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Case Report

Cardiogenic shock without hypotension in acute severe primary mitral regurgitation: a case report

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Abstract

A 60-year-old gentleman who presented with features of end-organ hypoperfusion despite initial hypertension was promptly diagnosed with cardiogenic shock following evidence of hyperlactatemia on biochemistry and left ventricular global hypokinesis with severe mitral regurgitation on transthoracic echocardiogram. He responded well to dobutamine and later underwent definitive surgical mitral valve replacement.

INTRODUCTION

Cardiogenic shock (CS) is a syndrome of inadequate tissue perfusion, secondary to reduced cardiac output [1]. CS in the absence of hypotension is relatively uncommon, accounting for ∼5% of cases in acute ischaemic cardiogenic shock trials, though they represent a cohort with poor outcomes [2–5]. We present a demonstrative case of non-ischaemic CS with normotension.

HISTORY OF CASE PRESENTATION

A sixty-year-old gentleman presented to the Emergency Department (ED) with a one-month history of malaise, anorexia, and 10 kg weight loss. On presentation to the ED his heart rate was 105 bpm, blood pressure was 167/117 mm Hg, and he required 4 L of oxygen via nasal prongs to maintain saturations >92%. Examination revealed an end-expiratory wheeze, right upper quadrant tenderness, peripheral fluid overload with pitting oedema to the knees and mottling of the lower limbs.

PAST MEDICAL HISTORY

Medical history was significant for peripheral vascular disease with iliofemoral balloon angioplasty, and gastroesophageal reflux disease. The patient took no regular medications, smoked approximately thirty cigarettes per day, and consumed five to six standard alcoholic beverages daily. He reported occasional cannabis use.

INVESTIGATIONS

An electrocardiogram (ECG) demonstrated sinus tachycardia with left atrial enlargement and a right bundle branch block (Fig. 1).

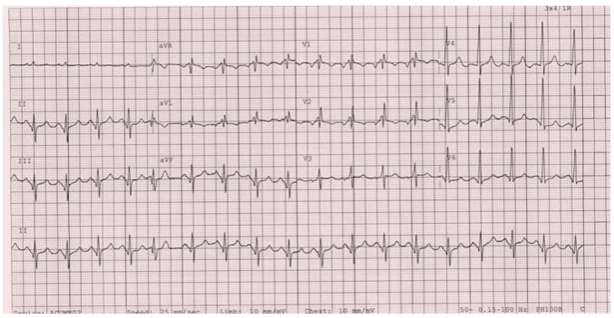

Figure 1. ECG on presentation. ECG showing sinus tachycardia, left atrial enlargement and right bundle branch block.

Initial bloods were grossly abnormal (Table 1). There was an AKI with hyperkalaemia, deranged liver function tests (LFTs) with an elevated international normalised ratio (INR), and a raised troponin and N-terminal-pro B-natriuretic peptide (BNP). A VBG revealed a partially compensated metabolic acidosis due to significant lactic acidaemia, with pH 7.21 (7.35–7.45), PCO_2 27 mm Hg (40–50 mm Hg), bicarbonate 10 mmol/L (22–32 mmol/L), and lactate 16 mmol/L (<2.0 mmol/L).

TTE showed a mildly dilated left ventricle with severe global hypokinesis; LVEF 25%–30%, severe eccentric anteriorly directed MR, severe pulmonary hypertension (right ventricular systolic pressure 66.5 mm Hg) and moderate TR (Fig. 2). An abdominal ultrasound revealed hepatic venous dilation consistent with congestive hepatopathy secondary to cardiac failure. There was also evidence of Child's Pugh B hepatic steatosis. Viral screens for myocarditis and hepatitis were negative. Cardiac magnetic resonance imaging (MRI) later showed a severely dilated LV with

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Table 1. Bloods on presentation

