Giant cell arteritis: advances in diagnosis and management

ABSTRACT

Giant cell arteritis has been widely studied throughout the world. Involvement of cranial vessels can lead to visual loss and strokes. This review primarily focusses on the presentation, diagnosis and treatment. The last 10 years have brought dramatic improvements in the imaging and medical therapies for this condition.

After the American College of Rheumatology suggested criteria for the diagnosis of giant cell arteritis, many studies have been performed to find alternatives to a temporal artery biopsy. There is growing evidence that a biopsy may not be needed when one can make a convincing clinical and radiological diagnosis.

Although glucocorticoids are the mainstay of treatment and their role has not changed, various biological and non-biological therapies are being used to reduce relapses and prolong remission of symptoms.

In the 19th century, Jonathan Hutchinson described a man who had difficulty wearing a hat because of his tender temporal area. Later giant cell arteritis was found to be accompanied by a spectrum of symptoms and signs of tender temporal area, fever, headache, malaise, visual problems and raised levels of inflammatory markers.

Giant cell arteritis is a chronic granulomatous inflammation of medium- to large-sized arteries involving the aorta, proximal upper limb, the neck and extracranial arteries. It is called temporal arteritis if this inflammation involves extracranial arteries, in particular those in the temporal region. The main concern is the occurrence of irreversible blindness from the occlusion of arteries supplying the optic nerve head (Schmidt and Ness, 2009). Steroid therapy is started immediately in most individuals with a clinical suspicion of temporal arteritis. Symptoms and signs of temporal arteritis are throbbing unilateral headaches, scalp sensitivity and tenderness to touch, visual symptoms (loss of vision, blurred vision, diplopia and amaurosis fugax) and jaw claudication. Once visual loss occurs, it is mostly irreversible and the aim of treatment is to reduce the inflammatory response and progression of damage, and to protect the contralateral eye.

The posterior ciliary arteries are the most common branches of the ophthalmic arteries involved. These supply the optic nerve head (*Figure 1*) and their occlusion can lead to acute anterior ischaemic optic neuritis.

A host of generalised symptoms and signs can be observed depending on the involvement of the arteries.

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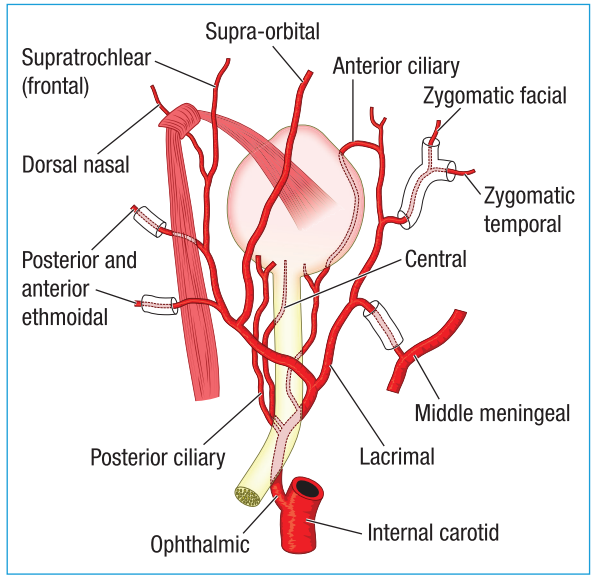
Incidence and epidemiology

Giant cell arteritis is more common in females. The incidence is 24.4/100 000 population in women and 10.3/100 000 population in men (Salvarani et al, 2004), and the mean age at diagnosis is 74.8 years (Gonzalez-Gay et al, 2010). The incidence is highest in people between 70 and 80 years of age. It rarely affects younger people.

Case series suggest that the disease affects certain families. Giant cell arteritis is more common in north European (Scandinavian) countries (Gonzalez-Gay et al, 2010). Figures from the UK suggest that the age-adjusted incidence of giant cell arteritis was 2.2/10 000 person-years from 1990 to 2001 (Smeeth et al, 2006). Both polymyalgia rheumatica and giant cell arteritis were seen more commonly in Caucasians originating in the south of the UK. Giant cell arteritis was more frequently

Figure 1. Ophthalmic arteries.



diagnosed in the summer times and in the southern counties.

