On-line Case Report

Diagnostic dilemma of adrenal enlargement after acute pancreatitis

R MOTALLEBZADEH¹, E GODFREY², NV JAMIESON¹

Departments of ¹Surgery and ²Radiology, Addenbrooke's Hospital, Cambridge, UK

A case is presented illustrating the diagnostic dilemma of adrenal enlargement following acute pancreatitis.

Keywords: Acute pancreatitis – Adrenal enlargement

A 69-year-old man presented to the accident and emergency department having sustained a fall at home, injuring his right leg. Examination revealed a pretibial haematoma and this was evacuated under general anaesthesia. He had an unremarkable past medical history and his only medication was Lansoprazole.

Twenty-four hours later, he complained of generalised abdominal pain and nausea. On examination, he was afebrile and haemodynamically stable, but tenderness and guarding were elicited on palpation of the abdomen. Laboratory investigations revealed a raised serum amylase 1122 U/l (normal range, 25–115 U/l) and increased C-reactive protein 170 mg/l (normal range, 0–6 mg/l). Haemoglobin, renal function, and liver function tests were normal. Ultrasound of the biliary tree was performed; this showed no evidence of gallstones or bile duct dilatation.

Because of the severity of the pancreatitis (Ranson score = 5) coupled with the development of pyrexia, computed tomography (CT) of the abdomen was performed. This revealed an enlarged and oedematous pancreas, consistent with acute pancreatitis (Fig. 1). Surprisingly, there was also enlargement of the right adrenal gland (3.4 × 2.5 cm, H44). The patient subsequently recovered and was discharged 2 weeks later. Follow-up abdominal CT 2

Figure 1 Curved coronal reformatted CT image of the upper abdomen obtained 5 days after admission. The adrenals are moderately enlarged and of similar attenuation to muscle.

months later (Fig. 2) showed a persistently enlarged right adrenal gland (3.8 × 2.1 cm, H38), but also an enlarged left adrenal gland (5 × 3.6 cm, H38). The attenuation and size suggested bilateral adrenal gland haemorrhage. Further imaging 5 months later (Fig. 3) confirmed reduction in the size of both adrenals and resolution of the attenuation, compatible with recent haemorrhage (right – 2.7 × 1.6 cm, H24; left – 3.3 × 2.2 cm, H11). Throughout this time, there

Correspondence to: Reza Motallebzadeh, Department of Surgery, Addenbrooke's Hospital, Box 202, Hills Road, Cambridge CB2 2QQ, UK
E: rmz1001@yahoo.co.uk

Figure 2 Curved coronal reformatted CT image of the upper abdomen obtained 2 months after admission. The adrenals are now grossly enlarged and are low attenuation.

Figure 3 Curved coronal reformatted CT image of the upper abdomen obtained 5 months after admission. The adrenals have reduced in size but remain enlarged. Their attenuation is again similar to that of muscle.

was no evidence to suggest the development of pancreatic necrosis, and a short Synacthen test confirmed normal adrenal function.

Discussion

Adrenal gland involvement is not a well-known complication of pancreatitis. In our case, the initial unilateral enlargement of the adrenal gland was thought to represent an incidentaloma, such as an adenoma. The subsequent imaging showed bilateral enlargement, and then regression in size, favouring evolving and resolving adrenal haemorrhage as the cause.

Adrenal haemorrhage can result from traumatic and non-traumatic causes, such as stress from surgery, Gram-negative septicaemia (Waterhouse–Friderichsen syndrome) or from coagulopathy.¹ Trauma accounts for 80% of cases of adrenal haemorrhage, with the right adrenal gland more commonly involved than the left. An increase in pressure in the inferior vena cava is directly transmitted to the right adrenal gland, as the right suprarenal vein drains into the

vena cava. The left suprarenal vein does not drain directly into the vena cava, possibly explaining the predominance of right-sided traumatic adrenal haemorrhage.

