

Vertebral Enhancement Due to Venous Congestion in a Patient with Innominate Venous Thrombosis: A Cause of False Sclerotic Bone Metastases*



Realce vertebral secundario a congestión venosa en paciente con trombosis de la vena innominada: una causa de falsas metástasis óseas escleróticas

Dear Editor,

Vertebral enhancement due to venous congestion (VEVC) has been described in some patients with mediastinal masses partially or totally occluding the superior vena cava (SVC) and in patients with intravascular devices (central venous catheters, pacemakers, etc.) who develop secondary thrombosis of the central veins of the chest.¹ When occlusion occurs in the SVC or other central veins of the chest, such as the innominate vein or the right brachiocephalic vein, venous collaterals develop. These venous collaterals ensure venous drainage from the head, neck, arms and upper thorax via the azygos/hemiazygos system, the internal mammary veins, and/or the epigastric veins. The intervertebral and basivertebral veins are tributaries of the azygos/hemiazygos system, and the VEVC phenomenon may occur if thrombosis of a central vein of the chest causes drainage difficulties. In VEVC, intravenous contrast medium travels in a retrograde direction until it reaches the spongy bone of the upper dorsal vertebrae, causing marked enhancement that can simulate focal sclerotic bone lesions.^{2–4} We report a case of VEVC in a cancer patient with chronic thrombosis of the innominate vein.

This was a 57-year-old woman with a personal history of breast cancer, with a Port-a-Cath reservoir implanted in the left prepectoral region. A chest computed tomography (CT) conducted for follow-up of her oncological disease with intravenous contrast medium administered via the left arm showed sclerotic lesions in several upper dorsal vertebrae, suggestive of bone metastases

(Fig. 1A, B). The CT also revealed chronic thrombosis of the innominate vein carrying the port and collateral venous circulation that had developed in the anterior chest wall (Fig. 1C) and in the posterosuperior mediastinum. Given the lack of chest pain, the chest CT was repeated without intravenous contrast, and the vertebral lesions seen in the previous study were not detected (nor were they seen in a dorsal spine magnetic resonance imaging (MRI) study performed a few weeks later), so the presence of vertebral bone lesions could be ruled out.

VEVC in patients with thrombosis of the central veins of the chest is a phenomenon rarely described in the scientific literature, although it has been documented mainly in patients with full or partial thrombosis of the SVC due to chest tumors (particularly in lung cancer), intravascular devices (pacemakers, ports), or mediastinal fibrosis.¹ The venous collaterals that develop in cases of chronic SVC thrombosis in the tributary veins of the azygos/hemiazygos system include the intercostal, the intervertebral, and the basivertebral veins.^{2–4} Our case is of interest because the chronic thrombosis affected only the innominate vein and not the SVC, and the VEVC of the upper dorsal was caused by the retrograde reflux of intravenous contrast medium via the collateral basivertebral veins (due to the difficulty of draining to the SVC). As the basivertebral veins drain the most posterior part of the vertebral bodies, VEVC preferentially affects the posterior margin of the dorsal vertebrae.

We believe that VEVC should be considered by radiologists and physicians in patients with thrombosis of the proximal veins of the chest (not only the SVC), particularly in cancer patients in whom this phenomenon can mimic sclerotic vertebral lesions and lead to the wrong therapeutic management. If sclerotic vertebral metastasis is suspected in patients with VEVC, we recommend repeating chest CT without contrast or performing other additional studies (MRI of the spine or PET/CT) to confirm the real etiology of the vertebral sclerosis and to avoid further aggressive diagnostic procedures, such as bone biopsy.

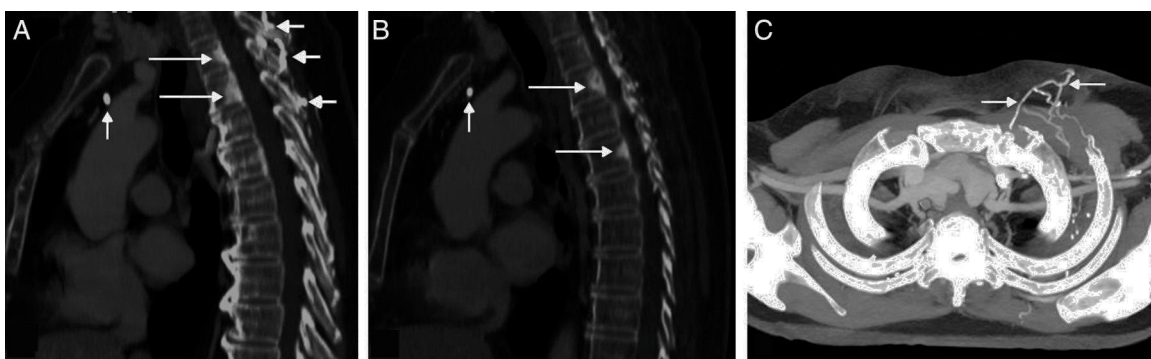


Fig. 1. (A) Sagittal reconstruction of the chest CT with intravenous contrast medium showing focal sclerotic bone lesions in the posterior margin of the T2 and T3 vertebral bodies, mimicking vertebral metastases. Note the port catheter in the theoretical position of the thrombosed innominate vein (short vertical arrow) and the development of collateral venous circulation in the perivertebral veins (short horizontal arrows). (B) Sagittal reconstruction of the chest CT with intravenous contrast medium showing focal sclerotic bone lesions in the posterior margin of the T3 and T5 vertebral bodies (long arrows). Note the port catheter in the theoretical position of the thrombosed innominate vein (short arrow). (C) Maximum intensity projection axial reconstruction of CT, showing development of collateral venous circulation in the anterior chest wall (arrows).

