

Does Phantom Tumor Really Exist?!

Dan-Nicolae TESLOIANU^a, Mihaela CHIOARTA^a, Dana CORDUNEANU^b,
Andreea-Mihaela IGNAT^a, Antoniu Octavian PETRIS^{a, c}, Anda TESLOIANU^d

^aDepartment of Cardiology, “St. Spiridon” Clinical Emergency Hospital, Iasi, Romania

^bDepartment of Internal Medicine, “St. Spiridon” Clinical Emergency Hospital, Iasi, Romania

^c“Gr. T. Popa” University of Medicine and Pharmacy, Iasi, Romania

^dDepartment of Pneumology, Clinical Hospital of Pneumophtisiology, Iasi, Romania

ABSTRACT

Localized interlobar effusion in congestive heart failure (known as phantom tumor or vanishing tumor of the lung) is an uncommon entity. We report a case of a 61-year-old man who presented to the Emergency Department with a two week history of dyspnoea, palpitations, dry cough and intermittent anterior chest pain. A posteroanterior chest radiography showed a nodular mass in the medium third of the right hemithorax suggestive of a pulmonary tumor. With this supposition of diagnosis, the patient was admitted to the Pneumology Department for further investigations. Left ventricular systolic dysfunction was identified on the echocardiographic examination, in the presence of atrial flutter with 2 to 1 block. Lateral chest X-ray confirmed the presence of a pleural effusion with complete regression of “the lung tumor” after ten days of congestive heart failure treatment, avoiding other expensive and unnecessary investigations.

Keywords: phantom lung tumor, congestive heart failure, atrial flutter, pulmonary mass.

INTRODUCTION

Phantom tumor is an uncommon entity, first described in 1950 by Geffer et al (1). It is caused by a localized interlobar pleural effusion that appears in congestive heart failure. Awareness of this form of pleural effusion is important in the differential diagnosis of a pulmonary mass on chest radiography (2). □

CASE REPORT

A 61-year-old man presented to the Emergency Department with a two week history of

dyspnoea, dry cough and intermittent anterior chest pain. The patient was an ex-smoker (ten pack-years) with no significant medical history. The physical examination on admission revealed rhythmic tachycardic sounds with a heart rate of 150 bpm, a mild elevation of blood pressure (148/100 mm Hg) and a peripheral oxygen saturation of 98%, measured in ambient air. Physical examination did not show either jugular venous distension or peripheral oedema, but the patient had subjective palpitations and orthopnoea. Auscultation of the lungs revealed bibasilar crackles. The patient was afebrile, with a mild increase of the respiratory rate (20 breaths/min). Biological

Address for correspondence:

Mihaela Chioarta

Department of Cardiology, “St. Spiridon” Clinical Emergency Hospital, Iasi, Romania

E-mail: dr.mihaelachioarta@gmail.com

Article received on the 6th of April 2017 and accepted for publication on the 18th of December 2017.

parameters indicated elevated levels of NTproBNP (9811 pg/mL), with normal values of troponin I, D-dimers, CK-MB and myoglobin. Arterial blood gas sampling revealed pH = 7.2, $p\text{CO}_2$ = 35 mm Hg, $p\text{O}_2$ = 76 mm Hg, HCO_3^- = 22.3 mmol/L.

Paraclinical investigations continued with a posteroanterior chest X-ray (Figure 1), which revealed a pathological aspect. A high mass with imprecisely defined border was occupying the right infrahilar region, and another nodular mass was detected in the medium third of the right hemithorax. The well defined, homogeneous opacity with increased density situated in the right costodiaphragmatic recess suggested a loculated pleural effusion. In the lower third of the left hemithorax, a high density opacity, probably of fluid nature, was also identified. The inferior left heart contour was enlarged.

