Letters to the Editors

Lymphocytic hypophysitis

Jenkins et al. (1995) have suggested that the entire removal of the pituitary in cases of lymphocytic hypophysitis is warranted both for accurate diagnosis and for definitive treatment. Based on our experience (Beressi et al., 1994) and data in the literature, we disagree with this conclusion. Some patients with lymphocytic hypophysitis can be managed medically. The diagnosis can be suggested by the pituitary appearances on MRI, or confirmed by pituitary biopsy in a suggestive clinical situation (e.g. woman, post-partum period, pituitary enlargement). The MRI characteristics of lymphocytic hypophysitis are a large pituitary mass, with a symmetrical extension (triangular shape) toward the chiasm. Gadolinium infusion shows a homogeneous enhancement with visualization of a thickened diaphragma sellae. This has been reported in many cases (reviewed in Modigliani et al., 1993; Jenkins et al., 1995; Powrie et al., 1995). This appearance in association with the clinical context was sufficient in our case to warrant steroid treatment. This treatment successfully reduced pituitary volume (two-thirds decrease) and clinical symptoms (disappearance of headaches and normalization of menstrual cycle) within 3 months. A relapse after steroid withdrawal prompted us to confirm our suspected diagnosis by pituitary biopsy. From this case report, we recommend a trial of steroids (1 mg prednisolone/kg/day) in patients without visual complications or suspected pituitary infection. Furthermore, the natural history of pituitary hypophysitis seems to be pituitary atrophy with an empty sella. Simple observation could be an alternative approach. These procedures avoid surgery with concomitant risk of panhypopituitarism.

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References


Sir, I reply to the letter by Cohen et al. suggesting that some patients with lymphocytic hypophysitis may be managed by steroid therapy. This is a course of action with which we do not agree, as our series of cases illustrates. The radiological appearances of lymphocytic hypophysitis are not diagnostic but usually show a pituitary mass with variable degree of superellar extension. In the case noted by the authors, a trial of steroids was given with decrease in size of the pituitary but which relapsed after discontinuing the steroids, necessitating a pituitary biopsy. In two of our cases, a biopsy may not have been sufficient and might have resulted in a wrong diagnosis. In one case lymphocytic hypophysitis coincided with a normal anterior pituitary gland and in the other case there were areas of lymphocytic hypophysitis within a somatotroph adenoma. These indicate that the complete removal of a pituitary mass is needed both for accurate diagnosis and treatment. Furthermore, although surgery may result in hypopituitarism, there is considerable morbidity associated with maintaining patients on several months of glucocorticoid therapy in the dose recommended by the authors (1 mg prednisolone/kg/day).

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Hypercalcaemia and secondary hypoadrenalinism due to Sheehan's syndrome

Sir, Hypercalcaemia is a well known complication of Addison’s disease. Vaskiakian et al. (1994) have reported a young woman with selective corticotroph failure causing secondary adrenal insufficiency who developed hypercalcaemia. The patient had also transient hyperthyroidism due to thyroiditis. According to the authors, it was unlikely that the hypercalcaemia was secondary to the thyrotoxicosis since the hyperthyroidism was both mild and transient. Two other patients with lymphocytic hypophysitis who had