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RMD Open
Rheumatic & Musculoskeletal Diseases

ORIGINAL RESEARCH

Imaging for diagnosis, monitoring and outcome prediction in polymyalgia rheumatica: a systematic literature review and meta-analysis informing an international task force

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ABSTRACT

Objective To summarise evidence on imaging for the diagnosis, monitoring and outcome prediction in polymyalgia rheumatica (PMR) to inform an international task force.

Methods Six research questions across diagnostic, monitoring, prognostic and technical aspects were defined. A systematic literature review searched Ovid MEDLINE, Ovid EMBASE, Epistemonikos and the Cochrane Library (through July 2025), including prospective and retrospective cohort studies (≥20 patients) as well as case–control and cross-sectional studies for diagnostic aspects. Test performance was summarised descriptively and meta-analysed using bivariate random-effects models when ≥2 low or some concerns on risk-of-bias (RoB) studies were available.

Results Forty-nine studies were included: 32 on diagnosis, 6 on monitoring, 13 on outcome prediction and 8 on technical aspects (some studies covered ≥1 aspect). No study was graded as low RoB. Diagnostic studies identified useful imaging lesions (eg, extracapsular and bilateral inflammatory patterns of shoulders and hips), but few were validated in independent cohorts. Diagnostic accuracy of positron emission tomography scores was reduced in the case of prior treatment and when used to differentiate PMR from other inflammatory diseases. Glucocorticoids substantially reduced sensitivity across modalities, highlighting the value of timely imaging when indicated. Data on monitoring and outcome prediction were sparse and heterogeneous.

Conclusion This systematic literature review serves as a basis for the formulation of international recommendations on the use of imaging in PMR. Available evidence was heterogeneous and of limited quality but revealed distinct imaging lesions and scores that can support PMR diagnosis. Well-designed prospective studies and external validations are needed.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Diagnosis and monitoring of polymyalgia rheumatica (PMR) are currently based predominantly on clinical evaluation.
⇒ In the absence of a specific laboratory test, differentiation of PMR from overlapping or mimicking conditions is challenging and misclassifications frequently occur.
⇒ Imaging is increasingly used in PMR; however, its value remains unclear.

WHAT THIS STUDY ADDS

⇒ We performed a systematic literature review of currently available evidence on imaging for the diagnosis, monitoring, and outcome prediction in PMR, as well as its technical aspects.
⇒ Imaging can help identify characteristic inflammatory patterns supporting PMR diagnosis, but diagnostic accuracy decreases after prior treatment and when differentiating PMR from mimicking conditions.
⇒ Data on monitoring and outcome prediction are heterogeneous and of limited quality, and the incremental value of imaging over clinical diagnosis remains unclear to date.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This SLR provides guidance to clinicians on the value of imaging in PMR. This SLR also informed an international task force formulating recommendations on the use of imaging for diagnosis, monitoring and outcome prediction in PMR.

INTRODUCTION

Polymyalgia rheumatica (PMR) is usually diagnosed and monitored clinically, but imaging modalities such as ultrasound (US), MRI and positron emission tomography (PET) have gained increasing attention in recent years both in research and clinical practice.[1 2] Current examples of imaging use in PMR include (1) the differentiation of PMR from mimicking inflammatory conditions,

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like late-onset rheumatoid arthritis (LORA) or axial spondyloarthritis (SpA) and (2) assessment of the overlap with giant cell arteritis (GCA) as well as confirmation of PMR in case of atypical presentations (eg, normal inflammatory markers).[3 4] Furthermore, PMR-like symptoms can arise as manifestations of other, non-rheumatological diseases, like malignancies or systemic infections.[5]

The current use of imaging for the assessment of PMR in clinical practice is, however, heterogeneous given the absence of clear guidance as well as the uncertainty about the incremental value of imaging beyond clinical assessment and laboratory markers. In addition, there are some practical challenges related to different imaging modalities, such as availability and costs (most relevant for MRI and PET), expertise and time constraints. The optimal placement of different imaging modalities within the diagnostic or prognostic work-up of patients presenting with suspected, newly diagnosed or relapsing PMR has not been defined, which constrains their most effective use.[6 7]

An international task force was formed to develop recommendations on the use of imaging in patients with PMR in clinical practice, focusing on diagnosis, monitoring and outcome prediction as well as on technical aspects of imaging. This systematic literature review (SLR) was undertaken to provide an up-to-date synthesis of available evidence on this topic as a basis for the discussion on the individual statements.

METHODS

Definition of research questions

We followed the European Alliance of Associations for Rheumatology (EULAR) standard operating procedures for task forces as well as the Cochrane Handbook for Systematic Literature Reviews and Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.[8 9]

The steering committee, composed of the convenors (MB, CD), two fellows (VK, EH), a methodologist (SR) and two experts in the field (KSMvdG, SES), drafted six research questions following the Population, Intervention, Control and Outcome (PICO) format.[10] These were circulated via e-mail among all task force members with the request for feedback. The task force consisted of 30 members from 12 countries and included rheumatologists, radiologists and specialists in nuclear medicine, a general practitioner, a rheumatology nurse specialist and two patient research partners. A virtual task force meeting was held on 11 November 2024, to finalise the PICOs. The six PICOs covered: (1) the diagnostic value of musculoskeletal imaging in patients with suspected PMR, (2) the diagnostic value of musculoskeletal imaging in patients in relapsing or refractory PMR (for confirmation of diagnosis), (3) the value of imaging for disease monitoring, (4) the value of musculoskeletal imaging for outcome prediction, (5) the value of vascular imaging for outcome prediction and (6) technical aspects of imaging in PMR (online supplemental tables S1–S6).

Systematic literature review

A search strategy was developed and carried out by an experienced librarian (LF). Ovid MEDLINE, Ovid EMBASE, Epistemonikos and the Cochrane Library were screened without language restrictions from database inception until 27 November 2024 (initial data-cut) (online supplemental text S1). The search was updated on 18 July 2025. Fully available primary research articles as well as conference abstracts from the 2023 and 2024 EULAR and American College of Rheumatology (ACR) conferences were considered, while SLRs were consulted exclusively for reference checking. The literature search was tested against key papers communicated by the task force. Screening of all records and full-text evaluation was performed independently by two researchers (VK and EH). Conflicts were solved by consensus or in consultation with the methodologist (SR).

We considered prospective and retrospective cohort studies that included ≥20 adult patients with suspected or confirmed PMR. For PICO 1, we also included case-control and cross-sectional studies with non-PMR controls (ie, people not suspected of having PMR). Prospective studies were considered only if ≥50% of patients completed follow-up. Studies not reporting the performance of musculoskeletal and vascular imaging for the diagnosis of PMR separately were excluded.

