

# Point-of-Care Ultrasound: A Case Series of Potential Pitfalls



Ghislaine Douflé, MD, MEd, Ricardo Teijeiro-Paradis, MD, Diana Morales-Castro, MD, MSc, Martin Urner, MD, Alberto Goffi, MD, Laura Dragoi, MSc, and Filio Billia, MD, PhD, *Toronto, Ontario, Canada*

## INTRODUCTION

In recent years, point-of-care ultrasound (POCUS) has gained wide acceptance among acute care physicians. POCUS facilitates the rapid diagnosis of several life-threatening conditions, potentially leading to changes in clinical decision-making.<sup>1</sup> Consequently, many critical care medicine, anesthesiology, and emergency medicine programs have integrated POCUS into their core training program.<sup>2</sup> However, the lack of available trained supervisors represents a major challenge for many programs.<sup>3-6</sup> As a result, various programs deliver training over a short period of time with workshops or elective rotations.<sup>4,5,7-9</sup> Recommendations for echocardiography laboratories' participation in POCUS training allude to the fact that some physicians may have misconceptions around POCUS training for image acquisition and interpretation, and warn against an expectation of quick mastery.<sup>10</sup> While many report the effectiveness of short courses with good long-term retention, others have found that long-term retention was poor.<sup>7,11-14</sup> After a short training period, physicians may not be sufficiently prepared to safely perform POCUS in a clinical setting.<sup>11,12,14,15</sup> However, data on diagnostic errors associated with the use of POCUS in the acute care setting are scarce.<sup>15-21</sup>

We report 3 cases where the conclusions based on cardiac POCUS were discordant with the diagnostic findings of a comprehensive echocardiogram. We provide examples of potential pitfalls of POCUS and discuss our quality improvement initiative.

From the Interdepartmental Division of Critical Care Medicine (G.D., R.T.-P., D.M.-C., M.U., A.G., L.D.) and Institute of Health Policy, Management, and Evaluation (G.D., M.U.), University of Toronto; Department of Anesthesia and Pain Management, Toronto General Hospital, University Health Network (G.D.); Department of Medicine and Li Ka Shing Knowledge Institute, Keenan Research Centre, St. Michael's Hospital, Unity Health Toronto (A.G.); and Division of Cardiology, Peter Munk Cardiac Center, University Health Network (F.B.), Toronto, Ontario, Canada.

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Correspondence: Ghislaine Douflé, Toronto General Hospital, 9 Mars Building, 585 University Avenue, Toronto, Ontario M5G 2N2. (E-mail: [ghislaine.doufle@mail.utoronto.ca](mailto:ghislaine.doufle@mail.utoronto.ca)).

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## CASE PRESENTATIONS

### Case 1

Our first patient was a 61-year-old man who presented to the emergency department with abdominal pain. On examination, he had a blood pressure of 107/69 mm Hg and a heart rate of 120 beats/min. The patient reported epigastric pain without tenderness. His extremities were warm and dry on examination, without peripheral edema. He complained of mild shortness of breath. His pulse oximetry was 98% on 3 L/min of supplemental oxygen. Pulmonary auscultation did not reveal any pathologic findings and cardiac auscultation was not documented. The chest radiography showed pulmonary venous congestion. The electrocardiogram showed atrial fibrillation.

A POCUS was performed by the emergency medicine resident, but no echocardiographic images were stored. The medical records stated: "The inferior vena cava (IVC) is not plethoric, there is no pericardial effusion nor abdominal aortic aneurysm." The patient had an acute kidney injury (AKI; creatinine 297  $\mu\text{mol/L}$ ,  $\text{K}^+$  6.4 mmol/L) and mild increase in liver enzymes (AST, 79; ALT, 95 units/L; bilirubin, 39  $\mu\text{mol/L}$ ). The troponin was within the normal range. A non-contrast abdominal computed tomography (CT) showed peripancreatic fat stranding and the radiologist raised concerns for acute pancreatitis; both amylase and lipase were normal. The emergency medicine resident concluded that the shortness of breath was unlikely to be due to congestive heart failure and attributed the AKI to hypovolemia. The patient was admitted to general surgery with a working diagnosis of acute pancreatitis.

Supported by the note summarizing the conclusions of the POCUS examination, the patient was given 4 L of fluids. The intensive care unit (ICU) team was consulted overnight on the day of admission because of worsening renal function with persistent oliguria. He received additional fluids (1 L) and subsequently diuretics but remained oliguric. The next day, a follow-up assessment noted an elevated jugular venous pressure (6 cm), and a grade 3/6 systolic murmur radiating to the axilla was heard upon cardiac auscultation. The transthoracic echocardiogram (requested 24 hours after admission to define the murmur) showed severe mitral regurgitation with a flail posterior leaflet (anterior scallop, P1) with signs of pulmonary hypertension and severe tricuspid regurgitation (Figure 1, Videos 1-4). The patient was transferred to the cardiac ICU. Aggressive diuresis was initiated with a rapid improvement of his dyspnea, AKI, liver congestion, and abdominal pain. The patient was eventually referred to cardiac surgery and underwent a successful mitral valve repair. The remainder of his clinical course was uncomplicated.

