

Pre-operative investigations confirmed elevated IGF-1: 240 ng/ml (NV: 40.2 - 225), random growth hormone (GH): 3.7 ng/ml (NV: <3) and prolactin (PRL): 805 mIU/L (NV: 45 -375). Thyroid and cortisol levels were normal. Pre-operative MRI of the pituitary revealed a heterogenous sellar mass with cerebrospinal fluid (CSF) fistulous connection between the floor of third ventricle and sella turcica. He underwent endoscopic chiasmopexy which revealed dense arachnoid adhesions in the sellar region with CSF gush on manipulation. No tumour was removed as a discrete adenoma could not be identified. Post-operatively, IGF-1 normalised to 184 ng/ml (40.2-225), but GH and PRL remained elevated at 4.9 ng/ml and 474 mIU/L, respectively. Failure of GH suppression following 75 g OGTT at 2.8 ng/ ml denotes persistent active acromegaly. As he had mild acromegaly with prolactin co-secretion and was unable to afford somatostatin receptor ligand therapy, he was commenced on cabergoline 0.25 mg twice a week.

CONCLUSION

Preceding case reports of spontaneous ventriculostomy secondary to obstructive hydrocephalus were due to neoplastic disease or benign aqueduct stenosis. We report a case of acromegaly with prolactin co-secretion who presented with a rare finding on pituitary MRI of spontaneous ventriculostomy.

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NON-FUNCTIONING PITUITARY ADENOMA COMPLICATING PREGNANCY

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INTRODUCTION

Pregnancies are rare in women with pituitary adenoma. We describe a patient with non-functioning pituitary adenoma who had spontaneous pregnancy. She developed progressive worsening of vision and underwent successful transsphenoidal surgery (TSS) in the second trimester.

CASE

A 35-year-old female presented with secondary amenorrhoea and galactorrhoea for 6 months. A large pituitary macroadenoma with mass effect coupled with mild increment in prolactin raised a suspicion of prolactinoma. Menstruation returned and galactorrhoea ceased following cabergoline therapy. She had a left nasal hemianopia, central hypothyroidism and hypogonadotropic hypogonadism. The other pituitary hormones were intact. Although prolactin remained suppressed for a year with cabergoline, there was no reduction in tumour size and serial perimetry showed gradual worsening of visual fields in both eyes. She was scheduled for TSS but was postponed. She presented at 17 weeks of a spontaneous pregnancy with left eye discomfort and no other symptoms of raised intracranial pressure. MRI of the pituitary showed unchanged tumour size exhibiting mass effect with no evidence of apoplexy. Perimetry showed worsening of peripheral scotoma on the left eye with optic atrophy. Hormonal evaluation showed new onset central hypocortisolism. She underwent TSS at 19 weeks POA. Histopathology confirmed pituitary adenoma but stained negative for prolactin. Post-operatively, she developed central diabetes insipidus requiring regular desmopressin. There was no improvement in vision.

CONCLUSION

Pregnancy is a known risk factor for pituitary apoplexy. However, worsening of mass effect could be due to physiological changes of pregnancy resulting in pituitary enlargement. Surgery is recommended early prior to pregnancy especially in those with large non-functioning pituitary adenoma for optimal pregnancy outcomes. Patients who become pregnant require multidisciplinary care and TSS in the second trimester is an option. Hormonal deficiencies and cranial diabetes insipidus should be addressed promptly.