Outcomes of interest were test performance reflected in sensitivity, specificity, positive and negative predictive values (PPV and NPV, respectively), positive and negative likelihood ratios (LR+ and LR−, respectively) for the diagnostic PICOs (PICO 1 and 2), while we included any measure of disease activity and outcome (eg, effect sizes, risk ratios (RR), HRs, or receiver operating characteristic (ROC) curve results) for the monitoring and prognostic PICOs (PICO 3–5). For PICO 6, we collected data on technical feasibility, reproducibility, reliability measures and measures of imaging quality. The study protocol has been published on PROSPERO (CRD420251022160).

Data extraction and risk of bias assessment

Data extraction of the first 20% of full-text articles was conducted by both reviewers (VK and EH). Since inter-rater agreement of data retrieved was good (Cohen's kappa $\kappa > 0.8$), the remaining articles were divided and extracted by a single fellow only. Uncertainties were discussed with the steering committee in monthly virtual meetings. Data were extracted onto prepared spreadsheets, which included study and patient characteristics, definition of the cases (reference standard for PICO 1), and imaging modalities applied as well as all outcome data relevant for the specific PICOs. In case of incomplete reporting of diagnostic test properties, these were calculated, if possible. We defined an LR+ of >4 and an LR− of <0.4 as indicators for an imaging test with a good diagnostic value.[11]

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Risk of bias (RoB) was assessed using the Quality Assessment of Diagnostic Accuracy Studies-2 (QUADAS-2) tool for PICOs 1, 2 and 6 (diagnosis and technical aspects),[12] and the Quality In Prognosis Studies (QUIPS) tool for PICOs 3–5 (monitoring and outcome prediction).[13] Randomised controlled trials were appraised using the Cochrane's RoB-2 tool.[14] All studies were graded as 'low', 'some concerns'/'moderate'/'unclear' ('some concerns' in RoB-2, 'moderate' in QUIPS, 'some concerns' or 'unclear' in QUADAS-2) or 'high' in the respective domains of the RoB scale (online supplemental tables S7-S9). These gradings were further summarised into an overall RoB assessment.

Specific aspects relevant for this SLR that were reflected in the RoB assessment were (1) the differentiation of studies recruiting patients with an initial suspicion of PMR from studies using healthy controls or patients suffering from other underlying diseases (ie, not suspected of having PMR or other musculoskeletal conditions) as comparators. While the former reflect more closely clinical situations where imaging is of potential value, studies where diagnosis was not blinded to the index test (particularly case–control studies) often lead to an overestimation of the true value of the imaging technique due to an unrealistically high specificity. Also, (2) diagnosis of PMR or other diseases could be made at baseline only, or confirmed in subsequent follow-up, which increased the strength of findings from a specific study with regard to bias from circular reasoning and frequent reclassification of PMR patients during follow-ups. Conference abstracts were not assessed for RoB.

Data analysis

Diagnostic test properties were compared descriptively and, when reported in at least two independent cohort studies, graded as low RoB or with some concerns, meta-analysed using bivariate random-effects models. Meta-analyses compared patients with suspected PMR who were clinically diagnosed with PMR and for whom the diagnosis was confirmed after 6–12 months, to patients initially suspected of having PMR but turned out to have an alternative diagnosis. As a sensitivity analysis, patients with PMR were compared with a subset of controls with an initial suspicion of PMR who were diagnosed with other inflammatory diseases (eg, LORA or SpA) at follow-up after 6–12 months (hence, to conditions that can mimic PMR). Some cohorts were reported in several publications; however, these data were included only once in case of meta-analysis, thereby ensuring that each patient could contribute only once. Studies on monitoring, outcome prediction and technical aspects of imaging were not meta-analysed due to a substantial heterogeneity in study design, populations and outcomes observed.

All analyses were conducted in R (V.4.4.1, Vienna, Austria).[15] The meta, metafor, lme4 and forestploter packages were used for meta-analysis and result visualisation.[16–19]

RESULTS

A total of 2909 references were screened after deduplication, of which 213 were selected for full-text reading and 49 articles were finally included: 31 investigated the diagnostic value of imaging in patients with suspected or recently diagnosed PMR (PICO 1), and two studied patients with a provisional diagnosis with PMR suffering a relapse (PICO 2). Six studies assessed imaging for disease monitoring (PICO 3), 10 studied outcome prediction using musculoskeletal imaging (PICO 4) and five studied outcome prediction using vascular imaging (PICO 5). Eight studies on technical aspects of imaging in PMR were further included (PICO 6). A total of 10 studies were considered for more than one PICO. Therefore, the sum of studies across all PICOs exceeds the total number of unique studies included. Details on the study selection process and PICO assignments are provided in online supplemental figure S1 and table S10.

Imaging for diagnosis of PMR (PICO 1 and 2)

Twenty-one studies were available on fluorodeoxyglucose (FDG)-PET imaging (referred to as PET in subsequent sections),[20–40] six on US[41–46] and four on MRI.[47–50] For PICO 2 (diagnostic evaluation in relapsing PMR), one PET and one US study were included.[24 51]

FDG-positron emission tomography

Of the 21 PET studies included, none was graded as low RoB, 7 with some concerns[20–26] and 14 with high RoB.[27–40] Of the seven studies with some concerns, three had a prospective[20–22] and four a retrospective study design (table 1),[23–26] while two high RoB studies had a prospective[27 28] and 12 a retrospective design (online supplemental tables S11–S13).[29–40] Four prospective studies included patients with suspected PMR and used the clinical diagnosis after 6 or 12 months as the reference standard,[20–22 28] while one study compared patients with suspected PMR with matched disease controls[27]; all enrolled treatment-naïve patients. Retrospective studies with high RoB typically compared PMR to controls undergoing PET for reasons unrelated to PMR (such as cancer or stroke).

Four studies with some concerns on RoB were proceeded to meta-analysis (in the absence of studies with low RoB), contributing a total of 340 PMR patients and 165 controls (online supplemental table S14). A detailed description of available PET scores is provided in online supplemental tables S15, S16; of these, only the Leuven and Leuven/Groningen scores could be meta-analysed (as validated in at least one independent cohort with low RoB or some concerns).[20 21 23 25] For the Leuven Score, meta-analysis yielded a pooled sensitivity of 89% (95% CI 85 to 92) and pooled specificity of 88% (70 to 96) (figure 1A). For the Leuven/Groningen score, pooled sensitivity and specificity of two independent cohorts were 93% (88 to 95) and 93% (86 to 97), respectively (figure 1B). Pooled LR+ of the Leuven score was 7.31 (2.68 to 19.95), pooled LR– was 0.13 (0.09 to 0.18).

