

A Case of a Functioning Gonadotroph Cell Adenoma of the Pituitary

H. Wood, A. Martin, T. Howlett & M.J. Levy

Department of Endocrinology, University Hospitals of Leicester NHS Trust

A 51 year old man was referred to the endocrine clinic with a recent history of dizziness (worse with walking and standing from sitting), tiredness and weight loss of 2 kg. There was no history of abdominal pain, vomiting and no change in skin colour and he reported no headaches or visual disturbances. He was on no medication.

On examination, he was of normal weight (BMI 24.1); there was no pigmentation or postural drop in blood pressure. Chest, cardiovascular and abdominal examinations were unremarkable. A possible mass was felt in the right scrotum. There was no visual field defect to confrontation with a red pin.

Initial investigations showed hyponatraemia of 120mmol/l (NR 135-145), normal potassium of 4.6mmol/l (NR 3.5-5.5). Chest x-ray was unremarkable. A short synacthen test performed during the clinic visit revealed hypoadrenalism (Cortisol: 47 nmol/L basally and 163 nmol/L at 30min)

Further hormone assays confirmed that hypoadrenalism was due to ACTH deficiency with TSH deficiency with a low free thyroxine of 9pmol/l and inappropriately normal TSH of 2.0umiu/L (NR 0.5-6.0). Growth hormone was 2miu/l and IGF1 below the age-related normal range at 74ug/L suggesting GH deficiency. Prolactin was normal at 378miu/l. In spite of this extensive hypopituitarism, testosterone was consistently normal (20.8 – 25 nmol/l; sex hormone binding globulin 37) with high normal LH (7.8-12.6 iu/l and elevated FSH (49-69 iu/L).

MRI pituitary revealed a large pituitary macroadenoma measuring 3.7 x 2.6 x 3.2cm with suprasellar and inferior extensions and optic chiasm compression. Ultrasound showed both testes appeared normal in size, outline and texture but did demonstrate several simple epididymal cysts. Formal visual field testing revealed a minor left upper outer quadrant defect only.

He was replaced with hydrocortisone, which reversed his weight loss, and subsequently trans-sphenoidal resection of the tumour was performed. Post-operatively, his basal cortisol remained low and he continued to have a TSH deficiency and a low IGF-1. Serum testosterone fell to 1.4 nmol/L with undetectable LH of <0.5 and FSH of 4.2. He was given levothyroxine and continued steroid replacement and reported symptoms of hot flushes and low libido, which responded to testosterone replacement with Testogel. Routine post-op MRI is awaited.

Histology of the tumour showed a pituitary adenoma with positive immunostaining for FSH and LH in keeping with a gonadotroph cell adenoma of the pituitary.

When pituitary adenomas are assessed by immunostaining, it has been shown that gonadotroph adenomas may account for 15 to 20 % of all pituitary adenomas¹. The majority of these are clinically non-functioning adenomas that present with symptoms due to the increasing size of the tumour. Typically patients present with headache, visual disturbance or symptoms of hypopituitarism. Rarely these tumours can secrete clinically significant quantities of LH or FSH or a combination of both. Testicular enlargement may also be reported. Very rarely do these adenomas secrete intact LH causing supranormal levels of testosterone.

Our case represents a rare example of a gonadotroph adenoma causing panhypopituitarism but with maintenance of normal circulating testosterone levels due to gonadotrophin secretion by the adenoma itself. Treatment with surgery successfully resolved the tumour mass but resulted in postoperative panhypopituitarism and revealed the underlying gonadotrophin deficiency. It is unclear whether sustained FSH hypersecretion was involved in development of his multiple epididymal cysts.

REFERENCES

1. Chanson P. Gonadotroph pituitary adenomas. *Ann Endocrinol.* 2000 Sep; 61(3):258-68