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Case Report

Biochemistry Section

Acute Decompensated Heart Failure Precipitated by Atrial Fibrillation with Rapid Ventricular Response in Severe Biventricular Systolic Dysfunction: A Case Report

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ABSTRACT

Acute Decompensated Heart Failure (ADHF) is a life-threatening clinical syndrome characterised by abrupt worsening of cardiac function and haemodynamic instability. The prevalence rate of ADHF in India is higher than in Western countries, around almost 1%, with younger age groups being more affected. A 65-year-old male presented to the emergency department with progressively worsening breathlessness, intermittent chest discomfort, and dizziness. Clinical examination revealed an irregular tachyarrhythmia, bilateral basal pulmonary crepitations, and mild peripheral oedema suggestive of cardiac decompensation. Electrocardiography demonstrated Atrial Fibrillation (AF) with rapid ventricular response, while transthoracic echocardiography revealed dilated cardiac chambers with severe global hypokinesia and markedly reduced Left Ventricular Ejection Fraction (LVEF) of approximately 20%. Laboratory evaluation showed markedly elevated N-terminal pro-B-type natriuretic peptide levels, acute kidney injury, and significant elevation of hepatic transaminases compatible with congestive hepatopathy with possible hypoxic hepatic injury. The patient was managed with intravenous rate control with amiodarone for AF, diuretic therapy, anticoagulation, fluid restriction, and initiation of guideline-directed therapy for heart failure with reduced ejection fraction. Gradual clinical and biochemical improvement occurred during hospitalisation. This case highlights AF with rapid ventricular response as a potentially reversible trigger of acute cardiac decompensation. Early recognition and prompt, targeted management of such precipitating factors are crucial to improving clinical outcomes and reducing morbidity and mortality in patients with heart failure.

Keywords: Acute kidney injury, Cardiomyopathy, Cardiorenal syndrome, Hepatitis, Multiple organ failure

CASE REPORT

A 65-year-old male presented to the emergency department with progressively worsening breathlessness over a period of approximately two weeks. The dyspnoea was initially exertional but gradually progressed to occur at minimal activity and at rest during the last 2-3 days prior to admission. His symptoms were associated with intermittent chest discomfort, productive cough, and occasional dizziness. During this period, his functional capacity had significantly declined, corresponding to New York Heart Association (NYHA) functional class III-IV [1].

The patient also reported a recent episode of symptomatic anaemia for which he had received a blood transfusion at a peripheral centre approximately 7-10 days prior to presentation. There was no documented history of similar acute decompensation in the past. He was a former bidi smoker who had quit nearly eight years earlier and denied alcohol consumption or tobacco chewing.

On arrival, the patient appeared dyspnoeic and anxious. Vital signs revealed blood pressure of 150/90 mmHg, an irregularly irregular pulse rate ranging between 150 and 160 beats per minute, respiratory rate of 29 breaths per minute, and oxygen saturation of 92% on room air. Cardiovascular examination demonstrated an irregularly irregular rhythm without additional murmurs or gallop sounds. Respiratory system examination revealed bilateral basal crepitations suggestive of pulmonary congestion. Mild bilateral pedal oedema was present.

On admission, haematological evaluation revealed anaemia with a haemoglobin of 8.4 g/dL, leucocytosis (total count 13,800/µL), and a raised Red Cell Distribution Width (RDW) of 15.3%, with indices suggesting a normocytic picture [Table/Fig-1]. Coagulation studies demonstrated a prolonged prothrombin time of 23.9 seconds, indicating hepatic synthetic dysfunction. Liver function tests showed markedly elevated transaminases - Aspartate Transaminase (AST) 396 U/L and Alanine Aminotransferase (ALT) 473 U/L - with a total bilirubin of 3.4 mg/dL, consistent with congestive hepatopathy. By Day 2, transaminases escalated dramatically (AST 6032 U/L, ALT 3320 U/L) and bilirubin rose to 7.2 mg/dL, reflecting ischaemic hepatitis superimposed on passive hepatic congestion. The total leucocyte count rose markedly to 24,600/µL on Day 2, attributed to stress leucocytosis in the setting of haemodynamic compromise and systemic hypoperfusion; concurrent infective aetiology could not be fully excluded and was considered clinically. The Day 2 MCV value of 61.3 fL was identified as a probable transcription error, as a decline of this magnitude within 24 hours is physiologically implausible given the 120-day lifespan of circulating erythrocytes; this value has been excluded from the report pending verification of the original laboratory record. Renal function tests showed progressive azotaemia with urea rising from 53 to 98 mg/dL and creatinine from 1.0 to 1.5 mg/dL, suggesting cardiorenal syndrome. Urine analysis demonstrated 2+ proteinuria and glycosuria (+1) to (+4), without any prior clinical history of diabetes mellitus, with microscopic haematuria (4-6 RBCs/hpf) and pus cells 1–2/hpf on urine microscopy. Urine sugar escalated from +1 on Day 1 to 4+ on Day 2 in the absence of any prior history of diabetes mellitus, attributed to stress hyperglycaemia secondary to catecholamine surge and systemic inflammatory response; random blood glucose was monitored and formal diabetes evaluation was planned for outpatient follow-up. The cardiac biomarker NT-proBNP was

