Anaesthetic management for caesarean section in a case of previously operated with residual pituitary tumour

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ABSTRACT

Successful anaesthetic management for caesarean section in a case with previous pituitary tumour resection, with residual tumour, is reported. The pituitary gland undergoes global hyperplasia during pregnancy. Functional pituitary tumours may exhibit symptomatic enlargement during pregnancy. Growth hormone secreting tumour is associated with acromegaly which has associated anaesthetic implications of difficult airway, systemic hypertension, and diabetes and electrolyte imbalance. Intracranial space occupying lesions can increase intracranial pressure and compromise cerebral perfusion or cause herniation. We report management of this case.

Key words: Anaesthesia, lower segment caesarian section, pituitary tumour

INTRODUCTION

Anaesthesia for pituitary tumours booked for non neurosurgical surgeries is a challenge to an anaesthesiologist with the risk of sudden change in intracranial dynamics during administration of spinal anaesthesia or during laryngoscopy, intubation and extubation. There is chance of increase in tumour size during pregnancy. Here we report anaesthetic management for elective lower segment caesarian section (LSCS) with previous pituitary tumour resection – one with residual tumour done under general anaesthesia.

CASE REPORT

A 24-year-old female weighing 65 kg and height of 156 cm, with 38 weeks of gestation with history of complete transphenoidal resection of functioning pituitary macroadenoma (Growth hormone and Prolactin secreting) two years ago was posted for elective LSCS. She was on cabergoline 1 mg twice weekly as she had history of infertility. Medical history was unremarkable. She was primi gravida. Airway examination done two days prior to LSCS suggested a difficult intubation with Mallampati Class III, macroglossia, prominent neck fold in the nape and a protruding mandible. She had no history of snoring. All hematological, biochemical and hormonal investigations were unremarkable. Magnetic resonance imaging (MRI) of the brain done at 30 weeks showed tumour encasing left internal carotid artery with no evidence of raised intracranial pressure (ICP). It also suggested macroglossia with normal pharynx and larynx. Patient’s refusal deterred us from using subarachnoid block.

Difficult intubation cart was kept ready. Aspiration prophylaxis was given 30 min prior. Monitors (pulse-oximeter, electrocardiogram, capnometry and non invasive blood pressure) were attached. Patient was pre-oxygenated for 5 min. Rapid sequence induction was done using thiopentone sodium and rocuronium, intravenous lignocaine 1.5 mg/kg was used to suppress the stress response. Laryngoscopy was done using Macintosh blade size 4 to find Cormacke Lehane grade II and cuffed endotracheal tube (ID 7.0 mm) was passed. Anaesthesia was maintained with controlled ventilation and endotracheal intubation.
using standard anaesthesia protocols. A 2.9 kg baby was delivered with Apgar score of 8 at birth. All the vitals, blood loss and urine output were maintained well. Intravenous lignocaine 1.5 mg/kg was used for smooth emergence and patient was extubated once fully awake. Postoperative period was uneventful and breastfeeding was initiated 4 h later.

**DISCUSSION**

The pituitary gland is located at the base of the skull in the sella turcica. It is divided into anterior (adenohypophysis) and posterior (neurohypophysis) lobes.[2] The hypothalamus regulates hormone release from the anterior pituitary through hypothalamic releasing and inhibiting factors that reach the anterior pituitary by a complex portal vascular system. The anterior pituitary secretes at least seven hormones while the posterior pituitary stores and secretes two hormones, antidiuretic hormone and oxytocin.

Functioning pituitary adenomas produce an excess of the anterior pituitary hormones. They are usually diagnosed when the tumours are small. Adenomas secreting both growth hormone (GH) and prolactin are common. Other less common pituitary tumours are growth hormone secreting lesions resulting in acromegaly; adencorticotrophic hormone (ACTH) secreting tumours causing Cushing's disease and a very rare thyroid-stimulating hormone (TSH) secreting lesion resulting in hyperthyroidism. Prolactinomas may produce the amenorrhea-galactorrhea syndrome in females. As the pituitary lesion compresses the pituitary tissue, the sequence in which hormonal function is lost is gonadotrophins; growth hormone; ACTH and TSH.[2]

