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CASE REPORT

Direct Left Ventricular Metastasis Reduction: 3D-Echo Monitoring For Management of Clinical Case

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ABSTRACT

A 52-year-old woman with a lung carcinoma metastatic to bone was admitted to the Cardiology Department for acute chest pain after 1 week of the oncologic therapy. Electrocardiographic examination (ECG) revealed ischemic picture with ST-T wave abnormalities in DI and aVL leads and poor progression of R wave in V1-V4 leads. Two- and three-dimensional transthoracic echocardiography (2D/3D TTE) showed myocardial involvement with infiltration of the anterolateral left ventricular (LV) wall from the epicardial to the endocardial layer, apical hypokinesia, LV ejection fraction (LVEF) and global 3D longitudinal strain reduction, but was absent pericardial effusion. Three months from the beginning of erlotinib, the patient showed a significant reduction in myocardial involvement with no ECG-ST elevation. Echo showed a mild regression of the wall infiltration and a slightly improvement of LVEF and strain. A computed tomography (CT) scan showed partial remission of the primary lung lesion, intracavitary and intramyocardial mass.

Key Words: Cardiac metastasis, CT scan, electrocardiography, echocardiography, lung tumor

INTRODUCTION

Metastatic cardiac involvement is reported in the literature highly variable, ranging from 2.3 to 18.3% in different reported studies.[1] Tumors can spread to the heart through direct extension of extra cardiac neoplasm, by bloodstream or lymphatic system diffusion and by intracavitary invasion through both the superior and inferior vena cava or pulmonary vessels. Lung cancer is one of the most common metastatic neoplasms to the heart, usually associated with malignant pericardial effusion. The presence of cardiac involvement is often only accidentally detected, prognosis is poor and therapeutic options limited.[2,3] Non-invasive imaging methods are available for evaluation of suspected cardiovascular complications of malignancy.[4]

CASE HISTORY

In May 2012, a 52-year-old Caucasian woman, with no significant past cardiovascular history and risk factors, was admitted to the department of Medical Oncology because a computed tomography (CT) scan, performed 1 month ago for worsening cough and fever, revealed a heterologous neoformation occluding the bronchial branch of the upper lobe, with extension at the parietal pleura, involvement of periaortic and precarenal lymph nodes, and another hypodense formation of about 2 cm with hyperdense rib in correspondence of the right supraspinatus muscle. There was no evidence of pleural or pericardial effusion. Bronchoscopy also, with bioptic sample of the mass, confirmed the diagnosis of bronchial spinocellular carcinoma, p63+, thyroid transcription factor-1 (TTF-1). On physical examination, were found two painful and non-mobile swellings localized in the right forearm and in the right scapula compatible with repetitive lesions, but not appreciable superficial pathological lymph nodes. The overall clinical condition of the patient were discrete and Karnofsky Performance Status (KPS) of 80/90. This clinical aspect refers to advanced clinical stage IV (cT3, cN2, and M1) with a poor prognosis, therefore the patient

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was not considered candidable for surgery, but it was considered appropriate to perform a chemotherapy scheme with carboplatin area under the time-concentration curve (AUC) 6 g1 + paclitaxel 175 mg/m^2 g1, every 21 days, and, in reason of the symptoms, also a radiotherapy treatment.

In June, after the second cycle of chemotherapy treatment, the patient was admitted to Emergency Care Unit for syncope associated with sphincters' incontinence and nosebleed. Electrocardiographic examination (ECG) revealed a sustained ventricular tachycardia (VT); the sinus rhythm was restored firstly with electrical shock of 150 J and then with amyodarone intra-venous infusion. The patient was discharge with clopidrogrel 75 mg/day, ramipril 5 mg/day, and bisoprololo 2.5 mg/day. A CT evaluation showed a progression of the disease with infiltration of the chest wall, mediastinal pleura and heart chambers [Figure 1]. It was decide to administer a second line of chemotherapy scheme with Vinorelbine 25 mg/mq g1, g8, and g15 every 21 days, started in July.

