Case Report
Journal of National Heart & Lung Society Nepal
Vol-3. No. 1, Jan-Jun 2024 DOI:10.3126/jnhls.v3i1.63818 ISSN: 2091-0657 (Online & Print)

Adrenal Insufficiency Presenting as Pulseless Ventricular Tachycardia

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Received: 5th May, 2024 Accepted: 27th May, 2024 Published: 10th June, 2024

ABSTRACT

Adrenal insufficiency (AI) is a severe sickness induced by adrenal gland or hypothalamic-pituitary-adrenal (HPA) axis dysfunction. It can induce a variety of symptoms, the most serious of which are potentially fatal adrenal crises. This case report describes a clinical scenario involving a 42-year-old male patient who had previously had brain tumor surgery. The patient had presented with fever and gastrointestinal symptoms. He developed pulseless ventricular tachycardia on day two of admission. Patient was promptly treated with electrical cardioversion. Workup revealed QT prolongation on ECG and adrenal insufficiency. Both the patient's arrhythmias and QT prolongation on ECG responded to intravenous hydrocortisone.

Key words: adrenal crisis; adrenal insufficiency; QT prolongation; ventricular tachycardia.

INTRODUCTION

Adrenal insufficiency (AI) is a dangerous illness characterized by adrenal gland pathology, hypothalamus or pituitary pathology, or exogenous glucocorticoid treatment leading to suppression of the hypothalamic-pituitary-adrenal (HPA) axis. AI has been linked to increased morbidity and mortality, as well as a decrease in quality of life. Furthermore, autoimmune adrenalitis, the most common cause of PAI, has been linked to a number of other autoimmune illnesses. Untreated AI can cause persistent exhaustion, weight loss, and infection. Failure to treat a significant sickness or infection might lead to a potentially fatal adrenal crisis. It is vital to acquire an accurate diagnosis in a timely manner in order to carry out appropriate therapy aimed at correcting chronic illness, avoiding acute crises, and restoring quality of life. The physiology of the HPA axis aids in understanding the clinical presentation of AI and serves as the foundation for biochemical testing that leads to AI diagnosis.[1] Adrenal insufficiency is attributed to numerous cardiac manifestations viz. cardiomyopathy, CHF, pericarditis, QT prolongation and cardiac arrest.[2] A potentially fatal cardiac arrhythmia is pulseless ventricular tachycardia that happens when coordinated ventricular contractions are replaced by very fast but inefficient contractions resulting in insufficient organ perfusion and heart failure. Pulseless ventricular tachycardia is a medical emergency. [3] Survival requires immediate emergency treatment and defibrillation. Prevention begins with heart-healthy habits and the careful management of pre-existing heart conditions. Ventricular tachycardia (VT) might resolve in 30 seconds or last longer (sustained V-tach or VT).[4] Laboratory tests play an important role in confirming the diagnosis. The ACTH stimulation test is a common diagnostic tool, which measures the adrenal gland's response to synthetic ACTH to assess cortisol production. Basal cortisol levels, along with ACTH levels, can provide further insight into adrenal function. Additionally, imaging studies such as CT or MRI scans may be used to identify any structural abnormalities in the adrenal glands or pituitary/hypothalamic areas, especially in cases of suspected secondary adrenal insufficiency.[5-7] Management of VT depends on patient's condition. Hemodynamically unstable patients may require immediate electrical cardioversion to restore normal rhythm. In cases where the patient is stable, antiarrhythmic drugs such as amiodarone, lidocaine, or procainamide can be used to suppress VT episodes. In cases of recurrent or refractory VT, catheter ablation may be considered. Implantable cardioverter-defibrillators (ICDs) are also important

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in the management of VT. These device prevent sudden cardiac death.[8-10]

CASE REPORT

We are presenting a case of 42-year-old male patient who was admitted from emergency department with the complaints of fever for one day, loose stool, and few episodes of vomiting for one day. Poor appetite was described since last two days. He had brain tumor operated 27 years back. His medical history in last months was complicated with headache, needle pain and myalgia. On examination, he was oriented to time, place and person and was cooperative. His vitals were: Pulse of 110 beats per minute. Blood pressure was systolic 88 and diastolic 50mmHg. He was febrile with documented temperature of 101.8 F. His respiratory rate was 20 breaths per minute. GCS was E3V5M6. Periphery was cold and clammy. His height was 149 cm and weight was 40 kg. On systemic examination: lung fields were clear, first heart sound and second heard sound were heard and there were no appreciable added sounds. Abdomen was soft and non-tender. There were no organomegaly. Bowel sounds were normal. On the basis of his history, examination, and preliminary investigations, he was accepted and monitored.

