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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 guidelines² 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