Patients with acromegaly exhibit a general overgrowth of skeletal, connective and soft tissues leading to enlargement of hands, prognathism and macroglossia. Thickening of laryngeal and pharyngeal soft tissues and vocal cords, reduction in size of laryngeal aperture, hypertrophy of uvula, epiglottic fold and recurrent laryngeal nerve palsy also occur.[3] All major organs increase in size, including the heart, lungs, liver, thyroid and kidneys. These patients require preoperative evaluation and management of coronary artery disease, cardiomyopathy with arrhythmias, hypertension (30%), diabetes (25%) and electrolyte imbalances.[3,4] Upper airway obstruction with sleep apnoea is a major cause of laryngeal airway obstruction and central depression.[3,4] Therefore, the risk of death from respiratory failure is threefold greater in patients with acromegaly.

Pressure effects on the normal pituitary gland from parasellar tumours can cause panhypopituitarism. Patients who have panhypopituitarism require replacement therapy with appropriate hormones. As glucocorticoids are also necessary to facilitate renal excretion of a water load, diabetes insipidus is usually not observed in the patient with pituitary insufficiency until cortisol replacement therapy is instituted. Preoperatively, the patient with panhypopituitarism will be receiving oral steroid and levothyroxine therapy and, when indicated, intranasal instillation of synthetic vasopressin. Other signs and symptoms in prolactin secreting microadenoma include amenorrhea, galactorrhea, anovulation, decreased libido, gynaecomastia and osteoporosis. These patients are usually women with secondary amenorrhea.

During pregnancy, the pituitary gland undergoes global hyperplasia and volume increases by 45%.[3] Tumour cells in patients with prolactinomas[3] and that with GH secreting tumours may exhibit symptomatic enlargement during pregnancy, probably due to the growth-promoting effect of estrogens. This may be because of an increase in either peritumoural edema secondary to increased sodium and water retention or blood volume in vascular tumours such as meningiomas. Accelerated tumour growth during pregnancy is likely, owing in part to the high levels of circulating progesterone that occur with gestation. As pregnancy itself is an insulin-resistant state, the pregnant acromegalic patient is at greater risk for hyperglycemia. There is also an increased incidence of hypertension and coronary artery disease in acromegalic patients, which poses potential risks to the fetus. However, none of these potential complications of elevated GH have been shown to have a deleterious effect in pregnant acromegalic patients.[2]

Active acromegaly during pregnancy does increase insulin resistance and thus increase the risk of gestational diabetes and hypertension. Underlying cardiac disease, from metabolic syndrome, hypertension, or acromegaly-associated cardiomyopathy, may become symptomatic during pregnancy. Pregnancy has not been found to alter the course of acromegaly other than in rare, reported cases of asymptomatic tumour enlargement. Whether tumour enlargement is more common in lesions that co-secrete prolactin remains unclear. If left untreated for the duration of pregnancy, it is thought not to have any adverse effects on the course of acromegaly, which otherwise is a chronic disease that can be addressed after delivery.
Diabetes insipidus (DI) can occur de novo during pregnancy or in the postpartum period, while pre-existing DI may be exacerbated during pregnancy. In addition, a transient DI may occur during pregnancy in the absence of a known defect in Arginine vasopressin (AVP) secretion. The increased demand for AVP during pregnancy may unmask subclinical or mild central DI. Glucocorticoid dose does not usually need to be changed during pregnancy, although symptoms occasionally indicate that a mild increase is required. Stress levels of cortisol or its equivalent are needed for labour and for any intercurrent major stress during the pregnancy. There is no mention of possible Sheehan syndrome. Important causes of postpartum pituitary necrosis include severe haemorrhage during and before parturition. The blood supply to the already enlarged pituitary gland is seriously compromised in times of acute volume depletion compounded by vasospasm due to circulating vasoconstrictors. The enlarged gland and low pressure in the portal system cause susceptibility to tissue hypoperfusion and infarction and it leads to an atrophic, hypofunctioning and a scarred gland. It has been suggested that patients with Sheehan syndrome have a small, rigid sella from the outset. The hyperplastic pituitary in this sella may be more likely to compress its blood supply, predisposing the gland to infarction if hypotension occurs. Pregnant women with type 1 diabetes, especially those with preexisting vascular disease, seem to be particularly at risk.