Table 1. Laboratory parameters of the patient

Parameters	Reference range	Patients value
Hb %	12-16 gm/dl	9.5 ↓
PCV (Hematocrit)	36-54%	28.8 ↓
WBC	4000-11000/cumm	6120
RBC	4.5-5.5 mil/mm^3	3.56
Neutrophil	40-70%	44.8
Lymphocytes	20-40%	49.1 ↑
Eosinophil	1-6%	0.3 ↓
Basophil	0-1%	0.2
Platelet	150000- 400000/cumm	159000
Na	135-150 mmol/L	133 ↓
K	3.5-5.5 mmol/L	3.58
Urea	15-45 mg/dl	23.44
Creatinine	0.4-1.4 mg/dl	1.02
Bilirubin Total	0.1-0.4 mg/dl	1.92 ↑
Bilirubin Direct	0.89-1.76 mg/dl	0.89
AST	40 IU/L	274.23 ↑
ALT	45 IU/L	111.38 ↑
Blood sugar (R)	70-140 mg/dl	72
Cortisol	4.30-22.40µg/dL	0.79 ↓

Patient was improving till next morning of admission when he developed ventricular tachycardia. (Figure 1) Patient became hemodynamically unstable and immediately was treated with DC shock (biphasic 200J).

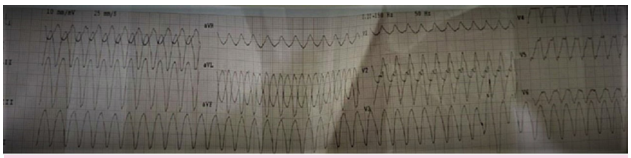

Figure 1. ECG of the patient showing ventricular tachycardia.

Patient reverted back to sinus rhythm. On further work up for this event his ECG revealed QT prolongation. Electrolytes and blood gas analysis were within normal limits. Serum cortisol was reduced. It was 0.79ugm/dL (normal range:4.30-22.40µg/dL). (Table1) On back ground of his brain tumor surgery, diagnosis of adrenal insufficiency was made. Patient was given intravenous hydrocortisone at the dose of 100mg tds. Subsequently QT prolongation reverted and patient did not develop ventricular arrhythmias in next 24 hours. Patient opted to get discharged on leave against medical advice.

DISCUSSION

In the current case, a 42-year-old male patient presented with medical history of worrying symptoms like fever, nausea and vomiting. Gastrointestinal symptoms with shock are established presentations in case of AI. Adrenal insufficiency (AI) and pulseless ventricular tachycardia stand out as important clinical issues in this instance. Ventricular tachycardia in the setting of adrenal insufficiency can arise from multiple mechanisms, including hypokalemia, electrolyte imbalances, inadequate stress response, adrenal crisis, and metabolic acidosis. These factors can individually or in combination lead to the electrical instability of the heart, predisposing patients to VT.[11-15] In our patient there was adrenal insufficiency and stress. ECG of our patient had prolonged QT (QTc-580). Prolonged QTc was present in a study done by Nishizawa S et also.[16] Pulseless VT was promptly treated with DC Cardioversion. DC Cardioversion

is most effective management strategy in pulseless VT.[8-10] Diagnosis of adrenal insufficiency was made on the background of history of his surgery and lab report in the form of decreased cortisol level. After administration of intravenous hydrocortisone, QT on ECG was corrected. Similar improvement in QT prolongation in patient of adrenal insufficiency was observed in study done by Komuro, J., Kaneko, M., Ueda, K. et al.[17]

CONCLUSION

Adrenal insufficiency should be considered an important differential diagnosis in patients presenting with GI symptoms and shock complicated by Ventricular Tachycardia. Prompt diagnosis and electrical cardioversion followed by corticosteroid replacement is lifesaving in such cases.

Conflict of interest: None

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Citation: Hussain A, Murarka R, Regmi SR, Dhital B, Shrestha A, Chaudhary R, Shrestha R, Shrestha S, Kafle A. Adrenal Insufficiency Presenting as Pulseless Ventricular Tachycardia. JNHLS. 2024; 3(1):49-52.