Lymphocytic Hypophysitis - A Report of a Case

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Key words: Lymphocytic hypophysitis, Sellar mass, Autoimmune basis.

Lymphocytic hypophysitis is a rare but well recognised clinicopathologic entity. The first case of lymphocytic adenohypophysitis was described in 1962 by Goudie and Pinkerton and since then 31 more cases have been described in the literature. Considering its rarity as well as unusual clinical presentation in a menopausal patient, we report here a case of lymphocytic hypophysitis.

CASE REPORT

A 46-year-old multiparous menopausal woman was admitted with history of continuous moderate to severe headache for the past two years as well as gradual deterioration of vision in both eyes for 2 months. There were no other symptoms suggestive of an endocrine dysfunction. On examination, the visual acuity was 6/12 in both eyes. Apart from bitemporal hemianopia, there were neither any focal neurological deficits nor any cranial nerve paresis. Routine haematological investigations revealed elevated erythrocytes sedimentation rate of 80 mm. A computed tomographic (CT) scan showed a well-circumscribed sellar mass. Endocrinological evaluations were normal. A diagnosis of endocrinologically inactive pituitary adenoma was made. By a transphenoidal route the sellar mass was removed piecemeal and the material submitted for routine histopathological examination. Sections showed extensive infiltration by chronic inflammatory cells predominantly composed of lymphocytes which were seen in and around atrophic cells in the adenohypophysis (Fig.1). A small component of plasma-cell population was also present in the sections. Caseation, multinucleated giant cells, epithelioid cells or histiocytes were not seen in the sections. Stains for bacteria, fungi and acid-fast bacilli were negative. In the sections there was no evidence of any neoplastic pituitary lesion and a secondary lymphocyte reaction was excluded. The above histopathological features were interpreted as "lymphocytic hypophysitis". Based on this histopathological diagnosis, the patient was further investigated for an autoimmune aetiology. Rheumatoid and antinuclear factors were negative. Antibodies to smooth muscle, thyroid microsomal, and thyroglobulin fraction and parietal-cells were negative. Indirect immunofluorescence on cryostat sections for antipituitary antibodies was also negative. The patient was treated with a course of corticosteroids. She was not given any radiotherapy. A CT scan done a year later did not show any increase in the size of the intrasellar SOL. Her vision and neurological status showed considerable improvement.
The histopathological features of lymphocytic adenohypophysitis are usually characteristic. However this entity needs to be distinguished from a granulomatous hypophysitis. Multi-nucleated giant cells and epithelioid macrophages are not seen in patients with lymphocytic hypophysitis. Secondly lymphocytic hypophysitis should be distinguished histopathologically from a healed case of post partum pituitary necrosis (Sheehan’s syndrome). In the latter, the lesion is characterised by extensive fibrosis with minimal lymphocytic reaction.

There are neither any specific clinical nor any laboratory investigations by which a diagnosis of lymphocytic hypophysitis can be precisely made. A possibility of this entity should be considered in a female patient with a sellar mass during post partum period or in menopausal age group. This diagnosis of the disease can only be established by histopathological examination of the tissue. A transphenoidal stereotactic biopsy is recommended for establishing the diagnosis in all clinically suspected cases.

REFERENCES
