

## IVC thrombosis: An unusual complication of metastatic prostate cancer

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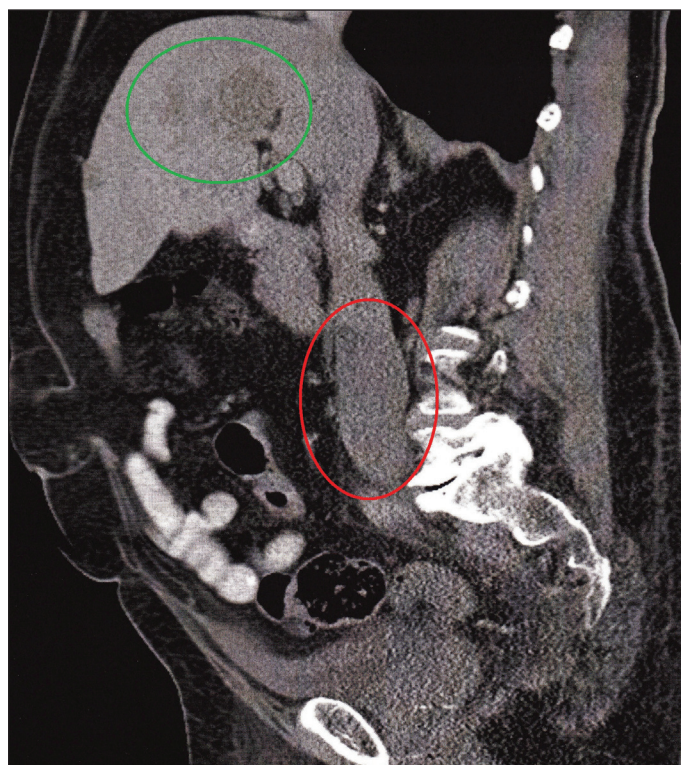
This case report identifies an IVC thrombosis in a patient with stage IV prostate cancer. The case demonstrates hypercoagulability as one of the many complications of malignancy. The patient presented clinically with bilateral pitting oedema to the groin and into the scrotum with dilated superficial abdominal veins. The prostate cancer was aggressive and unresponsive to anti-androgen therapy and brachytherapy. The latest staging CT and bone scans revealed diffuse disseminated disease and a caval thrombus. He is now receiving chemotherapy as an outpatient and unfortunately his prognosis is unfavourable.

### Case Introduction

Hypercoagulability is a known complication of malignancy and renal vein extension from renal cell carcinoma into the inferior vena cava (IVC) has also been extensively reported. However, IVC thrombosis (IVCT) is a rare complication of metastatic prostate cancer. Clinically, the venous obstruction presented with rapid onset bilateral pitting oedema extending to the groin. There were visible superficial abdominal veins. The swelling also caused pain and discomfort to the patient. The imaging appearances on ultrasound and computed tomography (CT) will be described in order to recognise this complication of malignancy.

### Case Presentation

MA, a 57-year-old male retired nurse, presented to the medical oncology department with gross bilateral pitting oedema extending into the scrotum. The swelling began suddenly two days prior to his



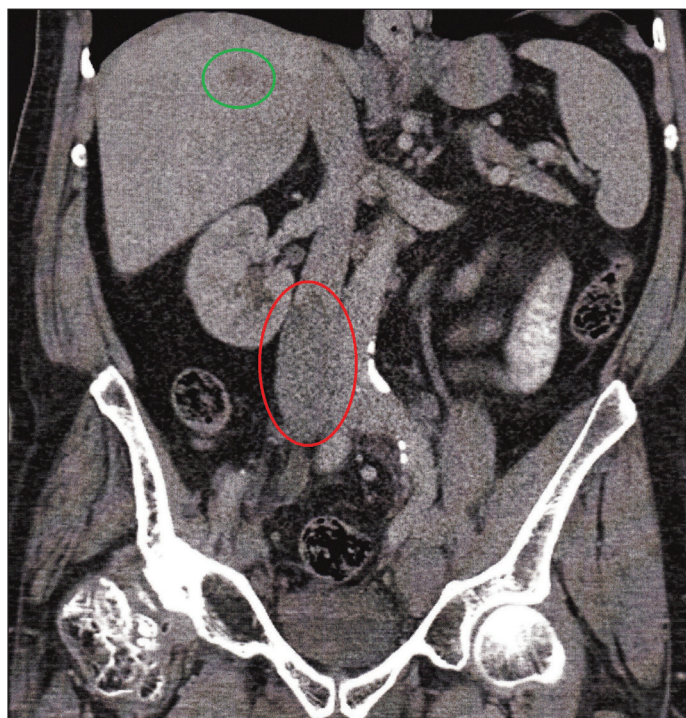
**Figure 2.** Contrast enhanced abdominal CT scan: sagittal section. This image demonstrates the ovoid hypodense filling defect in the expanded infra-renal IVC (red circle). Note also the low attenuating metastatic deposits in the liver (green circle).

