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Case Report

A woman with polyuria and polydipsia

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Abstract

Diabetes insipidus (DI) is characterized by polyurea and polydipsia which occurs due to inappropriate secretion or action of antidiuretic hormone (ADH). DI usually are of four types viz. Central diabetes insipidus, Nephrogenic diabetes insipidus, Dipsogenic diabetes insipidus and Gestational diabetes insipudus. Among them CDI is most importent and is caused by damage to hypothalamus or pituitary gland. Mrs Morium, a 35 years house wife, hailing from village Darial, Noongola of Bogra sador upazilla, admitted to TMSS Medical college (TMC) and Rofatullah Community Hospital (RCH) on 16.08.2016 with complaints of intense thirst (water intake ~ 21 L / 24 h) and large volume of urination (~ 18 L/24 h). Her urine osmolality was 72 mosmol/Kg, Serum osmolality was 292 mosmol/Kg, MRI of brain (pituitary gland) was normal, Radio immuno assay of plasma AVP was 2.99 pmol/L. With single dose of Desmopressin per nasally improved her complaints and two dose daily completely ceased her problems.

Key words: Diabetes insipidus, polyuria, polydipsia

Introduction

Inappropriate secretion or action of serum antidiuretic hormone (ADH) is termed diabetes insipidus (DI), characterized by polyurea (defined as 24 hour urine output in excess of 40 ml/kg) and polydipsia[1]. The hypothalamus, a small gland located at the base of the brain, produces vasopressin also called antidiuretic hormone (ADH), controls the fluid removal rate through urination[2]. A decrease of 75% or more in the secretion or action of ADH usually results Diabetes insipidus (DI)[3]. The patients may pass 5–20 L or more of urine in 24 hours[3]. The types of diabetes insipidus include–Central, Nephrogenic, Dipsogenic and Gestational[2].

Among them central diabetes insipidus (CDI) is most important. CDI happens when damage to a person's hypothalamus or pituitary gland causes disruption in the normal production, storage and release of ADH[2]. Damage can result from surgery, infection, inflammation, a tumor, head injury, inherited defect in the gene that produces ADH and in some cases cause is unknown[2]. CDI is characterized by poly urea, polydipsia, urinary frequency and nocturia[4].

Nephrogenic Diabetes Insipidus (NDI) occurs when kidneys do not respond to ADH[2]. In adults, NDI is opposed to CDI, tends to present more gradually than CDI; as sensitivity to ADH decreases, polyuria increases[5]. NDI can result from gene mutation, chronic kidney disease, certain drugs, lower potassium level in blood, high calcium level in blood and some cases cause is unknown. Lithium, widely used in therapy of bipolar disorder, is the commonest of acquired NDI[6]. Other cause of NDI are bilateral urinary tract obstruction[7] and polycystic kidney diseases[8].

In cases, where polyuria is due to vast amount of ingested fluid driven primarily by behavioral or thirst disorders, it is called primary polydipsia or Dipsogenic diabetes insipidus occurs due to defect in thirst mechanism. Primary polydipsia is characterize by the ingestion of vast amount of fluid, most frequently water, causes hypotonic polyuria[9].

During pregnancy, the placenta produces cystine aminopeptidases (vasopressinase) which metabolizes both oxytocin and ADH[10]. When cysteine aminopeptidase levels are greatly elevated ADH is degraded beyond the capacity of the hypothalamic/pituitary axis to secrete it sufficiently to maintain a proper urinary concentration; This results in GDI, also known as 'transient DI of pregnancy[10].

The relationship between plasma osmolality and urine osmolality is helpful in establishes a diagnosis[11]. In order to diagnosis of DI, urine osmolality should < 300 mosmol/kg. The gold standard is a dehydration test followed by plasma ADH levels can be indirect

measurement of urine concentration is usually used first[5]. An MRI imaging appearance as a bright spot in the sella turcica which represents stored ADH; the absence of a bright spot is not necessarily diagnostic of CDI[12].

Case history

Mrs Morium, 35 years, house wife, hailing from village Darial, Noongola of upazilla Bogra sador, admitted in TMSS medical college (TMC) and Rafatullah Community Hospital (RCH) on 16.08,16. She had complaints of excessive thirst and frequent passes of large volume of urine for last one years. She had to awake up from sleep 5 – 6 times or more per night for drinking water and voiding urine. She had predilection of cold water and she use jug or larger bottle for drinking water. She had history of recurrent headache associated with nausea or aura and subside spontaneously or after taking paracetamol for last five years. She has no history of hypertension, Diabetes mellitus, hypothyroidism, hyperparathyroidism or electrolytes imbalance. There was also no history of taking drugs like Lithium, Amphotericin B, Demiclocycline or other nephrotoxic drugs. There was no history of renal disease, head injury or intracranial surgery. She belongs to a low socioeconomic family. None of her family members are suffering from such type of illness. On Physical Examination, The patient body build was average. During examination she was anxious and ill looking. In general examination nothing abnormalities detected. Her body weight was 64 Kg. Her systemic examination including cardiovascular and renal system reveal normal. On average water intake was 21 liter and urine output was 18 liter.

Urine RE and ME was normal. Urine osmolality 72 mosmol/Kg. Serum osmolality was 292 mosmol/Kg. Serum electrolytes was within normal range. Random plasma glucose (6.9 mmol/L) was normal. Serum albumin was 2.30 gm/L, S. creatinine was 0.92, and blood urea was 15 mg/dl. S. total calcium were 10.5mg/dl. Thyroid function test (TSH & FT4 were 2.95 m IU/L & 1.39 m UI/L) was normal. Ultrasonogram of whole abdomen was normal. MRI of Brain shows no abnormalities in Sella, supra sellar region and pituitary gland. Radio immunoassy of plasma (done in ONCQUEST Laboratory limited, New Delhi, India.) AVP was 2.99 pmol/L. The gold standard dehydration as stated above was tried but the patient could not tolerate.

The patient ultimately diagnosed a case of Cranial Diabetes Insipidus (CDI). Treatment was started with Desmopressin. With single dose of Desmopressin (Minirin) per nasally her symptoms improved much. It was noticed that with single dose at morning her symptoms appeared in the night. After observing her clinical out come after two days, two dose was given; one in the morning and another in the evening. It was observed that with two dose 12 hourly completely ceased her symptoms.

Discussion

Mrs Morium, 35 years, admitted in TMC & RCH with complains of severe polydipsia and polyuria for last 1 years. She had no other physical abnormalities. Her average water intake was 21 L and urine output 18 L. Her serum osmolality was 292 mosmol/Kg. and corresponding urine osmolality was very low (72 mosmol/Kg). All other investigations including MRI of Brain reveled nothing abnormalities. Her basal plasma ADH was 2.99 pmol/L which was lower limit of normal (normal <13 pmol/L). With single dose of Desmopressin (Minirin) per nasally her symptoms improved and two dose 12 hourly completely ceased her symptoms. So, certainly this was a case of cranial diabetes insipidus (CDI). As there was no history of surgical intervention or radiotherapy in brain, feature goes in favor of essential CDI. It is more common in very earlier age but in this case it was middle aged patient. The history of her 5 years suffering from headache may reflect any disease causing destruction at the site of hormone genesis or drainage, which could not found during investigation. It was found in study[2] that up to 30% of the cases of DI that are eventually diagnosed do not have a contributory medical causes to it that has been discovered.

It can be mentioned here that follow up of the patient with two dose of Desmopressin after one year, showed she was very normal in every aspect of her life.

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