### Case 2

Our second patient was a 34-year-old man who had recently undergone pulmonary endarterectomy for chronic thromboembolic

## VIDEO HIGHLIGHTS

**Video 1:** Two-dimensional transthoracic parasternal long-axis view of patient 1 demonstrates a dilated left atrium and a physiologic pericardial effusion. There is no significant abnormality visualized on the portions of the mitral valve visible on this view (segments A2-P2). *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

**Video 2:** Two-dimensional transthoracic apical 4-chamber view of patient 1. There is biatrial enlargement. There is no significant abnormality visualized on the portions of the mitral valve imaged on this view. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

**Video 3:** Two-dimensional modified transthoracic parasternal long-axis view of patient 1 zoomed on the mitral valve. This view demonstrates anterior portions of the mitral valve leaflets exhibiting a flail posterior leaflet (*arrow* pointing at the anterior scallop of the posterior mitral valve leaflet: P1). *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

**Video 4:** Two-dimensional transthoracic apical 5-chamber view of patient 1 demonstrates the flail posterior mitral valve leaflet (*arrow*: anterior scallop P1). *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

**Video 5:** Two-dimensional transthoracic parasternal long-axis view of patient 2 demonstrates a dilated right ventricle and a small pericardial effusion. *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

**Video 6:** Two-dimensional transthoracic parasternal short-axis view of patient 2 demonstrates signs of RV volume and pressure overload with flattening of the interventricular septum throughout the cardiac cycle. *RV*, Right ventricle; *LV*, left ventricle.

**Video 7:** Two-dimensional transthoracic apical 4-chamber view of patient 2 demonstrating right atrial and ventricular enlargement. Right ventricular hypertrophy can also be appreciated. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

**Video 8:** Two-dimensional transthoracic modified apical 4-chamber view of patient 2 on postoperative day 14 post-pulmonary endarterectomy demonstrates the pericardial effusion mainly adjacent to the LV (*arrow*: pericardial effusion). *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

**Video 9:** Two-dimensional transthoracic parasternal long-axis view of patient 2 on postoperative day 19 post-pulmonary endarterectomy (15.5 hours after the POCUS). A large circumferential pericardial effusion can be seen along both ventricles (*arrows*: pericardial effusion). Adhesions can also be visualized between the parietal pericardium and the inferolateral wall of the LV (the adhesion is encircled by the *white circle*). *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

**Video 10:** Two-dimensional transthoracic parasternal short-axis view of patient 2 on postoperative day 19 post-pulmonary endarterectomy demonstrates a large circumferential pericardial effusion (*white arrows*: pericardial effusion). There is early diastolic collapse of the right ventricle and diastolic collapse of the left ventricular lateral wall (the *red line* helps identify the flattening of the anterolateral wall of the LV). The effusion is complex, and the adhesions between the pericardial sac and the inferolateral wall of the LV can be further appreciated (the adhesions are encircled by the *white circle*). *RV*, Right ventricle; *LV*, left ventricle.

**Video 11:** Two-dimensional transthoracic apical 4-chamber view of patient 2 on postoperative day 19 post-pulmonary endarterectomy. The view is focused on the LV and demonstrates the pericardial effusion adjacent to the left ventricular wall (*arrow*: pericardial effusion). *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

**Video 12:** Two-dimensional transthoracic subcostal 4-chamber view of patient 2 on postoperative day 19 post-pulmonary endarterectomy. The effusion is seen along both ventricles (*arrows*: pericardial effusion). *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium.

**Video 13:** Two-dimensional transthoracic subcostal view of the IVC of patient 2 on postoperative day 19 post-pulmonary endarterectomy. The IVC is dilated (2.2 cm), and there are no respiratory variations visualized.

**Video 14:** Monitor tracings of patient 2 upon ICU admission on postoperative day 19 post-pulmonary endarterectomy. Electrical alternans and pulsus paradoxus can be appreciated.

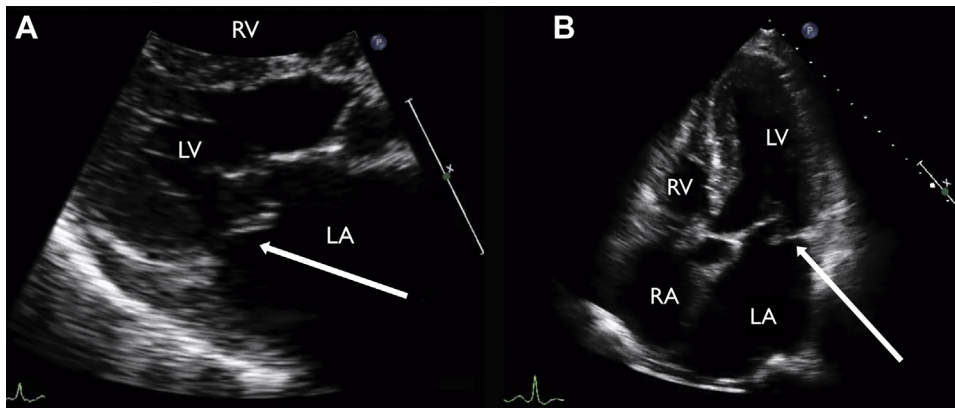
**Video 15:** Two-dimensional transthoracic parasternal long-axis view of patient 3 demonstrates a diastolic doming of the anterior mitral valve leaflet and left atrial enlargement (the *white line* is emphasizing the diastolic doming of the anterior mitral valve leaflet). *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

**Video 16:** Two-dimensional transthoracic apical 4-chamber view of patient 3 with color-flow Doppler across the mitral valve. Proximal isovelocity surface area hemispheres can be visualized at a Nyquist velocity limit of 60 cm/sec, indicating a significant acceleration of flow across the mitral valve (*arrow* pointing at the proximal isovelocity surface area hemispheres). *LA*, Left atrium; *RA*, right atrium; *RV*, right ventricle.