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Table 1 Prospective and retrospective studies on PET imaging with some concerns in risk of bias assessment*

Study ID	Study design	Population	N patients	N controls†	Reference standard	Structures investigated	Scoring method
Prospective studies (n=3)							
Henckaerts *et al*[20]	Prospective cohort study	Suspected PMR, treatment-naïve	67	32	Clinical diagnosis after≥6 months	12 MSK sites, 4 vascular sites‡	visual grading 0–2 vs liver; total visual score 0–24; positive≥16; 'Leuven Score'
Nielsen *et al*[21]	Prospective cohort study	Suspected PMR, treatment-naïve	66	27§	Clinical diagnosis after≥12 months	12 MSK sites‡	visual grading 0–2 vs liver; total visual score 0–24; positive≥16, 'Leuven Score'; dichotomous assessment (PMR yes/no)
Nielsen *et al*[22]	Prospective cohort study	Suspected PMR, treatment-naïve; investigation/ validation cohort	66 / 36	27§ / 21	Clinical diagnosis after ≥12 /≥6 months	12 MSK sites‡	visual grading 0–2 vs liver; total visual score 0–24; positive≥16, 'Leuven Score'; dichotomous assessment (PMR yes/no); combination of PET imaging and clinical diagnosis
Retrospective studies (n=4)							
Van der Geest *et al*[23]	Retrospective case–control study	Suspected PMR, treatment-naïve	39	19	Clinical diagnosis after ≥6 months	Sites for multiple scoring systems	Validation of Leuven, Besancon, Saint-Etienne, Heidelberg scores; definition of simplified 'Leuven/ Groningen score'
Moreel *et al*[25]	Retrospective cohort study	Suspected PMR, treatment-naïve	162	83	Clinical diagnosis after≥6 months	Leuven and Leuven/ Groningen score¶	Assessment of Leuven and Leuven/ Groningen scores; selection of new optimal cut-points
Van der Geest *et al*[24]	Retrospective cohort study	Suspected PMR; treatment-naïve and previously diagnosed (relapsing) PMR	45	23	Clinical diagnosis after≥6 months	Leuven and Leuven/ Groningen score¶	General assessment; validation of Leuven score and Leuven/Groningen score
Li *et al*[26]	Retrospective cohort study	Suspected PMR, mostly treatment naïve; investigation/ validation cohort	75/50	111/89	Clinical diagnosis after≥6 months	12 MSK sites**	Visual grading; proposal of integration of PET imaging in ACR/ EULAR classification criteria

*High risk of bias studies are shown in online supplemental tables S12,S13, and no low RoB studies were available.
†Initially suspected of having PMR, but receiving other diagnosis after 6 or 12 months.
‡12 MSK sites (shoulder joints, sternoclavicular joints, ischial tuberosities, hip joints, greater trochanters and cervical/lumbar interspinous bursae); 4 vascular sites (thoracic aorta, abdominal aorta, subclavian arteries, carotid arteries).
§13 with other inflammatory conditions.
¶Leuven Score = 12 MSK sites (shoulder joints, sternoclavicular joints, ischial tuberosities, hip joints, greater trochanters and cervical/lumbar interspinous bursae); Leuven/Groningen Score = 7 MSK sites (sternoclavicular joints, hips, ischial tuberosities, lumbar interspinous bursa).
**12 MSK sites (shoulder joint, acromioclavicular joint, sternoclavicular joint, costovertebral joint, sacroiliac joint, pubic symphysis, ischial tuberosity, greater trochanter of the femur, hip joint, knee joint, cervical interspinous bursa and lumbar interspinous bursa).
ACR, American College of Rheumatology; EULAR, European Alliance of Associations for Rheumatology; MSK, musculoskeletal; N, number of patients; PET, positron emission tomography; PMR, polymyalgia rheumatica; RoB, risk of bias.

For the Leuven/Groningen score, LR+ was 13.48 (6.59 to 27.59), LR− was 0.08 (0.05 to 0.13).

As a sensitivity analysis, the differentiation of PMR from other inflammatory diseases (diagnosed at follow-up, in patients initially suspected of having PMR) was meta-analysed in two independent cohorts.[21 23] Sensitivity for the Leuven score was 88% (80 to 93), while specificity decreased to 47% (27 to 69) (figure 1C). The pooled LR+ and LR− were 1.66 (1.08 to 2.57) and 0.26 (0.13 to 0.52), respectively (online supplemental table S17). Qualitative visual assessment (PMR yes/no/unclear) was assessed in three studies,[21 22 24] and three studies investigated the combination of PET with clinical or laboratory assessments or the inclusion of PET in classification criteria like the ACR/EULAR 2012 criteria.[22 26 36]

Musculoskeletal US

Six studies evaluated the diagnostic performance of musculoskeletal US in patients with suspected PMR (table 2).[41–46] Of the four prospective studies, one was rated with some concerns,[41] and three as high RoB.[42–44] The remaining two were case–control studies, both rated as high RoB (online supplemental table S19).[45 46] Overall, US enabled the detection of characteristic patterns of bilateral, proximal synovial inflammation, particularly involving the shoulders and the hips. The most frequent lesions included subacromial–subdeltoid bursitis and long-head biceps tenosynovitis (n=6), whereas hip synovitis (n=3), trochanteric bursitis (n=2) or knee synovitis (n=1) were reported in a subset of studies only.

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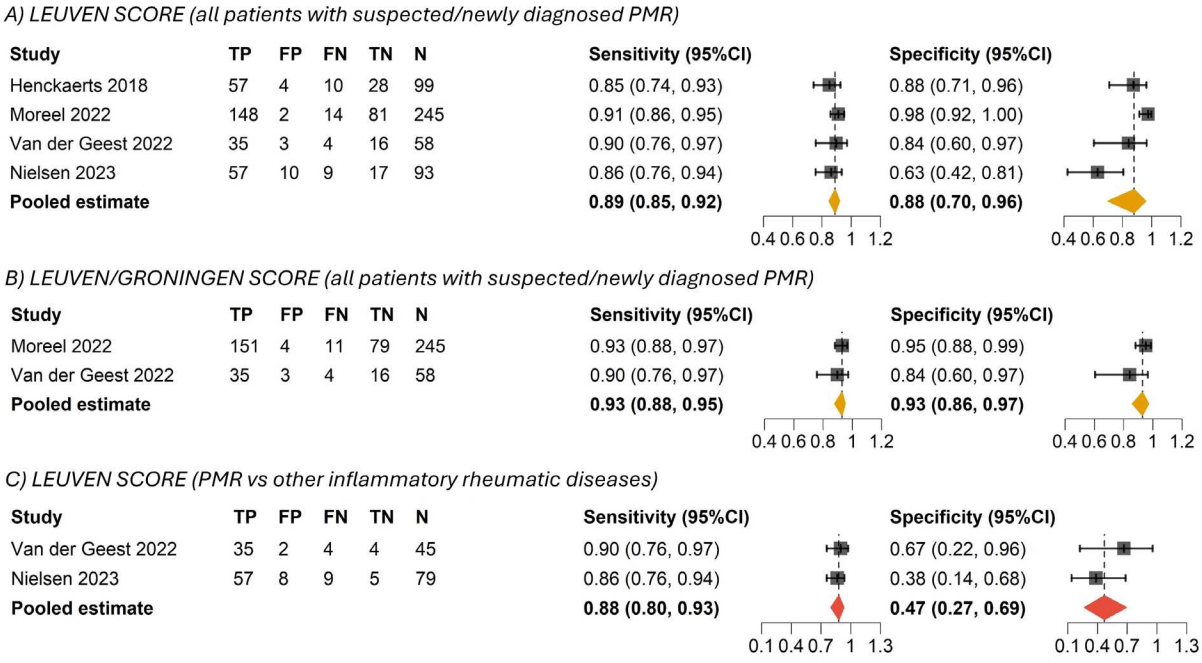