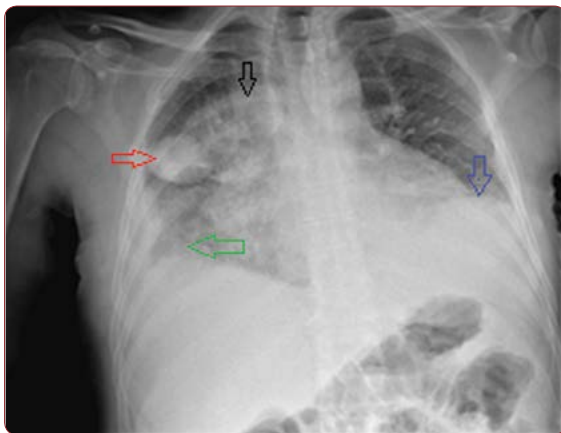


FIGURE 1. Posteroanterior chest radiograph of the patient at admission showing an imprecisely delineated mass, drop-like density, in the right infrahilar region (black arrow), and another one in the right middle lung field (red arrow). Green and blue arrows indicate a loculated pleural effusion in the right and left costodiaphragmatic recess, respectively.

Because the resting admission electrocardiogram (ECG) (Figure 2) showed atrial flutter with 2:1 block (ventricular rate of 150 bpm) and additional left bundle branch block at a symptomatic patient, a cardiology consultation was demanded.

An echocardiography was performed showing a moderately dilated left ventricle, with impaired systolic function (LVEF 35%-probably underestimated due to elevated heart rate), apex hypokinesia and septal dyskinesia

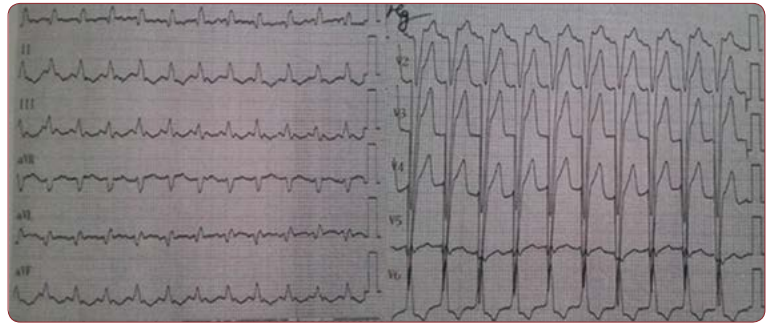


FIGURE 2. Atrial flutter with 2:1 conduction and left bundle branch block

due to left bundle branch block. Echo Doppler valve assessment revealed second degree mitral regurgitation and first degree aortic regurgitation (Figure 3 A, B). A pericardial effusion with 17 mm thickness was present at the apex and lateral from the left cavities (Figure 4 A, B).

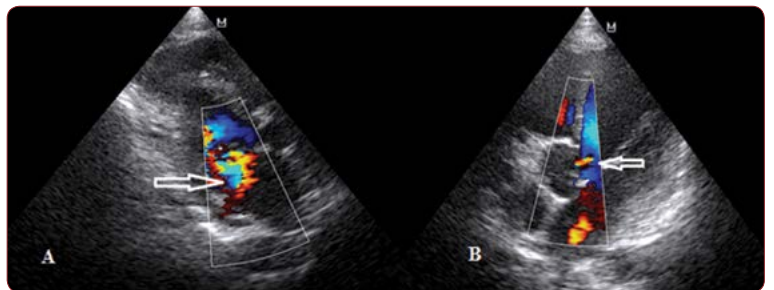


FIGURE 3. Echo Doppler assessment of the mitral (A) and aortic (B) jets of regurgitation (arrows)

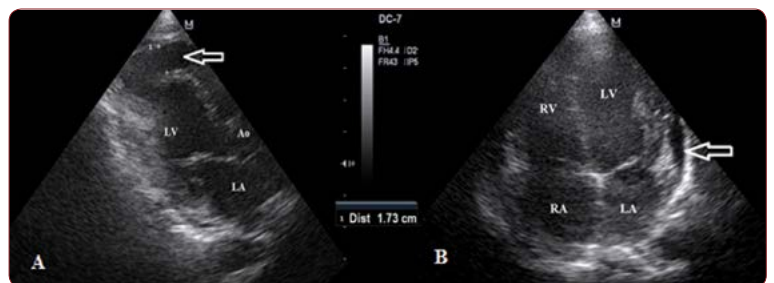


FIGURE 4. Transthoracic echocardiography indicating pericardial effusion (arrows) at the apex (A) and lateral from the left ventricle (B); LA=left atrium; RA=right atrium; LV=left ventricle; RV=right ventricle; Ao=Aorta

