Meetings International
SciTechnol

J Nephrol Ren Dis 2018, Volume: 2
DOI: 10.4172/2576-3962-C1-003

WORLD NEPHROLOGY CONGRESS

June 20-22, 2018 | Paris, France

Gitelman syndrom associated with growth hormone deficiency : A case report

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Introduction: Gitelman syndrom (GS) is an autosomal recessive salt-loosing renal tubulopathy. It is considered as one of the most common inherited tubular disease. The disorder is caused by inactivating mutations in the SLC12A3 gene that encodes the thiazide-sensitive sodium-chloride cotransporter (NCCT) expressed in the distal convoluted tubule of the kidney .The disease is characterized by hypokaliemic metabolic alkalosis and presence of normocalcemic hypocalciuria and hypomagnesemia.. Main symptoms consist in mild muscular weakness and cramps, constipation, abdominal pain and vomiting. Some cases of growth retardation was associated with this syndrom and was related to a growth hormon (GH) deficiency.

Case report: We report a case of a 21-year old girl admitted in our departement in December 2016 for a severe hypokalemia. Clinically, she presented with intense fatigue, weakness with cramps and paralysis . The patient's medical history revealed a low-birth weight, a pubertal delay and a growth failure with GH deficiency discovered at the âge of 14 requiring daily GH injections. She denied any medication usage including laxatives or diuretics. There was two cases of nephropathy disease recorded in her family history .Her blood pressure was 90/50 mmhg and pulse rate 80 beats/min.The cardiovascular examination was normal. She has no peripheral oedema and no oligoanuria. The electrocardiogram didn't show any sign of hypokaliemia .The laboratory findings revealed normal leukocyte,erythrocyte and platelet count. Creatinine was 7mg/L. Serum electolytes concentrations were as follows : sodium 134 mEq/L, potassium 2.4 mEq/L, calcium 95 mg/L, phosphorus 29 mg/L, Magnesium 17 mg/L. Analysis of 24h urine collection showed : an extremely decreased calcium excretion (30 mg/d), increased potassuim excretion (96 mEq/d) Magnesium (40mg/d) ,osmolality (284 mosmol/kg) .The analysis of the arterial blood gasses showed metabolic alkalosis (pH=7.50, Bicarbonate =28 mEq/L). Treatment was a combination of oral and intravenous supplementation of potassiun and magnesium, associated to spironolactone 100mg/d.The plasma potassium and magnesium levels were improved gradually but remain borderline low. Discussion :The diagnosis of (GS) was highly suspected in this patient based on clinical and laboratory findings, the main symtoms was related to the hypokalemia. The growth failure was related to a GH deficiency.Many factors may contribute to growth retardation in Gitelman syndrom such as prematurity,or marked polyuria .Some experimental studies suggested that potassium depletion could have a negative effect on pituitary GH secretion. The pathophysiologic relationship between hypokaliemic tubulopathies and GH deficiency is not clear yet. Most of reported cases had noted a good growth response to GH treatement if the GH deficiency was diagnosed early.GH treatement has also restored magnesium levels in children with (GS).Conclusion : Further clinical research on Gitelman syndrom patients is required for more comprehensive analysis of the relations among growth retardation,GH deficiency andresponse to GH treatement.

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