



Vasculitis

Systematic literature review informing the 2025 EULAR recommendations for the management of polymyalgia rheumatica and primary large vessel vasculitis: focus on referral and diagnosis

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ABSTRACT

Objectives: This study aimed to systematically review the literature on polymyalgia rheumatica (PMR) and large vessel vasculitis (LVV) referral and diagnosis to inform the 2025 European Alliance of Associations for Rheumatology recommendations on the management of PMR and primary LVV.

Methods: A systematic literature review focused on referral and diagnosis of PMR, giant cell arteritis (GCA), and Takayasu arteritis (TAK) was conducted across MEDLINE, Embase, and Cochrane for randomised trials, observational studies, and systematic reviews published from April 1, 2014 for PMR, and January 1, 2018 for LVV, to 31 December, 2024.

Results: Forty-one studies were included, all of which were observational. Fast-track clinics for suspected PMR shortened the time to diagnosis (80.53 days and 12.7 days, 2 studies, $P = .001$)

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and hospitalisations (20%-3.5%, $P = .001$). PMR-like symptoms with covert LVV at imaging were associated with a higher relapse risk compared with isolated PMR (odds ratio: 6.99, 95% CI: 2.30-21.74) and required more glucocorticoids (prednisone equivalents dose at 12 months 7.5 ± 13.5 mg/d vs 2.4 ± 3.4 mg/d, $P = .016$) and disease-modifying antirheumatic drugs (conventional synthetic 50% vs 13%, $P = .001$; biological 28% vs 2%, $P = .001$). Multimodal diagnostic approaches integrating clinical and laboratory assessment, imaging, and/or temporal artery biopsy accurately diagnosed GCA (sensitivity 92%-99%, negative predictive value 85%-99%). No evidence emerged concerning TAK referral and diagnosis.

Conclusions: Initial evidence on fast-track clinics emerged in PMR. Subclinical LVV with PMR-like presentation is increasingly recognised, but its long-term prognosis remains unclear. The value of multimodal diagnostic approaches has been shown in GCA; studies are lacking in TAK and PMR.

WHAT IS ALREADY KNOWN ON THIS TOPIC

- The latest European Alliance of Associations for Rheumatology (EULAR) recommendations for the management of polymyalgia rheumatica (PMR) and large vessel vasculitis (LVV) were published in 2015 and 2020, respectively. Since then, significant breakthroughs in the diagnosis and treatment of PMR, giant cell arteritis (GCA), and Takayasu arteritis (TAK) have been made.
- PMR and LVV were previously addressed separately. However, novel evidence about a diagnostic continuum between these diseases has emerged.

WHAT THIS STUDY ADDS

- The present systematic literature review (SLR) revealed the advantages of a quick diagnosis via fast-track pathways also in PMR.
- Preliminary data suggest that patients with PMR-like symptoms and subclinical LVV at imaging could have an increased risk of disease relapse compared with isolated PMR, especially if glucocorticoids are tapered too quickly. However, the long-term prognosis of subclinical LVV remains unknown. Multimodal diagnostic pathways comprising ultrasonography, temporal artery biopsy and PET/CT are useful in the diagnosis of GCA.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- The SLR identifies novel evidence regarding the referral and diagnosis of PMR and LVV to guide the 2025 EULAR recommendations on the management of PMR and primary LVV. The new recommendations will affect the clinical practice by improving the diagnosis, treatment and monitoring of PMR and LVV.

INTRODUCTION

The European Alliance of Association for Rheumatology (EULAR) recommendations for the management of polymyalgia rheumatica (PMR) and large vessel vasculitis (LVV) were updated in 2015 [1] and 2020 [2], respectively. Since then, significant breakthroughs in the diagnosis and treatment of PMR, giant cell arteritis (GCA), and Takayasu arteritis (TAK) have been achieved.

Fast-track clinics (FTC) for suspected GCA changed the prognosis of this disease, as earlier access to diagnosis and treatment reduced the risk of blindness [3]. The FTC paradigm often goes hand in hand with ultrasonography of superficial temporal and axillary arteries to confirm the diagnosis of GCA. Ultrasonography surpassed the gold standard temporal artery biopsy (TAB) as the first-line diagnostic modality in GCA for its accessibility and rapidity of the results [2,4]. Extracranial GCA may present without involvement of the temporal arteries or typical cranial

symptoms. It is associated with a higher risk of large artery aneurysms and more frequent disease relapses [5]. Vascular ultrasonography, 18-fluorodeoxyglucose positron emission tomography combined with computed tomography (FDG-PET/CT), computed tomography angiography (CTA), and magnetic resonance angiography (MRA) confirm the diagnosis and detect large vessel damage in this subset [4].

Access to an experienced centre is also crucial for TAK management: large vessel imaging expertise is required for diagnosis, whereas timely treatment is fundamental to prevent arterial aneurysms or stenosis [2].

GCA and PMR affect people aged >50 years, and their clinical phenotypes frequently overlap: GCA manifests in up to half of the cases with bilateral shoulder pain and stiffness, whereas about one-third of patients initially diagnosed with PMR have large vessel inflammation at imaging [6]. There is an ongoing debate on the value of screening for LVV in all patients diagnosed with PMR. Early identification and treatment of LVV might reduce relapses and complications such as large vessel aneurysms or stenosis, but it also raises concerns about potential overtreatment [6].

In recent years, there has been a renewed interest in exploring the possibility that PMR and GCA [5,6], alongside GCA and TAK [7], may lie on a disease spectrum, supported by the fact that treatment with interleukin 6 antagonists proved to be effective in both PMR and LVV [8,9].

