

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the web/Supplementary material).

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REFERENCES

1. Fernandez-Ortiz A, Nuñez Gil I, Ruiz Mateos B, Ibañez B. Propiedades de los diferentes inhibidores de la glucoproteína IIb/IIIa: ¿se puede aceptar el efecto de clase? *Rev Esp Cardiol Supl* 2011;11:3-7
2. Visentin G, Liu C. Drug Induced Thrombocytopenia. *Hematol Oncol Clin North Am* 2007; 21:685–vi. <http://doi.org/dppj6v>
3. Piątek L. Delayed severe abciximab-induced thrombocytopenia: A case report. *Heart Lung* 45:385-466, e1-e3.
4. Curtis BR. Drug-induced immune thrombocytopenia: incidence, clinical features, laboratory testing, and pathogenic mechanisms. *Immunohematology* 2014;30:55-65.
5. Aragonés Manzanares RM, Delgado Amaya M, Bullones Ramírez JA, Prieto Palomino MA, Arias García DM, Castillo Castro JL. Trombocitopenia extrema secundaria a abciximab. *Rev Esp Cardiol* 2004;57:885-8. <http://doi.org/cjj2>
6. Curtis BR, Divgi A, Garritty M, Aster RH. Delayed thrombocytopenia after treatment with abciximab: a distinct clinical entity associated with the immune response to the drug. *J Thromb Haemost* 2004;2:985–92.

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Pulmonary Vein Stenosis: A Rare Case of Heart Failure and Pulmonary Hypertension in a Cancer Patient

We report the case of a 55-year-old female patient with no cardiovascular risk factors or cardiac history. The disease began with recurrent facial palsy and left trigeminal neuralgia. Computed tomography scan revealed cervical lymphadenopathies and tumor of the left maxillary sinus. A biopsy of the maxillary sinus was performed, evidencing lymphoproliferative syndrome, which was sent for typification.

A few months later, the patient presented cough and fever together with progressive dyspnea to functional class III in the previous week.

Hospitalization was decided due to febrile syndrome and dyspnea. Presumptive diagnoses were B symptoms due to lymphoma versus right upper lobe pneumonia. The patient had leukocytosis (32,000 WBC) and poor general condition. Culture samples were collected and an empiric broad-spectrum antibiotic therapy was started. A computed tomography scan showed multiple supra- and infra-diaphragmatic enlarged lymph nodes at the mediastinal level, like a large mass that moved the trachea and compressed the superior vena cava entry into the right atrium, and was in close contact with the atria. Bilateral in-

filtrates in frosted glass (pulmonary edema), compression of the right upper lobe, moderate bilateral pleural effusion with passive atelectasis, and moderate pericardial effusion were also detected (Figure 1A).

Aspiration-biopsy of bone marrow was performed: flow cytometry revealed 37% large cells consistent with large B-cell non-Hodgkin lymphoma (LBCNHL). It was decided to start the pre-stage with glucocorticosteroid therapy.

The patient was transferred to the Coronary Care Unit two days after hospitalization due to FC III dyspnea and pericardial effusion. Given its semiology (bilateral crepitant rales) and X-ray images (venous-capillary hypertension pattern), it was interpreted as left heart failure (Figure 2).

The first transthoracic echocardiography (TTE) showed normal dimensions and thickness, left ventricular preserved ejection fraction (EF) and prolonged LV relaxation pattern (diastolic dysfunction grade I), moderate pericardial effusion, severe pulmonary hypertension (PHT) (60 mmHg), and no valve diseases (Figure 3).

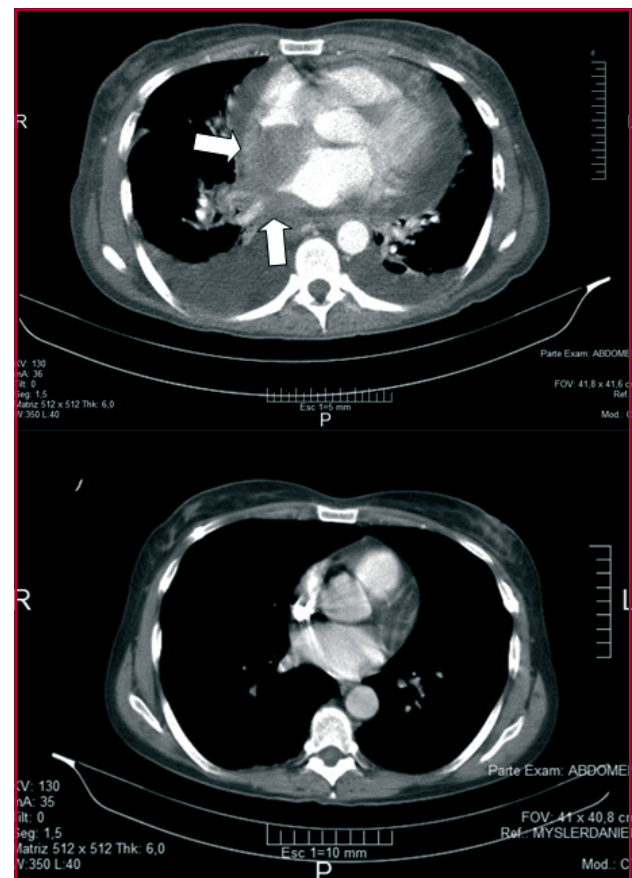


Fig. 1. Pretreatment chest computed tomography (CT) scan (1A top): mediastinal mass in close contact with right to left atria (arrows), bilateral pericardial and pleural effusion. Post-treatment chest CT scan (1B bottom): absence of enlarged lymph nodes, pleural or pericardial effusion.