Figure 1 Bivariate random-effects meta-analysis of the diagnostic accuracy of PET scores (Leuven score and Leuven/Groningen score); for comparing PMR to all patients with suspected or newly diagnosed PMR (A and B); for comparing PMR patients to patients diagnosed with other inflammatory conditions after 6 or 12 months (after initial suspicion of PMR) (C) Leuven score (A): 12 musculoskeletal sites (shoulder joints, sternoclavicular joints, ischial tuberosities, hip joints, greater trochanters, cervical and lumbar interspinous bursae); visual grading 0–2 vs liver uptake; total visual sum score of 0–24; applied cut-off≥16[20]; Leuven/Groningen score (B): 7 musculoskeletal sites (sternoclavicular joints, hips, ischial tuberosities, lumbar interspinous bursa); visual grading 0–2 vs liver uptake; total visual sum score of 0–14; applied cut-off≥8[23]; sensitivity analysis (C)**:** shows decreased specificity of the Leuven score when comparing PMR patients to patients diagnosed with other inflammatory conditions at follow-up, after an initial suspicion of PMR; clinical diagnosis after 6 or 12 months (considering also imaging data) was used as the gold standard; FP, false positive; FN, false negative; N, number of patients; PET, positron emission tomography; PMR, polymyalgia rheumatica; TP, true positive; TN, true negative.

The prospective study with some concerns included 77 patients with suspected PMR, with clinical diagnosis confirmed after 12 months serving as the reference standard; the US algorithm of the 2012 EULAR/ACR classification criteria was tested against clinical evaluation. Elementary lesions considered were subacromial-subdeltoid bursitis, long head biceps tendon tenosynovitis and glenohumeral or hip synovitis. With US, specificity increased from 74% to 90%, while sensitivity decreased from 90% to 79%. Notably, US performed worse for the discrimination between PMR and RA (bilateral shoulder lesions occurred in 61% of PMR patients and in 60% of RA patients), but presence of additional hip lesion markedly improved specificity (table 2).[41]

Another prospective study (high RoB) suggested acromioclavicular chondrocalcinosis as indicative for calcium pyrophosphate deposition disease (CPPD), though validity was limited by the inclusion of US in diagnosis and lack of hip assessment,[42] while one prospective study (high RoB) proposed a 'shoulder-knee' US model to distinguish PMR from other conditions but was only internally validated.[44] Earlier studies (all high RoB) likely overestimated US accuracy (>90%) due to the absence of controls initially suspected of having PMR, circular diagnostic reasoning or unblinded assessments.[43 45 46]

Magnetic resonance imaging

Four studies investigated MRI in patients with suspected PMR (table 3).[47–50] Collectively, they identified a predominant pattern of extra-articular inflammation, affecting peritendinous, capsular and bursal structures around the shoulder and pelvic girdles. However, the methodological quality varied: one study was rated with some concerns on RoB,[47] and three as high RoB (online supplemental table S20).[48–50]

In a prospective cohort of 137 patients with bilateral shoulder pain and inflammatory features suggestive of PMR (some concerns), contrast-enhanced MRI of the dominant shoulder detected capsulitis, rotator-cuff peritendinitis and focal bone oedema in 69%, 72% and 59% of patients with a final clinical diagnosis of PMR, respectively, compared with 35%, 32% and 19% observed in RA ($p<0.01$ for all comparisons). The reference standard was clinical diagnosis confirmed after 12 months, and the managing clinicians were blinded to MRI findings. Individual MRI lesions revealed only moderate diagnostic

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Table 2 Studies on diagnostic accuracy of US in suspected or newly diagnosed PMR

Study	Design	Population (PMR/ controls)	Ultrasound method evaluated	Reference standard	Assessed areas	SENS (%)	SPEC (%)	LR+	LR–	RoB
Lee *et al*[41]	Prospective	38/39	The full 2012 EULAR/ACR classification score, which includes points for US findings in both shoulders (bursitis, tenosynovitis) and hips (synovitis, bursitis).	Clinical diagnosis after 1 year	ACR/EULAR criteria without US	90%	74%	3.5	0.14*	Some concerns
					ACR/EULAR criteria with US	79%	90%	7.66*	0.24*	
					Any subdeltoid (SAD) bursitis	45%	77%	1.94	0.72	
					Bilateral subdeltoid (SAD) bursitis	24%	89.7%	2.30	0.85	
					Any biceps tenosynovitis	74%	74%	2.88	0.35*	
					Bilateral biceps tenosynovitis	47%	87%	3.70	0.60	
					Any glenohumeral (GH) synovitis	24%	87%	1.85	0.87	
					Any hip synovitis	21%	97%	8.12*	0.81	
					Bilateral hip synovitis	21%	100%	NA	0.79	
					Any trochanteric bursitis	32%	87%	2.47	0.78	
					Bilateral trochanteric bursitis	11%	100%	NA	0.90	
					At least 1 shoulder and at least 1 hip inflamed	45%	95%	8.80*	0.58	
Falsetti *et al*[43]	Prospective	29/32	Shoulders, hips, distal sites (lesions+PD grading); 1 experienced reader; 5–10 MHz transducer	Clinical diagnosis after 12 months (Bird criteria)	Subacromial subdeltoid bursitis	79%	59%	1.93	0.36*	High
					Biceps tenosynovitis	79%	38%	1.27	0.55	
					Glenohumeral synovitis	66%	47%	1.25	0.72	
					Hip synovitis	24%	88%	2.00	0.86	

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Table 2 Continued

Study	Design	Population (PMR/ controls)	Ultrasound method evaluated	Reference standard	Assessed areas	SENS (%)	SPEC (%)	LR+	LR–	RoB
Ottaviani *et al*[42]	Prospective	27/67	Shoulder and AC-joint ultrasound; 1 reader (blinded)	2012 EULAR/ACR classification for PMR and/or joint fluid analysis for CPPD	Subacromial/ subdeltoid bursitis (US performance for PMR diagnosis)	96%	36%	1.49	0.11*	High
					Biceps tenosynovitis (US performance for PMR diagnosis)	85%	45%	1.54	0.33*	
					Glenohumeral effusion (US performance for PMR diagnosis)	48%	64%	1.34	0.81	
					Humeral bone erosion (US performance for CPPD diagnosis)	41%	71%	1.40	0.83	
					AC synovitis (US performance for CPPD diagnosis)	59%	86%	4.10*	0.47	
					AC chondrocalcinosis (US performance for CPPD diagnosis)	85%	97%	29.40*	0.15*	
Kobayashi *et al*[44]	Prospective	60/81	A novel 'shoulder-knee US criteria', which combines US findings of shoulder bursitis/ tenosynovitis with synovitis in the knee joint.	Clinical diagnosis after 1 year	ACR/EULAR without ultrasound	87%	68%	2.72	0.19*	High
					ACR/EULAR criteria with knee ultrasound	90%	83%	5.29*	0.12*	
					Long head of biceps tendon tenosynovitis	88%	69%	2.86	0.17*	
					Subacromial/ subdeltoid bursitis	50%	88%	4.07*	0.57	

