

cause inhibition of ADH secretion and maintenance therapy with hydrocortisone perpetuates this effect. Adequate treatment directed toward the underlying cause of SIADH is the most appropriate, but strict limitation of fluid intake will correct all the physiological disturbances despite persistence of the source of excessive antidiuretic activity.<sup>2</sup>

We suggest that hypopituitarism should be considered as a possible cause of unexplained hyponatraemia<sup>1</sup> regardless of its response to corticosteroid therapy.

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#### References

- Oelkers, W. Hyponatremia and inappropriate secretion of vasopressin (antidiuretic hormone) in patients with hypopituitarism. *N Engl J Med* 1989, **321**: 492–496.
- Kleeman, C.R. & Berl, T. The neurohypophyseal hormones: vasopressin. In: De Groot, L.J. *et al.* (eds) *Endocrinology*. Grune and Stratton, New York, 1979, pp. 253–270.
- Ahmed, A.B., George, B.C., González-Auvert, C. & Dingman, J.F. Increased plasma arginine vasopressin in clinical adrenocortical insufficiency and its inhibition by glucocorticoids. *J Clin Invest* 1967, **46**: 111–123.
- Axelrod, L. Glucocorticoid therapy. *Medicine* 1976, **55**: 39–48.

### Phaeochromocytoma presenting with acute intestinal ischaemia and shock

Sir,

We have read with interest the case report of Carr *et al.* concerning small intestinal ischaemia occurring as a result of phaeochromocytoma.<sup>1</sup> We have recently had a similar experience with a patient who had a phaeochromocytoma in whom the presenting diagnosis was large bowel ischaemia with circulatory failure.

A 60 year old woman presented with a 4-hour history of colicky right hypochondrial pain and vomiting. She had noticed palpitations earlier that day; this symptom and mild hypertension had required  $\beta$ -blocker therapy in the past. Her only other medication was glibenclamide for the treatment of diabetes mellitus diagnosed 2 years previously.

On examination she was afebrile, oliguric, the systolic blood pressure was 80 mmHg and there was intense peripheral vasoconstriction. She had Kussmaul respiration and was tender in the right subcostal region; bowel sounds were sparse. Arterial blood gases showed metabolic acidosis. Abdominal X-ray films were normal. Bowel ischaemia was suspected and, whilst awaiting surgery, mesenteric angiography was performed. This showed moderate aortic atheroma but patency of the coeliac and both mesenteric axes.

At laparotomy after intravenous fluids the ascending colon was found to be ischaemic and this was treated by right hemicolectomy with terminal ileostomy and mucus fistula formation. Anaesthesia was complicated by hypertensive surges but the implication of this was not appreciated initially. Pathological examination of the surgical specimen confirmed ischaemic necrosis but there was no evidence of large artery occlusion although small fibrin

thrombi were seen in superficial sub-mucosal vessels. Post-operatively the patient required arterio-venous haemofiltration and ventilation on the ITU. Management was complicated by marked lability of blood pressure and the diagnosis of phaeochromocytoma was confirmed by assay of plasma catecholamines. Pulmonary gas exchange continued to deteriorate and the patient died 2 days later. At post-mortem a 10 cm diameter right adrenal phaeochromocytoma was identified which weighed 225 g.

Our patient presented with an acute abdomen and shock, and intestinal ischaemia was diagnosed at laparotomy. We suspect that the intestinal pathology resulted from catecholamine-induced vascular spasm. Indeed, severe vasoconstriction which has been found to be responsible for pulseless limbs and peripheral gangrene as well as permanent neurological deficits such as cortical blindness, is a well-recognized accompaniment of phaeochromocytoma.<sup>2</sup> Such intense vasoconstriction may have contributed to the acute renal failure that developed in this case, although other factors such as sepsis and shock were possibly more important. An alternative explanation of the intestinal ischaemia is a 'watershed-type' infarction as a consequence of hypotension (perhaps induced by tumour necrosis) in a patient with pre-existent atheromatous disease. Infarction of a phaeochromocytoma is well-recognized<sup>3</sup> and its presentation of shock and acute abdominal pain has resulted in several unexpected fatalities during the anaesthesia and surgery of an 'acute abdomen'.<sup>4</sup>

Although we do not feel that the outcome would have been altered in this case we wish to re-emphasize the need to consider a diagnosis of phaeochromocytoma in patients with acute abdominal signs whose presentation is atypical, and we would certainly support the view of other authors<sup>1,5</sup> that gastrointestinal symptoms occurring in association with phaeochromocytoma should indicate the necessity for urgent tumour removal, as gut ischaemia may be imminent.

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#### References

- Carr, N.D., Hulme, A., Sheron, N., Lees, W.R. & Russell, R.C.G. Intestinal ischaemia associated with phaeochromocytoma. *Postgrad Med J* 1989, **65**: 594–596.
- Radtke, W.E., Kazmier, F.J., Rutherford, B.D. & Sheps, S.G. Cardiovascular complications of phaeochromocytoma crisis. *Am J Cardiol* 1975, **35**: 701–705.
- Bergland, B.E. Phaeochromocytoma presenting as shock. *Am J Emerg Med* 1989, **7**: 44–48.
- Ledingham, J.G.G. Secondary hypertension-Phaeochromocytoma. In: *Oxford Textbook of Medicine*, 2nd Ed., vol. 2. 13, 1988, pp. 392–396.
- Fee, H.J., Fonkalsrud, E.W., Ament, M.E. & Bergstein, J. Enterocolitis with peritonitis in a child with phaeochromocytoma. *Ann Surg* 1977, **185**: 448–450.