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pulmonary hypertension (Videos 5-7). His preoperative mean pulmonary artery pressure was 50 mm Hg with a systolic pulmonary artery pressure of 79 mm Hg on right heart catheterization. His immediate postoperative course was uncomplicated and he was transferred to the surgical ward on therapeutic anticoagulation. Two weeks after

his surgery, he developed leukocytosis and antibiotics were started due to suspicion of surgical site infection. A comprehensive transthoracic echocardiogram was requested to rule out tamponade. It showed evidence of a moderate pericardial effusion without echocardiographic signs of tamponade (Figure 2, Video 8). The report

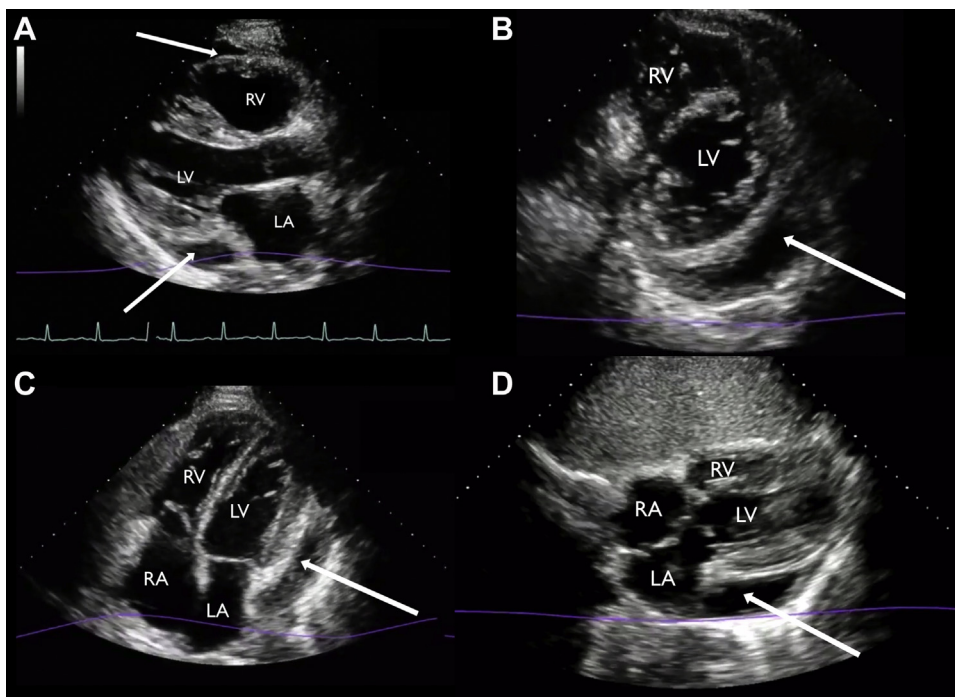


**Figure 1** Two-dimensional transthoracic views of patient 1. **(A)** Two-dimensional modified transthoracic parasternal long-axis view end-systolic frame zoomed on the mitral valve. This view demonstrates the anterior portions of the mitral valve exhibiting a flail posterior leaflet (*arrow*: anterior scallop of the posterior mitral valve leaflet P1). **(B)** Two-dimensional transthoracic apical 5-chamber view end-systolic frame demonstrates the flail posterior mitral valve leaflet (*arrow*). LA, Left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

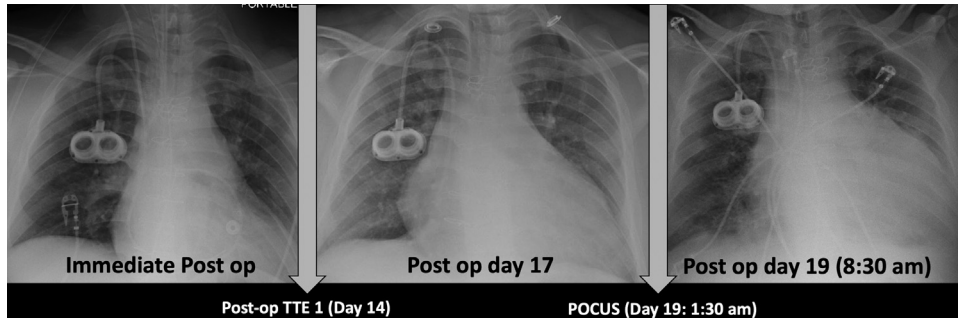
acknowledged that signs of tamponade may be masked in the setting of pulmonary hypertension.

