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Case Report

Large floating thrombus in the inferior vena cava

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ABSTRACT

A 32-year-old female presented to the emergency room with severe right loin pain. Abdominal color Doppler ultrasound (CDUS) observed a large thrombus in the inferior vena cava (IVC). Computed tomography with contrast described a large floating thrombus in the IVC with no visible wall attachment. Emergency transjugular suprarenal IVC filter was inserted and overnight infusion thrombolysis over 24 h. The patient started to show constitutional symptoms of pyelonephritis such as high-grade fever and pyuria post-thrombolysis. Immediate empiric antibiotic coverage was started. Overnight infusion thrombolysis resulted in resolution of the floating thrombus and the IVC filter was removed in the next day. Urine culture grew *Escherichia coli* and tailored antibiotic therapy was completed. Three months CDUS confirmed complete resolution of the IVC thrombosis.

Keywords: Angiography, endovascular therapy, pulmonary embolism

Background

Thrombosis of the inferior vena cava (IVC) is an under-recognized with a variable clinical presentation. Pulmonary embolism is a fatal consequence of IVC thrombosis, particularly when it is acute and floating.[1,2] The clinical outcome of IVC thrombosis is often determined by the underlying condition that initially caused the thrombosis. Extension or veno-venous embolization from lower limb deep vein thrombosis is the most common cause of IVC thrombosis. *In situ* IVC thrombosis is a rare entity of IVC thrombosis and most likely related to a localized disease either inside the IVC wall or compression outside the IVC wall. Acute pyelonephritis is a rare cause of IVC thrombosis. A small number of case reports are published, with a couple of cases identifying *Klebsiella pneumoniae* as the causative pathogen.[3-7] This case report describes *Escherichia coli*, the most common cause organism for pyelonephritis, causing a large floating IVC thrombosis successfully treated endovascularly.

Case Report

A 32-year-old healthy female presented to the emergency department complaining of severe right loin pain. The loin pain started 10 h ago and got progressively severe until the time of presentation. There is no history of fever or abnormal color of urine. She has medical history of recurrent urinary tract infections and pelvic inflammatory disease mainly during her pregnancies. She had two uneventful pregnancies and both cesarean section due to breach presentation. She had a history of C-section 3 months back.

Physical examination at presentation revealed a temperature of 37°C, blood pressure: 100/70 mmHg, respiratory rate: 26 breath/min, and heart rate 100 beat/min. Abdominal examination was remarkable for tenderness in the right hypochondrium; otherwise, the abdomen was soft, normal bowel sounds, and no rebound tenderness. She had normal cardiac and respiratory examination. Neurological examination was grossly intact.

Complete blood count showed leukocytosis (23,000 WBC/mm^3) with the left shift (neutrophils, 20,880/mm^3) otherwise normal hemoglobin and platelet levels. C-reactive protein was high (300 mg/L, normal value <5 mg/L). Kidney and liver function tests were normal. The blood sugar was normal. Chest X-ray was unremarkable. Color Doppler ultrasound abdomen showed echogenic mass in the IVC, consistent with floating thrombus at the level of the liver with patent renal veins and hepatic vein [Figure 1].

Emergency consultation to the vascular team was done. The floating thrombus was large enough to be potentially fatal; however, the exact cause of such floating thrombus was not known at that time. Therefore, emergency computed tomography (CT) of the abdomen and pelvis with contrast was done as well as venous duplex ultrasound of both lower limbs. The CT scan showed a large filling defect with surrounding contrast medium, consistent with floating thrombus of the IVC at the level of juxtarenal level with radiological signs of the right-sided severe pyelonephritis. Few cuts through the lung showed a suspicion of pulmonary

embolism in both lung bases [Figure 2]. The venous duplex ultrasound showed no evidence of deep vein thrombosis in both lower limbs.

Empiric parenteral antibiotic (amikacin and ceftriaxone) was started after obtaining septic workup including blood culture and urine culture. The vascular team opinion was to start anticoagulation and endovascular therapy because the IVC thrombus was floating with evidence of subsegmental pulmonary embolism. Transthoracic echo showed normal study notable for normal right ventricular size and function and normal estimated pulmonary artery pressure.

