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Original Article

Newly Diagnosed Patient with Central Diabetes Inspidous

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ABSTRACT

Central diabetes insipid us (CDI) is a syndrome by polyuria and polydipsia. This is attributed to arginine vasopressin (AVP), an anti-diuretic hormone that acts on kidney V2 receptors to enhance the re-absorption of free water. We reported a rare case of patient presenting with signs of polyuria and polydipsia which have a wide differential diagnoses of which CDI should not be neglected. Effective treatment can Improve the prognosis if diagnosed in time, We provide a summary of clinical findings, pathological features, brain imaging and laboratory investigations. We also discuss treatment strategies and approaches to monitoring the therapeutic response. lastly, we briefly summaries the current understanding of the path physiology ٦of the disease.

Key Words: *Central diabetes inspidious polyuria, polydipsia, Arginine vasopressin*

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INTRODUCTION

DI is a syndrome characterised by the excretion of abnormally large volumes of dilute urine[1]. Roughly 90% of the vasopressinergic neurons that connect the posterior pituitary to the hypothalamus must be injured in order to cause polyuric symptoms. Thus, there is always a strong correlation between central DI and severe hypothalamo-neurohypophysialdys function[2].

About 1% of cases have hereditary or familial CDI causes, while the majority of cases have acquired (idiopathic and iatrogenic) reasons[3]. The less common NDI is caused by a partial or complete resistance of AVP receptors to vasopressin. Some of the commonest NDI etiologies are electrolytic disturbances including hypokalemia and hypercalcemia. In our patient, both the potassium and calcium levels were within the normal.

Herein, we report a case of central diabetes inspidous likely secondary to Langerhans histiocytosis or lymphocytic hypophysitis and discuss the emerging difference in the incidence of hypophysitis/CDI .

Case presentation

A 22-year-old female was admitted to our ward with symptoms of nausea, epigastric pain and vomiting, she has episodes of acid reflux which has caused repeated vomiting , there is associated bloating which she puts down to drinking a lot of water. She also reported polyuria and polydipsia. She had been complaining about her extreme thirst for the entire day, continuous drinking water, and constant urination throughout the day for the last eight weeks—up to six litres per day, every forty to sixty minutes. Other than recurrent cellulitis, the patient had no known medical history and no drug history, with no history of a head injury.

On examination, she was comfortable not distressed, not dehydrated vital signs were stable apart from mild tachycardia , Cardio respiratory and abdominal examination were unremarkable, no muscle wasting or skin rashes. No focal neurological signs.

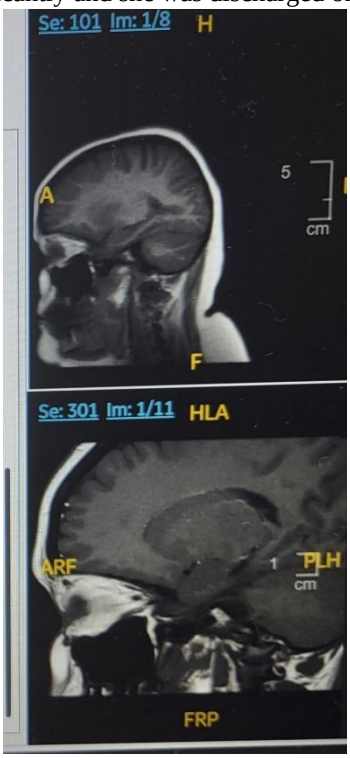
Investigation and progression during hospital stay

Initial blood tests showed WBCs of 15,3Hb of 152, K 3.7, Na 148.blood glucose was normal. RI head: bulkiness to the superior aspect of the infundibulum. The posterior pituitary bright spot is not seen.
US abdomen and pelvis: fatty liver infiltration, otherwise normal.

MRI pituitary: absent posterior pituitary bright spot ‘appearance of the Pituitary infundibulum represent Langerhans histiocytosis or lymphocytic hypophysitis, underlying neoplasm is unlikely.

Water deprivation test was done which showed serum osmolality of ranging from 299-309 and urine osmolality of 127 and 129. The patient was given 2 micrograms of desmopressin intravenously, serum osmolality decreased to 299 then 281, Serum sodium normalised (137), urine osmolality increased to 532., Copeptin was normal<1.2pmol/l.

The patient symptoms have improved significantly and she was discharged on 100 mcg desmopressin orally asm.



MRI: APBS and thickened centrally located infundibulum at the level of the optic chiasm.

Outcome and follow-up

She was reviewed in endocrine clinic two weeks later, stating, feeling almost back to normal, no polyuria, polydipsia or nocturia.

Repeated blood tests were normal apart from low fT4, She was advised to continue on the same dose of desmopressin100mcg tds‘started on 50 mcg levothyroxine and to be followed up in pituitary clinic.

