Massive pulmonary thromboembolism as the initial manifestation of pituitary tumor: a case report

Patients with brain tumors have a latent hypercoagulable state that predisposes to pulmonary thromboembolism (PTE). Risk factors for PTE in brain tumor patients include age \( \geq 60 \) years, large tumor size, and the presence of leg paresis. We presented a patient with an unremarkable medical history except hypertension who developed bilateral deep venous thrombosis (DVT) and PTE associated with pituitary adenoma. A 78-year-old woman with hypertension was brought to our emergency room (ER) with acute shortness of breath and chest pain. On initial evaluation in the ER, pulse rate was 120/min, respiratory rate 32/min and blood pressure was 74/42 mm Hg. Laboratory evaluation showed a white blood cell count of 9,270/mm\(^3\), and arterial blood gas measurements showed a pH of 7.56, PCO\(_2\) 21.9 mmHg, PO\(_2\) 74.2 mmHg, and bicarbonate 21.9 mEq/L on oxygen flow rate of 3 L/min nasal prong. Plasma D-dimer assay was 1.06 µg/L. A chest radiograph appeared normal, initial creatine kinase-MB was 4.05 ng/ml and pro-BNP was 5,898 pg/ml. An electrocardiogram showed sinus tachycardia of 120 beats/min and a S1Q3T3 pattern. Urgent bedside echocardiogram revealed moderate enlargement of right cardiac chambers, with a moderately hypokinetic right ventricle (RV) suggesting pressure overload. The estimated pulmonary artery pressure from tricuspid regurgitation was 70 mmHg. Chest computed tomogram (CT) showed multiple large filling defects of both main pulmonary arteries and both upper and lower lobe pulmonary arteries. The diagnosis of massive PTE with RV dysfunction and incipient cardiogenic shock was made. The decision to give thrombolytic therapy was made and immediately started. The patient subsequently had an uneventful recovery. Doppler ultrasonography of the lower extremities showed a left femoral to popliteal vein and distal branch. Tests for deficiencies of antithrombin III, protein C, protein S, antiphospholipid antibody were all negative. Brain CT showed a large pituitary adenoma, extending to 3rd ventricle and sphenoid sinus. Due to concern over the possibility of intracranial bleeding, a cava filter was successfully inserted for prevention of recurrent PTE.

Ergotism associated with ketoconazole: A case report

Introduction: Ergotamine is an alkaloid produced by Claviceps purpurea. It is widely used in treatment of migraine. It causes constriction of peripheral and cranial blood vessels. Ergotism is a rare cause of arterial insufficiency with an incidence of less than 0.01%. Delayed diagnosis leads to serious irreversible complications. Case report: A 63-year-old man with cyanosis in hands was transferred from outside hospital. His symptoms began 21 days ago and were performed right subclavian-radial artery bypass surgery. He had recovered temporary but recurred after 2 weeks. He was diagnosed migraine headaches, 20 years ago, for which he had taken ergotamine tartrate routinely. He had been prescribed ketoconazole for 2 weeks at local dental clinic 1 month ago. Angiogram revealed diffuse vasospasm in both upper extremities. Further history revealed the onset of symptoms began shortly after he finished to take ketoconazole. This revealed a possible interaction between ergotamine andazole derivatives, which may result in ergotism. Treatment consisted of sodium nitroprusside and heparin. Peripheral pulses recovered within a few hours. Follow-up angiogram revealed normally visualized both brachial, ulnar, radial arteries. Conclusion: The patient was misdiagnosed as PAOD at first and performed right subclavian-radial artery bypass surgery. Patient was responded well by iv nitroprusside. This case emphasizes the importance of obtaining a detail medication history, especially when new medications have been introduced into a patient's regimen.