

Editorial

Is the halo always holy? Glucocorticoid impact on detecting cranial large-vessel arteritis

The importance of temporal artery biopsy

In the developed world, GCA is the most common inflammatory vasculitis and may cause major morbidities, of which blindness is the most feared. Early diagnosis via temporal artery biopsy (TAB) and glucocorticoid treatment are still gold standard recommendations [1], but several issues may complicate this in real-life practice. First, TAB may be falsely negative due to skip lesions or inadequate material, truly negative in non-cranial large-vessel vasculitis (LVV) [2] or simply refused by the patient. Secondly, prior glucocorticoid exposure will influence the histological findings. This is particularly relevant in cases of proven and treated GCA with a suspected relapse. The development of non-invasive imaging techniques such as ultrasonography, MRI and PET has generated a large amount of data but has not eliminated the need for TAB, despite initial enthusiasm that it would [3]. However, as adjunct investigations in cases of high pre-test probability but negative TAB, all or one of these diagnostic techniques may be very informative. Although high-resolution MRI and colour-coded duplex sonography (CCDS) compare well in terms of sensitivity and specificity [4], neither has been prospectively compared with TAB [1]. Although it is clear that mural inflammatory changes resolve under glucocorticoid therapy, the extent and timing of this resolution varies. In classic cases of GCA, glucocorticoid treatment should commence immediately and a biopsy should be performed as soon as possible, ideally within 1–2 weeks [5]. However, biopsies may still remain positive up to 6 weeks after glucocorticoid exposure [6, 7]. We are also unclear how quickly glucocorticoid exposure eliminates MRI and CCDS signs, with some authors suggesting that there is no change for at least 1 week and that they can persist for up to 2 months after treatment begins [3, 8].

In this edition of *Rheumatology*, Hauenstein *et al.*'s retrospective study assesses the impact of prior glucocorticoid treatment on the ability of high-resolution MRI and CCDS to demonstrate mural inflammation of the cranial arteries in GCA. They found that within just a few days, the ability of both techniques to detect mural inflammation is markedly reduced [9].

Using ACR criteria, Hauenstein *et al.* identified 59 patients with possible GCA who had undergone both CCDS and MRI within 10 days of diagnosis. All patients had received glucocorticoids for <2 weeks. In the initial study population of 59 patients suspected of having GCA, the final diagnosis was only reached by the

rheumatologist at a follow-up visit of ≥ 6 months. This reduced the number of confirmed GCA cases to 36. Of these, 24 had positive TAB and 4 had false negative biopsies considered to be the result of skip lesions. The excluded 23 patients were used as controls in the analysis.

In the diagnosed patients, both CCDS (88%) and MRI (85%) performed well with regard to sensitivity at 1 day after glucocorticoid exposure. Both reduced rapidly to 50 and 56%, respectively, at >4 days after exposure. However, specificity remained high. When TAB was used as the reference point, the initial sensitivity was even higher: 92% for CCDS and 90% for MRI, remaining high at >4 days after exposure for MRI (90%) but dropping off for CCDS (50%). Specificity also remained high for MRI at 4 days (80%), but was reduced for CCDS (50%) due to more false positive results.

Hauenstein *et al.* conclude that while both CCDS and MRI have high sensitivity in detecting mural oedema in the first few days of glucocorticoid exposure in true GCA cases, this rapidly declines thereafter. The obvious limitations of this paper are its retrospective nature and rather small number of patients eventually entering the study. Additionally, it is assumed that all true GCA cases were confirmed, although this is uncertain without a positive TAB. However, the data do not contradict other prospective studies suggesting that both CCDS and MRI may remain positive for many weeks, since the specificity of both techniques was shown to be high.

As the authors rightly point out, studies of non-cranial LVV have shown the persistently high sensitivity of both techniques [10, 11] after glucocorticoid exposure, probably reflecting the larger mass of oedematous vessel wall. Also, as all those working in the field appreciate, the diagnosis of relapse in proven GCA cases during glucocorticoid therapy remains a major challenge. As yet, there are no scientific studies evaluating the performance of either CCDS or MRI in this situation.

In summary, while both CCDS and MRI offer attractive non-invasive options for assessing cranial arteries in suspected GCA, neither has yet replaced TAB, which is still recommended in all cases. Additionally, the application of glucocorticoids in patients strongly suspected of having GCA should not wait for either a TAB or non-invasive examinations, which should be performed as soon as possible and certainly within 1–2 weeks. Finally, it is never too late to perform these investigations in unclear

cases, remembering that although sensitivity may be lost, specificity is usually retained.

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