Profound Anterior ST-Segment Elevation in a Patient with Lung Cancer and Echocardiographic Evidence of Right Ventricle Metastasis

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INTRODUCTION

Electrocardiogram (ECG) is the initial key test in the timely diagnosis of ST-elevation myocardial infarction (STEMI). In the setting of STEMI, ST-segment elevation (STE) on the ECG is localizing, generally corresponding to the coronary distribution. However, STE mimicking STEMI ECG changes has been reported in patients with no STEMI. Here we report a case of a patient with small cell lung cancer who developed profound anterior STE due to cardiac metastasis involving primarily the right ventricle (RV).

CASE PRESENTATION

A 75-year-old man with no known cardiac history was initially evaluated for cough and generalized weakness for a year and shortness of breath for a week. He underwent computed tomography (CT) of the chest and subsequent needle biopsy of a left supraclavicular lymph node and was diagnosed with stage 4 small cell lung cancer with left pleural effusion. He underwent chemotherapy. The follow-up transthoracic echocardiography (TTE) obtained 9 months after initial diagnosis during chemotherapy showed small pericardial effusion but otherwise no significant abnormality, and an ECG showed no significant abnormality. A follow-up CT of the chest, 13 months after the initial lung cancer diagnosis, showed left hilar and mediastinal conglomerate mass with left bronchial invasion and an enlarged mass in the left upper lobe (Figure 1). Although no dedicated cardiac CT was performed, pericardial effusion and soft-tissue nodularity in the pericardium on the noncontrast CT suggestive of cardiac involvement was also seen (Figure 1).

Fifteen months after lung cancer diagnosis, the patient was hospitalized, this time with complaints of chest pain, worsening shortness of breath, and palpitations. The index ECG showed significant STE

VIDEO HIGHLIGHTS

Video 1: Transthoracic two-dimensional echocardiography in parasternal long-axis view. The suspected metastatic mass on the RV wall is shown by *asterisks* as ill-defined heterogeneous echodensities causing RV wall thickening. *AO,* Aorta; *LA,* left atrium; *PE,* pericardial effusion; *PLE,* pleural effusion.

Video 2: Transthoracic two-dimensional echocardiography in modified apical five-chamber view. The suspected metastatic mass on the RV wall is shown by *asterisks* as ill-defined heterogeneous echodensities causing RV wall thickening. *AO,* Aorta; *LA,* left atrium; *PE,* pericardial effusion; *PLE,* pleural effusion.

Video 3: Transthoracic two-dimensional echocardiography in parasternal long-axis RV inflow view. The suspected metastatic mass on the RV wall is shown by *asterisks* as ill-defined heterogeneous echodensities causing RV wall thickening. *PE,* Pericardial effusion; *RA,* right atrium.

Video 4: Transthoracic two-dimensional echocardiography in parasternal short-axis view at the AV level. The suspected metastatic mass on the RV wall is shown by *asterisks* as ill-defined heterogeneous echodensities causing RV wall thickening. *AV,* Aortic valve. *PE,* pericardial effusion; *LA,* left atrium; *RA,* right atrium; *RVOT,* right ventricular outflow tract.

Video 5: Transthoracic two-dimensional echocardiography in parasternal short-axis view at the mid-LV level. The suspected metastatic mass on the RV wall is shown by *asterisks* as ill-defined heterogeneous echodensities causing RV wall thickening. *PE,* Pericardial effusion.

Video 6: Transthoracic two-dimensional echocardiography in parasternal short-axis view at the LV apical level. The suspected metastatic mass on the RV wall is shown by *asterisks* as ill-defined heterogeneous echodensities causing RV wall thickening. *PE,* Pericardial effusion.

View the video content online at www.cvcasejournal.com.

