中文題目：原發性皮質醛酮過多症與糖分耐受異常 – 病例報告

英文題目：Primary aldosteronism associated impaired glucose metabolism: a unique and potentially curable cause of diabetes mellitus – A Case Report

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Background

Numerous epidemiologic researches have demonstrated the association between impaired glucose metabolism and primary aldosteronism (PA). The prevalence of impaired glucose tolerance in PA can reach up to 50% according to available literatures, making the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus to include PA as a specific form of type 2 DM. Surgical resection of aldosteronoma, which accounts for one-third of PA, can fully reverse patient's impaired glucose metabolism. This attractive finding is worth of our further exploration.

Case Report

Mr. Deng, a 49 year-old retired soldier, was sent to our ER on 97/09/03 due to sudden onset of shortness of breath and bilateral hand numbness while he was watching TV. He denied any chest discomfort and neither did he have fever, productive cough or recent trauma history. No body weight gain or decreased urine output were reported.

Tracing back his history, he had just been put on Metformin 500mg bid since two months ago after five years of fairly well controlled DM on lifestyle intervention. His last HbA1c on 97/05/05 was 8.7%. His hypertension was known for more than 10 years and was under Metoprolol, Amlodipine and Trichlormethiazide control during the time of his visit. Baseline systolic blood pressure was around 135~145 mmHg. Otherwise, no further underlying disease was mentioned.

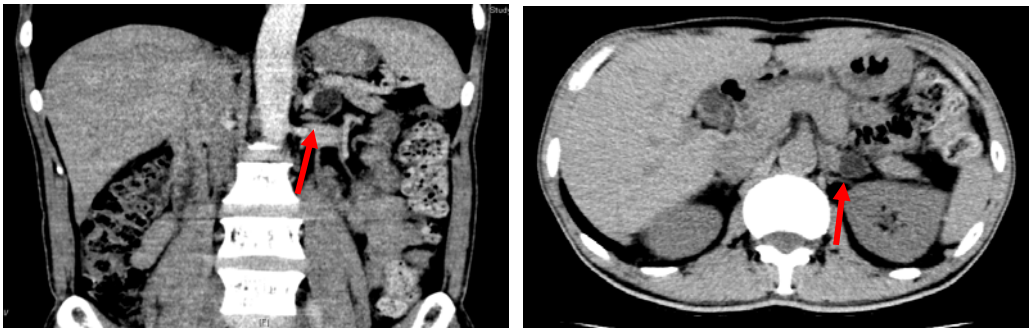
At ER, physical examination was within normal limit and there were no focal neurological signs. EKG disclosed sinus rhythm without any ST segment deviation. Lab work up was only significant for hypokalemia (K: 2.3 mEq/L). He was then admitted to our ward for further diagnostic evaluation because of his unique presentation of hypertension and hypokalemia.

Upon admission, his potassium was 2.6 mEq/L and TTKG was 9. The supine 8 am

plasma aldosterone concentration (PAC) / plasma rennin activity (PRA) was 47.13 (ng/dL) / 0.72 (ng/mL/hr) = 65. Saline infusion test with 2 liters of normal saline given in 4 hours showed non-suppressed PAC (29.5 ng/dL). Abdominal CT was then arranged and revealed one lipid rich hypodense tumor over left adrenal gland. Laparoscopic adrenalectomy was performed on 97/09/23 and the pathology confirmed it to be adrenal cortical adenoma.

His blood pressure and potassium level returned to normal range one month after the operation and no more hypokalemia was present during follow up. In addition, the patient's fasting blood sugar and HbA1c returned to normal range after surgical resection and he no longer required anti-diabetic agents ever since.

Abdominal CT



Discussion

In 1965, J. W. Conn hypothesized that hyperaldosteronism-related hypokalemia may play a key role by impairing insulin secretion via inhibition of K-ATP channel in pancreatic β cell. Nonetheless, the theory can't be fully validated since only half of the group has hypokalemia. Later researches have demonstrated that hyperaldosteronism may precipitate to insulin resistance via various cellular pathways. One of the major hypotheses is that aldosterone per se results in the increase of proteasomal degradation of Insulin Substrate 1 in vascular smooth muscle cell, hence leading to insulin resistance.

Base on these findings, it is reasonable to assume that serum glucose will return to normal level if adequate inhibition of aldosterone can be achieved. Indeed, as in our case, surgical resection of aldosteronoma successfully corrects hyperglycemia. However, pharmacological intervention targeting different sites of rennin-angiotensin-aldosterone system (RAAS) showed incoherent result. The prescription of spironolactone failed to show any benefit in idiopathic hyperaldosteronism group while several double blind randomized controlled trials have shown that ACEI or ARB is able to reduce the incidence of new onset DM. Although the reason of this discrepancy is incompletely understood, one can be sure

that aldosterone may still somewhat play its role.

Our case has demonstrated the reversibility of hyperglycemia after surgical resection of aldosteronoma. Thanks to the advancing screening tools, more and more primary aldosteronism will be detected. If aldosteronoma can be properly identified, these patients will be saved from not only receiving unnecessary medication but also a great deal of medical expense. Although the solution to hyperglycemia in idiopathic hyperaldosteronism has not yet been found, several studies are now underway. We hope that, with the development of newer RAAS blockade agent, we will find the answer in the near future.