(15%) and contributed in the diagnosis of neoplastic processes in 5 (4.8%). Table 2 shows the final diagnoses.

When analyzing the results of PET in patients with onset PMR vs. those who are corticosteroids resistant, no significant differences were observed in the final diagnoses ($p = 0.078$).

Table 1. Patient's features

Women, n (%)	73 (70.9%)
Disease duration (months), mean $\pm$ SD	24 $\pm$ 41.92
Shoulder pain, n (%)	100 (97.1%)
Hip pain, n (%)	89 (86.4%)
Amaurosis, n (%)	5 (4.9%)
Temporal artery tenderness, n (%)	2 (1.9%)
ESR (mm/s), mean $\pm$ SD	55.92 $\pm$ 31.09

Table 2. Diagnoses

	TOTAL	Onset PMR	Cortico- resistant PMR
PMR, n (%)	73 (70.9)	37 (50.7%)	36 (49.3)
Large vessel vasculitis, n (%)	16 (15.5)	6 (37.5)	10 (62.5)
Neoplasia, n (%)	5 (4.8)	5 (100)	0 (0)
EORA associated PMR, n (%)	6 (5.8)	2 (33.3)	4 (66.7)

Conclusion: PET-CT confirms the diagnosis of PMR in the significant majority of patients included in the study. In this cohort, PET-CT allowed to diagnose vasculitis of large vessels and neoplasms in 16% and 5% of the patients respectively. We did not observe differences in the PET findings in those patients who underwent a PET at the time of diagnosis vs. those who are corticosteroid resistant.

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THU0604

CARPAL TUNNEL SYNDROME: CAN ULTRASOUND PREDICT RESPONSE TO STEROID INJECTION?

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Background: Carpal tunnel syndrome (CTS) is the commonest nerve entrapment disorder of the upper limb. Diagnosis is often clinical, based on a suggestive history and physical examination. Neurophysiological studies correlate closely with clinical evaluation. The use of ultrasound has been evaluated, and a feature which consistently supports a clinical diagnosis of CTS is increased cross sectional area (CSA) of the median nerve within the carpal tunnel. Local injection with corticosteroid is generally accepted as the next step in those who remain symptomatic after conservative treatment with splinting and nonsteroidal anti-inflammatory drugs. If this is unsuccessful referral for consideration of surgery should be considered.

Objectives: To evaluate the utility of ultrasound in the assessment of CTS and identify predictors of response to injection.

Methods: Patients were recruited via primary care referrals to the rheumatology department. Inclusion criteria were age over 18 years, appropriate symptoms in the distribution of the median nerve for at least 3 months, and positive nerve conduction studies (NCS). Patients with evidence of thenar atrophy were excluded. At initial review a full symptom and medical history was taken. A Boston Carpal Tunnel Questionnaire

was completed, along with visual analog scale (VAS). Ultrasound assessment was carried out by a rheumatology physician who was blinded to the above information. CSA of the median nerve was measured at the level of the proximal third of the pronator quadratus muscle and the largest CSA within the carpal tunnel. The presence or absence of tenosynovitis, Doppler signal and a bifid median nerve were noted. Steroid injection was carried out under indirect ultrasound guidance with 20mg depomedrone. Follow-up assessment was carried out 12 weeks post-injection. Repeat ultrasound scan was performed to measure the CSA of the median nerve, as before. Repeat Boston Questionnaire and VAS were recorded. After the second assessment those who had not responded adequately were referred on for consideration of surgical release.

Results: 52 patients attended for initial assessment, with 47 patients reattending for follow up. 53% of patients were discharged at follow up; the remainder were referred for consideration of surgery. 78.9% of patients met the criteria for defining CTS with a median nerve CSA 0.1cm², with 77% meeting this at the entrance to the carpal tunnel, and 55.8% at the level of the pronator quadratus muscle. A bifid nerve was noted in 12 patients.

There was no statistically significant relationship between the initial size of the median nerve on ultrasound and the change in Boston score. There was a statistically significant correlation between a decrease in the size of the median nerve at the entrance to the carpal tunnel and an improvement in the Boston score ($r=0.42$, p -value 0.003). There was no correlation between change in median nerve measurement and final outcome (discharged or referred to surgery). There was no relationship between the degree of entrapment on NCS and pre-test Boston score. There was a significant change in the Boston score of 0.944 post-injection (95% CI 0.41-1.48, p -value 0.001). There was a statistically significant change in the VAS score of 9.87 post-injection (95% CI 0.78-18.95, p -value 0.03). No patients demonstrated evidence of surrounding tenosynovitis or positive power doppler signal.

Conclusion: There is an overall improvement in symptoms of CTS, based on the Boston questionnaire, following corticosteroid injection. There is a clear role for the use of ultrasound to confirm diagnosis of CTS in symptomatic patients by measuring the CSA of the median nerve at the entrance to the carpal tunnel. There are no definite ultrasound or clinical prognostic indicators of response to injection. References:

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SHEAR WAVE ELASTOGRAPHY MUSCLE STIFFNESS MAY DIMINISH AFTER CORTICOSTEROID TREATMENT

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Background: The use of corticosteroids is associated with several adverse effects including corticosteroid-induced myopathy (CIM). CIM may cause structural alterations to the myofibres, which support the hypothesis of altered muscle stiffness as seen in histological and preclinical studies [1,2]. Shear wave elastography is an ultrasound technology that can quantify tissue stiffness non-invasively.

Objectives: To investigate the changes in muscle stiffness as measured by SWE and muscle strength tests in giant cell arteritis (GCA) patients exposed to high doses (40–60 mg/day) of corticosteroid treatment.

Methods: Fourteen GCA patients (4 males, mean age 68.2 $\pm$ 4.3 years) were recruited and evaluated at baseline, after 3 months and 6 months on prednisolone. Shear wave velocity (SWV), as a surrogate for tissue stiffness, and muscle strength were evaluated at each visit. Baseline data were compared to frequency-matched healthy controls. Linear mixed models were used to analyse the longitudinal data.

Results: The patients did not have a significantly different muscle SWV to healthy controls (all $p > 0.05$) at baseline. However, after 3 months, the quadriceps and hamstrings SWV decreased on average by 14% (range 8.3%–17.3%; $p=0.001$) and after 6 months decreased by 18% (range 10.2%–25.3% $p<0.001$). The biceps brachii SWV did not change with time ($p=0.92$) (Fig 1). The baseline, 3-months and 6-months mean SWV for the vastus lateralis were 1.62 m/s, 1.40 m/s and 1.31 m/s