidelines.[6] The Naval Medical Center San Diego Institutional Research Board (IRB) has deemed this report IRB exempt. Written documentation of consent for publication was obtained from the patient and uploaded to the medical record.

CASE REPORT

A 62-year-old Hispanic man presented to the emergency department with one day of left flank and left lower quadrant pain. His urologic history was notable for a kidney stone that passed spontaneously and stable obstructive voiding symptoms managed with observation. Other

comorbidities included chronic kidney disease stage 3, pre-diabetes mellitus, hypertension and hyperlipidemia. Home medications included atorvastatin, losartan and amlodipine. He had a documented allergy to penicillins. He had no known family history of coagulopathy. He denied any history of tobacco use.

Physical examination demonstrated mild left flank tenderness with an otherwise benign abdominal exam. Initial lab showed an elevated serum creatinine to 1.5 and a urinalysis with greater than 50 red blood cells. A computed tomography (CT) scan with intravenous contrast was obtained given the broad differential with concomitant flank and abdominal pain and demonstrated a 6mm left distal ureteral stone near the distal inferior mesenteric vein (IMV) with proximal hydronephrosis (Fig 1). Abnormally decreased opacification of the IMV was also seen suggestive of IMV thrombophlebitis (Fig 2).

The patient was admitted and therapeutic anticoagulation via intravenous heparin drip was initiated. The following day, his heparin drip was held for eight hours after which treatment of the stone was attempted via ureteroscopy with laser lithotripsy. Extracorporeal shockwave lithotripsy was not considered as the location of the stone within the pelvis would not allow for effective extracorporeal lithotripsy. Semirigid ureteroscopy was therefore performed under general anesthesia. This demonstrated a tortuous left ureter and impacted stone. Due to the inability to safely perform lithotripsy secondary to ureteral tortuosity despite attempts at stone repositioning, an internal ureteral stent was placed and the patient was scheduled for second look ureteroscopy.

The patient was discharged the following day after transitioning his anticoagulation to oral apixaban. Hematology workup for Factor V Leiden, prothrombin gene mutation, JAK2 mutation, and antiphospholipid antibodies was unremarkable. Protein C/S and antithrombin III deficiency testing were not performed due to ongoing therapeutic anticoagulation. Second look ureteroscopy with laser lithotripsy was performed three weeks later after holding anticoagulation for 48 hours preoperatively with successful clearance of stone. Anticoagulation was resumed the following day. Renal ultrasound obtained one month postoperatively demonstrated resolution of hydronephrosis. Without a plausible alternate cause for the patient's thrombosis, the hematology team concurred with the proposed mechanism, deemed this event a provoked DVT, and prescribed a total of three months of anticoagulation.

DISCUSSION

DVT formation is predicated on venous stasis, endothelial injury and hypercoagulability. These are rare sequelae of urolithiasis. We believe the location of this particular stone together with the proximity of the IMV resulted in venous stasis secondary to mass effect, while impaction of the stone and consequent ureteral inflammation resulted in local hypercoagulability absent any systemic hypercoagulable state.

Hassan and colleagues described the first case of a similar process in which an impacted left ureteral stone was associated with formation of a DVT extending from the left common iliac to the left popliteal vein[5]. The location of this stone was correlated to the location of the iliac

vessels via observation of intraluminal pulsatility during ureteroscopy at the level of the stone. Given the severe degree of proximal hydronephrosis seen in their patient, they elected for immediate urinary drainage via percutaneous nephrostomy tube (PCNT) placement with ureteroscopy and stone manipulation one week later.

In our case, preoperative CT imaging was the most convincing evidence of proximity. Intraoperative findings that would support our claim of proximity include documented ureteral tortuosity at the level of the stone during index ureteroscopy (which could suggest deviation from a smoother, straighter course due to an adjacent impeding structure such as the IMV) as well as concordant documentation of stone location by intraoperative fluoroscopy at the distal ureter concordant.

While both PCNT and internal ureteral stent placement can establish upper tract drainage, we believe that the use of an internal ureteral stent has several advantages over PCNT in this unique setting. As one of the risks of PCNT placement is renal bleeding, ongoing anticoagulation is a relative contraindication to placement. Endoscopic intervention, by contrast, has a relatively low bleeding risk and is often performed without holding anticoagulation in patients in whom risk of VTE off anticoagulation is unacceptably high. It also provides an opportunity to potentially treat the stone primarily. In the case presented by Hassan and colleagues, low molecular weight heparin was initiated prior to PCNT placement, and it is unclear if this was held in the immediate preprocedural setting. Severe proximal hydronephrosis was also noted in their case, and this may have facilitated easier percutaneous access to the affected kidney than with the milder hydronephrosis that is typical of an acutely obstructing ureteral stone.

The case we report here is noteworthy not only due to the mechanism of DVT formation but also for the location of this thrombus which raised concerns for concomitant mesenteric ischemia. The presentation of mesenteric ischemia can differ depending on acuity (acute versus chronic) and location of occlusion (arterial versus venous).[7–9] Mesenteric venous thrombosis, such as that seen in our patient, tends to be a chronic process characterized by an insidious onset of abdominal pain over a period of several days to weeks. Management strategies for the above include anticoagulation, endovascular revascularization, and surgical intervention. While our patient did not present with abdominal pain, we maintained a low threshold for general surgery or vascular consultation in the event of worsening abdominal pain inconsistent with renal colic from urinary obstruction.

