An Unusual Case of Effusive-Constrictive Pericarditis in the Intensive Care Unit

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INTRODUCTION

Acute myeloid leukemia (AML) uncommonly causes a hemorrhagic pericardial effusion with tamponade effect. Effusive-constrictive pericarditis (ECP) development after pericardiocentesis in such clinical settings is exceedingly rare. We present a case of a patient newly diagnosed with AML who had a baseline echocardiogram that demonstrated a small pericardial effusion. During the early course of AML treatment, acute clinical deterioration occurred, and cardiac tamponade was detected by cardiac point-of-care ultrasound (POCUS). Despite successful pericardiocentesis, the patient continued to deteriorate. Constrictive physiology was appreciated on transthoracic echocardiogram, and ECP was diagnosed.

CASE PRESENTATION

A 39-year-old man with unremarkable past health presented with epigastric discomfort, acute dyspnea, and headache. Vital signs on presentation were normal, with a blood pressure of 123/59 mm Hg, heart rate of 95 per minute, oxygen saturation of 99% on ambient air, and temperature of 36.1 °C. Physical examination revealed a well-built man without apparent heart failure: jugular venous pressure was not elevated, pitting peripheral edema was absent, heart sounds were not muffled, and there was no pericardial rub. Laboratory tests showed a white blood cell count of 203 $\times$ 10^9/L (normal range [NR], 3.7-9.3 $\times$ 10^9/L) with 75% blasts and promonocytes in peripheral blood smear. Anemia and thrombocytopenia were also present, with hemoglobin of 8.7 g/dL (NR, 13.5-17.3 g/dL) and platelet count of 51 $\times$ 10^9/L (NR, 160-420 $\times$ 10^9/L). There was significant hypokalemia of 2.4 mEq/L (NR, 3.4-5.0 mEq/L), albumin-adjusted calcium level was 10 mg/dL (NR, 8.82-10.42 mg/dL), and phosphate level was 1.42 mg/dL (NR, 2.35-4.34 mg/dL). Creatinine was mildly elevated at 1.29 mg/dL (NR, 0.78-1.13 mg/dL). An electrocardiogram showed sinus tachycardia with normal voltage. Chest radiography showed cardiomegaly and a small left pleural effusion. Transthoracic echocardiogram demonstrated a small pericardial effusion and preserved left ventricular (LV) ejection fraction (Videos 1-3). Left ventricular global longitudinal strain was reduced at −14.4% (NR, ≤ −18.8%; Figure 1). He was diagnosed with AML with hyperleukocytosis by peripheral blood smear that showed greater than 20% of the leukemic population. The presence of promonocytes in peripheral blood smear and the expression of myelomonocytic markers from the flow cytometry study were consistent with the diagnosis of AML with monocytic differentiation. Standard 3+7 induction chemotherapy with cytarabine and daunorubicin was initiated. Aggressive hydration and prophylactic rasburicase were administered intravenously to prevent tumor lysis syndrome.

On day 4 of admission, the patient developed sudden deterioration with acute respiratory distress and profound hypotension. He was emergently intubated and put on invasive mechanical positive pressure ventilation (IMPPV). He required norepinephrine infusion at a high dose of 1.6 μg/kg/min and epinephrine infusion at 0.1 μg/kg/min to maintain his mean arterial pressure. Cardiac POCUS demonstrated a large pericardial effusion with fibrinous strands (Videos 4 and 5). Tamponade effect was evidenced by right atrial systolic collapse and beat-to-beat diastolic collapse of the right ventricle known as the "trampoline sign" (Videos 6 and 7). Shortly afterward, he developed pulseless ventricular tachycardia requiring defibrillation and cardiopulmonary resuscitation. Upon return of spontaneous circulation, urgent ultrasound-guided pericardiocentesis by apical approach yielded 500 mL of heavily blood-stained pericardial fluid. Blood tests showed a drop of hemoglobin level to 4.5 g/dL and platelet count of 17 $\times$ 10^9/L, for which urgent transfusion of packed cells and platelet concentrates was given. Pericardial fluid analysis was negative for bacterial and fungal cultures. Although pericardial fluid adenosine deaminase level was raised at 89 U/L (NR, <40 U/L), both smears for acid-base bacilli and mycobacterial culture were negative. The pericardial fluid cytology revealed the presence of suspicious cells with irregular nuclei with indentations. However, the nature of these suspicious cells cannot be ascertained since no further immunostaining was performed.

