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Low Libido Despite High Testosterone Levels in a Man With FSH-Secreting Pituitary Macroadenoma

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Background: Up to 60% of non-functioning pituitary adenomas stain for gonadotrophins on immunocytochemistry but do not secrete excess gonadotrophins. Functioning gonadotroph adenomas with clinical manifestations are extremely rare and majority of these are FSH secreting macroadenomas. Diagnosis may be difficult in pre-menopausal women with high oestrogen and variable FSH levels but in men, the unusually high gonadotrophins give the clues for diagnosis.

Clinical Case: We present the case of a 37-year-old Caucasian male with clinical manifestations of an FSH-secreting pituitary macroadenoma. He had sexual dysfunction with loss of libido for a year followed by bilateral testicular pain and enlargement (right >left). He was initially investigated under urology and had repeated treatment for suspected epididymitis. The diagnosis came to light many months later when he presented to emergency department with headaches and visual disturbance. Brain imaging confirmed a pituitary macroadenoma (3×2.5×3cm) with optic chiasm compression. He had bilateral superior temporal visual field defects. Pituitary profile showed unusually high FSH 200 IU/L (0.7-11.1), normal LH 7.6 IU/L (0.8-7.6) and a high normal testosterone 27 nmol/L (8.5-29). Prolactin was slightly elevated at 474mIU/L (0-350), cortisol and TSH were in normal range. He had polycythaemia (Haemoglobin 181g/l and HCT 0.51 L/L) and old blood results indicate his haemoglobin was above the upper limit of normal for previous 10 years.

This gentleman had a successful transsphenoidal hypophysectomy with near complete resection of pituitary adenoma. His vison improved immediately after surgery. Histology of the resected adenoma showed gonadotroph differentiation and immunoreactivity predominantly with FSH and also with LH and prolactin stains. A day after surgery, his FSH levels dropped to 18 IU/L and LH was undetectable. Symptoms of hypogonadism had gradually worsened post-surgery and 2 months later, his pituitary profile showed severe hypogonadotrophic hypogonadism (FSH 3.6 IU/L, undetectable LH and testosterone 0.9 nmol/L). He was started on testosterone replacement which he continues till date. He remains asymptomatic with no testicular pain and the last MR imaging, a year after his surgery showed no recurrence in the pituitary adenoma.

Conclusion: Testicular enlargement and hypogonadal symptoms with low testosterone levels are recognised features in FSH secreting pituitary adenomas. Our patient had testicular enlargement but consistently high testosterone levels prior to surgery and polycythaemia for many years prior to presentation. This may be due concomitant hypersecretion of both FSH and LH from his pituitary adenoma. The reason for low libido despite high androgen levels was not entirely clear.

Symptoms of hypogonadism overlap with many non-gonadal illnesses. A normal testosterone level in most cases would not lead to further endocrine investigations. Our case highlights the need to suspect such rare underlying pituitary pathology when dealing with unusual combination of hypogonadal symptoms, testicular enlargement and normal or high normal testosterone levels.

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