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Evaluation of Sodium Imbalances in Patients After Maxillofacial Trauma

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Abstract

Maxillofacial trauma patients are at risk of developing sodium imbalances due to the proximity of the maxillofacial skeleton to the brain. The force of impact from high-velocity injuries can cause concussion changes in the brain, leading to sodium imbalances that may not be easily detected on scans. Prompt monitoring and treatment of sodium imbalances are crucial, as these imbalances can have serious consequences if left untreated. This study aimed to determine the incidence of sodium imbalances in patients after maxillofacial trauma and to compare the occurrence of hyponatremia and hypernatremia in patients with or without head injury. Additionally, the study aimed to correlate sodium imbalances with factors such as age, sex, head injury, type of maxillofacial injury, and alcohol habits.

A sample of 300 patients with maxillofacial trauma above 5 years of age was evaluated, and their detailed medical history, physical examinations, and relevant blood tests were recorded. The patients were divided into groups based on the presence of head injury and sodium imbalance. The results showed that 21% of the patients had sodium imbalances, with hyponatremia being more common than hypernatremia. Patients with significant head injuries were more likely to develop sodium imbalances, with hyponatremia being an early manifestation. Alcohol consumption was also found to be associated with sodium imbalances.

The study highlights the importance of regular monitoring of serum sodium levels in patients with maxillofacial trauma, especially those with head injuries, as early detection and appropriate treatment are crucial to prevent serious neurological complications. Careful management of sodium correction is necessary to avoid adverse outcomes such as central pontine myelinolysis. Overall, this study emphasizes the need for close attention to sodium balance in maxillofacial trauma patients to ensure optimal outcomes and avoid potential complications.

Keywords: Sodium; Maxillofacial trauma; Head injury; Hyponatremia; Hypernatremia

Introduction

Maxillofacial trauma patients may develop sodium imbalances in their body. High velocity injuries are not confined to anatomical boundaries. Since maxillofacial skeleton is in close proximity to the brain, force of impact may cause concussion changes in the brain which may not be detected on scans and may cause sodium imbalances. Regular monitoring and understanding the condition with prompt treatment is extremely important as it can lead to grave consequences if goes unnoticed. Doczi [1] reported incidence of 17.33 % of patients with hyponatremia and 3.66 % of patients with hypernatremia after trauma. Stuart Lieblich [2] reported that maxillofacial trauma patient may have a severe hyponatremia secondary to the inappropriate secretion of antidiuretic hormone (SIADH) or a cerebral salt wasting disease (CSW).

Low sodium causes cellular edema. While this is tolerated by most cells in the body, it is not well tolerated within the rigid confines of the calvarium. Rapid declines in serum sodium can precipitate nausea, seizures, lethargy, headache, confusion and coma1. Rapidly accomplished corrections of serum sodium can result in pontine myelinosis or death [3]. This study was conducted to check sodium imbalance in maxillofacial trauma patient with or without head injury and the aims and objectives were as follows:

i. To find the incidence of Sodium imbalances in patients after Maxillofacial trauma

ii. To compare hyponatremia and hypernatremia in patients with Maxillofacial trauma, with or without head injury

iii. To correlate with factors of 1) age, 2) sex, 3) head injury, 4) type of maxillofacial injury 5) alcohol habits

Materials and methods

A sample size of 300 patients with maxillofacial trauma above 5 years of age were evaluated. Exclusion criteria included those with renal insufficiency, diabetes, hypertension, liver cirrhosis, patients taking diuretics or steroids, patients with abdominal or

chest trauma, those with previous treatment in other hospitals before reporting to us. The detailed history of the patient was recorded. The past medical history of the patient was recorded. Personal history and the alcohol habits of the patients were noted. General and local examinations were accurately done. Computed tomography (CT) brain and face was done (those patients who had cerebral hematoma, concussion, hemorrhage, or edema detected in the scans were grouped in the head injury category). Blood was investigated for Serum Sodium, Potassium, Chloride, Complete blood count, liver function tests, renal function tests, Random blood sugar.

