USE OF CLOZAPINE IN A PATIENT WITH SCHIZOPHRENIA AND POLYDIPSIA

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Abstract

Objective. Psychogenic polydipsia, although strongly associated with chronic psychosis, remains an underestimated and not clearly understood clinical condition in the psychiatric field. The aim of this study is to report a clinical case of potomania and highlight both the features and the treatment of the syndrome.

Method. Authors report a case of a 64-years-old man with a six month history of excessive water drinking. The anamnestic and clinical features of the patient, the diagnostic protocol used for the diagnosis and the clinical follow-up are described.

Results. A low-dosage treatment (350 mg/day) with clozapine allowed us to both resolve the negative symptomatology and control the abnormal fluid intake.

Conclusions. A possible etiological explanation of polydipsia among psychotic patients, involving dopaminergic circuits, is given. Clozapine can represent a valid tool to improve psychotic features and also to rapidly correct and stabilise the water/sodium metabolism, before emerging further complications.

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1. Introduction

An excessive fluid intake (>3L/days) is currently defined polydipsia: this syndrome can be frequently associated to many general medical conditions that produce primary polyuria. Polydipsia itself often implies a substantial risk of mortality [4] and morbidity [3], requiring an early diagnosis and a rapid establishing of a successful treatment. A psychogenic form of primary polydipsia (also named "potomania") has long been reported to be related to mental illnesses and can easily cause misdiagnosis [7,2], thus representing a relevant differential diagnostic problem. Furthermore, even if atypical antipychotics have been previously reported as a first choice treatment for this syndrome [5,8], no specific treatment for the syndrome has been identified yet [1].

2. Case report

F.L. is a 64 years old man, who had been admitted to a mental hospital since he was 34 years old. For more than 30 years, he showed a stabilized negative schizophrenic clinical picture [mainly characterized by affective flattening, alogia, avolition, anaedonia, and social withdrawal], receiving a DSM-IV diagnosis of "residual schizophrenia". For several years he was administered benzodiazepines, haloperidol and thioridazine, but during the year preceding the admission to our department he took diazepam only. After admission, he underwent a pharmacological three-week wash-out. The polydipsic symptomatology, with remarkable polyuria and polydipsia, started six months before the admission and remained stable, with an average estimated water intake of about 10 litres per day. We monitored the daily fluid intake and a catheter was used in order to measure the urine daily amount produced.

We performed endocrinological, haematological and neuroradiological investigations, but all the tests did not show the presence of a primary medical condition underlying polydipsia [ADH level was in the normal range (i.e.: 9,2 pg/mL; n.v. ≥8 pg/mL)].

Seriated plasma electrolyte, evaluated at least two times per week, both before and during our hospitalisation, showed a typical intermittent hyponatremia going from a minimum value of 117 mmol/L to a maximum value of 144 mmol/L (normal values range: 130-150 mmol/L), as depicted in figure 1.

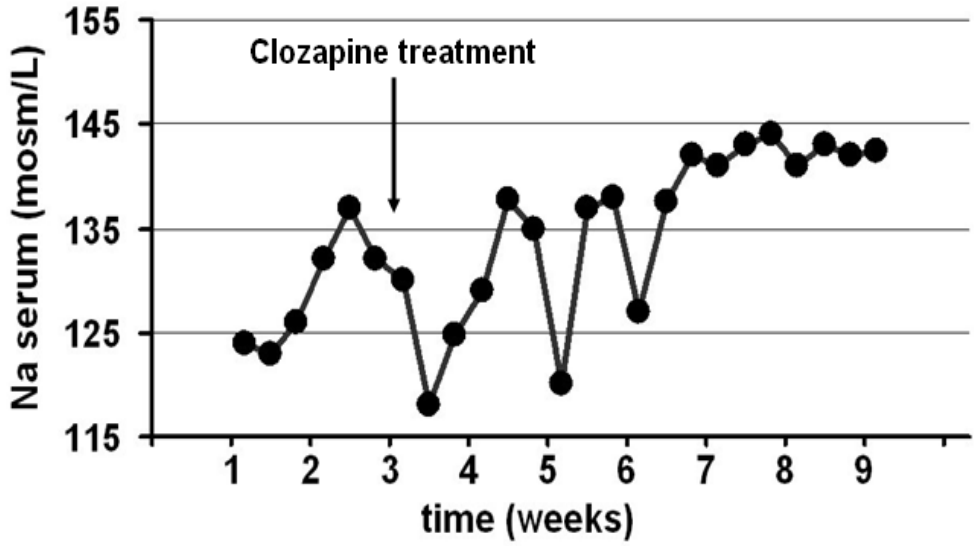

Figure 1. Seriated plasma sodium levels (mosm/L) three weeks before and six weeks after clozapine treatment.

After establishing the psychogenous nature of the observed polydipsia, our patient was started on clozapine (initial dose 12.5 mg), progressively increasing up to 350 mg/day in about 30 days (average clozapine increase: 12.5 mg/day).

We evaluated the clinical and organic picture before and after three weeks of clozapine treatment and observed a remarkable reduction of the fluid-seeking behaviour as well as the improvement of the psychotic "negative" symptomatology.

We also noticed a drastic reduction in fluid intake: the estimated amount after 21 days on clozapine treatment was about 2 litres per day, followed by a reduction of urine excretion up to 1,5 litres per day. Moreover, there was a normalisation of Na serum level, with an average value of 140,9 mmol/L.

3. Discussion

The aetiology of polydipsia is still unclear and it seems to involve behavioural, psychological, and neuroendocrine factors [3]. In the present case, we had no evidence of abnormalities of the ADH/kidney system, so that we hypotized that a central nervous system functional disorder has lead to the psychogenic polydipsia. Some previous studies alert about the risk of a water metabolism dysregulation due to the effect of psyhcotropic drugs (especially neuroleptics) [6]. Actually, only few patients describe thirst as the stimulus for their polydipsia and instead they attribute it to delusions [9] and polydipsia usually improves or at least remits, when the associated psychosis improve either spontaneously or in response to neuroleptic treatment [3]. However, in our case, we observed a patient with no psychotropic medications ongoing since at least one year (even if with the exception of diazepam). As a first therapeutic choice, in line with previous studies [8,10], we used clozapine: however, even using this drug

in monotherapy and at lower average dosages, if compared to previous studies, it resulted to be very effective both in rapidly stabilising the water/sodium metabolism and in treating polydipsia/hyponatremia and improving psychotic features before emerging further complications, which in extreme cases can be fatal.

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