Dear Editor,

Castleman’s disease is an uncommon lymphoproliferative disorder, usually accompanied with other autoimmune diseases. Autoimmune hypophysitis (AH) is a rare disease of the pituitary gland that presents as a sellar mass lesion and/or hypopituitarism, which is speculated to have an autoimmune basis. We reported the first case of Castleman’s disease accompanied with LH. This is a 34-year-old woman, who suffered from headache, diuresis, and irregular menses for 2 years since 2005. Laboratory examination showed hyperprolactinemia, anemia, and diabetes. Computed tomography (CT) scan of the lung revealed a lymph node enlargement in the mediastinum and multiple nodes in the lung. CT scan of the abdominal cavity showed a neoplasm located in the posterior abdominal wall. Cerebral magnetic resonance imaging (MRI) revealed intrasellar lesions (Fig. 1a, b).

The patient underwent a transnasal surgery and another surgery of the posterior abdominal wall neoplasm. The postoperative course was uneventful with partial recovery treated with desmopressin acetate tablets.

The neoplasm of the posterior abdominal wall was characterized by typical histological presentations of Castleman’s disease: large follicles with intervening sheets of plasma cells. As is well known, Castleman’s disease is classified into two histopathological types: the hyaline vascular type and the plasma cell type [1]. The plasma cell type of Castleman’s disease is less common and clinically more aggressive [2], which can have systemic manifestations including fever, excessive sweating, fatigue, arthralgia, anemia, loss of weight, and bone marrow plasmacytosis.

Histological examination of the intrasellar mass showed the defining pathological feature of AH: the infiltration of the pituitary gland mainly with plasma cells, which diffused throughout the gland, distorting the normal architecture. On MRI of AH, the symmetry of pituitary enhancement, the lack of erosive changes of the sellar floor, the homogeneity of the pituitary mass, and its intense enhancement after gadolinium can be diagnostic in the proper clinical context, which could be seen in this patient’s MRI images [1]. The clinical presentation of AH comprises four categories of symptoms: sellar compression, hypopituitarism, diabetes insipidus, and hyperprolactinemia [1].

As to this patient, based on previous histological diagnosis of Castleman’s disease, considering the clinical history, imaging features characteristic of AH, endocrine findings and histological findings of intrasellar mass, we made the final diagnosis as Castleman’s disease with AH.

Patients with Castleman’s disease could be treated with corticosteroid and antineoplastic chemotherapy [1, 3], but all these treatments have only demonstrated a limited effect. Appropriate management remains controversial for AH. Corticosteroid therapy has been advocated as a means of attenuating inflammation, but such therapy does not seem justified for most patients. Partial resection of the mass for accurate diagnosis and to decompress the surrounding structures is an optional surgical strategy [1].

We suggest that AH should be considered in the differential diagnosis of any pituitary mass, especially if presenting with Castleman’s disease. In the absence of surgical emergency, medical management combined with sequential MRI is preferable.
Fig. 1 MRI revealed intrasellar lesions, which were isointense on T1-weighted sequences, with intense and homogeneous enhancement after contrast administration (a, b).

References