Investigations and diagnosis

Giant cell arteritis can be diagnosed clinically with classical signs, symptoms and a raised erythrocyte sedimentation rate (*Table 1*). temporal artery biopsy is pivotal in confirming the diagnosis in cases where there is clinical uncertainty. For a long time temporal artery biopsy was considered the gold standard even in individuals with a classical presentation, sometimes leading to a delay in glucocorticoid administration. Imaging is important in diagnosis of large vessel and cranial giant cell arteritis (Gonzalez-Gay et al, 2010).

The American College of Rheumatology criteria

In 1990, after comparing 214 individuals with giant cell arteritis and 593 patients with other forms of vasculitis, the American College of Rheumatology identified five criteria (Hunder et al, 1990) to diagnose giant cell arteritis which are still applicable in a clinical setting:

1. Age ⩾50 years at disease onset
2. New headache localized to the temporal areas
3. Temporal artery tenderness or decreased temporal artery pulse
4. Elevated erythrocyte sedimentation rate 50 mm/hour
5. Biopsy sample including an artery showing necrotizing arteritis, classically with giant cells and inflammatory infiltrate.

The presence of three of the five criteria was considered adequate to make a clinical diagnosis of giant cell arteritis with a sensitivity of 93.5% and a specificity of 91.2% (Hunder et al, 1990). The differential diagnosis is outlined in *Table 2*.

Temporal artery biopsy

Temporal artery biopsy should ideally be requested in cases where there is doubt about the clinical diagnosis. Timing of surgery after clinical suspicion and the technical aspects of biopsy sampling can cause difficulty in prompt and accurate diagnosis in cases that do not fit the American College of Rheumatology criteria. Some district hospitals perform duplex ultrasound in clinic to help confirm the diagnosis. How duplex ultrasound affects the management depends on resources and clinical expertise.

Duplex ultrasound

The role of duplex ultrasound in diagnosing cranial or temporal arteritis has been widely studied. The British Society of Rheumatology (Dasgupta et al, 2010) and French Study Group for Large Vessel Vasculitis (Bienvenu et al, 2016) guidance suggests a limited role of duplex ultrasound as a first-line investigation for giant cell arteritis. Duplex ultrasound is operator dependent, relying on experience and the ability to identify hallmarks of arteritis. Guidance is based on studies that had heterogenous results several years ago (Dasgupta et al, 2010; Bienvenu et al, 2016).

In a meta-analysis of eight studies in 2010, duplex ultrasound findings were observed in individuals who had a positive temporal artery biopsy to try and define specific duplex ultrasound signs to help in the diagnosis. A 'halo' sign had a sensitivity of 68% and a specificity of 91% (Arida et al, 2010). Having a halo sign in several branches seems to carry a higher likelihood of a diagnosis of temporal arteritis in individuals with clinical evidence of giant cell arteritis.

In the TABUL study (temporal artery biopsy *vs* ultrasound), a prospective study of 301 patients, an ultrasound and biopsy were carried out within 7 days of definitive or probable diagnosis of giant cell arteritis

Table 1. Symptoms and signs of giant cell arteritis

Constitutional symptoms	Non-specific symptoms
	Fever
	Malaise
	Night sweats
	Weight loss
Myalgia and muscle stiffness	Proximal muscles (shoulder and pelvic girdle)
	Tender muscles and joints
	Contractures and atrophy of muscles
Joint symptoms	Tender joints (shoulder and hip joints).
	Synovitis (knees, shoulders and wrists)
Vasculitis involving branches of the external carotid artery	Headache and scalp pain
	Difficulty in combing hair
	Pain behind the ear
	Jaw pain and claudication
	Pain in jaw muscles and tongue on chewing and talking
Vasculitis involving branches of the ophthalmic artery	Decreased vision
	Amaurosis fugax
	Diplopia and ptosis
Vasculitis of larger arteries 9–18%	Thoracic aneurysms
	Arm or lower limb claudication
	Subclavian steal syndrome
	Aortic aneurysms
	Intestinal infarction
Polymyalgia rheumatica	40% of patients with giant cell arteritis have polymyalgia rheumatica
	Aches in shoulder and hip girdles
	Morning stiffness
	Intense fatigue

