Case Report

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Polyuria-polydipsia syndrome approach: clinical case and review

Abstract

Polyuria-polydipsia syndrome (PPS) compared three-pathologies: nephrogenic or central diabetes insipidus and primary polydipsia. The initial approach considers different causes, and it requires a complete evaluation of the fluid-status support by physician. The diagnosis must exclude frequent abnormalities. Clinical case: A 57-year-old male during his hospitalization documents polydipsia and polyuria with urine volume of 3.5 to 8.45 L/day. We did a test for identify to etiology and other causes was eliminated, concluding a primary polydipsia in a patient with the most important risk was his psychiatric component. An algorithm is proposed according to this experience obtained with the case presented with a review of the topic from the point of view of the internist physician in a patient with a PPS in a controlled-environment.

Keywords: Polyuria, Polydipsia, Diagnosis, Approach.

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Martínez-Romo ES,[1,2] Sánchez-Escobedo Y[1,3]
[1]Regional Hospital of Poza Rica, PEMEX, Department of Internal Medicine, Mexico
[2]National Autonomous University of Mexico, Mexico
[3]Meritorious Autonomous University of Puebla, Mexico

Correspondence: Sánchez-Escobedo Y, Regional Hospital of Poza Rica, PEMEX, Department of Internal Medicine, 16 Ote. Laredo, 93260 Poza Rica de Hidalgo,Veracruz, Mexico, Email yeiscimin@gmail.com

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Introduction

Polyuria-polydipsia syndrome (PPS) comprises the following pathologies as: diabetes insipidus (nephrogenic or central) and primary polydipsia. Polyuria is defined as a normal excretion of high volumes of dilute urine in adults is considered greater than 3 liters in 24 hours or greater than 40-50 milliters/kilograms in 24 hours, or a urinary osmolarity less than 300 mOsm/L. The origin is multifactorial, so it is initially considered as a syndrome. It is classified according to the amount of urine (liters) in a 24-hour period. Considered as: mild from 3 to 5 L, moderate from 5 to 7 L and severe when it's greater than 7 L1. The differential diagnosis of PPS lies in the etiology of each pathology. In general, it is due to a secondary alteration of vasopressin with modifications at different levels that obey to either alterations in the receptor or the inhibition of hypothalamic-pituitary axis at central level owing to structural or functional alterations. As a result, there is a change in solute and water concentration and consequently in the osmolarity generating a change in water movement. There is an ultra-excretion; so, it is vital and necessary to know the cause to stop the polyureic state of the patient.[1,2]

This is not a common condition found in a hospital environment where the variables of consumption and water intake are relatively controlled. So, physician must pursue a correct approach to this syndrome, consider all the possible causes and even delve into the complete pharmacological profile and drug interactions with potential effects or changes in increased lipid disorder or hyperglycemic states. We present a case of a man with polyuria in hospitalization, and a diagnostic pathway is made according to the latest suggestions on PPS approach.

Clinical case presentation

A 57-year-old male with a history of type 2 diabetes and psychiatric disorder due to schizophrenia in medical treatment of 5 years of evolution with quetiapine, denies use of magnesium valproate, lithium, or carbamazepine. He was admitted by urinary tract infection, during hospital surveillance he referred polydipsia as cardinal symptom. The patient did not present any state of acute metabolic imbalance. During his hospitalization he received resuscitation and water replacement by weight and treatment with an empirical and targeted antibiotic regimen with cephalosporin. Initial urinary volume was 3.5 liters per day, then with a tendency to increase despite water restriction and fluid balance control measures, reaching a maximum volume of 8.45 liters per day.

The diagnostic approach of PPS was developed during his in-hospital stay. Firstly, the total weight of the patient was established as 83.700 kilograms, then water and electrolyte requirements were adjusted. In addition, and as a part of the protocol, we did imaging studies were included to rule out structural and functional pathologies (includes MRI and hormonal profile), mainly renal ultrasound, which was reported without any pathological findings. During the follow-up, factors that could have intervened with the test results were excluded such as effective glycemia control, antipsychotic drugs use and water administration per kilogram of weight according to the basal requirements of liquids were adjusted. Nonetheless, we found the persistence of polyuria, therefore, a water restriction test was decided.

Paraclinical tests were taken to evaluate global alterations and pre and posttest follow-up. The serum osmolarity reported was 295 mOsm/kg, with urinary osmolarity of 145 mOsm/kg FENa greater than >2. A test with intranasal desmopressin was performed with new laboratory controls and urinary volume monitoring at determined times at 8 and 24 hours with partial response finding serum sodium of 145 mEq/L and urinary osmolarity of 380 mOsm/L (Table 1). A diagnostic approach is proposed and considering the results obtained after the tests performed, a primary polydipsia was concluded in the case presented (Figure 1).

