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EC CARDIOLOGY
Case Report

Reversible Dilated Cardiomyopathy Secondary to Cushing's Disease

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Abstract

Cushing's syndrome is a debilitating endocrine disorder characterized by excessive cortisol secretion that can affect the cardiovascular system, leading in rare cases to the development of dilated cardiomyopathy (DCM) [1]. A 48-year-old woman with a medical history of uncontrollable type II diabetes mellitus has been diagnosed 1 year ago with non-ischemic DCM, resulting in multiple hospitalization for recurrent decompensated heart failure. A review of the patient's medical history, physical examination and laboratory findings pointed towards the diagnosis of Cushing's disease (CS). Brain MRI revealed a pituitary macro adenoma, which was successfully resected via Trans-sphenoidal approach. After 8 months of follow up, restoration of euglycemia and normalization of the left ventricle function were successfully achieved.

This case clearly demonstrates the importance of recognizing CD as a potential reversible cause of dilated cardiomyopathy.

Keywords: *Cushing's Disease; Dilated Cardiomyopathy; Pituitary Macro Adenoma; Hypercortisolism; Cardiac Dysfunction*

Introduction

Dilated cardiomyopathy (DCM) is a cardiovascular condition that consist of the enlargement of the heart reducing the amount of blood pumped throughout the body. DCM is mostly an idiopathic disease characterized by a progressive and irreversible course that carries poor prognosis and outcome. DCM is mainly presented by fatigue, lower limbs edema and pulmonary complications (shortness of breath), in more severe cases, it can present with chest pain and fainting and in a more complicated cases it can present with heart failure, chronic arrhythmia and heart valve disease. Rarely, a reversible etiology that is amenable to specific therapy is identified [2]. DCM can be caused by genetic variations, cocaine abuse, alcoholism, pregnancy complications, and several infections (Tuberculosis), Chagas disease, Cushing's disease, thyroid disease and autoimmune mechanisms. There are other factors contributes to the development of DCM but are not considered as a main cause such as coronary artery disease and hypertension. Several lab methods can contribute to the diagnosis of DCM including Chest X-Ray, electrocardiogram (ECG or EKG) and cardiac echo-Doppler [3]. DCM treatment include beta-blockers along with diuretics, low sodium diet, blood thinners and in more severe cases; a heart transplant may be required [4].

Cushing's disease is an endocrine disorder characterized by an increased amount of cortisol in the body. It can be caused by an increased exposure to corticoids (Prednisone) or an adrenal gland tumor causing it to secrete excessive amounts. Symptoms ranges from hypertension to morbid obesity, type II diabetes mellitus, muscle and bone weakness, red stretch marks, acne and poor-healing skin. Women can also experience abnormal menstruation cycles and hair growth [5]. Cushing's syndrome treatment can be either by removal of the tumor that causes the adrenal gland to go overdrive or by reducing the amount of medications (corticoids) that are causing the disease until complete withdrawal. As well, CS is one of the disorders that contributes to the development of dilated cardiomyopathy; however, early detection of CS can reduce or eliminate these deleterious effects completely [6].

This case report will discuss and demonstrate how Cushing's syndrome can act as a reversible cause of dilated cardiomyopathy.

Case Report

A 48 year old woman, overweight, former smoker, non-alcoholic, has been diagnosed with uncontrollable type II diabetes mellitus 3 years ago, despite intensive insulin therapy with a Glycohemoglobin level of 12.8%.

A year prior to admission, she started experiencing progressive dyspnea and edema in the lower extremities, increased abdominal girth and proximal muscle weakness. According to these symptoms, she was diagnosed with dilated cardiomyopathy with an ejection fraction (EF) of 35% on her cardiac echo-Doppler (Table 1). Coronary angiography at this stage excluded ischemic heart disease with normal coronary arteries and cardiac MRI revealed a non-ischemic DCM.

	Before	After
LVEDD	61 mm	54 mm
LVESD	49 mm	39 mm
EDV	180 cc	91 cc
IVS	11 mm	11 mm
LPWT	11 mm	10 mm
FS	18%	29%
EF	35%	52%
Color Flow		
Mitral valve	2 - 3/4	1/4
Aortic Valve	0/4	0/4
Pulmonary Valve	1 - 2/4	0/4
Tricuspid Valve	1/4	1/4

Table 1: *Measurement 2D+M mode.*

Since then, she was hospitalized several times for decompensated heart failure despite optimal medical treatment at home, associated with a progressive unintentional weight gain.