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Table 2 Continued

Study	Design	Population (PMR/ controls)	Ultrasound method evaluated	Reference standard	Assessed areas	SENS (%)	SPEC (%)	LR+	LR–	RoB
Cantini *et al*[45]	Case–control	57/114	Bilateral shoulder US; glenohumeral joint, subacromial/ subdeltoid bursae, biceps tendon (MRI in 24 patients for validation)	Healey classification criteria	Bilateral bursitis (vs Healey classification criteria)	93%	99%	103.22*	0.07*	High
					Any bursitis (vs Healey classification criteria)	97%	78%	4.41	0.04	
					Joint synovitis (vs Healey classification criteria)	77%	42%	1.33	0.54	
					Biceps tenosynovitis (vs Healey classification criteria)	81%	47%	1.53	0.41	
					Biceps tenosynovitis (US vs MRI)	100%	100%	NA	0	
					Subacromial/ subdeltoid bursitis (US vs MRI)	93.7%	100%	NA	0.06	
					Glenohumeral joint synovitis (US vs MRI)	78.7%	93.3%	11.75	0.23	
Falsetti *et al*[46]	Case–control	50/100	Shoulders, hips, distal sites; 2 readers, blinded to clinical data	Clinical diagnosis/ classification (Healey, ARA, ESSG)	Bilateral subacromial bursitis	54%	97%	18	0.47	High
					Bilateral biceps tenosynovitis	46%	100%	NA	0.54	
					Bilateral trochanteric bursitis	70%	99%	70	0.3	
					Bilateral hip synovitis	60%	97%	20	0.41	

*good test performance (LR+ >4.0; LR– <0.4).
.AC, acromioclavicular; ACR, American College of Rheumatology; ARA, American Rheumatism Association; CPPD, calcium pyrophosphate deposition disease; ESSG, European Spondylarthropathy Study Group; EULAR, European Alliance of Associations for Rheumatology; GH, glenohumeral; LR+/-, positive/negative likelihood ratio; PD, Power Doppler; PMR, polymyalgia rheumatica; RoB, risk of bias; SAD, subacromial-subdeltoid; SENS, sensitivity; SPEC, specificity; US, ultrasound.

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Table 3 Diagnostic accuracy studies on MRI in suspected or newly diagnosed PMR

Study	Design	Population (PMR/controls)	Metrics based on unilateral vs bilateral sites	Reference standard	MRI test evaluated	SENS (%)	SPEC (%)	LR+	LR–	RoB
Studies assessing shoulder lesions										
Nakamura *et al*[47]	Case–control and prospective	58/79	Unilateral	Clinical diagnosis (unblinded)	Enhancement of shoulder joint capsule	69	65	1.97	0.48	Some concerns
					Enhancement of rotator cuff tendon	72	68	2.25	0.41	
					Focal bone oedema in humerus head	59	81	3.11	0.51	
					At least two of the three findings above	76	85	5.07*	0.28*	
					ACR/EULAR criteria+MRI; at least two of: shoulder capsule enhancement, rotator cuff tendon, focal bone oedema humeral head	93	81	4.89*	0.09*	
					ACR/EULAR criteria+US; at least one of: biceps tenosynovitis, subdeltoid bursitis, glenohumeral synovitis	86	81	4.53*	0.17*	
Fruth *et al*[50]	Retrospective	122/374	Unilateral	Clinical diagnosis (expert rheumatologist)	≥ 3 inflamed non-synovial sites on shoulder MRI	74	86	5.20*	0.31*	High
					≥2/6 non-synovial sites inflamed	84	74	3.22	0.21*	
					≥2/3 synovial sites inflamed	86	36	1.35	0.39*	
Studies assessing hip lesions										
Cantini *et al*[48]	Prospective	20/10	Unilateral	Healey's classification criteria	Trochanteric bursitis	100	80	5.00*	*0.00	High
		20/10			Hip synovitis	85	50	1.70	0.30*	
		20/10			Iliopsoas bursitis	50	80	2.50	0.63	
		20/10			Ischiogluteal bursitis	25	90	2.50	0.83	
		20/NA			Trochanteric bursitis	100	100	NA	0.00*	
		20/NA			Hip synovitis	53	100	NA	0.47	
		20/NA			Iliopsoas bursitis	60	100	NA	0.40	
		20/NA			Ischiogluteal bursitis	80	100	NA	0.20	
Fruth *et al*[49]	Retrospective	40/80	Bilateral	Bird's classification criteria	19 predefined tendinous/capsular pelvic structures; Threshold: ≥10 inflamed sites out of 19 possible locations	95.8	97.1	33.03*	0.04*	High
					19 predefined tendinous/capsular pelvic structures; criterium: bilateral inflammation of rectus femoris or adductor longus origins+3 other bilateral sites	100	97.4	38.46*	0.00*	

*Good test performance (LR+ >4.0; LR– <0.4).
ACR, American College of Rheumatology; EULAR, European Alliance of Associations for Rheumatology; LR+/–, positive/negative likelihood ratio; PMR, polymyalgia rheumatica; RoB, Risk of Bias; SENS, sensitivity; spec, specificity; US, ultrasound.

accuracy (sensitivity 59%–72%, specificity 65%–81%), while the presence of two or more lesions improved diagnostic performance (sensitivity to 76% and specificity to 85%). Incorporating MRI instead of US in the 2012 EULAR/ACR classification criteria increased overall sensitivity to 93%, while specificity remained at 81%.[47]

The remaining studies (all high RoB) supported the observation of a predominant extrasynovial pattern of PMR (as compared with RA, which involved mainly synovial structures), but were limited by smaller sample size, case–control designs or circular diagnostic reasoning.[48–50]

Diagnosis confirmation of recurrent or relapsing PMR

Only two studies were considered for PICO 2,[24 51] of which one was also included for PICO 1 and 6.[24] One retrospective study (some concerns) showed a sensitivity decrease in previously treated vs treatment-naïve patients, while specificity remained comparable[24]; another (high RoB) reported a high specificity of hand synovitis for the diagnosis of LORA in patients initially diagnosed with PMR after 1 year of follow-up (table 4).[51]

Imaging for disease monitoring in PMR (PICO 3)

Six studies investigated the role of imaging for monitoring treatment response or disease activity in PMR (table 5).[21 52–56] Overall, data were heterogeneous, and studies were graded with some concerns or high RoB (online supplemental tables S21, S22). Definitions of remission and relapse and the corresponding references are reported in online supplemental table S23.

