

Pitfalls of lung ultrasound interpretation in malignant pleural mesothelioma: an emergency department case report

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ABSTRACT

Lung ultrasound (LUS) is widely used in emergency departments (ED) for rapid evaluation of acute dyspnea and chest pain, but certain conditions may lead to misinterpretation of sonographic artifacts. We report a 70-year-old woman with malignant pleural mesothelioma (MPM) who presented with chest pain and dyspnea, in whom LUS demonstrated diffuse B-lines and pleural-based anechoic lesions mimicking cardiogenic pulmonary edema. The patient required non-invasive mechanical ventilation and was subsequently diagnosed with non-ST-elevation myocardial infarction. This case emphasizes that B-lines in oncologic patients may reflect pleural or subpleural pathology rather than interstitial edema and should be interpreted in conjunction with clinical and laboratory findings.

Keywords: Lung, ultrasonography, mesothelioma, emergency medicine, pleura

INTRODUCTION

Lung ultrasound (LUS) is increasingly utilized in emergency medicine (EM) as a rapid, bedside, radiation-free imaging tool for the evaluation of acute respiratory failure and chest pain. The fundamental principles and standardized interpretation of LUS artifacts were first systematically described by Lichtenstein,¹ forming the basis of modern LUS practice. The technique relies on the interpretation of pleural motion and ultrasound artifacts generated at the pleural-air interface rather than direct visualization of lung parenchyma.²

Under normal conditions, the presence of lung sliding and horizontal reverberation artifacts, known as A-lines, indicates normal lung aeration. In contrast, vertical hyperechoic artifacts extending from the pleural line to the bottom of the screen, referred to as B-lines, are typically associated with increased lung density and are most interpreted as interstitial syndrome, such as pulmonary edema. However, B-lines are not disease-specific and may also arise from non-cardiogenic causes, including pneumonia, pulmonary fibrosis, acute respiratory distress syndrome, and pleural-based malignancies.^{3,4} Malignant pleural mesothelioma (MPM) is an aggressive neoplasm originating from mesothelial cells and is frequently associated with pleural thickening, nodularity, and effusions, all of which may alter LUS findings.⁵

Although LUS has been described as a useful tool in detecting pleural masses and guiding biopsies in mesothelioma, reports focusing on LUS findings of mesothelioma patients presenting to the emergency department (ED) are scarce. In

this case report, we aim to emphasize the potential pitfalls in interpreting B-lines in patients with MPM from an EM perspective.

CASE

A 70-year-old woman presented to the ED with acute onset chest pain and progressive shortness of breath. Her medical history was significant for MPM, for which she was receiving active chemotherapy.

On arrival, her peripheral oxygen saturation was 88% on room air and increased to 94% with 2 L/min supplemental oxygen via nasal cannula. Vital signs revealed a blood pressure of 122/75 mmHg and sinus rhythm on electrocardiography, without ST-segment elevation. Physical examination demonstrated bilateral basal crackles without wheezing.

Initial laboratory analysis showed a serum creatinine level of 1.07 mg/dl and an elevated C-reactive protein of 84 mg/L. High-sensitivity cardiac troponin levels were elevated and demonstrated a rising trend (40 ng/L initially, followed by 57 ng/L and 85 ng/L on serial measurements).

Chest radiography findings are shown in **Figure 1**. Thoracic computed tomography revealed pleural irregularities and subpleural involvement consistent with known malignancy (**Figure 2**). LUS examination demonstrated multiple B-lines bilaterally. A diagnostic LUS was performed using a handheld ultrasound device (Butterfly iQ+, Butterfly Network Inc., Boston, MA, USA) with the lung preset and a depth set to

13 cm. **Figure 3** demonstrates the right lung, where multiple B-line artifacts attributable to pleural involvement secondary to malignant mesothelioma are observed. In contrast, **Figure 4** shows the left lung, with preserved A-lines interrupted by focal B-line artifacts.

