Rhabdomyolysis due to polydipsia in a patient with psychotic disorder

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Rabdomiólisis por polidipsia aguda en un paciente con trastorno psicótico

Summary

Polydipsia is a frequent clinical entity in psychiatric patients, especially in those with a psychotic disorder. Acute epidodes of polydipsia can produce important metabolic alterations and even coma and death. Psychogenic polydipsia is a underestimated diagnosis, due to multiple causal factors and an etiology that has not been clearly established. We present the case of a patient with psychiatric background who was seen due to a clinical situation of severe acute renal failure by high rhabdomyolysis that needed hemodialysis, due to acute polydipsia. We also review some of the epidemiological and clinical factors and etiopathogeny of the polydipsia. It is considered necessary to keep in mind the diagnosis of polydipsia in any psychiatric patient showing acute symptoms of confusion.

Key words: *Rhabdomyolysis. Polydipsia. Psychosis.*

Resumen

La polidipsia es un cuadro clínico frecuente en pacientes psiquiátricos, especialmente en pacientes psicóticos. En episodios agudos puede producir alteraciones metabólicas importantes, e incluso coma y muerte. La polidipsia psicógena es un diagnóstico infravalorado debido a tener múltiples factores causales y una etiopatogenia no claramente establecida. Se presenta el caso de un paciente con antecedentes psiquiátricos que es atendido por un cuadro clínico de insuficiencia renal aguda severa por intensa rabdomiólisis que requirió hemodiálisis, relacionado con conducta aguda de polidipsia. Se revisan también algunos de sus factores epidemiológicos, clínicos y etiopatogénicos de la polidipsia. Se establece la conveniencia de tener en cuenta el diagnóstico de potomanía ante cualquier paciente psiquiátrico que presente síntomas agudos de confusión.

Palabras clave: *Rabdomiólisis. Polidipsia. Psicosis.*

INTRODUCTION

Primary or psychogenic polydipsia is a clinical disorder characterized by excessive intake of water in the absence of physiological stimuli for this intake. This clinical picture is frequent in psychiatric patients, especially in schizophrenia. The presentation form can be both chronic as well as acute, and can produce important metabolic disorders, and even coma and death. The existence of multiple factors that can influence its presentation and an etiopathogeny that is not clearly established are the reason why its detection is underevaluated and, thus that an adequate treatment is not established.

CLINICAL CASE

A 47 year old male patient who came to the Emergency Service accompanied by his family due to presenting a picture of decreased awareness, disorientation and disorganization of behavior with agitation having a 24 hour evolution.

On evaluation, he was disoriented and confused, with psychomotor agitation, environmental disconnection and without the possibility of verbal contact. His vital signs were axillary temperature of 39.4º C, blood pressure 103/69 mmHg and heart rate of 115 bpm. He had several bruises on different parts of his body in relationship with falls to the floor and blows against furniture, reporting moments of vision loss. In the laboratory analyses performed in the Emergency Service, the following stand out: leukocytosis (24.8×10^9/l); creatinine, 2.8 mg/dl; sodium, 128 mEq/l; potassium, 5.4 mEq/l; calcium, 8.2 mg/dl; GOT, 1284 U/l; magnesium, 2.41 mg/dl, and acid-base balance with pH, 7.3; pCO_2, 42 mmHg; pO_2, 35 mmHg; bicarbonate, 2 mmol/l; BE, –4.1 mmol/l, and oxygen saturation of 62%. A very important elevation also stood out in creatine kinase (CK) enzyme) with concentrations of 129,931 U/l. The chest X-ray was normal. The brain CT scan showed a significant cortical atrophy for the patient's age without another significant finding. The lumbar puncture was normal. The presence of abuse drugs in urine was ruled out. Given the condition of the patient, who also presented a total anuria and renal function parameters that

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rapidly deteriorated, it was decided to transfer him to the ICU in order to perform adequate sedation of the agitation picture and organic control of the patient.

