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A 69-Year-Old Woman with Diabetes Presenting with Acute Dyspnea

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1. Introduction

Cardiogenic shock (CS) is a devastating syndrome with heterogenous etiology ranging from intrinsic acute or chronic myocardial damage to pericardial diseases, arrhythmias or conduction abnormalities and valvular heart disease [1]. Among these various etiologies, mechanical valve thrombosis is a difficult diagnosing condition and, although not common, is catastrophic leading to cardiogenic CS and death if its not suspected and diagnosed timely to provide the required treatment [2]. Difficulties in diagnosing mechanical valve thrombosis arise from variable clinical presentations, that is an incidental finding on an imaging examination to systematic thromboembolic events, pulmonary oedema and ultimately cardiogenic shock, mainly influenced by the acuity and the hemodynamic degree of obstruction [3]. Importantly, challenges in diagnosis are due to lack of awareness about the possibility of valve thrombosis, attributing patients' findings that may co-exist to another diagnosis. Diabetic ketoacidosis (DKA) is one of the two most serious complications of diabetes mellitus, presenting mainly with hyperglycemia and ketoacidosis. Although this condition is associated with type 1 diabetes, in type 2 diabetes can also occur but mainly associated with an underlying acute insult posing extreme stress such as serious infection, major trauma or cardiovascular emergency [4]. The principal findings at initial presentation are hyperglycemia (basically at levels of 350-500 mg/dl), increased anion gap metabolic acidosis and consequently compensatory hyperventilation, hypocapnia and ketonemia/ketonuria. Leukocytosis is almost invariably present in ketoacidosis patients, irrespective of underlying infection [5]. Sepsis and cerebral edema (CE) are the commonest causes of death in DK patients while respiratory failure is frequent and necessities intubation and invasive mechanical ventilation due to risk of aspiration if non-invasive ventilation is used [6]. Increased levels of lactic acid, although has been reported in DKA, their clinical significance remains elusive [7]. What is known is that increased lactate levels correlate with disease severity in multiple conditions such as septic or cardiogenic shock [8]. Now it is recognized that hyperlactatemia is frequently found in DKA and is reported to be associated with adverse outcomes [9], although elevations in blood lactate are considered a hallmark of hypovolemia, hypotension, and hyperventilation in addition to specific medications [10]. In the light of these information, we present a case of a woman admitted to the Emergency Department with initial working diagnosis of DKA, rapid deterioration of her clinical condition, intubation and ICU admission, where cardiogenic shock was diagnosed as the cause of her severe clinical condition and unfortunately her death.

2. Case Presentation

A 69-year-old female patient, was admitted to the emergency department of a tertiary hospital, presenting with fatigue and acute onset dyspnea and orthopnea started few hours before hospital admission. She denied any other symptoms in the previous days or any interruption of her medical treatment. Her past medical history includes arterial hypertension, insulin-treated diabetes

mellitus (DM), chronic kidney disease (CKD) without need for renal replacement therapy, mitral valve stenosis that 6 years ago underwent replacement with a metallic prosthesis, chronic atrial fibrillation and pulmonary hypertension with chronic hypoxia which required home oxygen therapy. She was on long-term anticoagulation therapy with acenocoumarol. Her previous echocardiogram five months ago reported that left ventricle was normal in dimensions with preserved ejection fraction, dilated RV but with preserved contractility, severe bi-atrial dilatation, severe tricuspid regurgitation and mechanical mitral valve without dysfunction. PASP had been estimated to 65mmHg.

3. Physical Examination Findings

At the initial evaluation in the emergency department, the patient was tachypneic (respiratory rate about 35 breaths per minute) with intense diaphoresis and reticular peliosis of the extremities. Her blood pressure was 140/70 mmHg, her heart rate was 110 beats per minute, her oxygen saturation was 60% and temperature was 36,8^{o}C. Pulmonary auscultation revealed extended rales till the mediation of both lungs. Rest physical examination revealed cold, pale extremities and no other significant findings.