Our case has several strengths. The involvement of the mesenteric vasculature as a site of DVT in setting of locally compressive urolithiasis is previously unreported in the literature. We also present radiographic evidence of the adjacency of the IMV to the purported compressive ureteral stone rather than inference of adjacency via ureteroscopic observation. Weaknesses include absence of radiographic confirmation of thrombosis resolution and, while convincing, ultimately circumstantial evidence of causation between our patient's ureteral stone and development his IMV thrombus.

Comparison of the management strategies and overall successful outcomes in both our case and that reported by Hassan and colleagues yields several takeaways. The first is that immediate therapeutic anticoagulation is a practical and effective first step that can be initiated with minimal delay. The second is that prompt establishment of upper tract drainage, preferably via ureteral stent placement, allows for protection of renal function and relief of renal colic without undue bleeding risk if performed with appropriate cessation and resumption of anticoagulation. Finally, the importance of multidisciplinary care is underscored to ensure proper evaluation of any occult coagulopathy and to facilitate prompt operative intervention if indicated.

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FIGURES AND TABLES

Figure 1. Representative coronal slice of presenting computed tomography abdomen/pelvis with IV contrast, portovenous phase. Red arrow indicates ureteral stone. Green circle drawn around inferior mesenteric vein (IMV). Note proximity of the stone to the IMV.

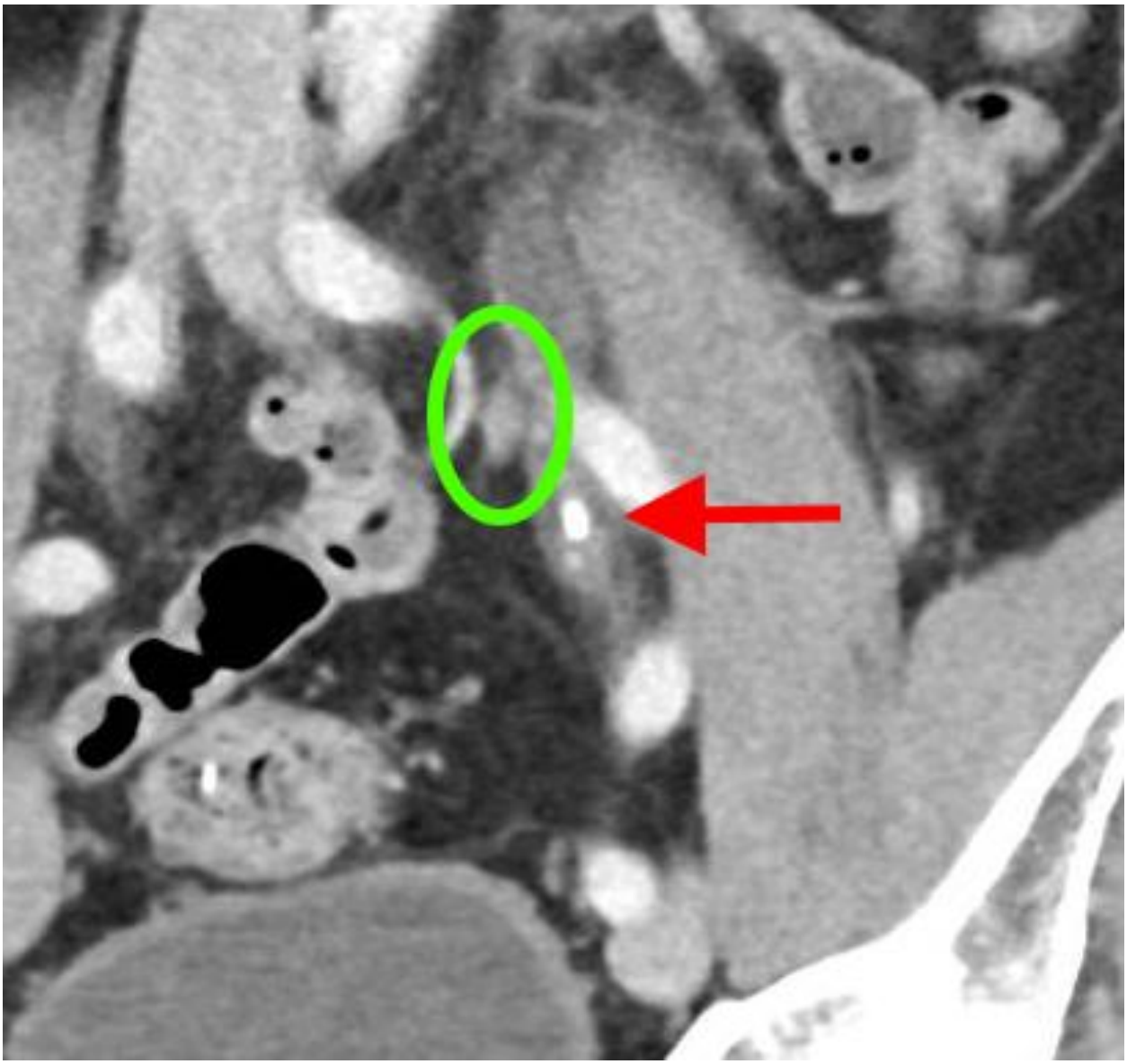

Figure 2. Representative coronal slice of presenting computed tomography abdomen/pelvis with IV contrast, portovenous phase. Red arrow indicates splenic vein. Green arrow indicates insertion of inferior mesenteric vein into the splenic vein. Note difference in enhancement of these structures.