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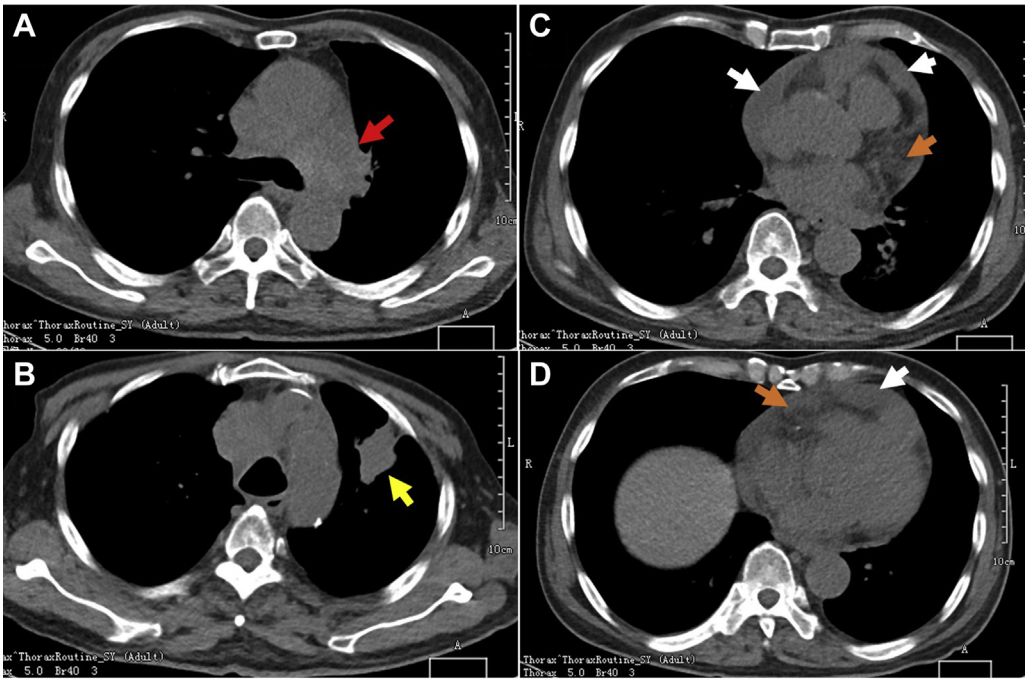

Figure 1 Axial noncontrast thoracic computed tomography, soft-tissue window, 2 months prior to the present hospitalization. **(A)** Superior mediastinal level shows a left hilar and mediastinal conglomerate mass or lymphadenopathy causing left bronchial obstruction (*red arrow*) and **(B)** shows a mass in the left upper lobe (*yellow arrow*). **(C)** Mid-thoracic level shows a pericardial effusion (*white arrows*) and **(D)** shows soft-tissue nodularity in the pericardium (*orange arrows*) suggestive of cardiac involvement.

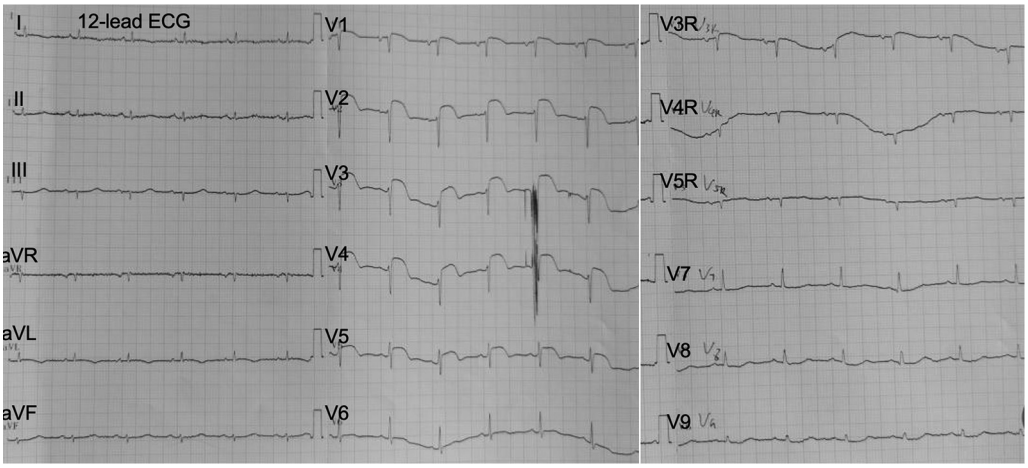

Figure 2 A representative 12-lead ECG, with right-sided (V3R-V5R) and posterior leads (V7-V9) recording, showing sinus rhythm, normal axis, poor R-wave progression, anterior STE, and low voltage in limb leads.