Hemodynamic instability persisted despite successful pericardiocentesis. A transthoracic echocardiogram was repeated, showing interval reduction in the size of pericardial effusion and resolution of tamponade features. Hyperechoic material was seen within the pericardial sac (Video 8, Figure 2). Respirophasic ventricular septal bouncing was appreciated: inspiration induced shift of the interventricular septum toward the left ventricle and upon expiration, toward the right ventricle (Video 9, Figure 3). Doppler interrogation of the mitral inflow showed a variation of E-wave peak velocity of 30%. Tissue Dopplers of the medial and lateral mitral annular tissue velocities (e′) were 12 and 8 cm/sec, respectively, illustrating annulus reversus.

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VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE, apical 4-chamber view at baseline prior to administration of chemotherapy demonstrates normal LV systolic function and a small pericardial effusion.

Video 2: Two-dimensional TTE, apical 2-chamber view at baseline prior to administration of chemotherapy demonstrates normal LV systolic function and a small pericardial effusion.

Video 3: Two-dimensional TTE, apical 3-chamber view at baseline prior to administration of chemotherapy demonstrates normal LV systolic function and a small pericardial effusion.

Video 4: Two-dimensional TTE, apical 4-chamber view after initiation of chemotherapy and hemodynamic deterioration demonstrates a large circumferential pericardial effusion with hyperechoic fibrinous stranding within the pericardial space *(arrow)*.

Video 5: Two-dimensional TTE, parasternal short-axis view at the papillary muscle level after initiation of chemotherapy and hemodynamic deterioration demonstrates a large circumferential pericardial effusion with hyperechoic fibrinous stranding within the pericardial space *(arrow)*.

Video 6: Two-dimensional TTE, parasternal long-axis view after initiation of chemotherapy and hemodynamic deterioration demonstrates a large pericardial effusion with beat-to-beat diastolic collapse of the right ventricle *(arrow)*.

Video 7: Two-dimensional TTE, parasternal short-axis view at the aortic valve level after initiation of chemotherapy and hemodynamic deterioration demonstrates a large pericardial effusion with beat-to-beat right atrial systolic collapse *(arrowhead)* and RV diastolic collapse *(arrow)*.

Video 8: Two-dimensional TTE, apical 4-chamber view after successful pericardiocentesis demonstrates a reduction in the size of the pericardial effusion. Hyperechoic material was seen within the pericardial space.

Video 9: Two-dimensional TTE, parasternal short-axis view at the mitral valve level after pericardiocentesis demonstrates a ventricular septal bounce.

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Pulsed-wave Doppler of the hepatic vein exhibited marked diastolic flow reversal (Figures 4-6). These echocardiographic findings were diagnostic of constrictive physiology. The presence of constrictive physiology despite drainage of the initial pericardial effusion suggested the diagnosis of ECP. Despite the timely diagnosis and supportive treatment, the patient developed an asystolic cardiac arrest within a few hours of diagnosis and failed resuscitation.