Table 2. Differential diagnosis of giant cell arteritis

Cranial large vessel vasculitis	Arterial	Takayasu's arteritis
		Atherosclerosis (carotid stenosis)
		Polyarteritis nodosa
	Neurological	Migraine
		Tension headache
		Trigeminal neuralgia
		Intracranial pathology
		Ear, nose and throat disease
	Musculoskeletal	Cervical spondylosis
		Temporomandibular disorders
		Polymyositis
	Rheumatic	Rheumatoid arthritis
		Seronegative rheumatoid arthritis
	Infections	Herpes zoster
		Systemic infections
	Neoplastic	
Large vessel vasculitis	Arterial	Takayasu's arteritis
		Coarctation of aorta
		Atherosclerosis
		Buerger's disease
		Kawasaki's disease
	Rheumatic	Rheumatoid arthritis
		Sarcoidosis
		Granulomatosis with polyangiitis
		Systemic lupus erythematosus
		Idiopathic aortitis
		IgG4 related disease
		ANCA-associated vasculitis
		Behçet's syndrome
	Infections	Herpes zoster
		Syphilis
		Epstein–Barr virus
		Cytomegalovirus
	Rheumatic fever	

and glucocorticoids were commenced immediately. The halo sign reduced in size until day 4 with glucocorticoid treatment, and reduction in oedema correlated with ischaemic cranial symptoms and also the response to treatment (Serafim et al, 2014). The halo appears darker (hypoechoic) as a result of oedema of the arterial wall. It has been suggested that temporal artery biopsy should only performed in those where clinical suspicion is low with a lack of signs on duplex ultrasound (Luqmani et al, 2016).

The inability to standardize the duplex ultrasound findings for diagnosing giant cell arteritis prompted the Outcome Measures in Rheumatology (OMERACT) group to provide a consensus expert opinion to standardize the duplex ultrasound findings into 'halo', 'compression', 'obstruction' and 'stenosis' of the arteries. The aim was also to assist in future research with clear definitions of positive findings in giant cell arteritis (Chrysidis et al, 2018).

The European League Against Rheumatism consensus statement for the diagnosis of giant cell arteritis recommends that a classical history, examination and positive finding on duplex ultrasound should be sufficient to confidently diagnose giant cell arteritis. A 'non-compressible halo sign' was the finding most suggestive of giant cell arteritis. A halo on duplex ultrasound was defined as: 'homogenous, hypoechoic wall thickening that is well delineated towards the luminal side that is visible both in longitudinal and transverse planes, most commonly appearing concentric in transverse scans.' Despite this, temporal artery biopsy is still the gold standard if adequate imaging is not available in cranial giant cell arteritis (Dejaco et al, 2018).

High resolution magnetic resonance imaging

High resolution magnetic resonance imaging has been investigated as the first-line imaging modality and as an alternative to temporal artery biopsy. Sensitivity of identifying signs of giant cell arteritis of above 80% can be achieved in patients with a positive temporal artery biopsy. A prospective study of 171 patients having magnetic resonance imaging and a concomitant temporal artery biopsy found a higher sensitivity of magnetic resonance imaging in diagnosing giant cell arteritis in individuals with a positive temporal artery biopsy. This was particularly advantageous in diagnosing intramural thickening of medium to large arteries. Magnetic resonance imaging was recommended as a first-line modality, with temporal artery biopsy to be requested in cases with positive findings (Rhéaume et al, 2017).

