ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 6, Issue – 6, November – 2021 , Page No. : 01 - 06

Dipsogenic Diabetes Insipidus Post-Acute Viral Meningoencephalitis – A Case Report

[1]Dr. Suneetha D K, MBBS, MD, Professor and Head, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India

[2]Dr. Bharath M. M., Senior Resident, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India

[3]Dr. Ravi Chethan Kumar A N, MBBS, MD, Associate Professor, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India

[4]Dr. Madhu Kumar R, MBBS, MD, DNB, Assistant Professor, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India

Corresponding Author: Dr. Bharath M. M., Senior Resident, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India

Citation this Article: Dr. Suneetha D.K, Dr. Bharath M.M, Dr. Ravichethan, Dr. Madhu Kumar, "Dipsogenic Diabetes Insipidus Post-Acute Viral Meningoencephalitis – A Case Report", IJMSIR- November - 2021, Vol – 6, Issue - 6, P. No. 01 – 06.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Dipsogenic diabetes Insipidus (DDI) is a subtype of primary polydipsia (PP), which occurs mostly in healthy people without psychiatric disease.[7] This case highlights the importance of detailed investigation to differentiate between central diabetes Insipidus and Dipsogenic polydipsia in the background of neuro infection, thereby avoiding misdiagnosis and iatrogenic hyponatremia which can have deleterious consequences.

Keywords: PP, ADH, DDI

Introduction

Primary polydipsia (PP) is a clinical disorder characterized by excessive thirst leading to excessive fluid intake and consequent excessive excretion of urine and has three subtypes: Dipsogenic diabetes Insipidus, psychogenic polydipsia and iatrogenic polydipsia.

Dipsogenic diabetes insipidus, subtype of primary polydipsia is defined as a rare non-psychiatric syndrome of disordered thirst, in which the osmotic threshold for thirst is abnormally low, below the threshold for antidiuretic hormone (ADH) release.

The diagnosis of dipsogenic diabetes insipidus requires a history of excessive thirst, no history of psychiatric illness, documentation of polyuria with low urine osmolality, and confirmation of intact urinary concentrating ability[7].

In the present case, we present a 26-year-old female with severe symptoms of polydipsia and polyuria in the background of neuro infection, and no psychiatric illness who underwent a thorough physical and

laboratory investigation finally leading to the diagnosis of dipsogenic diabetes insipidus

Report of a Case

The patient is a 26 year old female unmarried working as a teacher in a private sector.

The patient presented with complaints of headache and fever on day 1, for which she was taken to a hospital in her hometown and was symptomatically managed for 2 days

On day 3, patient had multiple episodes of vomiting and had about 4 episodes of generalised tonic clinic convulsions and was referred to our hospital for further management.

On day 4, patient was prophylactically intubated in intensive care unit. Neurologist opinion was taken and diagnosis of viral meningoencephalitis was made and treatment was started for the same. During the stay in ICU, patient vitals were stable and urine output 1700ml per day. Patient gradually recovered over the next 3 days and consciousness was improved, weaning was done.

On day 8 patient complained that she had increased frequency of micturition about 25 episodes per day, which was associated with increased thirst sensation.

On quantification, patient had passed around 17 litres of urine per day and around 15 litres of urine for next 3 days and the fluid balance assessment confirmed profuse intake of fluids around 9 litres of water each day.

Patient vitals were stable and routine blood and urine investigations were within normal range. (Table 1)

Patient was evaluated for polyuria based on the protocol (Figure 1) [5]

A 24-h urine volume was used to confirm polyuria which was 17 liters

Urine osmolality was 241.9mosm/l.

Water deprivation test was done as per the protocol for 8 hours and the results showed marked increase in urine osmolarity on water deprivation with serum osmolarity remaining the same.(Table 2) and was interpreted accordingly (Table 3)

On completion of water deprivation test, Plasma AVP levels were measured which was within normal range (43.3pg/ml)

Patient was diagnosed to have primary polydipsia and MRI brain was done which showed the presence of pituitary bright spot.

Desmopressin trial was given which showed decrease in serum sodium levels confirming the presence of primary polydipsia.

With no significant psychiatric history, primary polydipsia-dipsogenic diabetes insipidus subtype was confirmed.

The patient was advised Behavorial therapy and she noticed significant improvement in her symptoms within few weeks.

Discussion and Conclusion

In the present case, the major difficulty regarded the determination of the patient's cause for polyuria in the background of neuro infection.

Various etiologies and theories hypothesizing the etiology of dipsogenic diabetes insipidus include idiopathic, structural lesion similar to central diabetes insipidus, due to behavioral aspects, due to a belief that drinking excess water will protect against the formation of nephrolithiasis or have more pathologic benefits, lesions in the hypothalamus,[8] dopamine receptor super sensitivity [4].

Dopamine administration in animal models has been reported to initiate increased drinking.

We speculate that in this patient, acute neuro infection would have caused the stress resulting in increased dopamine signaling, thus triggering the polydipsia.[4]

Another hypothesis in our patient could be that viral meningoencephalitis might have caused a transient structural damage in the thirst centre of hypothalamus causing osmotic thirst threshold below that of ADH release, which is distinctly abnormal[8].

This case illustrates that the diagnosis of dipsogenic diabetes insipidus can be challenging.

Several clinical findings can aid in the differentiation: a sudden onset of polydipsia, excessive polyuria, drinking at night, nocturia, and persistence of symptoms are suggestive of cranial diabetes insipidus.

