Iatrogenic water intoxication in healthy parturient causing convulsions and fractured mandible

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ABSTRACT

نستعرض هنا حالة كسر في الفك السفلي سني سنخي الناجمة عن نقص الصوديوم الشديد علاجي المنشأ والذي أدى إلى حدوث تشنجات شديدة في سيدة حامل تبلغ من العمر31 عاماً. أدخلت من أجل الولادة المهبلية الطبيعية. أعطيت علاج اوكسيتوسين لتسريع عملية الولادة. عانت من تشنجات حادة في فترة ما بعد الولادة الفورية التي كانت راجعة إلى نقص صوديوم الدم الحاد. تمت السيطرة على اضطراب التمثيل الغذائي وتم تثبيت الكسر وإصلاحه بنجاح. خرجت المريضة بعد 48 ساعة وكان الشفاء جيدًا.

We report a case of a mandibular dentoalveolar fracture caused by severe iatrogenic hyponatremia-induced grand mal seizure in a 31-year-old pregnant lady who underwent normal vaginal delivery. She had oxytocin augmentation of her labor, and the seizure happened in the immediate postpartum period. The seizure was thought to be because of severe hyponatremia and prompt management controlled the metabolic disorder. The fracture was reduced and fixed successfully, and she was discharged after 48 hours, healing was uneventful.

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Water intoxication in a healthy parturient is not uncommon. It can be caused by psychiatric disease,[1] or high voluntary consumption of water.[2] It can also be iatrogenically caused by electrolyte-free fluid overload and prolonged administration of high dose oxytocin.[3] It can lead to dangerous electrolyte imbalances, as had been reported in multiple case reports. Severe hyponatremia (<120 mEq/L) may lead to serious neurological complications such as grand mal tonic clonic convulsions.[3,4] In this report, we present a case of hyponatremia-induced grand mal seizure resulting from water intoxication in the immediate postpartum period, after normal delivery in a healthy parturient. Our objective in presenting this case is to highlight the dangerous airway compromise that may results from mandibular fracture under such circumstances.

Case Report. A 31-year-old, 38+ weeks pregnant lady, gravida-3, para-2, weighing 83 kg (body mass index: 29 Kg/m^2), presented in labor to the obstetric department at Jordan University Hospital, Amman, Jordan. She had spontaneous vaginal liquor discharge with irregular labor pain. Her medical, including obstetric, history was unremarkable, and her current pregnancy was uneventful. On examination she was conscious, oriented, and vital signs were within normal limits. Obstetric examination revealed cervical dilation of 2 cm with 70% effacement. Normal vaginal delivery was planned and she was admitted to the obstetric ward. Laboratory investigations showed serum sodium of 137 mEq/l, serum potassium of 3.85 mEq/l, hemoglobin of 10.6 gm/dl, and a white cell count of 8.12 x 103/ml; in addition urine analysis was unremarkable. She was kept fasting as per obstetric ward policy and started on an intravenous infusion of 5% dextrose water at

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a rate of 150 ml/hr. Augmentation of her labor was commenced by continuous infusion of oxytocin to improve her weak uterine contractions. The solution was prepared in 5% dextrose water at a concentration of 10 mU/ml then administered at a rate of 7 mU/min and increased by 0.3 mU/min every 30 minutes according to response. The infusion was titrated up and down to get a uterine contraction rate of 3-4 /10 minutes with duration of about 30-40 seconds. Labor lasted around 9 hours, and she delivered a healthy male with Apgar score of 8/9. The oxytocin infusion was stopped 30 minutes post delivery. In total, she received 2,270 ml of 5% dextrose water and 9 units of oxytocin. She was transferred to the post-partum ward later, conscious, and with stable vital signs. Soon afterwards, and witnessed by the attending nurse and her mother, she became unresponsive, and started having tonic clonic convulsions, clenching strongly on her teeth while bleeding from the mouth. Diazepam 5 mg was administered intravenously. She was turned on her side, and ventilation with 100% oxygen by Ambu bag and mask was started using the jaw thrust maneuver. When the jaw became relaxed after abortion of her convulsion, an oral airway was inserted and pharyngeal suction was carried out. Her trachea was intubated with a 7.5 mm cuffed endotracheal tube to protect her airway due to the active bleeding and decreased level of consciousness. This was facilitated by the administration of propofol 100 mg and rocuronium bromide 1mg/kg. During resuscitation, her blood pressure was 100/60 mm Hg, heart rate was 130/min, and her hemoglobin-oxygen saturation was 95%. No extra fluid management was needed. We considered eclampsia as a possible cause of the convulsions and so administered 4 gram intravenous loading dose of magnesium sulfate. A blood sample was sent for electrolyte laboratory investigation. She was then transferred to the intensive care unit in our hospital, and was sedated with a continuous infusion of propofol and remifentanil. Magnesium sulfate infusion was commenced at a rate of 1gram/hour.