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Table 4 Studies on the value of imaging for diagnosis confirmation in patients with recurrent or relapsing PMR (PICO 2)

Study	Design	Population (PMR/controls)	Gold standard	Scoring system applied	Previous treatment exposure	N pts	N controls	SENS (%)	SPEC (%)	LR+	LR–	RoB
Van der Geest *et al*[24]	Retrospective cohort study	Suspected PMR (treatment-naïve) or previously diagnosed PMR	Clinical diagnosis after 6 months (considering also PET data)	Leuven score	All patients	45	23	80	74	3.07	0.27*	Some concerns
					Treatment-naïve	21	8	95	75	3.81	0.06*	
					Treatment-experienced	24	15	67	73	2.50	0.46	
				Leuven/ Groningen score	All patients	45	23	82	74	3.15	0.24*	
					Treatment-naïve	21	8	95	75	3.81	0.06*	
					Treatment-experienced	24	15	71	73	2.66	0.40	
Nawata *et al*[51]	Retrospective cohort study	PMR patients reassessed after 1 year and diagnosed either as PMR or LORA	Clinical diagnosis after 12 months	Grey scale synovitis (grade 2 or 3) of the wrists	Not specified	54	18	28	98	14.63*	0.74	High
				Power Doppler of the wrist		54	18	67	94	11.91*	0.35	

*Good test performance (LR+ >4.0; LR– <0.4).
LORA, Late-onset rheumatoid arthritis; LR–, negative likelihood ratio; LR+, positive likelihood ratio; N, number of patients; PET, positron emission tomography; PICO, Population, Intervention, Control and Outcome; PMR, polymyalgia rheumatica; pts, patients; RoB, risk of bias; SENS, sensitivity; SPEC, specificity.

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Table 5 Study characteristics and quality appraisal of studies assessing the value of imaging for monitoring disease activity in PMR

Study ID	Design	Population	Imaging	Follow-up	Main results	RoB
Macchioni *et al*[52]	Prospective cohort; prednisone monotherapy 12.5 mg/day (range 12.5–17.5), fixed taper after 1 month	57 PMR patients	US (shoulders)	41 months (assessments at baseline and week 24)	GC treatment resulted in significant reduction of patients with at least one sign of US-detected inflammation (bursitis, tenosynovitis, or synovitis) compared with baseline (98% vs 65%, p<0.001) Positive PD signals were also significantly reduced by treatment	Some concerns
					Subclinical greyscale inflammation persisted in 59% of patients who were in clinical remission or low disease activity	
					Persistent US inflammation did not correlate with future relapses (28% with persisting inflammation had relapses vs 39% without, p>0.05)	
Jiménez-Palop *et al*[53]	Prospective cohort; prednisone monotherapy 10–20 mg/day (mean 15 mg)	53 PMR patients	US (shoulders+hips)	12 weeks (assessments at baseline, week 4 and week 12)	US showed significant improvement parallel to clinical parameters at 4 weeks (all sites p<0.001)	Some concerns
					US showed similar or better sensitivity to change than ESR/CRP (SRM: LHB 1.43–1.50 vs ESR 1.41, CRP 0.99)	
					US bilateral inflammation normalised at week 4 and remained normal at week 12	
Nielsen *et al*[21]	Prospective, single-arm study; prednisolone monotherapy 15 mg/day with gradual taper	66 PMR patients	FDG-PET/CT	10 weeks (assessments at baseline, week 8 and week 10)	PET/CT showed a marked reduction in metabolic activity across PMR-typical regions (shoulders, hips, ischial tuberosities, spinous processes) The PMR-PET score significantly decreased between baseline and week 8 (p<0.001), with an average SUVmax reduction of 35%. Changes in PET parameters strongly correlated with improvements in CRP and PMR-AS (r=0.7)	Some concerns
Allard *et al*[54]	Ancillary study of the ALORS RCT (abatacept vs placebo)	30 PMR patients	FDG-PET/CT	12 weeks (assessments at baseline and after week 12)	PET/CT findings correlated with the overall clinical trial outcome: no significant difference in ΔSUVmax between abatacept and placebo, consistent with the trial's negative result	Some concerns
					No significant difference in PET changes between abatacept and placebo groups; (a subgroup analysis of patients without rescue GCs showed a significantly higher decrease in SUVmax in the left sternoclavicular joint for the abatacept group)	
Palard-Novello *et al*[55]	Single-arm intervention (tocilizumab)	18 PMR patients	PET-CT	12 weeks (assessments at baseline, week 2 and week 12)	FDG uptake decreased significantly but moderately after tocilizumab therapy (SUVmax from 5.8 to 5.2 at week 2 (p=0.026) and to 4.7 at week 12 (p<0.001)) At week 12, significant SUVmax decreases occurred in all regions except shoulders and cervical spinous processes. Few significant correlations found between ΔSUVmax and clinical parameters (ΔPMR-AS, ΔCRP, ΔESR) in both patient-based and region-based analyses.	High

Continued

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Table 5 Continued

Study ID	Design	Population	Imaging	Follow-up	Main results	RoB
Devauchelle-Pensec *et al*[56]	Single-arm intervention (tocilizumab)	20 PMR patients	PET-CT+MRI + US	12 months (assessments at baseline, week 2 and week 12)	No reduction of US detected inflammation despite major clinical improvement Change in shoulder US scores from baseline to week 12 not statistically significant (p=0.148) No hip inflammation detected at any time point	High
					Disconnect observed between clinical and imaging responses; while patients experienced major improvements in clinical and laboratory variables, PET/CT showed only 'moderate improvements' PET/CT showed a significant improvement in SUV in the hips, but not in the shoulders, by week 12 (SUV hips p<0.001, shoulders p=0.081)	
					Hips showed a statistically significant improvement in MRI scores from baseline to week 12 (p=0.005) Shoulders showed no significant improvement in MRI scores from baseline to week 12 (p=0.254)	

.ALORS, abatacept in early polymyalgia rheumatica; CRP, C reactive protein; ESR, erythrocyte sedimentation rate; FDG, fluorodeoxyglucose; GC, glucocorticoid; LHB, long head of biceps tendon; PD, Power Doppler; PET/CT, positron emission tomography/CT; PMR, polymyalgia rheumatica; PMR-AS, Polymyalgia Rheumatica Activity Score; RCT, randomised controlled trial; RoB, risk of bias; SRM, standardised response mean; SUV, standardised uptake value; SUVmax, maximum standardised uptake value; US, ultrasound; VAS, visual analogue scale.