DISCUSSION

Cardiac Tamponade

Understanding the pathophysiology of cardiac tamponade aids in the use of echocardiography for its diagnosis. Cardiac tamponade is defined as the accumulation of pericardial fluid that causes intrapericardial pressure to exceed myocardial transmural pressures and impede normal filling of cardiac chambers. As intrapericardial pressures continue to rise, the chambers with lower pressures are first affected compared with chambers with higher pressures. Thus, right-sided rather than left-sided chambers are usually involved, and atria are involved at an earlier stage than ventricles. The phase of the cardiac cycle in which a chamber is affected also matters. Compressive effect is more likely to be seen in the phase of the cardiac cycle with lower filling pressures, that is, during systole for atria and diastole for ventricles. Hence, right atrial collapse is more likely to be appreciated during systole and right ventricular (RV) collapse during diastole. Right atrial systolic collapse is an early echocardiographic sign to take notice of and has a specificity of 82% to 100% and a variable sensitivity that is reported to range from 64% to 100%.[1,2] Specificity for the diagnosis of cardiac tamponade improves with a longer duration of right atrial collapse, with the collapse time exceeding one-third of the cardiac cycle.[2] Right ventricular diastolic collapse occurs later and has a sensitivity of 92% and specificity of 100%.[1] As the RV free wall will stay collapsed as long as the intrapericardial pressure exceeds the RV pressure, the duration of RV collapse can reflect the severity of tamponade.[3] Plethora of the inferior vena cava is frequently described as an important finding in cardiac tamponade, defined by inferior vena cava dilatation of more than 2 cm and loss of physiological inspiratory collapse, indicative of a high central venous pressure. Other echocardiographic features include an inspiratory decrease in mitral inflow E-wave peak velocity of at least 30% and a corresponding expiratory decrease in tricuspid inflow E-wave peak velocity of at least 60%.[4] Although these Doppler flow velocity findings are important to highlight the interventricular dependence during cardiac tamponade, they may not be readily appreciated if only handheld devices are available for qualitative assessment.

Cardiac tamponade was a rare clinical manifestation of AML, limited to only isolated case reports.[5] In a case series involving 2,592 patients with AML, acute lymphocytic leukemia, or myelodysplastic syndrome, only 20% of patients had echocardiographic evidence of pericardial effusion, while only 3% of the cases required pericardiocentesis due to tamponade.[6] The event of hemorrhagic pericardial effusion with tamponade occurred acutely in our patient, as evidenced by a marked increase in pericardial effusion and new findings of tamponade effect 3 days after the initial echocardiographic assessment. The heavily blood-stained pericardial fluid and sudden drop in hemoglobin level confirmed the diagnosis of acute hemorrhage into the pericardial space. A few mechanisms may explain this. First, leukemic invasion of the pericardium may cause malignant pericardial effusion. In our case, only morphologic analysis was performed on the pericardial fluid, showing suspicious cells. Ideally, cytogenetic studies and immunohistochemical staining could be performed on pericardial fluid samples to confirm extramedullary involvement by AML, which may have prognostic implications.[7] Histologic findings were not available in our case as the patient's family refused postmortem examination. Second, the effect may be compounded by bleeding diathesis from thrombocytopenia. Of note, besides low absolute platelet counts in AML patients, defects in activation and aggregation of the existing platelets may contribute to life-threatening hemorrhage.[8] Third, chemotherapy-related pericarditis was possible, albeit unlikely in our case. Previous literature reported hemorrhagic pericardial effusion in patients receiving either high-dose cytarabine ($\geq$1,000 mg/m^2/day) for more than 2 days or low-dose cytarabine ($\leq$200 mg/m^2/day) for more than 1 week.[9] Our patient received intravenous cytarabine of 100 mg/m^2/day for 3 days prior to the event of cardiac tamponade, making cytarabine an unlikely culprit. Our patient was also given daunorubicin at a standard dose of 90 mg/m^2/day for 2 days. Although anthracycline-induced

