Secondary adrenocortical insufficiency and renal impairment in a patient presenting with hyperprolactinemia

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Introduction

Primary hypothyroidism has been described as a precursor to pituitary hyperplasia in literature.
Resulting pituitary hyperplasia can cause hyperprolactinemia by stalk effect or can affect other hormonal axis.
Following adequate hormone replacement with L-thyroxine both symptoms and pituitary hyperplasia
regressed on average within a few months

Case Report

We report a case of 19 year old lady who was referred to endocrine clinic with galactorrhoea associated with hyperprolactinemia. An MRI, arranged by GP, had had revealed an enlarged pituitary with a 14x8 mm macroadenoma slightly distorting the optic chiasma. Visual fields were normal on confrontation but showed restricted fields in both eyes on Goldman perimetry. She was commenced on cabergoline to which her galactorrhoea responded quickly. On subsequent visit her profile revealed severe primary hypothyroidism (TSH- 742mU/L and fT4<3.4). Cortisol was 283 nmol/L; gonadotrophins were normal, as was her IGF-1. Her creatinine was raised despite her slender built. Creatine Kinase was moderately elevated (297iU/L).
 She was commenced on Levothyroxine 50ug OD (later on increased) along with hydrocortisone (15mg am. 10 mg mid-afternoon). A short synacthen test, carried out less than 2 weeks after starting hydrocortisone, showed an abnormal response. Her subsequent ACTH (after omitting hydrocortisone the previous evening and delaying the morning does till after the test) was 17.1 ng/L; cortisol was 30 nmol/L at the time. Adrenal antibodies were negative. In due course with improvement in thyroid function her visual fields improved on perimetry with slight regression in size of pituitary gland.

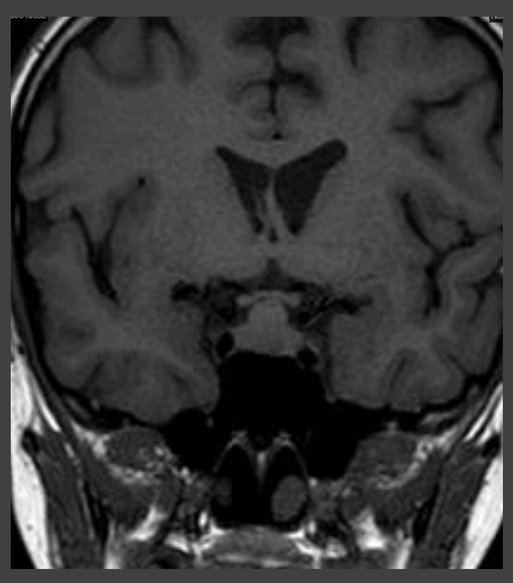
Conclusions

Her primary Hypothyroidism appears to have caused compensatory pituitary hyperplasia with associated hyperprolactinemia due to high TRH +/- pituitary stalk compression. Her pituitary hyperplasia appears to have caused secondary adrenal insufficiency. Pathophysiology of impaired renal function in hypothyroidism is multifactorial; the reduction in GFR due to the lower cardiac output and renal blood flow is likely to be the predominant mechanism

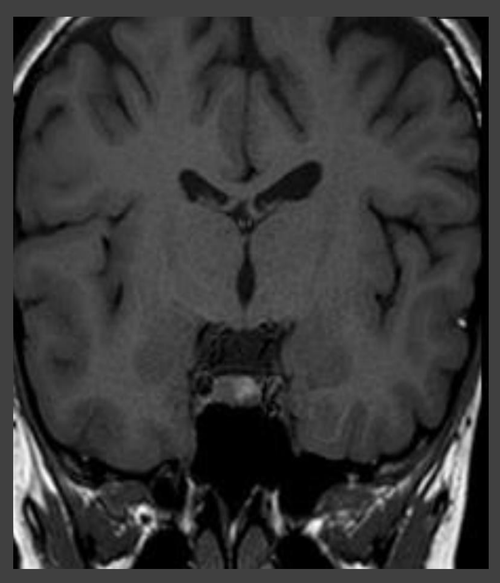
Investigations

	At presentation	After 7 months
Prolactin mu/L	1074	342
TSH mu/L	**742**	**71**
T4 pmol/L	<3.9	10.1
T3 pmol/L	<1.5	3.1
eGFR	45	76
Creatinine umol/L	148	88
Cortisol nmol/L	0min-108 30min-289	90min-390

Imaging



At Presentation



Follow up

References

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