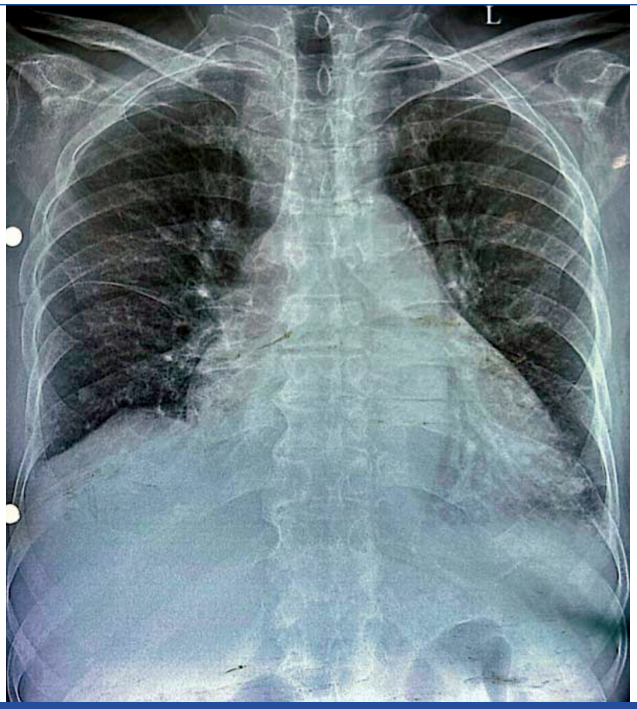
Parameters	Day 1	Day 2	Reference range
Haematology			
Haemoglobin (Hb)	8.4 g/dL	10.3 g/dL	M: 13-17 g/dL
			F: 12-15 g/dL
Total Count (TC)	13,800 /µL	24,600 /µL	4,000-11,000 /µL
MCV	83.1 fL	61.3 fL	80-100 fL
MCH	26.0 pg	18.3 pg	27-32 pg
MCHC	31.2 g/dL	29.8 g/dL	32-36 g/dL
RDW	15.3%	25.0%	11.5-14.5%
RBC count	3.22 ×10⁶/µL	3.17 ×10⁶/µL	M: 4.5-5.5×10⁶/µL
			F: 4.0-5.0×10⁶/µL
Coagulation			
Prothrombin Time (PT)	23.9 sec	—	11-13.5 sec
Liver function tests			
Bilirubin (Total)	3.4 mg/dL	7.2 mg/dL	0.2-1.2 mg/dL
SGOT/AST	396 U/L	6032 U/L	10-40 U/L
SGPT/ALT	473 U/L	3320 U/L	7-56 U/L
Renal function tests			
Urea	53 mg/dL	98 mg/dL	15-45 mg/dL
Creatinine	1.0 mg/dL	1.5 mg/dL	0.6-1.2 mg/dL
Electrolytes			
Sodium (Na^+)	135 mEq/L	131 mEq/L	135-145 mEq/L
Potassium (K^+)	4.4 mEq/L	4.0 mEq/L	3.5-5.0 mEq/L
Chloride (Cl^-)	98 mEq/L	101 mEq/L	98-107 mEq/L
Urine analysis			
Albumin (urine)	+2	—	Negative
Sugar (urine)	+1	4+	Negative
Pus cells (urine microscopy)	1-2/hpf	1-2/hpf	0-5 cells/hpf
RBC (urine)	4-6/hpf	—	0-2/hpf

[Table/Fig-1]: Blood investigations.
• Abbreviations: Hb: Haemoglobin; TC: Total (WBC) count; MCV: Mean corpuscular volume; MCH: Mean corpuscular haemoglobin; MCHC: Mean corpuscular haemoglobin concentration; RDW: Red cell distribution width; PT: Prothrombin time; AST/SGOT: Aspartate aminotransferase; ALT/SGPT: Alanine aminotransferase; hpf : high-power field; M: Male; F: Female