After 1 week of beginning the treatment, the patient presents chest pain and she was referred to the Cardiology Department. At clinical examination she was eupnoic, with a normal physical examination; ECG revealed sinus tachycardia but with ST segment elevation in leads DI and aVL [Figure 2a] and poor progression of R wave in leads V1-V4 [Figure 2b]. Two- and three-dimensional transthoracic echocardiography (2D/3D TTE) showed myocardial involvement with infiltration of the anterolateral left ventricular (LV) wall [Figure 2c] by the side of mass extending from the epicardial to the endocardial layer, with apical hypokinesia and reduction of the LV ejection fraction (LVEF, 45%); 3D global longitudinal strain [Figure 2d], obtained from the triplane cine-loops using the automated functional imaging (AFI) modality, was reduced (average 11.9%) especially at posterior wall; there was mild mitral insufficiency, but was absent pericardial effusion.

Under the impression of acute coronary syndrome we initially intent to perform emergency coronary angiography. However, the cardio-oncology team resolved to not perform the invasive cardiac procedure or coronary CT in view of the progressive decline in global performance status, of the serially followed cardiac enzyme levels within their normal range and also because the coronary CT do not take advantages for diagnostic approach in this clinical contest, the patient was already exposed to elevated ionizing radiation and, in order to evaluate the progression of disease, she could be exposed to other radiation.

It was decide to start the third-line of erlotinib, an oral epidermal growth factor receptor tyrosine kinase inhibitor (EGFR-TKI),[5] 150 mg/die, reduced to 100 mg daily after 1 month because of severe skin toxicity. Three months from the beginning of erlotinib, the ECG revealed a sinus rhythm with depth of negative T wave in leads DI and aVL [Figure 3a] and poor progression of R wave in leads V1 and V3 [Figure 3B], but without ST elevation. At this time 2D/3D-TTE showed a mild regression of the LV lateral wall infiltration [Figure 3c] and a slightly improvement of LVEF (50%). Global and regional strain [Figure 3d] increased (average 14.6%); the akinesia of the ventricular wall was unchanged. A CT scan showed partial remission of the primary lung lesion, of intracavitary

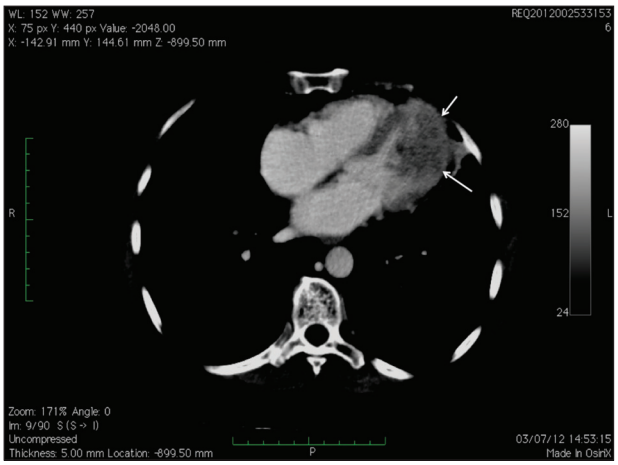

Figure 1: Thorax computed tomography (CT) scan at the time of ventricular tachycardia. The arrows show the infiltration of left ventricular lateral wall and apex

