Extrinsic Compression of the Inferior Vena Cava by a Lumbar Osteophyte: A Rare Cause of Pulmonary Embolism

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Abstract

Introduction: Pulmonary embolism results from thrombus migration into the pulmonary artery, with the most common cause being deep vein thrombosis. However, pulmonary embolism might not necessarily originate in the lower extremities, which necessitates specific diagnostic and therapeutic choices.

Case Presentation: An 84-year-old man presented with acute pulmonary embolism, but with no sign of deep vein thrombosis or a thrombophilic state. He experienced complete resolution with medical therapy involving parenteral and oral anticoagulants. During the patient’s hospital stay, an abdominal CT scan revealed a 23 mm lumbar osteophyte compressing and displacing the inferior vena cava. The turbulent blood flow through the stenotic area might have caused a thrombus and the consequent pulmonary embolism.

Conclusions: This is the first report of pulmonary embolism caused by inferior vena cava extrinsic compression due to an osteophyte. Such a diagnosis should be suspected if the patient lacks deep vein thrombosis and hypercoagulative states. Acute pulmonary embolism could be a rare consequence of osteoarthritis in the spine, although correct assessment is crucial to initiating lifelong oral anticoagulant therapy following the first episode of pulmonary embolism. Indeed, spinal surgery is generally avoided due to the high risks and the fact that extrinsic compression of the inferior vena cava cannot be radically resolved.

Keywords: Pulmonary Embolism, Osteophyte, Inferior Vena Cava, Anticoagulants

1. Introduction

Acute pulmonary embolism (PE) is a major cause of mortality and hospitalization, and it is typically a consequence of deep vein thrombosis (DVT) (1). Other sources of thrombi resulting in PE are extremely challenging to diagnose, and they account for only a small percentage of PE in clinical practice. The treatment of PE is based on oral anticoagulation, which should be administered for three months in patients with reversible risk factors and those who present with DVT, although lifelong therapy is required in the case of recurrent episodes, thrombophilia, or other irreversible risk factors so as to prevent the risks and the sequelae associated with recurrent PE (1).

2. Case Presentation

An 84-year-old Caucasian man was admitted to our department complaining of the sudden onset of a slight fever, fatigue, chest pain, and dyspnea on mild exertion. His relatives reported that the patient was previously in good health, and he was completely autonomous in terms of the basic and instrumental activities of daily living. His past medical history included hypertension and coronary heart disease with previous non-ST-segment elevation myocardial infarction (NSTEMI), which was completely resolved with medical therapy alone.

The patient’s vital signs and physical examination findings on admission were unremarkable, apart from a II/VI systolic murmur. His extremities were painless and they did not show swelling or pitting edema. The extremities were of normal color and the temperature of the skin was also normal. No signs of DVT were present. A chest X-ray showed a left pleural effusion, while echocardiography revealed right ventricular hypokinesia, with a 40 mmHg pulmonary artery systolic pressure (PASP). Blood tests showed elevated serum creatinine (1.5 mg/dL), C-reactive protein (52.26 mg/dL), erythrocyte sedimentation rate (> 120 mm/hour), D/Dimer (1840 ng/dL), and N-terminal of the prohormone brain natriuretic peptide (NT-
proBNP) (541 ng/dL). An arterial blood gas test revealed a hypoxemic hypocapnic state with an alkalotic pH (7.44). A ventilation/perfusion lung scan was performed and a diagnosis of pulmonary embolism was then confirmed.

The patient received low molecular weight heparin therapy with enoxaparin and, subsequently, oral anticoagulants according to established guidelines. Predisposing factors for DVT or PE, for example, recent surgery, trauma, autoimmune diseases, cancer, obesity, or prolonged immobilization, were absent. To identify the original site of the patient’s thrombus, a lower limb Doppler ultrasound (US) was performed, with no evidence of deep venous thrombosis being found. Thrombophilia testing was also negative. Therefore, a diagnosis of PE was made and appropriate treatment was initiated, although the primary cause of the PE remained unclear.