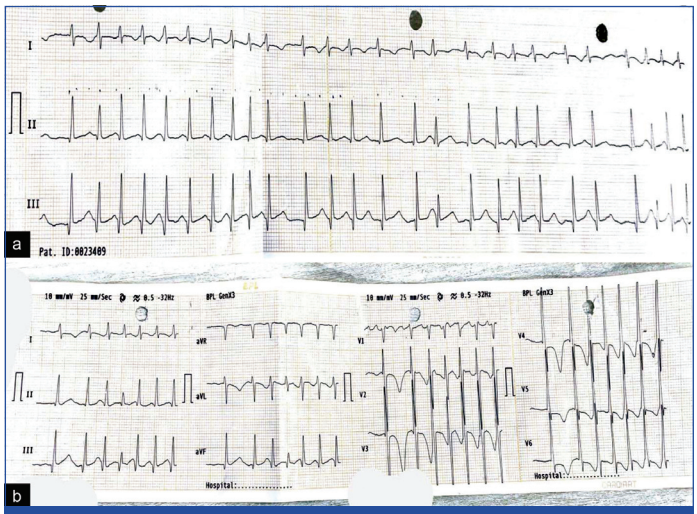
markedly elevated at 15,563 pg/mL, reflecting severe ventricular wall stress and significantly elevated cardiac filling pressures, in keeping with advanced decompensated heart failure. Chest radiograph confirmed cardiomegaly, pulmonary venous congestion, and bilateral pleural effusions [Table/Fig-2]. Electrocardiography showed AF with rapid ventricular response at approximately 150 bpm [Table/Fig-3]. Thoracic and abdominal ultrasonography corroborated bilateral pleural effusions and features of hepatic congestion. Two-dimensional echocardiography revealed severely dilated cardiac chambers with global hypokinesia, a critically reduced LVEF of 20%, severe mitral and Tricuspid Regurgitation (TR), and a Pulmonary Artery Systolic Pressure (PASP) of 48 mmHg, confirming severe biventricular systolic dysfunction with secondary pulmonary hypertension [Table/Fig-4].

The constellation of findings supported a diagnosis of ADHF precipitated by uncontrolled AF, complicated by cardiorenal syndrome and congestive hepatopathy. Differential diagnoses considered included acute coronary syndrome, myocarditis, and sepsis-associated cardiomyopathy.

Management was directed towards haemodynamic stabilisation, control of the arrhythmia, and treatment of volume overload. Intravenous amiodarone 150 mg in 100 mL normal saline over 30 minutes, followed by 900 mg in 50 mL normal saline at 3.6 mL/hour for six hours, then 1.8 mL/hour for the remaining 18 hours, was administered to achieve ventricular rate control in AF. Intravenous furosemide 80 mg was given immediately, followed by 40 mg thrice daily, to relieve pulmonary and systemic congestion. Intravenous heparin 5000 IU was initiated as a parenteral anticoagulant to



[Table/Fig-2]: Chest X-ray.
X-ray findings included cardiomegaly, pulmonary venous congestion, and bilateral pleural effusion.



[Table/Fig-3]: Electrocardiogram (ECG). ECG demonstrated Atrial Fibrillation (AF) with rapid ventricular response (~150 bpm); a) ECG findings; b) Different leads.

Parameter	Finding
Left Ventricular Internal Diameter (Diastole)	Dilated
Left Ventricular Internal Diameter (Systole)	Dilated
Left Ventricular Ejection Fraction (LVEF)	~20% (severely reduced)
Wall Motion Abnormality	Severe global hypokinesia
Mitral Valve	Severe Mitral Regurgitation (MR)
Tricuspid Valve	Severe Tricuspid Regurgitation (TR)
Right Ventricle	Dilated with dysfunction
Inferior Vena Cava (IVC)	Dilated; reduced inspiratory collapse
Pulmonary Artery Systolic Pressure (PASP)	48 mmHg (elevated)
Pericardial Effusion	Absent
Overall Impression	Severe biventricular systolic dysfunction with secondary pulmonary hypertension

[Table/Fig-4]: Summary of 2D echocardiographic findings. A 2D echocardiography demonstrated dilated cardiac chambers, severe global hypokinesia, LVEF of 20%, severe Mitral Regurgitation (MR), severe TR, and PASP of 48 mmHg.
Abnormal values are highlighted. LVEF: Left ventricular ejection fraction; MR: Mitral regurgitation; TR: Tricuspid regurgitation; PASP: Pulmonary artery systolic pressure; IVC: Inferior vena cava
Note: Original echocardiographic images could not be retrieved from the institutional archive. The above table summarises all quantitative and qualitative parameters documented in the echocardiography report at the time of admission and are presented in lieu of the image as per standard JCDR case report guidelines

reduce the risk of thromboembolic complications, and fluid intake was restricted to assist in volume management.

After initial stabilisation, guideline-directed pharmacological therapy for heart failure with reduced ejection fraction was introduced. Sacubitril/valsartan was initiated at 24/26 mg twice daily. Spironolactone was initiated at 25 mg once daily, with close monitoring of serum potassium and renal function given the presence of cardiorenal syndrome. Dapagliflozin was commenced at 10 mg once daily, with amiodarone tablet 100 mg twice daily. Additional medications included atorvastatin 40 mg once daily at night as part of lipid-lowering and cardiovascular risk management, and aspirin 75 mg once daily for antiplatelet therapy. Renal and hepatic biochemical parameters - including serum creatinine, blood urea, liver function tests, and serum electrolytes - were monitored regularly throughout treatment to assess organ function and therapeutic response.

