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Background: *Large vessel vasculitis (LVV), encompassing giant cell arteritis (GCA) and Takayasu arteritis (TAK), causes granulomatous inflammation of large- and medium-sized arteries with potentially devastating ischemic complications. Glucocorticoids remain first-line therapy but carry significant long-term morbidity.*

Methods: *This narrative review synthesizes current evidence on the classification, diagnosis, monitoring, and management of LVV, incorporating data from randomized controlled trials, meta-analyses, and international guidelines.*

Results: *Advances in vascular imaging have improved diagnostic accuracy and increasingly supplement or replace temporal artery biopsy. In GCA, phase 3 trials have demonstrated that tocilizumab and upadacitinib have superior remission rates and glucocorticoid-sparing effects. In TAK, tumour necrosis factor inhibitors remain the preferred biologic for refractory disease, with emerging evidence supporting Janus kinase (JAK) inhibition and novel disease-modifying anti-rheumatic drug combinations.*

Conclusion: *Targeted therapies have transformed LVV management, though reliable biomarkers for disease activity and consensus on optimal treatment duration remain unmet needs.*

Keywords: *large vessel vasculitis, giant cell arteritis, Takayasu arteritis, tocilizumab, upadacitinib, glucocorticoid-sparing, JAK inhibitors*

Updates on the Treatment and Management of Large Vessel Vasculitis

Stephanie Garner, MD, MSc, FRCPC

Introduction

Large vessel vasculitis (LVV) is characterized by granulomatous inflammation of large- and medium-sized arteries and manifests as giant cell arteritis (GCA) and Takayasu arteritis (TAK). GCA affects individuals over the age of 50 years, and is the most common systemic vasculitis in Western populations, with an incidence of approximately 25 cases per 100,000 persons aged ≥50.[1] TAK predominantly affects young women, often of Asian or Latin American descent, with a global incidence of approximately 1.1 per million per year (95% confidence interval [CI] 0.70–1.76), though prevalence is substantially higher in Asian populations (up to 40 per million in Japan).[2]

Although GCA and TAK share a common basis of granulomatous arterial inflammation, they differ in genetic susceptibility, pathogenesis, and vascular manifestations.[3,4] Resultant vascular injury can lead to stenosis, aneurysm formation, and ischemic complications.[5] In GCA, prompt diagnosis and treatment are essential to prevent irreversible vision loss and stroke.[6] Glucocorticoids have long been the cornerstone of therapy for both conditions, but long-term use carries substantial adverse effects, including osteoporosis, diabetes, cataracts, cardiovascular disease, and weight gain.[7,8]

Over the past decade, management strategies have increasingly shifted toward glucocorticoid-sparing regimens, driven by advances in understanding disease pathogenesis, the availability of targeted biologics, and growing recognition of the harm caused by corticosteroids.

This review offers a clinically relevant, evidence-based update on LVV management, diagnostics, disease monitoring, pharmacological advances, and future therapeutic directions.

Classification and Pathophysiology

The American College of Rheumatology (ACR)/European Alliance of Associations for Rheumatology (EULAR) 2022 classification criteria for GCA and TAK incorporate modern imaging modalities (positron emission tomography [PET], ultrasound), weighted scoring systems, and revised age thresholds.[9,10] For GCA, the updated criteria markedly increase the sensitivity for extracranial disease from 24% to 92%.[11] In TAK, the age threshold was raised to ≤60 years, acknowledging that onset after age 40 occurs in 9–32% of patients.[10] Imaging evidence of large vessel vasculitis is now a mandatory entry requirement, along with patterns of arterial involvement.[10,12] Although not intended for diagnosis, accurate classification remains clinically important because treatment protocols and prognoses differ.