4. Initial Diagnostic Studies

Arterial blood gases showed severe hypoxemia (P/F ratio 78), severe mixed acidosis (pH=6,9, pCO2=62mmHg, HCO^{3-}=12,4 mmlol/), increased anion gap (=26) and elevated levels of lactic acid (=8,4 mmlol/L]. Her ECG (Figure 1), showed atrial fibrillation with right axis deviation and repolarization abnormalities with ST depression in many leads. Initial laboratory values were remarkable for leukocytosis (WBCs=36 x 10^3), mildly elevated CRP (=0,3mg/dl), increased blood glucose levels (>500mg/dl) and very elevated urinary ketones. INR was 1,63 with 12,5 PT. Kidney biochemical parameters were abnormal (Urea 76 mg/dl, Creatinine 2,5 mg/dl) and rest laboratory values were within normal range. Transthoracic echocardiogram performed by cardiology resident reported that patient had poor acoustic windows, and there for gross examination revealed mildly depressed left ventricular ejection function (estimated ejection fraction 40-45%), normal left ventricular dimensions, severely dilated RV with compromised contractility and bi-atrial enlargement. Findings from cardiac valves were remarkable for tricuspid and mechanical mitral valve regurgitation. Inferior vena cava was 1,5cm in maximum diameter with >50% respiratory variation. PASP was estimated at 50mmHg. Pericardial fluid was not detected. Lung ultrasound at the bedside revealed bilateral B-lines and mild right pleural effusion without any other significant findings. At this point and before further diagnostic evaluation, the patient received initial therapy including intravenous fluids, bolus and drip insulin, and antibiotics (piperrakilin/tazobactam) after blood and urinary samples collection for cultures. For the treatment of acute respiratory insufficiency, physicians at the emergency department decided to use Non-Invasive Ventilation (NIV) with positive end-expiratory pressure. When NIV started, the patient collapsed hemodynamically, and vasopressor (norepinephrine) was initiated. Her clinical condition worsened, preventing her transfer for further diagnostic imaging. Vasopressor was rapidly escalated in dose and the patient fell into cardiac arrest with unshockable rhythm. She was intubated, invasive mechanic ventilation started, and she recovered after 8 minutes of CPR. She was transferred to the intensive care unit (ICU) for advanced post-resuscitation care. In the ICU, despite these therapeutic measures, acidosis could not be reversed despite continuation of insulin and sodium bicarbonate administration. Hemodynamic instability was continuously aggravated. These findings quickly prompted for re-evaluation of patient's initial diagnosis. Further laboratory exams revealed markedly elevated NT-proBNP levels (1237 pg/mL), consistent with significant cardiac strain. A central venous oxygen saturation (ScvO2) of 35% underscored inadequate oxygen delivery to tissues, procalcitonin was 0,35ng/ml that was not compatible with septic shock and could not explain patient's severe condition, at least solely. High-sensitivity troponin was negative at two sequential measurements 3 hours apart, excluding acute severe myocardial damage or acute coronary syndrome. These findings prompted a multidisciplinary discussion involving cardiologists and the critical care team. Based on the fact that patient had mechanical prosthetic valve at the mitral position and the fact that acute pulmonary embolism could not be investigated by CTPA due to patient's critical condition for transfer, team decided to proceed in TOE. TOE was performed by attending cardiologist-intensivist physician and revealed almost complete obstruction of left ventricular inflow due to large thrombus on the atrial surface of mechanical valve (Figure 2) further supported by the presence of "smoke" due to increased thrombogenicity and slow flow into the left atrium. Diagnosis was supported by patients subtherapeutic INR levels at the initial laboratory exams. Pressure half time on pulsed Doppler was increased to >200 ms, indicative of valvar severe obstruction. Systolic performance of left ventricle was severely impaired, stroke volume and cardiac output were severely reduced. Diagnosis of acute mechanical valve thrombosis was established as the cause of cardiogenic (obstructive) shock.

