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Sir,

Comment on 'Quantification of the role of temporal artery biopsy in diagnosing clinically suspected giant cell arteritis'

We read with interest the recent paper 'Quantification of the role of temporal artery biopsy in diagnosing clinically suspected giant cell arteritis'.[1] We welcome the data supporting the American Rheumatology guidelines[2] and that histological confirmation from a temporal artery biopsy is not essential for a diagnosis of giant cell arteritis (GCA). There are however two points that we wish to raise:

(1) One of the aims of the paper was to 'quantify the role of temporal arteritis in diagnosing GCA'. A positive biopsy confirms the diagnosis, but a negative biopsy does not exclude it. An incidence of false negative biopsies is well acknowledged with the length of the specimen being a vital factor.[3] While it was mentioned in the discussion that there is a false-positive rate for biopsies, no information is given upon the rate of any false-negative biopsy patients in this case series. Several previous authors have followed up biopsy negative patients, diagnosing GCA in a further 5–9%. They based this diagnosis on further symptoms, signs, response to steroids, or postmortem results.[4,5] Some authors have advocated taking a second biopsy in those with a negative result, increasing the yield of positive biopsies by 3%.[6] We suggest that follow-up data are essential to identify any false-negative results. In the Greenwich scheme such false-negative biopsies should be included in the 'adverse effect' group as they may lead to delay in diagnosis and intervention.

(2) The Greenwich grading scheme is specifically assessing the clinical application of an investigation.[7] While scalp necrosis is a serious complication, for the patient it does not constitute an adverse effect on the ability of the investigation to reach the correct diagnosis.

GCA is a clinical diagnosis which continues to be challenging to confirm.

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Sir,

Disseminated paracoccidioidomycosis with chorioretinal involvement

Paracoccidioidomycosis, also known as South American blastomycosis, is a systemic granulomatous mycosis caused by the dimorphic fungus *Paracoccidioides brasiliensis*. The infection is supposed to be acquired by inhalation, with a primary localization in the lungs.[1] It is commonly an endemic disease in Latin American, but several cases have been reported in North American, Asia, and Europe, in individuals who lived in endemic areas, sometimes many years before the development of clinical manifestations.[2]

The eye may be involved through haematologic spread of the fungus, and usually affects the eyelid and conjunctiva. Choroidal involvement is rarely described in the literature.[3] We report herein a case in which the ophthalmological findings are limited to chorioretinal disease in a patient with disseminated systemic paracoccidioidomycosis and its favourable response to trimethoprim–sulphamethoxazole therapy.

A 32-year-old rural worker man was referred for examination because of a 14-day history of pain and decreasing vision in his right eye. He also complained of a 4-kg weight loss associated with a productive cough. He denied any history of fever or night sweats.

His medical history was significant for chronic alcohol consumption and smoking habit. The respiratory examination revealed fine inspiratory crackles bilaterally. On ocular examination, the best-corrected visual acuity was 20/200 in the right eye and 20/20 in the left eye. The intraocular pressure was normal in both eyes. Results of biomicroscopic examination of the anterior segments were unremarkable. Examination of the right fundus revealed multiple, whitish-yellow elevated lesions at the choroid level. The lesions ranged from 0.5 to 4.0 disc diameters in size. The overlying neurosensory retina was edematous (Figure 1). The left fundus was normal.

Fluorescein angiography of the right eye revealed early hypofluorescence, followed by hyperflorescent staining of the choroidal lesions. Blood investigation showed a total white count of 7.08×10^9 per ml, and a haemoglobin level of 13.40 g/dl. An angiotensin-converting enzyme level, serum electrolyte, glucose, calcium, and phosphorus levels were normal. Serologic tests for syphilis, toxoplasmosis, cytomegalovirus, and HIV were negative. A purified protein derivative skin test was also negative. A chest X-ray showed a diffuse alveolointerstitial infiltrate with a right apical cavitation (Figure 2). The sputum analysis revealed the presence of *P. brasiliensis* and absence of BAAR. The double immunodiffusion test using GP 43 *P. brasiliensis* antigen-rich fraction was positive at a titer of 1:8. Diagnosis of disseminated ocular paracoccidioidomycosis with choroidal involvement was suspected. The patient was treated with trimethoprim–sulphamethoxazole (160/800 mg) 1 tablet b.i.d. and showed continuous improvement. The choroidal lesions decreased in size, forming atrophic chorioretinal scars at the end of 1 month. Treatment continued for 12 months. At follow-up 6 months after treatment, no reactivation of disease was noted. The subretinal fluid slowly reabsorbed, and visual acuity was 20/20 in both eyes.