Although clinical and paraclinical parameters indicated the presence of congestive heart failure (increased level of NT-proBNP, dilated cardiomyopathy, pleural and pericardial effusions), due to high suspicion of right lung neoplasia (pathological radiographic aspect), the patient was admitted to the Pneumology Department for further investigations. Previously,

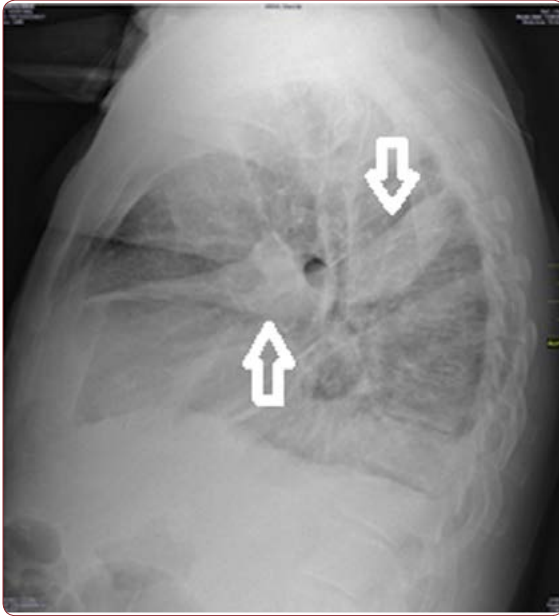


FIGURE 5. Right lateral thoracic radiography indicating the presence of interlobar pleural effusion (arrows)

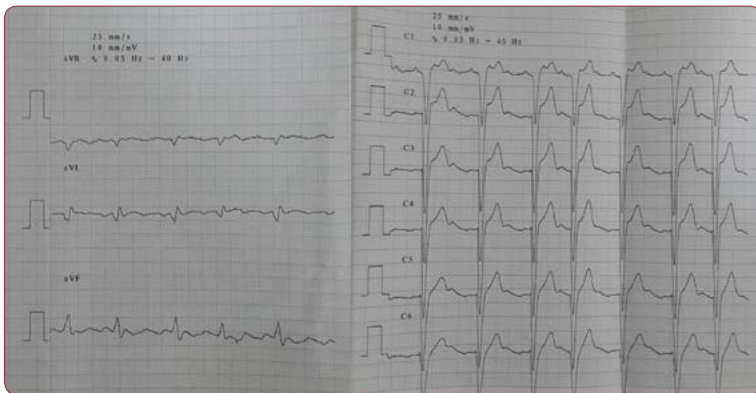


FIGURE 6. Atrial flutter with variable block 3:1 -2:1, left bundle branch block

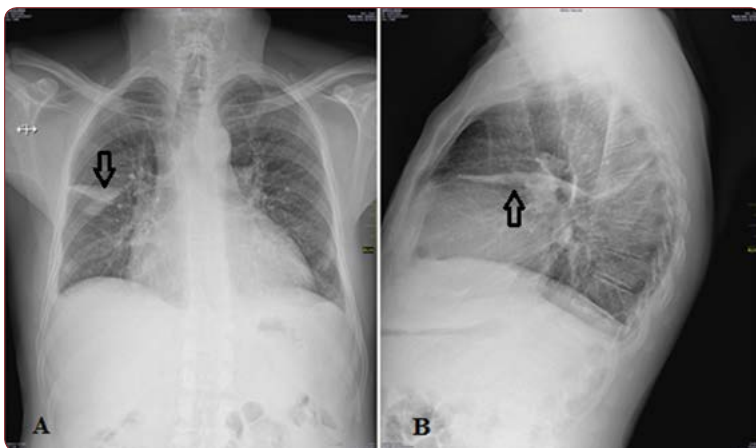


FIGURE 7. Posteroanterior and right lateral view chest radiograph after five days of congestive heart failure treatment: minimal pleural effusion in the right oblique interlobar fissure (black arrows)

an intravenous bolus of 300 mg of amiodarone was administered to the patient, which continued with Amiodarone infusion for the first 24 hours, with indication of switching to oral administration from the next day. The uncertain onset of arrhythmia, without a therapeutic anticoagulation before admission, determined electrical cardioversion temporization. It was decided to initially administrate intravenous Amiodarone, continued then orally, with loading dose in order to obtain sinus rhythm.