Serum Sodium was measured using the Ion Selective Electrode method. For patients with sodium imbalance, additional investigations of urine sodium, thyroid stimulating hormone levels and cortisol levels were checked. Clinical signs and symptoms shown by the patient (nausea, vomiting, headache, confusion, and convulsions) were closely monitored every day in all patients till discharge. For patients with maxillofacial injury and obvious head injury, serum Sodium values were checked every alternate day and for patients with maxillofacial injury and no obvious head injury, serum Sodium were checked every third day for at-least 3 days. For patients with detected sodium imbalance, sodium levels were checked every day till normal.

a. Normal serum sodium was defined as 135-145meq/L.

b. Hyponatremia was defined as serum sodium <135meq/L.

c. Hypernatremia was defined as serum sodium > 145meq/L.

Patients were grouped as follows

Group I- Patients with maxillofacial injury and obvious head injury and normal serum sodium

Group II- Patients with maxillofacial injury without obvious head injury and normal serum sodium

Group III- Patients with sodium imbalance.

III A- Patients with maxillofacial injury and obvious head injury

i. Patients with hyponatremia with head injury

ii. Patients with hypernatremia with head injury

III B- Patients with maxillofacial injury without obvious head injury

i. Patients with hyponatremia without head injury

ii. Patients with hypernatremia without head injury.

Results

Out of 300 patients, 63 (21%) patients had sodium imbalance which we included in group III.

217 patients had only maxillofacial trauma without any head injury and 83 patients had head injury with Maxillofacial trauma.

i. Age and sex distribution:

In patients with sodium imbalances detected, maximum number (45%) were of age group 21-30 years, followed by 20.66% of age group 31-40 years. 9.6% were in the 5–20-year range, 18% in 40–60-year range and 6.6% above 60 years of age. Overall sex distribution of entire study population was 261 (87%) males and 39 (13%) females. 53 patients were male in the group of sodium imbalances (84%), the rest were female.

ii. Maxillofacial injury with obvious head injury

Of the patients who had obvious head injury (83), 46.98 % of them developed sodium imbalance. 38.55% developed hyponatremia (group III A(i)) and 8.43 % developed hypernatremia. (group III A (ii). Chi square analysis of maxillofacial injury and obvious head injury patients was carried out between the patients with sodium imbalance and without sodium imbalance. It showed that the calculated χ 2 =14.46. The critical values are 3.841 at 1% level and 6.635 at 5% level. The computed value is very much greater than the critical value at 1% level and 5% level. Thus, there is a significant sodium imbalance in patients with head injury.

iii. Maxillofacial injury with no obvious head injury

Of the patients who had only maxillofacial injury and no obvious head injury and developed sodium imbalance (group III B), 14 had soft tissue injury, 7 had mandible fracture (7) and 3 had zygomaticomaxillary complex fracture.

iv. Alcohol Habits: 27 patients had consumed alcohol before trauma of which 19 of them had sodium imbalance. By Chi Square analysis, χ 2 =4.48. The critical values are 3.841 at 1% level and 6.635 at 5% level. The computed value is greater than the critical value at 1% level and less than 5% level. There is a relationship between alcohol consumption and Sodium imbalance.

Discussion

Sodium is the major extracellular cation in the body and is therefore one of the most important osmotically active solutes. It ensures a proper fluid and electrolyte or pH balance in our body, together with chlorine and potassium. Serum sodium concentration is regulated by stimulation of thirst, secretion of ADH, feedback mechanisms of the renin-angiotensin-aldosterone system, and variations in renal handling of filtered sodium [4]. Hyponatremia indicates a state of extracellular hypo-osmolarity and a tendency for free water to shift from the vascular space to the intracellular space. Although cellular edema is well tolerated by most tissues, brain is constrained inside a non-extensible envelope; thus, brain swelling carries a significant morbidity because of the compression of brain parenchyma over the rigid

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skull. Therefore, clinical manifestations of hyponatremia are related primarily to cerebral edema [5].