Investigation	Value (Reference Range)
Biochemistry	Sodium 131 mmol/L (135–145 mmol/L) Potassium 5.9 mmol/L (3.5–5.2 mmol/L) Creatinine 266 μmol/L (60–110 μmol/L) eGFR 21 ml/min/1.73m^2 (>60 ml/min/1.73m^2) Bilirubin 90 μmol/L (<20 μmol/L) Deranged LFTs, transaminases AST > ALT (ratio > 1.5)
Full blood count	White cell count 12.5 × 10^9/L (4.0–11 × 10^9/L) Neutrophils 11.2 × 10^9/L (2.0–8.0 × 10^9/L) Haemoglobin 133 g/L (130–180 g/L) Platelets 97 × 10^9/L (150–450 × 10^9/L)
Inflammatory markers	C-reactive protein (CRP) 42 mg/L (<5 mg/L)
Coagulation profile	INR 2.3
Troponin	711 ng/L → 842 ng/L → 1425 ng/L (<26 ng/L)
N-terminal-pro BNP	125 428 ng/L (<260 ng/L)

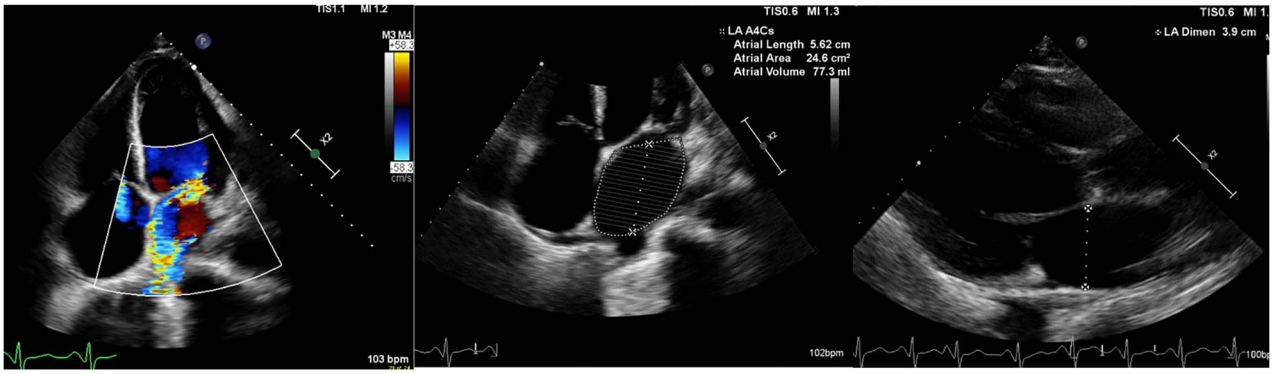

Figure 2. Transthoracic Echocardiogram. (**A**) Colour doppler four chamber apical view demonstrating severe mitral regurgitation and dilated right atrium and ventricle. (**B**) Four chamber apical view showing evidence of left atrial dilatation. (**C**) Parasternal long axis view demonstrating increased left atrium length, and right ventricular dilatation.

eccentric hypertrophy, moderate LV systolic dysfunction with diffuse hypokinesis plus segmental areas of wall thinning, akinesis, and scarring. The right ventricle was also dilated with severe systolic dysfunction and hypertrophy.

MANAGEMENT

He was promptly admitted to the Intensive Care Unit (ICU). Initial treatment included intravenous frusemide, bisoprolol, aspirin, and slow intravenous fluids. Over the next 24 h, his lactate improved from 16.1 mmol/L to 1.1 mmol/L and he was stepped down to the ward. Whilst awaiting improvement in renal function to allow for safe coronary angiography, the patient developed confusion and oliguria, accompanied by a rise in serum lactate to 14.7 mmol/L. Despite these clear clinical and biochemical signs of hypoperfusion, the patient maintained an SBP >90 mm Hg (Fig. 3). He was readmitted to ICU and commenced on a dobutamine infusion at 10 mcg/kg/min, with a subsequent improvement in cognition, renal function, and reduction in lactate to 3.2 mmol/L over the coming hours. Repeat echocardiography demonstrated slight improvement in his LVEF to 35%–40%, likely due to offloading with vasoactive substances, though, no change in the severity of MR. He subsequently underwent coronary angiography which demonstrated normal coronary arteries.

