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Comment on: Sailer C, et al. Primary polydipsia in the medical and psychiatric patient: characteristics, complications and therapy

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Primary polydipsia associated with hyponatraemia is a potentially life-threatening though often unrecognised condition, induced by different, poorly understood pathophysiological processes. Accordingly, the recently published review by Sailer and colleagues [1] has inspired me to share a few "psychiatric" comments.

The crucial clinical question is how to prevent the development of acute hyponatraemia and water intoxication. Polydipsia and changes in sodium levels correlate with diurnal weight gains [2]. Thus, prolonged monitoring of diurnal weight gains, at least twice daily, may help identify those patients at an increased risk of developing acute hyponatraemia, which is the main reason for hospitalising them. It is rarely mentioned in the literature that a major obstacle for more exact estimations of hyponatraemia occurrence rates is the observation that patients suffering from polydipsia typically exhibit significant diurnal oscillations in plasma sodium levels. For this reason, a single sodium measurement made on a fasting blood sample proves often inadequate, given that hyponatraemia is characteristically more severe in the afternoon [3].

Sailer and colleagues mentioned malnutrition as a known risk factor for hyponatraemia. Generally speaking, hyponatraemia is often found in conditions associated with lower food intake (e.g., in patients with tumours, alcoholism, anorexia nervosa, very advanced age, or in intensive care units) [4], and is more prevalent in patients on parenteral nutrition [5]. It should thus not be surprising that patients with hyponatraemia often exhibit additional electrolyte abnormalities unrelated to the arginine vasopressin secretion (e.g., hypophosphataemia, hypokalaemia, or hypomagnesaemia) [6]. Hypokalaemia possibly impacts the treatment outcome (increasing risk of demylineation) [7], whereas potassium levels may be associated with the development of polydipsia and hyponatraemia. Serum electrolyte monitoring should thus include potassium level measurements. First of all, patients with long-lasting disorders characteristically suffer from chronic euvolaemic hyponatraemia. Osmotic pressures in the extra- and intracellular compartments must thus be equalized, meaning that "visible" hyponatraemia must co-exist with an intracellular lack of K^+ and (mainly organic) anions. The main sources of organic anions are proteins. In this respect, the hypothesis that polydipsia and its progression to "polydipsia leading to acute or chronic hyponatraemia" may be related to inadequate nutrition has been put forth [4]. Basically, such hyponatraemia is considered a kind of physiological adaptation to the lack of intra-cellular ingredients (mainly K^+ and organic anions).

Besides, patients with euvolaemic hyponatraemia and low plasma osmolality (e.g., 250–260 mmol/l) probably display lower potassium content in cells or organs. Thus, the key question is whether a stable outcome is possible without stabilising intracellular contents and without potassium or protein input (intracellular anions). Furthermore, there is a decades-old finding that in hyponatraemic patients (regardless of the main diagnosis), "a significant rise in serum sodium concentration may follow oral potassium chloride administration" [8]. Potassium deprivation may impair the ability to concentrate urine in both humans and animals. Amlal and colleagues [9] have thus demonstrated a polyuria-polydipsia induced by K^+ deprivation after 24 hours in rats, with significant hypokalaemia after 6 days. Recently, Yang and Cheng [10] reported hyponatraemia in patients with schizophrenia to be associated with poor living conditions. If we assume that subjects belonging to low socioeconomic groups "have no money for meat," then this finding is in accordance with the above-mentioned hypothesis [4]. We reported improvement in hyponatraemia and stabilisation of diurnal weight gain in two schizophrenia patients who were given potassium- and protein-enriched food [4]. Taken together, the hypothesis that, in some patients, hyponatraemia may be nothing other than an easily "measurable outcome" of intracellular changes must still be rigorously tested.

I conclude that: (1) diagnosis of hyponatraemia should be based on multiple serum sodium measurements; (2) long-term follow-up should include measurements of diurnal weight gain; (3) monitoring of serum potassium must likewise be recommended; (4) patients should be informed about adequate nutrition rich in potassium and proteins.

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