Computed tomography angiography

Computed tomography angiography has been used to examine large vessels in individuals with giant cell arteritis, not only to identify any concomitant aortitis but also for individuals not having the classical cranial giant cell arteritis signs and symptoms. Aortitis can be diagnosed in 45–65% of individuals with giant cell arteritis on treatment. Owing to the frequency of large vessel involvement, imaging of the thoracic aorta has

been incorporated into the guidelines for treating patients with giant cell arteritis. This is particularly important in diagnosing other conditions like Takayasu's arteritis (Hiratzka et al, 2010).

18 fluorodeoxyglucose positron emission tomography

A meta-analysis concluded that arterial uptake of fluorodeoxyglucose equal to or greater than uptake by the liver in the presence of giant cell arteritis is the best criterion to detect large vessel inflammation compared to controls, with a pooled sensitivity of 90% (Soussan et al, 2015). There is currently no precise cut off to accurately diagnose aortitis in isolation although it can be helpful in ruling out aortic dilatation and other complications of giant cell arteritis.

Treatment

Giant cell arteritis requires immediate treatment, prolonging remission of the disease and preventing relapse. Immediate treatment aims to control the systemic inflammatory response and avoid complications like acute visual loss. Corticosteroids remain the mainstay of acute treatment for giant cell arteritis. The diagnosis should not be deferred until the results of an erythrocyte sedimentation rate or temporal artery biopsy if clinical assessment and duplex ultrasound findings indicate giant cell arteritis.

Management of uncomplicated giant cell arteritis

Figure 2 shows a management algorithm for patients with giant cell arteritis. Over the last 2–3 years biological and non-biological agents have shown promise in the management of polymyalgia rheumatica and giant cell arteritis but the role of glucocorticoids in immediate management remains unchanged. Glucocorticoids should be started immediately on the clinical suspicion of giant cell arteritis. Steroid therapy can continue for years and tapering the dose to reduce complications needs to be balanced with the risk of relapse of the disease. This has led to more research on the patterns of relapse of giant cell arteritis and various ways of reducing or tapering the optimum dose of glucocorticoids.

The British Society of Rheumatology and British Health Professionals in Rheumatology published guidelines for the management of giant cell arteritis recommending a dose of 60–80 mg/day of prednisone. An immediate improvement in clinical signs and symptoms should be observed with a reduction in inflammatory response (Dasgupta et al, 2010).

Similarly, European League Against Rheumatism recommends 1 mg/kg/day of glucocorticoids with a maximum of 60 mg/day (Mukhtyar et al, 2009).

A systematic review looked at pulsed intravenous hydrocortisone for the first 3 days in addition to high dose oral methylprednisone 40–60 mg per day from two randomized controlled trials. It reported no advantage in the use of pulsed intravenous glucocorticoid therapy in addition to oral glucocorticoids for uncomplicated giant cell arteritis (Buttgereit et al, 2016).

> "Steroid therapy can continue for years and tapering the dose to reduce complications needs to be balanced with the risk of relapse of the disease."

