Multiple pulmonary infarctions caused by giant cell arteritis

Sir,

A previously healthy 83 year old woman presented with a 4-week history of dyspnoea, weight loss, malaise and headaches. Apart from dyspnoea, systems examination revealed no positive physical signs. ESR was 80 mm in 1 hour. Haemoglobin, white cell count, blood glucose, urea, electrolytes, urinary microscopy, chest radiograph and ECG were all normal. Auto-antibody screen was negative. Giant cell arteritis was diagnosed and temporal artery biopsy showed florid inflammation of full thickness of vessel wall, infiltrated by lymphocytes, macrophages and numerous giant cells with fibroelastic connective tissue proliferation of the intima. Prednisolone 60 mg/day was commenced and the response was dramatic with complete remission of all her symptoms, including dyspnoea. The ESR decreased to 2 mm in 1 hour.

Two months later prednisolone was gradually reduced to 15 mg/day, following which dyspnoea recurred and progressively worsened, necessitating her hospitalization. There was no evidence of deep vein thrombosis, nor were there any new physical signs. ESR rose to 93 mm in 1 hour. ECG, chest radiograph and serum biochemistry all remained normal. Ventilation and perfusion lung scan were consistent with multiple pulmonary infarctions. Prednisolone was increased to 40 mg/day and she was initially anticoagulated. Dyspnoea improved over the following 4 weeks and she remained asymptomatic with ESR of 11 mm in 1 hour 4 months after her discharge from the hospital.

All the initial symptoms in this patient except the dyspnoea were typical of giant cell arteritis. The cause of dyspnoea was unclear at the initial presentation. Dyspnoea, along with other symptoms, responded well to steroid therapy but recurred and progressively increased on reduction of steroid dose.

Giant cell arteritis is a multisystem disease but pulmonary involvement is rare. A few cases of pulmonary nodules and interstitial infiltrates resolving with steroid therapy have been reported in association with giant cell arteritis. Lung biopsies in some such cases showed interstitial, non-caseating granulomas.

The histology of a lobectomy specimen in a suspected case of carcinoma of bronchus showed no tumour but revealed areas of infarction, fibrosis and features of giant cell arteritis of pulmonary artery. Aneurysm and narrowing of pulmonary artery due to giant cell arteritis have separately been reported in two cases. The striking response of dyspnoea to steroid therapy, its recurrence on reduced steroid dose and the absence of predisposing factors make pulmonary embolism as the aetiology most unlikely. On the other hand these features strongly favour giant cell arteritis of pulmonary arteries as the cause of pulmonary infarction. Giant cell arteritis, if diagnosed and treated early, has a good prognosis for complete recovery. Therefore physicians should be aware of pulmonary infarction as a complication in patients with giant cell arteritis with respiratory symptoms.

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