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CASE REPORT

MANAGEMENT OF INTRAOPERATIVE DIABETES INSIPIDUS DURING RESECTION OF CRANIOPHARYNGIOMA: A CASE REPORT

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Case Report

A 32 year old male patient presented with 2 months history of blurring of vision and headache , without any trigger , which improved partially with analgesics . He visited Vydehi hospital due to worsening of headaches.

Patient had history of diabetes mellitus 3 months back from the time of consultation and was taking metformin 500mg once a day , he also had history of newly diagnosed hypothyroidism past 12 days from the time of consultation and was taking thyroxine 75mcg once a day.

On general physical examination he had normal anthropometric measurements , bi-temporal hemianopia no pallor, icterus, cyanosis, clubbing, koilonychia, edema, lymphadenopathy. Airway examination normal.

After the admission MRI Brain was done and report showed multiloculated mixed intensity lesion with solid and cystic components and showing patchy blooming on gradient images suggestive of calcification in suprasellar region , superiorly the lesion indents hypothalamus and optic chiasma, posteriorly indents pituitary stalk and inferiorly indenting the pituitary gland.

The admission investigations showing diminished Cortisol 0.71 micro/dl, Testosterone < 10 ng/dl, Prolactin- 72.19micro g/L , HbA1C - 5.5%, FSH - 1.48, LH - 0.19, TSH- 1.41, T3- 1.18, T4- 3.32.

Standard IV induction was done with pre-medications - inj ondansetron 4mg , inj midazolam 1mg, inj glycopyrolate 0.2 mg , inj fentanyl 100mcg, inj propofol 100mg, inj atracurium 30mg , 1 hour into tumor resection , polyuria was noticed around 6-8ml/kg/hr . serum sodium and urine osmolality was sent , serum sodium - 155mEq/L and urine osmolality >330mOsm/kg

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Vasopressin infusion started at 0.1 milliunits/hour, urine output came down to 3ml/hour and tritrated to 0.025milliunits/hour.
Patient was extubated, uneventful and shifted to ICU.
In ICU T.Desmopressin 0.3mg in bedtime was given prophylactically.

Discussion

Craniopharyngiomas are uncommon, benign brain tumors that are typically derived from the pituitary gland, situated at the base of brain . Their exact cause is unclear, but they are believed to stem from remnants of embryonic tissue, particularly Rathke's pouch, which forms the pituitary gland during early development. The metaplastic transformation of adenophysis cells of the pituitary gland that leads to the development of craniopharyngioma.

These slow-growing tumors are more prevalent in children and adolescents, most commonly in children between the ages of 5 and 10 years. They represent about 2% to 6% of all paediatric primary intracranial tumours, manifest diverse symptoms based on size and location, including hormonal imbalances, vision issues, and headaches. Genetic factors and mutations may contribute to their development. The affected individuals can exhibit both pressure symptoms and endocrine derangements. There is a generalized deficiency of various pituitary hormones like growth hormone, gonadotropin, thyroid stimulating hormone and adrenocorticotropic hormone.Hypothalamic damage and endocrine disturbances are more common in children and typically ensue before the appearance of visual symptoms.

Generally present with

Intracranial hypertension: nausea, vomiting, headache, papilledema, altered level of consciousness .

Visual pathway disturbances: bitemporal hemianopsia, homonymous anopsia, optic nerve atrophy in severe cases.

Endocrine disturbances: hypothyroidism, growth failure, hypogonadism, syndrome of inappropriate antidiuretic hormone secretion, diabetes insipidus (DI), precocious puberty. Preoperative endocrine evaluation including measurement of growth hormone, thyroid stimulating hormone, cortisol, follicular stimulating hormone/luteinizing hormone, prolactin, and serum electrolytes should be performed and corrected as indicated prior to surgery.Consultation with an endocrinologist is recommended and preoperative considerations include administration of stress dose steroids and assessment of volume status and electrolyte disturbances.

Thalamus, hypothalamus, and/or frontal lobe disturbances: aphagia, obesity, poor energy, somnolence, emotional lability, hallucinations, autonomic disturbances

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Brain parenchyma disturbances: seizures, cognitive dysfunction.

