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Case Report Open Access

Stunned Myocardium due to Decompensation from Hypovolemic Shock in a Pregnant Woman with Uterine Atony Following Cesarean Section

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Abstract

Transient post ischemic left ventricular dysfunction has been called stunned myocardium. The stunned myocardium can lead cardiac problems such as arrhythmias, left ventricular dysfunction, and a myocardial infarction. Stunned myocardium is a reversible condition. In this case, a 25 year old female patient with a history of mild intermittent asthma became hypovolemic and got into shock after developing uterine atony following a cesarean section. The patient did not respond to rapid volume replacement therapy. Subsequently, the patient had acute pulmonary edema, hypotension, and tachycardia developed. Echocardiogram was done showing severe left ventricular dysfunction (ejection fraction (EF), 25-35%) with left inferobasal wall hypokinesis and no right ventricular dysfunction or severe tricuspid regurgitation or right ventricular hypertrophy. Chest x-ray showed newly diagnosed cardiomegaly and bilateral pulmonary congestion. Initial troponin I was elevated, however, the level of troponin I remained stable with the same baseline value. Patient was at high risk for pulmonary embolism (PE) based on risk assessment, therefore, anticoagulation was started. Chest CT for pulmonary embolism was performed, and the result was negative. Two days after, repeated echocardiogram showed improved EF was 35%. Patient was discharged with beta –blocker to reduce oxygen demand of myocardium. This case supports hypovolemic shock not responding to volume replacement therapy can cause myocardium damage which is able to be diagnosed with stunned myocardium.

Keywords: Hypovolemic shock; Stunned myocardium; Pulmonary embolism

Case Report

A 25 year old Hispanic female with history mild intermittent asthma was admitted for normal vaginal delivery. Obstetrician converted it to cesarean section due to severe bleeding secondary to uterine atony and inversion. Patient was already intubated for the cesarean section. Heart rate were 150/ min , O_2 saturation was 81% on FIO_2 100% , and systolic blood pressure was checked at 84 mmHg and diastolic was 40 mmHg, therefore, 2 units of packed red blood cell and 4 liters of Ringer lactate were given for hypovolemic shock secondary to bleeding. Blood pressure was not returned to normal range even if transfusion and intravenous fluid were given. Endotracheal tube position was checked by laryngoscopy, glydescope and fiber optic bronchoscopy. After completion of cesarean section in the operation room, patient was transferred to surgical intensive care unit (SICU) for post-surgical care. Patient was still tachycardic and hypotensive, therefore, patient was transferred to medical intensive care unit (MICU) for acute respiratory distress syndrome (ARDS) and presumed diagnosis of PE, which might be developed during cesarean section. Chest x-ray at that time showed bilateral pulmonary edema with cardiomegaly. Electrocardiogram (EKG) showed sinus tachycardia without specific ischemic changes (Figure 2). Initial troponin I was elevated up to 0.21, however, it was post-operative, moreover, the peak of troponin I was almost the same with initial value (0.22). So, it was not similar with pattern of acute coronary syndrome in terms of troponin level change. Also, d-dimer was elevated, which could be related to this surgery. DIC panel was within normal range. ABG was metabolic acidosis and respiratory acidosis with elevated alveolar –arterial gradient. Patient was at high risk of pulmonary embolism (PE) based on risk assessment such as recent surgery, tachycardia and hypotension, therefore, anticoagulation was started even if there was no definite evidence of PE from echocardiogram. The transthoracic echocardiogram (TTE) showed inferiobasal wall hypokinesis and apical wall normal motion (Figures 1A and 1B). Thrombolytics or thrombectomy should be considered because of hemodynamic instability [1], however, it was not done due to recent massive bleeding during cesarean section. After hemodynamic stabilization and extubation, chest CT for pulmonary embolism was performed, which came back negative for PE? Heparin drip was discontinued. Following TTE 2 days later showed improved EF (35%). Patient was discharged with beta –blocker to reduce oxygen demand of myocardium.