(Figure 2), indicating STEMI. Given the overall poor condition and prognosis of the metastatic small cell lung cancer, conservative management was pursued, and no coronary angiography was performed. However, the initial and serial myocardial cardiac biomarker levels were within normal limits and the STE was persistent on serial ECGs. The TTE showed that the right ventricular wall was significantly thickened with a heterogenous mass measuring 2 cm in the greatest dimension and characterized by ill-defined echodensities involving the anterior, lateral, and apical portion of the RV wall and compression of the RV cavity. There was no pressure gradient within the RV or right ventricular outflow tract, but the RV systolic function deteriorated, with a *fractional area* change of 36% compared with a normal *fractional area* change of 66% obtained 6 months prior. Although we did not use ultrasound-enhancing agents for a perfusion study, in the setting of metastatic lung cancer the echo findings were indicative of cardiac metastasis infiltrating the RV wall. The left ventricle (LV), however, remained grossly normal in size and morphology with no evident regional wall motion abnormality. The

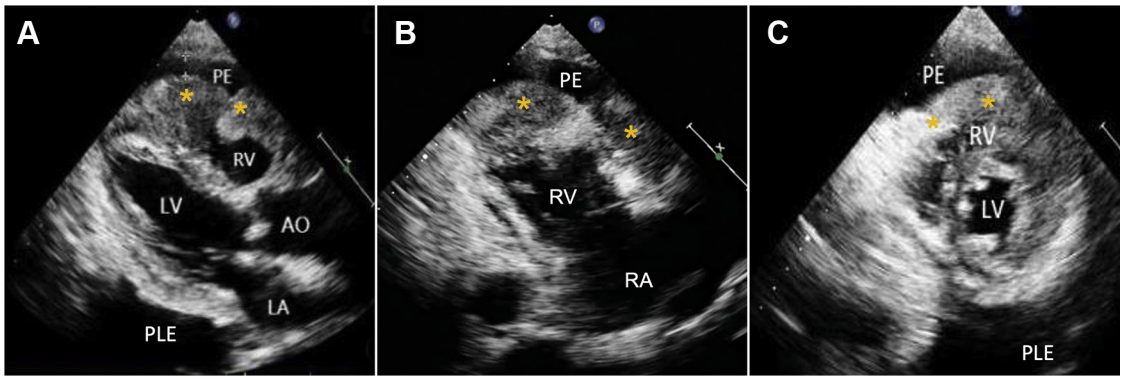

Figure 3 Transthoracic two-dimensional echocardiography obtained in parasternal long-axis **(A)**, parasternal long-axis RV inflow tract **(B)**, and parasternal short-axis mid-LV view **(C)**. The mass shown by *asterisks* as ill-defined heterogeneous echodensities resulting in RV wall thickening and distortion of the heart borders was consistent with myocardial metastasis. *AO*, Aorta; *LA*, left atrium; *PE*, pericardial effusion; *PLE*, pleural effusion; *RA*, right atrium.