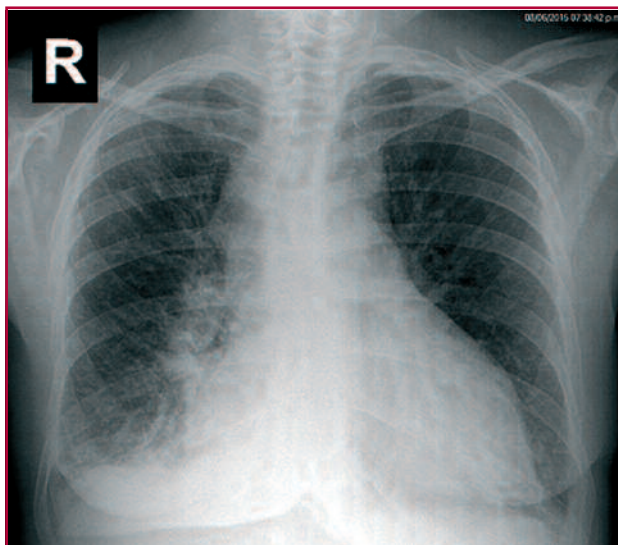


Fig. 2. Chest X-ray showing signs of bilateral venous capillary hypertension.

The patient presented significant clinical improvement with intravenous furosemide and corticosteroids, and a second TTE performed 5 days after treatment initiation revealed moderate acute PHT (44 mmHg). At the end of the course of antibiotics, cultures were negative. Lumbar puncture was positive for neoplastic cells. A few days later, the patient was started on chemotherapy with cyclophosphamide, and then rituximab, doxorubicin, vincristine, and prednisone, to complete the R-CHOP regimen, adding methotrexate due to central nervous system (CNS) involvement. The patient made good progress. A third TTE performed 12 days after admission showed EF of 65%, without PHT (29 mmHg). The patient was discharged a week later, and made good progress in outpatient follow-up. A follow-up CT scan was performed two months after discharge (Figure 3B): no axillary or mediastinal lymph nodes were identified and no pleural or pericardial effusion was observed either.

Final hematology-oncology diagnosis: large B-cell non-Hodgkin lymphoma (high histological grade and high proliferation rate) with CNS involvement. Cardiology diagnosis: left heart failure and PHT secondary to extrinsic pulmonary vein compression due to mediastinal mass.

Pulmonary vein stenosis is an uncommon entity. Until a few years ago, it was a condition appearing almost exclusively in children, associated to other forms of congenital heart disease, occurring as a primary condition or secondary to anomalous pulmonary venous return repair. (1) Its occurrence in adults is even rarer. The few cases reported in the literature are associated to mediastinal processes such as neoplasms, sarcoidosis, or fibrosing mediastinitis (a rare complication of tuberculosis or histoplasmosis). (2) However, its incidence has been increasing in recent years due

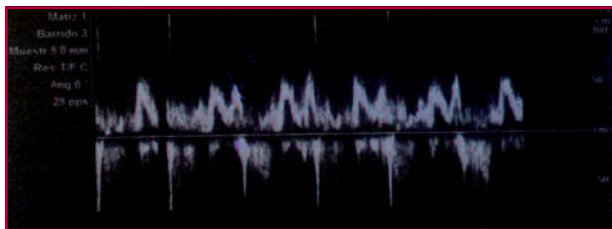


Fig. 3. First transthoracic echocardiography - Mitral flow.

to radiofrequency ablation procedures to treat atrial fibrillation, which may affect 1-3% of cases. (3-5) Symptoms in adult patients include dyspnea on exertion, cough, or even hemoptysis. Chest X-ray can show diffuse or localized pulmonary edema, depending on the veins involved. In most cases, echocardiography, particularly TTE, can evaluate pulmonary veins. Magnetic resonance imaging and computed tomography scan are very useful for the assessment of these patients. Treatment of cases due to extrinsic compression focuses on the underlying condition. Balloon angioplasty has shown positive outcomes in iatrogenic cases associated to radiofrequency ablation.

We present a case of heart failure and pulmonary hypertension in a female patient with mediastinal lymphoma, who first received diuretic therapy and, at the same time, corticosteroids and chemotherapy for her hematology-oncology condition. Images and clinical course supported the suspicion of extrinsic pulmonary vein compression as the pathophysiological mechanism of the process.

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None declared.

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REFERENCES

1. H1. Latson LA, Prieto LR. Congenital and Acquired Pulmonary Vein Stenosis. *Circulation* 2007;115:103-8. <http://doi.org/c8s75r>
2. Pazos-López P, García-Rodríguez C, Guitián-González A, Paredes-Galán E, Álvarez-Moure MÁ, Rodríguez-Álvarez M, et al. Pulmonary vein stenosis: Etiology, diagnosis and management. *World J Cardiol* 2016;8:81-8. <http://doi.org/cjnw>
3. Rostamian A, Narayan SM, Thomson L, Fishbein M, Siegel RJ. The incidence, diagnosis, and management of pulmonary vein stenosis as a complication of atrial fibrillation ablation. *J Interv Card Electrophysiol*. 2014;40:63-74. <http://doi.org/cf5t>
4. Holmes DR Jr, Monahan KH, Packer D. Pulmonary vein stenosis complicating ablation for atrial fibrillation: clinical spectrum and interventional considerations. *JACC Cardiovasc Interv*. 2009;2:267-76.