consultation.

The patient had a past medical history of non-Hodgkins lymphoma in 1992 and prostate cancer diagnosed in 2008. During this time he had experienced increased frequency, dysuria and poor urinary stream. His prostate cancer was diagnosed in 2009 after a PSA level of 7.2 ng/mL was found. Confirmatory prostate biopsy revealed high-grade adenocarcinoma with a Gleason score of 9. There were no obvious metastases found. He was treated with high dose brachytherapy to the prostate but despite this therapy his PSA continued to rise. In late 2010 his PSA had risen from 7 to 35.

A staging CT abdomen/pelvis and bone scan were performed at this time and showed only an enlarged prostate gland and degenerative changes in the lumbar spine and left hip. In 2011 the CT abdomen/pelvis and bone scans were repeated and revealed diffuse disseminated disease (stage IV). This included small nodules at the lung bases, extensive liver lesions and diffuse abdominal and pelvic lymphadenopathy. In addition, a caval thrombus was demonstrated (Figures 1 and 2) and confirmed with duplex Doppler ultrasound (Figure 3a). A direct comparison with a normal anechoic IVC in longitudinal section was also made (Figure 3b).

The thrombus was almost completely occlusive within the distal infrarenal IVC and extended into the right common iliac vein. Hepatic metastases and lymphadenopathy around the iliac vessels were again noted.

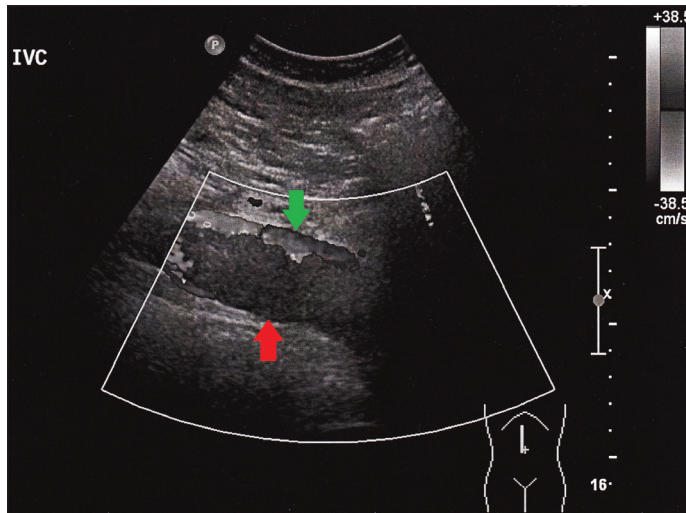


**Figure 1.** Contrast enhanced abdominal CT scan: coronal section. This image demonstrates the ovoid hypodense filling defect in the IVC distal to the renal veins. The thrombus is expanding the cava (red circle). Note also the hypodense metastatic deposit in the liver (green circle).

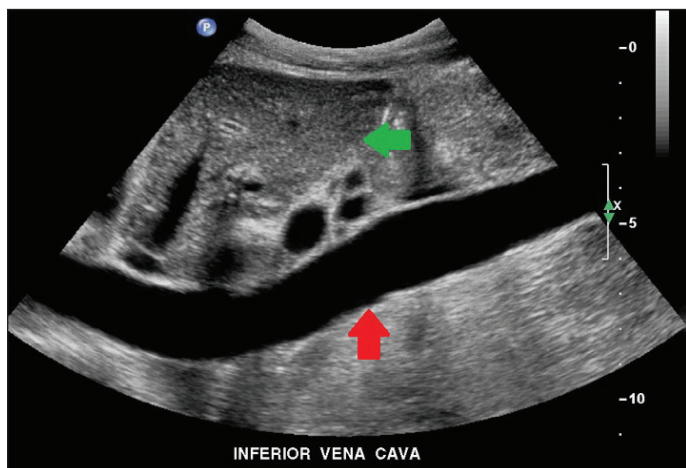
MA's medications included bicalutamide, leuprorelin, paracetamol, ibuprofen and oxycodone hydrochloride. He lived at home with his wife and was a non-drinker and non-smoker. There was no significant family history of malignancy.

