Silent Corticotroph Pituitary Adenoma 16

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Silent corticotroph pituitary adenomas are pituitary adenomas that require special attention. They are clinically and biologically silent but harbor ACTH-secreting cells at pathology. They may convert into active adenoma responsible for Cushing disease. Because they are endocrinologically silent, silent corticotroph adenomas are usually diagnosed when large and behave like nonfunctioning macroadenomas, being responsible for headaches, visual impairment, or pituitary deficiency (Fig. 16.1). Hemorrhage and apoplexy may be more prevalent that in nonfunctioning adenomas and ACTH-secreting adenomas (Fig. 16.2). Silent corticotroph adenomas can recur easily (Fig. 16.3), and present as aggressive tumors with frequent invasion of the cavernous sinus. Surgical treatment may fail, thus leading to different trials of multimodal therapy, including stereotactic radiosurgery, somatostatin analogs, and chemotherapy such as temozolomide.

Interestingly, a recent paper reported suggestive MRI features of silent corticotroph adenomas. Indeed, when compared with ACTH-secreting or nonfunctioning macroadenomas, silent corticotroph adenomas appeared significantly more frequently with a multimicrocystic pattern, best demonstrated on T2WI (Figs. 16.1, 16.3, and 16.4). In our experience there is a trend for silent corticotroph pituitary macroadenomas to harbor a multimicrocystic component, but this pattern is not pathognomonic, and may be observed in ACTH-secreting and gonadotropic adenomas as well. In general, corticotroph adenomas, when large, have a high chance of developing cysts, either macrocysts, microcysts, or a combination of both (Fig. 16.5). Apoplexy is another way to discover silent corticotroph adenoma. MRI is then nonspecific and the nature of the underlying adenoma remains unspecified, except if a nonnecrosed area of the adenoma appears with the suggestive multimicrocystic pattern.

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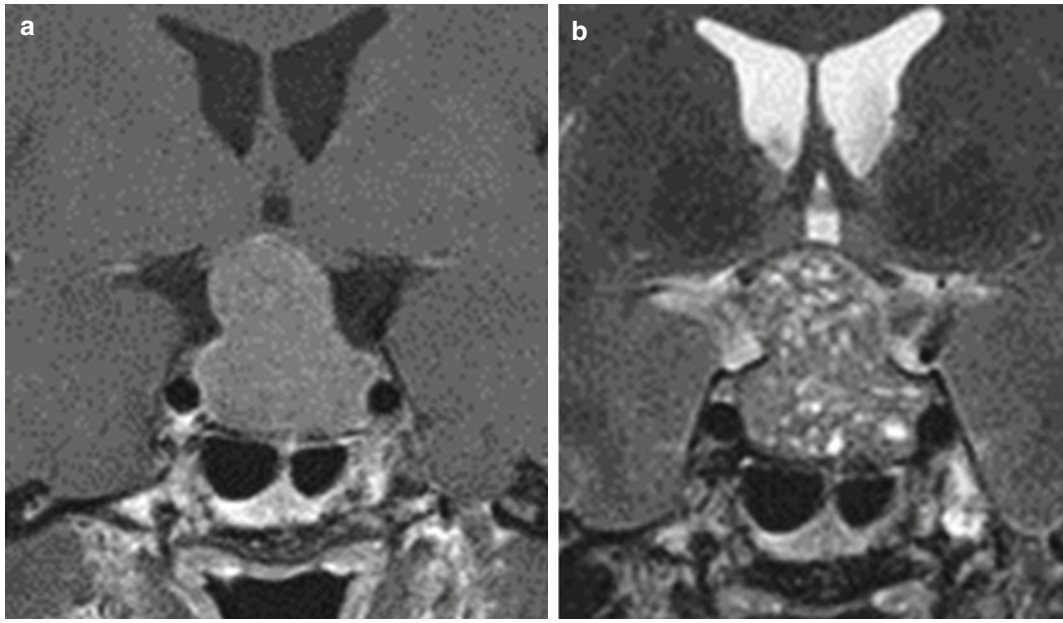