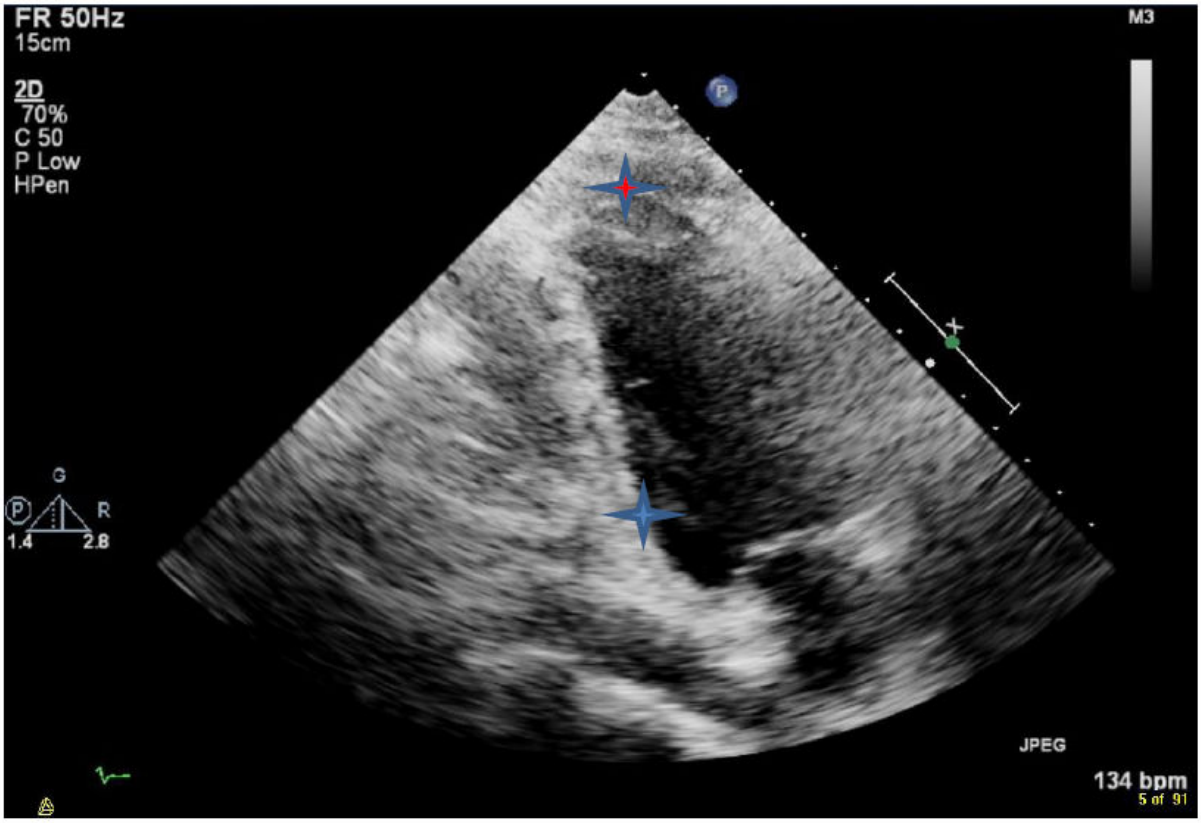

Figure 1A: Transthoracic Echocardiography two chamber view showed systolic phase of left ventricle. Apical wall was collapsed for contractile movement (See red star), however, hypokinesis of Inferior basal wall in the left ventricle was observed (See blue star).