This systematic literature review (SLR) aimed to identify new evidence on the referral and diagnosis of PMR and LVV to inform the 2025 EULAR recommendations for the management of PMR and primary LVV (under submission). A parallel SLR explored the treatment and management of relapses and complications in PMR and primary LVV (under submission).

METHODS

Search strategy and inclusion criteria

The protocol for this SLR was registered in the PROSPERO database of systematic reviews on June 21, 2024 (CRD42024560403) [10].

The research domains of interest were identified based on a survey among 26 expert rheumatologists forming the respective EULAR task force (Supplementary Material S1).

The scope of the SLR and eligibility criteria for studies were defined using the Population, Intervention, Comparator, Outcome (PICO) framework (Supplementary Material S1).

Relevant search terms to identify eligible studies were formulated starting from 12 PICO questions. Predefined relevant terminology for each database was used: Medical Subject Headings (MeSH) for MEDLINE and Cochrane Library, and Emtree terms

for Embase ([Supplementary Material S1](#)). Free-text words were searched for within keyword lists, titles, and abstracts.

Population of interest: PMR, GCA and TAK. Other LVV subtypes, such as isolated aortitis and immunoglobulin G4-related disease, were not included because it was beyond the scope of these EULAR recommendations. Age was limited to adults >18 years.

Interventions: referral from primary care, fast-track pathways, diagnosis with TAB or imaging (primarily ultrasonography, FDG-PET/CT, CTA, and MRA) alone or in combination.

Comparators: primary care management, regular (nonrapid access) referral pathways, clinical diagnosis, diagnosis based on another modality (TAB or imaging), different from the intervention.

Outcomes: diagnostic efficiency (sensitivity, specificity, positive/negative predictive value [NPV], likelihood ratio, accuracy, and area under the curve), time to diagnosis, effect on disease activity, disease-related complications (disease-related damage like blindness, vessel aneurysms or stenosis, mortality, hospitalisation), treatment-related complications, and healthcare and social costs.

Articles related to the sole use of imaging for diagnostic purposes were later excluded (PICO 4, 8, and 11), as imaging for the diagnosis of LVV was the objective of another recent SLR and related set of EULAR recommendations [4]. However, studies incorporating imaging in the frame of multimodal diagnostic algorithms or comparing the diagnostic performance of imaging with other diagnostic modalities were included.

Randomised trials, observational studies (cohort, case-control, cross-sectional), SLRs, and meta-analyses were considered. EULAR 2024 and American College of Rheumatology (ACR) 2024 conference abstracts were manually searched to identify completed manuscripts of relevant studies published after the end date of the SLR search. Studies with ≥ 20 participants and < 20% lost to follow-up were considered to limit potential bias.

The SLR output was cross-checked with the task force to ensure that key publications in the field of PMR and LVV were captured.

The search was run by a librarian (JL) on electronic databases including Ovid MEDLINE, Embase, and Cochrane Library. The search interval started from May 1, 2014 for PMR and January 1, 2018 for LVV, up to June 11, 2024. The search was later extended to December 31, 2024, to ensure inclusion of any recent relevant work. No language restriction was applied.

Study selection, data extraction, risk of bias assessment

Literature retrieved from the search was uploaded into End-Note V.X9 (Clarivate). Rayyan, an online software for citation management, was used for the screening process [11]. All duplicates were removed. Articles identified in both the 2018 LVV and 2015 PMR SLRs were excluded. Two reviewers (AB and MD) independently performed a round of title and abstract screening followed by full-text screening of the selected articles, both with 100% cross-validation. Any disagreement between the reviewers was discussed. Expert rheumatologists (CBM and BH) and methodologists (EN and PS) were consulted if the reviewers reached no consensus on article inclusion. After screening through titles and abstracts, definitive study inclusion was confirmed via full-text review.

Information from eligible papers was collected using a standardised data extraction sheet: study characteristics, study design, inclusion and/or exclusion criteria, population characteristics and demographics, intervention, comparator, and

outcomes. A 20% cross-validation of extracted information was performed.

Risk of bias (RoB) was assessed with the Cochrane Risk of Bias Assessment Tool for randomised controlled trials (RCTs) version 2 and the Newcastle-Ottawa scale for observational studies [12,13]. Each study was assigned a RoB rating of 'low', 'some concerns', or 'high'.

RESULTS

The literature search for the referral and diagnosis of PMR and LVV yielded 4833 records. By deduplication, 528 duplicates were removed. Abstract and title screening was performed on 4305 records, with 191 studies included for full-text screening. Thirty-five studies met the inclusion criteria for data extraction (34 from the literature search, 1 from the manual search). Six studies were added following the search update to December 2024 (3 from the updated literature search, and 3 from the manual search). All 41 selected studies were observational. No RCTs were found. Thirty articles included patients with GCA, 10 included patients with PMR, and 1 included patients with TAK. The PRISMA (Preferred Reporting Items for Systematic reviews and Meta-Analyses) flow diagram is shown in [Supplementary Figure S1](#).

The agreement between the reviewers was 97% for title and abstract screening and 93% for full-text screening. Agreement at data extraction cross-validation was 100%.

Meta-analysis was not performed because it was deemed inappropriate due to the large heterogeneity of the studies.

PMR

Referral of suspected PMR from primary care (PICO 1)

Three studies addressed the referral of suspected PMR cases from primary care, all of which included FTC ([Table 1](#), [Supplementary Table S1](#)) [14–16]. FTC was based on referral criteria such as age >50 years, raised inflammatory markers, symptoms of PMR, and the absence of cranial GCA symptoms, as well as a waiting time between general practitioner (GP) referral and first rheumatology visit of 3 [14,15] to 7 days [16].