Patient admitted to our hospital with acute decompensated heart failure as usual, echocardiographic examination showed severe left ventricle function impairment with ejection fraction of 35%, moderate mitral regurgitation with mild dilatation of LV cavity (Figure 1).

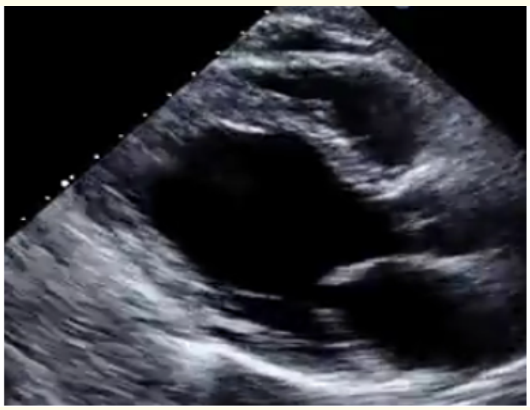

Figure 1: *Echocardiography of a parasternal long axis view showing dilated left ventricle.*

Laboratory tests upon admission are summarized in table 2.

	Results	Normal range
Hemoglobin	11.9 g/dl	12 - 15.5 g/dl
White blood count	7 x 10*9/L	4.5 - 11 x 10*9 / L
Creatinine	0.9 mg/dl	0.6 - 1.2 mg/dl
Sodium	139 mEq/L	135 - 145 mEq/L
Potassium	4 mEq/L	3.5 - 5 mEq/L
TSH	1.2 m IU/L	0.4 - 4 m IU/ L
HBA1C	10.90%	< 6%
Pro BNP	15400 pg/dl	< 450 pg/ml
Trop	0.5ng/ml	0 - 0.4 ng/ml

Table 2

On the basis of the current findings: new onset of uncontrolled diabetes mellitus, unintentional weight gain, proximal myopathy, truncal fat distribution and unexplained dilated cardiomyopathy. Cushing's syndrome diagnosis was highly suspected.

Therefore, initial laboratory testing to confirm excessive glucocorticoid production demonstrated a very high blood cortisol level associated with high ACTH level (Table 3).

	Results	Normal range
Urine free cortisol	385 mcg/24h	< 45 mcg/24h
8 am cortisol level	71 mcg /dl	10 - 20 mcg/dl
Overnight 1 mg DST	11.5 mcg/dl	< 1.8 mcg/dl
Late-night salivary cortisol	17.9 nmol/l	< 4 nmol/l
Basal ACTH level	120 pg/ml	10 - 55 pg/ml

Table 3

Magnetic-resonance imaging of the pituitary gland revealed a macro adenoma measuring 11 mm in diameter (Figure 2).

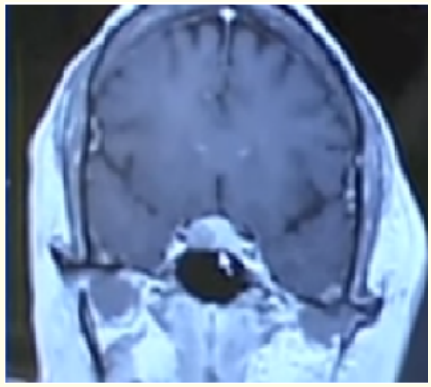

Figure 2: *Coronal T2 weighted magnetic resonance image showing a macro adenoma (white arrow).*

As such, a diagnosis of ACTH-dependent Cushing's disease was made.

Administration of adrenolytic drugs (ketoconazole and metyrapone) was initiated for the preparation before surgery.

Following reduction in her cortisol level and stabilization of her cardiac status, trans-sphenoidal adenomectomy was performed successfully.

6 to 8 months post-surgery, clinical features of Cushing's have almost resolved and cardiac function has been improved significantly (repeat echocardiography showed EF (45 - 50%)).

Discussion

Studies examining the relationship between hypercortisolism and cardiac dysfunction suggests that excess cortisol is contributory to cardiac remodeling and dilated cardiomyopathy. The pathophysiology of cardiac remodeling involves complex mechanisms including activation of neuro-hormonal factors, alpha adrenergic and renin-angiotensin-aldosterone systems [7].

It is clear that diagnosing Cushing's disease in heart failure patient requires a very high index of suspicion and careful evaluation of the patient's history and clinical findings [8].

Despite the paucity of cases reporting, the association between dilated cardiomyopathy and Cushing's disease in the literature demonstrated remarkable improvement in cardiac function post-surgical resection [9].

Conclusion

With this case report, we want to emphasize that physicians *must* at all times, be alert to recognize Cushing's disease as a reversible cause of dilated cardiomyopathy that can be successfully treated with surgical therapy.

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