Case Report

Cardiogenic Shock with High Pressure: A Case Report

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Introduction

Shock is defined as a state of tissue hypoperfusion due to reduced supply, increased consumption, inadequate use of oxygen, or a combination of these processes. When it is of cardiogenic etiology, it occurs as a result of left or right ventricular or biventricular dysfunction with reduced cardiac output (CO) or increased filling pressures and normal intravascular volume.[1] Although the most classic manifestation of shock is arterial hypotension, it is essential to recognize that a patient in shock may present with normal, elevated, or decreased blood pressure (BP). Therefore, early identification of clinical, laboratory, and hemodynamic signs of tissue hypoperfusion is crucial. Timely and appropriate treatment can prevent deterioration and signs of multiple organ dysfunction.

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Female patient, 69 years old, with a history of high BP, smoking, and coronary artery disease who underwent surgical myocardial revascularization in 2019, arrived at the emergency room reporting oppressive chest pain lasting 2 hours, started at rest, with irradiation to the back and upper limbs, associated with non-specific malaise, a higher Verbal Numerical Pain Scale (VNS) of 8 and partial improvement after using captopril and losartan while still at home. She regularly used losartan 50 mg twice a day, acetylsalicylic acid 100 mg a day, and rosuvastatin 20 mg a day. She arrived at the hospital with mild chest discomfort.

On physical examination, ectoscopy was altered by skin pallor and cold extremities. Normal cardiovascular auscultation. Respiratory auscultation showed fine crackling rales at the base of the right hemithorax and BP of 170/90 mmHg. Resting electrocardiogram without changes. After initial measures for Acute Coronary Syndrome (ASA 200 mg and Ticagrelor 180 mg) and initiation of nitroglycerin due to clinical signs of

pulmonary congestion, signs of hypoperfusion developed (thin pulses, cold and clammy skin, skin-mucosal pallor), despite BP remaining elevated (170/90 mmHg). Arterial blood gas analysis with evidence of metabolic acidosis and elevated lactate (2.7 mmol/L).

After starting dobutamine at a continuous flow rate of 7.5 mcg/kg/min, the patient was taken to the ICU, where the drug was suspended due to hypertension and improvement in signs of hypoperfusion. The patient developed acute pulmonary edema a few minutes after discontinuation of dobutamine, BP 210/130 mmHg, being submitted to orotracheal intubation and referred to emergency cardiac catheterization, where a severe lesion was seen in the proximal segment of the left mammary artery - anterior descending, submitted to angioplasty with drug-eluting stent, intra-aortic balloon implantation (IABP) and Swan-Ganz catheter.

Swan-Ganz measurements showed cardiogenic shock due to left ventricular (LV) dysfunction with CO of 3.59 L/min, Cardiac Index (CI) of 2.3 L/min, Cardiac Power Output (CPO) of 0.29, BP 140/60 mmHg (MBP 86 mmHg), Systemic Vascular Resistance (SVR) of 2495 Dyn.s and Pulmonary Artery Capillary Pressure of (PCAP) 32 mmHg.

After examination, she was kept in the ICU with dobutamine 7.5 mcg/kg/min, nitroglycerin 16 mcg/min, and IAPB 1:1, removed the next day due to hemodynamic improvement. There was rapid weaning of vasoactive drugs, with initial suspension of dobutamine and, on the second day of hospitalization, nitroglycerin. Transthoracic echocardiogram on the second day of hospitalization showed LVEF 48% by Simpson and inferolateral and lateroapical hypokinesia. Repeated echocardiogram on the third day of hospitalization with LVEF 75% by Teicholz (Simpson not calculated) and anterior medioapical and inferior apical akinesia. She was weaned from mechanical ventilation and was discharged from the hospital after 15 days of hospitalization.

Discussion

The patient reported in the case was admitted to the emergency department due to acute coronary syndrome without ST elevation, leading to sudden cardiogenic shock, characterizing a very high-risk acute coronary syndrome. She presented clear clinical signs of tissue hypoperfusion, such as cold extremities, slow capillary refill time, and tachypnea; however, BP remained high. Blood gas analysis with signs of metabolic acidosis and increased lactate collected in the emergency department increased the probability of

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cardiogenic shock, and confirmation occurred through cardiac catheterization and introduction of Swan-Ganz, showing low CI, CO, and CPO 0.29 (normal when higher than 0.6). Given the ischemic etiology, after percutaneous revascularization and initial circulatory support with vasoactive drugs and an intra-aortic balloon, the treatment was successful, and the patient was discharged from the hospital within a few days.