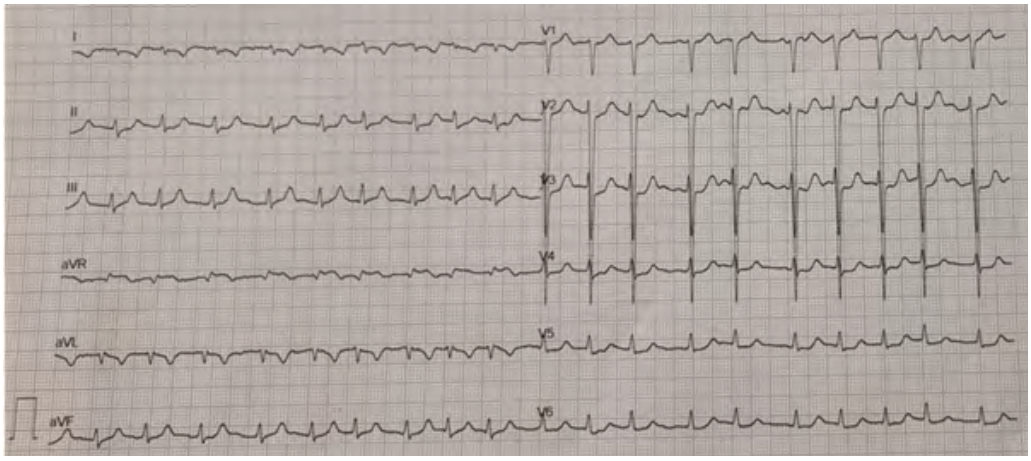

Figure 1: Patient's initial ECG.

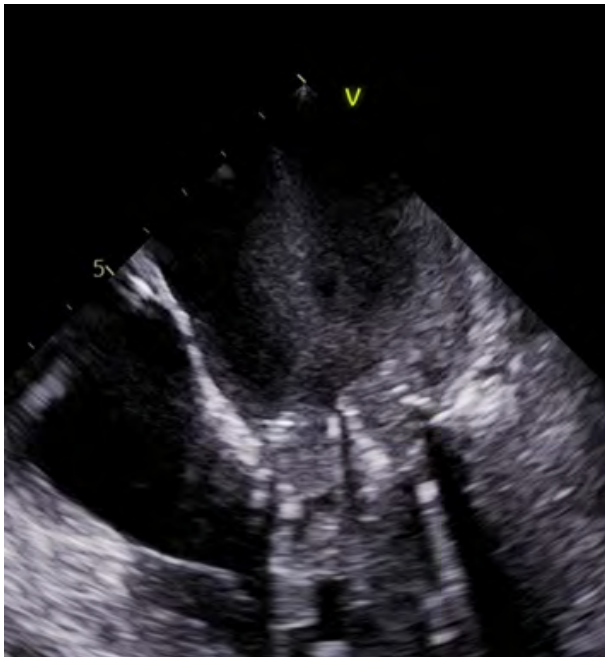

Figure 2: TOE zoomed on left cavities showing obstructive thrombus on the atrial surface of mechanical valve along with "smoke" into left atrium. This image quickly setting the diagnosis of cardiogenic shock due to acute thrombosis of mechanical valve and obstruction of left ventricular inflow tract.

5. Discussion

Based on initial findings, pulmonary oedema due to acute cardiac decompensation is the most probable diagnosis and further diagnostic evaluation should be conducted to identify the exact underlying cause. Diabetic ketoacidosis could explain patient's metabolic findings (acidosis and urine ketones) but could not explain, at least solely, tissue hypoperfusion, severe lactic acidosis and severe hemodynamic shock. Although this condition is associated with type 1 diabetes, in type 2 diabetes can also occur but in such cases, it is mainly associated with an underlying acute insult posing extreme stress such as serious infection, major trauma or cardiovascular emergency and so key element in patient's treatment is the identification of this acute insult. Lactic acidosis present from the beginning implies severely reduced organ perfusion despite initial "good" blood pressure that is maintained possibly due to patient's hyperadrenergic state. Indeed, hypoperfusion is not a hallmark of hypotension. Blood pressure may be initially normal due to compensatory vasoconstriction, albeit at the cost of impaired tissue perfusion [11] and oxygenation, reflected by increased values of lactic acid and patients reticular peliosis. In accordance with these data, cardiogenic shock now is defined as hypoperfusion due to decreased or not enough cardiac output, but with adequate preload [12,13] with hypoperfusion being a hallmark of the diagnosis even in the absence of hypotension [14]. When NIV initiated, hemodynamic collapse was the result implying acute decompensation of cardiac output of an already severely dysfunctional heart due to positive intrathoracic pressures that reduced cardiac preload (venous return).