Over the following days, the patient developed an AKI with a creatinine that reached 239  $\mu\text{mol/L}$  from a baseline of 60  $\mu\text{mol/L}$ . He was also transiently hypotensive. There was concern that he may have

developed cardiac tamponade, and the on-call cardiology team was consulted for hypotension and worsening AKI. A POCUS was performed overnight (postoperative day 19 at 1:30 a.m.) by the junior cardiology resident on call. The written POCUS report concluded the following: “Limited views were obtained due to post surgical



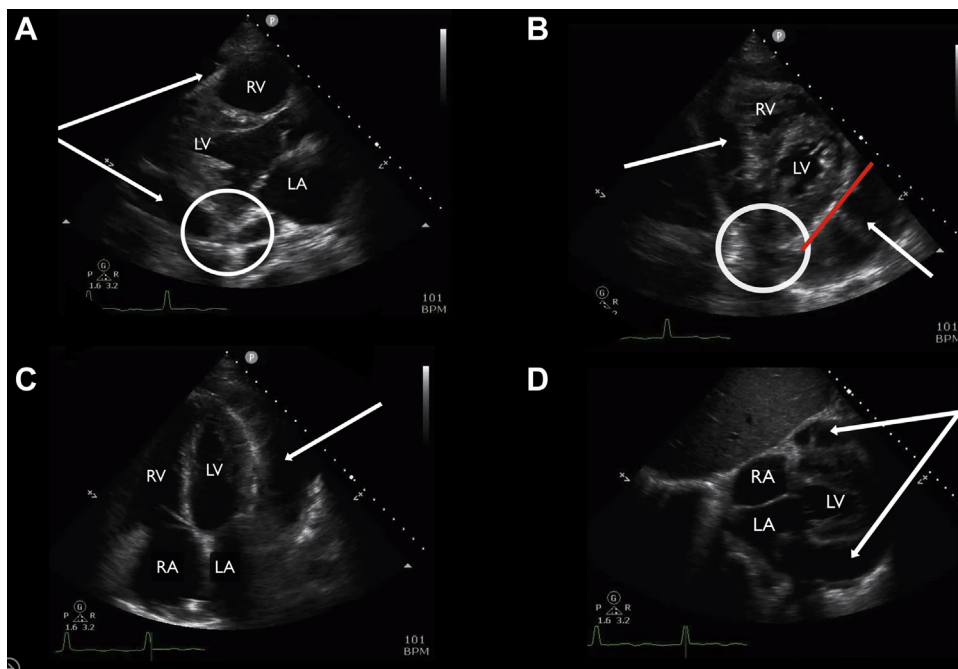
**Figure 2** Two-dimensional transthoracic views of patient 2 on postoperative day 14 post-pulmonary endarterectomy. **(A)** Two-dimensional transthoracic parasternal long-axis view end-diastolic frame with a pericardial effusion anterior to the RV and along the LV (*arrows*: pericardial effusion). **(B)** Two-dimensional transthoracic parasternal short-axis view end-diastolic frame demonstrates the pericardial effusion adjacent to the LV (*arrow*: pericardial effusion). **(C)** Two-dimensional transthoracic apical 4-chamber view end-diastolic frame with the pericardial effusion adjacent to the lateral wall of the LV (*arrow*: pericardial effusion). **(D)** Two-dimensional transthoracic subcostal 4-chamber view demonstrates the pericardial effusion along the lateral wall of the LV (*arrow*: pericardial effusion). LA, Left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.



**Figure 3** Chest radiographies of patient 2 post-pulmonary endarterectomy. *Left panel:* immediate postoperative chest radiography. *Middle panel:* chest radiography on postop day 17 (this chest x-ray was obtained 3 days after the first postoperative transthoracic echocardiogram, which was requested to rule out tamponade). There is a significant enlargement of the cardiac silhouette compared to the immediate postoperative chest radiography. *Right panel:* chest radiography on postop day 19 (7 hours after the POCUS) demonstrates further enlargement of the cardiac silhouette compared to the previous chest radiography.

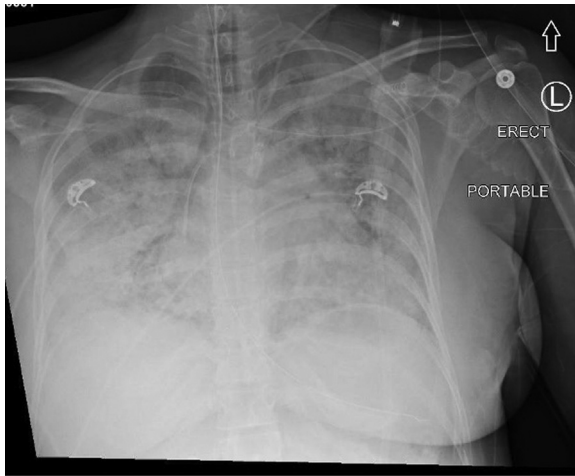
incision. The left ventricle (LV) is grossly normal, while there is right ventricular (RV) dilation and septal dyssynergy. The IVC measures 2.0 cm with more than 50% collapse on inspiration. There is no pericardial effusion visualized, nor inversion of the right atrium (RA) or RV. There is 12 and 21% inflow variation across the mitral and tricuspid valves, respectively.” No images from this study were stored for future review. The resident concluded that the AKI was secondary to RV failure and recommended a repeat comprehensive echocardiogram in the morning. The on-call resident also ordered a chest radiography, which was obtained in the morning (Figure 3). The repeat comprehensive echocardiogram was not ordered by the surgical team.

