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Letter to the Editor

Before Hyponatriemia and Rhabdomyolysis can be Attributed to Psychogenic Polydipsia, Alternative Causes must be Excluded

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Letter to the Editor

We read with interest Minhas *et al*'s article about a 31-year-old male with a history of schizophrenia who was treated with risperidone (5mg/d) and diagnosed with hyponatriemia due to psychogenic polydipsia and subsequent rhabdomyolysis [1]. The patient benefited from the slow substitution of sodium and fluid supplementation [1]. The study is excellent but some points need discussion.

The first point is that the full spectrum of differential causes of polydipsia in addition to psychogenic polydipsia has not been considered and thoroughly excluded. The first differential cause is diabetes insipidus due to pituitary dysfunction with decreased antidiuretic hormone (ADH) secretion with consecutive volume and sodium loss. Pituitary microadenoma, macroadenoma, hypophysitis, or pituitary apoplexy should preferably be ruled out by pituitary MRI with contrast. A cerebral CT scan is not sufficient to assess subtle pituitary pathology. In addition, it is imperative to measure the serum levels of all hormones released by the pituitary gland, releasing factors secreted by the hypothalamus, and aldosterone secreted by the adrenal cortex, also to rule out dipsogenic polydispia. A second differential cause of polydipsia is diabetes. A normal blood sugar level does not rule out diabetes. To rule out diabetes, HbA1c levels and an oral glucose tolerance test must be performed. A third differential cause is certain medications. There is a report that risperidone causes syndrome of inappropriate ADH secretion (SIADH) [2]. Lithium is known that to cause polydipsia. It should be ruled out that the patient suffered from hyperhidrosis.

The second point is that causes of rhabdomyolysis other than hyponatriemia have not been largely ruled out. The primary cause of rhabdomyolysis in the index patient is a seizure. Prior to his admission to the hospital, he had suffered an unwitnessed fall. We should know whether the individual history was positive for epilepsy, alcohol abuse, drug abuse, traumatic brain injury, or birth trauma. Was the family history positive for epilepsy? An increase in creatine-kinase with a delay of several days after a seizure is typical of seizure-associated rhabdomyolysis [3]. Did the patient complain of myalgias after the fall?

The third point is that no EEG was recorded. The patient had a history of an unwitnessed fall, which prompts the exclusion of a seizure. We should know whether the patient reported loss of consciousness before or after the fall, tongue bite, urinary secession, post-fall confusion, or postictual muscle pain.

The fourth point is that alcoholism and illicit drug use has not been adequately ruled out as causes of hyponatriemia. Several of these drugs are known to cause rhabdomyolysis (e.g. cocaine, ecstasy) [4].

The fifth point is that the current medication in addition to risperidone was not reported. Did the patient take medications known to cause rhabdomyolysis? Even risperidone can cause rhabdomyolysis [5]. Has muscle injury from the fall been ruled out as the cause of the increase in creatine-kinase?

A sixth point is that pontine myelinolysis has not been definitively ruled out. Pontine myelinolysis occurs particularly in patients with severe hyponatriemia and is characterised by T2 hyperintesities in the pons on cerebral MRI. Was an MRI performed and was there any evidence of pontine myelinolysis on MRI?

In summary, the excellent study has limitations that should be addressed before final conclusions are drawn. Clarifying the weaknesses would strengthen the conclusions and improve the study. Before hyponatriemia can be attributed to psychogenic polydipsia, alternative causes must be thoroughly ruled out.

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