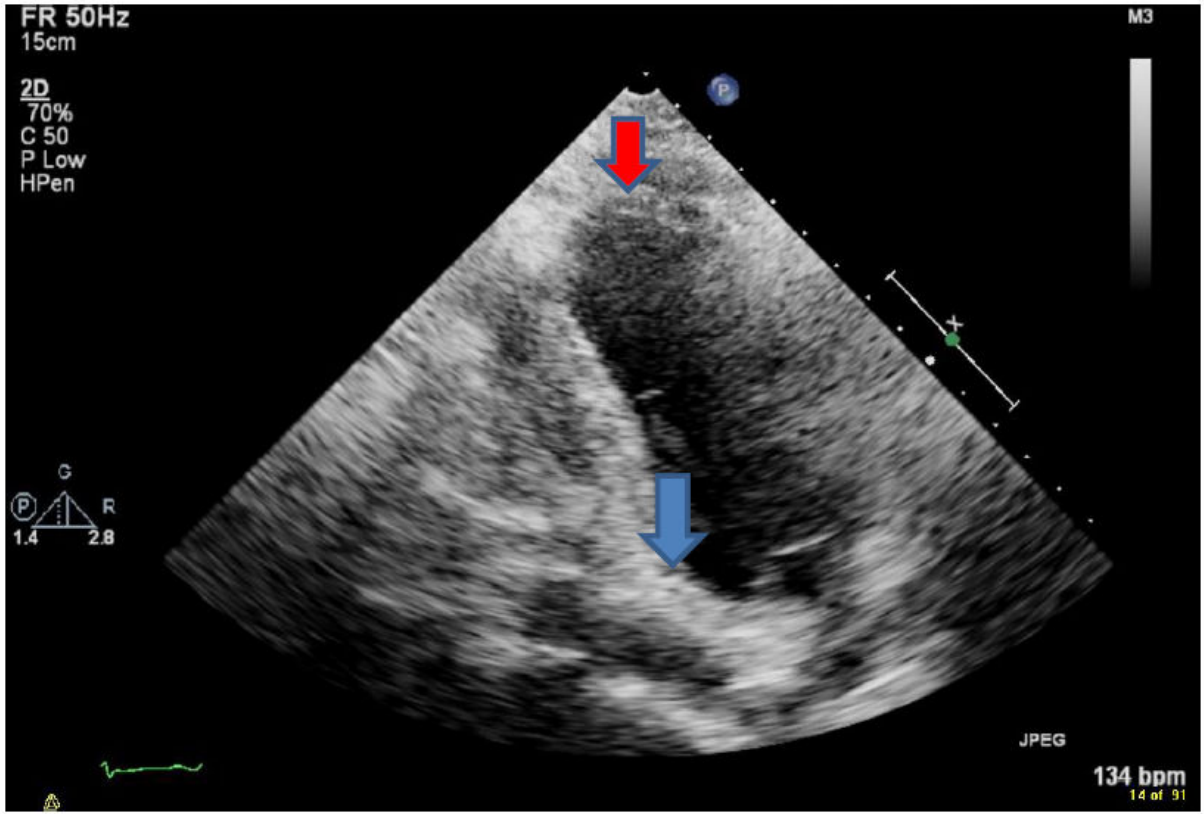

Figure 1B: Transthoracic Echocardiography two chamber view showed diastolic phase of left ventricle, and inferior basal wall did not show any movement. Only apical wall moved up compared to Figure 1, (See red arrow), however, inferior basal wall did not move well. It suggests hypokinesis of inferior basal wall in left ventricle.

Discussion

We report a case of stunned myocardium following hypovolemic shock which is reported for the first time. Stunned myocardium is called when heart has dysfunction after transient ischemia [2,3]. Myocardial dysfunction persists for a variable period after acute coronary ischemia, uncontrolled heart failure, subarachnoid hemorrhage and tachycardia induced cardiomyopathy. The term "stunned" myocardium originally arose from observing the wall motion of canine hearts after occluding coronary blood flow shorter than necessary to cause cell death [3]. The time that myocardium is at risk of being stunned is when it has a low reserve flow [4]. Acute emotional or physical stress can trigger a catecholamine-mediated myocardial stunning [5]. Extremely high plasma catecholamine levels and their metabolites elevation can cause stress cardiomyopathy resulting in stunned myocardium with transient ischemic change [6]. This is also related to autonomic dysfunction which is potentially resulting in tachycardia and hypotension in our study. Activation of sympathetic nerve has affected left ventricle myocardium, which might result in stunning [7]. In addition, this patient had pro-inflammatory status such as severe bleeding and surgery. It could be resulting in oxidative stress and then cardiac dysfunction like hypokinesis of left ventricle [8]. Adrenergic cardiac innervation through alpha-lipoic acid (ALA) may give benefit from patient's myocardium LV recovery in stunned myocardium [9] because it is also transient ischemic injury and ALA may play a role as anti-oxidative therapy in myocardium.