As the AKI continued to worsen over the course of the day, the ICU team was consulted (postoperative day 19 at 5 p.m.). A critical care echocardiogram was repeated by the ICU team, and a large circumferential complex pericardial effusion was found (maximal diameter 3.5 cm lateral to the LV; Figure 4, Videos 9-13). There was no RA inversion and only a subtle early diastolic inversion of the RV. There was evidence of flattening of the anterior and anterolateral wall of the LV. The effusion had significantly increased in size compared with the previous echocardiogram performed a few days prior. Therapeutic anticoagulation was immediately discontinued, and the patient was admitted to ICU for further management. Upon admission, he was hypotensive with a mean arterial pressure



**Figure 4** Two-dimensional transthoracic views of patient 2 on postoperative day 19 post-pulmonary endarterectomy. **(A)** Parasternal long-axis view (diastolic frame) demonstrates a large circumferential pericardial effusion (arrows). Adhesions visualized between the parietal pericardium and the inferolateral wall of the (LV; white circle). **(B)** Parasternal short-axis view (diastolic frame) demonstrates a large circumferential and complex pericardial effusion (arrows) with adhesions (white circle). Hemodynamic significance is demonstrated by flattening of the anterolateral LV wall (red line). **(C)** Apical 4-chamber view (diastolic frame) focused on the LV demonstrates the large pericardial effusion seen along the left ventricular wall (arrow). **(D)** Subcostal 4-chamber view (diastolic frame) demonstrates the large pericardial effusion along both ventricles (arrows). LA, Left atrium; RA, right atrium; RV, right ventricle.





**Figure 5** Chest radiography of patient 3 admitted for acute respiratory failure demonstrates bilateral infiltrates and air bronchograms.

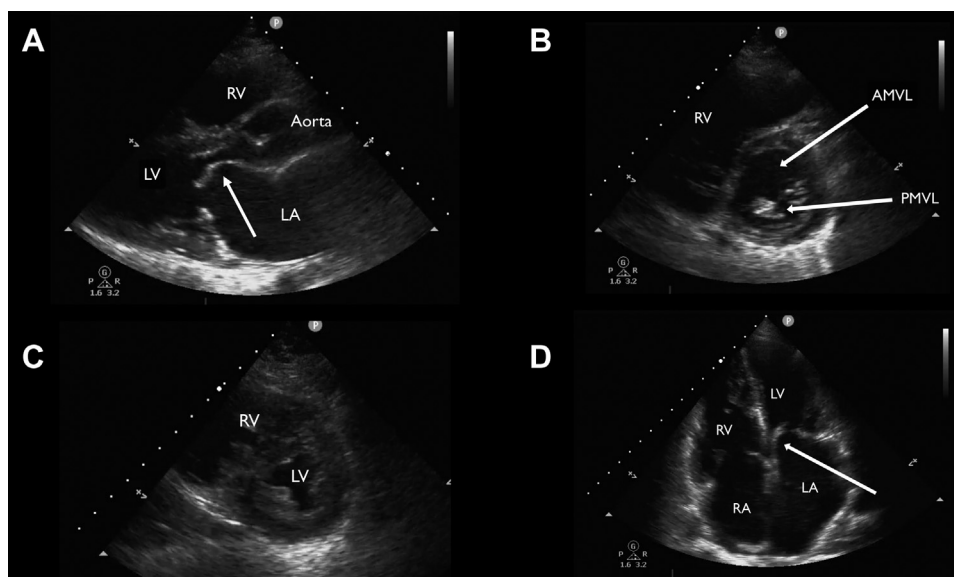
of 60 mm Hg, and support with vasopressor had to be initiated. The arterial line tracing showed a significant pulsus paradoxus, and the electrocardiogram exhibited electrical alternans (Video 14). Given the location of the effusion, the clinical team decided to proceed with a CT-guided pericardiocentesis. Approximately 1 L of blood-tinged fluid was drained with subsequent hemodynamic improvement and progressive resolution of the AKI. A repeat transthoracic echocardiogram postpericardiocentesis showed a trivial residual pericardial effusion. The patient was discharged from the ICU within 48 hours and subsequently home.

### Case 3

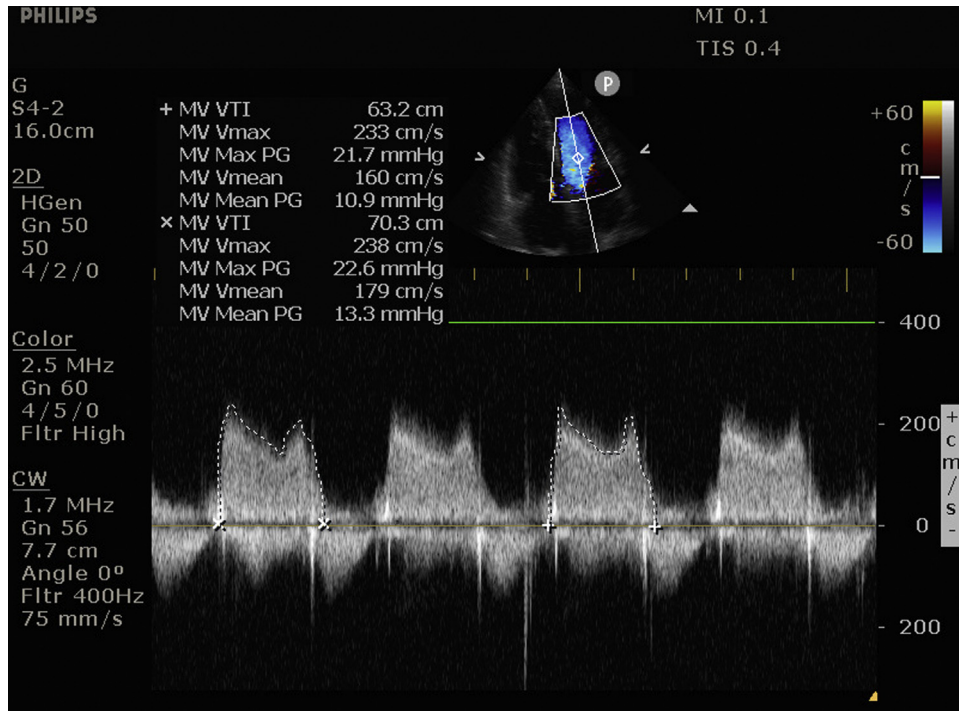
Our third case was a 33-year-old pregnant woman (29-week pregnancy, G4P0) who presented to a peripheral hospital with acute onset dyspnea and hemoptysis. The chest radiography documented bilateral interstitial infiltrates and alveolar consolidation (Figure 5).

