Chronic inferior venacava thrombosis in membranous nephropathy

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A 30-year-old gentleman, a known membranous nephropathy patient since 2002, presented a month ago for recurrent bipedal oedema and dilated veins in front and back of abdomen (Figure 1). After contrast prophylaxis, a venogram was done with the intention of intervention in the same sitting if required. His venogram showed non-visualization of the entire infra-hepatic inferior venacava (IVC) that was replaced by collaterals with reformation of the supra-hepatic segment of the IVC (Figure 2). He was treated with placement of an IVC filter and anticoagulation.

Patients with nephrotic syndrome have an increased incidence (10–40%) of arterial and venous thrombosis particularly of the deep veins and renal veins. A variety of haemostatic abnormalities contribute to the hypercoagulable state including loss of antithrombin, hyperaggregability of platelets and clotting factor abnormalities such as elevation of factors V, VII, VIII and fibrinogen [1].

Thrombosis of the adjacent IVC at or near the renal vein origin causes complications in 40–50% of patients with thrombosis of renal veins. Acute thrombosis of IVC should be treated as a medical emergency as pulmonary embolism and deterioration of renal function may follow [2]. Chronic IVC thrombosis is difficult to treat as they have well-established collaterals making intervention difficult.

Conflict of interest statement. None declared.

References


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Fig. 1. Dilated veins in front of chest (white arrows) and abdomen (black arrows).

Fig. 2. Venography right lateral view showing the collaterals (black arrows) in the hepatic segment of IVC with reformation of the supra-hepatic segment (thin black arrow). Note the posterior collaterals extending into the chest (white arrow).