Pulmonary artery obstruction mimicking the clinical features of acute pulmonary embolism

C W Siu, M-H Jim and H F Tse

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IMAGES IN CARDIOLOGY

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A 44 year old woman, who had enjoyed good health up until recently, presented with a three month history of progressive shortness of breath and bilateral ankle oedema. Physical examination revealed tachycardia of 125 beats per minute and a blood pressure of 100/60 mm Hg on admission. Elevated jugular venous pressure with giant CV wave pattern was observed at the level of the earlobe. Hepatomegaly and bilateral pitting ankle oedema were also noted on palpation. There was a soft, grade 2/6 ejection systolic murmur best heard over the pulmonary area. The oxygen saturation was about 95% on room air. Chest *x* ray showed cardiomegaly and widened mediastinum. ECG revealed sinus tachycardia, right axis deviation and S_I Q_{III} T_{III} pattern (below left). The clinical presentation and the ECG pattern were typical of acute pulmonary embolism. Computed tomography with contrast of the thorax strikingly showed no evidence of pulmonary embolism but a huge, solid mediastinal tumour encroaching on the heart. The main pulmonary artery and the right ventricular outflow tract was grossly compressed and squeezed, leaving only a slit-like lumen (below right). The entire heart was pushed aside towards the left hemithorax. Serum α-fetal protein (αFP) and human chorionic gonadotrophin (HCG) concentration were normal. Mediastinoscopy and biopsy of the tumour was subsequently performed which yielded malignant spindle cell tumour by histology. In view of the probable invasion of the heart and great vessels by the tumour, complete curative surgical resection would not be possible. Chemo-irradiation was offered to the patient as an alternative but she finally declined this treatment option after knowing and understanding all the side effects of the treatment and the prognosis of her disease.

Spindle cell tumour is a rare disease of mesenchymal origin. This case is unusual because this rapid growing tumour caused external compression and luminal obstruction of the main pulmonary trunk, the latter being exactly the same pathological mechanism as pulmonary embolism.

C W Siu
M-H Jim
H F Tse
hftse@hku.hk