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Case Report

Intersecting emergencies: acute adrenal crisis in a patient with acute coronary syndrome

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ABSTRACT

Acute adrenal crisis is a rare but life-threatening endocrine emergency that can present with non-specific symptoms and may mimic or coexist with other critical conditions such as acute coronary syndrome (ACS). We report a case of 48-year-old male with no known comorbidities who presented with left-sided chest pain, restlessness, and gastrointestinal symptoms. He was initially diagnosed with Non-ST Elevation Myocardial Infarction (NSTEMI) and managed for Acute coronary syndrome (ACS) and dyselectrolytemia. Upon referral to our tertiary care centre, the patient was found to have complete heart block, severe hyperkalemia, hyponatremia, and hypotension. Despite correction of hyperkalemia and initiation of standard therapy for ACS with cardiogenic shock, the patient's condition failed to improve. Further evaluation revealed critically low serum cortisol (1.9 µg/dl) and elevated plasma adrenocorticotropic hormone (ACTH-630 pg/ml), confirming a diagnosis of primary adrenal insufficiency (Addison's disease) with acute adrenal crisis. Initiation of intravenous hydrocortisone and fludrocortisone led to rapid clinical stabilization, normalization of electrolytes, and improvement in hemodynamic status of the patient. The patient was discharged in stable condition on maintenance corticosteroid therapy. This case highlights the importance of considering adrenal insufficiency in patients with persistent hypotension and dyselectrolytemia, particularly when presenting atypically in the context of acute coronary syndrome.

Keywords: Acute adrenal crisis, Acute coronary syndrome, Dyselectrolytemia, Hypotension

INTRODUCTION

Acute adrenal crisis, also known as Addisonian crisis, is a life-threatening condition resulting from a sudden and severe deficiency of adrenal corticosteroids (glucocorticoids and mineralocorticoids) most commonly in the setting of previously undiagnosed or poorly managed primary adrenal insufficiency (Addison's disease).[1,2] It can be precipitated by physiological stressors such as infection, trauma, surgery, or cardiovascular events, and is often under recognized due to its nonspecific clinical presentation, which includes hypotension, fatigue, electrolyte disturbances, and gastrointestinal symptoms.[1-3] Acute coronary syndrome (ACS) encompasses a spectrum of conditions, including unstable angina and myocardial infarction, that results from myocardial ischemia due to sudden reduction in coronary blood flow.[4] The physiological stress induced by ACS- including hypotension, systemic inflammation and catecholamine surge–can precipitate adrenal decompensation in susceptible individual.[5] In such a setting, the signs and symptoms of adrenal crisis such as hypotension, hyponatremia, hyperkalemia, chest and abdominal discomfort and altered mental status may overlap with or be masked by clinical presentation of ACS, complicating the diagnostic process. The interplay between endocrine and cardiovascular systems is complex. In patients with adrenal insufficiency, the stress of a myocardial infarction can precipitate an adrenal crisis due to the increased demand for cortisol during acute illness, compounded by the absence of physiological corticosteroid response.[5] Additionally, hyperkalemia from mineralocorticoid deficiency can lead to life-threatening arrhythmias,

including complete heart block.[6] Authors report a rare case of a 48-year-old male with no known comorbidities who presented with features suggestive of ACS and was later found to be in acute adrenal crisis. This case underscores the importance of considering adrenal insufficiency in patients with unexplained hypotension, electrolyte disturbances, and poor response to standard ACS management.

CASE REPORT

48-year-old male with no known comorbidities presented with complaints of non-radiating left-sided chest pain for the past 8 days. The chest pain was associated with restlessness, shortness of breath and diaphoresis. He also reported abdominal discomfort, vomiting and reduced appetite for the last 5 days. The patient was initially admitted to a private clinic, where he was diagnosed with acute coronary syndrome (ACS) and dyselectrolytemia. An electrocardiogram (ECG) done at the referring centre was suggestive of Non-ST Elevation Myocardial Infarction (NSTEMI). Initial treatment was initiated. After 3 days, he was referred to Parul Sevashram Hospital for further management. On arrival, the patient was conscious and oriented. Vital signs were as follows: pulse rate: 38 beats/min in right radial artery in supine position, BP: 90/60 mmHg, respiratory rate: 18/min, temp: 98.4 F and SpO2- 96% on room air. Physical examination showed acutely ill appearing cachexic patient with shrunken eyes, conjunctival pallor and dark discoloration of skin. There was no associated icterus and limb edema. ECG showed complete heart block (Figure 1).

