VANISHING PITUITARY LESION WITH TRANSIENT CENTRAL ADRENAL INSUFFICIENCY

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Introduction

The differential diagnosis for sellar and suprasellar lesions is broad and includes neoplastic entities such as pituitary adenoma, metastases, Rathke's cleft cyst, craniopharyngioma, and meningioma but also vascular lesions and inflammatory lesions such as sarcoidosis and autoimmune hypophysitis. We present the case of a patient with a vanishing pituitary mass of unknown etiology and transient, recurrent, central adrenal insufficiency.

Case presentation

A lady aged 47 years presented sudden secondary **amenorrhea and galactorrhoea** discovered incidentally during mammography. The local endocrinologist diagnosed her with **central hypothyroidism, chronic autoimmune thyroiditis and mild hyperprolactinemia** (2.5xULN). She received LT4 replacement. One year later (Nov.2020), she performed an MRI which revealed a **1.1 cm low-uptake pituitary mass** in contact with the internal carotid arteries and optic chiasm; left deviation of the pituitary stalk (figure 1a, 1b). PRL levels were still elevated, thus, CAB 0.5 mg x1/week was initiated, but amenorrhea persisted.

Jan 2021: was diagnosed in our clinic with **central hypothyroidism and hypogonadism** (no stimulation on Diphereline 0.1 mg test); **normal adrenal function and borderline GH insufficiency**. PRL levels were normal on 0.25 mg CAB/week. We excluded sarcoidosis and germinoma - on normal ACE, bHCG, CEA, thorax CT.

May 2021, after 7 months of low dose CAB and E-P, the pituitary gland was mildly increased and homogeneous at MRI, **the mass was no longer visible** (figure 2a, 2b).The patient had asymptomatic central adrenal insufficiency.
Diagnosis: LYMPHOCYTIC HYPOPHYSITIS ?

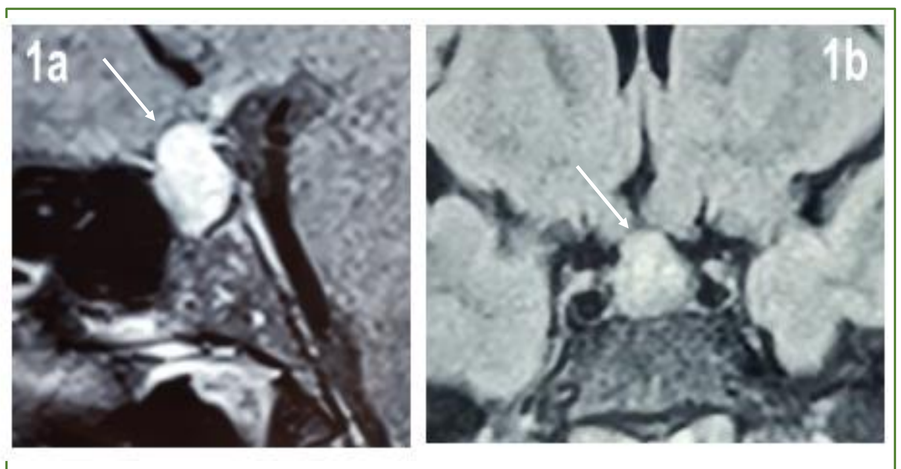

Figure 1a, 1b. Initial imaging on contrast enhanced sagittal and coronal MRI

Antiphospholipidic antibodies for ant and post pituitary were negative. She received glucocorticoid (GC) replacement until January 2022 when **baseline** and **Synacthen-stimulated cortisol levels were normal;** GC were withdrawn.

May 2022: again asymptomatic **low cortisol levels**, normal ACTH. No detectable intake of exogenous GC.

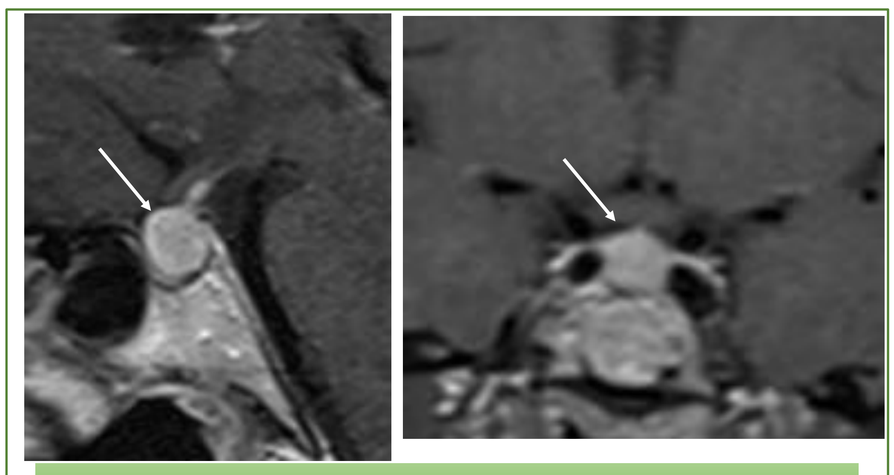

Figure 2a, 2b. Imaging after 7 months of CAB on contrast enhanced MRI

	January 2021-Initial evaluation	May 2021	June 2021	October 2021	January 2022	May 2022
Cortisol 8 a.m. (N: 6-22 ug/dL)	12.33	3.67 -3.53 Start GC	4.8 - on Prednisone 5 mg/day	7.37 – on PDN	7.42 on PDN. Stop GC	0.99 - 3.54
ACTH (N: 3-66 pg/mL)		9.75				9.95

Jan 2022 Synachten test 0.25 mg i.m	Base line	30'	60'	90'
Cortisol (4.8 – 19.5)	6.35	14.7	17.8	19.3

May 2022: She was recommended Prednisone 5 mg in stressful situations or adrenal insufficiency symptoms, LT4 and estro-progestin therapy. Long-term follow-up.

Discussion

- Primary lymphocytic hypophysitis (LH) typically presents as a homogeneous pituitary enlargement with intense and homogeneous enhancement post-gadolinium and no deviation of the stalk, features that can help differentiate from pituitary adenomas at MRI[1] – however our patient had pituitary stalk deviation, thus the diagnosis was not clear.
- Primary LH can be self-limiting and spontaneous remission may occur, however it may evolve to fibrosis and pituitary atrophy in the chronic stages of the disease, which often impair pituitary function. Caturegli et al. reported that in 379 patients with primary LH only 4% of patients had spontaneous remission with recovery of pituitary function, while most patients required long-term replacement of one or more pituitary axes[2].
- On the other hand, cases of spontaneous regression of pituitary adenomas are rare but have been reported in patients after pregnancy, with primary hypothyroidism after thyroxine treatment and with pituitary macroprolactinoma.[3]
- A pituitary biopsy would have been be useful for the histological diagnosis, but the patient refused the procedure.

Conclusion

We present a female patient with a vanishing pituitary mass, established central hypothyroidism and hypogonadism, mildly increased PRL (probably due to pituitary stalk compression), and late, intermittent, asymptomatic central adrenal insufficiency. While it may be interpreted as a nonfunctioning pituitary adenoma shrunken by cabergoline treatment, a more likely possibility is an evolutive pituitary inflammatory process, e.g., primary lymphocytic hypophysitis.

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The authors declare no conflicts of interest.