



Case Report

Reversible Stress Induced Cardiomyopathy in Adrenal Insufficiency

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Citation: Salman M, AlKhoury J, AlSherooqi R, Assar W. Reversible Stress Induced Cardiomyopathy in Adrenal Insufficiency (2018)

Clinical Cardiology and Cardiovascular 1: 1-3

Received: Feb 21, 2018

Accepted: Apr 07, 2018

Published: Apr 12, 2018

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Abstract

It's evident in the literature that Addisonian crisis is tightly linked with cardiovascular manifestations, with very few cases reporting an associated between adrenal insufficiency and reversible cardiomyopathy. We report a case of a 57 years old lady who presented with an acute onset of adrenal insufficiency then developed severe heart failure that resolved post hydrocortisone administration. The clinical presentation, course of management and outcome will be discussed.

Introduction

Addison's disease, also known as primary adrenal insufficiency, is an endocrinological condition with a deficiency in the production of glucocorticoids and mineralocorticoids from the adrenal cortex [1]. It's a very life-threatening disease that is often overlooked due to the unspecific range of symptoms patients usually present with, thus a prompt diagnosis is essential.

There have been multiple etiologies attributed to the development of Addison's disease, including infections, drugs and even genetic factors. However, the most common cause remains to be an autoimmune destruction of the adrenal cortex [2].

Despite the well-established relationship between cardiovascular complications and Addisonian crisis, it's thought to be limited mainly to hypovolemic hypotension, syncope and arrhythmias, resolving with volume repletion and glucocorticoids administration [3].

The presentation of cardiomyopathy and heart failure in patients who have no previous history of adrenal or cardiac abnormalities is not very common and only 6 cases are reported as such in the literature [4-10]. However, in all cases cardiomyopathy seemed to have been improved post glucocorticoids.

We report a case of a 57 years old lady who presented with an acute onset of adrenal insufficiency then developed severe HF that resolved on serial echocardiography post hydrocortisone administration. The clinical presentation, course of management and outcome will be discussed.

Case

A 57 years old female not known to have any medical complaints, was brought in to the emergency department with a few hours history of agitation, restlessness and disturbed level of consciousness. She was completely alert the night before until the morning of her admission when she was confused, unable to recognize her family and very agitated. There was no documented loss of consciousness or any seizure attacks. There were also no complaints of any chest symptoms or abdominal pain.

The patient's only past history was chronic back pain on analgesia. On initial assessment the patient had a GCS of 8, then was very agitated and sedated by the emergency department with midazolam. She was hypotensive with a blood pressure of 90/60, afebrile, maintaining saturation on a face mask attached to 8L oxygen. No abnormal skin pigmentations were noted, and no obvious dysmorphic features. Her initial biochemical panel showed a hyponatremia of 121mmol/l and hyperkalemia of 8.15 mmol/l, as well as a sight elevated Creatinine of 117 umol/l (Table 1). Her VBG showed deteriorating mixed acidosis and the patient was awaiting intubation after the CT brain.

The patient also underwent a cardiology review while in the emergency department, her initial ECG showed normal sinus rhythm with down-sloping ST elevation in V1-V2 attributed to hyperkalemia with no reciprocal changes. A repeat ECG after hyperkalemia correction showed a descend in the down-sloping ST elevation to isoelectric line, T wave inversion in anterior leads (**Figure 1**). Her bedside troponin was negative and her bedside ECHO showed an ejection fraction of 60% with no regional wall motion abnormalities.

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Investigations	On presentation	At discharge	In outpatient follow-up
Na	121 mmol/L	140 mmol/L	139 mmol/L
K	8.15 mmol/L	3.75 mmol/L	3.92 mmol/L
Creatinine	117 umol/L	60 umol/L	78.6 umol/L
Troponin I	0.0 ng/ml	0.004 ng/ml	0.0 ng/ml
CKMB	0.84 ng/ml	-	0.34 ng/ml
TSH	6.7 uIU/ml	-	1.43 uIU/ml
Cortisol (AM)	On Day 6 0.82 ug/dl		53.3 ug/dl
ACTH	On Day 6 1250 pg/ml		9 pg/ml

Table 1: Investigation readings.



