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AN EXCEPTIONAL CASE OF SEIZURE
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Abstract :
Primary polydipsia refers to a condition of inappropriate ingestion of excess water. The excess water ingested leads to suppression of posterior pituitary hormone, arginine vasopressin (AVP) resulting in polyuria. Excess water ingested also leads to extracellular fluid dilution resulting in electrolyte abnormalities. Psychogenic polydipsia is a subset of primary polydipsia, where the excess water intake is secondary to certain psychiatric disorders. We present a case of psychogenic polydipsia which presented initially with seizure and on further evaluation found to have this disorder.

Keyword : psychogenic polydipsia, polyuria, seizure, vasopressin.

A 27 year old unmarried male came to the medical casualty with complaints of lethargy and giddiness of two days duration. It was not associated with head ache, vomiting, fever, weakness of limbs, visual disturbances, ear discharge, hard of hearing, swallowing difficulties or tremors. Patient was diagnosed as a case of seizure disorder elsewhere few months back. History was suggestive of Generalised Tonic Clonic Seizures. Computed Tomography (CT) brain taken showed a focal calcification in the right frontal lobe with perilesional edema suggestive of granuloma. Patient was advised antiepileptics and was on tablet phenytoin 100mgBD. While on treatment, patient developed seizures again. Patient then discontinued the treatment . Patient was not a known diabetic, hypertensive, asthmatic or a case of cardiac disease. There was no history of any chronic drug intake, alcoholism, Intravenous drug abuse, tuberculosis, renal or liver disease. On examination, his blood pressure was 120/80mmHg, pulse rate 86/min with normal volume, respiratory rate was 14/min, temperature was 98°F.

Further probing in to the history revealed that the patient was apparently normal two yrs back. The patient had gradually started to withdraw himself from his family and friends for the past one year. He also quit his job and started to get into spiritual ideas. His food habits changed and he became a vegetarian. He believed that he was losing energy and started to consume lots of water to restore it back. There was increased frequency and of micturition with increase in volume of urine. He also started complaining of giddiness and lethargy on and off.

As the patient had polyuria and polydipsia, input output chart was maintained.

<table>
<thead>
<tr>
<th>Day</th>
<th>Input</th>
<th>Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>15 litres</td>
<td>14 litres</td>
</tr>
<tr>
<td>Day 2</td>
<td>13.5 litres</td>
<td>12 litres</td>
</tr>
</tbody>
</table>

Investigations revealed normal blood sugar, urea, creatinine. Ultrasonogram of abdomen, Electrocardiogram and Chest X ray taken at admission were normal. Mantoux was showing 6mm induration. Thyroid function test was normal. Serum electrolyte screening showed the following values.

- **Serum sodium-** 127 meq/L (136-146 meq/L)
- **Serum potassium-** 3.8 meq/L (3.5-5 meq/L)
- **Serum chloride-** 97 meq/L (101-109 meq/L)
- **Serum calcium-** 9.2 mg/dl (8.7-10.2 mg/dl)
- **Serum magnesium-** 1.8 meq/dl (1.5-2.3 mg/dl)
- **Serum osmolarity-** 150 mosm (285-295 mosm)
- urine osmolarity - 56 mosm (300-900 mosm)

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Electroencephalogram was normal. Magnetic Resonance Imaging (MRI) of the brain taken showed presence normal bright spot of posterior pituitary. No mass lesion was detected . As the initial urine osmolarity was very low, water deprivation test was performed.

Water deprivation test showed that the patient was able to adequately concentrate urine and raise serum sodium values without the administration of desmopressin.

<table>
<thead>
<tr>
<th>Time</th>
<th>Body weight (kg)</th>
<th>Urine output(mL)</th>
<th>Serum sodium (meq/L)</th>
<th>Serum osmolarity (mosm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7 am-9 am</td>
<td>58 kg</td>
<td>1.50</td>
<td>127</td>
<td>80</td>
</tr>
<tr>
<td>9 am-12 pm</td>
<td>55.7 kg</td>
<td>0.75</td>
<td>138</td>
<td>345</td>
</tr>
<tr>
<td>12 pm-3 pm</td>
<td>55.5 kg</td>
<td>0.50</td>
<td>141</td>
<td>320</td>
</tr>
</tbody>
</table>

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Differential diagnosis for polyuria and polydipsia include Diabetes mellitus, increased sodium loss, urea, mannitol use (increased renal water loss from excessive solute), Diabetes insipides both central and renal (increased water permeability from low secretion or impaired response to ADH), neurological or endocrine problems (cushing’s syndrome, glucocorticoids, CNS sarcoidosis).

The gold standard for diagnosis is the water deprivation test. The urine osmolarity is low (<100 mosm) prior to the test in PPD also the plasma ADH[15]. In diabetes insipides, the urine will be dilute before the start of the test and the ADH levels depend on whether the defect is central (low) or renal (high). Central diabetes can appear like PPD prior to water deprivation test (low ADH and dilute urine). After water restriction test, PPD alone shows remarkable rise in urine osmolarity and serum ADH levels. In Diabetes insipides, there is little raise in urine concentration even with fluid restriction (<600 mosm). If water restriction test is not diagnostic, exogenous ADH is given and the urine osmolarity is measured. In PPD, no increase in urinary concentration occurs even after exogenous ADH. In central diabetes, immediate response occurs in the form of increase in urine concentration.

An alternative method of differential diagnosis is MRI of the pituitary and hypothalamus. In most healthy adults and children, the posterior pituitary emits a hyperintense signal in T1-weighted midsagittal images. This "bright spot" is almost always present in patients with primary polydipsia but is invariably absent or abnormally small in patients with pituitary DI. It is usually also small or absent in nephrogenic DI presumably because of high secretion and turnover of AVP. Thus, a normal bright spot virtually excludes pituitary DI, argues against nephrogenic DI, and strongly suggests primary polydipsia.[16]

Treatment of hyponatremia is needed only if severe symptoms arise. Correction should be <12 meq/litre on day one and <6 meq/litre for every day after that.[12]. Correction should be continued till sodium levels raise to safer levels. (>118-120 meq). Rapid correction is needed if severe symptoms like seizure and coma arise. Hypertonic saline is given in a dose from 1ml/kg/hr up to 6ml/kg/hr if symptoms like seizure and coma arise. Therapeutic fluid restriction is an inexpensive form of treatment of PPD. It may take several days for effect to be seen.[12]. Behavioural programme to restrict water intake is also tried. Drugs like clozapine, low dose olanzapine, risperidone, beta blockers, clonidine, enalapril, ibersartan are used in the management of PPD.

CONCLUSION:
Psychogenic polydipsia should be considered after carefully ruling out the other causes of polydipsia. Careful history and water deprivation test will help in early diagnosis. Recognition and management of underlying psychiatric disorder is important. Life threatening complications like severe hyponatremia presenting as seizure and coma should be identified and managed accordingly. Regular follow up is mandatory as the disease has got a tendency to relapse.

BIBLIOGRAPHY:
1. Brian Dundas, MD, Melissa Harris, BS, and Meera Narasimhan, MD; Psychogenic Polydipsia Review; Etiology, Differential, and Treatment.