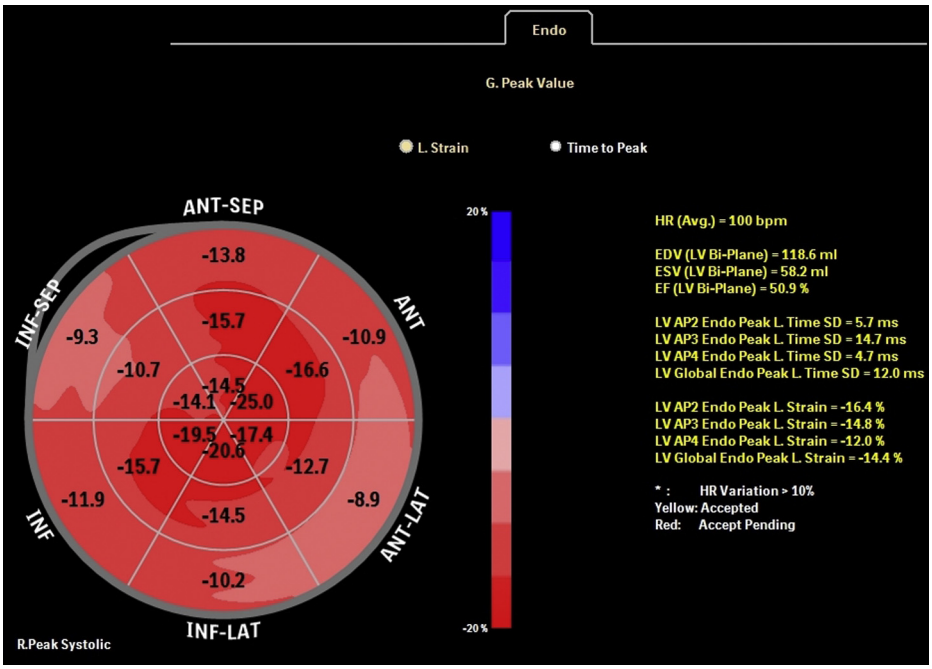

Figure 1 Bull's-eye strain display for our patient with AML at baseline prior to administration of chemotherapy. There was reduction in longitudinal strain values especially over the basal inferoseptal and anterolateral regions.

cardiotoxicity is a well-known entity, it manifests as a reduction in LV ejection fraction and dilated cardiomyopathy rather than hemorrhagic pericardial effusion.[10] The short duration and low cumulative dose of daunorubicin used in our patient also made anthracycline-induced cardiotoxicity unlikely.[10]

Effusive-Constrictive Pericarditis

Effusive-constrictive pericarditis is a rare entity in the spectrum of pericarditis syndromes, with an incidence of 6.9% among patients with clinical tamponade.[11] It is a transitional occurrence between acute effusive pericarditis with tamponade and chronic constrictive pericarditis (CP), while showing features of both entities. The proposed pathophysiology of ECP is the presence of pericardial effusion that is associated with edema, inflammation, or thickening of the visceral pericardium, leading to a constrictive physiology. Another possible mechanism is the presence of fibrinous strands that cause loculation of pericardial effusion to the visceral pericardium leading to constriction.[4] Consistent with previously reported cases, our patient showed the presence of hyperechoic intrapericardial material at the time of diagnosis of ECP, which contributed to the constrictive physiology.[4] Such material was not visible on the initial echocardiogram on day 1. At the time of cardiac tamponade, fibrinous stranding that was hyperechoic was already visualized. As discussed previously, this could be due to spontaneous hemorrhage into the pericardial cavity from leukemic invasion and bleeding diathesis. The presence of hyperechoic material after pericardiocentesis can either be residual

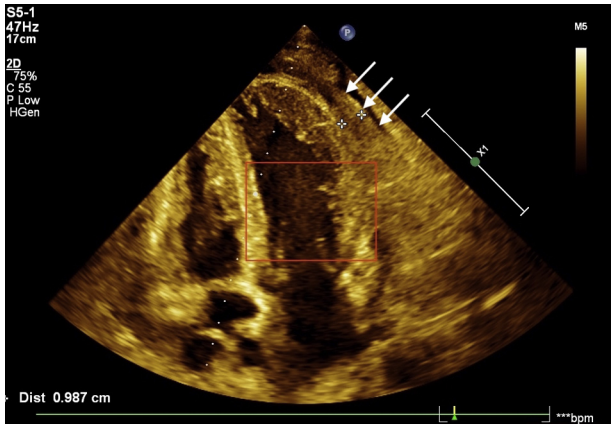

Figure 2 Two-dimensional TTE in the apical 4-chamber view demonstrated hyperechoic material *(arrows)* in the pericardial space after successful pericardiocentesis.