Patients after maxillofacial trauma need to be monitored regularly for serum sodium levels. Sodium level can change due to incipient head injury either cerebral salt wasting disease or syndrome of inappropriate antidiuretic hormone secretion which can lead to disastrous complications if not watched for and timely corrected. Signs and symptoms of hyponatremia include lethargy, nausea, vomiting, anorexia, irritability, headache, muscle weakness and in severe cases, hyporeflexia, drowsiness/confusion, seizures, and coma. Signs and symptoms of hypernatremia include lethargy, thirst, irritability, restlessness, cramps and in severe cases hyperreflexia, ataxia, seizures, and coma. Prompt treatment is indicated in the presence of acute symptomatic hyponatremia to minimize the risk of significant neurological complications and adverse outcome, including an increased risk of death. However, the correction of hyponatremia can itself lead to neurological sequelae, particularly central pontine myelinolysis [6], and these risks should be minimized by gradual correction of sodium deficits. In most circumstances, serum sodium should be increased by no more than 0.5 mmol /litre/h or 8–10 mmol/ litre /day.

A total of 300 patients were observed admitted in our hospital with a history of maxillofacial trauma (hard or soft tissue), with or without head injury. In the 300 patients 217 had only maxillofacial trauma and 83 had head injury with Maxillofacial trauma. Totally, 63 (21%) patients had sodium imbalance. Doczi [1] reported 17.33 % of patients had hyponatremia after trauma. 3.66 % of patients had hypernatremia after trauma. Of the patients who had head injury, 46.98 % of them developed sodium imbalance.38.55% developed hyponatremia and 8.43 % developed hypernatremia.

Of the patients who had only maxillofacial trauma, 11% developed sodium imbalance, 9.21% developed hyponatremia and only 1.84 % had hypernatremia.

Age

In the present study, out of 300 patients, most patients were in the age group of 21-30 years, followed in frequency by 31-40 years. This implies that road traffic accidents, assaults and falls are more common in this age group. Sodium imbalance was also common in patients in the age group of 21-30 years in both the group of patients having maxillofacial trauma with or without head injury.

7.2. Sex

In the present study, out of 300 patients, 261 were males and 39 were females. This may be as males encounter more falls or assaults or accidents than females. Thus, in this study, males developed imbalances post trauma more commonly. According to this study, patients with significant head injury are prone to develop sodium imbalance. And hyponatremia is more common than hypernatremia. It was also noted that hyponatremia manifested early, on the first day of admission except in one case when it developed on the 6th day in a patient with only maxillofacial trauma. Hypernatremia developed on the first day except in 3 patients with head injury on 3rd, 5th, and 7th day respectively. Clinical signs of hyponatremia ideally developed below 125, which appeared in only 4 patients.

Hypernatremia was rare, resolved within 2 days. Also, there was a greater incidence in those patients who had concomitant head injury, reason being thirst response is active in most patients might be deranged in patients with head injury to varying levels and so patients do not generally develop hypernatremia. According to Adiga [7] et al., in patients with head injury, 64% had hyponatremia and 4% had hypernatremia. Dong Ki Kim [8], reports that Hyponatremia is the most common and important electrolyte disorder affecting patients with head injury. Cerdà-Esteve [9] reported hyponatremia is common in head injury patients. Umberto et al. [10] reported an incidence of hypernatremia in 51.5% of the patients after a history of head injury.

1.3. Maxillofacial injury with no obvious head injury

In the present study, by analysis, no specific maxillofacial injury be it soft tissue or mandible or mid face fractures could be attributed to be more likely to develop sodium imbalance. All patients need to be investigated for serum sodium for all patients irrespective of perceivable injury. Thus, it can be concluded that mild concussion injury might not be detected perceivably as head injuries but, as maxillofacial region is very close to the brain, there might be subtle injury which is of serious concern and major consequence if overlooked. Robert Steelman [11] reported a case of a 9-year-old boy, with history of fall from bike facial lacerations but no signs on CT to develop hyponatremia. It was diagnosed as cerebral salt wasting disease and treated accordingly.

