

Diabetes insipidus and pediatric craniopharyngioma excision: perioperative challenges

Short communication

Craniopharyngiomas are rare, histologically benign, recurrent brain tumors, derived from pituitary gland embryonic tissue, that are seen most commonly in children between the ages of 5 and 10 years.¹ Their incidence ranges from 0.13 to 2 per one lakh population per year, with no variance by gender or race. Distribution by age is bimodal with the peak incidence in children at 5 – 14 years and in adults at 65 – 74 years.² They represent about 2 – 6% of all paediatric primary intracranial tumours.¹

Craniopharyngiomas are slow-growing, but locally invasive tumors and clinical presentation varies with the age at presentation, site and size of the tumor, structures compressed by it, changes in intracranial pressure (ICP) and presence of endocrine abnormalities. The most common clinical features are visual disturbance and endocrine deficiencies. Visual disturbance (sometimes blindness) is caused by compression of the optic nerve by the tumor. Hormonal manifestations may include hypothyroidism, growth failure, hypogonadism and diabetes insipidus (DI). Other manifestations are non-communicating hydrocephalous and raised ICP (nausea, vomiting, headache, altered sensorium), hypothalamic insufficiency (obesity, poor energy, somnolence, emotional lability, hallucinations) and cerebral parenchyma involvement (seizures).¹

Management traditionally involves decompressive surgery (gross total resection or subtotal resection) followed by radiation therapy with a five-year progression free survival rate exceeding 90%. The advent of newer techniques like endoscopic surgery and precision radiotherapy (especially, intensity modulated photon radiation therapy) have led to a significant decrease in the long-term side effects related to the traditional surgical approach.³

Preoperatively, a thorough preanesthetic multisystem assessment and airway evaluation is required. Investigations must focus on complete blood counts, blood sugar levels, liver function tests, kidney function tests, serum electrolytes and chest radiography. Complete endocrinological assessment including thyroid function tests, cortisol, growth hormone, sex hormones, adrenocorticotropic hormone and prolactin levels measurement is essential. Preoperative ophthalmologic assessment for visual acuity, field of vision and papilledema should be performed. A detailed medical history, focusing on antiepileptic drugs and hormonal therapy, should be obtained and these medications should be continued till the day of surgery. In view of extensive nature of the tumor and the risk of bleeding, availability of adequate blood and blood products needs to be ensured beforehand.

Induction and maintenance of anaesthesia depends on the patient's hemodynamic status and associated comorbidities. The prime goal is to maintain cerebral perfusion pressure by avoiding increase in ICP and major variations in mean arterial pressure.¹ Intraoperatively, standard monitoring, including electrocardiography, pulse oximetry, invasive blood pressure monitoring, temperature and end-tidal carbon dioxide (CO₂) monitoring is recommended. Catheterization for urine output monitoring is necessary to detect perioperative DI.

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Serial intraoperative arterial blood gas (ABG) analyses are needed to monitor partial pressure of CO₂ in arterial blood, serum osmolality, serum electrolytes and hemoglobin levels.

Proper positioning of the patient during the intraoperative period is important in preventing complications. Supine with lateral head tilt is the most commonly used position. Care should be taken while fixing the head with Mayfield head pins. Position of the endotracheal tube should be verified after the final patient positioning to detect any inadvertent migration.

Intraoperative complications most commonly include blood loss, diabetes insipidus, hypothalamic injury (temperature dysregulation), seizures, brain stem injury (comatose state) and adrenal insufficiency. Perioperative steroid supplementation with intravenous hydrocortisone infusion should be considered for preventing adrenal insufficiency. DI is rarely seen intraoperatively but is common in the postoperative period (70 – 90%).⁴ Copious urine output towards the end of surgery may be a early indicator. Management of DI is very challenging, especially in the paediatric cases with refractory hypernatremia, so much so that it becomes the main cause of poor outcome in these patients. DI is diagnosed when the patient develops polyuria (urine volume > 4 – 5 ml/kg/hour or >250 ml/hour for 2 or more consecutive hours) and plasma osmolality increases over 300 mOsm/kg, with the corresponding urine osmolality of less than 300 mOsm/kg.⁵ Plasma sodium concentration of more than 145 mEq/L is an important parameter indicating early intervention. DI should be promptly treated with fluid replacement and pharmacological agents like vasopressin or its synthetic analogue desmopressin.⁴ Frequent serial monitoring of plasma sodium levels is important for fluid management.⁵ 0.45 % saline should be used if plasma sodium concentration is more than 145 mEq/L, and it should be replaced with 5% dextrose solution if the concentration further rises above 150 mEq/L.⁵ Continuous intravenous infusion of aqueous vasopressin may be started at a dose of 0.5 mU/kg/hour and titrated to maintain urine output of over 2 ml/kg/hour. Desmopressin may also be used in a dose of 5 – 10 µg intranasally or 50 – 200 µg orally.⁵

Prolonged postoperative ICU care and ventilation with vigilant monitoring may be required. Serial measurements of urine output, osmolality and serum sodium levels along with ABG is recommended.

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