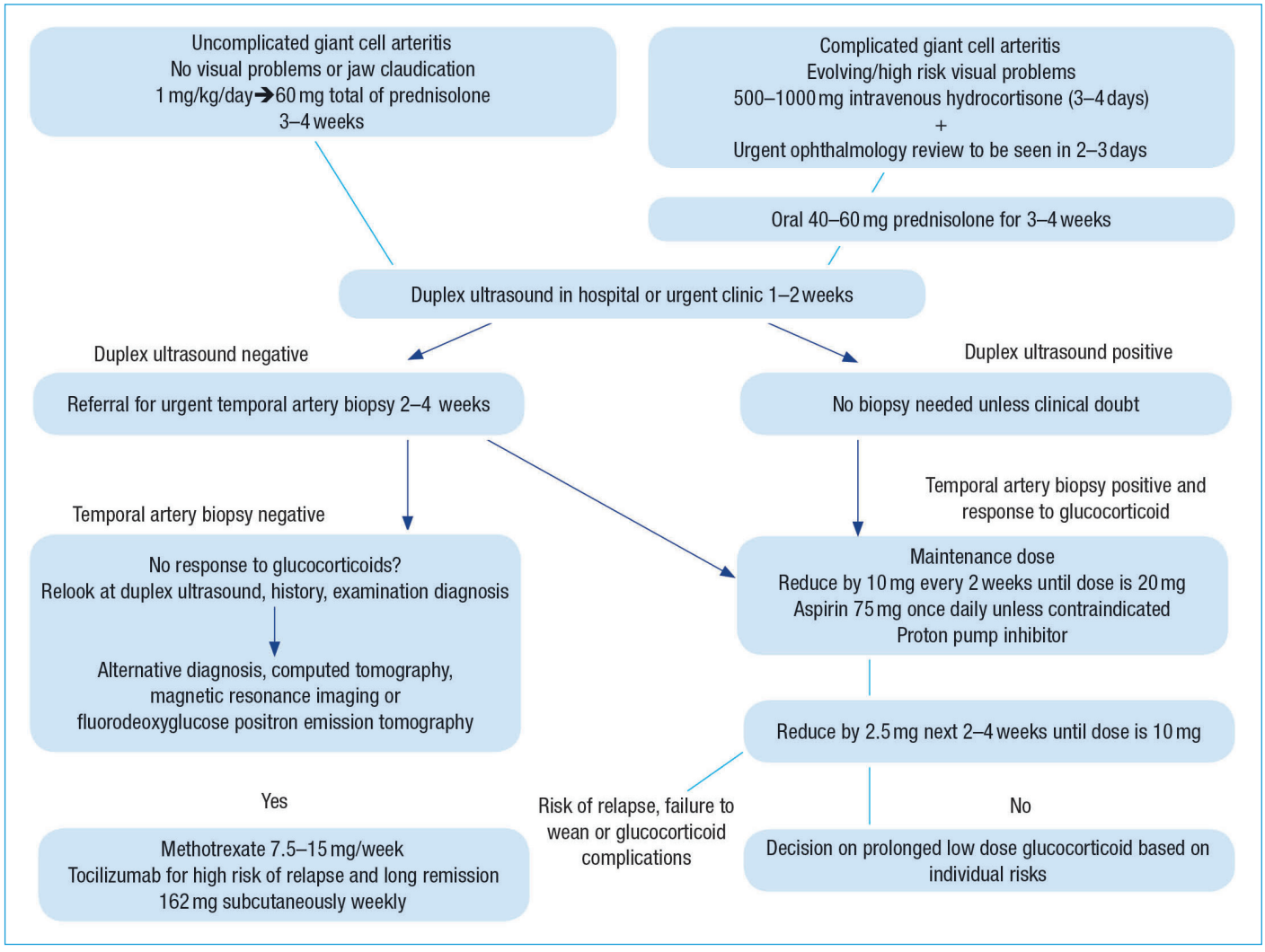
More than 50% of flares occur when steroids are tapered down after initial induction of therapy. In addition, most relapses in patients with giant cell arteritis occur in the first 6 months of therapy. One randomized controlled trial assessed the use of initial high-dose intravenous induction glucocorticoid therapy for 3 days and oral 40 mg methylprednisolone with the intention to rapidly taper oral glucocorticoid doses and to sustain remission. (Remission is defined as the absence of clinical symptoms and normal values for erythrocyte sedimentation rate and C-reactive protein.) More individuals in the intervention group were able to maintain remission with less than 5 mg/day at 36 weeks and even up to 72-week follow up. The treatment arm only had 14 patients but 10 achieved rapid tapering of oral glucocorticoids. They also achieved a longer sustained remission. Pulsed therapy is currently not recommended in the UK for uncomplicated giant cell arteritis (Mazlumzadeh et al, 2006).

Management of complicated giant cell arteritis

In clinical practice individuals do not always present with the classical signs and symptoms of complicated giant cell arteritis or temporal arteritis. In the authors' experience, referrals for temporal artery biopsy in those suspicious of ischaemic ophthalmological or intracranial signs and symptoms can vary from 'blurring of vision', 'diplopia', 'visual impairment', or visual loss to classical signs of acute anterior ischaemic optic neuritis or amaurosis fugax. Some individuals have chronic visual changes as a result of type 2 diabetes making it difficult not only to diagnose but also to treat with high dose glucocorticoids.