Given the severity of MR, and presumed consequent LV dysfunction and pulmonary hypertension, collectively resulting in marked reduced cardiac output and recurrent CS, the patient underwent surgical bioprosthetic mitral valve replacement and tricuspid valve annuloplasty with opportunistic left atrial appendage closure. The posterior mitral valve cusp biopsy exhibited myxoid and degenerative changes without evidence of infective endocarditis. He was extubated two days post-procedure. His post-operative course was complicated by ventilator associated pneumonia, and consequent atrial flutter, but was successfully medically managed. He was stepped down from ICU one-week post-op and recovered well on the ward before eventual discharge home with cardiac rehab.

FOLLOW-UP

The patient received 4-week post-op follow up from his Cardiothoracic Surgeon, and ongoing review by his Cardiologist and Renal Physician. Fig. 4 summarises key events throughout the patient's clinical course.

DISCUSSION

This case report describes a patient with overt clinical and biochemical signs of organ hypoperfusion secondary to cardiogenic shock, evidenced by clinical response to inotropes and ultimately, mitral valve replacement, despite normotension throughout admission. It serves as a reminder that marked peripheral vasoconstriction, either intrinsically or as a result of vasoactive therapies, may preserve peripheral blood pressures but does not

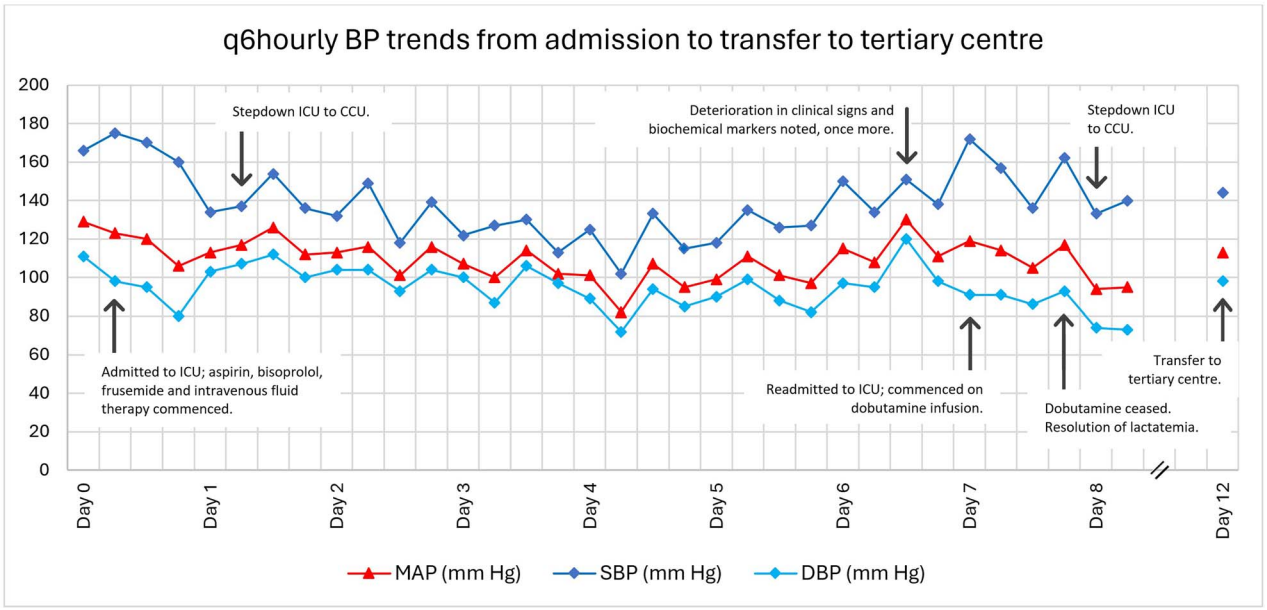

Figure 3. q6hourly BP trends from admission to transfer to tertiary centre. Abbreviations—MAP: mean arterial pressure; SBP: systolic blood pressure; DBP: diastolic blood pressure; ICU: intensive care unit; CCU: coronary care unit.

