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Case Report

Acute symptomatic hyponatremia following elective rhinoplasty: A case report

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ABSTRACT

We present the first reported case of symptomatic hyponatremia after elective rhinoplasty. A 42-year old female underwent cosmetic rhinoplasty without complication and was discharged home after an uneventful recovery from general anesthesia. Just prior to midnight on the day of surgery, she reported nausea, which was treated with supportive care. Four hours later, she developed emesis, altered mental status, and seizure-like activity prompting medical transport to the emergency department. Upon arrival, she was hypotensive (BP 78/54), tachycardic (HR 112 bpm), hyponatremic (116 mmol/L), hypoosmotic (239 mOsm/kg), and had decreased consciousness (GCS = 10). She was admitted to the intensive care unit and had a central line placed for hypertonic saline infusion. Urinalysis was suggestive of SIADH (UrNa 111 mmol/L, UrOsm 546 mOsm/kg) and Nephrology was consulted. Her serum sodium was corrected over three days and her mental status improved. Surgeons should maintain a low threshold for further evaluation in patients who deviate from the expected postoperative recovery pathway. This report demonstrates that normal postoperative symptoms may mask underlying physiological abnormalities

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that can progress to acute life-threatening illness and underscores the importance of direct patient observation in the immediate postoperative period.

Background

With over 218,000 procedures performed in 2017, rhinoplasty is the fourth most common aesthetic surgical procedure performed in the United States.[1] Major complications resulting in 30-day reoperation, readmission, or emergent room visits are rare and occur in less than 2% of patients[2]; however, minor complications are more common, occurring in up to 8%.[3] Patient dissatisfaction occurs in 15% and contributes to a surgical revision rate of 10–15%.[3,4] Unique complications have been reported and include visual loss, benign paroxysmal positional vertigo, pseudoaneurysm, pneumocephalus, Tapia's syndrome (unilateral recurrent laryngeal and hypoglossal nerve paralysis), and cavernous sinus thrombosis.[5]

The Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH) is reported to occur following traumatic head injury and transsphenoidal pituitary surgery, but it is not known to occur after rhinoplasty.[6,7] The purpose of this report is to increase awareness of this potential complication and to emphasize that its symptoms may be masked by normal symptoms of surgical recovery (i.e. nausea, fatigue, etc.).

Case presentation

A 42-year old female with no medical history or nasal airway complaints elected to undergo rhinoplasty for nasal reshaping. The procedure consisted of dorsal reduction, refinement of the tip, and nasal length shortening. General anesthesia was induced using sevoflurane and maintained with desflurane, two grams of cefazolin were administered for surgical site infection prophylaxis, and the patient was placed in the supine position. The surgical site was prepped with Povidone-Iodine and draped in the routine sterile fashion. A total of 8.5 mL of 2% lidocaine with 1:100,000 epinephrine were injected during the case. The procedure was performed through a stair-step transcolumellar incision followed by infracartilaginous extensions. The bony dorsum was reduced 3.5 mm using a rasp followed by bilateral medial oblique and lateral osteostomies. Tip shaping sutures were placed to refine the nasal tip and a Denver splint was applied at the end of the case. She tolerated the procedure well and without complication. She was discharged home following an uneventful recovery in the post-anesthesia care unit.

Prior to midnight on the day of the procedure, she reported nausea and fatigue. Both were initially managed with supportive care; however, her symptoms continued to worsen. Over the next four hours, family members witnessed her vomit, develop acute onset altered mental status, and convulse. Emergency medical services then transported the patient to the hospital. Upon arrival in the emergency department she was diaphoretic, tachycardic (heart rate 112 bpm), hypotensive (blood pressure 78/54) and had decreased level of consciousness (Glasgow Coma Score of 10). On exam, she was euvolemic and her pupils were reactive and dilated. An EKG and head CT were unremarkable. Initial laboratory studies were suggestive of SIADH (serum sodium of 116 mmol/L, serum osmolality of 239 mOsm/kg, urine sodium of 111 mmol/L, and urine osmolality of 546 mOsm/kg). Lactic acid was 7.1 mmol/L prior to fluid resuscitation (Table 1).