Frølund et al [16] (retrospective study, some concerns RoB) found that, upon FTC implementation, fewer patients were started on glucocorticoids by their GP (11% FTC vs 42% before FTC, $P < .001$). However, a significant difference in glucocorticoid dosage before and after FTC implementation was not found ([Table 1](#)) [14–16].

FTC for suspected PMR (PICO 2)

Three studies showed a favourable impact of FTC on PMR diagnosis and outcomes ([Table 1](#), [Supplementary Table S1](#)) [14–16]. Chrysidis et al [15] showed that, compared with historical data between 2013 and 2017, FTC implementation since 2018 decreased the mean (SD) time to diagnosis from 12.3 (12.3) to 6.8 (0.7) days, $P = .001$ (retrospective study, some concerns RoB). FTC also reduced the need for patients' hospitalisation from 20% to 3.5% ($P = .001$). The number of inpatient days decreased from a mean (SD) of 4 (3) days to 1 (0) day ($P < .00001$). As a result, FTC implementation reduced the annual patient cost by 65%. Similarly, Frølund et al [16] found that the median (IQR) time to diagnosis with FTC between 2016 and 2019 was 53 (31–83) days compared with 80 (58–132) days in 2014 to 2016 before FTC implementation, $P = .001$. The number of hospital contacts before reaching the diagnosis was also

Table 1
Fast-track pathways for PMR referral

	Study, year	Population	Intervention	Comparator	Primary outcome		Key secondary outcomes		Risk of bias
Prospective observational	Cowley, 2024 [14]	Suspected PMR referred to FTC (N = 172)	Referred to FTC steroid naïve (N = 48)	Initial primary care management of suspected PMR (N = 39)	Alternative diagnosis, n (%)	67 (39)	Starting PDL dose, mg/d, mean (SD)	16.77 (6.14) in steroid naïve 23.18 (10.06) in patients commencing steroids < 30 d in primary care P = .54 63.5 (30-95)	Some concerns
Retrospective observational	Chrysidis, 2021 [15]	PMR diagnosis (N = 310)	PMR diagnosis with FTC (N = 56)	PMR diagnosis before FTC implementation (N = 254)	Hospitalisation, n (%)	2 (3.5) in FTC 52 (20.4) in controls P = .001	Starting PDL dose, mg/d, mean (SD)	18.4 (8.3) in FTC 19.2 (9.3) in controls P = .6 6.8 (0.7) in FTC 12.3 (12.3) d in controls P = .001	Some concerns
	Frølund, 2021 [16]	PMR diagnosis (N = 180)	PMR diagnosis with FTC (N = 83)	PMR diagnosis before FTC implementation (N = 97)	Time to diagnosis, days, median (IQR)	53 (31-83) in FTC 80 (58-132) in controls P < .001	Alternative diagnosis, n (%)	29/112 (26)	Some concerns
					Starting PDL dose, mg/d, median (IQR)	15 (15-15) in FTC 15 (15-20) in controls P < .001			
					PDL prescribed before rheumatological assessment, n (%)	9 (11) in FTC 41 (42) in controls P < .001			

FTC, fast-track clinic; PDL, prednisolone; PMR, polymyalgia rheumatica.

reduced (median [IQR] 1 [0-17] historical cohort vs 0 [0-8] FTC, $P < .001$).

Protocolised diagnostic workup for PMR (PICO 3)

Three studies on FTC in PMR integrated clinical history, physical examination, and laboratory markers of inflammation (Table 1, Supplementary Table S1) [14–16]. Furthermore, Cowley et al [14] (prospective study) included shoulder X-rays and vascular ultrasonography in the panel. Of 172 patients referred to FTC for suspected PMR, 67 (39%) had an alternative diagnosis, mainly inflammatory arthritis, crystal arthropathies, myositis, vasculitis, osteoarthritis, fibromyalgia, and malignancy. The study lacked a comparator group with standard referral and diagnostic workup; therefore, the RoB was ‘some concerns’.

Chrysidis et al [15] additionally performed musculoskeletal ultrasonography and vascular ultrasonography.

The protocol from Frølund et al [16] also included a low-dose chest CT, abdominal ultrasonography, and, according to clinical suspicion, musculoskeletal ultrasonography and joint X-rays. Of the 113 patients referred via FTC, 1 was lost to follow-up, and 29 (26%) had an alternative diagnosis, the most common being GCA, rheumatoid arthritis, other inflammatory disorders, degenerative joint disease, or neoplasia.

Diagnosis of LVV in PMR (PICO 5)

Nine observational studies on the detection of LVV in patients presenting with a PMR-like picture were retrieved [14,17–24].

The prevalence of covert LVV, ie, LVV at imaging in patients with suspected PMR (presenting with limb girdle pain and stiffness, in the absence of cranial symptoms of GCA), was largely variable across 6 studies (Table 2, Supplementary Table S2) [14,17–21]. Nielsen et al [19] in a prospective study with low RoB found that systematic screening of newly diagnosed PMR with clinical examination, labs, ultrasonography of superficial temporal and axillary arteries, and FDG-PET/CT resulted in 3% being reclassified as subclinical GCA. In addition, 3% patients with an initial diagnosis of PMR developed covert as well as clinically overt LVV later in the course of the disease (IR 32/1000 patient-years).

Vascular ultrasonography found covert LVV in 17% to 23% newly diagnosed PMR (2 prospective studies, RoB: some concerns) [14,17].