Musculoskeletal US

Two prospective studies (both some concerns) demonstrated a rapid reduction of inflammatory US lesions and Power Doppler activity within 4–12 weeks of glucocorticoid (GC) treatment.[52 53] However, residual abnormalities persisted despite clinical remission in a substantial proportion of patients (eg, US lesions persisted in 59% of patients in clinical remission, but did not predict relapse in one study[52]). In another study (high RoB), US abnormalities persisted in patients achieving clinical remission with tocilizumab treatment,[56] but the prognostic relevance of these findings remains unclear.

FDG-positron emission tomography

A prospective study (some concerns) of 66 treatment-naïve PMR patients undergoing up to three PET scans (baseline, after 8 weeks of GC treatment and after 1 week of tapering to 5 mg followed by 1 week of GC discontinuation; n=47 completed all three) showed a sensitivity decrease from 86% to 36% from baseline to week 8, which strongly correlated with CRP and the PMR activity score (r=0.7), with a partial recovery of sensitivity to 66% observed at week 10 after GC withdrawal.[21]

In contrast, findings from single-arm tocilizumab studies (n=18 and 20; both high RoB) indicated only modest PET improvements after 12 weeks of treatment without strong correlations with clinical findings,[55 56] and FDG-PET imaging data from the randomised abatacept in early polymyalgia rheumatica (ALORS) trial on abatacept (n=30; some concerns) showed no significant difference in SUVmax between treatment and placebo from baseline to week 12; these findings, however, have to be interpreted with caution because (1) the trial failed also its clinical endpoint and (2) more patients in the placebo than in the intervention group used rescue GCs (53% vs 31%).[54]

Magnetic resonance imaging

One small study (n=20; high RoB) evaluated MRI longitudinally in PMR patients treated with tocilizumab. Hip inflammation improved (p=0.005), but shoulder lesions did not (p=0.254) after 12 weeks; interpretation is limited by the single-arm design, small sample size and lack of correlation with clinical findings.[56]

Musculoskeletal imaging for outcome prediction in PMR (PICO 4)

Ten studies investigated musculoskeletal imaging for outcome prediction in PMR; four on US,[52 57–59] four on PET,[21 60–62] one on MRI[63] and one evaluating both MRI and US.[47] (online supplemental tables S24, S25). Follow-up durations ranged from 10 weeks to 5 years, and methodological quality was heterogeneous, with most studies rated as some concerns or high RoB (online supplemental table S24).

Musculoskeletal US

Five studies evaluated US with mixed results.[47 52 57–59] In prospective cohorts (all some concerns), baseline grayscale shoulder findings did not predict relapse, GC

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response or treatment duration, whereas baseline Power Doppler was linked to higher relapse rates.[47 52 57] In one retrospective study (some concerns), a semiquantitative shoulder grayscale score ≥5 independently predicted an inadequate GC response at 1 year (relapse or inability to taper prednisolone to ≤5 mg/day),[58] and another retrospective cohort (some concerns) indicated that bilateral long-head-of-biceps tenosynovitis may predict persistence of PMR diagnosis, while glenohumeral synovitis was associated with a later diagnostic shift to inflammatory arthropathies.[59]

Magnetic resonance imaging

Two small prospective studies investigated MRI: a characteristic extracapsular pattern of inflammation on whole-body MRI was associated with initial GC response but also with a higher likelihood of prolonged treatment >1 year (22 patients; some concerns),[63] and synovial hypertrophy on gadolinium-enhanced MRI was linked to a higher relapse risk (OR 7.6), whereas rotator-cuff tendon enhancement seemed protective (OR 0.2) (54 patients; high RoB).[47]

FDG-positron emission tomography

Four studies assessed PET, with mixed and largely negative results.[21 60–62] Baseline PET findings were not associated with relapse or treatment response in prospective studies (one some concerns, two high RoB),[21 60 61] in contrast to positive associations with GC dependence after 1 year in one retrospective study (high RoB),[62] and a potential predictive value of the Leuven PET during treatment, but not at baseline, for short-term relapses (seen in one single-arm with high RoB).[21]

Vascular imaging in outcome prediction in PMR (PICO 5)

Five studies evaluated the predictive value of vascular imaging in PMR, mainly assessing relapse, treatment response or GC persistence (online supplemental table S26); four studies were available on PET,[60 61 64 65] and one on vascular US.[66] Two studies were rated with some concerns and three with high RoB, mainly due to lack of blinding, treatment confounding and retrospective designs (online supplemental table S27).

Vascular US

One prospective study (some concerns) stratified 150 newly diagnosed patients at baseline into isolated PMR (n=100) and PMR with subclinical GCA (n=50), defined by the presence of intimamedia thickening in temporal, axillary or subclavian arteries. The subclinical GCA group showed a fourfold higher relapse after 22 months of follow-up (62% vs 16%, p<0.001), despite higher GC exposure at baseline (37 vs 20 mg; p<0.001); however, unblinding of treating physicians may have introduced bias.[66]

Positron emission tomography

Four PET studies (one some concerns, three high RoB) found no predictive role of baseline vascular FDG uptake for relapse or treatment response over mean follow-up durations of 10 to 37 months,[60 61 64 65] although interpretation is limited by lack of standardised vascular scoring, frequent absence of blinding and treatment confounding.

Technical aspects of imaging in PMR (PICO 6)

Eight studies were considered for PICO 6, of which seven covered technical aspects of PET imaging,[21 24 30 37–39 67] and one covered MRI.[68] Two studies were graded with some concerns,[21 24] five as high RoB[30 37–39 68] and one abstract was not assessed for RoB.[67]

GC treatment consistently reduced sensitivity and diagnostic accuracy across studies, with partial recovery observed after tapering or withdrawal (table 6).[21 24 37] Cross-sectional analyses showed significantly lower overall PET scores in GC-treated compared with GC-naïve patients as well (online supplemental table S28, table S29).[30 38 39 67] Regarding inter-rater agreement, one study (some concerns) reported highest Leuven scores at baseline (intraclass correlation coefficient (ICC) 0.87, 0.65–0.93), while GC treatment reduced agreement (ICC after 8 weeks of GCs: 0.66, 0.13–0.85), and tapering/withdrawal was again positive (ICC at week 10 after 2 weeks of GC tapering/withdrawal: 0.79, 0.36–0.91).[21]

For MRI, retrospective data (high RoB) showed that contrast-enhanced imaging detected more extracapsular inflammatory lesions than oedema-sensitive imaging (13.2% vs 5.4% inflamed sites), but T2-weighted oedema-sensitive imaging identified four of five PMR cases as well.[68]

DISCUSSION

This SLR summarises available evidence on imaging for diagnosis, monitoring and outcome prediction in PMR, to inform a task force formulating international recommendations. While US, MRI and PET imaging consistently identified characteristic periarticular inflammatory patterns across studies, current evidence is limited by heterogeneity in study designs, patient populations and outcome definitions as well as methodological concerns reflected in RoB assessments.

