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Short Note

A Narrative Review on Neuroendocrine Abnormalities in Traumatic Brain Injury: The Dread of Hyponatremia

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Abstract: Head trauma or any traumatic injury can cause damage to parts of the brain. The most important part regulating the body are the hormones and the two centers of brain which are main ones for this regulation are the most prone to getting injured. The hypothalamus and pituitary regulate a lot many functions and the loss of these organs can cause Adrenal Insufficiency, SIADH, Diabetes Insipidus, and many more.

Keywords: head trauma; neuroendocrine; hormones; hypothalamus; pituitary

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Introduction:

Traumatic Brain Injury holds the potential to cause several Neuroendocrine abnormalities due to the peculiar location of two main endocrine structures i.e., the Hypothalamus and the Pituitary gland[1], [2].

This article mainly focuses on the hyponatremia caused as a consequence of neuroendocrine abnormalities due to traumatic brain injury. Four main conditions causing hyponatremia due to traumatic brain injury are discussed in this text[3–5].

1) ADRENAL INSUFFICIENCY:

Signs and symptoms include anorexia, nausea, vomiting, fatigue, postural dizziness, abdominal pain, limb and back pain, and impaired consciousness. The biochemical abnormalities include hyponatremia, hyperkalaemia, and hypoglycemia[6].

2) DIABETES INSIPIDUS:

It is associated with fractures near Sella Tursica, which may damage the stalk of the pituitary gland, disrupting ADH secretion from the posterior pituitary gland. It is characterized by passage of large volume (>3 liters/24 hours) or 40 mL/kg/24 hrs, urine osmolality <300 mOsmol/kg[7].

3) SIADH

Head trauma can cause damage to the posterior pituitary which can cause inappropriately increased secretion of ADH. It is characterized by hyponatremia and serum hypoosmolality along with a continuous excretion of sodium[8].

4) CEREBRAL SALT WASTING SYNDROME:

This is an acute clinical manifestation that happens often in traumatic head injuries—abnormalities in renal sodium transport cause extracellular water depletion in patients with intracranial disease or injury. Hyponatremia is a main characteristic feature of this condition[9].

The clinical conditions causing hyponatremia in Traumatic Brain Injury are chosen because this can lead to a rapid deterioration of consciousness and may even lead to seizure, coma, and death. Clinicians must be aware that Traumatic Brain injury can cause neuroendocrine manifestations[10].

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Detailed Analysis:

Traumatic Brain injury is caused when something hits the head with a force strong enough to damage the internal structures, when the head hits recurrently, or when an entity enters the skull and into the brain. This has the potential to cause hormonal problems soon after or months/years later as the two most important structures of the endocrine system i.e., the pituitary gland and hypothalamus are situated near the brain[11].

This article highlights the significance of being aware of the endocrine consequences after a Traumatic brain injury.

The hypothalamus and Pituitary glands constitute the central neuroendocrine system. The hypothalamus is a part of the forebrain which lies below the thalamus. Its function is to synchronize the interconnections of the neuroendocrine system. The pituitary gland lies in the Sella Tursica. The anterior lobe of the Pituitary is called Adenohypophysis. It stores hormones and releases them which is mainly mediated by the Hypothalamus. These hormones are as follows: follicle-stimulating hormone (FSH), luteinizing hormone (LH), adrenocorticotropic hormone (ACTH), thyroid-stimulating hormone (TSH), prolactin, and growth hormone (GH). The posterior lobe is called Neurohypophysis, which is responsible for storing and releasing Oxytocin and Antidiuretic hormone. The hypothalamus synthesizes these 2 hormones. They traverse through a pituitary stalk to the posterior pituitary, where they finally get released. This gives a basic picture of how damage to these sites due to Brain trauma can cause major Neuroendocrine abnormalities[12].

This article mainly focuses on the hyponatremia caused as a consequence of neuroendocrine abnormalities due to traumatic brain injury. Hyponatremia leads to a rapid deterioration of consciousness which can progress to seizure, coma, and death eventually[13].

ADRENAL INSUFFICIENCY:

In an article published in 1855, Thomas Addison described a group of patients suffering from a constellation of symptoms, including "general languor and debility, remarkable feebleness of the heart's action, and a peculiar change in the color of the skin, associated with disease of the suprarenal capsules, or in modern parlance, the Adrenal glands." [14]

SIGNS AND SYMPTOMS: Adrenal insufficiency results in anorexia, nausea, vomiting, fatigue, postural dizziness, abdominal pain, limb and back pain, and impaired consciousness. The biochemical abnormalities include hyponatremia, hyperkalemia, and hypoglycemia.

INVESTIGATION: The cosyntropin test is a safe and definitive method of diagnosing this condition.

TREATMENT: This condition is treated by administration of exogenous glucocorticoid or cortisol at a dose that is sufficient enough to meet the daily physiological needs of the body. 15-20 mg hydrocortisone in 2 to 3 divided doses daily is the standard protocol.[15], [16]

DIABETES INSIPIDUS:

Traumatic Brain Injury causes Central Diabetes Insipidus. It is associated with fractures near Sella Tursica, which may damage the stalk of pituitary gland, disrupting ADH secretion from the posterior pituitary gland. It is characterised by the passage of large volume (>3 litres/24 hours) or 40 mL/kg/24 hrs, urine osmolality <300 mOsmol/kg.