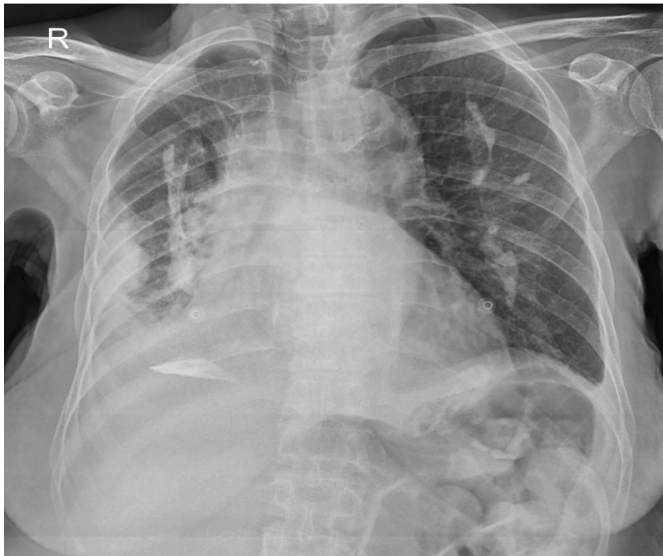


Figure 1. Posteroanterior chest radiograph demonstrating asymmetric right-sided pleural abnormalities with associated volume loss, consistent with known pleural disease



Figure 3. Lung ultrasound image obtained from the right upper lung zone demonstrating multiple vertical hyperechoic artifacts consistent with B-lines, attributed to pleural involvement secondary to malignant pleural mesothelioma

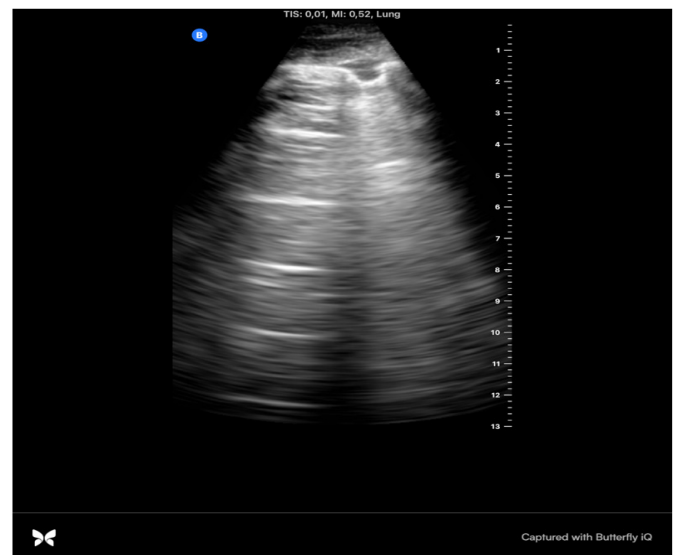


Figure 4. Lung ultrasound image obtained from the left upper lung zone demonstrating preserved A-lines with focal interruption by vertical hyperechoic B-line artifacts

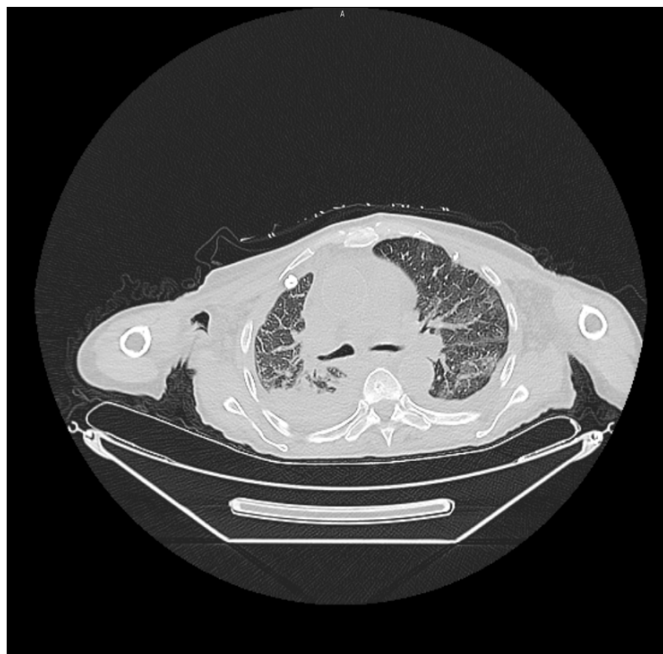


Figure 2. Non-contrast axial chest computed tomography image showing irregular pleural thickening and pleural-based lesions consistent with malignant pleural mesothelioma