PET shows high diagnostic accuracy for PMR, but its specificity declines when differentiating PMR from other inflammatory diseases, which is a relevant application in clinical routine. Of note, pooled PET estimates are based on a small number of studies from selected centres, and their generalisability remains uncertain at this stage. US findings seem most reliable when detected bilaterally in shoulders and hips, while MRI may detect extracapsular inflammatory patterns characteristic of PMR; however, prospective studies are needed to confirm this. Across modalities, diagnostic performance is reduced by prior or ongoing GC treatment, which supports early imaging or GC tapering (although often challenging in routine practice). Overall, the incremental diagnostic value of imaging beyond clinical assessment remains unclear, as existing studies suffer from incorporation bias and

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Table 6 Comparison of diagnostic accuracy of FDG-PET/CT imaging in treatment-naïve and treatment-experienced patients

Study	Design	Population (PMR/ controls)	Gold standard	Scoring system applied	GC exposure	N pts	N controls	SENS (%)	SPEC (%)	LR+	LR–	RoB
Nielsen *et al*[21]	Prospective cohort study	Suspected PMR; treatment naïve	Clinical diagnosis after 12 months	Leuven score (PMR vs non-PMR)	PET at baseline (all GC naïve)	66	27	86%	63%	2.34	0.22*	Some concerns
				Leuven score (PMR vs non-PMR)	Week 8 (8 weeks of GC treatment 10–15 mg prednisone/day)	66	27	36%	41%	0.61	1.57	
				Leuven score (PMR vs non-PMR)	Week 10 (1 week of tapering +1 week of discontinuation)	66	27	66%	60%	1.65	0.57	
Van der Geest *et al*[24]	Prospective and retrospective cohort study (mixed)	Suspected PMR (treatment-naïve) or previously diagnosed PMR	Clinical diagnosis after 6 months (considering also PET data)	Leuven score	All patients	45	23	80%	74%	3.07	0.27*	Some concerns
					Treatment-naïve	21	8	95%	75%	3.81	0.06*	
					Treatment-experienced	24	15	67%	73%	2.50	0.46	
				Leuven/ Groningen score	All patients	45	23	82%	74%	3.15	0.24*	
					Treatment-naïve	21	8	95%	75%	3.81	0.06*	
					Treatment-experienced	24	15	71%	73%	2.66	0.40	
Brinth *et al*[37]	Retrospective cohort study	Age>40; referral for PET (2/3 with suspected PMR/ GCA)	Clinical diagnosis after 6 months (considering also PET data)	Copenhagen visual score	GC naïve	42	46	92%	86%	6.57*	0.09*	High
					GC experienced	36	74	38%	80%	1.90	0.78	
				Copenhagen semiquantitative score	GC naïve	42	46	89%	87%	6.85*	0.13*	
					GC experienced	36	74	43%	72%	1.54	0.79	
				Leuven score	GC naïve	42	46	81%	95%	16.2*	0.20*	
					GC experienced	36	74	24%	93%	3.43	0.82	
				Leuven/Groningen score	GC naïve	42	46	84%	89%	7.64*	0.18*	
					GC experienced	36	74	35%	83%	2.06	0.78	
				Saint-Etienne algorithm	GC naïve	42	46	84%	69%	2.71	0.23*	
					GC experienced	36	74	53%	65%	1.51	0.72	
				Heidelberg algorithm	GC naïve	42	46	86%	91%	9.56*	0.15*	
					GC experienced	36	74	36%	77%	1.57	0.83	

*Good test performance (LR+ >4.0; LR– <0.4).
FDG, fluorodeoxyglucose; GC, glucocorticoid; GCA, giant cell arteritis; LR–, negative likelihood ratio; LR+, positive likelihood ratio; N, number of patients; PET, positron emission tomography; PMR, polymyalgia rheumatica; pts, patients; RoB, risk of bias; SENS/SPEC, sensitivity/specificity.

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circular reasoning given the absence of a true gold standard beyond clinical diagnosis (often made by physicians unblinded to the imaging findings).

For monitoring, imaging modalities are sensitive to change following GC therapy but often reveal poor concordance with clinical status, particularly in patients receiving IL-6 receptor blockers. The predictive value of persistent subclinical inflammation is unclear; however, clinicians might be tempted to treat the 'image' rather than patients' complaints, with the inherent risk of overtreatment. In contrast, higher relapse rates and GC persistence in PMR patients with subclinical vasculitis according to US at baseline might suggest that imaging-based screening for large vessel inflammation has a clinical impact; however, these data result from a single study with several methodological limitations such as unblinding of the investigators to US results. In contrast, two PET studies (also with some methodological concern) did not reveal any differences between patients with and without subclinical large vessel vasculitis in regard to treatment response and relapse rate. These studies, however, are not directly comparable given that US and FDG-PET address different vascular territories, and that the former is used to evaluate vessel wall thickening while FDG-PET investigates metabolic activity. Further research is, therefore, needed to clarify the potential relevance of different imaging modalities for the assessment of subclinical arterial inflammation in PMR patients.

The strengths of this SLR lie in its comprehensive scope across all clinical domains, the rigorous RoB assessment and the integration of diagnostic, monitoring and prognostic data into a comprehensive interpretation of the imaging landscape in PMR. Its limitations reflect those of the primary literature: small and mostly single-centre cohorts with considerable selection bias, heterogeneous imaging protocols and outcome definitions, frequent lack of blinding, overfitting in primary studies without predefined statistical analysis plans and a subsequent lack of validation cohorts, and a scarcity of data on relapsing or refractory disease. In addition, PMR research remains challenged by the lack of validated reference standards and broadly accepted definitions for remission and relapse. Across many studies, PMR patients were compared with patients with non-inflammatory conditions without symptoms suggestive of PMR, and the comparison of imaging to clinical diagnosis, or the combination thereof, suffered from bias by circular reasoning as the subsequent clinical diagnosis (unblinded to imaging findings) was used as the gold standard. Due to these reasons, no study was graded with a low RoB, and meta-analyses were only feasible for a subset of PET studies with comparable methodology and patient populations. Regarding methodological rigour, a random 20% sample of studies underwent independent duplicate data extraction by two reviewers (VK, EH). The remaining studies were extracted by a single reviewer after substantial agreement had been established, but data extraction errors cannot be fully excluded.

In summary, this SLR depicts an evolving field: imaging in PMR has progressed from a purely descriptive tool to an important instrument for characterising disease anatomy, pathophysiology and underlying inflammatory biology. However, the incremental value of imaging over clinical assessment has not yet been robustly established, and translation of imaging findings into clinically meaningful outcomes will require well-designed prospective studies including external validation cohorts.

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Data availability statement All data relevant to the study are included in the article or uploaded as supplementary information. All data used in the current analysis are publicly available from the respective publications of the included trials, and are summarised in tabular form in the manuscript and supplementary appendix.

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