(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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THU0605

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

respectively ($p < 0.001$). Muscle strength was generally preserved at follow-up. However, there were moderate to strong correlations ($r = 0.54-0.96$) between weaker muscle strength at follow-up and greater reduction in SWV.

Figure 1. Muscle stiffness changes in the quadriceps, hamstrings and biceps brachii.

Conclusion: The GCA patients showed a significant loss of muscle stiffness after 3 and 6 months of corticosteroid treatment. With further validation in larger samples, shear wave elastography may be useful for detecting subclinical CIM.

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THU0606

PROLIFERATIVE GLOBULAR SYNOVITIS, A CHARACTERISTIC ULTRASONOGRAPHIC PATTERN OF SEROPOSITIVE RHEUMATOID ARTHRITIS

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Background: Rheumatoid Arthritis (RA) and Psoriatic Arthritis (PsA) have a different ultrasound (US) patterns. Synovial changes are characteristic of RA patients and soft tissue changes are more frequently found in PsA. However, no previous studies have analysed if US findings differ between seropositive and seronegative RA patients.

Objectives: To analyse differences in the ultrasound pattern among patients with seropositive and seronegative RA. To assess if proliferative globular synovitis is characteristic of seropositive RA.

Methods: Retrospective Analysis. We collected clinical, epidemiological and ultrasound images of patients with RA who met American College of Rheumatology/European League Against Rheumatism 2010 criteria¹ with bilateral carpal and hand ultrasonography carried out during the last five years. Synovial hypertrophy (SH) and Power Doppler signal (PD) in wrist and hand (1-5 metacarpophalangeal [MCP]) were evaluated. We calculated the SH score (sum of the SH degrees of each joint), PD (sum of the PD degrees of each joint) and the total score (sum of the score of SH and PD) for each patient. We also evaluated the presence of proliferative globular synovitis, defined as big synovial hypertrophy with exophytic growth and a convex upper limit.

Results: 145 RA patients were collected. 80% were women. Mean age was 59.06 (14.8) years and the mean time of disease evolution was

114.6 (112.8) months. 68.3% were RF positive and 74.5% ACPA positive. Overall, 115 of the 145 (79.3%) patients were seropositive for RF/ACPA. 53.1% had radiographic erosions, 73.1% used conventional synthetic Disease-modifying drugs (DMARDs), 29.7% biological therapy, and 57.2% low doses of corticosteroids (<5 mg prednisone). The mean DAS28 was 2.81 (1.14), the number of swollen joints was 3 (3.4), and the C reactive protein (CRP) was 0.99 mg/dl (1.6).

No significant differences between seropositive and seronegative patients in terms of disease activity (swollen joints count [SJC], tender joint count [TJC], CRP, DAS28), treatment (use of corticosteroids, DMARDs, biological), time of evolution or US scores (SH, PD and total scores) were found. Globular synovitis was present in 62% and 13.7% of seropositive and seronegative RA patients, respectively ($p < 0.0001$).

Globally, 75 (51.7%) out of 145 patients had "globular" synovitis by US (Figure 1). 71 out of 75 patients were RF/ACPA positive (94.6%). Only four patients with seronegative RA had this US pattern ($p < 0.0001$). Furthermore, patients with "globular" synovitis had more erosions (72% vs 33%, $p < 0.0001$), higher SJC (3.3 vs 2.5, $p = 0.013$) and higher SH and PD scores ($p = 0.0001$).

Conclusion: The presence of proliferative globular synovitis was significantly associated with the presence of RF/ACPA in patients with RA. This US pattern identified a subgroup of RA patients with poor prognosis: more erosions and greater inflammatory activity both at clinical and ultrasound level.

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THU0607

CORRELATION BETWEEN ULTRASOUND AND STANDARD RADIOGRAPHY AND BETWEEN ULTRASOUND AND CLINICAL DATA IN RHEUMATOID ARTHRITIS

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Background: Early diagnosis in Rheumatoid Arthritis (RA) is essential in order to quickly introduce a background treatment to prevent joint destruction. In recent years, ultrasound has become an essential part of the initial assessment of polyarthritis because of its superior sensitivity to clinical examination and standard imaging for the investigation of synovitis, tenosynovitis and erosions.

Objectives: To evaluate concordance between clinical examination, standard radiography and joint ultrasonography in a heterogeneous group of patients with RA.

Methods: 40 patients were included in a prospective, transverse and monocentric study conducted in the radiology department of Farhat Hached University Hospital in Sousse over a period of 03 months whatever the level of activity of the disease, the duration of evolution or the treatment received. For each patient, 24 joints were evaluated for a total of 960 joints. Synovitis was scored using clinical examination, B-mode ultrasound, and pulsed Doppler. Concordance between joint swelling assessed by clinical examination, synovial thickening assessed by E-B and inflammation assessed by PD was evaluated by calculating the Kappa coefficient (k). A comparison between the reliability of ultrasound versus standard imaging was evaluated for the detection of bone erosions. The onset of RA, stage, and ultrasound score were assessed by Spearman coefficient.

Results: 960 joints were studied. The mean age was 54.6 years. The sex ratio was 0.29. The mean duration of illness was 3.4 months. Clinical examination revealed 207 active synovitis versus Ultrasonography: 474 active synovitis. The concordance between clinical examination and ultrasound was very low at the metacarpophalangeal (MCP) (<0.1), low at