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ENDOCRINE IMAGING

Ectopic posterior pituitary causing hyperprolactinemia

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Hyperprolactinemia may recognize different causes. Many physiological conditions (pregnancy, breast-feeding, stress, exercise, sleep) can cause hyperprolactinemia as medications that interfere with the hypothalamic-pituitary dopaminergic pathways (e.g., antipsychotics or prokinetics). A number of pathological conditions (e.g., hypothyroidism or renal failure) can also raise prolactin levels. Increased autonomous prolactin secretion occurs from lactotroph adenomas, which account for ~40 % of all pituitary tumors. Because prolactin secretion is tonically inhibited by hypothalamic dopamine, disruption or compression of the pituitary stalk by a pituitary tumor, or other parasellar mass will lead to hyperprolactinemia [1–3].

Monomeric prolactin is the most common and biologically active form of circulating prolactin in healthy individuals and most patients with true hyperprolactinemia, but forms with higher molecular mass are also present, such as big prolactin and big–big prolactin or macroprolactin. Molecular aggregates as macroprolactin are regarded to have low biologic activity, so that macroprolactinemia should be suspected when typical symptoms of hyperprolactinemia are absent.

We report the case of a 43-year-old woman with history of oligomenorrhea and mild hyperprolactinemia since her puberty. In 2000, a pituitary MRI showed a 3 mm T1-hyperintense lesion adherent to the pituitary stalk. PRL was raised at 48 ng/ml (n.v. 5–25). Treatment with cabergoline was started (0.5 mg weekly) with normalization of prolactin levels and oligomenorrhea resolution. Periodic MRI evaluations documented the stability of the lesion. In February 2011, cabergoline was discontinued on the advice of another endocrinologist.

In February 2012, she came to our attention due to relapse of her symptoms. Blood tests, performed in our lab, showed hyperprolactinemia (56.3 ng/ml, n.v. 3.5–26.5) in the absence of biochemical and clinical signs of anterior hypopituitarism. A new MRI documented a normal anterior pituitary gland with normal enhancement and confirmed the presence of a 3-mm nodular lesion hyperintense in T1 and hypointense in T2 localized at the medium portion of the pituitary stalk (Fig. 1a, b), compatible with ectopic posterior pituitary (EPP). The hypothesis of an ectopic pituitary adenoma was ruled out because of the absence of the typical radiological findings of microadenomas and considering the stability of the lesion during a 12-year-follow-up despite the previous medical treatment. EPP was considered as the origin of hyperprolactinemia as other causes were excluded, including the presence of macroprolactin and hypothyroidism. Treatment with cabergoline was reintroduced with regularization of menses.

EPP is a rare morphological alteration of the hypothalamic-pituitary region that can be associated with isolated growth hormone deficiency or combined pituitary hormone deficiency. EPP may be associated with an abnormal pituitary stalk and a small anterior pituitary gland and is seen in septo-optic dysplasia [4]. Some mutations in genes (e.g., HESX1, LHX4, SOX3) involved in the development of the pituitary region have been described [5].

We can suppose that, in our case, hyperprolactinemia was due to the presence of EPP adherent to the pituitary stalk, probably because of alterations in the dopaminergic action.

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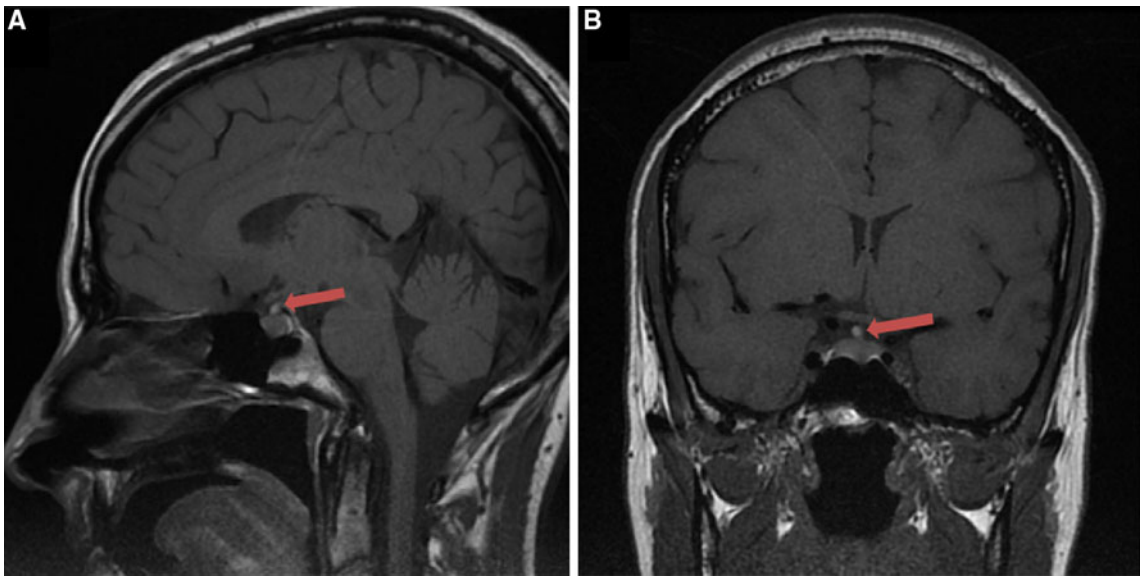

Fig. 1 **a** Sagittal T1 MRI, **b** coronal T1 MRI, both showing an hyperintense nodule (*arrow*) localized at the medium portion of the pituitary stalk compatible with EPP

To our knowledge this is the first report of hyperprolactinemia associated to EPP, therefore EPP should be considered in the differential diagnosis of hyperprolactinemia.

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