The initial diagnosis was not clear or easy to establish in our patient. In the emergency department, clinical and laboratory findings were considered diabetic ketoacidosis. Red flags that should prompt further evaluation were 1) Diabetic ketoacidosis could not solely explain patient's clinical condition especially severe elevation of lactic acid and hemodynamic collapse after NIV initiation, unless a serious underlying condition exists. 2) The dyspnea that the patient complained about was considered as probable respiratory infection on top of the pre-existing respiratory insufficiency and sepsis due to that was thought to be the causative factor of ketoacidosis. But patient was afebrile, CRP was not high enough to direct the diagnosis to septic shock, at least initially, and lung ultrasound, although with 70% sensitivity, did not reveal findings compatible with lung infection 3) At the initial TTE, pathologic findings are reported but with the limitation of poor acoustic windows. These findings should prompt quick further characterization, probably by a more experienced physician and advanced echocardiographic measures before a patient's situation is further compromised. 4) Although NIV is appropriate therapeutic measure in hypercapnic respiratory insufficiency, it is important to evaluate patient' s cardiac condition thoroughly before its application. Hemodynamic collapse after applying positive end-expiratory pressure indicates severely reduced cardiac output further aggravated by reduced preload (venous return). Although hemodynamic decompensation can occur in any patient with reduced volume status (e.g dehydration as is true in diabetic ketoacidosis) after NIV initiation, this hemodynamic instability is rarely severe, requiring very high doses of vasopressors, rapidly responds to fluids administration and if no other cause exists, there is no need for vasopressors dose escalation. The only treatment options for this patient were surgery and thrombolysis, but with very high risk whatever the option taken [15]. Surgery is only suitable for patients without serious contraindications as per ESC guidelines statement [16]. The extremely severe, uncompensated, hemodynamic shock along with severe metabolic acidosis that never reversed despite treatment, along with other serious problems of our patient (severely dysfunctional RV with pulmonary hypertension, chronic respiratory insufficiency with LTOT) prevented patient from surgery due to contraindication by consulting cardiac surgeons stated that patient was at unacceptable high risk for surgery. The second therapeutic option of fibrinolysis carries risks of bleeding, systemic embolism, and recurrent thrombosis [17]. In our evaluation for fibrinolysis, our patient had increased thrombus size carried increased risk for embolic events after fibrinolysis. A second laboratory evaluation was performed before thrombolysis that revealed highly abnormal coagulation times probably due to severe shock liver and hepatic insufficiency due to hypoperfusion. In light of increased risk for embolic events and increased bleeding risk, contraindication to thrombolysis was settled and slow infusion of unfractionated heparin was decided as despair solution but patient unfortunately died after 2 hours in ICU.

6. Clinical Pearls

1. Key features for proper and timely diagnosis derives from the ESC guidelines which state that obstruction of mechanical prosthetic valve should always be suspected in any patient who presents with recent dyspnea or embolic event [18] as exactly in our case. Diagnosis should be confirmed at the earliest stages of patient evaluation by TOE if TTE is of low diagnostic value.

2. When mechanical valve is present, evaluation should always be conducted at least with the presence of a highly experienced attending physician to ensure diagnostic accuracy and to mitigate the risk of being erroneously directed toward alternative diagnoses, along with resident' s education.

3. Challenges in diagnosis of mechanical valve thrombosis are due to lack of awareness about the possibility of valve thrombosis, attributing patients' findings that may co-exist to another diagnosis. This lack of awareness arises from variable clinical presentations, that is an incidental finding on an imaging examination to systematic thromboembolic events, pulmonary oedema and ultimately cardiogenic shock, mainly influenced by the acuity and the hemodynamic degree of obstruction

4. Cardiogenic shock can mimic various other conditions at initial presentation and diagnosis is obscured by superimposed medical problems that these patients usually have. Increased awareness is needed for quick diagnosis and proper therapeutic measures before patient is non-reversible compromization. Cardiogenic shock is a very serious medical emergency with very poor prognosis, basically affected by the correct diagnosis and proper management.

5. It is important to access patients preload and cardiac condition before use NIV for respiratory failure. PEEP could severely compromise these patients and alternative measures should be considered such as High-Flow Nasal Oxygen.

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