Hypophysitis is a chronic inflammation of the pituitary gland of complex and still incompletely defined pathogenesis [1]. It belongs to the group of non hormone-secreting sellar masses, sharing with the other diseases in this group similar clinical presentation and radiographic appearance. These similarities make it often difficult to establish a diagnosis of certainty before pituitary surgery and pathological examination of the resected pituitary tissue [2] (pathologically-proven forms). Nevertheless, more and more cases are diagnosed nowadays solely on clinical and imaging grounds (clinically-suspected forms). Hypophysitis can present with sellar compression, hypopituitarism, diabetes insipidus, and/or hyperprolactinemia [1,3], signs and symptoms that are, however, common to those seen with other non-secreting sellar masses. The clinical course of hypophysitis is unpredictable: it resolves spontaneously in a minority of patients, is cured by transient glucocorticoid treatment in another minority, or requires long-term hormone replacement in the majority of patients because of the eventual destruction of the pituitary endocrine cells [1]. In some cases, death has been reported, the most recent one in 2009 [4].

Hypophysitis has been classified in a number of ways, none of them particularly useful to the clinician or the researcher. Classifications are based on the location of pituitary involvement (adenohypophysitis, infundibulo-neurohypophysitis, or panhypophysitis), the histopathological appearance (lymphocytic, granulomatous, xanthomatous, plasma cell rich, and necrotizing), or the cause (primary hypophysitis to indicate the forms of unknown etiology, and secondary hypophysitis to indicate those where a casual connection can be made). In recent years, the observation that hypophysitis can appear as a consequence of immune-modulatory therapies like CTLA-4 blockade has increased the awareness about this disease in the medical community.

This presentation will focus on two main aspects of hypophysitis: the identification of pituitary autoantigens and the association between hypophysitis and CTLA-4 blockade.

Disclosure of interest

The author declare that he has no conflicts of interest concerning this article.

References