SIGNS AND SYMPTOMS: Patients present with sudden onset of polyuria, polydipsia, and nocturia. Children may develop enuresis. Hypernatremia occurs if fluid intake is not adequate. In traumatic DI, the triphasic response may occur. Initial polyuria, prolonged antidiuresis and final polyuria.

INVESTIGATIONS:

Fluid deprivation/desmopressin test

Patient preparation consists of allowing the patient to consume normal fluids overnight, avoid caffeine and smoking, and weigh the patient.

The next steps are as follows:

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1) Obtain blood and urine for osmolality measurements and urine volume at 8 am
2) Restrict fluid for up to 8 hours
3) Weigh patient every 2 hrs
4) Obtain blood and urine for osmolality and urine volume
5) Stop the test if wt. loss > 5% of starting wt. or intolerable thirst
6) Inject desmopressin 2 µg IM.
7) The patient is allowed to eat and drink up to 2 × volume passed during the deprivation phase.
8) Obtain blood and urine for osmolality and urine volume at 8 pm and 8 am the next day.

RESULTS OF THIS TEST:

1) After fluid deprivation, if the urine osmolality is <300 mOsmol/kg, and after administering desmopressin, the urine osmolality is >750 mOsmol/kg, then the diagnosis is Central Diabetes Insipidus
2) After fluid deprivation, if the urine osmolality is <300 mOsmol/kg, and after administering desmopressin, it is still <300 mOsmol/kg, then the diagnosis is Nephrogenic Diabetes Insipidus.
3) After fluid deprivation, if the urine osmolality is >750 mOsmol/kg, and after administering desmopressin, it is still >750 mOsmol/kg, then the diagnosis is primary polydipsia.

TREATMENT: Desmopressin is the drug of choice. Daily dose – Oral 50–1200 µg in divided doses, intranasal 5–40 µg by spray in one to three doses, parenteral 1–2 µg intramuscular. Desmopressin in excess over a prolonged period causes hyponatremia.[17–20]

SIADH

It is the most common neuroendocrine disorder noticed in patients with traumatic brain injury. It is characterized by hyponatremia and serum hypoosmolality along with a continuous excretion of sodium. Head trauma can cause damage to the posterior pituitary which can cause inappropriately increased secretion of ADH.

DIAGNOSTIC CRITERIA:

1) Dilutional hyponatremia (plasma osmolality appropriately low compared to plasma sodium).
2) Urine osmolality > plasma osmolality.
3) Persistent renal sodium excretion.
4) Absence of hypotension, hypovolaemia, and edema-forming states.
5) Normal renal and adrenal function.

A mainstay of treatment in most cases is fluid restriction to about 1.0 to 1.2 L per 24 hours with daily serum sodium monitoring. Strictly monitoring fluid inputs and outputs helps to ensure fluid intake is less than the combined loss through urine output and insensible loss.[8]

CEREBRAL SALT WASTING SYNDROME:

Abnormalities in renal sodium transport cause extracellular water depletion in patients with intracranial disease or injury. This is an acute clinical manifestation that happens often in traumatic head injury. Hyponatremia is a main characteristic feature of this condition.

SIGNS AND SYMPTOMS: lethargy, agitation, headache, altered consciousness, seizures, and coma. The severity of symptoms typically reflects the magnitude and rapidity of the decrease in serum sodium concentration.

INVESTIGATION: the laboratory finding includes elevated urinary sodium concentrations and low serum osmolality.

TREATMENT: Management of Cerebral salt wasting is focused on the correction of intravascular volume depletion and hyponatremia and on the replacement of ongoing urinary sodium loss, usually with intravenous (IV) hypertonic saline solutions.[21]

CONCLUSION:

The clinical conditions causing hyponatremia in Traumatic Brain Injury are chosen because this can lead to a rapid deterioration of consciousness and even may lead to seizure, coma, and death.

Hyponatremia leads to cellular swelling, a result of water movement down the osmotic gradient from the hypotonic ECF to the ICF. The symptoms of hyponatremia are primarily neurologic, reflecting the development of cerebral edema within a rigid skull. Acute hyponatremic encephalopathy ensues when these volume regulatory mechanisms are overwhelmed by a rapid decrease in tonicity, resulting in acute cerebral edema. Early symptoms can include nausea, headache, and vomiting. However, severe complications can rapidly evolve, including seizure activity, brainstem herniation, coma, and death. A key complication of acute hyponatremia is normocapnic or hypercapnic respiratory failure; the associated hypoxia may amplitude the neurologic injury.

Clinicians must be aware that Traumatic Brain injury can cause neuroendocrine manifestations.

Ethical Statement: Being a Short note, there were no ethical issues and IRB permission is not required.

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Conflicts of interest: The authors declare no conflict of interest.

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