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Case Report

Clinical approach: hypokalemia paralysis periodic with type 2 diabetes mellitus and insulin

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ABSTRACT

Hypokalemic paralysis periodic (HypoKPP) also known as familial hypokalemic periodic paralysis (FHPP) or primary FHPP is rare case characterized by periodic muscle weakness and estimated prevalence of around 1 in 100.000. Insulin is a treatment for diabetes that also decreases blood potassium levels, therefore, it is necessary to further investigate the causes of hypokalemia. This will be the topic of further discussion if there is a correlation with FHPP. A 32-years-old Indonesian woman with diabetic who has symptoms indicate HypoKPP, and her twin-sister suffered same symptoms is suspected of having FHPP. FHPP is difficult to diagnose due to genetic, other causal factors that must be eliminated, and uncommon references.

Keyword: FHPP, Diabetes, Insulin

INTRODUCTION

Hypokalemia defined as a plasma potassium concentration below than 3.5 mmol/L, whereas low potassium intake, transcellular potassium shift and increased potassium loss lead to hypokalemia. As we know, insulin therapy can cause hypokalemia by redistributing potassium from the extracellular to the intracellular fluid compartment.[1,2] FHPP is caused by mutations in the genes for calcium or sodium ion channel CACNA1S and SCN4.[3] For a definitive diagnosis of FHPP, a genetic test is required, but other methods, such as measuring urine excretion, electrolytes, blood gas analysis, and the trans-tubular potassium gradient (TTKG), can also be used.[4] However, in this case, clinical and A1C result indicate that patient required insulin. FHPP will be the topic of further discussion if there is a correlation with insulin-dependent diabetes.

CASE REPORT

A 32-year-old Indonesian female to the emergency department with complained of feeling weak in both hands and legs, but the heaviest in both legs since early morning. She did not have problem related to nutritional intake. As usual, the history of urinating felt complete and not more concentrated. She did not feel any pain or heat. He denied any history of fever, cough, trauma, diarrhea. She doesn't smoke and her husband doesn't either. She had diabetes mellitus type II (T2DM) with insulin prescription two month ago and she have been hospitalized twice with the same symptoms in six months. The patient's family, her twin-sister found similar signs and symptoms. The physical examination showed muscle strength decrease in the lower and upper extremities, but upper extremities relative strongest than lower extremities. Laboratory examination showed hypokalemia of 1.3 mmol/L, TTKG <2. Blood gas analysis shown pH 7.46, pCO_2 26 mmHg, $CHCO_3$ 19 mmol/L and ABE -5 mmol/L. she had abnormal result HbA1c 9.5%. Complete blood count, thyroid functions, liver function and kidney function were normal. The patient was we diagnosed with HypoKPP caused by suspected FHPP. The patient received KCL of 50 mEq/24 hours, a high potassium diet, lantus 1×4 IU, and novorapid 3×4 IU for T2DM and then the patient's potassium levels were monitored. The results were 1.8

mmol/L (the second day), 2.5 mmol/L (the fourth day). On the fifth day the potassium serum was 3.2 mmol/L, the patient was treated as an outpatient and given KSR therapy of 3×600 mg/24 hours. On the 14th days, the potassium was normal 3.7 mmol/L, she continued medication insulin, KSR of 3×600 mg/24 hours and high potassium diet. She did not complain weakness.

DISCUSSION

FHPP is classified as primary HypoKPP. The patients who got HypoKPP usually have generalized weakness in upper or lower extremities with a normal neurological examination once the episode resolves. As well as primary HypoKPP; FHPP can be suspected if similar symptoms and signs are present in family history. The diagnosis of FHPP is confirmed with genetic mutation analysis.[3,6] The episodes of weakness in HypoKPP triggered by carbohydrate-rich meals, cold, salt intake, alcohol, stress, anesthetic procedures and certain medications such as steroids, insulin and β_2 Agonist. The typical duration of an attacks is a few hours, sometimes minutes to days in some cases. Laboratory results during an attack of weakness are hypokalemia however can be normal in some cases The most common genetic abnormality in HypoKPP is gene mutations encoding the voltage-dependent L-type calcium channel subunit alpha 1s (CACNA1), the voltage gated sodium channel type IV alpha subunit (SCN4A).[6-8] Mechanism of weakness can be happened due decreased ATP-sensitive potassium current, abnormalities in excitation-contraction, altered Ca^{2+} homeostasis resulting from the calcium channel mutation and secondary calcium-sensitive channelopathy.[3,9]

The clinical approach of patient who got HypoKPP one should evaluate whether renal wasting is contributing, a spot urine collection can be informative alternative to the 24 hours urine collection. Blood gas analysis can be measured to help gather information about the case, whether the patient had metabolic alkalosis with high K^+ excretion aim Gitelman syndrome, or Bartter syndrome. This case has not relevant of blood gas analysis result. Alkalosis respiratory can happened due anxiety. Among the etiologies of HypoKPP most cases are the result of FHPP, thyrotoxic periodic paralysis (TPP) and sporadic periodic paralysis (SPP) are 2 common causes in Asia.[4,10]

Treatment of acute attacks includes oral potassium chloride 1 mEq/kg body weight per day (maximum, 200-250 mEq/day). Patient who cannot swallow oral tablets, intravenous potassium chloride may be administered maximum, 20 mEq/h and 200 mEq/d. Prophylaxis against recurrent attacks has been successful with various treatment modalities, including dichlorphenamide and acetazolamide.[6,11] The patient was prescription with Insulin as indicate. As we know insulin make hypokalemia with mechanism shift K^+ intracellular by activating the Na^+-K^+-ATPase pump. Ten units estimated to lower serum potassium by 0.6 to 1.2 mEq/L when administered IV.[12] The limitation of our study is genetic analysis to confirm the diagnosis of FHPP due to family refused any further investigation.

CONCLUSION

FHPP has a clinical of hypokalemia and pattern of inheritance. Acute attacks of weakness resolved with potassium replacement. The clinical approach and management of HypoPP in general is challenging due limitation of source. FHPP with administration of insulin in this case needs to be explored more deeply, so that we know who first made HypoKPP and management approach is needed individualism.

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