Three days later, a contrast-enhanced thoracic-abdominal computed tomography (CT) scan was performed, which showed a complete recovery from PE, as well as evidencing a massive (23 mm) right anterior osteophyte at the L3 - L4 intersomatic space that was compressing and locally displacing the inferior vena cava (IVC) (Figures 1 and 2). The caval diameter at the compression site was 5 mm, with a pre-stenotic and post-stenotic value of 19 mm. Due to both the patient’s age and the co-morbidities, treatment with spinal surgery was not advised, and the patient was discharged in good condition on lifelong oral anticoagulant therapy. Some 12 months later, the patient still takes oral anticoagulants and he is in good clinical condition, with no recurrence of PE and no decrease in his functional status.

3. Discussion

Thrombus formation is a consequence of abnormalities in the vessel wall, blood flow, or blood constituents (2). Therefore, it can occur in any segment of the venous circulation. Although unusual, a thrombus did originate in the inferior vena cava as a result of it being compressed by a lumbar osteophyte. When the dimensions of the osteophyte caused a critical level of IVC compression, a thrombus was generated by turbulent flow. It then migrated into the pulmonary circulation, leading to PE. A possible explanation for this hypothesis and the observed phenomenon can be supported by fluid dynamics. In a normal venous blood flow setting in the IVC (3) (peak systolic velocity of 0.30 - 0.45 m/sec, a blood density of 1.05 × 10^3 kg/m^3, and a viscosity of 4 × 10^-3 N × sec/m^2), the Reynold’s number is 1496 and the flow is linear. However, with a 5 mm diameter, the blood flow velocity increases to 4.33 - 6.5 m/sec, while the Reynold’s number in the stenotic area rises to 5643, which means that the flow is turbulent. Based on these observations, in the current case report we can reliably speculate that turbulent flow resulting from extrinsic compression of the IVC could be responsible for thrombus formation and the consequent pulmonary embolism. In fact, turbulent flow determines countercurrents that promote endothelial cell activation and local areas of stasis, and these factors allow the activation of the platelet clotting cascade, which ultimately results in thrombosis (4-6).

Osteophytes have been classically related to aging and osteoarthritis in the spine, and their development usually takes many years. We can therefore imagine that the condition of our patient gradually worsened over the years, while the turbulence generated by the osteophyte only re-
cently exceeded the threshold for thrombus generation. Considering that the patient was not eligible for spinal surgery, as well as the fact that this extrinsic compression is not reversible, lifelong oral anticoagulant therapy seems mandatory for preventing the recurrence of PE and its associated complications.

Giant osteophytic spurs are known to be responsible for the compression of the jugular foramen (7), airway obstruction (8), and dysphagia (9). Yet, the extrinsic compression of the IVC has been reported to be responsible for PE in a few cases. Reports in this regard include hepatic hemangioma (10), non-parasitic hepatic cyst (11), extremely distended urinary bladder (12), right renal cysts (13), and giant mesenteric desmoid tumor (14). To the best of our knowledge, this is the first case report to describe osteophytes as being responsible for extrinsic caval compression.

Acute pulmonary embolism resulting from extrinsic IVC compression is rare but possible, and a lumbar osteophyte might well be the cause. The correct diagnosis is crucial because patients should receive lifelong oral anticoagulant therapy since spinal surgery cannot usually be performed and this predisposing factor cannot be removed. Additionally, patients with PE but without deep vein thrombosis or hypercoagulable states should undergo an abdominal CT scan to check for extrinsic caval compression.

Footnote

Authors’ Contribution: Study concept and design: Antonio Nenna, Nicola Papapietro, and Massimo Chello; acquisition of data: Antonio Nenna, Filippo Barberi, Cristiano Spadaccio, and Mario Lusini; analysis and interpretation of data: Antonio Nenna and Francesco Nappi; drafting of the manuscript: Antonio Nenna, Filippo Barberi, and Mario Lusini; critical revision of the manuscript for important intellectual content: Cristiano Spadaccio, Nicola Papapietro, Francesco Nappi, and Massimo Chello; statistical analysis: Antonio Nenna; administrative, technical, and material support: Mario Lusini and Massimo Chello; study supervision: Massimo Chello.

References