With adequate rate control and decongestive therapy, the patient demonstrated progressive clinical improvement. The heart rate stabilised between 60 to 70 beats per minute with controlled ventricular rate by approximately five days of hospitalisation, and respiratory distress gradually subsided. Laboratory investigations showed stabilisation of renal function and a declining trend in hepatic transaminase levels.

The patient was discharged in a haemodynamically stable state with advice regarding strict adherence to prescribed medications. Regular cardiology follow-up was recommended, along with continued monitoring of renal and hepatic function and periodic reassessment of ventricular function. The patient was last followed up after 10 days with AF with controlled ventricular rate, haemodynamically stable with adequate urine output.

DISCUSSION

The ADHF is one of the leading causes of emergency hospitalisation worldwide and is increasingly recognised as a multisystem syndrome characterised by haemodynamic instability, neurohormonal activation, systemic inflammation, and venous congestion resulting in end-organ dysfunction [2,3]. AF with rapid ventricular response is a well-established precipitating factor for acute decompensation, as tachyarrhythmia reduces diastolic filling time, impairs stroke volume, and increases myocardial oxygen demand, particularly in patients with pre-existing systolic dysfunction [4].

The present case illustrates this complex pathophysiological interaction in a 65-year-old male who presented with ADHF precipitated by AF with rapid ventricular response in the setting of severe biventricular systolic dysfunction with a LVEF of 20%. The clinical course was further complicated by cardiorenal syndrome, congestive hepatopathy, and markedly elevated natriuretic peptide levels, reflecting advanced haemodynamic compromise. Rate control was achieved using a structured intravenous amiodarone infusion protocol in accordance with contemporary European Society of Cardiology recommendations for acute AF management [5]. Progressive ventricular rate stabilisation contributed significantly to haemodynamic improvement and symptom resolution.

Renal dysfunction observed in this patient was consistent with cardiorenal syndrome type 1. Ronco C et al., proposed a five-subtype classification emphasising that acute deterioration in cardiac function may lead to renal impairment independent of intrinsic renal pathology [6]. Furthermore, Damman K et al., demonstrated that elevated central venous pressure is a major determinant of worsening renal function in acute heart failure [7]. Improvement in renal parameters following aggressive decongestion supports this haemodynamic mechanism.

Marked elevation of hepatic transaminases reflected combined congestive hepatopathy and hypoxic hepatitis. Ischemic hepatic injury secondary to systemic hypoperfusion has been well described [8], while chronic venous congestion contributes to hepatocellular dysfunction and cholestasis [9]. Abnormal liver function tests have also been shown to independently predict adverse outcomes in acute heart failure, as demonstrated in the PROTECT trial [10].

Significantly elevated NT-proBNP levels quantified the severity of myocardial wall stress and haemodynamic burden. Natriuretic peptide testing has been consistently shown to improve diagnostic accuracy and risk stratification in acute heart failure [11,12]. In addition, haemodynamic profiling studies have demonstrated that patients presenting with combined congestion and hypoperfusion represent the highest-risk subgroup with significantly increased morbidity and mortality [13].

Following haemodynamic stabilisation, guideline-directed quadruple neurohormonal therapy was initiated. Sacubitril/valsartan has been shown to significantly reduce cardiovascular mortality and heart failure hospitalisation compared with angiotensin-converting enzyme inhibition in the PARADIGM-HF trial [14]. Similarly, sodium-glucose cotransporter-2 inhibition with dapagliflozin reduces worsening heart failure events and improves survival irrespective of diabetic status [15]. Mineralocorticoid receptor antagonism with spironolactone has also been shown to reduce all-cause mortality in severe systolic heart failure [16].

Anticoagulation was initiated to mitigate AF-related thromboembolic risk, and rate control was preferred over rhythm control given the presence of advanced structural heart disease. The favourable clinical outcome - including resolution of respiratory distress, stabilisation of ventricular rate, improvement in biochemical markers, and successful initiation of disease-modifying therapy - highlights the importance of early recognition of tachyarrhythmia-triggered decompensation and implementation of a structured multidisciplinary management strategy in advanced ADHF.

CONCLUSION(S)

This case illustrates AF with rapid ventricular response as an important reversible precipitating factor of ADHF in patients with advanced systolic dysfunction. Early identification of tachyarrhythmia-induced haemodynamic compromise and prompt initiation of appropriate therapy are essential to prevent multiorgan dysfunction and improve clinical outcomes.

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