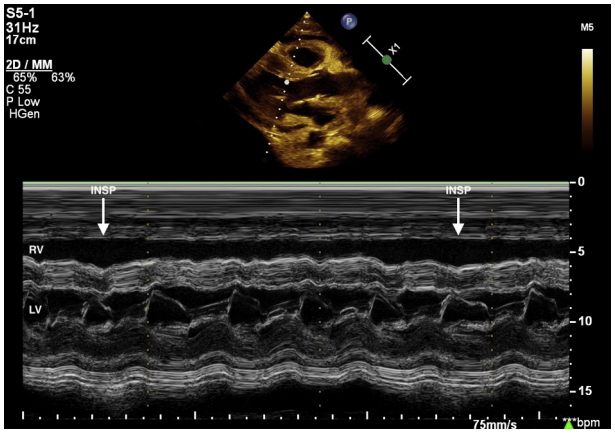

Figure 3 M-mode echocardiogram at the parasternal long-axis view demonstrated a shift of the interventricular septum toward the left ventricle on inspiration *(arrows)*, which signified increased interventricular dependence.

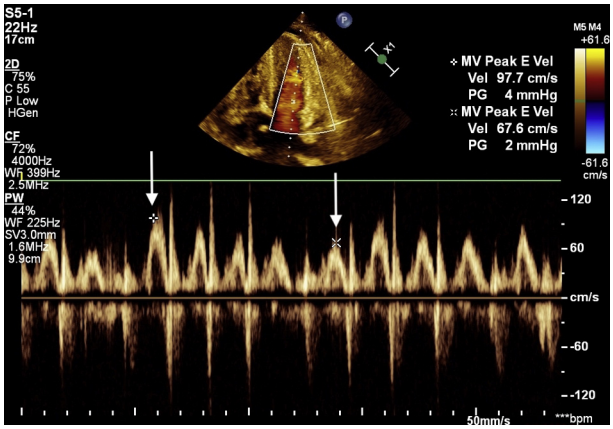

Figure 4 Pulsed-wave Doppler of the mitral valve E-wave peak velocity demonstrated a marked respiratory variation of >30% *(arrows)*.

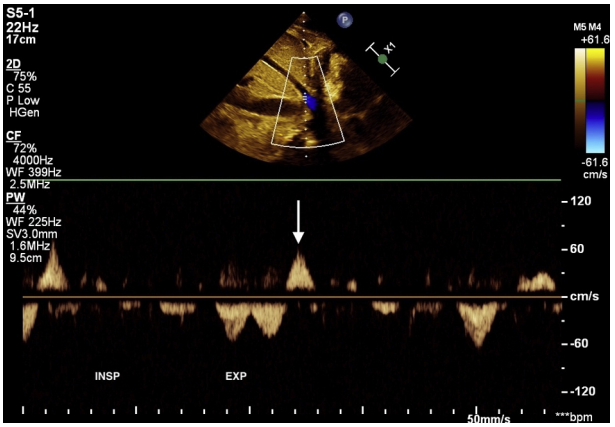

Figure 6 Pulsed-wave Doppler of the hepatic vein demonstrated marked diastolic flow reversal during expiration *(arrow)*.

hemorrhagic pericardial effusion or iatrogenic bleeding that occurred because of the pericardiocentesis.

The gold standard for diagnosis of ECP used to be cardiac catheterization showing the hallmark feature of persistent elevation of right atrial pressure despite normalization of intrapericardial pressure. Persistent RV and LV end-diastolic pressures are also observed. Display of LV pressures at cardiac catheterization shows the classical "dip-and-plateau" morphology.[4,11] Echocardiography has been increasingly used for noninvasive diagnosis, and invasive hemodynamic assessment for ECP diagnosis is currently reserved for a subgroup of cases with inconclusive echocardiographic findings. The diagnostic criteria proposed for the demonstration of constrictive physiology included respirophasic ventricular septal shift, normal or increased medial mitral annular tissue velocity (e') ≥ 9 cm/sec, hepatic vein expiratory diastolic flow reversal, variation in mitral inflow E-wave peak velocity of at least 14.6%, and the ratio of medial to lateral e' ≥ 0.9.[12] The first 3 criteria were considered the most important, as each of these were independently associated with the presence of CP.[12] The presence of all 3 features had a sensitivity of 64% and specificity of 97% in diagnosing ECP. Our patient met all 5 diagnostic criteria of constrictive physiology in the setting of postpericardiocentesis for cardiac tamponade, confirming the diagnosis of ECP. Cardiac catheterization was not performed in our patient due to his rapid clinical deterioration and significant bleeding risk due to severe thrombocytopenia.