The pathophysiological differences between GCA and TAK have direct therapeutic implications. In GCA, the human leukocyte antigen (HLA) class II/CD4+ T-cell/IL-6 axis underlies the efficacy of tocilizumab and Janus kinase (JAK) inhibitors, while in TAK, the HLA class I/CD8+ T-cell/tumour necrosis factor (TNF)-α axis in TAK explains the clinical effectiveness of TNF inhibitors, which have not demonstrated similar benefit in GCA.[3,5]

Diagnosis and Disease Assessment

Clinical Presentation

GCA classically presents with new-onset headache, scalp tenderness, jaw claudication, and visual symptoms in patients aged over 50 years.[5,13] Visual complications are broad and can include arteritic anterior ischemic optic neuropathy, amaurosis fugax, and diplopia.[14] Early ophthalmology involvement is critical. Systemic features (fever, weight loss) are common, and polymyalgia rheumatica co-occurs in 40–60% of cases.[15] Jaw claudication has the highest positive likelihood ratio (+LR 4.90) for predicting GCA, though diagnostic delay is frequent given the often non-specific symptom profile.[16]

In contrast, TAK presents insidiously, with constitutional symptoms, limb claudication, asymmetric blood pressures, diminished pulses, bruits, and hypertension.[5,12] Diagnostic delays are common, particularly given the younger demographic, and the disease is often discovered incidentally on imaging.

Biomarkers and Laboratory Assessment

C-reactive protein (CRP) and erythrocyte sedimentation rate are standard surrogate markers for disease activity in LVV. A normal CRP provides the strongest negative likelihood ratio (−LR 0.40; 95% CI 0.29–0.56) for excluding GC.[16] In TAK, however, active disease can occur despite normal inflammatory markers. Disease activity determined solely by clinical assessment and acute-phase reactants correlates poorly with histopathologic activity, and new arterial lesions have been observed in 61% of patients considered clinically inactive.[12,17] Notably, CRP is unreliable for monitoring in patients receiving interleukin (IL)-6 inhibitors, as CRP production is directly IL-6–dependent.

Temporal Artery Biopsy

Temporal artery biopsy (TAB) has a sensitivity of approximately 85–95% in clinically suspected GCA, though diagnostic yield is diminished by short segments, skip lesions, and extracranial disease.[6] Contrary to traditional teaching, the 2-week window for biopsy following steroid initiation is not supported by evidence. A prospective study of follow-up biopsies in treated patients demonstrated unequivocal vasculitis in 70% at 3 months, 75% at 6 months, and 44% at 12 months after treatment initiation,

with lymphocytic infiltrates persisting even as granulomatous features diminished over time.[18] Additional studies have shown that biopsies are consistently positive for at least 6 weeks after treatment initiation.[19] Glucocorticoids should never be delayed while awaiting biopsy. Although imaging has an increasing role in diagnosis, biopsy-confirmed GCA has been associated with higher rates of visual loss (9.7% vs 2.4%).[20]

Imaging in LVV

Imaging has increasingly replaced TAB and is recommended over other imaging modalities for diagnosing GCA.[21] Ultrasound of the temporal and axillary arteries demonstrating the "halo sign" (hypoechoic mural edema) achieves sensitivity and specificity comparable to biopsy.[21] "Fast Track" ultrasound clinics using this approach have been implemented in several centres to reduce diagnostic delay and support point-of-care diagnostics.[22]

PET offers excellent sensitivity for large vessel involvement in both GCA and TAK and is increasingly used for diagnosis and monitoring.[21] Magnetic resonance imaging/magnetic resonance angiography (MRI/MRA) is preferred for aortic and branch-vessel assessment in TAK, given its spatial resolution and avoidance of ionizing radiation in a younger population. computed tomography angiography (CTA) remains valuable for emergency evaluation and structural vascular complications. Temporal artery MRA (TAMRA) shows promise, particularly as a rule-out test, but uptake is limited by the availability of 3T MRA and radiologist expertise.[21]

Currently, no standardized guidelines exist for the frequency of monitoring using imaging in these populations; therefore, decisions should be made on an individual basis.

Investigative Algorithms

Given the heterogeneous presentation of GCA, pre-test probability tools have been developed to improve diagnostic accuracy. The halo count-GCA (HAS-GCA) algorithm combines the giant cell arteritis probability score (GCAPS) clinical score with temporal artery ultrasound (TAUS) to classify 74% of patients as either low or high probability, thereby reducing the need for additional testing (**Figure 1**).[23]