Table 1: Serial laboratory investigations.

	Day 1	Day 2	Day 3	Day 4	Day 6	Day 8	Day 9	Day 12	Day 14
Hemoglobin (g/dl)	10.8	11.4	11.0			10.7	10.9	10.1	12.2
WBC count (cells/mm^3)	6500	8240	5200			5400	4590	4690	4780
Platelet count (×10^6 cells/^{3})	3.73	3.85	2.44			2.01	2.11	2.64	2.86
Sr. Sodium (mmol/l)	112	118	122	125	125	126	129	133	136
Sr. Potassium (mmol/l)	7.4	4.4	4.5	4.8	4.6	3.6	3.7	3.7	3.8
Sr. Chloride (mmol/l)	96	98	91	93	96			94	93
BUN (mg/dl)	108	78	55						40
Creatinine (mg/dl)	2.2	0.9	1.4			0.7	1.2		0.7

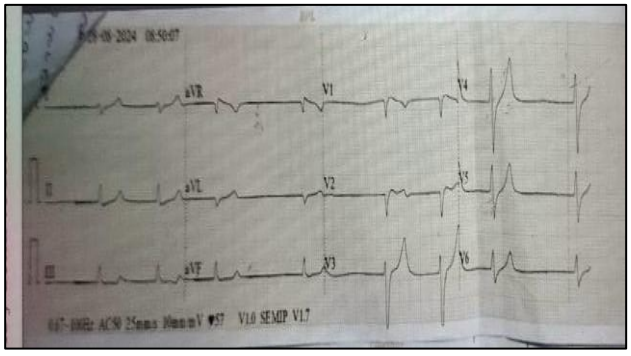

Figure 1: Electrocardiogram showing complete heart block.

Routine blood investigations were sent, and the patient was transferred to the intensive care unit (ICU) for further management. In the ICU, the patient's condition rapidly deteriorated. He became drowsy and hypotensive with a blood pressure of 70/40 mmHg. Intravenous fluids and noradrenaline infusion were initiated. He was managed as a case of ACS with cardiogenic shock and treatment was started accordingly. Initial laboratory investigations revealed Hb-10.8 g/dl, white cell count 6500/mm^3 and platelets 373000/mm^3. Serum sodium 112 mmol/l, serum potassium- 7.4 mmol/l, Urea-108 mg/dl, serum creatinine -2.2 mg/dl (Table 1). Troponin I was positive (24.6 ng/l) and NT-pro BNP was 647 ng/l (Normal range: 12-133 ng/l). Liver function test, HbA1C and Lipid Profile were within normal limits. Hyponatremia correction was initiated promptly with 3% sodium chloride infusion. Hyperkalemia correction was initiated with 10 ml of 10% calcium gluconate IV, Glucose insulin infusion and oral potassium-binding resin. Complete heart block was attributed to hyperkalemia. Urgent nephrologist consult was taken and Hemodialysis was initiated. Post Dialysis, serum potassium levels were 4.4 mmol/l, serum creatinine-0.9 mg/dl and heart rate gradually increased to 68-70 beats/min. 2D Echo cardiology showed regional wall motion abnormality, apical anterior wall hypokinesia with ejection fraction of 45%.