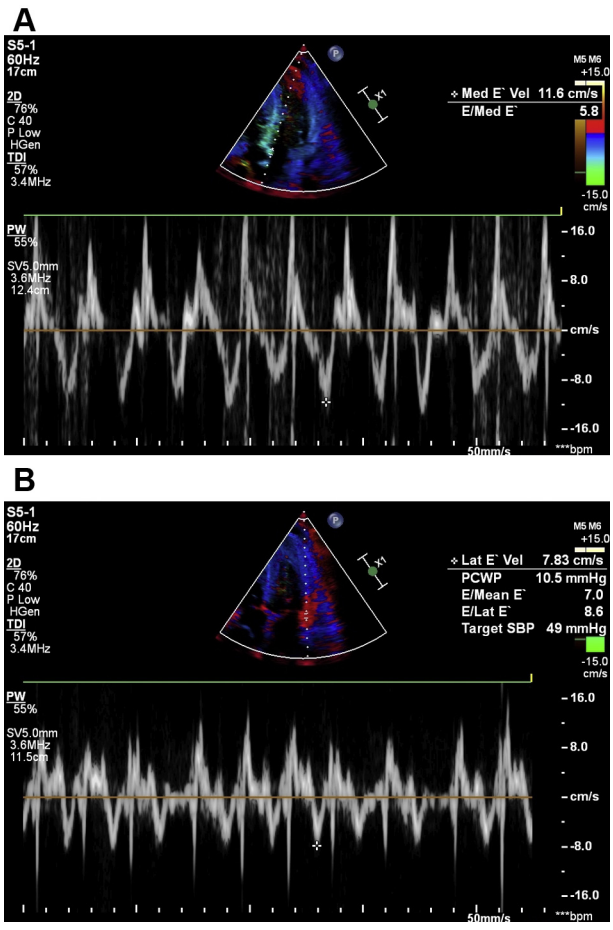

Figure 5 Tissue Doppler interrogation of the mitral annulus early diastolic velocity demonstrated annulus reversus with an increased medial e' of 12 cm/sec **(A)** and lower lateral e' of 8 cm/sec **(B)**.

Most patients with ECP had favorable outcomes.[5,13] Mortality has been reported in patients with underlying malignancy.[13] Spontaneous resolution of ECP was observed in cases of idiopathic etiology.[11] The use of colchicine, nonsteroidal anti-inflammatory drugs, or steroids in ECP were reported in existing literature.[5,13] In a study of 33 patients with ECP, 92% (24 of 26 patients who followed up and had progress echocardiography) showed clinical and echocardiographic resolution of ECP features following medical therapy.[13] Surgical pericardiectomy was performed in selected candidates with poor response to medical treatment.[4,13] The unfavorable outcome in our case could be attributed to multiple factors. Cardiac arrest on presentation of cardiac tamponade led to impaired LV systolic function. Limitation of preload due to ECP by hyperechoic material in the pericardium on top of an impaired systolic function post–cardiac arrest may have caused further hemodynamic compromise.

