Visual field compression by a non-secreting pituitary tumour during pregnancy

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The pituitary gland enlarges physiologically in pregnancy, mainly because of lactotroph hyperplasia. Pre-existing pituitary tumours can likewise increase in size, but compressive effects may be due to displacement rather than enlargement.

CASE HISTORY

A woman of 29 was referred to the endocrine clinic for investigation and management of oligomenorrhea and infertility. Adrenal function was normal, she was euthyroid, and visual fields were normal to confrontation. Investigations suggested anovulatory cycles in the presence of adequate oestrogens (serum prolactin 430 mU/L [normal range 150–800], testosterone 1.4 nmol/L [0.3–2.5], sex hormone binding globulin 130 nmol/L [30–90], oestradiol 300 pmol/L; a menstrual bleed followed administration of medroxyprogesterone acetate 10 mg daily for 5 days). She was treated with clomiphene 50 mg on days 5–10 of the cycle and became pregnant that month.

At 18 weeks’ gestation she noticed abnormal vision in the left eye. On examination acuity was reduced on the left (6/12 compared with 6/5), with a central scotoma, loss of vision in the temporal field and an afferent pupil defect on the affected side. An MRI scan showed a pituitary mass extending into the suprasellar cistern, displacing and stretching the optic chiasm (Figure 1). The vertical height of the lesion was 2.0 cm. Serum prolactin, measured several times, had risen by about 100 mU/L—no more than is usual in pregnancy. Other endocrine investigations were likewise normal so she was diagnosed as having a non-functioning pituitary tumour. The obstetric team were reluctant for her to undergo pituitary surgery in the early middle trimester since, if surgery precipitated labour, the fetus would not be viable.

The presentation in pregnancy was thought to be due to upward displacement of the tumour by physiological pituitary enlargement; thus dopamine agonist therapy might allow surgery to be deferred. She was started on bromocriptine (5 mg on day 1, 5 mg twice daily on day 2 and 5 mg three times daily thereafter). Five days after the beginning of treatment the visual symptoms had disappeared, and on a repeat MRI scan the optic chiasm was no longer compressed, although there was negligible change in the size of the tumour (Figure 2). The visual fields returned to normal. Bromocriptine therapy was continued and the pregnancy proceeded without incident; a healthy child was born at 39 weeks’ gestation.

Postpartum, the patient was keen to breast-feed and elected to stop her bromocriptine. Within 2 days the visual field defect returned and she then underwent transphenoidal decompression of the tumour. On histological examination it proved to be a pituitary adenoma in which only about 5% of cells stained for prolactin and growth hormone. Postoperative endocrine assessment gave normal results except for hypoadrenalism. A year later she had an uncomplicated pregnancy.
COMMENT

We suspect that the beneficial effect of bromocriptine was achieved by an effect on the physiologically enlarged pituitary. A prolactinoma would be expected to respond to this agent, but this patient’s tumour was non-secretory; although 5% of cells stained for prolactin, serum prolactin concentrations were normal before and after pregnancy. 8–16% of non-secretory pituitary adenomas do respond to dopamine agonist therapy, so a direct effect cannot be ruled out; however, the rapidity of the therapeutic response makes this unlikely.

This is not the first report of successful use of bromocriptine to relieve compression of the optic chiasm by a non-secretory pituitary adenoma. In the previous cases, however, the effect was thought to be due to tumour shrinkage. Grossman and colleagues suggest that in these circumstances dopamine agonists work by shrinkage of normal lactotrophs, and recommend only temporary use (for about 2 weeks) before surgery. We could find no other cases where the physiological lactotroph hyperplasia of pregnancy had caused upward displacement of a clinically silent pituitary tumour.

REFERENCES


Mesh repair of sacral hernia following sacrectomy

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Sacrectomy is used in the management of sacral or advanced pelvic tumours. A sacral hernia is a rare complication of the procedure.

CASE HISTORY

A man aged 68 underwent resection of a sacral chordoma. The tumour involved all sacral segments and nerve roots below S1, therefore the sacrum was divided at the S1/S2 junction. The anus and rectum were excised, and an end colostomy was fashioned in the left iliac fossa. Postoperatively he developed a sacral wound infection that required formal incision and drainage. He also had a prolonged ileus.

Four months later he reported sacral pain and swelling, and on examination he had a large sacral hernia. Initially he refused surgical repair, but 2 years later he developed gallstone pancreatitis and requested sacral hernia repair at the time of his open cholecystectomy. The hernia was repaired via a transabdominal approach. The hernial sac contained small intestine, which was reduced, and two sheets of polypropylene mesh were used to repair the hernial defect and reconstruct the pelvic floor (Figures 1 and 2). The caecum and omentum were used to cover the mesh, to lessen the risk of small-bowel adhesion. A single suction drain was used and prophylactic antibiotics were administered. There were no postoperative complications and at 2-year follow-up there was no evidence of hernial recurrence.

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