The procedure was started by the right internal jugular vein access under ultrasound guidance under local anesthesia. The ultrasound was very crucial to minimize the risk of access site bleeding that can be fatal in the setting of thrombolytic therapy. The internal jugular access was chosen over femoral access to minimize the risk of embolization of the floating thrombus while crossing by the IVC filter and its own sheath. A Celect (Cook Medical™, Bjaeverskov, DENMARK) IVC filter is chosen due to shorter height to position it just below the renal vein with no pressure exerted near the pericardial space to avoid pericardial injury. A 10 mm infusion catheter was placed across the IVC filter within the floating thrombus, Figure 3. Then, 10 mg tissue plasminogen activator (t-PA) was infusion over 10 min followed by infusion of thrombolytic therapy at a rate of 1 mg/h and to follow the institutional protocol for infusion thrombolysis therapy [Table 1]. To note at night, the patient started to manifest the constitutional symptoms of sepsis with high-grade fever (40°C).

The patient was brought back to the catheterization laboratory after 24 h of thrombolytic therapy. The infusion catheter is confirmed by fluoroscopy to be in place. Then, it was exchanged over 0.035″ Terumo soft guidewire together with the pre-exiting sheath to 45 cm destination sheath. Abdominal ultrasound showed no visible thrombus in the IVC filter or IVC. In addition, venogram through 5-Fr pigtail catheter showed no significant filling defect within the IVC filter. In addition, addition thrombectomy using 8-Fr Pronto thrombectomy catheter retrieved no thrombi. Therefore, the IVC filter was removed using the 6-Fr destination sheath to avoid seeding the microorganism over the IVC filter. The patient tolerated the procedure very well and the sheath was removed immediately. Ultrasound after removal of the IVC filter is performed and showed small visible thrombus in the juxtarenal IVC [Figure 4].

Table 1: Pharmacomechanical thrombolysis protocol

Pharmacomechanical thrombolysis protocol
t-PA 1 mg/h, repeat CBC and fibrinogen 6 h later, then every 12 h in case of no concerns of active bleeding
In case of fibrinogen dropped to <200 mg/dl, consider reducing the dose of t-PA by 50%
In case of fibrinogen dropped to <100 mg/dl, call the physician operator immediately
Unfractionated heparin 500 units/h. Repeat aPTT 2 h later, then every 6 h and follow the low-intensity unfractionated heparin protocol. Note, repeat aPTT 2 h after each change of infusion rate of heparin
Check vital signs, pedal pulses, limb sensation, and observe the site for bleeding, hematoma, and color every 15 min for 1 h then hourly for 4 h
If there are any signs of hematoma, bleeding, severe oozing, unexplained hypotension, or hypertension after sheath removal, please notify the physician immediately

t-PA: Tissue plasminogen activator

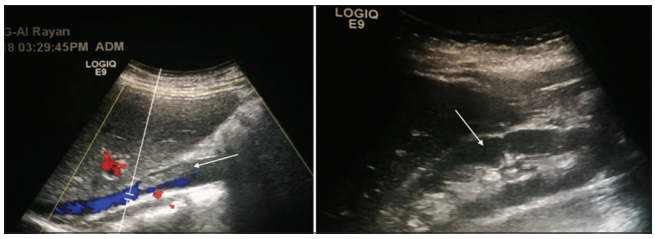

Figure 1: Ultrasound abdomen showed large floating thrombus in the juxtarenal inferior vena cava, see arrow. There is evidence of the right kidney congestion suggestive of pyelonephritis with perinephric fluid collection, see arrow

