ORIGINAL RESEARCH PAPER

Neurosurgery

TRIPLE PHASE RESPONSE IN TRAUMATIC BRAIN INJURY-A RARE PRESENTATION

KEY WORDS:

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INTRODUCTION

Triple phase response in trauma suggests a pituitary stalk injury encompasses an initial phase of Diabetic inspidus followed by ADH mediated Hyponatremia and a subsequent phase of chronic Diabetes inspidus

Here I am presenting the classic triphasic response post traumatic injury

Case Description

A 23 years male ,resident of Chhattisgarh state came to our emergency department after a road traffic accident due to fall from bike.He sustained frontal bone depressed fracture with contusion for which he was operated and multiple faciomaxillary injuries bilaterally. He had initial phase of hypernatremia 171 meq/l

Serum osmolality 356 mmol/kg,urine 220mmol/kg .Patient improved with hypotonic fluid administration and desmopressin therapy no signs of pituitary injury on Ct

On day 7,he developed hyponatremia with sodium 124 meq/l and osmolality 286 ,urine osmolality 780 suggesting Adh mediated hyponatremia managed with fluid restriction aggressively after one week polyuria developed treated by desmopressin.

Following desmopressin administration Patient recovered well and discharged after electrolyte correction .

DISCUSSION

Diabetes insipidus (DI) is a rare disorder of water homeostasis characterized by the excretion of abnormally large volumes of hypotonic urine. Over 90% of the vasopressinergic neurons which project from the supraoptic and paraventricular nuclei to terminate in the posterior pituitary must be destroyed in order to produce AVP deficiency sufficient to cause polyuric symptoms. Central DI (CDI) therefore always represents significant hypothalamo-neurohypophysial dysfunction.

History and clinical examination occasionally yield useful diagnostic clues. The first step in diagnosis is to confirm the presence of hypotonic polyuria. Approximately 15% of patients referred for investigation of polyuria have normal urine volume, but present with urinary frequency, due to infection, prostatism or irritable bladder. In an adult patient, if the daily urine volume is <2.5 L, no further investigations of osmoregulatory function are required, he most commonly used test for DI is the two-step water deprivation test. The first step is an 8-hour period of water deprivation; the physiological basis for this step is that dehydration stimulates the secretion of vasopressin, with consequent reduction in free water clearance. Urine volumes fall and urine osmolality should rise to over 750 mOsm/kg, provided that a sufficient osmotic stimulus to AVP secretion (plasma osmolality > 295 mosm/kg) is attained at the conclusion of dehydration. This step should differentiate patients with primary polydipsia, in whom vasopressin secretion and function are normal, from patients with either central and nephrogenic diabetes insipidus, who do not concentrate urine at the end of dehydration.

The second part of the test is designed to differentiate central diabetes insipidus from nephrogenic diabetes insipidus. Desmopressin (dDAVP), an AVP analogue, is administered by intramuscular or subcutaneous injection, and the urine osmolality response is measured. As patients with central DI are simply missing AVP, they respond to exogenous desmopressin with a rise in urine osmolality to over 750 mOsm/kg. Patients with nephrogenic DI are resistant to the antidiuretic effects of desmopressin and do not respond with urinary concentration

Once a diagnosis of CDI is made, MRI imaging of the hypothalamo-pituitary region is required to establish whether there is a structural lesion which is responsible for AVP deficiency. It is important to note that lesions such as histiocytosis and germinoma may not manifest on the initial scans; normal imaging should be repeated within 6 months. Treatment of diabetes insipidus involves replacement of the free water deficit, replacement of the deficient hormone (in the case of CDI), and treatment of the underlying condition

Acute onset of CDI usually occurs in the neurosurgical setting, 2-3 days following hypothalamic/pituitary insult. Hypernatraemia can ensue if the patient has impaired cognition, absent thirst or cannot freely access water because of immobility. Urinary water loss should be clamped by administrating desmopressin (dDAVP), and water deficit should be replaced with hypotonic fluids. Acute CDI is usually transient and often responds to a single dose of parenteral dDAVP. Further doses are administered only if polyuria recurs. Rarely, a triphasic response can occur, in which initial DI is followed by an antidiuresis and hyponatraemia, with a subsequent return to permanent DI. For this reason, and due to the fact that CDI is often transient, regular dDAVP is only prescribed if polyuria persists beyond 48 hours, with ongoing monitoring of pNa and urine volume advised. Withdrawal of dDAVP prior to discharge from hospital is useful in identifying recovery of endogenous AVP secretion. Patients who are discharged on dDAVP should be advised about the potential risk of the triphasic response, and the need for urgent blood draw for pNa measurement should they develop symptoms suggestive of hyponatraemia, such as headache and bloating.

Long-term treatment with desmopressin (dDAVP) can control the thirst and polyuria of CDI and, in the absence of other pituitary hormone deficiencies, can allow the patient a normal quality of life. The dose of dDAVP required is proportional to the degree of AVP deficiency. In patients with mild CDI, a starting dose of 100-200 g orally at night is often sufficient to control nocturia. The dose may be titrated until symptom control is achieved; severe disease may require up to 200 g two to three times daily.

Hyponatraemia is a relatively common complication of dDAVP treatment of CDI. In normal physiology, water drinking suppresses endogenous AVP to allow excretion of excess free water and maintenance of eunatraemia. However, pharmacological vasopressin analogues are non-suppressible. Water intake which is in excess of physiological needs cannot be lost through the kidneys and accumulates, causing dilutional hyponatraemia