

## Chronic Hypoadrenocorticism Protecting Cardiac Structure and Function Potentially: A Case Report

Kang Meini\* and Wang Peixian

Department of Internal Medicine, Tianjin United Family Hospital, Tianjin, China

\*Corresponding author: Kang Meini, Department of Internal Medicine, Tianjin United Family Hospital, Tanjiang Road, Hexi, Tianjin 300221, China, Tel: +862258568500; E-mail: konniedoc@163.com

Rec date: Mar 20, 2017; Acc date: Mar 31, 2017; Pub date: April 03, 2017

Copyright: © 2017 Meini K, et al. This is an open-access article distributed under the terms of the creative commons attribution license, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

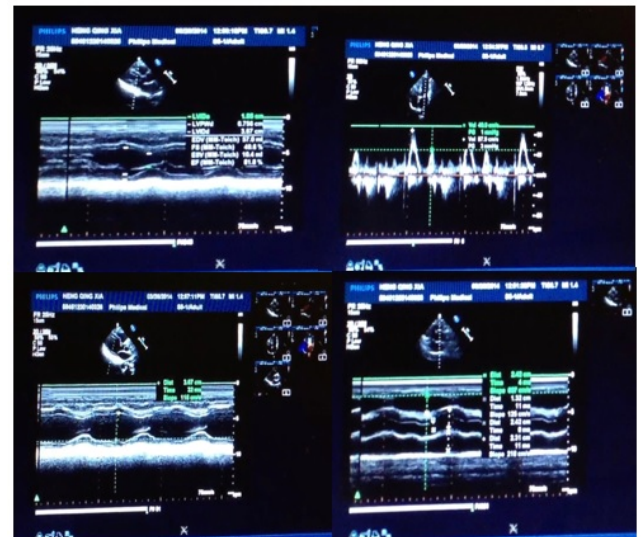
### Abstract

Addison's disease, or primary adrenal insufficiency, results in glucocorticoid and mineralocorticoid deficiency. Classically, patients affected with Addison's disease develop weakness, fatigue, anorexia, weight loss, electrolyte imbalances, hypotension, hypoglycemia and hyperpigmentation of the skin and mucous membranes. Here in this case report, we focus on the interesting finding of good cardiac structure and function of patient with Addison's disease contrast with her senile condition.

**Keywords:** Addison's disease; Cardiac structure; Cardiac function

### Case Report

A 77-year-old woman presented to our cardiology department for a two-week history of intermittent generalized weakness and fatigue. She had past history of tuberculosis of lung, kidney and adrenal glands. She subsequently developed chronic adrenal insufficiency (Addison's disease) and has had this for 51 years. She reported hyperpigmentation of the gingival and mucosal surface of tongue, dorsum of digit joints, palmar creases, nipples, areola of breast, umbilical region and skin of cicatrix as well as fatigue and weight loss. She reported several episodes of hypoglycemia and hypotension (sometimes too low to be measured) which were relieved by fluid therapy. Addison's disease was diagnosed based on the above symptoms and decreased 24-hour urine 17-hydroxysteroid and 17-ketosteroid. Dexamethasone was given (4.5 mg daily as highest dose and 1.5 mg daily as maintenance dose) for replacement therapy until now. Pigmentation of skin and mucosa almost disappeared after dexamethasone therapy. Generalized weakness and fatigue occasionally ever occurred because she decreased the dose of dexamethasone by herself during these 51 years. The above symptoms reappeared two weeks ago, after she caught a cold but she denied decreasing the dose of dexamethasone (1.5 mg daily), no chest pain, shortness of breath and paroxysmal nocturnal dyspnea. The patient was thin with aged appearance. Her blood pressure was 94/56 mmHg. No significant pigmentation of skin and mucosa was observed. The remain of her physical exam was unremarkable. The basal serum cortisol concentration was 180 nmol/L (normal, 275 nmol/L to 555 nmol/L) and a 12-lead electrocardiogram was normal. Echocardiogram revealed aortic dimension was 27 mm; left atrium dimension was 25 mm, right atrium dimension was 29 mm, left ventricle dimension was 48 mm and right was 20 mm during the phase of diastole; left ventricle wall thickness was 10 mm; all cardiac valves showed good motion without reflux or calcification additionally only mild aortic valve fibrosis. Left ventricle ejection fraction was 61.6%, M-mode anterior mitral valve leaflet showed E peak was higher than A peak (Figure 1). Based upon the above findings, her dose of dexamethasone was increased to 2.25 mg daily. Her symptoms subsequently disappeared.