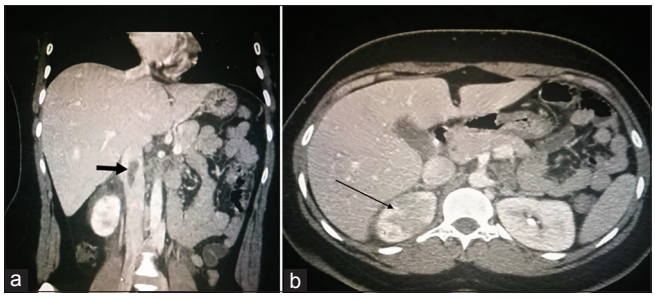

Figure 2: (a and b) Computed tomography showing large floating thrombus in the juxtarenal inferior vena cava, see arrow. The right kidney pyelonephritis, see arrow

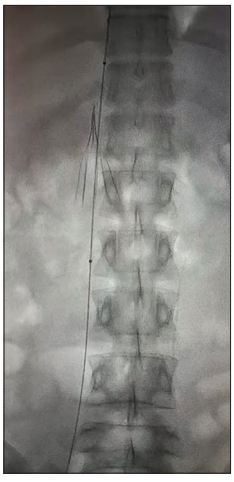

Figure 3: Fluoroscopic image of suprarenal inferior vena cava (IVC) filter (CellCept) showing the infusion catheter (Cragg-McNamara) 10 cm length within the IVC filter

One day later, urine culture grew *E. coli* and the antibiotic therapy was tailored according to the sensitivity. She completed 4 days of amikacin therapy and 10 days of ceftriaxone. Anticoagulation using rivaroxaban 15 mg twice daily (BID) for 3 weeks then 20 mg OD for 3 months. Abdominal ultrasound after 3 months of anticoagulation showed no more thrombus in the IVC [Figure 5]. The D-Dimer was normal; therefore, the anticoagulation was stopped after 3 months of anticoagulation therapy using rivaroxaban. The patient was screened for hypercoagulable state, which was negative/normal (plasma protein C and S, factor V, anti-thrombin III anti-phospholipid, and anti-cardiolipin antibodies).

Discussion

This case report illustrates a rare but lethal complication of acute severe pyelonephritis. Inflammation is closely related to hypercoagulability state. Pulmonary embolism is a lethal consequence of vein thrombosis if not recognized in a timely fashion and dealt with caution due to the longer delay of diagnosis the higher cost of treatment in terms of risk of complications of any form of therapy. To the best of our knowledge, this the first case report of IVC thrombosis presentation for *E. coli* acute pyelonephritis treated endovascularly.

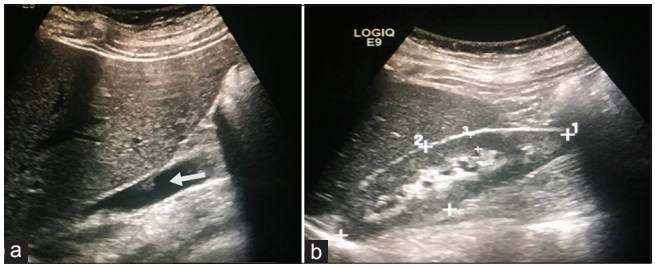

Figure 4: (a and b) Abdominal ultrasound right after pharmacomechanical thrombolysis showed significant reduction of the inferior vena cava thrombus, see arrow. There is also marked reduction of the right kidney congestion, see arrow

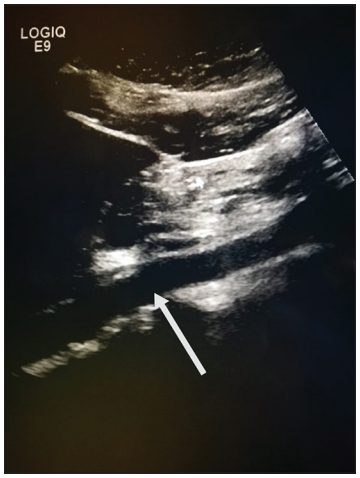

Figure 5: Abdominal ultrasound showed no visible thrombus in the inferior vena cava, see arrow