The chest CT reported diffuse parenchymal opacities and the absence of pulmonary embolism. The differential diagnoses included pulmonary edema, multifocal pneumonia with or without pulmonary hemorrhage, and diffuse alveolar damage. Her COVID test was negative. The patient eventually required intubation and was subsequently transferred to our center for further management of high-risk pregnancy. On examination, she was sedated, afebrile, and tachycardic (heart rate, 118 beats/min) and required vasopressors to maintain a mean arterial pressure above 65 mm Hg. Her ratio of partial pressure of arterial oxygen-to-inspiratory fraction of oxygen ( $\text{PaO}_2/\text{FiO}_2$  ratio) was 118 mm Hg. Cardiac auscultation was not performed. The clinical examination of the abdomen was unremarkable besides the gravid uterus. A complete septic workup was ordered, including repeat COVID testing. The patient's blood work only showed leukocytosis (white cell count,  $15 \times 10^9/\text{L}$ ) with the remaining values within normal ranges.

A cardiac POCUS performed on admission by the critical care fellow was verbally reported as normal. No images were recorded for future review. Based on these findings, the patient was treated as septic shock secondary to a pneumonia with administration of fluids, antibiotics, and vasopressors. Within the 48 hours following admission, the patient's cumulative fluid balance was positive 5 L. A repeat CT with contrast excluded a pulmonary embolism. As she continued to deteriorate with new-onset AKI, additional differential diagnoses were being considered, including pulmonary-renal syndrome secondary to autoimmune vasculitis. A comprehensive transthoracic



**Figure 6** Two-dimensional transthoracic views of patient 3 admitted for acute respiratory failure. (A) Parasternal long-axis view (diastolic frame) demonstrates the diastolic doming of the AMVL (arrow) and left atrial enlargement. (B) Parasternal short-axis view at the mitral valve level (systolic frame) demonstrates thickened mitral valve leaflets (arrows). (C) Short-axis view at the midpapillary level (systolic frame) demonstrates a septal flattening indicative of RV pressure overload. (D) Apical 4-chamber view (diastolic frame) with significant biatrial enlargement and doming of the anterior leaflet of the mitral valve (white arrow). AMVL, Anterior mitral valve leaflet; LA, left atrium; LV, left ventricle; PMVL, posterior mitral valve leaflet; RA, right atrium; RV, right ventricle.



**Figure 7** Two-dimensional transthoracic transmitral flow tracing of patient 3 measured on the apical 4-chamber view with continuous-wave Doppler across the mitral valve. The mitral valve mean pressure gradient (MV mean PG) is between 10 and 13 mm Hg (heart rate, 87 beats/min at the time of the echocardiogram).

echocardiogram was requested 36 hours after admission for respiratory failure and documented 48 hours after admission; it showed severe mitral stenosis with features of rheumatic heart disease (Figures 6 and 7, Videos 15 and 16). All septic and autoimmune workups were negative; fluids and antibiotics were replaced with diuretics and  $\beta$ -blockers for heart failure management. Despite aggressive attempts to medically optimize her clinical condition, she failed to improve. A balloon valvuloplasty of the mitral valve was performed 13 days postadmission. Subsequently, her clinical condition rapidly improved and she was extubated 3 days later. She delivered a full-term healthy baby and was discharged home with a planned cardiology follow-up.

## DISCUSSION

POCUS is a valuable noninvasive diagnostic tool that can facilitate rapid decision-making at the bedside. In the present work, we present 3 cases illustrating the potential pitfalls of POCUS performed with suboptimal integration of the clinical context with other diagnostic tests.

