Adrenal insufficiency masquerading as an acute abdomen

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CASE REPORT

A 29-year-old woman was admitted under the care of the general surgeons with a 3-week history of increasing colicky epigastric and retrosternal pain with severe nausea, profuse bile-stained vomiting, constipation and weight loss of 1.5 stone. In contrast, for the preceding 6 months she had been suffering from weight gain, 'indigestion', malaise, lethargy and syncopal attacks. There was no previous history of note and she had had two uneventful deliveries 3 and 5 years previously.

On examination, she looked unwell, was severely dehydrated and apyrexial. Her skin was pale with a yellow tinge. Mucous membranes were pink and sclera were not pigmented. Her pulse was 92 beats per minute and her blood pressure was 93/51 mmHg. Abdominal examination revealed epigastric tenderness with guarding, but no rebound tenderness. There was no abdominal distension or succussion splash. She had marked abdominal striae which were pigmented.

Laboratory results were as follows: haemoglobin 15.9 g/dl (normal range (NR) 11.0–16.5 g/dl), white cell count 10.8x10^9/litre (NR 3.5–11.0x10^9/litre), neutrophils 5.7x10^9/litre (NR 1.5–7.5x10^9/litre), haematocrit 0.478 (NR 0.36–0.45), sodium 133 mmol/litre (NR 135–146 mmol/litre), potassium 4.1 mmol/litre (NR 3.6–5 mmol/litre), urea 5.9 mmol/litre (NR 2.6–6 mmol/litre), creatinine 153 mmol/litre (NR 62–124 mmol/litre), amylase 70 U/litre (NR <300 U/litre), aspartate transaminase 35 IU/litre (NR 0–40 IU/litre), alkaline phosphatase 43 IU/litre (NR 40–105 IU/litre), bilirubin 21 mmol/litre (NR 0–17mmol/litre), calcium 2.53 mmol/litre (NR 2.12–2.62mmol/litre), albumin 41g/litre (NR 32–45g/litre), urine dipstick negative for blood and protein, and pregnancy test negative. Abdominal and chest X-rays were unremarkable. An ultrasound scan of the biliary tract revealed a solitary gall stone with a normal calibre common bile duct.

Surgical differential diagnoses included biliary colic or peptic ulceration but because of the low sodium in the presence of clinical and biochemical/haematological dehydration a diagnosis of adrenocortical failure was considered. A random cortisol was performed at presentation with a value of 40 nmol/litre. A short synacthen test revealed a basal cortisol of 41 nmol/litre and virtually no response to adrenocorticotrophic hormone (ACTH) with cortisol values of 43 nmol/litre at 30 minutes and 51nmol/litre at 60 minutes confirming adrenocortical failure. A simultaneous serum ACTH was grossly elevated at 834 ng/litre (NR 0–50 ng/litre), showing this to be primary adrenocortical failure and thus Addison's disease. Subsequent investigations showed her to be hypothyroid with a thyroid-stimulating hormone level of 74 mU/litre (NR 0.35–5.5 mU/litre) and free thyroxine (T4) of 5 pmol/litre (NR 10–22 pmol/litre) and to have circulating antithyroid microsomal and adrenal antibodies.

INTRODUCTION

This case report highlights an emergency presentation of endocrine catastrophes to the general surgeon. It also demonstrates the importance of identifying the cause of hyponatraemia and full interpretation of biochemical and haematological data.

DISCUSSION

Many conditions can present to the general surgeon which are primarily medical in origin. The consequences of overlooking a diagnosis of adrenal insufficiency has disastrous if not fatal consequences and surgeons should be wary of this uncommon condition. It has been calculated that Addison's disease is present in up to 1 in 1000 surgical admissions (known or undiagnosed) (Wheeler, 1994). Addison's original description associated 'slight pain or uneasiness from time to time referred to the region of the stomach and there is actual vomiting' with profound weight loss, lethargy and skin pigmentation (Addison, 1855).

Abdominal pain as the primary complaint in hypoadrenalism occurs in approximately 10% of cases, although gastrointestinal symptoms are common (Turnipseed et al, 1976). Severe abdominal pain and tenderness, even mimicking peritonitis, occurs in about 7% and heralds the onset of acute adrenal insufficiency (Tobin et al, 1989).

Although in this case the sodium levels were just below the reference range, it was felt that a low level of sodium in the presence of dehydration was significant. Causes of hyponatraemia in this setting of a decreased extracellular volume include gut losses of salt and water such as vomiting and diarrhoea and renal losses from tubulointerstitial disease, excessive use of diuretics and adrenocortical failure.