FDG-PET/CT detected covert LVV in 8% to 9% (2 retrospective studies, RoB: some concerns, mainly driven by possible selection bias) [20,21]. An explanation of the discrepancy between the findings on ultrasonography and FDG-PET/CT could be that the latter is less sensitive for cranial-limited covert LVV. No studies comparing the diagnostic performance of ultrasonography and FDG-PET/CT for covert LVV were found. However, when FDG-PET/CT was performed in PMR with atypical features or refractory/relapsing disease, the prevalence of covert LVV was up to 65% (3 studies, RoB: some concerns) (Table 2, Supplementary Table S2) [22–24].

Clinical features and outcomes in patients with covert LVV vs isolated PMR (PICO 6)

Six studies with RoB ‘some concerns’ found no unequivocal features distinguishing subclinical LVV from isolated PMR at presentation (Supplementary Table S3) [17,18,20,22,24,25]. Possible red flags for an underlying LVV in a patient with a PMR-like presentation included older age, higher levels of C-reactive protein (CRP), constitutional symptoms, neck pain, pelvic girdle pain, relapsing disease, or refractoriness to glucocorticoid treatment [17,18,20,22,24,25].

Covert LVV differed from isolated PMR in 3 studies that looked at outcomes (Table 3, Supplementary Table S4) [20,22,25]. De Miguel et al [25] documented that PMR with covert LVV prospectively recruited into an international cohort had a greater risk of relapse compared with isolated PMR (odds ratio: 6.99, 95% CI: 2.30-21.74) (RoB: some concerns). Covert LVV received more glucocorticoids and immunosuppressants compared with isolated PMR (mean [SD] dose in prednisone equivalents at diagnosis 36.6 [14.3] mg/d vs 19.5 [7.4] mg/d, $P = .001$; mean [SD] dose in prednisone equivalents at 12 months 7.5 [13.5] mg/d vs 2.4 [3.4] mg/d, $P = .016$; 50% vs 13% on conventional synthetic disease-modifying antirheumatic drugs [DMARD], $P = .001$; 28% vs 2% on biological DMARD, $P = .001$). These findings were in line with a prior retrospective study by Arévalo-Ruales et al [22] (RoB: some concerns), where 73% patients with covert LVV, compared with 25% isolated PMR, were started on an additional immunosuppressant to achieve disease control. Moreel et al [20] also found that covert LVV were administered a greater glucocorticoid dose than isolated PMR at diagnosis and in the first year of treatment, but did not find any difference in DMARDs use (10% vs 10%, $P = 1$) (retrospective study, RoB some concerns). Notably, the greater immunosuppressive burden of the covert LVV group can be biased by the tendency to treat LVV more aggressively than PMR.

Laboratory biomarkers in the diagnosis of PMR (PICO 12)

No studies were found.

GCA

Referral of suspected GCA from primary care (PICO 1)

One retrospective study with low RoB by Van Nieuwland et al [26] addressed the diagnostic delay in patients with GCA referred to FTC from primary care (Table 4, Supplementary Table S5). The median (IQR) time between symptom onset and the visit was 31.0 (13.0-108.8) days. There was still a considerable waiting time between symptom onset and the first GP visit (median [IQR] 10.5 [4.0-36.3] days), and between GP evaluation and referral to FTC (median [IQR] 10.0 [1.0-47.5] days). Median [IQR] referral time was shorter in cranial GCA (21.0 [11.0-73.5] days), especially with ocular symptoms (14.5 [4.3-29.5] days), compared with extracranial GCA (57.0 [33.0-105.0] days).

FTC for suspected GCA (PICO 2)

Two observational studies regarding FTC in GCA were found (1 with low RoB and 1 with some concerns) (Table 4, Supplementary Table S5) [27,28]. Compared with historical cohorts, implementation of an ultrasonography-based FTC reduced by nearly half the rate of visual loss: from 27% to 13% ($P = .03$) [28] and from 19% to 12% [27]. Monti et al [28] also found a reduction in the occurrence of all GCA-related ischaemic events (29%-13%, $P = .02$). Time to diagnosis was reduced from a median (IQR) of 62 (20-122) days in the historical cohort to 33 (15-83) days with FTC ($P = .04$). However, an earlier diagnosis and treatment with FTC did not seem to reduce the relapse risk (Supplementary Table S5).

Protocolised diagnostic workup for GCA (PICO 7)

Four studies exploring multimodal pathways combining clinical, laboratory, and imaging findings for the diagnosis of GCA were retrieved (Table 5, Supplementary Tables S6 and S7) [29–32].

Table 2

Prevalence of subclinical LVV in patients presenting with a PMR-like picture in the absence of symptoms of cranial arteritis