The first investigation in the Pneumology Department was a right lateral thoracic radiography (Figure 5) in order to establish the localization of the pulmonary mass. The lateral view identified an interlobar homogenous opacity with increased density, indicating pleural interlobar effusion. Pulmonary ultrasound examination confirmed the pleural fluid collection on both lungs, with approximately 7 cm thickness on the left side and 3.5 cm within oblique interlobar fissure of the right pulmonary lobe. Because the aspect of the right profile view and the pulmonary ultrasound were highly suggestive of pleural effusion, conservative treatment was adopted. No further imagistic investigation, such as a thoracic computed tomography examination, was demanded.

The patient received, besides Amiodarone, an anticoagulant agent (Enoxaparin 60 mg twice a day) for prophylaxis of thromboembolic events, a loop diuretic (Furosemide 40 mg per day), an angiotensin converting enzyme inhibitor (Perindopril 5 mg per day) as an antihypertensive agent, and Ibuprofen (400 mg three times a day) as an anti-inflammatory agent for the pericardial effusion.

During admission in the Pneumology Department, repeated ECG revealed persistent atrial flutter with variable block (3:1 -2:1) (Figure 6).

After five days of congestive heart failure treatment, the radiologic follow up showed partial regression of lung opacities (Figure 7). Five days later, complete regression of lung opacities was demonstrated (Figure 8).

After ten days of hospitalization, the patient was transferred to the Cardiology Department for atrial flutter management. Once a thyroid disorder was excluded, we continued with Amiodarone oral administration (1000 mg per day) based on heart rate and QT length. No sinus rhythm was obtained during admission, so it was



FIGURE 8. Chest X-ray aspect after ten days of treatment with diuretics and ACE-inhibitor, showing complete regression of lung opacities

decided to discharge the patient with Amiodarone 600 mg *per day* for another four days, then 300 mg *per day*, with two days a week pause, Clopidogrel 75 mg *per day*, Perindopril 5 mg *per day*, Ibuprofen 400 mg three times a day for 10 days, with gradual tapering over 3–4 weeks and Pantoprazole 40 mg *per day* until the next visit. Because the efficiency of Amiodarone is proved after four to six weeks, we opted for pharmacological cardioversion until the next follow-up visit.

The resting electrocardiogram performed after another four weeks indicated persistent atrial flutter with variable conduction (ventricular rate of 100 bpm). An echocardiographic re-examination revealed an improved ejection fraction (45%) but with mildly dilated left atrium and a thin rim of pericardial effusion (10 mm). Since no sinus rhythm was obtained during this period of antiarrhythmic treatment, we decided to replace Amiodarone with an alpha-beta adrenergic blocker (Carvedilol 6.25 mg twice a day) for ventricular rate control. ■

DISCUSSION

Phantom tumors predominantly occur in men, commonly found within the transverse fissure (three-quarters of the reported cases), less frequently within the oblique fissure (3-5). In this case, the chest X-ray identified a 3.5 cm tumor within the oblique interlobar fissure, which was confirmed later by pulmonary ultrasound as being pleural effusion. The pathoge-

nesis of this atypical intrafissural distribution of pleural effusions is related to adhesions and obliteration of the pleural space around the edge of the fissure due to pleuritis, so phantom tumors appear whenever transudation from the pulmonary vascular space exceeds the resorptive ability of the pleural lymphatics. Another mechanism is related to local increase in elastic recoil by adjacent, partially atelectatic lung that yields a “suction cup” effect and favors loculation of liquid (6, 7). Most frequently, their appearance is associated with congestive heart failure, but there are also other disorders such as hypoalbuminemia, renal insufficiency or pleuritis that can cause this radiological aspect (6, 8).

The atrial flutter with rapid heart rate (150 bpm) determined decreased systolic function of the left ventricle and cardiac decompensation. Although pericardial and pleural effusion were revealed, as signs of congestive heart failure, by transthoracic echocardiography and posteroanterior chest radiography respectively, the multiple mass identified on the right lung demanded further investigations at the moment of admission.