However, clinical findings can be misleading as shown in a recent prospective study wherein more than 60% of patients with PP reported nightly drinking, most of them preferred cold beverages, and nearly 80% indicated a sustained character of symptoms[4]. In this patient, the age, the absence of a psychiatric background, the diuresis of 17 liters, nocturia, and presence of neuro infection hinted at a diagnosis of central diabetes insipidus, however the detailed laboratory investigations proved otherwise.

This case report thus illustrates that clinical signs and symptoms are not specific or sensitive enough to differentiate between the different causes of polyuria and polydipsia.

Our patient underwent a water restriction test, the gold standard to which measures the ability of the CNS to produce AVP and of the kidney to respond to it, thus helping in differentiation of etiologies of polyuria. Based on the interpretation of results from water deprivation test, the patient had increasing urine osmolarity with normal serum osmolarity suggestive of primary polydipsia.

The further evaluation showed normal plasma AVP levels and presence of posterior pituitary bright spot viewed as high intensity signal on T1 weighted images on MRI brain which is believed to be from the storage of vasopressin.

The detailed investigation led to the diagnosis of primary polydipsia of dipsogenic diabetes insipidus subtype as the patient has no significant psychiatric history.

Unfortunately, the treatment options for dipsogenic polydipsia are scarce. The perfect therapy would be a voluntary reduction of water intake, but this is often not attainable as patients will not comply with this strategy. Behavioral therapy that includes education on the disease, relaxation therapy using biofeedback and conditioning of desired behavior is a valid alternative[4].

This case also underscores the importance of differentiating between dipsogenic diabetes insipidus from central diabetes insipidus as a wrong diagnosis and treatment can cause harm to patients.

The main risk of wrong use of desmopressin in primary polydipsia leads to the development of potentially fatal fluid overload, profound hyponatremia which acutely leads to increased risk of serious complications such as brain edema, seizures, falls, and fractures as well as rhabdomyolysis.

In conclusion, this case report shows that dipsogenic diabetic insipidus can present atypically in a patient with acute viral meningoencephalitis with sudden onset of polydipsia and very large urine voids, mimicking central diabetes insipidus. To differentiate from partial CDI, a water restriction test can be helpful when performed within the first week of onset.

Although the water restriction test is the golden standard, additional tests such as plasma AVP levels, desmopressin trial test and MRI brain increases the diagnostic accuracy, in particular. It also highlights the importance of Behavorial therapy in this subset of patients as it has significant effect in improvement of symptoms.

Acknowledgements

Dr. Himamani, Consultant Nephrologist, INU, Mysore.

Dr. Rekha Bhat. V, Consultant endocrinologist. Coloumbia Asia hospital, Mysore

References

1. Primary polydipsia – A case report by Kalliopi Tournikioti, MD, Konstantinos Voumvourakis, MD, PhD

2. Diabetes insipidus in the diagnosis of polyuria by Michał Graczyk1, Anna Kamińska2 et al.,

3. A 27-Month-Old Boy with Polyuria and Polydipsia by Yvonne Lee, Erica Winnicki et al.,

4. Psychogenic polydipsia in a female adolescent without a psychiatric background: A case report by Elise Nauwynck , Karolien Van De Maele et al.,

5. Indian journal of endocrinology and metabolism Diabetes insipidus: The other diabetes by S kalra et al., Year : 2016 | Volume : 20 | Issue : 1 | Page : 9-21

6. Diagnostic Testing for Diabetes Insipidus by Sriram Gubbi, Fady Hannah-Shmouni et al.

7. Dipsogenic diabetes insipidus: report of a novel treatment strategy and literature review by Robert M. Perkins • Christina M. Yuan • Paul G. Welch

8. A Subset of Primary Polydipsia, “Dipsogneic Diabetes Insipidus”, in Apparently Healthy People Due to Excessive Water Intake: Not Enough Light to Illuminate the Dark Tunnel Krishnaraju Venkatesan 1, Kumarappan Chidambaram et al.,

Legend Tables

Table 1

Hb(g/dl)	11
WBC (/µl)	4500
S. Sodium (mEq/L)	138
S. Potassium (mEq/L)	4.6
S. Calcium(mg/dl)	9
S. Creatinine(mg/dl)	0.7
RBS (mg/dl)	97
Urine pus cells	2-3
Urine OSM	241
Urine albumin	Nil
Urine specific gravity	1.007

Table 2

Time	Body Weight	Urine Osmolarity (Mosm/Kg)	Serum Osmolarity	Blood Pressure Mmhg	Urine Volume (Ml)
10 Pm	34.5 Kg	241	278	110/70	
11 Pm	34.5kg				
12am	34.3kg	278	276	110/70	250
1 Am	34.3kg				
2am	34.2kg	1252	279	106/70	200
3am	34.2 Kg				
4am	34.2kg	1266	282	106/70	200
5am	34.1kg				
6am	33.9kg	1304	278.1	100/60	160

Results of Water Deprivation Test

Table 3 [5]

Table 3: Interpretation of the water deprivation test and the desmopressin challenge test in the diagnosis of diabetes insipidus

Diagnosis	Urine osmolality (mOsm/kg)	
	After desmopressin	After fluid deprivation
CDI	>750	<300
NDI	<300	<300
PP	-	>750
? partial CDI or ? partial NDI or ? PP	<750	300-750

Interpretation of the water deprivation test results.

Figure 1[5]