Treatment of Giant Cell Arteritis

Glucocorticoids

High-dose glucocorticoids remain the first-line treatment for GCA: oral prednisone 40–60 mg/day without visual involvement, or intravenous methylprednisolone (500–1000 mg/day for 3 days) when visual loss or ischemic complications are present, followed by oral therapy.[6] Traditional tapering over 12–18 months carries high relapse rates (40–50%), and cumulative steroid exposure drives significant morbidity.[24,25] Vision loss during treatment is uncommon (2.2%); new visual symptoms should prompt a broad evaluation, including consideration of corticosteroid-related causes such as cataracts and glaucoma.[26]

Steroid-Sparing Agents

Steroid-sparing therapies are now considered the standard of care for patients with GCA, with current treatment options outlined in **Table 1**. The ACR guidelines suggest starting steroid-sparing therapies in all patients.[6]

Other targeted therapies remain under investigation. Abatacept demonstrated improved relapse-free survival in a small, randomized trial of GCA, supporting the role of T-cell co-stimulation in disease pathogenesis, although evidence remains limited compared with that for IL-6 and Janus kinase (JAK) inhibition.[27]

Data from the GiACTA and SELECT-GCA extension studies support the concept that GCA requires prolonged immunosuppression (prednisone, steroid-sparing agents) in many patients.[28,29] Current guidelines recommend individualized decision-making regarding treatment duration, with some patients discontinuing after 1–2 years while others require indefinite therapy to prevent relapse.[6]

Treatment of Takayasu Arteritis

Conventional Immunosuppression

Glucocorticoids (1 mg/kg/day; maximum 60 mg/day) remain the cornerstone of initial treatment for TAK; however, relapse rates of 50–80% during dose tapering necessitate the early addition of a steroid-sparing agent.[6,30]

