Case Report

Intraoperative central diabetes insipidus in a postpartum patient during decompression of base of brain lesion: Missing out the diagnosis of Sheehan's syndrome?

ABSTRACT

A 35-year-old female presented with headache in the third week postpartum period following uneventful cesarean delivery. She had left sided ptosis, pain, and numbness over left face since third trimester. Post-delivery magnetic resonance imaging revealed invading left sphenoid sinus meningioma. She was planned for combined endonasal and pterional craniotomy. Her preoperative investigations including sodium, glucose, and liver functions were normal. Intraoperatively during endonasal phase a high urine output (UO) with rising sodium were noticed which continued with worsening sodium (156 mEq/L after 3 h). Desmopressin 1 mcg IV administered which normalized UO for the rest of surgical duration with trends of declining sodium (149 mEq/L at the end of procedure). Her postoperative MRI was normal however desmopressin could not be discontinued because of increasing sodium and UO without it. She was discharged on oral desmopressin, hydrocortisone and levothyroxine. On her follow-up 3.5 months later she had normal sodium and normal UO.

Key words: Central diabetes insipidus, desmopressin, puerperium, Sheehan's syndrome

Introduction

Central diabetes insipidus (CDI) results from surgery involving hypothalamic–pituitary–axis (HPA) and can be transient or permanent. Transient condition resolves in vast majority of patients within first postoperative week and only 2.7% develop permanent CDI.[1] It almost always develops 24–48 h postoperatively.[2]

Sheehan's syndrome (SS), a known complication of pregnancy usually follows massive bleeding causing hypotension, however it may result after uncomplicated delivery and produces CDI in 5% of them.[3] Latter category of patients has mild unrecognized panhypopituitarism and when they receive glucocorticoids CDI becomes overt.[4]

We report a patient with presumably unrecognized mild SS with panhypopituitarism who presented with intracranial lesion, during postpartum period. Patient developed CDI during surgery likely from steroids administration.

Access this article online	
Website: www.saudija.org	Quick Response Code
DOI: 10.4103/sja.SJA_885_20	

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How to cite this article: Bithal PK, Jan R, Butt YM, Alshuaibi K. Intraoperative central diabetes insipidus in a postpartum patient during decompression of base of brain lesion: Missing out the diagnosis of Sheehan's syndrome? Saudi J Anaesth 2021;15:204-6.

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Submitted: 20-Aug-2019, **Revised:** 10-Sep-2020, **Accepted:** 17-Sep-2020, **Published:** 01-Apr-2021

Case Report

A 35-year-old, 79 kg female developed left sided ptosis with ipsilateral facial pain and numbness during third trimester of pregnancy but was not investigated. Following uneventful cesarean delivery in a peripheral hospital, her symptoms improved only to worsen subsequently. Her brain magnetic resonance imaging (MRI) revealed sphenoid sinus meningioma invading suprasellar, left cavernous sinus and left orbital areas. During the third postpartum week she presented to emergency department of our hospital with increasing headache. Neurosurgeon administered her dexamethasone and planned for combined endonasal and left pterional craniotomy in collaboration with ENT surgeon. Her investigations revealed normal liver functions and blood glucose, Na 140 mEq/L and K 3.6 mEq/L. Following anesthesia induction and tracheal intubation, 8 mg of dexamethasone was administered. Anesthesia was maintained with 50% oxygen and infusion of fentanyl and propofol. Intrarterial blood pressure, neurophysiology monitoring beside routine monitoring was instituted. Approximately 30 min into endonasal phase, her Na was 144 mEq/L and K was 3.7 mEq/L with normal glucose. Urine output (UO) at that time was 200 ml, which increased to 250 ml in the next hour. She was receiving 5 ml/kg/h of crystalloids. No diuretic or mannitol was administered during the procedure. Nearly 30 min before the completion of nasal procedure, Na was 146 mEq/L with 300 ml UO. ENT surgeon handed over the patient to neurosurgeon after 90 min. Serum Na kept on worsening and reached 156 mEq/L after 3 h with increasing UO. Therefore, a presumptive diagnosis of CDI was made in consultation with endocrinologist and 1 mcg of desmopressin was given IV. UO during the remaining surgical period got reduced to 70–100 ml/h with decline in Na to 149 mEq/L at the end of nine hours of procedure. Total UO was 3,000 cc (most of which was in pre-desmopressin phase). Owing to the stormy intraoperative course she was ventilated overnight in the neurocritical care unit (NCCU) and was extubated next day after her check MRI which was normal. In NCCU, discontinuation of desmopressin resulted in recurrence of polyuria, hypernatremia, and high serum osmolality (309 mOsm/kg). Desmopressin was restarted 1 mcg twice daily IV and later on changed to oral route 60 mcg morning and evening with 30 mcg in the afternoon. In addition, she was advised hydrocortisone and levothyroxine. She was discharged home after 2 weeks, with normal Na and normal UO. On her last visit to the clinic after 3.5 months, her Na and UO were controlled on desmopressin.

Discussion

High UO with increasing Na raised suspicion of CDI which was established by robust response to desmopressin. Common surgical reason for its occurrence is HPA injury and it manifests postoperatively in most patients.[2] There are report of transient CDI developing during nasal sinus surgery and during dexmedetomidine infusion, from unknown etiology.[5,6]

CDI during pregnancy and puerperium is extremely rare and can be difficult to recognize as polydipsia and polyuria are common in pregnancy. CDI can get exacerbated or become apparent during pregnancy from an accelerated catabolism of antidiuretic hormone (ADH) by placental vasopressinase enzyme. Most cases of gestational CDI are from conditions which impair the hepatic clearance of vasopressinase.[7] Even massive release of placental vasopressinase into circulation from placental abruption has also caused postpartum CDI.[8] However, CDI does not follow the placental manipulation during uncomplicated cesarean delivery. Furthermore, gestational CDI does not last beyond 6 weeks of puerperium.[9]

An uncommon complication of pregnancy is Sheehan's syndrome (SS), characterized by varying degree of anterior pituitary dysfunction. Most have mild disease which goes untreated. Though massive hemorrhage producing hypotension is the cause, it rarely presents even after uncomplicated delivery and produces CDI in 5% of them.[3] Many SS patients have impaired neurohypophyseal functions.[10] Thus, CDI may be a feature of postpartum panhypopituitarism which is infrequently recognized due to wide spectrum in severity of CDI in postpartum hypopituitarism, as well as its masking from concomitant glucocorticoids insufficiency.[11] ADH deficiency is unveiled by glucocorticoids administration from unknown mechanism.[4]

Our patient was 3 weeks into postpartum and had none of the risk factors to suggest heightened vasopressinase activity. She had uncomplicated cesarean delivery and was not given dexmedetomidine during surgery. We speculate that she probably suffered a brief period of hypotension, either during cesarean or during postpartum period, which was dismissed as insignificant but caused mild posterior pituitary damage. Administration of dexamethasone before and during surgery unmasked neurohypophysis dysfunction with resultant CDI. Other possible reason is that endonasal phase of surgery triggered transient CDI from some obscure etiology which was superimposed by trauma to HPA, converting thereby, temporary condition into permanent.[5] However, this theory looks untenable because injury to HPA does not induce CDI intraoperatively.[2] Moreover, there was no intraoperative CSF leak and most significantly postoperative MRI was normal.[12]

In conclusion, one should suspect SS of some degree causing panhyopituitarism in a postpartum patient who develops CDI

following administration of steroids during surgery and CDI in such patients is permanent.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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