The pre-anæsthetic considerations are related to the endocrine and the tumour status. During the pre-anæsthetic evaluation, the size and location of the tumour and its effect on intracranial dynamics should be determined by preoperative MRI of brain. Difficulty with endotracheal intubation should be anticipated in an acromegalic patient. Mask ventilation may be difficult in acromegalic patients requiring oral airway.[3] There are four grades of airway involvement described in acromegaly; grade 1- no significant involvement; grade 2- nasal and pharyngeal mucosa hypertrophy but normal cords and glottis; grade 3- glottic involvement including glottic stenosis or vocal cord paresis; grade 4- combination of grade 2 and grade 3.[6] For patients with difficult airway and glottic abnormalities, awake fiber optic intubation is the method of choice. This obviates the need for a tracheostomy in all but the most severe cases.[7] The intubating laryngeal mask airway is another alternative in a patient with acromegaly. Raised ICP can result in either reduction in perfusion pressure with concomitant ischaemic injury or herniation of brain tissue causing brain injury.[8] The main aim of the anaesthesiologist is smooth induction of anaesthesia by avoiding coughing, straining by maintaining patient in deeper plane of anaesthesia, avoiding hypo or hypertension. Intravenous induction of anaesthesia with either thiopentone or propofol may be used. It will produce a fall in ICP by lowering the cerebral metabolic rate (CMRO₂) and the cerebral blood flow (CBF).[8] It is essential for pregnant patients to undergo MRI after four months gestation[8] especially those who have undergone pituitary surgery in the past to look for any residual tumour which can increase in size. Unlike computerized tomography (CT), MRI avoids the use of ionizing radiation. Risk benefit ratio is to be taken into consideration. However, it is to be avoided during pregnancy in the first trimester.

We suggest that any pregnant patient with history of pituitary tumour or resection of the same should undergo preoperative extensive evaluation and those with acromegaly for detailed airway assessment.

REFERENCES

1. Herman-Bonert V, Seliverstov M, Melmed S. Pregnancy in acromegaly: Successful therapeutic outcome. J Clin Endocrinol Metab 1990;65:72-31.
2. Bendo AA, Kass IS, Hartung J, Cottrell JE. Anesthesia for Neurosurgery. In: Barash PG, Cullen BF, Stoelting RK, editors. Clinical Anaesthesia, 5th Ed. Philadelphia: Lippincott Williams and Wilkins; 2006. p. 775-6.
3. Southwick JP, Katz J. Unusual airway obstruction in acromegalic patient—indications for tracheostomy. Anesthesiology 1979; 51:72-3.
4. Melmed S. Disorders of anterior pituitary and hypothalamus. In: Braunwald E, Fauci AS, Hauser SL, Longo DL, Kasper DL, Jameson JL, editors. Harrison’s Principles of Internal medicine, 5th Ed. New Delhi: McGraw-Hill; 2005. p. 2029-52.
5. Gonzalez JC, Elizondo G, Saldivar D, Nanez H, Todd LE, Villareal JZ. Pituitary gland growth during normal pregnancy: An in vivo study using magnetic resonance imaging. Am J Med 1988;85:217-20.
6. Hakala P, Randell T, Valli H. Laryngoscopy and fibreoptic intubation in acromegalic patients. Br J Anaesth 1998;80:345-7.
7. Ovassapian A. Fibreoptic Airway Endoscopy in anesthesia and Critical Care. New York: Raven Press, 1990. p. 57-79.
8. Drummond JC, Patel PM. Neurosurgical Anesthesia. In: Miller RD, Editor. Miller’s Anesthesia; 6th Ed. Philadelphia: Elsevier Churchill Livingstone; 2005. p. 2127-73.
9. Ravussin P, Guinard JP, Ralley F, Thorin D. Effect of propofol on cerebrospinal fluid pressure and cerebral perfusion pressure in patients undergoing craniotomy. Anesthesia 1988;43 (suppl):37-41.
10. Yamashita Y, Namimoto T, Abe Y, Takahashi M, Iwamasa J, Miyazaki K, et al. MR imaging of the fetus by a HASTE sequence. AJR Am J Roentgenol 1997;168:513-9.

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