Caval umbrella causing obstructive uropathy

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Summary

A 49-year-old woman had a vena caval filters inserted having suffered multiple pulmonary emboli and a large upper gastrointestinal bleed. She re-presented five years later with loin pain and obstructive uropathy. She was found to have a right pelvi-ureteric obstruction due to inferior vena caval wall perforation from the vena caval filter.

Keywords: obstructive uropathy, vena caval filter

Vena caval filter placement is an accepted form of treatment for the prevention of pulmonary embolism, particularly in patients at risk of recurrent emboli despite anticoagulation or where anti coagulation is contraindicated. However, there are a number of potential complications associated with their use, including recurrent pulmonary embolism, thrombus formation on the filter, filter migration and caval perforation by the legs of the filter. We report a case of pelvi-ureteric obstruction secondary to perforation of the inferior vena cava by a Kimray–Greenfield filter.

Case report

A 49-year-old woman presented in 1988 with fever, sore throat, arthralgia and a rash suggestive of acute vasculitis. This was associated with acute renal failure (serum creatinine 600 µmol/l) and a strongly positive c-type anti-neutrophil cytoplasmic antibody (ANCA) consistent with the diagnosis of ANCA-positive vasculitis. She was treated conventionally with steroids and azathioprine. Unfortunately, her recovery was complicated by recurrent pulmonary emboli with multiple ventilation/perfusion mismatches on imaging. Venography confirmed thromboses in the deep veins of both legs. Simultaneously, she suffered a large upper gastrointestinal bleed secondary to multiple gastric ulcers and therefore, anticoagulation was contraindicated. A Kimray–Greenfield vena caval filter was inserted through the right sapheno-femoral junction to prevent further pulmonary emboli. At operation the long saphenous vein was confirmed to be occluded with thrombus. Her renal function stabilised on immunosuppressive treatment with a serum creatinine of 180 µmol/l on discharge eight weeks later.

She represented five years later with right loin pain. Renal ultrasound and intravenous urography showed a moderate right hydro-

nephrosis secondary to pelvi-ureteric junction obstruction. One leg of the vena caval filter was seen to lie in close proximity to the right pelvi-ureteric junction. Subsequent computed tomography scanning showed that the lower portions of five legs of the filter had perforated the wall of the inferior vena cava (figure) with one leg eroding the anterior margin of the vertebral body of the third lumbar vertebra and a second had embedded in the right psoas muscle. Two legs passed anteriorly into the wall of overlying duodenum. The fifth leg was lying adjacent to the pelvi-ureteric junction of the right kidney. The inferior vena cava was patent.

It was felt that the caval filter should not be removed due to the risk of damage to adjacent organs. At the present time her renal function remains stable (serum creatinine 150 µmol/l) and she is having infrequent right loin pain.

Discussion

Vena caval filters have been available for the prevention of pulmonary embolism since the late 1960s. The stainless steel Kimray–Greenfield filter was one of the first to become widely clinically accepted, although it has subsequently been modified. The number of filters implanted each year continues to rise.¹ However, there is a marked geographical variation in the use of these devices with a 10-fold difference between the numbers used in the US and UK.²

The indications for vena caval filters were well defined; these include patients at risk of pulmonary emboli where anticoagulation is contraindicated, or where pulmonary emboli occurs despite adequate anticoagulation.³ Despite this, the decision to insert a filter may still be difficult as there are a number of significant acute or longer-term risks associated with filter placement. The common routes for insertion

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Complications of vena caval filters
<i>Short term</i> <ul style="list-style-type: none">• complications of venepuncture• filter migration
<i>Longer term</i> <ul style="list-style-type: none">• recurrent pulmonary embolism• thrombus formation on the filter• filter migration up or down the inferior vena cava• caval perforation by the legs of the filter• lower limb oedema

Figure Targetted CT section through lower portion of vena caval filter. Duo = duodenum, IVC = inferior vena cava, AA = abdominal aorta, RK = right kidney, Ps = psoas, L3 = body of third lumbar vertebra.

are via the internal jugular vein or femoral vein and in the majority of cases the filter is placed intra-renally. Fortunately, death directly associated with filter insertion is rare. Longer term risks of filter insertion include recurrent pulmonary emboli, caval thrombosis, filter migration and caval perforation.

The ultimate function of a vena caval filter is to prevent sudden death from massive pulmonary embolism. In two large series successful prevention of pulmonary embolism was reported in 96% of patients who had inferior vena caval filters inserted (150/156 and 451/469 patients, respectively).^{4,5}

Vena caval thrombosis is associated with insertion of inferior vena caval filters in up to 25% of cases.⁴ However this figure may be an overestimate as it is difficult to determine whether the filter itself caused the thrombosis or trapped emboli from the deep veins as intended. It may also simply represent extension of thrombus in a subject already at risk. Fortunately the degree of thrombosis is rarely haemodynamically significant.