Individuals with complicated giant cell arteritis and diagnosed ophthalmological or intracranial findings should be given intravenous steroids (hydrocortisone 500–1000 mg/day) if features of impending cranial or optic ischaemia are identified. A management pathway should include urgent ophthalmological review. Signs of impending cranial and optic ischaemia include amaurosis fugax, evolving visual loss and jaw claudication. The aim is to prevent progression and control the inflammatory process by aggressive immunosuppression via intravenous glucocorticoids. These individuals need to be admitted to an acute hospital bed to monitor their clinical status. An urgent ophthalmological review is needed

Figure 2. Suggested algorithm for the acute management of giant cell arteritis.



to investigate any evolving ischaemic event. Judicious use of steroids is warranted and only patients with clear clinical signs of evolving visual problems should be given high-dose intravenous steroids. High-dose oral glucocorticoids up to 60 mg/day should be prescribed to individuals with established visual loss to protect the contralateral eye (Dasgupta et al, 2010; Hayreh and Biousse, 2012).

The consequences of visual loss can be irreversible if diagnosis and treatment are delayed. European League Against Rheumatism guidelines on the diagnosis and management of giant cell arteritis suggest, controversially, that high-risk patients should be diagnosed with imaging in preference to a temporal artery biopsy. This has not been the practice previously but might help expedite urgent treatment with glucocorticoids without waiting for an emergency temporal artery biopsy (Dejaco et al, 2018).

Outcomes of urgent glucocorticoid therapy vary but a high erythrocyte sedimentation rate, headaches, temporal inflammation and jaw claudication may resolve in several days. If high-dose steroids are started within 72 hours of visual symptoms, partial improvement of visual function can be expected (Ness et al, 2013).

Maintenance therapy and prevention of relapse

Glucocorticoid doses and duration of therapy are based on consensus guidelines because of the lack of statistically significant trials, heterogeneous individual presentation and the need to individualize treatment for each patient (Dasgupta, 2010; Dejaco et al, 2015). The British Health Professionals in Rheumatology and British Society of Rheumatology advises tapering glucocorticoids in the absence of clinical symptoms and signs, and with normal levels of serum inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein. 'The lowest effective dose', patient choice and side-effect profile should be considered when tapering the glucocorticoid dose. Prescribing 40–60 mg of glucocorticoids is advised for a minimum of 3–4 weeks and then reducing this by 10 mg every 2 weeks to 20 mg. Following this a reduction of 2.5 mg is advised for the next 2–4 weeks until a 10 mg

dose is reached. The eventual dose depends on the risk of relapse (Dasgupta et al, 2010).

Disease relapse is defined as recurrence of symptoms or worsening of symptoms after immediate treatment. *Table 3* suggests options for patient care and management of relapse. The signs and symptoms can be constitutional symptoms, polymyalgia rheumatica-like symptoms and giant cell arteritis with or without cranial (headache, cerebrovascular ischaemia, temporal tenderness, jaw claudication and visual impairment) or extracranial aortic branch involvement. Most relapses present with symptoms similar to the first episode. Pulsed intravenous glucocorticoid therapy allows rapid tapering of oral glucocorticoids and maintenance of remission (Mazlumzadeh et al, 2006).

In a study from Spain, a standard protocol of glucocorticoid therapy and tapering doses was used and intravenous 1 g glucocorticoid used for those with evolving visual symptoms. Most of those who relapsed did so within 2 years and mostly with the same symptoms as their initial presentation. The cumulative glucocorticoid dose was higher in those relapsing with symptoms, as was the risk of osteoporosis (Alba et al, 2014).

Relapse can be difficult to diagnose because symptoms might not be associated with a raised erythrocyte sedimentation rate or C-reactive protein level which can make the diagnosis difficult for clinicians (Kermani et al, 2015).