	Study, year	Population	Intervention	Outcome	Risk of bias		
Prospective observational	Nielsen, 2024 [19]	Newly diagnosed PMR without cranial symptoms (N = 64)	Screening for subclinical GCA: clinical examination, labs, STA + AX US and FDG-PET/CT at diagnosis (N = 64)	(S) Subclinical GCA, n (%)	2 (3)	Low	
		Newly diagnosed PMR without LVV at diagnosis (N = 62)	Monitoring for late-onset GCA (N = 62) • Labs every 4 wk • Clinical examination, STA + AX US and FDG-PET/CT at week 8 (on GCs) and 10 (short-term GC discontinuation) • Clinical examination, STA + AX US at 1 y	(P) Late-onset GCA (clinically overt or subclinical), n (%)	2 (3) IR 32/1000 person-years		
	Cowley, 2024 [14]	Newly diagnosed PMR without cranial symptoms (N = 84)	US of STA + AX (N = 84)	(S) Subclinical GCA, n (%)	14 (17)	Some concerns	
	De Miguel, 2024 [17]	Newly diagnosed PMR without cranial symptoms (N = 346)	US of STA, AX, CA, SC (N = 346)	(P) Subclinical LVV, n (%)	79 (22.8) Isolated extracranial LVV: 31 (39.2) Cranial + extracranial LVV: 26 (32.9) Isolated cranial LVV: 22 (27.9)	Some concerns	
Retrospective observational	Moreel, 2024 [20]	Newly diagnosed PMR without LVV symptoms (N = 337)	FDG-PET/CT (N = 337)	(P) Subclinical LVV, n (%)	31 (9) Isolated extracranial LVV: 21 (58) Cranial + extracranial LVV: 7 (23) Aorta only: 5 (20) Isolated cranial LVV: 3 (10)	Some concerns	
Prospective observational	Lund-Petersen, 2017 [21]	Newly diagnosed PMR (N = 50)	FDG-PET/CT (N = 50)	(S) Subclinical LVV, n (%)	4 (8)	Some concerns	
	Lavado-Pérez, 2015 [24]	PMR with suspected LVV (N = 40)	FDG-PET/CT (N = 40)	(P) Subclinical LVV, n (%)	26 (65)	Some concerns	
Retrospective observational	Serrano-Combarro, 2024 [23]	PMR with suspected LVV (N = 75)	GC-resistant PMR (N = 28)	GC-naïve PMR (N = 12)	(S) Subclinical LVV at PET/CT, n (%)	20 (71) in GC-resistant PMR 6 (50) in GC-naïve PMR	Some concerns
			FDG-PET/CT (N = 75)	(S) Subclinical LVV, n (%)	49 (65.3)		
				GC-resistant PMR (N = 61)	GC-naïve PMR (N = 14)	(S) Subclinical LVV at PET/CT, n (%)	38 (62.3) in GC-resistant PMR 13 (92.9) in GC-naïve PMR P = .03
	Arévalo-Ruales, 2019 [22]	PMR with suspected LVV (N = 23)	FDG-PET/CT (N = 23)	(P) Subclinical LVV, n (%)	11 (48)	Some concerns	

AX, axillary artery; CA, carotid artery; FA, facial artery; FDG-PET/CT, 18-fluorodeoxyglucose positron emission tomography with computed tomography; GC, glucocorticoid; GCA, giant cell arteritis; IR, incidence ratio; LVV, large vessel vasculitis; MSK, musculoskeletal; P, primary outcome; PMR, polymyalgia rheumatica; S, secondary outcome; SC, subclavian artery; STA, superficial temporal artery; US, ultrasonography; VA, vertebral artery.

Table 3

Subclinical LVV vs isolated PMR outcomes

	Study, year	Population	Intervention	Comparator	Outcomes					Risk of bias
						Intervention	Comparator	P value	Effect (95% CI)	
Prospective observational	De Miguel, 2024 [25]	Newly diagnosed PMR (N = 150)	Subclinical LVV (N = 50)	Isolated PMR (N = 100)	(P) GC dose at baseline, mg/d PDN equivalents, mean (SD)	36.6 (14.3)	19.5 (7.4)	.001	–	Some concerns
					(P) GC dose at 12 mo, mg/d PDN equivalents, mean (SD)	7.5 (13.5)	2.4 (3.4)	.016	–	
					(P) csDMARD, n (%)	25 (50)	13 (13)	.001	–	
					(P) bDMARD or tsDMARD, n (%)	14 (28)	2 (2)	.001	–	
					(P) Relapse, n (%)	31 (62)	16 (16)	<.001	OR: 6.99 (2.30-21.74)	
					(P) GC dose at 3 mo in relapsing patients, mg/d PDN equivalents, mean (SD)	13.4 (7.1)	18.8 (7.5)	.016	OR: 0.90 (0.81-0.99)	
Retrospective observational	Moreel, 2024 [20]	Newly diagnosed PMR (N = 337)	Subclinical LVV (N = 31)	Isolated PMR (N = 306)	(S) GC starting dose, mg/d MPDN equivalents	24.3	14.6	<.001	–	Some concerns
					(S) GC dose during follow-up	Higher in the first 12 mo		<.001	–	
					(S) Need for DMARD, n (%)	3 (10)	31 (10)	1	–	
					(S) Relapse	–	–	–	Hazard ratio: 0.82 (0.50-1.36)	
	Arévalo-Ruales, 2019 [22]	Newly diagnosed PMR (N = 23)	Subclinical LVV (N = 11)	Isolated PMR (N = 12)	(S) Time to first relapse, months	14	11	.03	–	Some concerns
					(S) Need for DMARD, n (%)	8 (73)	3 (25)	–	–	

bDMARD, biological disease-modifying antirheumatic drug; csDMARD, conventional synthetic DMARD; DMARD, disease-modifying antirheumatic drug; GC, glucocorticoid; HR, hazard ratio; LVV, large vessel vasculitis; MPDN, methylprednisolone; NS, not significant; OR, odds ratio; P, primary outcome; PDN, prednisone; PMR, polymyalgia rheumatica; S, secondary outcome; tsDMARD, targeted synthetic DMARD.