Post-mortem migration of the filter up or down the inferior vena cava occurs in up to 50% of cases⁶ but is rarely of clinical significance.^{7,8} A small number of reports have described migration to the renal vein, to the right side of the heart^{10,11} and to the pulmonary artery.¹²

Caval perforation by a leg of a Kimray–Greenfield filter has been estimated to occur in 15–27% of patients after filter insertion^{12–15} with penetration of adjacent structures such as duodenum, psoas muscle, abdominal aorta¹⁶ and vertebral bodies.¹⁷ Reported rates of perforation by the Kimray–Greenfield filter may be inaccurate as CT scanning, the most accurate method of assessing perforation, was not performed in all series. Modifications to the original steel Kimray–Greenfield filter have now been made to reduce the incidence of

perforation and migration. Fortunately inferior vena cava penetration is rarely symptomatic. In this case, five of the six legs of the filter had penetrated the inferior vena cava, encroaching on the lumbar vertebra, the duodenum, the right psoas muscle and the right kidney. Prolonged use of corticosteroids may have contributed to weakening of the wall of the inferior vena cava leading to more extensive penetration.

Long-term evaluation of the risk/benefits of inferior vena caval filters is difficult as the patients in whom they are used are often in poor general health and thrombo-embolism is often one of the terminal events in advanced illness such as carcinoma.¹⁸

For this patient the decision now as to whether to remove the filter is difficult. The illness predisposing to venous thrombo-embolism and peptic ulceration is now quiescent so the continued presence of a filter may be unnecessary. However, the position of the filter is not at present life threatening and removal would be a major surgical procedure. At present her loin pain is intermittent and adequately controlled with simple analgesia. We have elected to treat her conservatively.

Summary points

- vena caval filters are accepted treatment for: current pulmonary embolism despite anticoagulation, pulmonary embolism where anticoagulation is contraindicated
- filters are inserted percutaneously under local anaesthetic
- there are a number of associated complications (see above)
- CT scanning is the most accurate method of assessing perforation of the inferior vena cava by the filter

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Acute pancreatitis due to zinc phosphide ingestion

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Summary

The case of a young woman is described who suffered from acute pancreatitis related to the ingestion of zinc phosphide. This unusual complication was successfully managed with conservative treatment.

Keywords: pancreatitis, zinc phosphide

Zinc phosphide is a commonly used rodenticide in India. When exposed to moisture, or gastric juice hydrochloride, it liberates highly lethal phosphine gas (PH_3), producing various metabolic and non-metabolic toxic effects.^{1–3} Mortality due to PH_3 is very high (37–100%).¹ The clinical and pathological features are shown in box 1. The poisoning may also result in hypoglycaemia,² but not to date, acute pancreatitis. We report a case of acute pancreatitis due to zinc phosphide ingestion that was successfully treated.

Case report

A 21-year-old woman was hospitalised six hours after ingestion of zinc phosphide. On examination, she appeared drowsy and her vital signs were normal. Twelve hours after admission, she was found to be stuporose but irritable, febrile (37.4°C), jaundiced and hypotensive. Signs of metabolic acidosis and peripheral circulatory failure were present. Epigastric tenderness and generalised abdominal distension with absent intestinal peristaltic sounds were noted. Laboratory studies showed haemoglobin 110 g/l, hyperglycaemia (glucose 161 mmol/l), raised blood urea nitrogen 7.1 mmol/l, creatinine 124 $\mu\text{mol/l}$, hyperbilirubinaemia (bilirubin 72 $\mu\text{mol/l}$), raised hepatic enzymes (aspartate aminotransferase 706 U/l, alanine aminotransferase

617 U/l), hyperamylasaemia (2132 U/l), hyperkalaemia (6.2 mmol/l), hypoalbuminaemia (32 g/l), hypocalcaemia (1.9 mmol/l), glycosuria and ketonuria. An abdominal X-ray showed generalised ileus with air-fluid levels. Ultrasound demonstrated an oedematous, enlarged pancreas. She was treated conservatively with intravenous fluids, fasting, nasogastric suction, antibiotics, H_2 -blockers, analgesics, and crystalline insulin injections guided by blood glucose estimations.

Over the next four days, she became normotensive; cyanosis and jaundice disappeared; tenderness and abdominal distension decreased. Blood glucose and potassium were reduced to 60 mmol/l and 40 mmol/l, respectively, glycosuria and ketonuria disappeared but urinary amylase levels rose to 11873 U/l. Repeat ultrasound performed on the seventh hospital day showed marked decrease in pancreatic oedema. After 17 days serum albumin rose to 38 g/l, and calcium to 2.13 mmol/l. Blood urea nitrogen, creatinine, bilirubin, aspartate aminotransferase, and alanine aminotransferase were reduced to 4.26 mmol/l, 88.4 $\mu\text{mol/l}$, 18.1 $\mu\text{mol/l}$, 50 U/l, 50 U/l, respectively and normalised after an additional week with the exception of a slight rise in amylase to 66 U/l, and urinary amylase to 575 U/l.

The patient is well two months after discharge. Ultrasound and computed tomography showed a normal pancreas.

Discussion

PH_3 causes non-competitive inhibition of cytochrome oxidase, and insect catalase, and a change in the dichroic spectrum of haemoglobin, suggesting a valency change in haem accompanied by conformational changes in the prosthetic group.^{1,4} The exact pathogenesis of PH_3 -induced organ toxicity is not well under-

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