Hypokalemic paraparesis: Presenting feature of previously undiagnosed celiac disease in celiac crisis

Sir,

Celiac crisis is a life-threatening cause of acute diarrhea and multiple metabolic emergencies.[1] Clinically, it is characterized by severe diarrhea, dehydration and metabolic disturbances such as hypokalemia, hypomagnesemia, hypocalcemia, hypoproteinemia, etc. Various precipitating factors identified for the crisis are severe malnutrition, infections, hypoproteinemia, and poor compliance with gluten-free diet, bacterial overgrowth in setting of altered motility in celiac disease and anticholinergic drugs. Hypokalemia, a prominent feature in celiac crisis can lead to muscle weakness. Potassium is needed to repolarize the cell membrane to a resting state after an action potential has passed. Lower potassium levels in the extracellular space cause hyperpolarization of the resting membrane potential. This hyperpolarization is caused by the effect of the altered potassium gradient on resting membrane potential. As a result, a greater than normal stimulus is required for depolarization of the membrane to initiate an action potential leading to muscle paralysis. Paralysis usually starts in the legs and progresses to involve the upper limbs. There are only a few reports of celiac crisis presenting as quadriparesis and that too in adults.[2-4]

We present a 3 years old thriving boy who was admitted with complaints of watery diarrhea and vomiting with dehydration. He was managed with 75 ml/kg RL correction with which dehydration improved, but loose stools persisted though the frequency decreased. On day 2 of admission, the child developed flaccid paraparesis with abdominal distension attributable to hypokalemia. Laboratory reports of electrolytes revealed severe hypokalemia (1.4 meq/L), which was confirmed by an electrocardiogram. Hence, the child was given rapid potassium correction at the rate of 0.3 meq/kg/h for 3 h with subsequently maintenance therapy up to 4 meq/kg/day. Despite potassium replacement paraparesis and hypokalemia did not show improvement. Even with repeated rapid corrections and maintenance dose increased to 6 meq/kg/day hypokalemia, as well as paraparesis persisted (serial potassium values of 1.4, 1.7, 2.3, 2.9, 2.1, 3.2 meq/L) and was accompanied by hyponatremia (serum sodium 124, 131, 134 meq/L) and glycosuria. Review of history elicited intermittent loose stools. At this point of time, the possibility of the celiac crisis was raised. Plasma tissue transglutaminase IgA was returned positive (22.14 U/ml). With a diagnosis of the celiac crisis, the child was started on steroids and gluten-free diet after which he showed significant improvement. A duodenal biopsy in a referral hospital confirmed the diagnosis of celiac disease. The child is currently doing well on a gluten-free diet. Timely diagnosis of such a case is the key to prompt management. Though corticosteroids are the mainstay of management, supportive care, particularly management of electrolyte imbalance is very important.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Access this article online	
Quick Response Code:	Website: www.ijccm.org
	DOI: 10.4103/0972-5229.162478

How to cite this article: Kumar P, Badhan A. Hypokalemic paraparesis: Presenting feature of previously undiagnosed celiac disease in celiac crisis. Indian J Crit Care Med 2015;19:501.