CASE REPORT PROF-1395

HEAT STROKE WITH DIABETES INSIPIDUS;
A CASE REPORT

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CASE REPORT

A young soldier 20 years of age was brought with 02 hour history of sudden loss of consciousness associated with high grade fever while working in the field for several hours in hot and humid weather in month of August. There was no history of head trauma.

General physical examination revealed an average built man deeply comatose having GCS 5/15. His BP was 140/100, pulse 120 beats/min, axillary temperature 106ºF and respiratory rate 40 breaths /min. His skin was very hot and dry showing severe degree of dehydration. Examination of nervous system revealed nonreactive dilated pupils, with absent deep tendon reflexes. Planters were equivocal bilaterally . Fundi oculi were normal. Neck was supple. Examination of respiratory, gastrointestinal and circulatory system was normal .

Provisional diagnosis of "heat stroke" was made however possibility of acute pyogenic meningitis , cerebral malaria and acute viral encephalitis were also kept in mind. Tepid sponging with ice cold water was started immediately to lower the body temperature. Intravenous line was established and IV fluids given to him. Oxygen inhalation was started. He was given IV paracetamol, dexamethasone, quinine, and third generation cephalosporin. Later he started having recurrent generalized tonic clonic fits He was then shifted to Intensive care unit where he was put on ventilatory support, catheterized and nasogastric tube passed. He was also given intravenous Acyclovir and tablet sodium valproate through nasogastric tube.

Blood complete count showed haemoglobin 14g/dl, total leucocyte count $7.2\times10^3/\mu l$ with 70 % neutrophils and platelets $180\times10^3/\mu l$. Blood film was negative for malarial parasite. His coagglution profile was normal. ECG showed sinus tachycardia. His cerebrospinal fluid (CSF) examination revealed increased CSF pressure, having proteins 33mg/dl , glucose 62mg/dl and normal cell count. No AFB was seen in CSF. Serum urea was 70 mg/dl, creatnine 1.2 mg/dl ,sodium 149 mmol/dl and potassium 4.0 mmol/dl. Rest of the base line investigations were with in normal limits. His CT scan brain was also done which revealed normal study. Next day his fits were controlled so he was weaned off ventilatory support.

During the next three days, there was no apparent change in his condition but he developed polyuria.

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Following day he started responding to painful stimuli, pupils were also showing sluggish response. On 5th day he started opening and blinking his eyes, started responding to verbal command. However polyuria persisted with daily urine output ranging between 6000 to 11000ml. Intravenous fuids were given to replace fluid losses. One week after admission he became fully conscious, responding to verbal command but his speech was incoherent. Serum sodium was144µmol/L.

Plasma osmolality was high (298 Osm/kg) and urine osmolality was low (180 Osm/kg). To confirm diagnosis of central diabetes insipidus he was subjected to water deprivation test. Result of water deprivation test was consistent with diagnosis of central diabetes insipidus. After water deprivation his plasma osmolality rose to 310 m Osm/kg however urinary osmolality was low (240 m Osm/kg). His urinary osmolality increased by more than 50 % after DDAVP was given. He was started on oral desmopressin 200 µg bid to which he responded well and urine output became normal. Two weeks after admission his condition was much improved, he started speaking single words and orientation also improved. Later he was discharged and is being followed up in outdoor and is asymptomatic presently on treatment. His urine out put and serum osmolality are presently within normal limit.

DISCUSSION

Heat stroke is defined as a core body temperature in excess of 40.5ºC (105ºF) with associated central nervous system dysfunction in the setting of a large environmental heat load that cannot be dissipated[1]. Exertional heat stroke generally occurs in young, otherwise healthy individuals who engage in heavy exercise during periods of high ambient temperature and humidity[2]. Typical patients are athletes and military recruits in basic training[3]. Our patient was also a recruit undergoing physical activity in hot weather.

Complications of heat stroke including acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation, renal or hepatic failure, hypoglycemia, rhabdomyolysis, and seizures have been reported in literature[4]. Although diabetes insipidus(DI) has not been documented as complication of Heat stroke in literature, it can be associated with any condition causing hypothalamic damage.

Central diabetes insipidus (CDI), is associated with deficient secretion of antidiuretic hormone (ADH). Central diabetes insipidus is characterized by decreased release of antidiuretic hormone, resulting in a variable degree of polyuria. Lack of ADH can be caused by disorders that act at one or more of the sites involved in ADH secretion: the hypothalamic osmoreceptors; the supraoptic or paraventricular nuclei; or the superior portion of the supraopticohypophyseal tract[5]. The most common causes of central DI accounting for the vast majority of cases, are neurosurgery or trauma, hypoxic or ischemic encephalopathy , primary or secondary tumors or infiltrative diseases (such as Langerh ans cell histiocytosis), and idiopathic DI(possibly due to autoimmune injury to the ADH-producing cells)[6]. A case of transient diabetes insipidus has been reported following cardiopulmonary bypass[7]. Our patient developed diabetes insipidus most likely due to damage to hypothamalus due to severe hyperthermia. Approximately 30 to 50 percent of cases of CDI are idiopathic, being associated with destruction of the hormone-secreting cells in the hypothalamic nuclei. It has been suggested that an autoimmune process is involved in many, if not most, patients[8].

In a polyuric patient a high-normal plasma sodium concentration (greater than 142 meq/L, due to water loss) points toward DI[9] .The diagnosis of diabetes insipidus is confirmed in the face of elevated plasma osmolality (i.e >300mOsm/kg) and a low urine osmolality (i.e <600 Osm/kg). It is further confirmed by water deprivation test.

Since the primary problem in central DI is deficient secretion of ADH, control of the polyuria can be achieved by hormone replacement. In the past, this was achieved by intramuscular injections of vasopressin (Pitressin) tannate in oil, which is no longer available. Intramuscular vasopressin has been replaced by desmopressin, a two-amino acid substitute of ADH that has potent antidiuretic but no vasopressor activity[10]. Desmopressin can be given intranasally or orally[11], however absorption of

desmopressin in normal persons is decreased by 40 to 50 percent when taken with meals[12]. Oral Desmopressin has been successfully used in treatment of diabetes insipidus[13]. Our patient also responded well to oral desmopressin and his symptoms improved with treatment.

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