On physical examination MA appeared well. His abdomen on inspection had distended superficial abdominal veins and was generally tender to palpation. There was bilateral pitting oedema to the groin extending into the scrotum. He was tender on palpation over his lumbar spine. The remainder of the examination was unremarkable.

He received palliative radiotherapy at 20Gy in 5 fractions for metastatic deposits in the sacrum and lumbar spine and is currently receiving palliative chemotherapy with docetaxel every three weeks. His disease is incurable.



**Figure 3a.** Duplex Doppler ultrasound: longitudinal section of the IVC. This image demonstrates the hypoechoic partially occlusive thrombus within the IVC (red arrow). There is some peripheral flow, most notably anterior to the thrombus (green arrow).



**Figure 3b.** Ultrasound: longitudinal section of a normal IVC. Note the vessel is anechoic with no thrombus present (red arrow) posterior to the left lobe of the liver (green arrow).

## Discussion

IVCT is an under-recognised pathology and may develop from many conditions including malignancy. Whilst other causes exist such as

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trauma and a dysfunctional coagulation system, it should be noted that IVCT is frequently idiopathic. In Nepal it is endemic, with a suspected association with infections. [1]

Thrombotic disease has been reported in all types of malignancy especially with advanced disease. Patients with haematological malignancies such as acute leukaemia, lymphoma and multiple myeloma are also at high risk of thrombotic or haemorrhagic complications. [2] In a study by Ege *et al.*, [3] the majority of thromboses in patients with malignancies were localised to the femoral vein (85.7%) rather than the IVC. Virchow's triad of stasis, hypercoagulability and vessel wall damage underpin the pathophysiology behind IVCT. [4] The combination of thrombosis and malignancy is also known as Trousseau syndrome. Notably Trousseau first appreciated this in 1865. [5]

The most familiar tumour linked with IVCT is renal cell carcinoma where the tumour directly invades the renal vein. Other reported tumours include teratomas and seminomas. Any tumours anatomically related to the IVC may result in direct compression causing stasis or turbulent blood flow, potentially inducing thrombosis. [4]

The pathogenesis of the hypercoagulable state in malignancy involves multiple variables. Intact tumour cells may express pro-coagulant activity that can directly induce thrombin generation; in addition, normal host tissues may express pro-coagulant activity in response to the tumour. [6] Expression of activated MET oncogene causes transformation of somatic cells and tumour growth. This may result in upregulation of PAI-1 and COX-2 resulting in inhibition of fibrinolysis and activation of platelets respectively. This interaction may lead to disseminated intravascular coagulopathy (DIC). [7]

IVCT may be treated medically or surgically and treatment is based on the underlying pathophysiology. Medically, therapy involves anticoagulation and thrombolytic therapy. In this patient, heparin was commenced immediately to decrease the risk of embolism. The merits of thrombolytic therapy must be weighed up against the risks of haemorrhage. Thrombolytic agents include streptokinase, urokinase and tPa. [4]

Used less frequently is surgical caval interruption and thrombectomy. Although caval filters allow central flow there is a risk of thrombus formation at the filter site. Re-thrombosis rates are significant with thrombectomy and the procedure usually does not remove the whole thrombus. Several interventional modalities are available to treat IVCT such as percutaneous balloon angioplasty, wall stents and Z stents. [4]

## Conclusion

This case report outlines one of the rarer complications of malignancy. It is relevant as the prevalence of cancer within society is high.

## Acknowledgements

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## Conflict of interest

None declared.

## Consent Declaration

Informed consent was obtained from the patient for publication of this case report and accompanying images.

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overview.

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