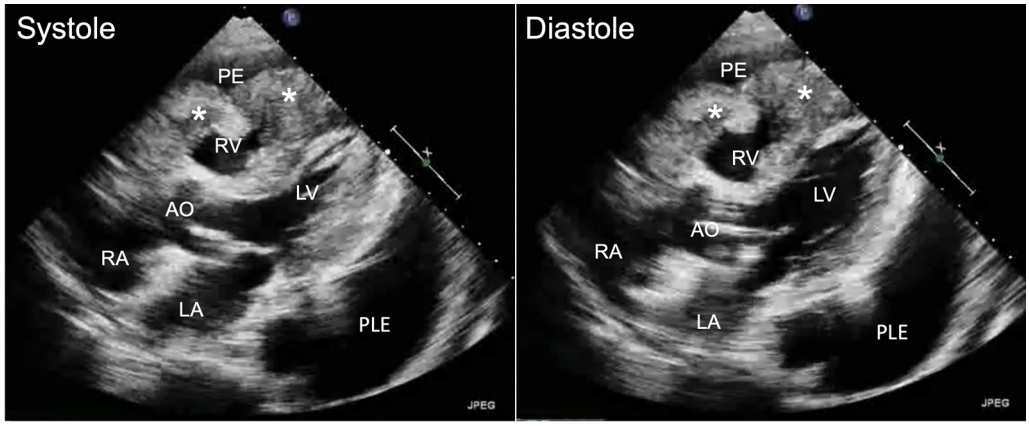

Figure 4 Transthoracic two-dimensional echocardiography in modified apical five-chamber view obtained during systole and diastole. The mass shown by *asterisks* as ill-defined heterogeneous echodensities resulting in RV wall thickening and distortion of the heart borders was consistent with myocardial metastasis. *AO*, Aorta; *LA*, left atrium; *PE*, pericardial effusion; *PLE*, pleural effusion; *RA*, right atrium.

LV was hyperdynamic in systolic function, with a left ventricular ejection fraction of 77% by biplane Simpson measurement (Figures 3 and 4 and Videos 1–6).

Unfortunately, the patient died 4 weeks later due to multiorgan failure from metastatic lung cancer.

DISCUSSION

Here we report a case of a patient diagnosed with stage 4 small cell lung cancer with metastatic cardiac involvement. The patient developed anterior STE pointing to ongoing extensive anterior STEMI unless proven otherwise. Although no coronary angiography was performed, unremarkable serial levels of cardiac biomarkers in the setting of persistent STE ruled out STEMI. The STE on the ECG reflected the underlying malignant cardiac metastasis.

STEMI requires timely diagnosis and emergent coronary revascularization including percutaneous coronary intervention. In some cases, thrombolytic therapy is needed; therefore, ruling out STEMI-mimicking situations is mandatory before thrombolytic administration. STE can occur on several occasions other than STEMI, including acute pericarditis, early repolarization, and STE secondary to QRS complex abnormality due to left bundle branch block, left ventricular hypertrophy, or preexcitation, as well as other processes such as electrolytes disturbances, pulmonary embolism, and Brugada syndrome.[1,2] In the setting of STEMI, the STE is localizing, which is believed to result from an injury current due to transmural myocardial ischemia that causes a potential gradient at the boundary between the ischemic and normal regions.

However, localizing STE can develop in nonischemic cardiac conditions.[2-5] Cardiac metastatic metastasis has been rarely reported to cause STE, which could reflect the extent and location of the malignant cardiac infiltration. In the case reported here, the STE predicted a process involving the anterior wall of the LV. However, the STE was secondary to the extensive RV metastasis and there was no STE in right-sided ECG leads (Figures 1 and 2). As seen on the echocardiogram, the metastasis involved mainly the apical, lateral, and the anterior wall of the RV, as well as the right ventricular outflow tract (Figure 2 and Video 1). Cardiac metastasis led to significant focal

thickening with heterogeneous echodensities, which caused a change in the electrical property of the myocardium and subsequently a change in the electrical potential between the involved and normal myocardium engendering injury current, manifesting as STE on ECG. The extensive RV metastasis wrapped around the LV in part and caused distortion and clockwise rotation of the heart, which shifted the electrical vector as reflected by extensive anterior STE in the precordial leads, mimicking a pattern seen in the setting of anterior STEMI. This case provided a learning point and reinforced the importance of point-of-care ultrasound in real-time clinical decision-making.

CONCLUSION

Malignant cardiac metastasis can cause significant ECG abnormality including STE. Extensive RV wall involvement can lead to heart distortion and rotation masquerading as anterior STEMI. Echocardiography is a convenient, noninvasive, high-yield imaging modality in the timely diagnosis of cardiac metastasis to guide critical clinical decision-making.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2021.11.001.

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