Diagnosis heavily relies on MRI scans. It is the investigation of choice for precise diagnosis of the tumor location and to supplement the central-type visual field defect diagnosis during ophthalmic examination.Radical surgery is often necessary but carries risks like neuronal damage to the parts of the brain including thalamus, mammilo-thalamic tract and basal forebrain. The morbidity and mortality due to radical surgical treatment is quite high, to an extent of 40–50%. Prognostic criteria for better outcomes include age >5 years, tumor size <4 cm, complete removal, and minimal endocrinological dysfunction. Treatment involves surgery, radiation therapy, and sometimes hormone replacement therapy, tailored to the tumor's characteristics and the patient's condition. The management of craniopharyngioma was via a transcranial approach with the aim of complete resection, but this was associated with greater injury to vital tissues, resulting in a high frequency of visual impairment, hypothalamic dysfunction, central diabetes insipidus, and anterior pituitary hormone deficiencies. In recent years, less invasive surgical approaches followed by adjuvant therapies have gained acceptance. This includes an endoscopic endonasal rather than open approach to tumor resection, partial resection with or without radiation therapy to eradicate residual tumor, or procedures aimed at tumor decompression in patients with a significant cystic component, including endoscopic cyst fenestration or placement of an Ommaya reservoir into the tumor cyst for drainage with or without subsequent instillation of antineoplastic agents. This shift in surgical treatment approach away from gross total resection has led to better preservation of the pituitary stalk and improved endocrine outcomes, with a significant reduction in the occurrence of central DI .

For surgical resection, Secure an intravenous access . Having at least two large-bore peripheral intravenous lines is recommended for prompt resuscitation, given the proximity to critical vessels such as the internal carotid artery and the circle of Willis. consider inhalational induction for those who are appropriately fasting and lack significant intracranial hypertension. When choosing maintenance options for anesthesia, the focus should be on facilitating a rapid awakening post-surgery for neurological examination, especially when intraoperative neurophysiological monitoring is planned.
Arterial cannulation is advisable for both hemodynamic monitoring and close tracking of blood gases and serum electrolytes, particularly in patients at high risk of intraoperative diabetes insipidus. Additionally, placing a urinary catheter is recommended to promptly detect diabetes insipidus.

Perioperative Management of Central Diabetes Insipidus

Diabetes insipidus results due to deficiency of antidiuretic hormone, or arginine vasopressin, produced in the hypothalamus, stored and released from the posterior pituitary, acts on the distal tubules and collecting ducts of the kidneys to promote reabsorption of water. A lack of antidiuretic hormone due to surgical resection therefore results in diuresis and electrolyte imbalance. It is characterized by the inability to concentrate urine resulting in polyuria and subsequent polydipsia.

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Polyuria is defined as excretion of a urinary volume greater than 2 L/m2 /day and 40–50 ml/kg/day in adults . A serum osmolality (Sosm) > 300mOsm/kg at the same time as inappropriately dilute urine (Uosm 4 ml/kg/hr of urine for two or more hours, specific gravity.

Signs suggestive of DI include the following:

- Urine output ≥ 4 mL/kg/h
- Serum Na ≥ 145 mEq/L
- Serum osmolality >300 mOsm/kg
- Urine osmolality < 300 mOsm/kg
- Polyuria ≥ 30 minutes
- Other causes of polyuria ruled out (eg, mannitol, saline, osmotic contrast agents, glucose, diuretics).

Approximately 75% of patients develop DI following extended surgical resection of a pituitary tumour and DI occurs in 10% to 44% of patients after transsphenoidal pituitary surgery. Managing DI can be challenging, and a multidisciplinary discussion should be held during the preoperative evaluation for the management. Prior approaches for the perioperative management of central DI relied on the replacement of fluid losses with normal saline or lactated Ringer solution, After a reduction in the rate of diuresis, the intravenous fluids rate should be at two-thirds maintenance as needed to support blood pressure and to match urinary output plus insensible losses until antidiuresis is established .Replace blood loss with normal saline, lactated Ringer solution, 5% albumin, or blood products as appropriate. Perioperative use of intravenous vasopressin for central DI is considered the treatment modality of choice,The standard vasopressin concentration is 30 milliunits/mL, infusion is started at 1 milliunit/kg/h and the rate is slowly (every 5 to 10 minutes) increased (maximum 10 milliunits /kg/h) to avoid hypertension resulting in lesser fluctuations in serum sodium as long as care is taken to limit fluid intake while under full anti-diuresis and to avoid iatrogenic water intoxication with hypotonic fluids.The goal is to decrease the urine output to less than 2 mL/kg/h.

Urine output and serum electrolytes to be closely monitored.Monitoring serial serum and urine osmolality and electrolyte levels can be helpful in differentiating the etiology of polyuria.

Administration of osmotic diuretics such as mannitol can result in a brisk diuresis and cloud the diagnosis or management of central DI.

Postoperative challenges include managing pituitary dysfunction, such as severe diabetes insipidus, get accentuated in almost 70–80% of the cases necessitating careful preoperative preparation. A multidisciplinary approach, considering both surgical and adjuvant therapies, is vital for effective craniopharyngioma management . Our patient also suffered from severe diabetes insipidus during the

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postop period, and the excessive urinary output was controlled only after the administration of oral desmopressin.

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