On admission, arterial blood gas analysis revealed significant respiratory acidosis with a pH of 7.18 and a PaCO₂ level of 68 mmHg. Following initiation of non-invasive mechanical ventilation, repeat arterial blood gas analysis demonstrated improvement in gas exchange, with the pH increasing to 7.30 and PaCO₂ decreasing to 41 mmHg. Due to the continued rise in cardiac troponin levels, cardiology consultation was obtained. The patient was diagnosed with non-ST-elevation myocardial infarction and transferred to the coronary intensive care unit, where coronary angiography was subsequently performed.

DISCUSSION

This case highlights a key diagnostic challenge in the ED: the interpretation of LUS artifacts in patients with underlying pleural malignancy. Although B-lines are frequently associated with pulmonary edema in acute care settings, they are nonspecific artifacts reflecting increased acoustic interfaces caused by alterations in lung aeration or pleural integrity rather than a single pathological process.

In MPM, pleural thickening, nodularity, subpleural infiltration, and loculated effusions disrupt the normal pleural surface and may generate multiple B-lines that mimic diffuse interstitial syndrome.⁶ Additionally, pleural-based anechoic or hypoechoic lesions, as observed in our patient, are atypical for cardiogenic pulmonary edema and should prompt consideration of alternative diagnoses. These findings underscore the risk of misinterpretation when LUS is used in isolation, particularly in oncologic patients with complex pleuropulmonary pathology.

MPM is an aggressive disease with nonspecific clinical manifestations, often presenting with dyspnea, chest pain, or pleural effusion—symptoms that overlap with more common cardiopulmonary conditions encountered in the ED.⁷ While LUS provides valuable real-time bedside information regarding pleural morphology and subpleural involvement, its findings must be interpreted within the broader clinical context. Previous studies have shown that LUS can reliably detect pleural thickening, nodularity, and pleural-based masses, and in selected settings may even surpass computed tomography in identifying chest wall invasion.⁸ However, the presence of B-lines in malignant pleural disease should not be automatically attributed to pulmonary edema, as these artifacts may instead reflect pleural surface disruption or adjacent compressive atelectasis.

In our patient, although the presence of diffuse B-lines initially suggested an interstitial syndrome such as cardiogenic pulmonary edema, several clinical and sonographic features argued against volume overload as the primary mechanism. The patient did not demonstrate signs of acute fluid overload, such as hypertension, peripheral edema, or rapid radiographic progression compatible with pulmonary congestion. Moreover, LUS revealed that many of the B-lines originated directly from irregular, thickened pleural surfaces and pleural-based anechoic lesions corresponding to known malignant involvement on computed tomography. This spatial relationship between the pleural masses and the vertical artifacts supports the interpretation that the B-lines were generated by pleural and subpleural disruption caused by malignant mesothelioma rather than by diffuse interstitial edema. While concomitant pulmonary edema cannot be completely excluded in the setting of acute coronary syndrome, the overall clinical picture and imaging findings favored malignancy-related ultrasound artifacts as the predominant cause.

From an EM perspective, this case reinforces the importance of integrating LUS findings with clinical history, laboratory data, and complementary imaging modalities. Awareness that B-lines are not pathognomonic for pulmonary edema is essential to avoid diagnostic pitfalls and inappropriate management decisions, particularly in patients with known or suspected pleural malignancy.

CONCLUSION

This case highlights the potential for misleading LUS findings in patients with MPM presenting to the ED. Awareness of pleural-based pathology as a cause of B-lines is essential to avoid diagnostic pitfalls, and LUS findings should always be interpreted in conjunction with clinical context and complementary investigations.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The author declare no conflicts of interest.

Financial Disclosure

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Author Contributions

The author is solely responsible for the conception, design, data collection, analysis, interpretation, literature review, writing, and critical review of the article.

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