Alcohol habits

In the present study, by chi square analysis, there is a relationship between alcohol consumption and Na imbalance Steven M Joyce [12]. Reported a case of beer potomania, a rare disorder in which dietary sodium and protein insufficiency lead to dilutional hyponatremia. Fenves AZ, Thomas S [13] reported two cases of beer potomania. Alcoholic patients might develop hyponatremia. This is due to inability to excrete free water. Thus, protein rich diet must be given to these patients. The two main causes of hyponatremia in patients with head injury might be SIADH or hyponatremia. The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) was originally described by Schwartz [14] in 1957. Because of the excess secretion of antidiuretic hormone, ingestion of water is not followed by its excretion, and the patient develops water retention state, a

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dilution of sodium and subsequent hyponatremia [15]. Treatment of SIADH focuses on water restriction [16].

Evidence has arisen that cerebral salt wasting (CSW) syndrome is also a cause of hyponatremia. It was first described by Peters and colleagues [17] in 1950. CSW is a disorder that can cause the kidneys to be unable to conserve salt. CSW is believed to be caused by renal salt wasting. A decrease in fluid volume and salt balance set these two processes apart [18]. Signs of clinical dehydration, weight loss, orthostatic hypotension, and a negative water balance are key to the diagnosis of CSW [19,20]. A water input to output ratio of less than one represents a negative water balance. Elevations in serum blood urea nitrogen, serum potassium, and protein concentration serve as additional support for a diagnosis of CSW [21]. Serum uric acid is usually decreased in SIADH and likely normal in CSW 6. According to Doczi et al. [1] eighty-four patients (4.6%) developed the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). Wenchuan Zhang [22] reported SIADH being the common cause of hyponatremia in head injury patients. Hegde [23] reported a case of cerebral salt wasting disease after subarachnoid hemorrhage. Paul Vespa [24] reported cerebral salt wasting as a common cause of hyponatremia in head injury patients. Daniel C Lu [25] reported 2 cases of cerebral salt wasting in patients after head injury.

According to Robert Steelman11, early onset of hyponatremia of less than 3 days has been associated with SIADH whereas a later onset (3 weeks) has been associated with CSW. Robert Steelman11 reported urine output needs to be closely monitored in conjunction with serum sodium concentrations. If serum sodium is less than or equal to 125 mmol/liter, then 3% NaCl should be administered to increase the sodium level to 130 mmol/liter. Attention to volume status also needs to be addressed. Meticulous calculation of fluid status and daily weights should be done. Generally, with an adequate urine output, and low serum sodium, CSW may be presumed to be the clinical problem rather than SIADH. In the later stages of SIADH, especially with fluid restriction, urinary output will increase with a concurrent increase in serum sodium. Cerebral salt wasting, however, results in low serum sodium despite an adequate urine output.

Also, patients do not develop neurologic manifestations until the serum sodium drops below 125meq/l. So, to prevent this from occurring, we need to keep monitoring on a regular basis and correct immediately as and when required. If the patient is hypervolemic, or output is less and has hyponatremia, correct with fluid restriction. If euvolemic, treat with isotonic saline. If hypovolemic, appropriate fluids must be resuscitated. Hypernatremia patients must have more water intake and salt restricted diet. Importantly, Sodium correction must be gradual. Based on the clinical signs, investigations, and input output status, hyponatremic cases were managed by 3% hypertonic saline to increase sodium to 125meq/L. Further management was done according to etiology. For hypervolemic cases or patients with low output, fluid restriction was done. For dehydrated patients, isotonic saline was given. Hypernatremia was managed by increasing the water intake.

Conclusion

There was a significant incidence of sodium imbalance in patients with maxillofacial trauma. It was more common in male patients between 21-30 years of age. Patients with noticeable head injury on scans were more prone to develop imbalance. All patients of maxillofacial trauma should be closely monitored as they may develop sodium imbalances. Alcoholic patients have a greater tendency to develop imbalance.

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