In the operating room, oral and maxillofacial examination revealed a dentoalveolar fracture carrying all mandibular incisors associated with muco-gingival lacerations. Reduction and fixation of the dentoalveolar segment and fixation was achieved using the readily available Erich arch bar and interdental wiring, the lacerated mucosa was sutured with 4-0 Vicryl and the bleeding stopped. Blood results revealed serum sodium of 118 mEq/L, potassium of 3.1 mEq/L, blood sugar of 279 mg/dl, normal kidney and liver function tests, hemoglobin of 10.6 gm/dl, and white cell count of 22,600/ml. Treatment of hyponatremia with normal saline of 0.9% was started. She kept intubated and mechanically ventilated overnight. Serial measurements of serum sodium showed a gradual increase reaching 127 mEq/L after 6 hours, and 134 mEq/L 18 hours from the seizure attack. Remifentanil and propofol infusions were then stopped, and she was weaned from mechanical ventilation within 3 hours. She had an uneventful recovery without general or focal neurological deficits. Dental occlusion was satisfactory and the panoramic radiograph showed acceptable reduction (**Figure 1**). She was discharged home after 48 hours. She resumed dental follow up, and the dental splint was removed after 4 weeks. She required no further active treatment.

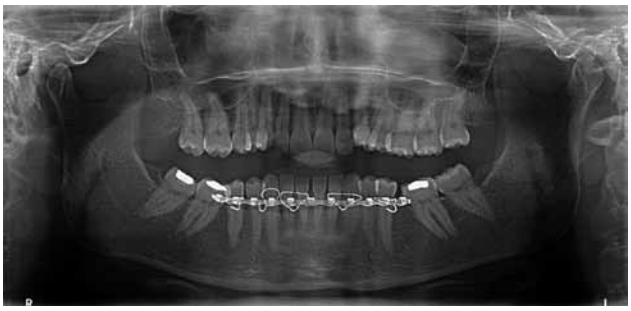

Figure 1 - A postoperative dental panoramic radiograph of the patient showing fixation of the dentoalveolar fracture.

Discussion. The likelihood of a grand mal seizure during normal labor in a known epileptic is estimated at 1.3%.[4] Convulsions in the peri-partum period can be precipitated by multiple causes, underlying convulsive disorder, eclampsia, stressful physiological changes, labor complications, coexisting diseases, and iatrogenic mishaps.[4] Water intoxication as a cause of convulsions was described in 1923 by Rowntree.[5] The cerebral hyperexcitability leading to convulsions after toxic water loads is believed to be due to the consequent dilutional hyponatremia and/or cerebral edema.[1] The toxic water load can be psychogenic with impulsive water intake,[1,6] voluntary after consumption of large amounts of water,[2] or iatrogenic.[1] Water retention during labor can be a side effect of oxytocin,[3] the main function of which is stimulation of uterine smooth muscle contraction at birth and stimulation of milk ejection. However, because of the structural similarity to antidiuretic hormone (ADH) it exerts an antidiuretic activity. This hormone was infused into this patient throughout her labor to augment her uterine contractions. The risk of hyponatremia in association with oxytocin administration is increased when oxytocin is diluted in 5% dextrose water, and when intravenous hypotonic fluids are given.[3] Tolerance to water load is diminished

during labor, and even a moderate fluid volume may cause hyponatremia; therefore, administration of hypotonic fluid should be avoided.[8] The oxytocin solution was prepared, against recommendations, in 5% Dextrose water, which is hypo-osmolar in itself and deficient in necessary electrolytes, especially sodium. The previous factors could have led to the fall in serum sodium concentration leading to convulsions in our patient.

Patients with poorly controlled epileptic seizures have an increased rate of injuries. Dentoalveolar and other maxillofacial injuries have been described during epileptic seizures,[8,9] and are commonly due to falls during the attack.[9] When such injury occurs in a bed ridden convulsing patient like our case, it could be caused by a forceful jaw thrust maneuver or a forceful insertion of oropharyngeal airway during airway management. However, the mouth bleeding that was observed before any intervention was found later to originate from the dentoalveolar trauma site. We were unable to source any reports of a similar condition under such circumstances via Medline literature search, and hope to raise awareness of this rare but potentially serious complication.

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