

## Acromegaly due to Primary Somatotroph Hyperplasia

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The most common cause for acromegaly is a somatotroph (GH-secreting) adenoma of the anterior pituitary gland. Acromegaly resulting from ectopic GH or GHRH hyper-secretion is exceedingly rare (< 1%). We present a patient with classical acromegaly whose pituitary histology showed somatotroph hyperplasia rather than typical adenoma.

A 21 year old male was referred to this tertiary centre in October 2007 with classical clinical and biochemical features of acromegaly (random GH >25 µg/L and IGF-1 >208 nmol/L). Cranial MRI suggested a 1.4 by 2.5 cm pituitary macroadenoma with supra-sellar extension making contact with the optic chiasm and bilateral cavernous sinus invasion. He was hypertensive, euglycemic and had no visual field defects. Other anterior pituitary hormones including prolactin were normal. He underwent trans-sphenoidal exploration in November 2007 during which complete macroscopic excision of a firm, bulging tumour was carried out. Pathological examination of the resected gland showed primary somatotroph hyperplasia with reactive changes and no clear evidence of a pituitary adenoma. The reticulin network was intact with expansion in places. Immuno-histochemistry showed weak GH positivity while isolated cell nests were positive for FSH, LH, TSH, prolactin and ACTH.

Post-operatively, his acromegaly remained active (random GH 15 µg/L; IGF-1 > 208 nmol/L) while MRI scan in February 2008 suggested residual tissue now confined to the sella and with right sided cavernous sinus invasion. Acromegaly was treated medically with dopamine agonist (Bromocriptine 10mg twice daily) and long acting somatostatin analogue (Lanreotide Autogel® 60mg monthly). He received pituitary radiotherapy in April 2008. During his most recent clinic visit in September 2008, while his clinical symptoms and hypertension had resolved completely, his biochemical parameters remain elevated (random GH 6.2 µg/L and IGF-1 153 nmol/L).

In view of somatotroph hyperplasia seen in the excised pituitary, further investigations to look for a source of GHRH were undertaken. His GHRH levels were in the normal non acromegalic range: 26 pg/ml, [normal < 49 pg/ml, Quest Diagnostics, California USA]. Fasting gut hormones and CT imaging of his chest, abdomen and pelvis failed to reveal any evidence of a carcinoid tumour. A second histo-pathological review of the pituitary tissue was again consistent with primary somatotroph hyperplasia.

### **Questions:**

1. Should further investigations be undertaken to locate a source of GHRH (e.g. In<sup>111</sup> Octreoscan)
2. What further treatment should be considered to achieve biochemical control of his acromegaly (e.g. Pegvisomant)