High risk of relapse seems to be more frequent in women with a history of hypertension and diabetes mellitus. High-dose immediate glucocorticoid therapy (>40 mg) is also thought to reduce the risk of relapse (Labarca et al, 2016).

Regular clinic follow up and review of glucocorticoid treatment is important to prevent relapse.

Table 3. After care and management of relapse

Rheumatology clinic review for temporal arteritis, discharge if negative	
Temporal arteritis screened for hyperlipidaemia or hypertension	
Bone protection or proton pump inhibitor	
Screen for proximal vessel involvement if systemic symptoms continue with glucocorticoids	
Monitoring of therapy and relapse	Clinical assessment and inflammatory markers
	Glucocorticoid complications assessment (diabetes mellitus, osteoporosis, renal problems, obesity, menstrual problems etc.)
	Giant cell arteritis complications: visual or jaw claudication, polymyalgia rheumatica, osteoporosis, vascular bruit, claudication of limbs
	Other symptoms suggest other diagnosis
	Each visit erythrocyte sedimentation rate and C-reactive protein level, renal function tests, glucose
	2 yearly chest X-ray, computed tomography or magnetic resonance imaging
	Bone mineral density assessment
Management of relapse	Return of symptoms, ischaemic complications, unexplained fever and polymyalgia rheumatica (not all have raised erythrocyte sedimentation rate or C-reactive protein level)
	Return of headache needs higher glucocorticoid dose
	Symptoms of large vessel disease computed tomography or magnetic resonance imaging
Recurrent relapse	Early introduction of methotrexate or other immunosuppressants should be considered
	Tocilizumab with a tapering dose 162mg subcutaneous every week for up to 1year and no more (NHS)

Biological and non-biological agents

Methotrexate

Immunosuppressive agents and methotrexate are recommended for the treatment of relapsing giant cell arteritis in the UK or in individuals who cannot be weaned to a reducing dose of glucocorticoid (Dasgupta et al, 2010).

The joint European League Against Rheumatism and American College of Rheumatology recommendations give advice on the early introduction of methotrexate in the following conditions: high risk of disease relapse, need for prolonged glucocorticoid therapy, individuals with significant comorbidities and those on medications where glucocorticoid side effects are more likely to occur. This includes relapses seen on follow up despite adequately initiating glucocorticoids (Dejaco et al, 2015).

A systematic review showed that addition of methotrexate reduced the risk of first relapse by 35%, and second relapse by 51%. The review included 161 patients of which 84 received methotrexate and 77 received placebo. In addition to achieving a reduction in relapse rates, the review also showed the sustained ability of methotrexate to allow discontinuation of glucocorticoid for up to 24 weeks. The side-effect profile was acceptable.

French consensus guidelines also mention the use of methotrexate based on a systematic review of three randomized controlled trials. Using oral methotrexate 7.5–15mg per week they saw a modest reduction in the risk of relapse and the cumulative glucocorticoid dose (Yates et al, 2013, 2014).

Azathioprine

The earliest trial on the use of azathioprine showed a significant reduction in glucocorticoid requirements in the treatment group, but later studies and meta-analyses showed no significant advantage of adjuvant azathioprine (Muratore et al, 2017).

Tocilizumab

The first phase 2 randomized controlled trial testing the efficacy and safety of tocilizumab, an IL-6 receptor inhibitor monoclonal antibody, for treating relapsing

giant cell arteritis was published in 2016. Out of a total of 30 patients, 17 (85%) in the tocilizumab and glucocorticoid arm (n=20) achieved remission within 12 weeks compared to only 4 out of 10 (40%) in the glucocorticoid plus placebo group. Time to remission was also shorter in the treatment group. Additionally, 17 out of 20 patients in the treatment group achieved relapse-free survival. Individuals on tocilizumab managed to wean off glucocorticoids earlier by a mean of 12 weeks, thus having a lower cumulative dose of glucocorticoids. This was a small study and the design was not robust enough to demonstrate the desired results although it showed promise in the use of tocilizumab (Villiger et al, 2016). A later review of data from previous trials confirmed that a 2–4-fold remission rate was achievable with the use of tocilizumab with glucocorticoids (Buttgereit et al, 2016).