Gram-negative bacteria release endotoxin into bloodstream, the lipopolysaccharide can change endothelial lining of blood vessels from an anticoagulant profibrinolytic surface into one that promotes thrombosis. Bacterial endotoxin potently stimulates expression of the gene encoding tissue factor, a procoagulant molecule that multiplies many fold the activity of coagulation factors VIIa and Xa. Endotoxin also can augment endothelial cell production of the fibrinolytic inhibitor plasminogen activator inhibitor-1.[8] Acute pyelonephritis is a rare cause of IVC thrombosis. Some may argue that it is a late complication, although thrombosis as a consequence of a concomitant thrombophlebitis of the IVC in pyelonephritis could be a possible explanation.[9] In our case, the diagnosis of acute pyelonephritis was made radiologically by the CT scan findings before the onset of fever by at least 12 h. The local infusion of recombinant t-PA can be considered as a second-line treatment for renal vein thrombosis and/or IVC thrombosis when a satisfactory response is not obtained with anticoagulation.[9] However, in our case, the IVC thrombosis is large enough to occupy 70% of the central IVC lumen with no clear wall attachment, i.e., floating, with 7 cm length in very close vicinity to the heart making it a growing monster that if dislodged would be most likely fatal (pulmonary embolism). Therefore, a decision was made to do local thrombolysis. In addition, the decision of retrievable IVC filter was challenged by two factors: (1) Suprarenal position and (2) presence of infectious milieu. Therefore, a 24 h IVC filter fulfilled the task to trap the thrombus until the infusion thrombolysis acts effectively while we are moving the guidewires, infusion catheter, thrombectomy catheter, and others, such as pigtail catheter inside the floating IVC thrombosis. The IVC filter was removed as soon as possible to prevent seeding of the organism in the metallic frame of the IVC filter.

A limited number of case reports are published describing the association between renal vein thrombosis and IVC thrombosis in the setting of *K. pneumoniae* infection.[4] Bassilios *et al*. described renal vein thrombosis extended to the IVC and right atrium with pulmonary embolism. This was for a 45-year-old diabetic female patient presented with fever and dysuria. The urine and blood cultures grew *K. pneumoniae*. In our case, the presentation was severe right loin pain and the fever started at night of the same admission. Harris *et al*. described renal vein thrombosis in a 62-year-old female with acute pyelonephritis. The urine culture grew *K. pneumoniae*.[5] Our patient was a younger female with no comorbidities and the pyelonephritis was caused by the most common pathogen for urinary tract infection and the IVC thrombosis was isolated with no involvement of renal vein.

Such complication of IVC thrombosis secondary to acute pyelonephritis may have more sinister course. In 1986, Eijsten reported a 54-year-old male who presented with

fever, weight loss, and a painful right upper quadrant mass. A workup confirmed a heterogeneous mass in the right kidney with vessel irregularity and a central calculus. A right nephroadrenalectomy was performed to rule out malignancy with subsequent renal vein and IVC wall removal due to occluding thrombotic masses found intraoperatively. Blood cultures grew *K. pneumoniae*. Pathology revealed multiple renal and perinephric abscesses with extensive thrombosis into the renal vein and IVC wall.[6] Eijsten's case represents the variety and severity of the presentation of *K. pneumoniae*-induced renal vein thrombosis.

IVC thrombosis secondary to pyelonephritis should include antithrombotic management because antibiotic therapy is not enough to prevent IVC thrombosis. It has been reported that the diagnosis of renal vein thrombosis was made 8 days after commencing antibiotic therapy.[7]

In our patient, pharmacomechanical thrombolysis plus antibiotic therapy resulted in rapid resolution of symptoms with short hospital stay. "Previously mentioned collection in the right kidney on the CT scan is not seen on ultrasound" and no floating IVC thrombus is seen.

Conclusion

IVC thrombosis should be suspected as a complication of *E. coli* pyelonephritis which might lead to life-threatening complications, namely, pulmonary embolism and intra-abdominal abscess. Pharmacomechanical thrombolysis with retrievable IVC filter is feasible and effective in the treatment of IVC thrombosis secondary to pyelonephritis. Duplex ultrasound should be considered in case of pyelonephritis and severe abdominal pain to rule out IVC and renal vein thrombosis.

Patient Consent

The patient was consented to have her case published in literature.

Competing Interest

There are no potential conflicts of interest in regard to this case for all authors.

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