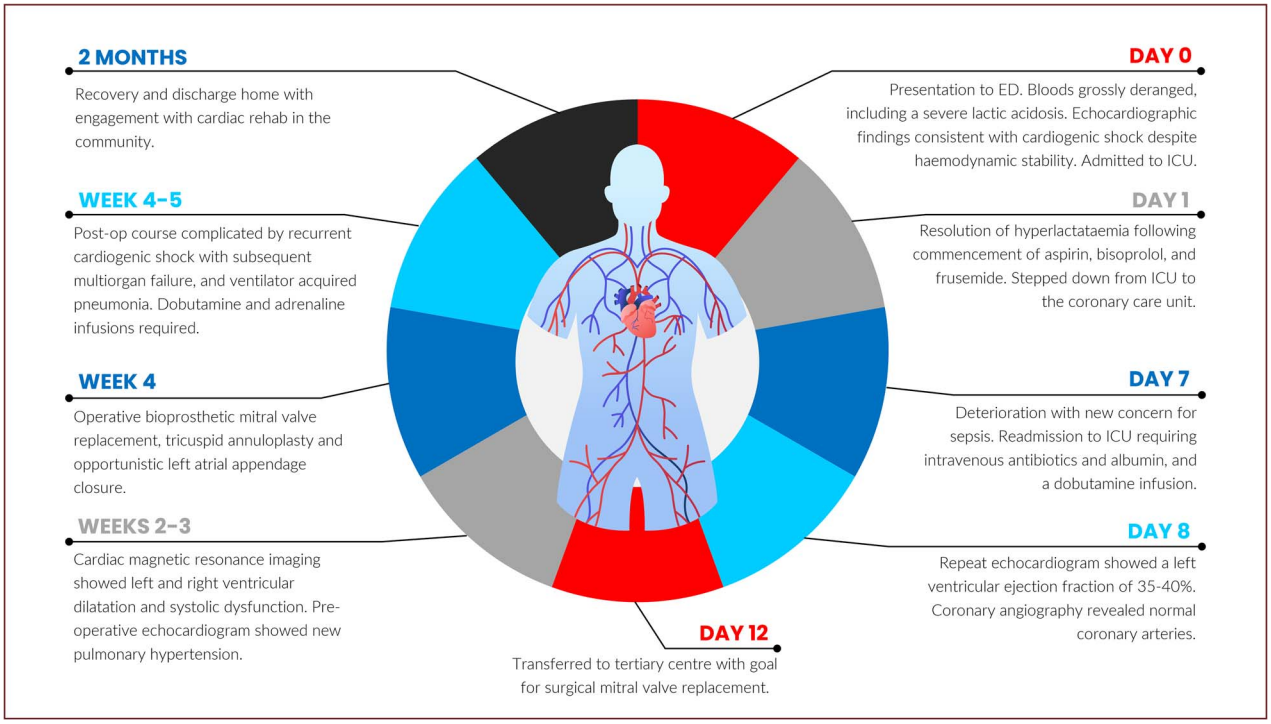

Figure 4. Timeline of Key Events. Abbreviations—ICU: Intensive Care Unit; CCU: coronary care unit.

necessarily ensure adequate tissue perfusion [3]. This report challenges the conventional understanding of shock to include hypotension which may otherwise cause a delay in identifying deteriorating patients and may preclude such from receiving timely treatment.