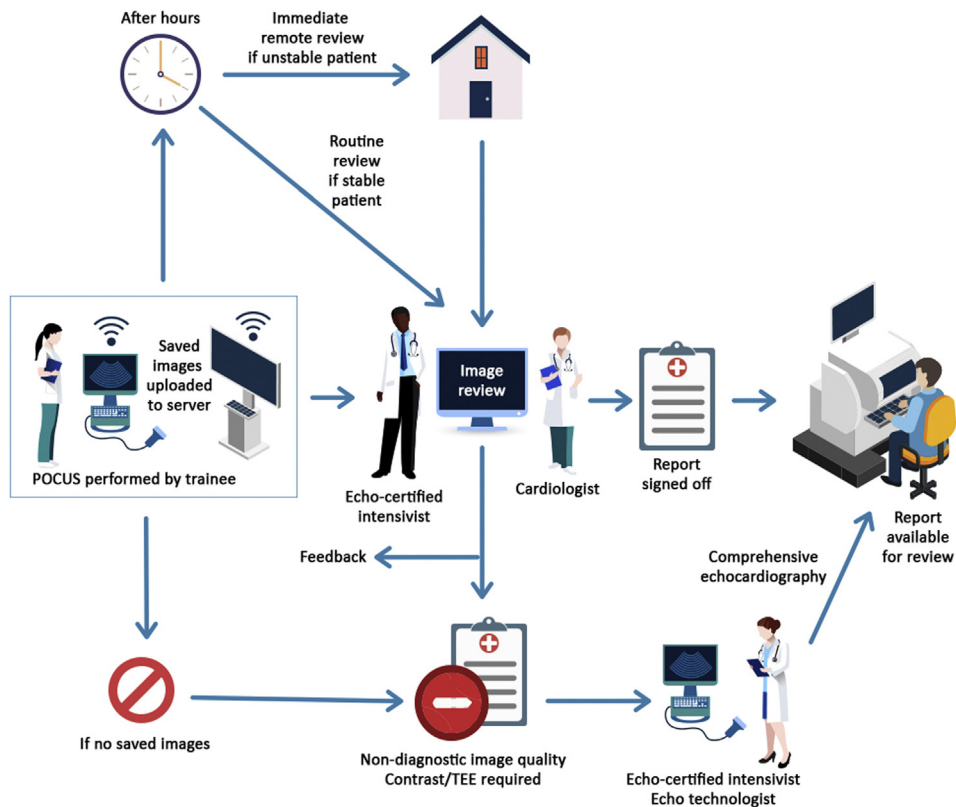
International recommendations emphasize that POCUS should be used as an adjunct to clinical examination with the goal to identify a defined list of potential clinical diagnoses and that POCUS is not equivalent to comprehensive echocardiography.<sup>22,23</sup> Hence, any suspicion of abnormalities beyond the scope of POCUS should be followed with a comprehensive echocardiogram.<sup>22,24</sup> Furthermore, experts warn that competency in POCUS cannot be achieved in a few days.<sup>22</sup> Although training can be delivered over a short period of time, it should be followed by longitudinal supervised practice. Finally, it is recommended that POCUS studies be archived and available for review by a trained echocardiographer.<sup>25,26</sup> These recom-

mendations were only partially followed in the cases presented. Our cases highlight 3 key issues: (1) integration of POCUS findings with pretest probability, clinical findings, and other diagnostic tests; (2) variability in training; (3) lack of archiving and documentation.

### Integration of POCUS Findings in the Clinical Context and with Other Diagnostic Tests

Clinical examination was incomplete in all 3 patients. In the 2 patients with valvular disease, cardiac auscultation might have detected a murmur and prompted a comprehensive study, but it was either not documented or not performed in our cases. Admittedly, it may be challenging to hear a diastolic murmur on a mechanically ventilated patient, and a murmur may not be audible in acute mitral regurgitation. Nevertheless, while in the setting of acute mitral regurgitation, the left atrium might not be dilated, one would expect a clinical presentation in keeping with an acute pathology of the mitral valve (such as severe respiratory failure, unilateral opacity on the chest x-ray, and possibly circulatory shock).<sup>27</sup> Our first patient exhibited echocardiographic signs suggesting either significant diastolic dysfunction or chronic valvulopathy (i.e., left atrial dilation), which could have been detected on any type of cardiac ultrasound. Also, the symptoms and clinical context were not consistent with an acute mitral insufficiency.

For the patient in tamponade, the presence of pulsus paradoxus, electrical alternans, and the significant increase in the size of the cardiac silhouette on the chest radiography (Figure 3) should have raised serious concerns and resulted in the necessary escalation of care. It is important to note, however, that even a comprehensive transthoracic



**Figure 8** Flow chart of archiving and review system. *TEE*, Transesophageal echocardiogram.

echocardiogram may fail to visualize a loculated effusion on a postsurgical patient and a transesophageal echocardiogram may be required.

The first case illustrates one of the potential inherent limitations of POCUS. As the anterior portion of the posterior leaflet was flail, it was not visualized on a limited POCUS protocol—which usually images the A2/P2 segments of the mitral valve. The flail leaflet was evident on the apical 5-chamber and on the remainder of a comprehensive echocardiogram. As outlined in the recommendations, POCUS practitioners may occasionally diagnose gross valvular abnormalities, but it is recommended that these patients should be referred for a comprehensive echocardiogram.<sup>22</sup> Additionally, since POCUS does not detect all pathologies (e.g., valvular pathologies, diastolic dysfunction), a comprehensive echocardiogram might still be needed despite a POCUS examination not displaying direct signs of valvular disease. Furthermore, integration with all forms of cardiac testing and cautious clinical reasoning related to POCUS findings are crucial.<sup>28</sup> Importantly, given the lack of consistent standards and the significant variability in POCUS protocols (number of views obtained, optional assessment of gross valvular pathologies, and use of Doppler), POCUS practitioners should be explicit about their scanning protocol, specify which structures were assessed, and mention structures that were not evaluated.<sup>8,29,30</sup> For example, in our first and third cases, a statement such as “valves not assessed” may have been helpful and prompted the team to request a comprehensive echocardiogram for specific valvular assessment.