Contrary to hibernating myocardium [10], the stunned myocardium could have more acute change from transiently impaired coronary blood flow [11]. As long as the ischemic myocardium remains viable, the LV dysfunction can be partially or completely restored to normal by improving blood flow or by reducing oxygen demand. Usually, for hypovolemic condition, rapid volume repletion is indicated in patients with severe hypovolemia [12]. If blood pressure or heart rate is not corrected by rapid volume infusion, we need to rule out other conditions related to decompensation [13]. During cesarean section, patient condition was deteriorated, as a result, amniotic fluid embolism and PE were also considered. But DIC panel was negative, and that means amniotic fluid embolism [14,15] was less likely considered at that time. PE was also ruled out by chest CT angiogram. The echocardiogram showed inferobasal hypokinesis and normal apical wall motion which was similar to other reports [16-18], which suggested that this patient might have transient ischemic injury on myocardium. In this case, the cause of stunned myocardium could be decompensation from hypovolemic shock occurred after severe bleeding, and there are no similar previous reports. Severe LV dysfunction with heart failure feature was assumed to develop from hypovolemic shock in this patient. Stunned myocardium with severe heart failure following hypovolemic shock is rarely described; hence, we hope this report helps for differential diagnosis related to hypovolemic shock (Figure 3).

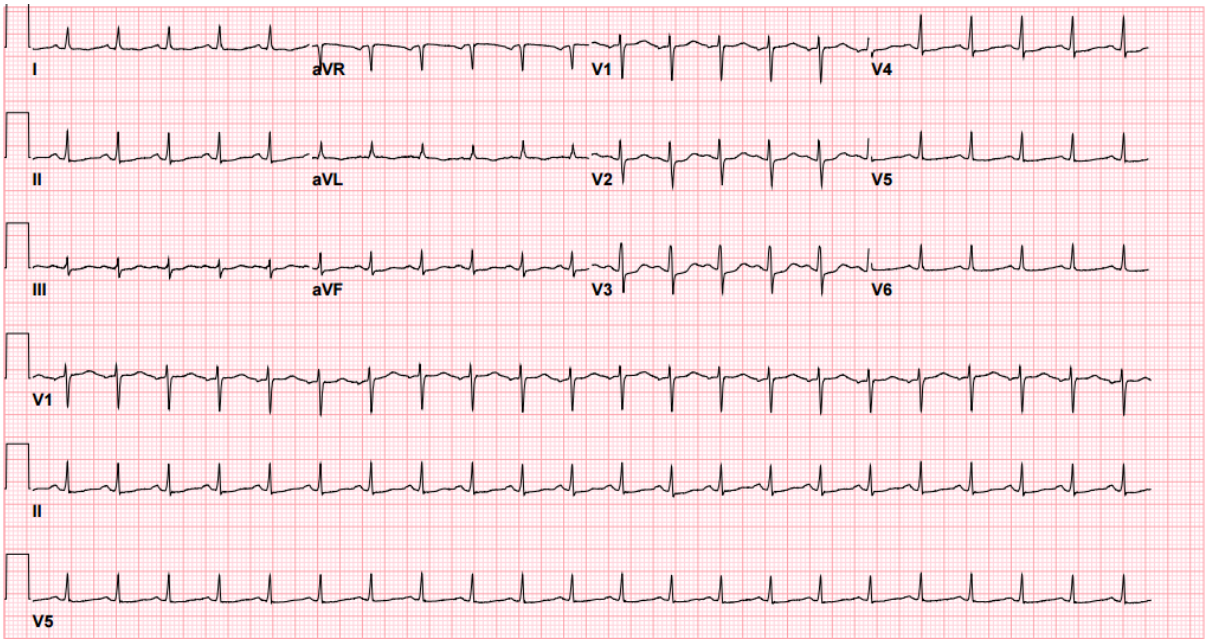

Figure 2: Electrocardiogram at the time of hypovolemic shock, Legend: Sinus tachycardia, no ischemic change for this hypovolemic shock.