Figure 1A



Figure 1B

Figure 1: ECG after hyperkalemia correction showed a descend in the down-sloping ST elevation to isoelectric line, T wave inversion in anterior leads



Figure 2: ECG showed poor R wave progression with T wave inversion in anterior leads.

The patient was later on intubated, admitted to the intensive care unit and started on hydrocortisone (200mg IV stat then infusion) during her hospital stay in the intensive care unit, the patient was started on continuous renal replacement therapy (CRRT), and her hydrocortisone infusion was converted to boluses of 100mg IV every 8 hours after an endocrine review. Her blood pressure was maintained on the hydrocortisone and intravenous fluids. CT brain ruled out any brain

lesions, and a CT abdomen was also done which ruled out any adrenal masses. She remained hyponatremic, on mechanical ventilation and sedated on Propofol. TSH was 6.7 uIU/ml while LH and FSH were both normal (**Table 1**). On her second admission day, a departmental echocardiography showed severe hypokinesia of mid to apical LV segments and an overall systolic function of 20%. A further cardiology review was requested as the Troponin was slightly elevated (highest of 5 ng/L), ECG showed poor R wave progression with T wave inversion in anterior leads (**Figure 2**). An impression of stress induced cardiomyopathy secondary to Addisonian crisis was made and the patient was kept on full anti-ischemic measures.

On Day 5, the patient was extubated, and started self-ventilation on minimal O2 support, her clinical condition started to improve and her BP remained adequate, thus she was transferred to the medical ward.

On Day 6, her hydrocortisone was stopped for 24 hours before cortisol levels were sent. The patient's BP was maintained above 90 mmhg systolic off the hydrocortisone, cortisol levels came back as 0.82 ug/dl, ACTH was 1250 pg/ml and the patient was started on hydrocortisone tablets (10mg in the morning and 5mg in the evening). A repeat echocardiography was done which showed an improvement in the ejection fraction, with hypokinetic mid anteroseptum, mid inferoseptum and entire apex with an overall systolic function of 40%-45%.

The patient was discharged on day 12 with further outpatient review in both endocrinology and cardiology clinics. Follow up echocardiography studies were done which demonstrated improvement in the ejection fraction with the latest one showing an ejection fraction of 60% with well-preserved contractility. The patient also underwent further cardiology assessments as an outpatient, including a CT coronary angiography and a dobutamine stress test which were all normal.

Discussion

Primary adrenal insufficiency is an endocrinological condition with a decrease in the production of glucocorticoids and mineralocorticoids from the adrenal cortex, most commonly secondary to an autoimmune destruction of the adrenal cortex. Cardiovascular manifestations of Addison's mainly involve hypotension due to volume depletion, syncope and arrhythmias secondary to electrolytes imbalance.

There have been few cases in the literature demonstrating some sort of a relationship between Addisonian crisis and reversible cardiomyopathy that usually resolved after a stress-dose of glucocorticoids, and this case is an evident demonstration of that. The cause of left ventricular dysfunction in Takotsubo cardiomyopathy is still unknown, It may be caused by a catecholamine-mediated myocardial stunning secondary to either a myocardial ischemic injury or epicardial spasm with metabolic injury. Catecholamines can directly damage the myocardium. In Takotsubo cardiomyopathy, physical stress is considered as a major predisposing factor [11,12].

Conclusion

This case demonstrated a fairly uncommon presentation of stress induced cardiomyopathy secondary to adrenal insufficiency, which resolved after high doses of glucocorticoids. This is still a field of mystery and interest for many in the medical field, more research is needed to demonstrate the mechanism of such phenomenon and methods to tackle it.

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