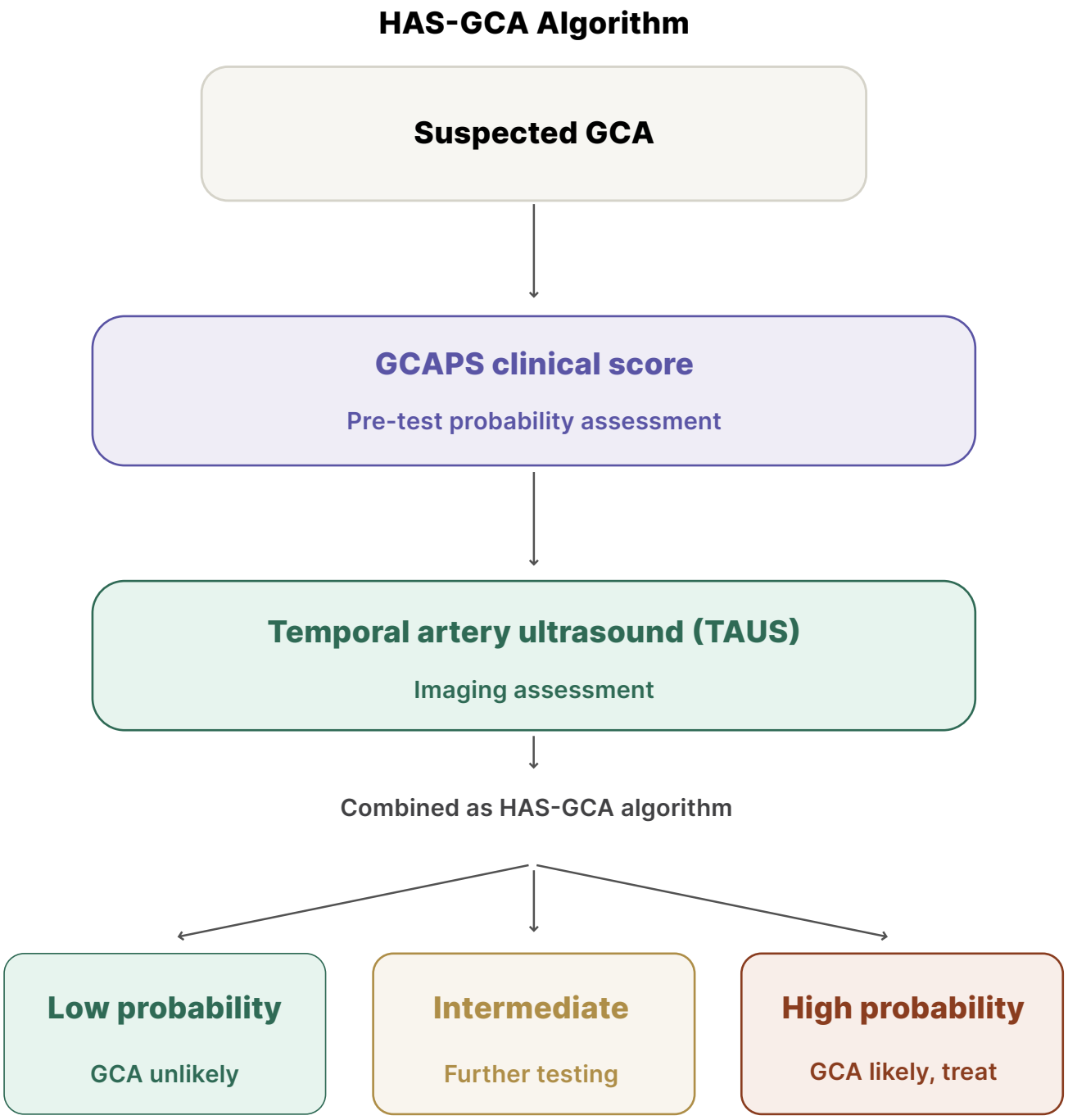

Figure 1. HAS-GCA Algorithm; *adapted from Sebastian, 2024.*[23]

Abbreviations: GCA: Giant Cell Arteritis; **GCAPS:** Giant Cell Arteritis Probability Score

Feature	Tocilizumab	Upadacitinib	Methotrexate	Leflunomide
Drug class	Anti-IL-6R monoclonal antibody	Selective JAK inhibitor	Conventional DMARD (antimetabolite)	Conventional DMARD (pyrimidine synthesis inhibitor)
Route	SC (162 mg weekly)	Oral (15 mg daily)	Oral/SC (10–25 mg weekly)	Oral (10–20 mg daily)
Landmark trial	GiACTA (phase 3, n=251)	SELECT-GCA (phase 3, n=428)	3 RCTs (n=21–98); IPD meta-analysis	Observational studies only
Sustained remission at 52 weeks	56% vs 14% placebo (P=0.001)[28]	46.4% vs 29.0% placebo (P=0.002)[29]	Modest relapse reduction (HR 0.65, P=0.04)[38]	~60% achieved at least partial remission (meta-analysis of 7 studies)[11]
GC-free remission	42% maintained drug-free remission over a 2-year extension	50.2% vs 19.6% at week 52 (P0.001)[29]	HR 2.8 vs placebo (P=0.001)[38]	53% discontinued GCs entirely[11]
Median cumulative GC dose (52 weeks)	1,862 mg vs 3,296 mg placebo[28]	1,615 mg vs 2,882 mg placebo[29]	−1.1 g reduction at 96 weeks (20–44% reduction)[38]	Mean reduction of 15.6 mg/day[11]
GC taper in trial	26-week taper[28]	26-week taper[29]	Variable (concurrent with GCs)[38]	Variable[39]
Key safety concerns	Infections, GI perforation, hyperlipidemia; masks CRP[40]	Herpes zoster, theoretical MACE/VTE (none observed in trial)[29,37]	Hepatotoxicity, cytopenias, nausea,	GI symptoms, hepatotoxicity, 19–25% discontinuation rate[11]
CRP monitoring reliability	Unreliable (IL-6 pathway suppressed)	Partially suppressed but more reliable than TCZ[37]	Reliable	Reliable
Level of evidence	High (phase 3 RCT + 3-year extension)[28,36]	High (phase 3 RCT + extension ongoing)[29,37]	Moderate (3 small RCTs with mixed results)[38]	Low (observational only; phase 3 RCT recruiting)[41]
Guideline positioning	First-line steroid-sparing agent[6]	Alternative to TCZ	Option if biologics inaccessible/contraindicated	Alternative if MTX is not tolerated

Table 1. GCA Treatment Options; *courtesy of Stephanie Garner, MD, MSc, FRCPC.*

Abbreviations: CRP: c-reactive protein; **DMARD:** disease-modifying anti-rheumatic drugs; **GC:** glucocorticoid; **GCA:** giant cell arteritis; **GI:** gastrointestinal; **HR:** hazard ratio; **IL:** interleukin; **IPD**: individual participant data; **JAK:** Janus kinase; **MACE:** major adverse cardiac events; **MTX:** methotrexate; **RCT:** randomized controlled trial; **SC:** subcutaneous; **TCZ:** tocilizumab; **VTE:** venous thromboembolism