Effect of Mechanical Ventilation on Pericardial Diseases

Lutz *et al.*[5] previously reported a similar case of AML presenting as cardiac tamponade and ECP. In contrast to this previous case report, our patient was critically ill and required IMPPV, which could demonstrate the interesting interaction between mechanical ventilation and

pericardial diseases. The initiation of IMPPV in the setting of cardiac tamponade may have contributed to the cardiac arrest that occurred shortly after intubation in our patient. The positive intrathoracic pressure throughout inspiratory and expiratory phases in mandatory ventilator breaths reduces RV preload. The use of high positive end expiratory pressure may also cause alveolar overdistension and increase in RV afterload. In combination with impaired right-sided filling during tamponade, a sudden reduction in RV output due to mechanical ventilation and consequent reduction in LV output may lead to shock and cardiac arrest. In cases when the diagnosis of cardiac tamponade has already been established, preload augmentation is of utmost importance, and pericardial drainage should precede the initiation of IMPPV to prevent fatal hemodynamic collapse.

The use of IMPPV may also have interesting implications for the echocardiographic diagnosis of cardiac tamponade and CP. In spontaneously breathing subjects with cardiac tamponade, negative inspiratory intrapleural pressure augments venous return. Increase in right heart volume is accommodated by left shift of the interventricular septum due to the reduced pericardial compliance, causing marked ventricular septal shift. In contrast, mechanically ventilated patients have positive intrapleural pressure throughout the respiratory cycle. The subsequent reduction in RV preload results in a less marked shift of interventricular septum. For similar reasons, in spontaneously breathing subjects, inspiration induces a marked increase in transtricuspid flow and a corresponding reduction in transmitral flow, forming the basis for diagnostic criteria in transvalvular flow velocity for cardiac tamponade.[4] Patients under IMPPV experience less marked variation in transvalvular flow velocities compared with spontaneously breathing patients. This concept was shown in a canine model that demonstrated nonexistent respiratory variation in transmitral flow velocity in mechanically ventilated dogs with cardiac tamponade.[14]

Constrictive pericarditis shares similarities with cardiac tamponade in terms of reduced pericardial compliance and increased ventricular interdependence. However, the pathophysiology is markedly different: cardiac tamponade is a pathology of RV filling, while CP is a pathology of LV filling. In spontaneously breathing subjects, pericardial pressure is isolated from the change in intrathoracic pressure induced by inspiration, due to the thickened pericardium. Spontaneous inspiration decreases pulmonary venous pressure and hence the pressure gradient of LV diastolic filling and lowers the transmitral flow velocity during spontaneous inspiration. During positive pressure ventilation (PPV), the positive intrathoracic pressure increases LV filling and increases transmitral flow velocity during the inspiratory phase of mechanical breaths. This reversal in the pattern of respiratory variation of transmitral flow velocities during PPV was delicately described by Abdalla *et al.*[15] Despite the reversal in pattern, the authors reported a similar percentage change in the respiratory variation induced by PPV compared with that in spontaneously breathing subjects[15]; however, there is no validation of the diagnostic value of septal bouncing in CP for patients on mechanical ventilation.

To conclude, patients with cardiac tamponade have diminished ventricular septal shift and respiratory variation of transvalvular flow velocities when being put on PPV. In CP, reversal in pattern of respiratory variation in transvalvular flow is observed during PPV, although the magnitude of respiratory variation remains similar to that during spontaneous breathing. Currently, there are no specific guidelines to diagnose these conditions in mechanically ventilated patients. We must consider these variations in the diagnosis of pericardial disease in mechanically ventilated patients.

Limitations

Electrocardiogram and respirometer recording were not included in our images due to the urgent situation of resuscitating the unstable and deteriorating patient.

CONCLUSION

We report a rare case of ECP occurring in an AML patient. The use of cardiac POCUS can aid the timely diagnosis of cardiac tamponade, which uncommonly occurs in patients with hematological malignancies. Effusive-constrictive pericarditis should be suspected in patients without clinical improvement following successful pericardiocentesis. Echocardiographic assessment aids in the noninvasive diagnosis of ECP. This uncommon pericardial syndrome should be looked for precisely and should not be missed, as it is potentially reversible and the outcome is favorable in most patients is possible. Important heart-lung interactions occur in patients on IMPPV with pericardial diseases.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2022.04.011.

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