#### Variability in Training

In our second case, it is unlikely that the pericardial effusion was not evident on the POCUS views, given its size and the widening of the

cardiac silhouette on chest radiography. An experienced echocardiographer would have recognized the presence of the pericardial effusion, compared its current size with the size on the previous echocardiogram, and immediately alerted the responsible clinical team. In both cases describing patients with mitral pathologies, the significant biatrial enlargement should have raised clinical suspicion. Furthermore, in the patient with rheumatic mitral stenosis, the typical diastolic doming of the mitral valve and pulmonary hypertension were present. While these findings would not have presented diagnostic challenges to the experienced eye, they were not recognized in the cases presented. POCUS practitioners are not expected to be experts in cardiac ultrasound, and certain POCUS protocols do not involve valvular assessment. Nevertheless, significant abnormalities such as severe atrial dilation and paradoxical septal motion are an integral part of any POCUS protocol but were not identified in our patients. Left atrial dilation is indicative of chronically elevated left atrial pressure. Paradoxical septal motion throughout the cardiac cycle denotes RV volume and pressure overload. In the setting of respiratory failure, presence of atrial dilation, and RV overload (cases 1 and 3), administration of large volume of fluids should have been avoided.

Given the absence of the POCUS images and specific information on the training undergone by the POCUS users in our cases, one can only speculate that insufficient training and experience may have contributed to the failure to identify and integrate critical findings in our patients.<sup>17,29</sup>

#### Lack of Archiving

In our institutions, all ultrasound systems have the capability to store images, yet POCUS images were not recorded in any of the cases

presented. All 3 comprehensive echocardiograms subsequently performed exhibited good echogenicity but could not be compared with the POCUS views. Without archived images, it is impossible to retrospectively understand whether the inconsistencies were secondary to poor image acquisition (i.e., nondiagnostic images on which the effusion or the flail leaflet were not visible) or to incorrect interpretation (findings were adequately captured but not recognized) by the physician performing the POCUS.

Systematic archiving allows ongoing education and quality assurance of POCUS practice. While some centers are able to archive POCUS images on a hospital-based server, it comes with a significant financial cost.<sup>31</sup> Furthermore, images saved by one department are not always accessible to other departments. Physicians who cannot access a hospital-based archive should still strive to record images on the ultrasound machine itself and review their recordings with experts. However, review of POCUS examinations by cardiology consultants may result in increased workload for the echocardiography laboratory. This may also lead to legal implications when a consultant is asked to interpret a suboptimal or incomplete POCUS.

In one of our ICUs, we have implemented a systematic archiving and review of POCUS performed by ICU physicians in collaboration with the echocardiography department (Figure 8). Intensive care unit trainees are instructed to save all their cardiac ultrasound images and upload them to our local echocardiographic data system. The cardiology, anesthesiology, and ICU departments share the same system and can access all studies performed within the institution.

Once uploaded, the images are reviewed by a National Board of Echocardiography–certified ICU attending who appraises the quality of the study and interpretation of the findings. If the ICU attending is not available, images are reviewed with an attending cardiologist.

After hours, the timing of the review depends on the patient's clinical condition. Should the patient be unstable, the images are immediately reviewed remotely by the ICU attending (or by an on-call cardiologist). The images are reviewed the following day for more stable patients.

Studies that were not saved are systematically repeated. It is difficult to quantify how often images are not saved, as there are no retrievable records. Reasons for not saving the images may include lack of familiarity with the protocol to save images, differences in expectations at other clinical sites, and reluctance to save suboptimal images.

Additionally, cardiac ultrasound images that do not reach diagnostic quality (or that require additional views or measurements) are repeated by an echo-certified intensivist (or cardiology trainee overnight), either immediately (depending on the patient's conditions) or the following morning. Comprehensive echocardiograms are performed by the echocardiography laboratory when there is no echo-certified intensivist available.

All cardiac ultrasounds performed by ICU physicians are reviewed and signed off by an echo-certified ICU attending. Once the report is validated, it is automatically transferred to the patient's medical record.

Our process has been particularly valuable during the COVID pandemic, as exposure of health care workers has been a concern.<sup>26</sup> Since the implementation of systematic archiving, only studies that are incomplete or suboptimal had to be repeated. Additionally, it has allowed ongoing education and assessment of ICU physicians performing POCUS.

Of note, none of the cases presented occurred in the ICU where this review process has been implemented.

## LIMITATIONS

Our report has several limitations. First, the lack of comparative images allowed us to only make assumptions on the underlying reasons for the diagnostic discrepancies. Second, we did not have detailed information on the ultrasound training undergone by the physicians involved in our cases. Lastly, although we report cases pertaining to POCUS, comprehensive echocardiography is not exempt from diagnostic errors and may equally fail to diagnose subtle findings.<sup>21</sup>

## CONCLUSION

In the presented cases, the initial interpretation of the cardiac ultrasound may have contributed to suboptimal therapeutic strategies and a delay in the provision of necessary treatments, which could have potentially resulted in catastrophic consequences. For our patients, a repeat echocardiogram was eventually performed, and appropriate treatment subsequently initiated.

While we only presented 3 exemplary cases, in our experience, discrepancies between POCUS findings and comprehensive echocardiogram are not infrequent.

While POCUS can be a powerful adjunct to clinical examination, it should not be used as a substitute for or as equivalent to a comprehensive echocardiogram. Physicians who want to incorporate POCUS in their clinical practice should be aware of its limitations, seek appropriate training, carefully integrate the findings within the clinical picture, and review their images with an expert to ensure patient safety and ongoing quality assurance.

## SUPPLEMENTARY DATA

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.case.2022.05.002>.

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