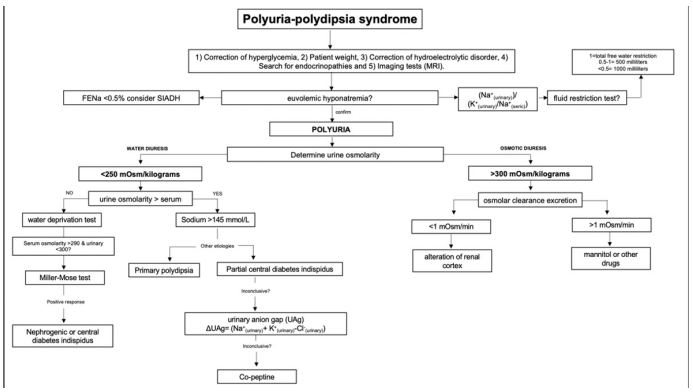

Figure 1

Discussion

The cause of PPS is variable, the initial approach starts from the clinical history since most of the cases have been associated with psychogenic causes. Hence, the differential diagnosis starts from the adequate interrogation and ruling out other type of alterations in a relatively controlled environment making use of imaging studies.[3] The first test to be performed should be water deprivation where vasopressin activity is indirectly evaluated, however, it has a low diagnostic accuracy (41%). Thus, the etiological diagnosis of PPS depends on the diagnostic standard which has been recently established and according to guidelines are copeptin levels with a performance of area under the curve >90% conjugated with vasopressin levels increases sensitivity. Due to the limitations in our environment to make use of this type of test we are in need to use other exclusion tools.[3,4]

Table 1 Patient's paraclinical results (Miller-Mose Test)

Variables	Pre	Post
Serum osmolarity (mOsm/kg).	286	304
Urinary osmolarity (mOsm/kg).	131.6	381.51
Fractional excretion of sodium (FENa) %	>2	>2
Creatinine:		
Serum (mg/dl)	1.2	1.16
Urinary	18.8	28.2
Serum electrolytes (mmol/L):		
Sodium	144	145
Potassium	3.6	3.8
Chlorine	110	106
Magnesium	1	1.1
Calcium	7.2	7.3
Phosphorus	3.2	3.8
Index Potassium urinary/Creatinine urinary	<0.2	<0.2
Urinary delta GAP	43.3	66.5
Urinary electrolytes (mmol):		
Sodium	107	173
Potassium	8.3	16.5
Chlorine	72	123

In our case, urinary delta gap allowed us to direct the pathology towards the primary cause. Another obstacle for the diagnosis is the need for documenting polyuria and maintain strict control balances with the use of serum and urinary electrolytes, when we go through the control phase and there is persistence of alterations, the first test to perform is water restriction; it is suggested to individualize in the context of other comorbidities such as in patients with heart failure or chronic kidney disease where the enteral intake is less than 1000 milliliters of water per day. The clinical and biochemical criteria for adequate response are: urinary osmolarity greater than 800 mOsm/kg after two measurements, weight loss greater than 3% of past weight, increased sodium level greater than 150 mmol/L; when any of these criteria is inconclusive, a Miller-Mose test should be performed, even if other peptide measurements are available.[5,6]

The desmopressin test has a sensitivity of 93% and a specificity of 83%, in consequence, in many hospital centers is the first test to differentiate the etiology of PPS. This test works by producing a gradient of urine concentration elimination in the renal medulla through the aquaporin 2 receptor. The values after the test broaden the possibilities and rule out others (Table 2).[6] For the interpretation, we must take into account false positives, for example: administration of platelet-rich plasma, among others.

Table 2 Interpretation of results of different diagnostic tests

Nephrogenic Diabetes Insipidus	<300	<50	>21.4
Complete Central Diabetes Insipidus	<300	>50	<2.6
Partial Central Diabetes Insipidus	300-800	Oct-50	<5
Primary Polypsia	300-800 o >800	<10	>5

Additionally, there are other diagnostic tools which are not yet available in our environment which have demonstrated superiority in test performance curves. For instance, measuring co-peptin; where a value greater than 5 pmol/L is considered with a sensitivity of 82%, specificity 92% whereas the sensitivity and specificity increase to 96% and 81% respectively if the acute deprivation test is added (co-peptin delta) considering positive as more than 21.4 pmol/L6,7.

The patient it was considered as partial responder when finding persistent hydro electrolytic alteration after intranasal desmopressin test with a single dose. Here by, this pattern would obey to a psychogenic primary cause.

Conclusion

PPS should have a multidisciplinary approach, the internist should rule out structural and functional causes, from the most frequent to referral to the subspecialist physician. The only evaluation of serum and urinary osmolarity is enough to have a more directed diagnostic pathway.

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Declaration of conflicts of interest

The authors declare no conflict of interest.

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