Once the diagnosis of Addison's disease is suspected, and if the patient is unwell, it is essential that treatment is started as soon as pretreatment blood samples, including cortisol, have been taken. A random cortisol in an ill patient of less than

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200 nmol/litre confirms adrenocortical failure. Emergency treatment consists of 0.9% saline and intravenous hydrocortisone 100 mg every 6 hours. Long-term treatment involves replacement doses of hydrocortisone and fludrocortisone.

Primary adrenocortical failure is usually caused by autoimmune adrenal destruction, which accounts for up to 90% of cases. Women are affected two to three times more commonly. As with this case patients may have other autoimmune endocrine deficiencies, such as autoimmune thyroid disease (Schmidt, 1926), type I diabetes mellitus and premature gonadal failure. Other causes include infections such as tuberculosis and tumours (most notably metastatic carcinoma of the breast) (Burke, 1992).

CONCLUSION

Hypoadrenalism is an uncommon condition but it commonly presents with gastrointestinal symptoms and occasionally as an intra-abdominal catastrophe. A low level of sodium in the presence of a decreased extracellular volume has a narrow differential diagnosis. Failure to diagnose or treat Addison's disease may be fatal. **HM**

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Tobin MV, Aldridge SA, Morris AI, Belchetz PE, Gilmore IT (1989) Gastrointestinal manifestations of Addison's disease. *Am J Gastroenterol* **84**: 1302–5

Turnipseed WD, Madura JA, Luther K (1976) The acute abdomen in undiagnosed Addison's disease. *Wisconsin Med J* **75:** S104–106

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IN THE PUBLIC'S VIEW...

Quashing the MMR hype: can we overcome the media's influence?

Mid-January was a bad time in the media. Many birds came home to roost. These weren't the type of bird the phrase is meant to apply to; they were birds that should have been shot long ago, birds at which this column has fired both barrels a number of times.

The latest figures on the falling uptake of the measles, mumps and rubella (MMR) vaccine were accompanied by dire warnings of likely epidemics of measles, but the media couldn't resist interviewing some parents convinced their children had been damaged by the vaccine. It will not do for the media to point to their reasoned editorials and say they are doing their best.

It is human nature that parents take more notice of one mother's tearful condemnation than of public health doctors, or of the reported Finnish study of nearly two million children that turned up no excess cases of autism or bowel disease. 'My baby developed autism after his inoculation' is, to far too many, an undeniable truth. A worrying aspect of the story was the large number of GPs, and even larger number of nurses, who believe that MMR is harmful.

The same 'it's obvious' logic sprang up on the front pages in the worry about depleted uranium ammunition. In a replay of 'Gulf war syndrome', ill British servicemen said they'd been ill ever since they came back from the Balkans, and reporters rushed around Kosovo finding grieving relatives convinced their loved ones had died because of radioactive contamination.

Uranium is known to cause lung and bone cancer, and it damages the kidneys. It is unlikely to cause excess brain cancer, breast cancer or leukaemia. The newspapers reported the story ('She was a fit and healthy 25-year-old until she visited the shell crater') as if every cancer death since the Balkan conflict was obviously and incontrovertibly caused by uranium.

Uranium-238 is mildly radioactive. Cancer is a common cause of death. From what I've read it is beyond reason that the reported ills are caused by U^{238}. If there really is an excess of diseases known to be caused by radioactivity then the conclusion should be that someone is telling porkies about what types of ammunition were used, but the media are too concerned with the obvious, even if it's wrong.

Ignoring recent medical evidence and opinion (Inskip et al, 2001; Rothman, 2000), the *Guardian* (13 January) preferred that well known dispenser of truth, the US legal system, and asked the question, 'Are mobile phones as dangerous as smoking?' A case is about to start in California in which the usual experts (all paid up and guns smoking) will line up to tell half-truths through their teeth. The latest scare is that mobile phones cause eye tumours. I've not seen details of this yet but I predict that it will all come to nothing, and that there is more danger from poking your eye out with the aerial. **HM**

Inskip PD, Tarone RE, Hatch EE et al (2001) Cellular-telephone use and brain tumors. *N Engl J Med* **344:** 79–86

Rothman KJ (2000) Epidemiological evidence on health risks of cellular telephones. *Lancet* **356:** 1837–40

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