Inferior vena cava thrombosis following rectus sheath haematoma

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ABSTRACT
Inferior vena cava thrombosis following benign extrinsic compression has been infrequently described. It responds well to early recognition and management. A rare presentation of inferior vena cava thrombosis following a rectus sheath haematoma, and successful management, in a 46-year-old woman is reported.

Keywords: extrinsic caval compression, inferior epigastric vein rupture, inferior vena cava thrombosis, rectus sheath haematoma

INTRODUCTION
Inferior vena cava thrombosis (IVCT) is linked to a triad of stasis, vessel injury, and hypercoagulability. Benign intra-abdominal causes of external inferior vena cava (IVC) compression as a predisposition to deep vein thrombosis have not been widely described. IVCT secondary to bladder distension, polycystic kidney disease, hepatic cyst and hydronephrosis have been described in case reports, and have been treated successfully with decompression and anticoagulation. A rare presentation of IVCT, secondary to an inferior epigastric artery rupture and rectus sheath haematoma (RSH), is presented.

CASE REPORT
A 46-year-old postmenopausal woman was referred with sudden loss of consciousness and rapidly-progressive swelling of the lower limbs for one day. There was no history of seizures or focal neurological deficits, but she was disoriented and restless following the episode. She was anuric and had haematuria for a day prior to admission. Blood was transfused and she was on ventilatory and ionotropic support on referral. On examination, she was pale with unrecordable blood pressure and had swelling of her lower limbs with darkening and cyanosis. The distal pulses were well felt and no pre-gangrenous changes were observed. An ill-defined suprapubic swelling with irregular margins was felt on abdominal examination, with free fluid and distended superficial abdominal veins on the flanks. A vaginal speculum examination was unremarkable.

Blood investigations revealed haemoglobin 7.7 g/dL, total leucocyte count 18,200/mm³, platelet count 78,000/mm³ and prothrombin time 18 seconds (control 11 seconds). There was acute renal failure with blood urea 104 mg/dL, serum creatinine 3.7 mg/dL, severe metabolic acidosis and serum potassium 7.1 meq/L. Liver function test was suggestive of ischaemic hepatitis. A detailed malignancy screening was negative. Ultrasonography of the abdomen showed a mass measuring approximately 8.4 cm x 8.6 cm x 7.0 cm in the pelvis that was distinct from the uterus. An echogenic thrombus was also seen in the IVC with minimal filling on colour Doppler imaging. The thrombus in the IVC appeared to extend to the left internal iliac and femoral vein. A detailed history did not suggest any previous cannulation or instrumentation of these veins. No history was present to suggest obstructive lung disease, liver disease, inflammatory bowel disease, intravenous drug abuse or trauma. No history of hormone replacement therapy or surgery was present, and she had an uneventful obstetrical history.

Computed tomography (CT) of the abdomen showed a heterogeneous density mass in the left iliac fossa, measuring approximately 11.4 cm x 11.0 cm x 10.6 cm in size, and compressing the left internal iliac vein (Fig. 1). There was an IVC thrombus extending from the bifurcation up to the intrahepatic portion, and involvement of the right renal vein. Fine needle aspiration of the mass yielded blood. Exploratory laparotomy showed a RSH, measuring 10 cm x 10 cm, in the infra-umbilical area,
anti-coagulant, Nicoumalone (Acitrom, Nicholas Piramal, India) after a suitable overlap. The lower limb swelling subsided thereafter. She was on intermittent haemodialysis for six weeks, and improved thereafter with normalisation of renal function after eight weeks. MR imaging done after three months showed recanalisation of the thrombosed IVC, with a part of the thrombus visualised in the subhepatic portion of the IVC (Fig. 3). A normal study of the IVC was seen after two years.

**DISCUSSION**

RSH is associated with blunt trauma in the abdomen and anticoagulation. Rare associations with severe exertion, pregnancy, laparoscopy, insulin injections and coughing have been reported. In the clear absence of any of the preceding causes other than a recent upper respiratory infection, the possibility of a bout of coughing precipitating the RSH was considered. Coughing has been associated with RSH due to intense contraction of the rectus muscle, and consequent tearing and bleeding from the perforating branches of the inferior epigastric vessels within the muscle. Sudden abdominal pain after coughing should alert the physician to this possibility. Ultrasoundographically, these haematomas may be confused with abdominal wall tumours. On CT, a hyperdense mass posterior to the rectus abdominis muscle with ipsilateral anterolateral muscular enlargement is considered characteristic of acute RSH, although chronic RSH may be isodense or hypodense relative to the surrounding muscle.

Berna et al classified RSH into three types using CT, and it has been used in the prognostication and management of RSH. Type I is characterised by unilateral intramuscular haematoma not dissected along fascial planes. Type II is intramuscular with unilateral or bilateral haematoma and blood in between the muscle and transversalis fascia, but not in the perivesical space. In type III, the haematoma may or may not affect the muscle, and blood is seen in the peritoneum and perivesical space with occasional haemoperitoneum. Type III is the most severe and may present as an acute abdomen with anaemia and haemodynamic disturbance. It usually requires hospitalisation and blood transfusion; its resolution may take more than three months.

CT has improved diagnosis and helped avoid the need for surgery in RSH. In a study of 30 patients by Moreno Gallego et al, 22 patients were managed conservatively and eight patients required surgery: four for diagnosis and four for treatment. MR imaging is very useful in the diagnosis of RSH; this is demonstrated as a high signal intensity area on both T1- and T2-weighted images, especially when the CT findings are not specific for RSH. Surgical intervention may be required when the symptoms are severe, as is in the case of a spreading haematoma with

![Fig. 2 Coronal T1-W MR image shows abnormal hyperintense signal within the IVC, suggestive of thrombus, extending from the level of the bifurcation to the subhepatic portion (arrow).](image)

![Fig. 3 Repeat MR image after three months shows recanalisation of the thrombosed IVC, seen as a profuse hypointense flow signal void. A part of the thrombus is still visualised as a soft tissue thickening in the subhepatic portion of the IVC.](image)
haemodynamic instability or an infected haematoma, although the latter may be drained percutaneously (1). IVCT and renal vein thrombosis, secondary to an inferior epigastric artery rupture and RSH, has not been described, to the best of our knowledge. The possibility of thrombosis, induced by compression of the left internal iliac vein and blunt endothelial damage, compounded with vascular stasis, was suspected to be the cause of the IVCT and renal vein thrombosis in this patient. Spontaneous recanalisation was noticed after surgical evacuation of the haematoma and anticoagulation. Renal function was normalised with resolution of the renal vein thrombosis. IVCT secondary to extrinsic compression has significant complications, and early recognition with imaging and management with decompression and anticoagulation are successful.

REFERENCES