Life-threatening electrolyte disturbance in a child with urinary tract infection: a case report

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INTRODUCTION

Severe hyperkalaemia is a worrying condition in children population because of its wide differential diagnostic range and potential life-threatening consequences. Hyponatraemia is another dangerous electrolyte disturbance that needs to be recognized early to avoid possible outcomes, such as brain oedema and others. Hyperkalaemia together with other electrolyte and fluid disorders is usually a result of acute syndromes, such as gastrointestinal or adrenal insufficiency.¹ Pseudohypoaldosteronism (PHA) is a rare but important syndrome to recognize that was first described by Rodriguez-Soriano et al. in 1983.² Type-1 PHA is a salt wasting syndrome with constant (also called primary) or transient (also called secondary) receptor resistance to aldosterone. Transient/secondary type-1 PHA is strongly associated with urinary tract infections (UTI), especially when urinary tract abnormalities are present.

CASE REPORT

A 7-month old boy with previously diagnosed hydronephrosis was admitted to paediatric department of Vilnius University Children hospital after finding changes in urine analysis (leucocytosis, positive nitritic reaction) with no fever, persistent poor weight gain, loss of appetite, exacerbation of severe atopic dermatitis. The patient has had 3 fever-less urinary tract infections caused by various atypical agents since the age of two months. All of them have been incompletely treated with antibiotics because of mother's own initiative and patient's allergy to antibacterial drugs, such as *trimethoprim*. Patient's mother noticed poor weight gain that occurred after the first episode of UTI, since which his weight grew only 300 grams per month. He was exclusively breastfed with mother being on a strict diet because of child's skin condition. No additional food was given to the patient due to his refusal to eat. On admission, he was seriously underweight and dehydrated with poor skin condition. Normal vital signs and delayed neurologic development were documented. An enlarged pulsatile fontanelle was visualized. Laboratory workup showed metabolic acidosis, severe hyperkalaemia, hyponatraemia and hypochloraemia, with normal renal function (see Table 1), urine culture analysis was positive for *Staphylococcus spp.* Serum aldosterone level was obtained suspecting hypoadrenalism, showing high elevation of the hormone >1000ng/dL. All other blood biochemistry was normal. As a result, a diagnosis of pseudohypoaldosteronism was made.

Table 1: Clinical biochemistry results at presentation

Laboratory data	Blood level detected
K, mmol/l	8,3
Na, mmol/l	110
Cl, mmol/l	90
pH	7,34
Creatinine, mmol/l	30
Aldosterone, ng/l	>1000

HOSPITAL COURSE

The patient was put on oral nitrofurantoin for UTI. Homeostasis and electrolyte disorders were corrected using both oral and intravenous normal saline together with sorbisterit for hyperkalaemia. Additional food was introduced into his diet after consulting a dietologist. Patient's electrolytes became normal several days after initial treatment (see Table 2). He started gaining weight and was discharged home with additional 500 grams of weight and obviously better skin condition. Persistent leucocyturia was seen in urine analysis so further prophylactic antibacterial treatment was prescribed. Patient had several exacerbations of UTI during the last few months showing the same symptoms (failure to thrive, lethargy, anxiety), and electrolyte and homeostasis disturbances. Urine cultures were positive for *Staphylococcus spp.*, so patient was put on continuous prophylaxis and follow up interventional tests were planned.

Table 2: Clinical biochemistry results at discharge

Laboratory data	Blood level detected
K, mmol/l	6,4
Na, mmol/l	130
Cl, mmol/l	104
pH	7,36
Creatinine, mmol/l	Not obtained
Aldosterone, ng/l	Not obtained

DISCUSSION

Severe hyperkalaemia together with hyponatraemia and metabolic acidosis might be a life-threatening situation which needs urgent actions.⁴ Apart from gastrointestinal, other causes for such electrolyte abnormalities are adrenal failure, either form congenital or acquired factors, especially if changes in mineralocorticoids, specifically aldosterone, are seen. In Lithuania, neonatal screening for congenital adrenal hyperplasia (CAH) was implemented in 2015.⁵ Together with CAH, other common causes of adrenal insufficiency are aldosterone deficiency, pseudohypoaldosteronism (PHA). Pseudohypoaldosteronism is a salt wasting syndrome, which leads to hyperkalaemia, hyponatraemia, and dehydration. Typical finding for diagnosing PHA is highly elevated level of aldosterone due to congenital (primary Type-1), or secondary (transient Type-1) renal tubular resistance to the hormone.⁴ Transient PHA is commonly seen in children with UTI, especially with anatomical abnormalities in urinary tract. We found seven different cases since 2013 analysing analogical conditions of transient pseudohypoaldosteronism in children less than 1 year of age.^{1,6,7} All children, similarly to our patient, were admitted to hospital due to failure to thrive or even weight loss, where severe electrolyte disturbances together with UTI complicating urinary anomalies were found. Our patient was known to have unilateral hydronephrosis in prenatal state. Several articles imply, that urinary tract infection itself even without structural abnormalities could be the cause of aldosterone resistance.^{6,8} Our patient showed no electrolyte disturbances in later follow-up stages, distinguishing the condition from any congenital causes of pseudohypoaldosteronism. He was continued on antibacterial prophylaxis because of constantly positive *Staphylococcus spp.* growth and is now waiting for further radiological work-up.

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