Discussion

Dilute urine excretion in abnormally large volumes is the hallmark of the DI syndrome[3]. In addition to thirst, enuresis, polydipsia, nocturia, and urine incontinence, a patient with DI may also exhibit these symptoms. Diagnosing diabetic ketoacidosis (DI) in an adult patient entails assessing urine volume >2 mL/kg/hour or >300 mL/hour in two consecutive hours, hypotonic urine < 300 mOsm/kg, increasing plasma osmolarity > 300 moSm/L, ruling out hyperglycemia, and starting mannitol therapy[3]. For safe and efficient management, it might be caused by a variety of flaws, each of which must be identified. The aetiology of central DI is insufficient antidiuretic hormone (ADH) synthesis and secretion. The cause of gestational DI is an enzyme generated in the placenta that breaks down ADH. It's critical to distinguish between different varieties of DI in order to properly and successfully treat the illness[3].

Regardless of the cause, the primary MRI result linked to CDI is typically the lack of a normal T1 posterior pituitary bright spot (PPBS). It is optimal to evaluate the PPBS using a T1 sagittal precontrast sequence. In addition, up to 20%–30% of the normal population may not have PPBS in the absence of documented hypothalamic–pituitary dysfunction, particularly in older adults. Therefore, only in the appropriate therapeutic situations is the lack of PPBS meaningful in central DI[4]. Our brain imaging studies revealed no abnormalities on CT, and the lack of neurohypophyseal hyperintensity on T1 weighted images in the MRI suggested posterior pituitary dysfunction. To sum up, DI is a disorder of osmoregulation and water-electrolyte.

DI is an uncommon ailment that impacts one in every 25,000 individuals. The most prevalent type of DI is called CDI, and it usually results from hypothalamic-neurohypophysial dysfunction, which impairs the posterior pituitary's or the hypothalamus's ability to secrete enough arginine vasopressin (AVP)[1,5]. About 1% of cases have hereditary or familial CDI causes, while the majority of cases have acquired (idiopathic and iatrogenic) reasons[6]. When over 80% of the neurons that secrete AVP are injured, CDI occurs. The aetiology of the less prevalent NDI is partial or whole AVP receptor resistance to vasopressin. The most frequent causes of non-dissociative illness (NDI) are electrolytic abnormalities, such as hypokalemia and hypercalcemia.

DI is a rare condition that affects one in 25,000 persons (CDI) is the most common form of DI and is generally the result of hypothalamic-neurohypophysial dysfunction leading to inadequate arginine vasopressin (AVP) secretion from the posterior pituitary or inadequate production from the hypothalamus[6]. The majority of the causes of CDI are acquired (idiopathic and iatrogenic) whereas inherited/familial CDI causes account for approximately 1% of cases[6]. CDI develops when more than 80% of the AVP-secreting neurons are damaged. The less common NDI is caused by a partial or complete resistance of AVP receptors to vasopressin. Some of the commonest NDI etiologies are electrolytic disturbances including hypokalemia and hypercalcemia.

Based on serum and urine osmolalities, the diagnosis of DI may be clear in many cases of suspected patients. There might be no need for additional testing if there is a significant increase in serum osmolality and a corresponding decrease in urine osmolality. The diagnostic difficulty occurs when serum osmolality or sodium levels are inappropriately normal or "almost normal," or when primary polydipsia or NDI should be ruled out in the presence of polyuria and polydipsia symptoms. Dynamic tests, such the water deprivation test, are necessary in these situations because direct measurement of plasma AVP is rarely done due to its quick clearance. Nevertheless, even in the best of circumstances, the water deprivation test sometimes necessitates extended periods of observation and has modest sensitivity (86%) and specificity (70%)[6,7].

A pituitary "bright spot" absence on an MRI may or may not be accompanied by a 2-3 mm enlargement of the pituitary stalk; nevertheless, this finding is not always enough to confirm the diagnosis of CDI. Although it is absent in 20% of the general population, the posterior pituitary bright spot is a sign of stored vasopressin, and its absence on MRI is consistent with CDI. There was not a single encouraging thing about our patient that supported the CDI. Contrarily, no additional abnormalities were seen on the MRI of the pituitary gland, supporting the diagnosis of hypophysitis, which may manifest as a mild to moderate widespread swelling of the pituitary gland[8].

Ethics approval and consent to participate: ‘‘Not applicable’’, my manuscript does not report on or involve use of animal or human data or tissue.

List of abbreviations: Cdi(central diabetes insipidus),NDI(nephrogenic diabetes insipidus),AVP (Arginine vasopressin),APBS(Absent posterior pituitary spot)

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