Paracoccidioidomycosis is a multisystem infection that affects predominantly males from rural areas of South America, but several cases have been reported outside these areas in returning travellers, aid workers, and immigrants.[2] Although, in most cases, the primary site of infection is in the lung, haematogenous spread may occur with the formation of multiple metastatic foci.[4]

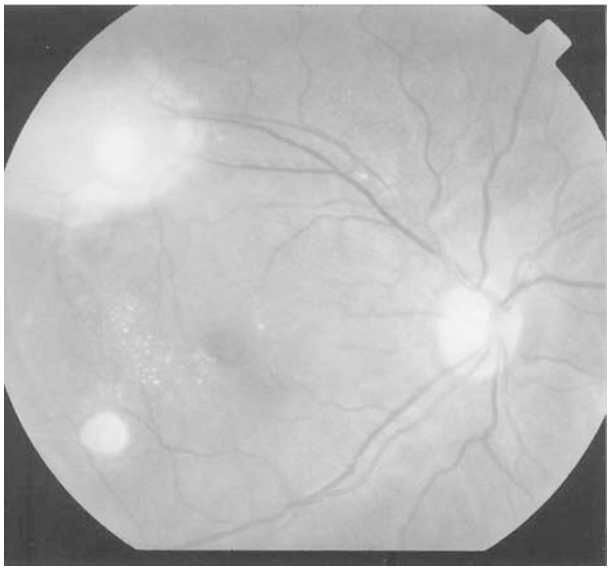

Figure 1 Fundus photograph of the right eye shows a large well-circumscribed choroidal lesion in the superotemporal arcade associated with oedema of the underlying retina and subretinal fluid, and a smaller lesion in the inferior temporal area.

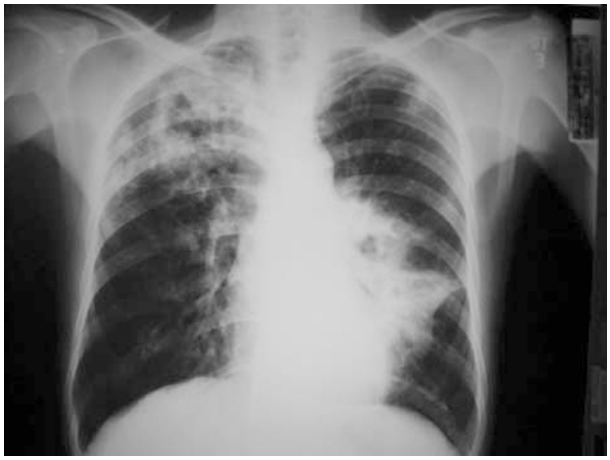

Figure 2 Radiograph of the chest shows alveolointerstitial infiltrate with a right apical cavitation.

Lung involvement usually presents nonspecifically with cough, dyspnoea, and inspiratory crackles on examination. Chest radiograph often reveals nodules and/or cavities superimposed on a diffuse interstitial or fibrotic pattern.[5]

The incidence of ocular involvement is low, but if present may lead to mutilation and even to blindness.[1] Of 50 cases described by Belfort Jr *et al*,[6] 38% had lid

lesions, 24% palpebral–conjunctival involvement, 12% conjunctival, and 4% palpebral–conjunctival–corneal. Choroidal involvement alone or associated with that of other ocular structures, as retina and iris, has been described even less frequently and is seen clinically as yellowish-white subretinal lesions, as shown earlier in the course of the disease in our patient.[7]

The diagnosis of this disease is possible by the detection of the fungus on specimens of body fluids or biopsies of lesions.[4] The main other entities that can produce similar ocular picture are syphilis, sarcoidosis, and tuberculosis.[3] The diagnosis of systemic paracoccidioidomycosis was reached in this case by finding the fungus in the sputum and confirmed by double immunodiffusion test. Since other possible causes for similar choroidal lesions had been eliminated, and since the patient positively responded to the specific treatment, the final diagnosis made was of disseminated choroidal paracoccidioidomycosis.

Our patient had a good response to trimethoprim–sulphamethoxazole therapy. The systemic and ocular manifestations resolved, and no evidence of reactivation has been noted on follow-up examination.

Paracoccidioidomycosis should be suspected in patients who lived in endemic areas or with an appropriate travel history. Although rare, ocular dissemination of *P. brasiliensis* also be considered in patients with posterior chorioretinitis and previous or active pulmonary lesions of equivocal nature. Early diagnosis and adequate therapy are essential.

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Sir,

Patient alert system: the Edinburgh experience

We read with interest the article and accompanying editorial on 'The Patient Alert System' (PAS).[1,2] The Edinburgh system was developed 3 years ago and incorporates tactile vibrating feedback through the hand piece once activated. The prototype lacked this feature and was similar to the Manchester device. Evaluation of the prototype showed patients were unsure if staff had noticed an audible alarm amidst the background theatre noises of the phacomachine, music, and conversation. Deaf patients found the tactile vibrating feedback device in the hand piece of particular benefit.

We agree that patient choice should determine if hand-holding or the patient alert system should be used. The latter was the preferred option in approximately 40% of patients in an evaluation of 50 consecutive patients undergoing cataract surgery in our unit. It is preferred by patients who may have poor hand-grip strength, arthritis, or who are unsure about the procedure of increasing grip as a means of attracting attention. This may in part reflect patient anxiety, cognitive dysfunction, and conflicting patient advice. For example, patients are instructed not to move under the drapes, yet when anxious, distressed, and sensorially deprived, moving a limb rather than increasing a squeeze on a hand is an instinctive method of requesting assistance.

The patient alert system is a tool for reducing patient anxiety by ensuring a clear method of communicating distress from patient to surgeon. In order to pre-empt the possibility of patient movement in a population who are likely to have communication difficulties and poor appreciation or ability of increasing hand grip as a method of signifying distress, we suggest the Edinburgh patient alert system as a simple cost-effective strategy.