What is the diagnosis?

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CLINICAL PRESENTATION

A 69-year-old hypertensive gentleman (on amlodipine and metoprolol) presented with atrial fibrillation. Amiodarone and low molecular weight heparin (LMWH–enoxaparin 60 mg twice daily) were commenced. On the third day he developed right (Rt) flank pain, shock (50 mmHg systolic), low oxygen saturation (80%), peri-umbilical ecchymotic patches, and palpable Rt retroperitoneal mass. Laboratory investigations revealed low haemoglobin, 8.3 g%; normal platelets, 210,000/mm³; leucocytosis, 31,000/mm³; azotemia, urea 170 mg%, creatinine, 4.3 mg%; and

deranged coagulation, international normalised ratio (INR) 2.7. Computed tomography (CT) of the abdomen was also performed (Figures 1 and 2).

WHAT IS THE DIAGNOSIS?

- Dissecting aneurysm of renal artery.
- Haemorrhagic pancreatitis.
- Spontaneous peri-nephric haematoma.
- Right adrenal haemorrhage.

Figure 1 Non-contrast computed tomography (NCCT) image at the level of kidneys.

Figure 2 Reconstructed contrast-enhanced computed tomography (CECT) image of abdomen. Hounsfield value of the area of pathology is 61 HU.

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Spontaneous perinephric haematoma

INTERPRETATION OF THE IMAGE

Computed tomography reveals right (Rt) perinephric haematoma (H), 61 Hounsfield unit (HU), measuring $13 \times 12 \times 16 \text{ cm}^3$, with supero-medial displacement of Rt kidney (K) (Figure 3).

CLINICAL COURSE

Chronic kidney disease (CKD) grade IV (glomerular filtration rate 28.4 mL) was confirmed using diethylene triamine penta-acetic acid (DTPA) scan. Patient was stabilised on prompt cessation of LMWH/anti-hypertensives and resuscitation using oxygen, intravenous fluids, blood/fresh frozen plasma (FFP) and inotropes (dobutamine and dopamine). Anti-hypertensives were resumed and the patient was followed up by serial imaging using ultrasonography (USG)/magnetic resonance imaging (MRI). Magnetic resonance imaging scan obtained after six months revealed partial resolution of the haematoma (H) measuring $7.4 \times 9 \times 7.2 \text{ cm}^3$ which appeared hyperintense both on T1 and T2 weighted images, surrounded by hypointense thick wall (3.6 mm) and internal septae (Figure 4). Right kidney (K) measured 7 cm with blurred cortico-medullary differentiation. Cysts (C) were seen in both the kidneys.

Haematoma resolved uneventfully over 12 months.

DISCUSSION

Anticoagulants are employed in the management of myriad medical conditions.¹ In anticoagulated patients, appearance of flank pain, shock, and palpable retroperitoneal mass—the triad of Lenk or Wunderlich—is classical of spontaneous retroperitoneal haemorrhage (SRH); in this case it is perinephric.² Peri-umbilical/flank ecchymosis (Cullen's/Grey Turner's sign) appears infrequently.²

Early diagnosis of SRH is rendered difficult by its varied presentations, which depend up on the location of bleed.^{1,2} The presentations could be: Friderichsen–Waterhouse syndrome in adrenal haemorrhage; flank pain, obstructive uropathy/azotaemia in perinephric bleed; and flank/hip/thigh pain and monoparesis in ilio-psoas bleed.² Hypotension, anaemia, and abdominal compartment syndrome may complicate bleed from any of the sites, when massive and unrecognised early.^{1,2}

The severity of bleed is further aggravated by platelet dysfunction either because of uraemia or because of the use of anti-platelet agents, especially when INR reaches the therapeutic range.^{1,3}

Figure 3 Reconstructed contrast-enhanced computed tomography (CECT) image of abdomen showing right perinephric haematoma (H) (61 HU) with supero-medial displacement of right kidney (K).

Figure 4 Magnetic resonance imaging scan of abdomen showing partial resolution of the haematoma (H) surrounded by thick wall. Cortical cysts (C) seen in both the kidneys.

Low molecular weight heparins are rapidly replacing conventional anticoagulants as agents of choice due to the advantages it offers over the later essentially, safety on adoption of fixed dosage schedules without the need for regular monitoring.² However, it is becoming increasingly evident by cases akin to ours, that LMWHs are not as safe in patients with CKD due to reasons more than 1. First, in CKD grade III–V, LMWHs have prolonged half-lives leading to major haemorrhages when fixed dose schedules are employed without appropriate dose reduction.⁴ Second, unlike conventional anticoagulants, effects of which can be readily reversed using protamine/FFP/vitamin K, effects of LMWH do not as readily reverse owing to lack of

specific antagonists.^{2,4} Third, unlike conventional anticoagulants which can be monitored by prothrombin time (PT)/activated partial thromboplastin time (APTT)/INR, monitoring LMWH entails the use of surrogate markers of activity such as anti-Xa activity, which are ambivalent in the prediction of bleeding and are not freely available in laboratories of developing countries.⁴

Routine serial haemoglobin estimation in all anticoagulated patients, in addition to monitoring the coagulation profile, may help in identifying bleeds early.³ Ultrasonography/CT/MRI are the different imaging modalities of choice used in the visualisation and monitoring of SRH.^{1,3}

Intervention, by way of embolisation, percutaneous drainage or convention surgery, is required only in cases of exsanguinating bleed despite apt management, compressive neuropathy, obstructive uropathy, and infected haematoma.¹⁻⁴ Most bleeds are fortunately self-limiting, and are therefore amenable to conservative management. However, despite all, about 20% patients with SRH eventually die either because of the severity of bleed or due to the aggravation of co-morbidities such as coronary artery disease.^{1,2,4}

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