Table 4

Fast-track pathways for GCA referral

	Study, year	Population	Intervention	Comparator	Primary outcome		Key secondary outcomes		Risk of bias
Retrospective observational	Van Nieuwland, 2024 [26]	Suspected GCA referred to FTC (N = 205)	Cranial GCA (N = 41)	Extracranial GCA (N = 20) Cranial GCA with visual symptoms (N = 16)	Time between symptom onset and FTC visit ^a , days, median (IQR)	31.0 (13.0-108.8)	Delay from symptom onset to treatment initiation, days, median (IQR)	21.0 (11.0-73.5) in cranial GCA 57.0 (33.0-105.0) in extracranial GCA (P = .02) 14.5 (4.3-29.5) in cranial GCA with visual symptoms (P <.001)	Low
					Time between symptom onset and first GP visit, days, median (IQR)	10.5 (4.0-36.3)			
					Time between first GP visit and FTC referral, days, median (IQR)	10.0 (1.0-47.5)			
	Mansfield Smith, 2023 [27]	GCA diagnosis (N = 350)	GCA diagnosis with FTC (N = 243)	GCA diagnosis before FTC implementation (N = 107)	PVL, n (%)	28 (11.5) in FTC 20 (18.7) in controls			Some concerns
	Monti, 2020 [28]	GCA diagnosis (N = 170)	GCA diagnosis with FTC (N = 63)	GCA diagnosis before FTC implementation (N = 97)	PVL, n (%)	8 (12.7) in FTC 26 (26.8) in controls P = .03	Time to diagnosis, days, median (IQR)	33 (15-83) in FTC 62 (20-122) in controls P = .04	Some concerns

FTC, fast-track clinic; GCA, giant cell arteritis; GP, general practitioner; PVL, permanent visual loss.

^a Patients were generally seen at the FTC within 1 d after referral.

Table 5
Diagnostic pathways for GCA

	Study, year	Population	Intervention	Comparator	Outcomes							Risk of bias			
						Sens (%)	Spec (%)	PPV (%)	NPV (%)	Acc (%)	AUC				
Prospective observational	Denis, 2024 [29]	Suspected GCA (N = 165)	Diagnostic pathway: STA US → TAB → other imaging/ expert opinion	Diagnosis by an expert clinician	(P) cranial GCA diagnosis		99.2	100	100	99.3	–	–	Some concerns		
	Estrada, 2024 [30]	Suspected GCA (N = 69)	Diagnostic pathway: clinical suspicion → US → FDG-PET/CT → TAB	Diagnosis by an expert clinician	(P) GCA diagnosis	High suspicion US + FDG-PET/CT + TAB	92.3	100.0	100.0	71.4	–	–	Some concerns		
	Mukhtyar, 2024 [31]	Suspected GCA (N = 1000)	Diagnostic pathway: US → TAB or FDG-PET/CT if CRP > 20 mg/L → TAB or FDG-PET/CT if still high clinical suspicion and previous studies were negative/ inconclusive	Diagnosis by an expert clinician at 6 mo	(P) GCA diagnosis		95.7	–	–	98.0	–	–	Low		
						STA US with/without other arterial districts	Diagnosis by an expert clinician at 6 mo	(S) GCA diagnosis	STA US	62.3	–	–	84.7	–	–
									STA + AX US	78.7	–	–	90.1	–	–
									STA + AX + other artery US based on clinical picture	86.1	–	–	93.8	–	–
	Ludwig, 2023 [32]	Suspected GCA (N = 21)	Diagnostic pathway: clinical suspicion → STA US → FDG-PET/CT → expert opinion	TAB	(P) GCA diagnosis	Pathway	92.3	100	100	88.9	95.2	–	Some concerns		
						TAB	38.5	100	100	50.0	61.9	–			

AX, axillary artery; CRP, C-reactive protein; FDG-PET/CT, 18-fluorodeoxyglucose positron emission tomography with computed tomography; GCA, giant cell arteritis; P, primary outcome; S, secondary outcome; STA, superficial temporal artery; TAB, temporal artery biopsy; US, ultrasonography.

A pathway by Denis et al [29] based on superficial temporal artery ultrasonography, followed by TAB in case of inconclusive ultrasonography, optional additional imaging, and, finally, diagnosis based on an expert's opinion had 99% sensitivity and 96% NPV for the diagnosis of cranial GCA (RoB: some concerns). The reported specificity for the diagnosis of GCA was 100%, but this value is difficult to contextualise because there are no data about the general population.

Estrada et al [30] presented a pathway based on clinical suspicion, followed by ultrasonography, FDG-PET/CT, and TAB if the previous modality was negative or inconclusive (RoB: some concerns). Among patients with a high clinical suspicion of GCA based on symptoms and laboratory tests, a combination of the 3 modalities resulted in a sensitivity for the diagnosis of GCA of 92%, 100% reported specificity, and 71% NPV.

Mukhtyar et al [31] tested the diagnostic performance of a protocolised multimodal pathway for the diagnosis of GCA with ultrasonography, followed by TAB and/or FDG-PET/CT if ultrasonography was negative but the clinical suspicion remained high in the presence of CRP > 2 g/L; 2 mg/dL (low RoB). The pathway provided high sensitivity (96%) and NPV (98%): of 1000 patients investigated, only 7 were initially classified as not having GCA but were diagnosed with GCA at a subsequent re-evaluation. It was also confirmed, as previously reported that extending ultrasonography to the axillary arteries and, based on the clinical picture, to other vascular districts, improved the diagnostic yield (sensitivity 62.3% and NPV 84.7% superficial temporal artery alone, sensitivity 78.7% and NPV 90.1% superficial temporal and axillary artery, sensitivity 86.1% and NPV 93.8% extended ultrasonography).