Concurrent with the above workup, she received a one-liter bolus of normal saline which lowered the lactic acid to 2.7 mmol/L. She was subsequently admitted to the intensive care unit where arterial and central venous lines were placed followed by initiation of hypertonic saline infusion. Nephrology

Table 1
Admission and discharge laboratory studies.

Laboratory study	Admission	Discharge
White blood cells	14.3	N/A
Hemoglobin	11.3	N/A
Platelets	206	N/A
Serum electrolytes		
Sodium (mmol/L)	116	137
Chloride (mmol/L)	85	100
Potassium (mmol/L)	3.6	4.0
Bicarbonate (mmol/L)	17	31
Urea nitrogen (mg/dL)	7.0	9.0
Creatinine (mg/dL)	0.60	0.55
Glucose (mg/dL)	129	102
Calcium (mg/dL)	7.7	9.0
Serum osmolality (mOsm/kg)	239	275
Urinalysis		
Sodium (mmol/L)	111	N/A
Osmolality (mOsm/kg)	546	N/A
Protein (mg/dL)	12	N/A
Creatinine (mg/dL)	49.0	N/A
Urea nitrogen (mg/dL)	469	N/A

was also consulted to assist with reversal of her hyponatremia. Over the course of the next three days, she experienced large volume diuresis, her serum sodium slowly corrected, and her symptoms resolved. She was discharged to home on hospital day three with a serum sodium of 135. She was seen for surgical follow up on post-operative day eight, and the Denver splint was removed. She had an appropriate amount of swelling at the surgical site and was not experiencing any more symptoms consistent with hyponatremia.

Discussion

Under normal physiologic conditions, antidiuretic hormone (ADH) is secreted from the posterior pituitary in response to decreased extracellular volume or increased plasma osmolality, resulting in a compensatory increase in free water resorption from the distal renal collecting ducts. SIADH occurs when ADH is continuously secreted, even in the absence of normal physiologic stimuli, thereby decreasing serum osmolality and sodium concentrations. Patients who develop SIADH may present with a spectrum of symptoms ranging from fatigue and nausea in mild cases (serum sodium >125 mmol/L) to seizures and respiratory arrest in more severe cases (serum sodium <125 mmol/L).[8] Patients who develop symptoms acutely (<48 h) are at higher risk for serious morbidity.[8] Special consideration should be given to the rate of correction as rapid increases in serum sodium can precipitate symptoms of osmotic demyelination.[8] Importantly, SIADH should be distinguished from cerebral salt wasting, as symptoms may overlap and treatments differ. Cerebral salt wasting most often presents in hypovolemic patients who require fluid and salt replacement whereas patients with SIADH require fluid restriction and typically present clinically euvolemic or hypervolemic.[9]

SIADH is a major cause of hyponatremia following traumatic brain injury and craniofacial procedures including cranial vault reconstruction, transsphenoidal pituitary gland resection, and cleft palate repair.[9,10] The mechanism by which SIADH occurs in these patients remains undefined, but an association with procedures in the midline of the skull exists. Following larger, more extensive surgical procedures, ADH may be released in response to the physiologic stress of surgery; however, patients undergoing craniofacial procedures may have a lower threshold for ADH release given the operative proximity to the pituitary gland. As our report indicates, this may also be the case for nasal reconstructive procedures. Aside from her age (>40 years), our patient had no risk factors for major complications.[2] She received minimal intravenous fluids during the case which was performed without complication.

This report underscores the importance of identifying patients who may be deviating from the expected postoperative recovery pathway and demonstrates that SIADH can occur following short outpatient aesthetic surgical procedures. Expected symptoms of postoperative recovery (i.e. nausea, headache, fatigue, etc.) may mask potentially life-threatening illness and should be followed to resolution. Critical thinking and a low threshold for evaluation may minimize or prevent serious complications following rhinoplasty.

Conclusions

Even though major complications are rare following rhinoplasty, critical thinking and early identification of patients who acutely decompensate may reduce the severity of or help avoid potentially life-threatening complications. This case report of acute symptomatic hyponatremia after elective rhinoplasty supports direct patient observation in the immediate postoperative period and serves to increase awareness about this previously unreported complication.

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Conflicts of interest

All authors have no financial conflicts of interest.

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