Further exploration of haemodynamic state

Left ventricular outflow tract (LVOT) diameter and LVOT velocity time integral parameters from the patient's echocardiograms permitted the calculation of cardiac output and subsequently, systemic vascular resistance (SVR) [6, 7]. At initial presentation, there was considerably reduced cardiac output of 3.19 L/min (5–6 L/min) with an elevated SVR of 2731 dynes/sec/cm^{-5} (700–1500 dynes/sec/cm^{-5}). In the more common ischaemic aetiology of CS, an appropriately raised SVR helps to maintain tissue perfusion by compensating for the lower cardiac output [8, 9]. However, in acute MR, it unfavourably increases the regurgitant fraction, reduces forward flow through the LVOT, and worsens pulmonary oedema, paradoxically worsening shock [9]. This is likely to have been the case, here. Additionally, our patient's markedly elevated systolic blood pressure to almost 180 mm Hg on two occasions (Fig. 3) each coincided with his requirement for ICU admission.

This is likely not by chance; hypertension is reported to cause acute worsening of MR by means alike to that of a raised SVR which are aforementioned, and also by increasing mechanical stress on the valve [10], again reducing forward flow. Furthermore, on Day 8 of admission, a repeat echocardiogram after dobutamine administration demonstrated mild improvement in cardiac output to 3.52 L/min, with a corresponding drop in SVR to 2478 dynes/sec/cm^{-5}. There was a concordant improvement in clinical signs with a downward trend in systolic blood pressure and mean arterial pressure, prompting stepdown from ICU to the ward.

Treatment options for cardiogenic shock secondary to acute MR

There is limited evidence regarding the use of inotropes in hypoperfusion due to cardiac failure in the setting of haemodynamic stability. One multicentre observational study recruited patients with clinical and biochemical features of CS both with or without hypotension [5]. Patients without hypotension were more frequently managed with diuresis, and less so with inotropic or vasopressor support, yet outcomes between the two groups—thirty-day mortality rate and frequency of heart transplantation—were not dissimilar. This suggests that normotension alone does not reflect an adequately compensated cardiovascular state.

In the context of relative hypertension, as seen here, vasodilators would likely have been a viable option to lower blood pressure and hence, SVR; they also improve coronary blood flow directly via their action on vascular smooth muscle [11]. Studies show that sodium nitroprusside is beneficial in acute MR by reducing afterload; the LV volume is transiently reduced due to reduced pressures across the aortic valve, which leads to a smaller mitral annulus size, and subsequent reduction in mitral effective regurgitant orifice area and regurgitant volume [12, 13].

CONCLUSION

This case is a timely reminder of the breadth of presentation of CS and the need to consider the multitude of mechanisms of reduced cardiac output. It highlights the importance of early treatment of tissue hypoperfusion, irrespective of normotension, and the crucial need for identifying the underlying pathophysiology for definitive treatment of this life-threatening clinical syndrome.

LEARNING OBJECTIVES

1. Consider the multitude of mechanisms of reduced cardiac output and the associated physiological compensatory efforts; hypotension may not be observed in some patients who otherwise exhibit evidence of poor end-organ tissue perfusion.
2. Understand the necessary investigations and treatment required in cardiogenic shock—both short-term and definitive—to optimise patient outcomes.
3. Understand the effect of hypertension and systemic vascular resistance on forward flow in the setting of severe mitral regurgitation.

ABBREVIATIONS

Acute kidney injury (AKI); acute myocardial infarction (AMI); B-natriuretic peptide (BNP); cardiogenic shock (CS); electrocardiogram (ECG); Emergency Department (ED); Intensive Care Unit (ICU); international normalised ratio (INR); left ventricular ejection fraction (LVEF); left ventricular outflow tract (LVOT); liver function tests (LFTs); mitral valve regurgitation (MR); systemic vascular resistance (SVR); transoesophageal echocardiogram (TOE); transthoracic echocardiogram (TTE); tricuspid valve regurgitation (TR); venous blood gas (VBG).

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CONFLICT OF INTEREST STATEMENT

None declared.

FUNDING

No funding was received for the present case report.

CONSENT

Informed written consent was obtained from the patient for whom this case study pertains to. As this is a case report and does not meet the definition of research, it is exempt from review by a Human Research Ethics Committee.

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