As the head of the pancreas lies antero-inferiorly to the right adrenal gland, and the tail lies anterior to the left adrenal gland, pancreatic inflammation can involve the perirenal spaces. The proteolytic enzyme-rich inflammatory process can penetrate Gerota's fascia and, therefore, come into contact with the adrenal glands themselves.² The precise mechanism by which the pancreatic exudate stimulates adrenal haemorrhage is not known. In fact, the mechanism may not be due to direct involvement. The adrenal gland has a complex arterial supply network, in comparison to its limited venous drainage which is critically dependent on a single vein. This pattern of blood supply has been described as a 'vascular dam'. In stressful situations (e.g. pancreatitis), adrenocorticotrophic hormone secretion increases, which stimulates adrenal arterial blood flow that may overwhelm the limited venous drainage capacity of the organ, leading to infarction and haemorrhage.³

Acute adrenal insufficiency can occur in association with massive bilateral adrenal haemorrhage, and maybe fatal if unrecognised and untreated. Bilateral haemorrhage occurs in 10% of all cases, but adrenal insufficiency secondary to bilateral haemorrhage is relatively unusual.¹ More than 90% of the adrenal cortex must be destroyed before there is evidence of adrenal insufficiency.⁴ In contrast, unilateral adrenal haemorrhage is not usually associated with acute adrenal insufficiency. Overall, adrenal haemorrhage is associated with a 15% mortality rate, which varies according to the severity of the causative factor underlying adrenal haemorrhage.³

Clinical findings in patients with adrenal haemorrhage are non-specific, and depend on extent of haemorrhage, whether it is bilateral or unilateral, and the underlying cause. Fever, nausea, tachycardia, hypotension, and abdominal pain can occur; coma can ensue in severe cases.⁴ However, most patients with adrenal haemorrhage do not have clinical signs of adrenal insufficiency. The diagnosis is often suggested at imaging performed for another reason.

The most reliable method of identifying adrenal haemorrhage is with CT. The adrenals appear as hyper-dense round/oval structures. The attenuation value depends on the age of the lesion. In the acute setting the adrenal gland has high attenuation (50–90H). As the haemorrhage gets absorbed over time, there is decrease in attenuation to near that of water. Associated findings include peri-adrenal fat stranding. Over the longer term, the adrenals show progressive atrophy and, in some cases, calcification.

Magnetic resonance imaging can age the haemorrhage into acute (24–72 h after onset), subacute (3–7 days after

onset), and chronic (> 7 weeks) depending upon the signal intensity yielded by T1/T2-weighted sequences.¹

Ultrasound is the preferred imaging investigation in neonates because of the small patient size, the relatively large size of the adrenals, and the absence of ionising radiation. In early adrenal haemorrhage, the adrenal glands appear as a solid, hyperechoic mass. As the haemorrhage resolves, the glands reduce in size and are hypo-echoic as the haematomas become cystic.¹

Adrenal haemorrhage is usually managed conservatively, with follow-up imaging to determine the size and progression of the haematoma, and to exclude an underlying lesion. In cases complicated with adrenal insufficiency, treatment with hydrocortisone is required to prevent an Addisonian crisis. In the rare instance of adrenal haemorrhage where intervention is necessary, endovascular therapies such as superselective trans-

catheter embolisation with a variety of agents has been used to treat adrenal haemorrhage.⁵ Surgery is not needed for non-traumatic adrenal haemorrhage, except in patients with primary adrenal tumours or if there is extensive retroperitoneal haemorrhage.

References

1. Kawashima A, Sandler CM, Ernst RD, Takahashi N, Roubidoux MA, Goldman SM *et al.* Imaging of nontraumatic hemorrhage of the adrenal gland. *Radiographics* 1999; **19**: 949–63.
2. Farman J, Morehouse H, Amis Jr ES, Newhouse JH. CT of pancreatitis with renal and juxtarenal manifestations. *Clin Imaging* 1997; **21**: 183–8.
3. Vella A, Nippoldt TB, Morris 3rd JC. Adrenal hemorrhage: a 25-year experience at the Mayo Clinic. *Mayo Clin Proc* 2001; **76**: 161–8.
4. Chin R. Adrenal crisis. *Crit Care Clin* 1991; **7**: 23–42.
5. Igwilo OC, Sulkowski RJ, Shah MR, Messink WF, Kinnas NC. Embolization of traumatic adrenal hemorrhage. *J Trauma* 1999; **47**: 1153–5.