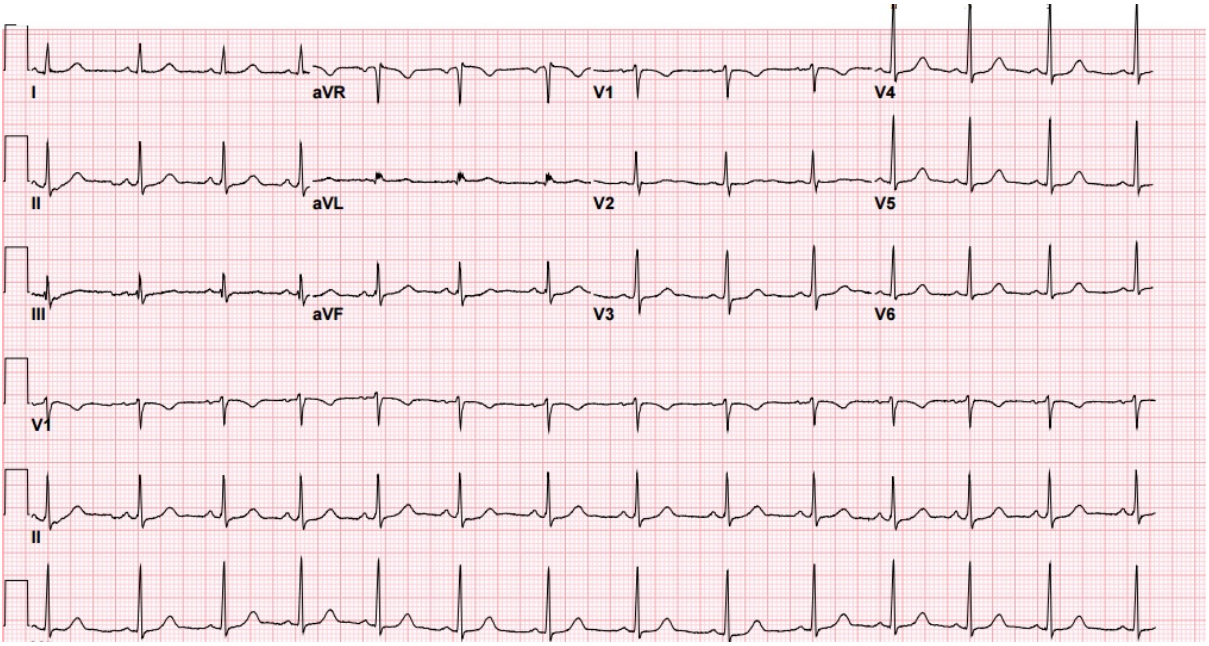

Figure 3: Electrocardiogram at the time of admission, Legend: Normal sinus rhythm without ischemic change before delivery in this admission.

Conclusion

The case emphasized the importance of differential diagnosis for hypovolemic shock from severe bleeding. Even if patient did not have any risk factors for cardiac problems, this patient developed stunned myocardium from hypovolemic shock, and then her left ventricular function recovered very quickly. There is no clear answer for this recovery in stunned myocardium. We may hypothesize the myocardial function recovery in hypovolemic and ischemic myocardial condition can be affected by endothelial progenitor cells differentiation and mobilization [19]. Therefore, no invasive procedure was needed and supportive treatment was necessary in this case. From this study, a case of suspicious PE can be diagnosed with stunned myocardium with

echocardiogram and this supports hypovolemic shock not responding to volume replacement therapy probably can cause transient ischemic injury resulting in stunned myocardium.

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