**Figure 1:** M-mode anterior mitral valve leaflet showed E peak was higher than A peak.

### Discussion

Addison's disease (primary adrenal insufficiency) is most frequently caused by autoimmune adrenalitis, and hypothalamic-pituitary tumors represent the most frequent cause of secondary adrenal insufficiency. In the past, adrenal tuberculosis was a more common cause of Addison's disease in China. Currently tuberculosis is a less common cause of chronic adrenal insufficiency due to decreased incidence of severe tuberculosis. In this case, definitive diagnosis is established with clear cause, typical clinical manifestations (mucocutaneous pigmentation, hypoglycemia, hyponatremia, hypovolemia, episodes of hypotension consistent with Addisonian crisis) and effective replacement therapy.

Of interesting note in this case, the structure and function of the patient's heart exhibited characteristics expected to be seen in a patient of much younger age, in contrast with the rest of her physical condition and appearance. Cardiac ultrasound showed normal left atrial and ventricular dimension and ventricular wall thickness. Aortic diastolic wave was normal with good elasticity. All the cardiac valves showed good movement. Both left ventricular ejection fraction and E/A ratio was observed good systolic and diastolic function. Ventricular wall exhibited normal motion which was symmetric and without any movement defect of abnormality. The patient took dexamethasone intermittently during these 51 years according to her history, we speculate that long-term hypotension and low blood volume caused by Addison's diseases possibly reduced cardiac preload and afterload. Additionally, long-term hypoglycemia may have helped avoid cardiovascular damage that can be caused by hyperglycemia [1]. Therefore, hypotension and hypoglycemia may play an important role in protecting heart and blood vessels from the oxidative stress, inflammation and apoptosis which are often seen with hypertension and hyperglycemia [2,3]. The levels of blood pressure and glucose in humans tend to increase with age deviating from the norm specified for the young adults. Such elevation is often considered as a factor contributing to an increase in risks of disease and death. Yashin etc. [4] has done some research in general people that some of the variables of body mass index, diastolic blood pressure, pulse pressure, pulse rate, blood glucose, hematocrit, and serum cholesterol describing individual dynamics of the age-associated changes in physiological indices influence human longevity and exceptional health more substantially than the variables describing physiological state. About the patient with Addison's disease in this case, she also has very stable low blood pressure and glucose, which possibly can help her to keep good healthy

condition especially heart function. Several cases were reported Takotsubo cardiomyopathy in patient with Addison's disease could be reversible [5-7]. It maybe indicates that the cardiac function of patient with Addison's disease is good enough to live through this severe attack.

Patients with Addison's disease don't typically survive to such advanced age and receive evaluation by advanced echocardiography, making observation of the possible protective effect of Addison's disease on the heart. The observation seen in this case may suggest new insights into mechanisms of human longevity and may suggest that further investigation at this phenomenon is warranted.

## References

1. Fiorentino TV, Prioleta A, Zuo P, Folli F (2013) Hyperglycemia-induced oxidative stress and its role in diabetes mellitus related cardiovascular diseases. *Curr Pharm Des* 19: 5695-703.
2. Akash MS, Rehman K, Chen S (2013) Role of inflammatory mechanisms in pathogenesis of type 2 diabetes mellitus. *J Cell Biochem* 114: 525-531.
3. Urbański K, Nowak M, Guzik TJ (2013) Oxidative stress and vascular function. *Postepy Biochem* 59: 424-431.
4. Yashin AI, Arbeev KG, Akushevich I (2010) Dynamic determinants of longevity and exceptional health. *Curr Gerontol Geriatr Res* 381637.
5. Punnam SR, Gourineni N, Gupta V (2010) Takotsubo cardiomyopathy in a patient with Addison disease. *Int J Cardiol* 144: 34-36.
6. Eto K, Koga T, Sakamoto A, Kawazoe N, Sadoshima S, et al. (2000) Adult reversible cardiomyopathy with pituitary adrenal insufficiency caused by empty sella: A case report. *Angiology* 51: 319-323.
7. Derish M, Eckert K, Chin C (1996) Reversible cardiomyopathy in a child with Addison's disease. *Intensive Care Med* 22: 460-463.