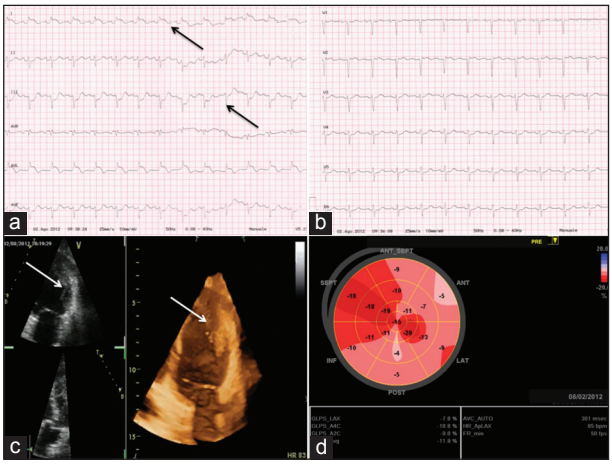

Figure 2: Time of chest pain. (a and b) Electrocardiographic examination (ECG): ST segment elevation in DI and aVL leads (arrows) and R-wave poor progression V1-V4 leads. (c) Two- and three-dimensional transthoracic echocardiography (2D/3D TTE): Anterolateral left ventricular wall and apex infiltration. (d) A lesser 3D global longitudinal strain at posterior (POST), lateral (LAT), and aterior (ANT) wall

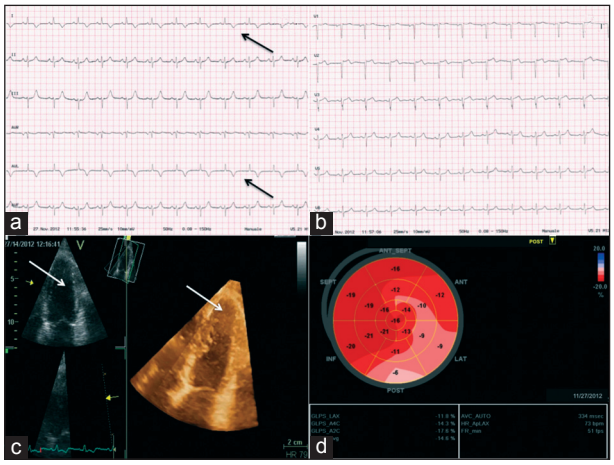

Figure 3: Three months from the beginning of chemotherapy. (a and b) Negative T wave in DI and aVL leads (arrows), but without ST elevation and R-wave poor progression V1-V3 leads. (c) 2D/3D TTE: Mild regression of the left ventricle lateral wall infiltration. (d) Recovery of 3D global longitudinal strain

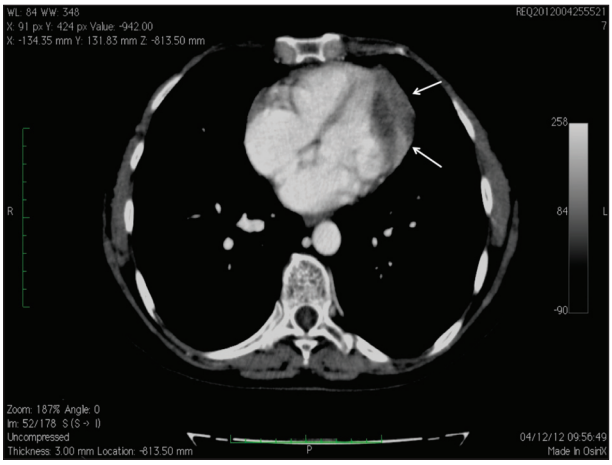

Figure 4: Thorax CT scan at 3 months from the beginning of chemotherapy. Reduction of intracavitary and intramyocardial mass (arrow)

and intramyocardial mass and an assessment of the size and morphology of the cardiac chambers [Figure 4]. We subsequently performed EGFR mutation analysis that showed single-point substitution mutation L858R in exon 21, specific types of activating mutations that confer sensitivity to EGFR-TKIs.

DISCUSSION

Lung cancer is one of the most common metastatic neoplasms to the heart, usually associated with malignant pericardial effusion.[4] Metastases to the heart and pericardium are much more common than primary cardiac tumors. Clinical manifestations are extremely variable; some common presentations of cardiac metastases include atrial and ventricular tachyarrhythmia or premature beats, conduction disorders and blocks, syncope and sudden death as result of LV metastatic infiltration causing episodes of VT or LV outflow tract dynamic obstruction, myocardial dysfunction, angina pectoris, and myocardial infarction as result of coronary embolism, but also of invasion or compression of a coronary artery.[6-12] When a cardiac metastasis is diagnosed antemortem, signs and symptoms of the primary cancer are usually the presenting features, the presence of cardiac involvement is often only accidentally detected, prognosis is poor, and therapeutic options limited.[1,2]