Fig. 16.1 Silent corticotroph pituitary macroadenoma in a 35-year-old woman with headaches, vertigo, and tinnitus. Laboratory studies revealed mild hyperprolactinemia and hypothyroidism. Surgery was performed and the diagnosis of silent corticotroph adenoma confirmed by pathology. (**a**) Coronal CE T1WI demonstrates a typical figure-of-8 shaped intra-suprasellar homogeneous adenoma. (**b**) Coronal T2WI depictes a unique multimicrocystic pattern, a feature frequently observed in silent corticotroph adenomas

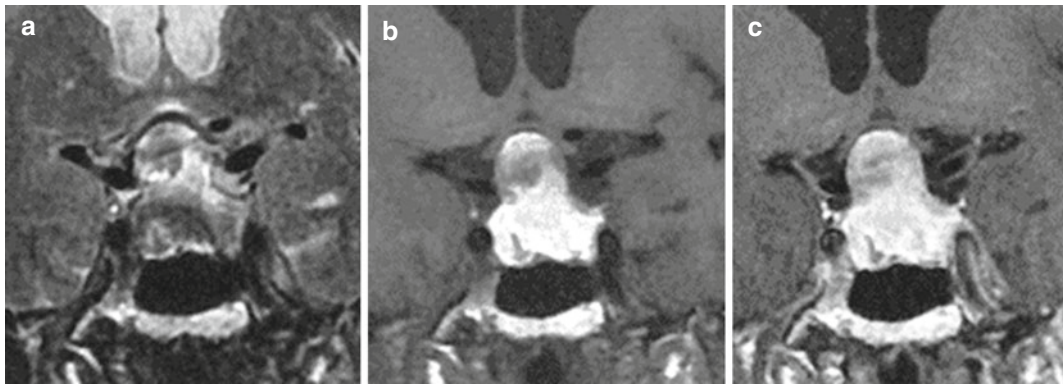

Fig. 16.2 Apoplexy of a silent corticotroph pituitary macroadenoma in a 71-year-old woman with visual impairment and headaches lasting for a few weeks. Coronal T2, T1, and CE (**a**–**c**) T1 WIs reveal heterogeneous signal intensity with predominant T1 hyperintensity and T2 hypointensity of an intra-suprasellar adenoma impinging on the optic chiasma. Pathological examination of the resected tissue demonstrated positive ACTH expression

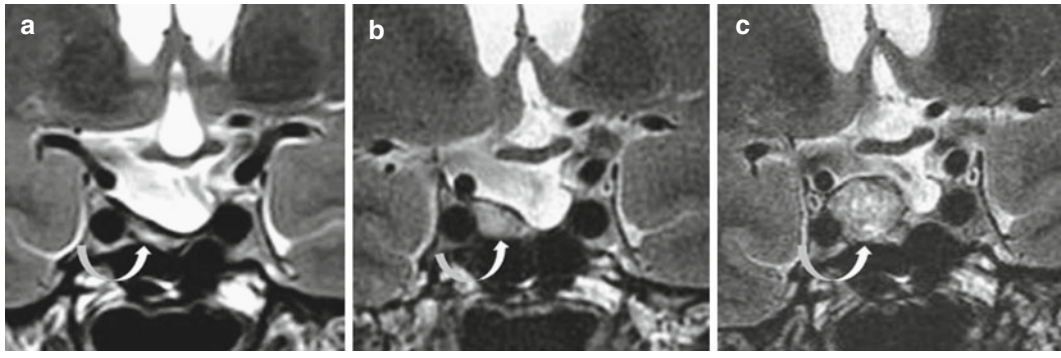

Fig. 16.3 Recurrent silent corticotroph pituitary adenoma in a 52-year-old man. (**a**–**c**) Coronal T2WIs. Episode of severe headache evoking a pituitary apoplexy 4 months previously with subsequent pituitary deficiency. (**a**) Small right T2-hyperintense tumoral remnant (*arrow*). (**b**) Two years later, increased size of the remnant (*arrow*). (**c**) Frank recurrence with obvious increased of tumoral volume 8 months later (*arrow*). Microcystic MRI pattern. Adenomectomy was performed and the diagnosis of corticotroph adenoma obtained by pathological examination