Ludwig et al [32] demonstrated that a pathway using superficial temporal artery ultrasonography, followed by FDG-PET/CT and a second opinion by an expert clinician in case of inconclusive findings but high suspicion of LVV, was more sensitive than the sole use of TAB (92% vs 39%) (RoB: some concerns). NPV was 89%, whereas the reported specificity was 100%. The algorithm also decreased the time to diagnosis (median [IQR] 1.1 [0.9-1.5] days for ultrasonography, 1.9 [1.6-2.7] days for FDG-PET/CT, 10.8 [6.4-14.6] days for TAB).

TAB technique (PICO 9)

The benefit of specimen length on the diagnostic performance of TAB was unclear (Supplementary Table S8) [33–35]. One prospective study documented a higher sensitivity with specimens ≥ 15 mm in length (52% vs 14% with TAB 10-14 mm) [33], but 2 other retrospective studies did not [34,35]. Two studies had a low RoB, and one had RoB with 'some concerns'.

Three retrospective studies (2 low RoB and 1 RoB with some concerns) evaluated the potential advantages of performing bilateral TAB to improve the sensitivity of this procedure (Supplementary Table S9) [34–36]. The discordance rate between the 2 sides of the biopsy was 3% to 12%. Frozen section TAB can be useful to rapidly identify negative samples and proceed with contralateral biopsy, resulting in an overall sensitivity of 83% (Supplementary Table S10) [36].

Five studies (2 low RoB and 3 RoB with some concerns) found that TAB sensitivity was higher if performed before initiating glucocorticoids or within a few days thereafter (Supplementary Table S11) [33,34,37–39]. Each study used different timing cut-offs: TAB sensitivity was as high as 74% on glucocorticoids ≤ 3 days, but it rapidly dropped within the first week of therapy.

TAB vs ultrasonography for the diagnosis of GCA (PICO 9)

Nine studies compared the diagnostic performance of TAB with ultrasonography of superficial temporal with/without axillary and other extracranial arteries (Supplementary Table S12) [33,37,40–46]. Mourot et al [45] and Zarka et al [44] reported comparable diagnostic performance of superficial temporal and axillary arteries ultrasonography and TAB when compared with clinical diagnosis as a reference standard (ultrasonography sensitivity 82%-93% and specificity 90%-99%, TAB sensitivity 66%-77% and specificity 100%) (RoB: some concerns) [44,45]. Another prospective comparative study by Oshinsky et al [41] showed good agreement for GCA diagnosis between TAB and cranial and extracranial arteries ultrasonography (low RoB). Of note, ultrasonography also detected an isolated extracranial LVV that would otherwise be missed by TAB. Other studies reported variable sensitivity of superficial temporal artery ultrasound for the diagnosis of GCA (3 low RoB and 3 RoB with some concerns) [33,37,40,42,43,46].

Preoperative superficial temporal artery ultrasonography was positive in only 27% patients who turned out to have a positive TAB (RoB some concerns) [34]. Conversely, sequential TAB performed when superficial temporal artery ultrasonography was inconclusive or negative still resulted in a sensitivity of 39% for GCA diagnosis (low RoB) [33].

TAB vs FDG-PET/CT for the diagnosis of GCA (PICO 9)

A prospective study by Lambæk et al [39] assessed the diagnostic potential of TAB vs FDG-PET/CT (Supplementary Table S13). Using clinical diagnosis by an expert physician at 6 months as a reference standard, FDG-PET/CT had a sensitivity of 76% (95% CI: 63%-90%) and a specificity of 100% (95% CI: 92%-100%), whereas TAB had a sensitivity of 63% (95% CI: 48%-78%) and a specificity of 100 (95% CI: 92%-100%). Interestingly, 10 of 29 patients (34%) with a positive FDG-PET/CT had a negative TAB, despite 4 of 10 having FDG-PET/CT uptake at the level of the cranial arteries. FDG-PET/CT was negative in 9 patients with a clinical picture compatible with GCA, 5 of whom had arteritis at TAB. This study had RoB with 'some concerns' because of poor comparability between the cohorts.

Outcomes of cranial vs extracranial GCA (PICO 10)

De Boysson et al [47] reported that extracranial GCA (N = 183) had more cardiovascular (49% vs 11%, $P < .0001$) and cerebrovascular events (10% vs 3%, $P = .02$) compared with cranial GCA (N = 105) (retrospective study, low RoB) (Supplementary Table S14). Extracranial disease had also more relapses (68% vs 50%, $P = .002$) and a glucocorticoid-dependent disease course (49% vs 36%, $P = .04$). Extracranial GCA experienced more frequent aortic dilatation (23% vs 4%, $P < .0001$), dissection (4% vs 0%, $P = .03$), and vascular surgery procedures (19% vs 0%, $P < .0001$).

Delaval et al [48] reported that patients with LVV diagnosed between the ages of 50 and 60 years presented less frequently with cranial symptoms (73% vs 90%, $P < .0001$) but had peripheral limb ischaemic manifestations more often (23% vs 5%, $P < .0001$) (case-control study, RoB: some concerns) (Supplementary Table S14). This younger subgroup had higher rates of extracranial vessel inflammation at FDG-PET/CT (90% vs 72%, $P = .01$) and lower frequency of positive TAB (59% vs 72%, $P = .03$). Patients aged 50 to 60 years received more lines of immunosuppressive treatment compared with the > 60 g; 60 years subgroup (median [IQR] 2 [1–5] vs 1 [1–4], $P = .006$).

Laboratory biomarkers in the diagnosis of GCA (PICO 12)

No studies were found.

