Neuroimaging Highlight

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Corticotroph Pituitary Stone

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CASE REPORT

A 65-year-old woman, with a history of type II diabetes and high blood pressure, was admitted for diverticulitis. Clinical examination showed a central obesity and a moon face. Neurological examination did not reveal any abnormality. Endocrine studies revealed Cushing’s syndrome with an increase of urinary free cortisol at 1578 nmol/24h (normal values: 30-220 nmol/24h), and a midnight salivary cortisol at 23.53 nmol/l (normal value at midnight: <6.1 nmol/l). Adrenocorticotropic hormone (ACTH) was increased at 71 ng/l (normal value: 10-60 ng/l), the high dose dexamethasone suppression test (8mg) and the corticotropin releasing hormone (CRH) test were consistent with Cushing’s disease. Indeed we noticed a cortisol suppression in response to the high dose test and not to the low dose dexamethasone test. There was also a significant increase of ACTH and cortisol following intravenous administration of a CRH bolus. Clinico-biological status suggested a diagnosis of Cushing’s disease. Magnetic resonance imaging (MRI) and computed tomography (CT) (Figure 1), revealed a calcified intrasellar mass, extending to the suprasellar space, without invasion of the optic pathways or cavernous sinus. This mass appeared heterogeneously hypointense in T1 and T2 weighted-images (wi). The periphery of the mass was slightly hyperintense in T1 and T2 wi with an enhancement after gadolinium administration (Figure 2). In addition petrosal sinus sampling was performed, showing an abnormal ACTH secretion in the left side. Investigations suggested a largely calcified pituitary macroadenoma; also named pituitary stone. The hyperintense enhanced peripheral zone corresponds to non-calcified adenoma tissue.

Trans-sphenoidal hemi-hypophysectomy, taking the calcified area, was performed. The histological study confirmed a calcified pituitary macroadenoma with a strong immunocytochemistry expression of ACTH within the calcified area; ACTH positivity was also noted in the surrounding tissue.

The post-operative course was uneventful and the Cushing’s syndrome resolved.

DISCUSSION

Calcified sellar lesions are rare. The differential diagnosis includes: craniopharyngioma, pituitary adenoma, Rathke cleft cyst, meningioma and chordoma. Craniopharyngioma is the most common diagnosis.

There are mainly two types of calcifications: capsular curvilinear calcifications and nodular central calcifications. The nodular pattern suggests craniopharyngioma and the curvilinear pattern suggests pituitary adenoma (Figure 1 and 2) or rarely a calcified Rathke cleft cyst.

Calcifications are present in 15-25% of pituitary adenoma. The calcifications are radiologically detected in 0.2 to 14% of cases. Largely calcified pituitary adenomas are very rare (see video on-line).

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Calcification in adenomatous tissue could be secondary to necrosis during silent pituitary apoplexy. In fact, old bleeding was found around and inside the calcified adenomas. It may appear following a bromocriptine treatment or radiation therapy. Calcifications are most common in prolactin pituitary adenomas, and very rare in corticotroph. The first corticotroph pituitary stone was reported in 1989 with a spontaneous resolution of a Cushing’s disease. Authors supposed that the large calcifications destroyed the ACTH-producing tissue.

An amyloid deposition is present in 71% of the pituitary adenoma, probably related to the peptide-hormonal synthesis process and the degenerative changes of the tumor. The amyloid deposition occurs commonly with a stellar-perivascular distribution and rarely with a spherical presentation. The spherical presentation appears almost exclusively in prolactin adenoma, and can appear as high density spots on CT, simulating an extensive calcified pituitary adenoma.

The treatment of choice of pituitary adenoma is surgery. Transsphenoidal approach showed excellent results regarding safety and efficacy. The success of this approach depends on many factors, including the size, the location, the subtype, the invasiveness and the histopathological markers of the tumor.

**REFERENCES**