Our patient also exhibited findings suggestive of myocardial ischemia with a previous episode of VT, but without pericardial effusion. The explanations for the ECG findings are the probably occlusion of the circumflex epicardial artery by the neoplasm infiltration, tumor microembolization occluding minor branches of myocardial vessels, expression of myocardial lesions currents, or coronary artery disease (CAD) causing myocardial infarction. We could not exclude the presence of CAD because the patient did not undergo coronary angiography mainly for ethical reasons. However, considering the clinical context and the temporal duration of ECG alterations, the presence of CAD was very unlikely. According to Lim[7], previous VT episode might also be related to focal myocardial ischemia, but we rather think that it is mainly due to spatial heterogeneity of refractoriness or localized conduction delay caused by direct invasion mass.

After treatment with erlotinib, the patient showed a significant clinical improvement and we observed a reduction in lung carcinoma and myocardial involvement. We are following up on this case with careful observation since 6 months. We conclude that the noninvasive strategy in this context was more suitable for patient's quality life. Noninvasive imaging methods are useful for evaluation of suspected cardiovascular complications of malignancy and to guide proper management of these patients.

REFERENCES

1. Chiles C, Woodard PK, Gutierrez FR, Link KM. Metastatic involvement of the heart and pericardium: CT and MR imaging. Radiographics 2001;21:439-49.
2. Bussani R, De-Giorgio F, Abbate A, Silvestri F. Cardiac metastases. J Clin Pathol 2007;60:27-34.
3. Weg IL, Mehra S, Azueta V, Rosner F. Cardiac metastasis from adenocarcinoma of the lung. Echocardiographic-pathologic correlation. Am J Med 1986;80:108-12.
4. Jimenez-Juan L, Leen J, Wald RM, Nguyen ET, Yan AT, Kirpalani A, *et al*. Multimodality Imaging in the Evaluation of Cardiovascular Manifestations of Malignancy. Cardiol Res Pract 2011;2011:378041.

5. Zhang J, Zhang W, Huang S, Li H, Li Y, Chen H, *et al*. Maintenance erlotinib improves clinical outcomes of unresectable advanced non-small cell lung cancer: A meta-analysis of randomized controlled trials. Exp Ther Med 2012;4:849-58.
6. Mager A, Strasrerg B, Tikamien B, Kaplinsky C, Sclarovsky S. Life-Threatening Ventricular Tachycardia as the Presenting Symptom of Metastatic Cardiac Disease. Clin Cardiol 1991;14:696-8.
7. Lim HE, Pak HN, Jung HC, Kim JS, Shim WJ, Kim YH. Electrocardiographic manifestations of cardiac metastasis in lung cancer. Pacing Clin Electrophysiol 2006;29:1019-21.
8. Casella M, Carbucicchio C, Dello Russo A, Tundo F, Bartoletti S, Monti L, *et al*. Radiofrequency catheter ablation of life-threatening ventricular arrhythmias caused by left ventricular metastatic infiltration. Circ Arrhythm Electrophysiol 2011;4:e7-e10.
9. Coller JM, Parente P, Esmore D, New G, Murugasu A, Cooke JC. Large left ventricular metastasis causing left ventricular outflow tract obstruction and haemolysis. Eur J Echocardiogr 2009;10:456-8.
10. Son JW, Hong GR. Unusual left ventricular endocardial metastasis from primary lung cancer. J Cardiovasc Ultrasound 2012;20:157-60.
11. Oliveira SM, Gonçalves A, Cruza C, Almeidac J, Madureira AJ, Amendoeira I, *et al*. Cardiac metastasis from epidermoid esophageal cancer mimicking anterior myocardial infarction. Rev Port Cardiol 2012;31:163-6.
12. Alhakeem M, Arabi A, Arab L, Guerra RA. Unusual sites of metastatic involvement: Intracardiac metastasis from laryngeal carcinoma. Eur J Echocardiogr 2008;9:323-5.

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