**CASE REPORT**

A 64-year-old male with a medical history of chronic obstructive pulmonary disease was referred by the ophthalmologist to the rheumatology department under suspicion of giant-cell arteritis (GCA). The patient presented with acute blurred vision related to bilateral retinal artery occlusions (RAO). Apart from an unintentional 25 kg weight loss, there were no other related symptoms such as headache, jaw claudication, fever, morning stiffness, nor pain and stiffness of the shoulder and pelvic girdle. At physical examination no abnormalities were discovered and pulsations of the temporal artery were normal with absence of tenderness. Laboratory tests revealed a raised erythrocyte sedimentation rate of 108 mm/hour, C-reactive protein of 122 mg/l and anaemia (haemoglobin 6.2 mmol/l). An electrocardiogram showed sinus rhythm. Because of the preliminary diagnosis of GCA and potential risk of permanent blindness, the patient was immediately treated with a high dose of prednisolone. However, a temporal artery biopsy did not confirm this diagnosis. The chest X-ray showed a central mass of the left lung with pleural effusion (**figure 1**). Computed tomography (CT) was performed.

**WHAT IS YOUR DIAGNOSIS?**

See page 231 for the answer to this photo quiz.
DIAGNOSIS

A CT scan showed a tumour of the left upper lobe invading the left pulmonary vein and atrium, slightly enlarged adrenal glands and a large (tumour) thrombus in the left atrium (figure 2). The latter was confirmed by transthoracic ultrasonography. Oncological work-up led to the diagnosis of a stage IV squamous cell lung carcinoma.

The retinal artery occlusions (RAO) were caused by embolism from the left atrial (tumour) thrombus. Prednisolone was discontinued and low-molecular-weight heparin (LMWH) was initiated. This led to a serious haemoptysis and because of the high risk of a fatal bleeding LMWH had to be discontinued. Salvage surgery was not an option due to the extension of the tumour into the mediastinum and the radiotherapist was afraid of massive bleeding complications when applying radiotherapy. The patient refused palliative chemotherapy and died several months later.

The differential diagnosis of RAO is extensive, including serious systemic disease. RAO affects 10-13% of those with visual loss in GCA. GCA is only the cause in 1-4.5% of the cases of central RAO. The combination with elevated inflammatory parameters makes GCA more likely, although this does not exclude other causes, such as the one in the above-mentioned patient. A cardiac tumour related embolism as cause of RAO is rare and mainly related to myxomas. A few cases have been reported regarding spontaneous systemic tumour embolism secondary to lung cancer with invasion of the left atrium and pulmonary vein, but these patients presented with ischaemic stroke. We are not aware of a case with bilateral RAO as presenting symptom.

RAO in the presence of elevated inflammatory parameters should raise the suspicion of GCA. However, other causes, such as a cardiac origin of a (tumour) embolism, should always be considered. In the absence of contraindications, treatment with corticosteroids is warranted to prevent ongoing ocular damage. Waiting for a definite diagnosis of GCA before initiating therapy might result in permanent vision loss. Furthermore, this case taught us that an obvious diagnosis should always be confirmed by a proper clinical investigation.

REFERENCES