The National Institute for Health and Care Excellence (2018) has issued new guidance for treating relapsing giant cell arteritis with tocilizumab, based on the Giant-Cell Arteritis Actemra (GiACTA) trial. This included a randomized controlled trial based on four arms in the trial which included tocilizumab in the first two arms comparing with those on glucocorticoid only. This study included 251 patients with known giant cell arteritis with biopsy-proven cranial or radiological giant cell arteritis involving proximal arteries. Individuals were randomly assigned to four groups: those receiving a weekly 162 mg subcutaneous tocilizumab plus a tapering dose of prednisone over 26 weeks (100 patients), those receiving 162 mg subcutaneous tocilizumab every other week plus a tapering dose of prednisone (50 patients), weekly subcutaneous placebo plus a 26-week tapering dose of prednisone (50 patients) and weekly subcutaneous placebo plus a 52-week tapering dose of prednisone (51 patients). Use of tocilizumab after glucocorticoid tapering caused earlier sustained remission and increased the time until the next flare of disease.

More than 50% achieved sustained glucocorticoid-free remission in the first two groups that were given tocilizumab and the overall cumulative dose of glucocorticoid was significantly reduced during the 52-week follow-up period. Patients in the two tocilizumab arms experienced similar adverse events than those in the other two arms in the study but fewer serious adverse events (12–14% *vs* 22–25%). The safety profiles were acceptable. It took longer to see any positive outcome in those receiving tocilizumab and thus it should be prescribed for at least 6 months to 1 year. It is licenced for treatment for 1 year in the UK (Stone et al, 2017).

According to the National Institute for Health and Care Excellence (2018) guidelines, tocilizumab can be used with a tapering dose of glucocorticoid or immediately after glucocorticoid use in relapsing or refractory disease in those who have not already had tocilizumab. It is stopped after 1 year of uninterrupted treatment.

Other biological agents

Use of abatacept, etanercept, adalimumab and infliximab has been studied but larger studies are needed to be able to compare these outcomes to those of tocilizumab.

Adjunctive treatment

Aspirin

Most evidence for the use of anticoagulants to prevent ocular or intracerebral events is from retrospective studies. The British Society of Rheumatology advise the cautious use of aspirin in individuals at risk of ischaemic ocular or intracranial events and avoidance in cases where the risk of bleeding is higher. The recommended dose of aspirin is 75–150 mg once a day (Dasgupta et al, 2010).

The decision to prescribe aspirin is based on individual presentation and risks of cranial ischaemia (Dejaco et al, 2015). Previous history including medications being taken, the risk of evolving ischaemic symptoms and severity of inflammatory response should be considered when prescribing aspirin.

Bone protection

Individuals taking steroids should be considered for calcium supplements and vitamin D. Bisphosphonates should be considered in suitable candidates with no contraindications (Dasgupta et al, 2010).

Proton pump inhibitors

Individuals with a high risk of upper gastrointestinal acid peptic disease, or those who have been prescribed aspirin or non-steroidal anti-inflammatory drugs, should be prescribed concomitant proton pump inhibitors.

Conclusions

Giant cell arteritis can be difficult to diagnose. Duplex ultrasound is a useful tool that can be used in clinic settings. The mainstay of treatment and the role of these treatments has not changed – various biological and non-biological therapies are being used. Methotrexate and tocilizumab are used for recurrent relapse and to reduce remission. Acute management of complicated giant cell arteritis may require hospital admission. BJHM

Conflict of interest none.

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KEY POINTS

- Giant cell arteritis can be difficult to diagnose. Duplex ultrasound is a useful tool that can be used in clinic settings.
- The mainstay of treatment and their role has not changed, with various biological and non-biological therapies being used. Methotrexate and tocilizumab are used for recurrent relapse and to reduce remission.
- Acute management of complicated giant cell arteritis may need hospital admission.