Lateral views of the chest X-ray are useful for an easy differential diagnosis in patients with presumed diagnosis of pulmonary mass on the front view; they are also helpful for a better localisation on the pulmonary field (9, 10). In the presented case, noninvasive, nonionizing pulmonary ultrasound established the diagnosis of pleural effusion. Rapid resolution of the pseudotumor after management of the left ventricular failure provided additional evidence for the diagnosis. Despite the demonstrated need of long-term anticoagulation for atrial fibrillation (class I level C) (11, 12), the patient had an improved ejection fraction (45%) at the follow up visit, with no other risk factors for embolic events, so we opted for an antiplatelet drug (Clopidogrel).

Pulmonary infarction, pneumonia, tuberculosis, lobe collapse, malignant mass or metastatic neoplasm, abscess, emphysema, cyst and arteriovenous aneurysm are the differential diagnosis for this radiographic finding (13). A loculated pleural effusions within the fissure should be differentiated from transudates due to renal failure, exudates (parapneumonic pleural effusions, malignant pleural effusions,

benign asbestos-related pleural effusions), and hemothorax, chylothorax, and fibrous tumors originating from the visceral pleura of the interlobar fissure (3).

There are no better imagistic techniques for the confirmation of a phantom tumour of the lung than a marked response to diuretic therapy, but some case reports showed that a thoracic computed tomography was performed to allow an easier differential diagnosis (14). □

cians should consider a phantom tumour as a possible differential diagnosis for a lung opacity on a chest radiography in a patient with symptoms of heart failure.

Our case report highlights the importance of a correct management of this entity that avoids unnecessary diagnostic procedures and therapeutic errors, and confirms the efficiency of conservative medical treatment for congestive heart failure. □

CONCLUSION

In conclusion, phantom tumor really exist, and even though it is easy to treat, establishing this diagnosis can be a true challenge. Physi-

Conflicts of interest: none declared.?????????

Financial support: none declared.??????????????

REFERENCES

1. Geffer WI, Boucot KR, Marshall EW. Localized interlobar effusion in congestive heart failure; vanishing tumor of the lung. *Circ* 1950;3:336-343.
2. Ardic I, Yarlioglues M, Celik A. Vanishing or phantom tumor of the lung. *Texas Heart Institute Journal* 2010;6:730-731.
3. Haus BM, Stark P, Shofer SL, et al. Massive pulmonary pseudotumor. *Chest* 2003;2:758-760.
4. Buch KP, Morehead RS. Multiple left-sided vanishing tumors. *Chest* 2000;5:1486-1149.
5. Oliveira E, Manuel P, Alexandre J, et al. Phantom tumour of the lung. *The Lancet* 2012;9858:2028.
6. Lazović B, Stajić Z, Putniković B. Rapidly vanishing lung pseudotumor in a patient with acute bilateral bronchopneumonia. *Vojnosanit Pregl* 2013;9:878-880.
7. Stark A, Leung A. Effects of lobar atelectasis on the distribution of pleural effusion and pneumothorax. *J Thorac Imaging* 1996;2:145-149.
8. Lozo M, Loze E, Ivancevic Z. Phantom tumor of the lung: Localized interlobar effusion in congestive heart failure. *Case Rep Cardiol* 2014.
9. D'Avila Melo BS, Martins Serra AC, Carreira Teixeira Belo MT, et al. Phantom tumor of the lung. *Rev Assoc Med Bras* 2012;58:517-518.
10. Ye C, Wang J, Chai Y. Vanishing lung syndrome with air-fluid levels. *Am J Med Sci* 2016;352:1.
11. Kirchhof P, Benussi S, Kotecha D, et al. 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. *Eur Heart J* 2016;38:2893-2962.
12. Ghali WA, Wasil BI, Brant R, et al. Atrial flutter and the risk of thromboembolism: a systematic review and meta-analysis. *Am J Med* 2005;118:101.
13. Mikaeili H, Baghbani JM. Multiple phantom tumor of the lung: A complex appearance resolving with appropriate intervention. *Tanaffos* 2016;4:243-245.
14. Althomali SA, Almalki MM, Mohiuddin SA. Phantom tumour of the lung in a patient with renal failure misdiagnosed as chest infection. *BMJ Case Rep* 2014.