Information was gathered about the patient through his family, in which the presence of excessive intake of water, about 10 liters/day, in the last week stands out. Among the personal background, they reported usual alcoholism in the last 5 years with approximate consumption of one liter of wine a day and presence of psychiatric background that initiated 20 years ago with a progressive decrease in usual functioning and performance in his activity. He initiated antidepressive and anxiolytic treatment, that was maintained until 24 years of age, when he was admitted to the hospital for a psychiatric study, and was diagnosed of possible non-specific psychotic picture. Antipsychotic treatment was initiated, which he abandoned shortly after, also abandoning the psychiatric control. Since then, he has only taken anxiolytic medication. There is progressive functional deterioration, he has not been able to establish stable work activity and he has important social withdrawal and thus has hardly left his home in the last 5 years, with progressive loss of interest and motivation in regards to the surroundings.

In the ICU, venovenous hemodiafiltration was initiated with improvement of renal parameters in the first days, decrease of CK level until normal values as well as normalization of the hepatic tests and the ionogram. His fever abated at 48 hours and the leukocytosis disappeared. After reduction of his stage of agitation, the patient remained conscious, oriented and without signs of neurological focality. He is sometimes irritable and even verbally aggressive, with speech impoverishment, short responses and total amnesia of what happened in the days prior to admission. Occasionally he reports some visual hallucinations in form of animals that are self-limiting. In order to favor sedation state, treatment was initiated with haloperidol up to 10 mg/day, initially by parenteral route and later orally. Other complementary diagnostic examinations were performed, standing out among them negative antinuclear and Jo-1 antibodies, normal ADH, normal abdominal ultrasonography and MRI in which only cortical atrophy having mild parieto-occipital predominance stands out. After several days, an attempt was made to remove the hemodiafiltration but adequate levels of diuresis could not be achieved and progressive deterioration of the renal parameters was observed so that treatment was proposed with dialysis that was performed for four weeks, achieving adequate recovery of the renal function.

As discharge diagnosis, a picture of renal failure secondary to rhabdomyolysis due to hyponatremia, caused by an acute polydipsia in a patient with psychiatric diagnosis of residual schizophrenia was proposed.

DISCUSSION

Psychogenic polydipsia has been related both with psychiatric as well as organic causes. It is relatively frequent (3-39%) in patients with psychiatric disease[1], schizophrenia standing out in 70% of the cases, although its presence in affective disorders, stress, alcohol abuse, eating disorders, mental retardation, personality disorders and organic mental disorders has also been described[2]. A profile of characteristics associated with the presence of polydipsia has been indicated among schizophrenic patients, so that these are general patients having a long evolution, with maintained metabolic disorders and presence of cortical atrophy and cognitive deficits[3].

Organic factors that can occur with alteration of the water elimination capacity have been established. These can be pain or the administration of drugs such as amitriptyline, fluphenazine, thiothixene, carbamazepine, barbiturics, clofibrate, isoproterenol, diuretic agents, chloropropamide and morphine[4]. It has also been related with tobacco, coffee and alcohol abuse[5,6]. Regarding psychotropic drugs, there are contradictory data on whether they act as a precipitant of polydipsia or may have a therapeutic function. Thus, it has been observed that these are generally patients who take psychotropic drugs with anticholinergic properties, which can cause mouth dryness and thirst as a factor influencing the polydipsia[7]. A certain coincidence between intoxications due to water intake and exacerbation of the psychosis has also been detected, and, even though the exact mechanism of this association is not known, it must be considered that dopamine plays an important role in the control of thirst and secretion of antidiuretic hormone. It has been verified that dopaminergic D2 blockage increases type II angiotensin, a potent dipsogen[8]. The possible influence of antipsychotics in the presence of rhabdomyolysis secondary to hyponatremia or a direct effect of psychotropics in the appearance of hyponatremia has also been pointed out[9]. On the contrary, there are authors who recommend administering neuroleptics for the treatment and control of psychogenic polydipsia in patients with psychosis[10]. In any event, faced with the hypothesis of the implication of neuroleptic treatments in the physiopathology of polydipsia-hyponatremia, it must be remembered that the first descriptions of polydipsia in psychiatric patients go back more than 70 years when antipsychotic drugs had still not been synthesized[11].