According to the SHOCK Trial, the concept of cardiogenic shock involves three hemodynamic criteria: persistent hypotension (SBP < 90 mmHg or Mean BP < 30 mmHg in relation to baseline), CI < 1.8 L/min/m² without circulatory support or < 2.2 L/min/m² when using circulatory support and increased filling pressures (LV end-diastolic pressure > 18 mmHg or right ventricle end-diastolic pressure > 10-15 mmHg).[2]

With the passage of time and the understanding of the complexity of the concept and management of cardiogenic shock, other more detailed classifications became necessary to understand the severity and prognosis of patients better, thus guiding therapy. The SCAI Shock classification classifies cardiogenic shock into five stages: stage A are patients with acute cardiac conditions who demonstrate a risk of progressing to shock; Stage B is known as pre-shock and encompasses patients with intact tissue perfusion, but with signs of hemodynamic instability, such as compensatory tachycardia or hypotension. Stage C refers to patients in clear cardiogenic shock with signs of tissue hypoperfusion (such as increased lactatemia) and the need for pharmacological or mechanical circulatory support. Stage D refers to patients receiving increased doses of vasoactive drugs or mechanical circulatory support, and stage E translates into patients refractory to all these measures.[3]

Cardiogenic shock resulting from acute coronary syndrome can present a dramatic clinical picture with a rapid decline in tissue perfusion, consequent renal and hepatic dysfunction, and reduced level of consciousness. In the pre-shock state, the body can use compensatory mechanisms to maintain adequate output, such as tachycardia and peripheral vasoconstriction, generating normal or even increased BP.[2]

In the patient report, we observed that cardiogenic shock occurred suddenly and that she was in stage C of the SCAI classification, as she presented clinical and laboratory signs of tissue hypoperfusion (arterial lactate above 2 mmol/L) despite her high BP. This fact draws attention to the importance of investigating incipient signs of cardiogenic shock in patients at risk, even with apparently "stable" vital signs, as studies using SCAI Shock show a higher rate of negative outcomes in patients with tissue hypoperfusion without hypotension when compared to hypotensive patients without hypoperfusion.[4]

Given the real possibility of diagnosing cardiogenic shock without hypotension, patients at risk of shock, that is, those classified as SCAI stage A, need a careful approach and a high degree of suspicion for progression to more advanced stages of the disease since there is evidence that this patient profile has a longer delay in diagnosis and transfer to specialized services. Recognizing the correct shock stage in these cases may mean a greater possibility of defining the therapeutic strategy, as patients with shock and normotension or hypertension take longer for clinical deterioration compared to hypotensive patients.[5]

In patients with previous advanced heart failure, cardiogenic shock may have more indolent clinical presentations, such as the "cold and damp" hemodynamic profile, in which there are signs of low output and increased cardiac filling pressures due to volume overload, and the "cold and dry" with reduced intravascular volume. In both situations, due to a state of compensation, patients can maintain SBP above 90 mmHg. Around 5% of patients in this profile present cardiogenic shock with arterial normotension.[1]

Given a wide spectrum of presentations of cardiogenic shock, it is necessary, in addition to a thorough anamnesis and physical examination, to use invasive hemodynamic monitoring for an accurate diagnosis, mainly by calculating the CI, CO and CPO, which is obtained by the CO X MBP/451 calculation, which reflects the cardiac hydraulic pumping capacity, with high accuracy in measuring LV function and prognostic correlation with in-hospital mortality according to SHOCK Trial.[5,6] The calculation of PAPi (Pulmonary Arterial Pulsatility index) through the ratio between the pulsatility pressure of the pulmonary artery and the pressure of the right atrium is capable of providing right ventricular function.[2] Early diagnosis helps in quickly choosing the best circulatory support, whether through vasoactive drugs or ventricular assist devices, revascularization in cases of acute myocardial infarction, increasing the probability of reversibility of the shock state, despite studies such as FRENSHOCK showing that there is no difference in outcomes when comparing patients with cardiogenic shock with hypotension or normal BP.[2]

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