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Case Report

Congenital Nephrogenic Diabetes Insipidus and the Trauma Injured Brain: A Case Report

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Abstract

Diabetes insipidus is commonly attributed as post-surgical complication of neurosurgical procedures. The case presented here describes the antithesis, where a young man with nephrogenic diabetes insipidus requires emergency neurosurgical intervention. Complex treatment goals and fluid strategies are discussed.

Keywords: Case Report; Nephrogenic Diabetes Insipidus; Fluid management; Traumatic Brain Injury

Introduction

Diabetes Insipidus (DI) is not a common pathology, with an overall prevalence of 1 in 250,000. The congenital forms are even more infrequent accounting for less than 10% of these cases. In both central and nephrogenic DI patients produce large quantities of dilute urine resulting in electrolyte abnormalities. Hypernatremia and hypokalaemia being the most prominent abnormalities observed [1].

More than 20% of patients present with central DI after neurosurgical intervention [2]. The case below speaks to the inverse, where a young man with congenital nephrogenic DI presents for neurosurgery after trauma. This rare presentation juxtaposes treatment goals for the dual pathology as the fluid management of any traumatic brain injured patient is contentiously debated, compounded here by the pre-existing water and electrolyte derangement in the form of nephrogenic diabetes insipidus. The discussion to follow identifies the specific treatment goals for the perioperative management of this complex patient, furthermore, proposes a pragmatic approach to fluid and electrolyte management in this acute setting.

Case Presentation

A 13-year-old male was involved in a high velocity motor vehicle accident. Upon arrival in the emergency department, he was found to have a decreasing level of consciousness as well as features in keeping with shock, presumed at the time to be hypovolemic in nature. Immediate resuscitatory efforts were launched. Which included crystalloid and colloid resuscitation as well as the initiation of inotropy. His initial vital signs indicated Blood pressure 91/54, HR 162 bpm, Oxygen Saturation of 91% on 40% facemask oxygen at respiratory rate of 32 bpm. The young man's Glasgow coma scale (GCS) continued to fall prompting intubation and ventilation. Basic bedside imagery, primary and subsequent surveys could not account for the ongoing decreasing GCS as well as the haemodynamic instability. Urgent radiological investigation was requested revealing that our patient had a large subarachnoid hemorrhage and was in need of urgent decompressive craniectomy. At the time of obtaining consent for this emergent procedure, it was uncovered that our patient has congenital nephrogenic diabetes insipidus, managed on outpatient basis utilizing diuretics and tap water self-correction of sodium. Prior to surgery, our patient had a serum sodium of 192mmol/l and in need of ongoing fluid resuscitation.

The operative management was uncomplicated. The anaesthetist remarked a difficult resuscitation as fluid choices, electrolyte management and excessive fluid losses in the form of urine and blood proved onerous. During the 60 minute procedure the patient received 1800ml of Sodium Chloride 0.45% solution, as well as 2 units of packed red cells intravenously as well as 500ml tap water enterally. Electrolytes were checked every fifteen minutes. He did not need inotropy and mean arterial pressures were maintained at sufficient levels throughout. His urine output was 5ml/kg/hr and serum sodium at the end of the procedure was 189mmol/l. The patient was transferred to a neurosurgical intensive care unit, where he was extubated and deemed neurologically intact with GCS 15/15. He was successfully discharged from the ICU 3 days later.

Discussion

Whole body water and electrolyte balance is regulated by the antidiuretic hormone (ADH) [1]. The physiological response to ADH is to concentrate urine via the V2 receptors on the renal tubules. Nephrogenic diabetes insipidus is characterized by the body's inability to adequately respond to ADH with resultant high outputs of dilute urine. In most instances of congenital nephrogenic DI, there is abnormal gene coding for the V2 receptors, or the aquaporin channels themselves and as such, treatment with desmopressin (DDAVP) has negligible effects [4]. The urine osmolality in these patients is typically less than <200mOsm/kg with serum hypernatremia [4].

The majority of patients with nephrogenic DI are able to manage their condition themselves with the use of diuretics and oral replacement of their free water deficit [1]. However, the trauma situation often prevents this management strategy, compounded hereto significant blood and fluid losses. During the resuscitation phase the timing, dose and type of fluid has to be individualized in order to achieve haemostasis, organ perfusion and restore circulatory volume [5]. Minimizing large fluctuations in serum sodium levels is an added treatment target. Resuscitatory fluids, as well as raised

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intracranial pressure treatment aides such as mannitol and Hypertonic Saline (HTS) contain large sodium loads [2,3]. Despite this, there is insufficient evidence to make recommendations on the use of osmotic agents in TBI. Furthermore, the side effects of these drugs may have deleterious effects - especially in patients with already high urine outputs and sodium abnormalities like nephrogenic diabetes insipidus [8-12]. With desmopressin, mannitol and hypertonic saline demonstrating more harm than benefit in this condition, the treatment strategy hinges upon careful fluid management.

The complexity in the resus of a patient with inherited nephrogenic diabetes insipidus is underpinned by the paucity of evidence with which to provide recommendations of fluid management in such cases. Some authors recommend that 5% dextrose water be administered in volumes to match urine output. 0.9% normal saline, in the dose of 10ml/kg in children, is then reserved for resuscitation in the shocked patient. In theory, this appears to a viable solution. It would require frequent serum sodium monitoring to avoid the risk of rapid shifts in serum sodium should the administration of 5% dextrose water far exceed the urinary loss. For similar reasons loading with 0.9% saline may also result in rapid changed in sodium level [6].

A proposed recommendation, particularly in the resource limited setting, where resuscitation of such patients is needed is as follows:

- Admission to a unit where strict input and output can be monitored.
- Replace urine losses equally with 5% dextrose water. If patient can take free water enterally, then use the enteral route in combination with 5% dextrose water to match urine losses.
- Twice a day serum sodium levels.
- Resuscitation should be achieved using 0.45% saline and only in the shocked patient.
- Multi-disciplinary team approach-paediatrician/intensive care specialist/nephrologist to assist with the fluid management.

Conclusion

Treatment options are few in the scenario provided. Careful fluid choices, strict fluid balance and meticulous electrolyte checks are proposed as the mainstay of therapy in patients with congenital nephrogenic diabetes presenting for operative management.

Declarations

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