The clinical characteristics, treatment, and outcomes of giant cell arteritis are dependent on histological subtype

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Giant cell arteritis (GCA) can cause sudden and potentially bilateral sequential visual loss in the elderly. Therefore, it is considered a medical emergency in ophthalmology and a significant cause of morbidity in an increasingly aging population. Our study goal was to determine the clinical characteristics, treatment, and outcomes of the two histopathological patterns considered positive for giant cell arteritis: active arteritis and healed arteritis. A retrospective chart review was performed on 22 patients with biopsy-proven GCA. Eleven patients had active arteritis and eleven patients had post-inflammatory alterations consistent with healed arteritis. We sought to compare presenting symptoms, ischemic ocular events, inflammatory markers (erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and platelet count), relapses, and dosage requirements of long-term steroids between the two groups. Seven of 11 patients with active arteritis had an initial ocular ischemic event while 3 of the 11 patients with healed arteritis had an initial ischemic event to the eye. There was no statistical difference in initial ESR between the two groups, but CRP and platelet counts on initial presentation were statistically higher in the active group (p=0.0002 and p<0.0001 respectively). Patients with active arteritis on biopsy required higher doses of steroids over a 2-year follow-up compared to the healed group: on average 11 mg/day vs. 1 mg/day at 1 year (p=0.0008), and 7 mg/day vs. 0.5 mg/day at 2 years (p=0.0208), respectively. During the follow-up period, 2 of the 11 patients in the active group demonstrated a recurrent ischemic event to the same or fellow eye while in the healed group there were no recurrent ischemic events. Patients with healed arteritis on pathological examination of temporal artery biopsy appear to have better prognoses and may require less aggressive treatment than those with active inflammation.

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Biography

Joseph G Chacko, MD has been the Director of Neuro-Ophthalmology at Harvey and Bernice Jones Eye Institute, University of Arkansas for Medical Sciences (UAMS) since 2005. He is Professor of Ophthalmology, Neurology, and Neurosurgery in the UAMS College of Medicine. He received his medical degree from the Medical College of Pennsylvania in Philadelphia (now Drexel University College of Medicine) in 1991. He completed Residency in Ophthalmology at the Medical College of Georgia in Augusta in 1995. He then went on to complete a Fellowship in Neuro-Ophthalmology at Bascom Palmer Eye Institute, University of Miami in 2005. His research interests include ischemic optic neuropathy, giant cell arteritis, divergence insufficiency, and pseudotumor cerebri. He has been board-certified by the American Board of Ophthalmology since 1996. He became a Fellow in the North American Neuro-ophthalmology Society (NANOS) in 2010.

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