Conventional disease-modifying anti-rheumatic drugs (DMARDs) remain first-line, with methotrexate most frequently used.[6] In a recent randomized trial (n=111), mycophenolate mofetil plus methotrexate was superior to cyclophosphamide/azathioprine (overall response 55% vs 32% at 52 weeks, P=0.022), providing the first high-quality evidence favouring a specific conventional DMARD combination in TAK.[31]

Biologic DMARDs are recommended for refractory or severe disease. TNF inhibitors have the strongest supporting evidence and are preferred in the 2021 ACR guidelines[6], with a large multicenter study (n=209) demonstrating 3-year relapse-free survival of 90.9% versus 58.7% with conventional DMARDs.[32] Tocilizumab showed benefit in the TAKT trial extension despite failing its primary endpoint, and a 2023 meta-analysis reported comparable efficacy between TNF inhibitors and tocilizumab.30 JAK inhibitors are an emerging option, though long-term data remain limited.[5,30,33]

Vascular Interventions

Surgical and endovascular interventions, including angioplasty, stenting, and bypass grafting, may be required for hemodynamically significant stenoses or occlusions in TAK. These procedures should be performed during disease remission, when possible, to reduce the risk of restenosis, with perioperative glucocorticoid coverage and postoperative surveillance.

Monitoring of Disease Activity

In GCA, ongoing imaging monitoring is indicated primarily for those with documented large-vessel disease to identify stenoses, aneurysms, or structural progression.[6] FDG-PET can assess inflammatory burden and identify large vessel involvement; however, findings are not specific to active inflammation and require clinical correlation.[6,34] The 2026 French recommendations propose Doppler ultrasound as the first-line imaging modality for suspected relapse and structural monitoring, with FDG-PET, MRI, and CT as alternative options.[34]

A caveat to this is that monitoring disease activity during tocilizumab therapy is challenging because CRP, no longer a reliable marker, and PET findings often show clinical-radiographic dissociation. Complete PET normalization occurs in only a minority of patients, even in clinical remission.[35] Treatment decisions should therefore be guided by clinical assessment and structural imaging rather than isolated PET findings in asymptomatic patients.

For TAK, regularly scheduled non-invasive imaging (MRA, CTA, or FDG-PET) is conditionally recommended in addition to clinical assessment, as vascular changes can occur during clinically quiescent disease.[6] Follow-up imaging frequency ranges from every 6 to 24 months, depending on disease activity, with shorter intervals early in the disease course.[5]

Special Considerations

Glucocorticoid Toxicity Mitigation

All patients initiated on glucocorticoid therapy for LVV should receive concurrent prophylaxis for glucocorticoid-induced osteoporosis, along with blood pressure monitoring, glycemic surveillance, and ophthalmological follow-up. Lipid profiles, bone mineral density assessment, and vaccinations (pneumococcal, influenza, and herpes zoster) should be addressed at disease onset.

Pregnancy and LVV

TAK disproportionately affects women of reproductive age, and pregnancy in the setting of active or recently active vasculitis carries elevated maternal and fetal risks, including hypertension, preterm delivery, and small-for-gestational-age infants. Close collaboration among rheumatology, obstetric medicine, and maternal-fetal medicine is essential.

Conclusion and Future Directions

The management of LVV has evolved substantially with advances in imaging, disease classification, and targeted therapies. In GCA, IL-6 and JAK inhibition have significantly reduced glucocorticoid exposure while improving disease control. In TAK, biologic therapies, particularly TNF inhibitors, have expanded treatment options for patients with refractory disease.

Despite these advances, important challenges remain, including the lack of reliable biomarkers for disease activity, uncertainty regarding optimal treatment duration, and the need for better decision-making tools to monitor vascular inflammation and damage. Future research will focus on precision medicine approaches, novel targeted therapies, and improving long-term outcomes while minimizing treatment-related toxicity. Continued advances in

immunopathogenesis and imaging are expected to further refine the diagnosis and management of both GCA and TAK.

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