Early improvement of radiological signs of large-vessel inflammation in giant cell arteritis upon glucocorticoid treatment

Sirs, We read with interest the recent article by Hauenstein et al. [1] reporting, in a retrospective study, that the sensitivity and specificity of colour-coded duplex sonography and high-resolution MRI for the diagnosis of GCA rapidly declines within a few days after the initiation of glucocorticoid (GC) treatment.

We would like to remark that similar results are obtained when assessing large-vessel involvement in GCA by PET or computed tomography angiography (CTA) [2, 3]. These techniques have revealed that large-vessel involvement in GCA is more prevalent than previously anticipated, and imaging of large vessels has received a great deal of attention as a potential additional tool for GCA diagnosis [4-6]. However, we have recently shown that radiological signs of large-vessel involvement may decrease rapidly after the initiation of GC treatment. In a prospective study of 40 biopsy-proven GCA patients treated with GC for 0–3 days, we found that CTA signs of large-vessel vasculitis (concentric wall thickening) were significantly more frequent among treatment-naïve patients than in patients who had received GC, even for such a short period of time (77% vs 29%, \( P = 0.005 \)) [3]. The prevalence of CTA-defined aortitis in our series (65%) was higher than that reported in a previous study where patients were imaged within the first month after the initiation of GC and consequently were treated for longer periods of time (45%) [7]. Similarly, in the study by Fuchs et al. [2], the diagnostic accuracy of PET in detecting large-vessel involvement was significantly higher in patients not receiving immunosuppressive therapy (93.3% vs 64.5%, \( P = 0.006 \)). Metabolic activity detected by PET and vessel wall oedema leading to concentric vessel wall thickening detected by CTA may rapidly decrease upon GC treatment.

In contrast, Achkar et al. [8] reported that GC treatment for up to 6 weeks did not reduce the diagnostic efficacy of temporal artery biopsy. Although GC reduces inflammatory infiltrates over time, minor remaining clusters of inflammatory cells and vascular remodelling changes may persist for months or years, still allowing the recognition of treated GCA [9]. Therefore histopathological examination is remarkably more sensitive than imaging in detecting vascular inflammation in GC-treated patients.

However, follow-up studies reveal that in some patients fluorodeoxyglucose uptake detected by PET or vessel wall thickening detected by CTA may persist for months after the initiation of GC treatment, although generally at lower intensity than at the initial evaluation [6, 10]. It is currently unclear whether these changes reflect persisting inflammation or vascular remodelling or scarring.

Taken together, these findings emphasize that in order to achieve maximal diagnostic accuracy, imaging must be performed before or shortly after the initiation of GC treatment since sensitivity and specificity seem to suffer a sharp decline within a very few days. However, even with decreased diagnostic accuracy, imaging may still detect changes useful for diagnosis in some treated patients. Moreover, imaging is essential to detect complications and sequelae such as dilatation or stenoses over time [11].

**Rheumatology key messages**

- Imaging signs of vasculitis may rapidly decline upon glucocorticoid treatment.

**Acknowledgements**

**Funding:** Supported by the Ministerio de Ciencia e Innovación (SAF 08/04328) and Ministerio de Economía y Competitividad (SAF 11/30073).

**Disclosure statement:** The authors have declared no conflicts of interest.

**References**

Pulmonary impairment, not muscle injury, is associated with elevated ESR in the idiopathic inflammatory myopathies

Sr., Despite the presence of systemic inflammation with muscular and extramuscular disease manifestations, the ESR is elevated in only about half the patients with DM and PM [1, 2]. Interstitial lung disease (ILD), an extramuscular manifestation of DM and PM, is common, with a reported prevalence of 40–65% [3, 4]. It is especially high among the idiopathic inflammatory myopathy (IIM) subgroup with antisynthetase autoantibodies, which can manifest with a severe form of ILD and increased mortality. Given that the lung is an extramuscular target of immune-mediated injury in IIM, we hypothesized that ESR elevation is associated with ILD.

In the present report, 48 patients with IIM according to the Bohan and Peter criteria, without overlapping rheumatic diseases [5, 6], evaluated at the Johns Hopkins Myositis Center between March 2007 and April 2009, were analysed. The presence of ILD was confirmed by typical high-resolution CT evidence and pulmonary function testing [7]. Mann–Whitney and $\chi^2$ tests were used for comparison between groups of continuous and categorical variables, respectively. Spearman correlation was performed to examine the association between ESR and the duration of ILD, or creatine phosphokinase (CPK) and aldolase.

Mean age of patients was 48.3±1.6 years, with the majority being Caucasian (63%) and female (81%). Mean disease duration from diagnosis of IIM to evaluation at our centre was 19.9±3.1 months. There were 21 (44%) patients with and 27 (56%) without ILD. Age, gender and race did not differ between these groups. Notably, 9 (43%) were newly diagnosed with ILD while 12 (57%) had established ILD at their first centre visit. The mean haematocrit level was 40.5±0.6%. Antisynthetase antibodies were present in 14 (67%) of 21 ILD patients and uniformly absent in the non-ILD group. Anti-Jo-1 was most common, present in 9 (69%) of the 14 patients, followed by 3 with anti-PL-12 (23%) and 1 each with anti-PL-7 and anti-EJ. Malignancy was present in 5 (10%) of the 48 IIM patients and distributed as follows: 4/28 (14.3%) ILD-negative and 1/21 (4.3%) ILD-positive patients had a malignancy ($P = 0.38$). The prevalence of inflammatory arthritis and RP did not differ between the ILD and non-ILD groups, but mechanic’s hands were more common in the ILD group (33% vs 7%, $P = 0.022$). Mean levels of CPK (normal range 24–170 U/l) and aldolase (normal ≤8.1 U/l) did not differ between the non-ILD and the ILD group (1100 ± 2650 vs 1740 ± 3281 U/l, $P = 0.83$ and 17.5 ± 22.5 vs 26.6 ± 37.3 U/l, $P = 0.68$, respectively).

In contrast, mean ESR was significantly higher in the ILD group (42 ± 38 vs 17 ± 16 mm/h, $P = 0.027$) (Fig. 1A); however, there was no correlation between ILD duration and ESR. The six outliers in the ILD group with the highest ESR were older (54.8 ± 8.0 vs 46.1 ± 9.4 years, $P = 0.06$), African American (83.3% vs 26.7%, $P = 0.046$), female (100% vs 66.7%, $P = 0.26$), with lower haematocrit (35.0 ± 1.3 vs 41.6 ± 2.87, $P = 0.001$) and lower forced vital capacity (FVC) (55.3 ± 13.7 vs 70.9 ± 14.7, $P = 0.038$). As antisynthetase antibodies can be associated with severe lung disease, the ILD group was further divided into the antisynthetase-negative ($n = 7$) and antisynthetase-positive ILD groups ($n = 14$). As expected, ESR was significantly higher in the antisynthetase-positive ILD group (50 ± 41 vs 17 ± 16 mm/h, $P = 0.029$), whereas there was no difference between the antisynthetase-negative ILD group and the non-ILD group (Fig.1B), suggesting that the ESR elevation in ILD is driven by the antisynthetase-positive group. Strikingly, ESR levels did not correlate with the extent of muscle injury as CPK or aldolase levels (Fig. 1C and D).

The daily prednisone dose did not differ between the non-ILD and antisynthetase-positive ILD group

---