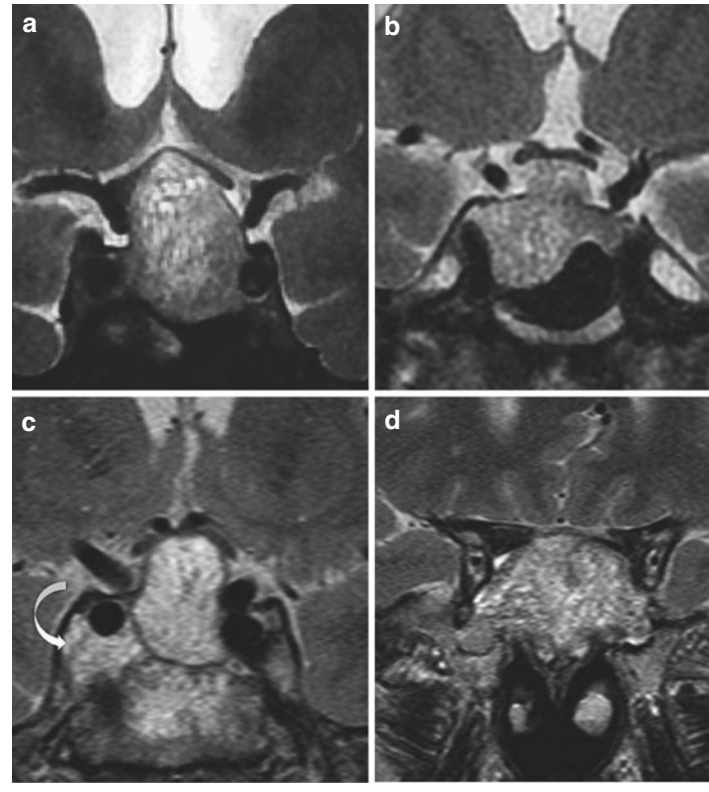

Fig. 16.4 Coronal T2WIs (**a**–**d**) in different patients with pathologically proven silent corticotroph pituitary adenomas. All adenomas harbor a multimicrocystic pattern, a feature suggestive of this entity. Cavernous sinus invasion is obvious in (**c**) (*arrow*)

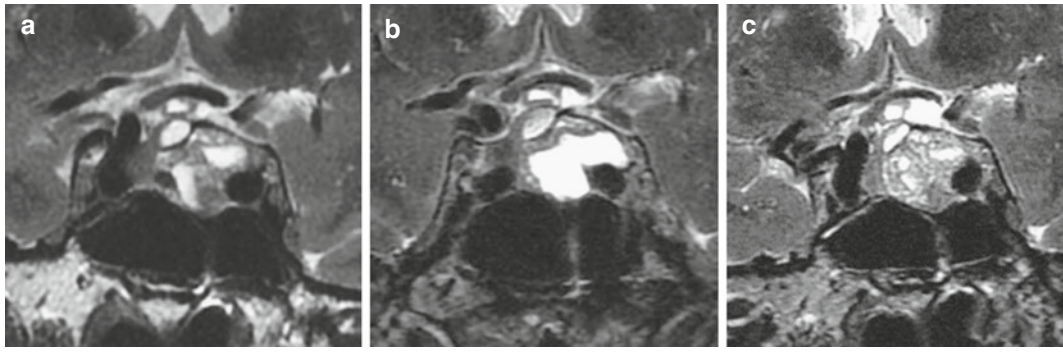

Fig. 16.5 Recurrent silent corticotroph pituitary adenoma in a 67-year-old woman. Visual field defect. (**a**–**c**) Coronal T2WIs. Rapid changes of the cystic MR pattern. (**a**) Four years after surgery. (**b**) Six months and (**c**) 12 months later

Further Reading

Cazabat L, Dupuy M, Boulin A et al (2014) Silent, but not unseen: multimicrocystic aspect on T2-weighted MRI in silent corticotroph adenomas. Clin Endocrinol 81:566–572

Cooper O (2015) Silent corticotroph adenomas. Pituitary 18:225–231

Nishioka H, Inoshita N, Sano T, Fukuhara N, Yamada S (2012) Correlation between histological subtypes and MRI findings in clinically nonfunctioning pituitary adenomas. Endocr Pathol 23:151–156