

Letters to the Editors

Secondary hypoadrenalism with hypercalcaemia

Sirs, We read with interest the case report of secondary hypoadrenalism presenting with moderate hypercalcaemia (Vasikaran *et al.*, 1994) in which the authors speculate that hypercalcaemia may be exacerbated by hyperthyroidism in the presence of glucocorticoid deficiency. We believe that hypercalcaemia, albeit less severe, can occur in secondary hypoadrenalism without overt hyperthyroidism, as apparently in that case report (Vasikaran *et al.*, 1994). We have recently presented a series of five women whom we believe to have lymphocytic hypophysitis presenting either in the last trimester of pregnancy or early puerperium (Patel *et al.*, 1994). All had secondary adrenal insufficiency at presentation, and 4/5 had secondary hypothyroidism with normocalcaemia. The fifth patient, a 41-year-old woman, presented 2 months after her sixth uncomplicated pregnancy and delivery. Since parturition she had felt generally unwell with lethargy, fainting episodes (BP 90/60, 80/50 lying), aching joints, muscle pains, proximal myopathy and limb girdle muscle wasting; nausea, vomiting, anorexia; and weight loss of 10 kg. She was able to lactate but suffered from recurrent breast abscesses requiring surgical drainage. Short Synacthen test (250 mg i.m.) basal cortisol <50 nmol/l; 60 min, 75 nmol/l. The patient had mild and persistent elevation of serum calcium (see Table 1) from the time of presentation for which she was investigated. PTH was undetectable at <1.5 pmol/l (normal range 2–5). Her free thyroid hormone levels were normal, although her TSH was suppressed.

Hydrocortisone replacement resulted in prompt normalization of serum calcium despite increasing biochemical hyperthyroidism, which by 6 months post partum was clinically obvious and required treatment (see Table 1). This woman too would seem to have had both postpartum hypophysitis and thyroiditis and mild hypercalcaemia, despite initially normal free thyroid hormone levels. It could be argued that the suppressed TSH indicated tissue hyperthyroidism and that this, combined with glucocorticoid deficiency, was the cause of the patient's mild hypercalcaemia, by the mechanisms discussed by Vasikaran *et al.* However, the serial calcium measurements show that glucocorticoids alone normalized the serum calcium, despite increasing biochemical hyperthyroidism. Whilst accepting that hyperthyroidism combined with glucocorticoid deficiency may result in exacerbation of hypercalcaemia of adrenal failure, it is possible that, just as in primary adrenal insufficiency, mild hypercalcaemia

can occur in secondary adrenal insufficiency without hyperthyroidism.

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References

- Patel, M.C., Guneratne, N.D., West, T.E.T., Drury, P. & Clayton, R.N. (1994) Maternal malaise: consider lymphocytic hypophysitis. *Journal of Endocrinology*, **140** (Suppl.), 155.
Vasikaran, S.D., Tallis, G.A. & Braund, W.J. (1994) Secondary hypoadrenalism presenting with hypercalcaemia. *Clinical Endocrinology*, **41**, 261–264.

Sirs, Patel and Clayton have presented a series of five patients with lymphocytic hypophysitis all of whom had secondary hypoadrenalism. Four of these five patients also had secondary hypothyroidism and were normocalcaemic whereas the only patient who had sufficient (or arguably marginally elevated) thyroid hormone activity was hypercalcaemic. This is further evidence for the hypothesis that thyroid hormone action is important in the aetiology of hypercalcaemia of hypoadrenalism.

Our contention is that hypercalcaemia is not usually seen in secondary hypoadrenalism because the latter is usually accompanied by overt or subclinical secondary hypothyroidism. All the cases of secondary hypoadrenalism consequent on lymphocytic hypophysitis which presented with hypercalcaemia presumably had thyroiditis, and therefore autonomous thyroid hormone secretion. Irrespective of whether the hypoadrenalism is primary or secondary, the important factor in the aetiology of hypercalcaemia is the presence of sufficient thyroid hormone action.

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Sirs, Vasikaran *et al.* (1994) outline how the hypercalcaemia which may develop in adrenocortical deficiency is largely the result of altered renal handling of calcium, and of unopposed osteocytic osteolysis. We have previously described how these processes may combine to mask calcium deficiency in a