The presentation form of primary or psychogenic polydipsia may be either a maintained excessive water intake with normal kidney excretory capacity that gives rise to hypotonic polyuria or an intermittent intake of large quantities of liquid, that can cause water intoxication and dilutional hyponatremia if the excretory capacity of the kidney is exceeded. This latter case is not frequent in healthy adults because they have the capacity to excrete important amounts of water (from 10 to 14 ml/min), unless there is a renal disorder that makes this excretion difficult, as, for example, during treatment with thiazidic diuretics. Chronic excessive intake of liquids, characteristic of psychogenic polydipsia, may also limit the capacity of renal excretion as it produces a lavage of medullary osmotic gradient. Thus, a decrease of the

osmotic threshold for secretion of antidiuretic hormone (ADH), an increase in sensitivity to the natriuretic effect of the ADH in the kidney, even at low concentrations of it[5] and an increase in the levels of cerebrospinal fluid of the angiotensin converting enzyme has also been observed in psychotic patients treated with neuroleptics, with polydipsia and hyponatremia compared with psychotic patients without neuroleptic treatment[12].

Hyponatremia is a change that is frequently associated with excessive intake of liquid. It may either be due to inadequate secretion of ADH or to internal factors of the kidney such as decrease in glomerular filtrate derived from severe renal failure. Association between psychogenic polydipsia and rhabdomyolysis[13] with very significant increases in CK, and even clinical situations of coma[14] is relatively frequent. CK concentrations for cases of hyponatremia may vary from 1,800 to 100,000 U/l. The physiopathological condition that relates hyponatremia and rhabdomyolysis is not clear, but two hypotheses have been supplied: the first establishes that the exit of intracellular potassium to compensate the cellular edema caused by hyponatremia could cause a decrease in the transmembrane potential with rupture of the muscular cell[14]. The second hypothesis proposes that the correction rate of hyponatremia is the cause of the cellular lesion, so that the cellular balance that is established during prolonged hyponatremia may not be very stable during its recovery treatment[15]. One author has mentioned the role of the antipsychotic, clozapine, on the increase of the permeability of the muscular cellular wall[9].

Between the beginning of the polydipsia habit and the appearance of symptoms suggestive of water intoxication, a relatively moderate time period, between 5 and 10 years, generally passes[16]. In the case of the patient reported, the behavior of excessive water intake as well as the symptoms presented, are acute. This agrees with that described for a cerebral edema due to water intoxication, in which headache, blurred vision, nausea, vomiting, diarrhea, muscular cramps, restlessness, confusion, convulsions, and even coma and death can appear[17]. In our case, the absence of a well defined history of psychiatric disorder, of recent neuroleptic treatment and a background of excessive water intake behavior made it difficult to reach a diagnosis. The presence in this patient of a factor considered to be at risk for the appearance of behaviors of excessive water intake and hyponatremia, such as excessive alcohol consumption during the last years, must be mentioned[13]. It also stands out that while the hyponatremia in situations of rhabdomyolysis was important in other cases previously described[14], it was only moderately low in our patient, considering the levels of tissue destruction observed.

While the treatment of acute water intoxication is more related with hydroelectrolytic control until its recovery, there is major difficulty to treat chronic psychogenic polydipsia. In spite of there being very few controlled studies of drug treatments for polydipsia in mental patients, some antipsychotics such as clozapine, propanolol, angiotensin converting enzyme inhibitors and opiate antagonists such as naloxone have been proposed[10,18,19].

Finally, we want to stress that primary polydipsia is an underevaluated diagnosis, partially due to the fact that its causal and etiopathogenic factors have not been established which, however, can give rise to important physical complications, even with life risk. This diagnosis must be taken into account, therefore, as a possibility in any psychiatric patient who presents acute confusion symptoms.

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