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Bronchitis Obliterans Due to Influenza B Pneumonia Complicated With *Staphylococcus aureus* Infection[☆]



Bronquitis obliterante secundaria a neumonía por virus Influenzae B y sobreinfección por *Staphylococcus aureus*

Dear Editor,

Despite the high incidence of bronchiolitis among children, post-infective bronchiolitis obliterans (PIBO) is an uncommon pediatric complication. The microorganism most frequently associated with bronchiolitis obliterans is adenovirus, followed by *Mycoplasma pneumoniae*, parainfluenza virus, influenza virus, etc.¹ The obliterative fibroblastic process in the bronchioles causes air trapping and respiratory failure that can improve with time, leaving severe obstructive functional impairment.² In children older than 12 months, fibroblastic scarring of the bronchial lumen has been described after infectious processes, an entity known as bronchitis obliterans. In this case, occlusion of the lumen can be associated with atelectasis of the pulmonary anatomical region. However, this radiological finding is not exclusive to bronchitis obliterans, and is common in PIBO. Local treatment consists of partial or complete lung resection. When involvement is generalized, the disease course is fatal, unless lung transplantation can be performed.³

We report the case of a 13-year-old girl, with no significant clinical history, who consulted due to a catarrhal syndrome with fever, treated with amoxicillin-clavulanate acid. Twenty-four hours later, she attended the emergency room due to progressive dyspnea. On arrival, the patient was showing signs of severity with raised inflammatory markers, and the chest radiograph showed bilateral alveolar consolidation. Treatment was started with cefotaxime and levofloxacin, and she was admitted to the intensive care unit for monitoring. Her clinical situation and arterial blood gases quickly deteriorated, and intubation was required. Influenza B virus RNA was identified in the nasal swab. Blood cultures and antigen detection in urine for pneumococcus and *Legionella* were negative. Given her poor response to mechanical ventilation and the possible need for extracorporeal membrane oxygenation (ECMO) support, the patient was transferred to our hospital.

After methicillin-resistant *Staphylococcus aureus* was isolated from the blood culture and the bronchoalveolar lavage, the cephalosporin was discontinued and treatment continued with clindamycin. After 7 days of intubation, 2 days of non-invasive ventilation, and 17 days of oxygen therapy, the patient was discharged, 28 days after admission, receiving bronchodilation with salbutamol.

Outpatient monitoring confirmed radiological resolution of the infiltrates and persistent retrocardiac segmental atelectasis. The patient had a slight cough, persistent wheezing, and dyspnea on moderate exertion, so the bronchodilator treatment was intensified and treatment began with inhaled corticosteroids. However, limited clinical improvement and severe functional obstructive impairment (FVC 1.9 l (55%); FEV1 0.76 l (26%); MMEF 0.22 l/s (9%); FEV1/FVC 48%) led to the initiation of oral corticosteroid treatment. PIBO was suspected, so a pulmonary CT was performed, revealing tracheobronchomegaly, bilateral diffuse bronchiectasis, and generalized air trapping.

Three months after discharge she developed clinical worsening with a normal chest radiograph, requiring hospitalization. She was given methylprednisolone iv (500 mg/day/3 days) and nebulized bronchodilators. After initial improvement, she developed respiratory failure due to atelectasis of the left lung and consequent pneumothorax that did not require a chest tube. A bronchoscopic study was performed, in which the findings shown in Fig. 1 were observed. After the membranes that were occluding the segmental bronchi of the left upper lobe were perforated, the patient showed transient resolution of the atelectasis, and then of the pneumothorax, so perforation of the endobronchial septa was repeated, and the patient was treated with CPAP positive pressure with an oronasal mask. Scintigraphy with quantification of pulmonary perfusion showed hypoperfusion in the left lung and to a lesser extent in the right lower lobe: left lung: 17% (upper 10%, lower 7%), right lung: 83% (upper 51%, lower 32%) (Fig. 1C).

The patient was included in the lung transplantation wait list, and the procedure was performed 6 weeks later. Immediate post-surgical progress was good. Four months after transplantation, she did not require oxygen, and lung function and clinical examination were normal. Histology of the explant showed bronchial and bronchiolar ectasias, patchy bilateral constrictive bronchiolitis with fibroblasts and foamy histiocytes, and adjacent cystic formations caused by dilation of the mucinous glandular component of the airway.

Pulmonary complications associated with influenza virus infection include necrotizing tracheobronchitis, acute respiratory distress syndrome, PIBO, and secondary bacterial pneumonia. *Staphylococcus aureus* is the most common coinfection in influenza virus pneumonia, and is associated with an increased rate of suppurative complications.⁴

Initial clinical manifestations of bronchitis obliterans are non-specific: persistent cough, wheezing, tachypnea, dyspnea, and loss of appetite. Although it has been suggested that bronchitis obliterans is a different entity from PIBO,³ the histological study of our patient's lungs showed associated bronchiolar scarring. A diagnosis of PIBO based on spirometric and radiological findings (CT) after a severe pulmonary infection is now generally accepted.^{1,2,5} Septation caused by bronchial scarring might be diagnosed only in the presence of complications such as lobar or pulmonary atelectasis

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