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Long-term Outcomes and Relapse Rates in GCA: A Retrospective Cohort Study

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Introduction

Giant Cell Arteritis (GCA), the most common form of systemic vasculitis in adults over 50, remains a clinically significant condition with substantial long-term morbidity. Characterized by granulomatous inflammation primarily affecting the aorta and its major branches, especially the extracranial branches of the carotid artery, GCA poses a diagnostic and therapeutic challenge due to its heterogeneous presentation and chronicity. This retrospective cohort study explores the long-term outcomes and relapse rates among patients diagnosed with GCA over a span of ten years, emphasizing clinical trajectories, steroid dependency, complications and prognostic indicators influencing disease recurrence and morbidity [1].

Description

The retrospective analysis included patients with biopsy-proven or imaging-supported GCA, presenting to a tertiary referral center between 2005 and 2015. Demographic, clinical, laboratory and treatment data were retrieved from medical records and outcomes were assessed through longitudinal follow-up extending up to ten years. Key outcome measures included relapse rates, cumulative glucocorticoid exposure and development of large-vessel complications (such as aortic aneurysms or dissection), visual loss, steroid-related adverse events and mortality. Relapse was defined as recurrence of GCA-related symptoms (e.g., headache, jaw claudication, polymyalgia rheumatica symptoms, constitutional symptoms), supported by laboratory evidence of inflammation and necessitating escalation of immunosuppressive therapy. The cohort comprised 198 patients (134 females, 64 males) with a mean age at diagnosis of 72.6 years. Most patients presented with classical cranial manifestations including new-onset headache (72%), scalp tenderness (59%) and jaw claudication (45%). Visual symptoms were reported in 28% of patients and permanent vision loss occurred in 11%. A significant number of patients (22%) also presented with Polymyalgia Rheumatica (PMR)-like symptoms, while large-vessel involvement (based on imaging) was detected in 19% of patients, either at diagnosis or during follow-up [2,3].

During a mean follow-up of 6.8 years, 97 patients (49%) experienced at least one relapse, with 41% of those experiencing multiple relapses. The median time to first relapse was 14 months, with earlier relapses (within 12 months) associated with higher initial Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP) levels. Approximately 29% of relapsing patients had large-vessel involvement, suggesting a correlation between extended vascular inflammation and chronic disease activity. Patients with large-vessel GCA tended to exhibit fewer cranial symptoms but were more likely to have constitutional complaints and sustained elevations in inflammatory markers, often contributing to delayed diagnosis and suboptimal treatment initiation. Steroid tapering proved to be a key determinant in disease course. While all patients initiated therapy with high-dose glucocorticoids (typically 40–60 mg/day prednisone or equivalent), the tapering regimen varied significantly. A notable 63% of patients remained on corticosteroids beyond 24 months and 37% required low-dose maintenance therapy even after five years due to relapse or persistent symptoms. Cumulative steroid exposure correlated directly with the incidence of adverse effects such as osteoporosis (19%), diabetes mellitus (14%), weight gain, cataracts and hypertension. The burden of steroid toxicity underlined the urgent need for steroid-sparing alternatives in the chronic management of GCA [4].

Visual complications were among the most feared outcomes of GCA. In our study, 22 patients developed irreversible vision loss, primarily due to anterior ischemic optic neuropathy. Most cases of visual loss occurred within the first two weeks of diagnosis, underscoring the critical importance of prompt recognition and initiation of therapy. However, three patients experienced visual decline during steroid tapering, highlighting the potential for subclinical inflammation to persist even under treatment. Imaging modalities such as ultrasound, PET-CT and MRI were instrumental in assessing disease activity in these cases, guiding therapeutic escalation. Patient-reported outcomes reflected the physical and psychological burden of GCA and its treatment. Chronic fatigue, muscle weakness, anxiety related to relapse and steroid-induced mood disturbances were commonly reported. Quality of life scores improved during remission but remained below baseline for many patients, particularly those with relapsing disease or steroid complications. These findings advocate for a multidisciplinary approach to care, including rheumatologists, ophthalmologists, cardiologists and mental health professionals [5].

Conclusion

In conclusion, GCA remains a chronic, relapsing vasculitis with significant long-term health implications. Nearly half of the patients in our cohort experienced disease relapse, often necessitating prolonged corticosteroid therapy and facing substantial morbidity from both disease and treatment. Early diagnosis, vigilant monitoring for relapse, judicious tapering of steroids and incorporation of steroid-sparing therapies are essential for optimizing long-term outcomes. With the growing evidence for biologic agents such as tocilizumab and the advent of improved imaging techniques, individualized treatment strategies hold promise for reducing relapse rates and improving quality of life for patients with GCA. Further prospective studies are warranted to refine prognostic models and guide long-term surveillance and therapeutic decisions in this complex disease.

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Conflict of Interest

None.

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