Despite giving adequate treatment with high doses of inotropes, vasopressors, IV fluids and reversing the heart block patient's blood pressure remained low and electrolyte imbalance persisted. This raised suspicion of an underlying chronic endocrine disorder. An 8 AM serum cortisol level was sent and found to be 1.9 µg/dl (normal: 3.7–19.4 µg/dl), which was significantly low. Serum ACTH level was found to be 630 pg/ml (reference: 5–52 pg/ml), suggesting a diagnosis of primary adrenal insufficiency (Addison's disease). Empirical intravenous hydrocortisone 100 mg stat followed by 50 mg QID and oral fludrocortisone 0.1 mg OD were started. Within 48 hours of initiating corticosteroid therapy, the patient's condition improved markedly suggesting that the patient

was in acute adrenal crisis. Inotropes were gradually tapered, and his serum electrolytes normalized. (Table 1) A CT abdomen with adrenal imaging revealed no adrenal mass or hemorrhage. Coronary Angiogram was planned for a later date. The patient was discharged on Day 14 in a stable condition with oral corticosteroid replacement therapy.

DISCUSSION

This case illustrates a rare and diagnostically challenging presentation of acute adrenal crisis (Addisonian crisis) precipitated by acute coronary syndrome (ACS) in an otherwise healthy middle-aged male. The coexistence of these two critical conditions-each capable of producing overlapping cardiovascular and systemic manifestations-can obscure timely diagnosis and complicate management.

Adrenal insufficiency, particularly the primary form (Addison's disease), is a rare endocrine disorder characterized by deficient production of cortisol and aldosterone. While chronic adrenal insufficiency may present with fatigue, hyperpigmentation, hypotension, and gastrointestinal disturbances, acute adrenal crisis manifests abruptly with profound hypotension, shock, severe hyponatremia, hyperkalemia, and even altered sensorium.[3,7] In our patient, persistent hypotension, bradycardia, and refractory electrolyte imbalance despite treatment for ACS with cardiogenic shock prompted further endocrine evaluation.

Cardiovascular stress, such as myocardial infarction or surgery, is a known precipitating factor for adrenal crisis in undiagnosed cases of Addison's disease.[8] During acute illness, the hypothalamic-pituitary-adrenal (HPA) axis is expected to respond by increasing cortisol production to maintain vascular tone and metabolic homeostasis. In adrenal insufficiency, this response fails, leading to circulatory collapse and electrolyte derangements, especially hyponatremia and hyperkalemia due to aldosterone deficiency.[6]

Significant electrocardiographic disruptions can be seen in untreated adrenal insufficiency partially due to hyperkalemia and partially due to glucocorticoid deficit, which prolongs the duration of the cardiac action potential resulting in conduction abnormalities. Even rare, isolated cardiac rhythm anomalies have been documented in adrenal insufficiency, including cardiac arrest, torsade de pointes (QT prolongation), and sinus bradycardia Hyperkalemia in our patient led to complete heart block, a rare but recognized cardiac manifestation of adrenal crisis.[9,10]

Although initial management focused on ACS-evidenced by positive troponin I, ECG changes, and regional wall motion abnormalities, persistent hypotension despite correction of hyperkalemia and improvement in heart rate highlighted a non-cardiac etiology for the shock. This diagnostic pivot was critical. The low cortisol level of 1.9 µg/dl and elevated ACTH (630 pg/ml) confirmed primary adrenal insufficiency, aligning with typical Addisonian crisis findings.

Notably, dark skin discoloration and cachexia had been present but overlooked due to the acute cardiac symptoms. Prompt initiation of stress-dose corticosteroid therapy with intravenous hydrocortisone and fludrocortisone led to a dramatic improvement in hemodynamics and resolution of dyselectrolytemia. This reinforces the lifesaving role of early steroid replacement in suspected adrenal crisis, even before confirmatory testing is complete.[2] The absence of adrenal mass or hemorrhage on CT imaging pointed toward an autoimmune or idiopathic cause, the most common etiology of Addison's disease in both developed and developing countries.[7,8]

This case underscores the importance of maintaining a high index of suspicion for adrenal insufficiency in any patient with refractory shock, persistent electrolyte imbalance, or poor response to standard interventions, especially in the context of a known stressor like ACS.

CONCLUSION

This case highlights the diagnostic complexity when acute adrenal crisis presents in the setting of acute coronary syndrome. Clinicians must maintain a high index of suspicion for adrenal insufficiency in any patient with refractory shock, persistent electrolyte imbalance, or poor response to standard interventions, especially in the context of a known stressor like ACS. Early recognition and prompt initiation of corticosteroid therapy can be lifesaving.

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