TAK*Referral of suspected TAK from primary care (PICO 1), FTC for suspected TAK (PICO 2), protocolised diagnostic workup for TAK (PICO 10)*

No studies were found.

Laboratory biomarkers in the diagnosis of TAK (PICO 12)

One study on the usefulness of diagnostic serum biomarkers for TAK was found ([Supplementary Table S15](#)) [49]. Eighteen cytokines were tested to distinguish between TAK and healthy controls. Tissue inhibitor of metalloproteinase 1 was significantly higher in TAK. This work had some concerns regarding RoB, largely driven by selection bias. Nevertheless, novel diagnostic biomarkers need extensive validation in large cohorts before being widely used in clinical practice.

DISCUSSION

The present SLR has informed the development of a new set of EULAR recommendations for the management of PMR and primary LVV (under submission). The SLR identified new evidence regarding the referral for diagnosis of PMR and GCA, but not of TAK.

The SLR confirmed the pivotal role of a strategy combining vascular ultrasonography with FTC for the diagnosis of GCA. This approach reduced the time to diagnosis and the occurrence of severe complications like blindness. Imaging modalities, primarily ultrasonography and FDG-PET/CT, are increasingly replacing TAB due to their noninvasiveness, shorter times to obtain the results, comparable diagnostic accuracy, and lower costs. Unlike TAB, both ultrasonography and FDG-PET/CT detect not only cranial but also extracranial arteritis and vessel wall damage. This is reflected in both the 2018 EULAR recommendations for the management of LVV and the 2023 EULAR recommendations for the use of imaging in LVV, suggesting ultrasonography as the first-line diagnostic modality for suspected GCA [2,4]. The 2021 ACR guidelines recommend initial TAB over imaging for the diagnosis of GCA, mainly due to a higher expertise in TAB than ultrasonography in the United States [50].

Moreover, protocolised multimodal diagnostic pathways for GCA, integrating clinical, laboratory, TAB, and imaging, improved the diagnostic accuracy and minimised misdiagnosis. Several combinations proved their usefulness in clinical practice, so that pathways can be tailored according to the resources and expertise available at any centre.

The SLR identified new concepts that were not explored by the latest guidelines. In recent years, FTC has also been implemented for PMR diagnosis with excellent results, although high-quality evidence showing the superiority of FTC for PMR vs conventional pathways in terms of clinically relevant outcomes is still lacking. However, the advantages of rapid access to rheumatology specialist care must be balanced with the resources available, as PMR is much more common than GCA, and the differential diagnosis of bilateral shoulder pain is widespread. The role of the primary care physician becomes crucial in the referral of suspected PMR and LVV: patients should be easily referred by their GPs to tertiary centres, and GPs should be trained to minimise inappropriate referrals and prioritise cases of suspected GCA.

There is a growing interest in the need to recognise covert LVV in patients presenting with a PMR-like picture in the absence of cranial symptoms of GCA. The urge to treat these patients more aggressively is still under debate, as there are yet no data from high-quality clinical trials showing superiority of more aggressive treatment of covert LVV in PMR vs PMR-only in terms of clinical outcomes. Future research on the management of covert LVV and perhaps a change in the clinical practice are expected to occur in the next few years, as this entity has been increasingly recognised and studied.

This SLR had some limitations. No RCTs were retrieved, and the evidence found was too heterogeneous to perform a meta-analysis. GCA and TAK are rare conditions, where it is difficult to produce large amounts of data and plan RCTs. Despite PMR being a common condition, studies on systematic referral to a rheumatology specialist are only emerging in recent years. This SLR identified several high-quality observational studies on GCA and PMR, even though they provided a lower level of evidence compared with RCTs. We found no high-quality articles regarding the referral and diagnosis of TAK, underscoring the need for additional studies in this field. Moreover, there were several research domains that found no matching articles, such as the development of new diagnostic biomarkers, the utility of FTC and/or multimodal pathways for TAK, and the differential assessment of suspected cranial vs extracranial LVV. These unmet needs have been discussed during the task force meeting for the development of the recommendations, in the context of building a future research agenda.

In conclusion, this work, together with the SLR on treatment and management of relapses and complications (under submission), formed the basis for the new 2025 EULAR recommendations for the management of PMR and primary LVV. This SLR detected paradigm shifts potentially reshaping the diagnostic approach to PMR and LVV. For the first time, the literature draws attention to extracranial LVV and covert LVV with initial PMR-like presentations. The implementation of the FTC, combined with a sequence of diagnostic modalities in the frame of a protocolised step-by-step pathway, will enhance the diagnostic performance in clinical practice.

Competing interests

PS has received speaker fees from BMS, CSL Vifor, and AbbVie; consulting fees from AbbVie; and travel support from J&J. BH has received speaker fees from AbbVie, AstraZeneca, Boehringer, CSL Vifor, GSK, InflaRx, Janssen, MSD, Pfizer, Novartis, and Roche; honoraria for consultation services from AbbVie, AstraZeneca, Alexion, Boehringer, CSL Vifor, GSK, Pfizer, Novartis, and Sanofi; and funding for guideline development from European Alliance of Associations for Rheumatology (AAV, LVV) and DGRh (LVV). CBM contracted services and expenses from Takeda UK Ltd. EN has received speaker honoraria/participated in advisory boards for UCB, Pfizer, Gilead, Galapagos, AbbVie, Eli Lilly, Alfasigma, Fresenius, and Novartis, and received research grants from UCB, Pfizer, and Eli Lilly. All other authors declare that they have no competing interests.

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Data available on reasonable request.

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