International Journal of Medical Ophthalmology

E-ISSN: 2663-8274
P-ISSN: 2663-8266
www.ophthalmoljournal.com
IJMO 2022; 4(2): 34-36
Received: 12-10-2022
Accepted: 14-11-2022

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Giant cell arteritis with bilateral vision loss: A case report

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DOI: https://doi.org/10.33545/26638266.2022.v4.i2a.125

Abstract
Giant cell arteritis (GCA) also known as temporal arteritis, is a systemic vasculitis of medium and large-size vessels. It can lead to permanent visual loss in elderly patients. Here we report an old patient presenting with bilateral loss of vision later diagnosed as giant cell arteritis.

Keywords: Giant cell arteritis, vasculitis, optic atrophy

Introduction
Giant cell arteritis (GCA) is a most common primary systemic vasculitis less frequently seen in Asian adults [1-3]. Most common presentation of GCA is permanent visual loss (85%) secondary to arteritic anterior ischemic optic neuropathy (AAION) [4]

Case presentation
An 80 years old patient came to Eye OPD with chief complaint of loss of vision both eyes since around one week, which was sudden in onset, progressive in nature. There was history of headache & jaw claudication, for which patient also visited dentist around 5-6 days back and treatment was given in the form of oral analgesic and antibiotic, but symptoms persisted. The patient went to private hospital for these complaints. He was given injection methyl prednisolone stat and MRI Brain was advised. MRI Brain was done two times and it was seen that there were atrophic and small vessel ischemic changes and area of encephalomalacia in right parieto-occipital lobe. The patient went to neurologist and later on referred to Eye OPD. There was no history of any chronic disease. On examination patient was alert and oriented. On examination visual acuity was no perception of light bilaterally. The pupils were not reacting to light. The extra-ocular movements were full & unrestricted in both eyes. On complete eye examination it was found that there was pseudophakia both eyes and a macular corneal opacity in inferior quadrant of left eye (Fig. 1A & 1B). On fundus examination there was slight media haze left eye, bilaterally swollen optic disc with blurred margins and chalky white appearance (pallid disc oedema) (Fig. 2A & 2B). The bilateral superficial temporal arteries were palpable. On routine laboratory tests, fasting blood sugar was 94 mg/dl, erythrocyte sedimentation ratio (ESR: 80 mm/h) and C-reactive protein (CRP: 30 mg/L) was elevated. The patient was diagnosed as giant cell arteritis and was started on oral prednisolone 60 mg and which was tapered on afterward. The vision acuity remained no perception of light after treatment, whereas the headache and jaw pain disappeared. The patient was then kept under regular follow ups and prognosis explained to the patient.

Discussion
Giant cell arteritis also known as temporal arteritis is a most common primary systemic vasculitis, less frequently seen in Asian adults [1-3]. The average age of presentation is 72.5 years for women (2-6 times), and 70.3 years old for men [5, 6]. Histopathologically, GCA is marked by generalized granulomatous inflammation of medium- to large-sized vessels that occurs in the elderly. The most common vessels affected are the superficial temporal artery, the ophthalmic artery, the posterior ciliary arteries, and the vertebral arteries [7] less commonly, the aorta, coronary arteries, intracranial arteries are typically spared [8]. Although ocular involvement is common in GCA, the incidence of ocular findings (14-70%) in all GCA patients is not well-defined [6, 9]. The visual loss from AAION can initially be transient, and can become bilateral and permanent. The AAION is characterized by acute monocular vision loss accompanied by optic disc edema.

The optic disc edema in AAION is usually diffusely "chalk white", and may be accompanied by disc hemorrhage, retinal whitening or cotton wool spots which raise a clinician's suspicion for GCA. Systemic symptoms are present in most patients, may be acute or gradual, and often precede the ocular manifestations [10]. The most common systemic complaint of GCA patients is a new onset headache (90% - temporal or occipital), often associated with localized or diffuse scalp tenderness [11]. The patients may complain of jaw claudication, diplopia, myalgias and constitutional symptoms. GCA is considered an ophthalmologic emergency & treatment with corticosteroids is indicated on an urgent basis, as further vision loss and fellow eye involvement are usually preventable. According to the American College of Rheumatology (1990), the following are classification criteria for GCA (sensitivity of 93.5% and specificity of 91.2%). The criteria include the presence of at least 3 of the following 5 findings [12]:

1. Greater than 50 years old
2. New onset of headache
3. Temporal artery abnormality
4. Elevated erythrocyte sedimentation rate (ESR) (>50 mm/h)
5. Abnormal arterial biopsy showing a necrotizing vasculitis.

   There is no single lab test, imaging result which will always be positive in all patients which makes diagnosis of GCA difficult. Each patient with GCA can present with a wide range of symptoms and examination findings, and many of the symptoms may be transient, patients must be questioned directly about symptoms of GCA.

The most common differential diagnosis of GCA is non-arteritic ischemic optic neuropathy (NAION). AAION patients do not have vascular risk factors whereas NAION is associated with hypertension, diabetes, and hyperlipidemia [13]. In addition, patients with GCA are typically older (8th-9th decade) than those with NAION (6^{th}-7^{th} decade). In NAION, there will be no associated systemic symptoms such as headache, jaw claudication, scalp tenderness, etc. In AAION the vision loss is more severe [14]. AAION patients demonstrate a diffuse "chalky" white edema [15], eventual cupping of the disc, and possible coexistent retinal ischemia but NAION demonstrates segmental optic nerve edema. Also laboratory tests e.g. ESR and CRP are usually normal in patients with NAION. Other differential diagnoses are rheumatoid arthritis, systemic lupus erythematosis, polyarteritis nodosa, polymyositis, and takasayu arteritis. malignancy, systemic infections, dental disease, trigeminal neuralgia, etc.

Conclusion

GCA can lead to permanent blindness in elderly patients caused by anterior ischemic optic neuropathy. Based on history, examination findings, and lab tests (an elevated ESR, C-reactive protein), the initiation of corticosteroid treatment and a temporal artery biopsy should be considered. In addition, several other tests may confirm the diagnosis.

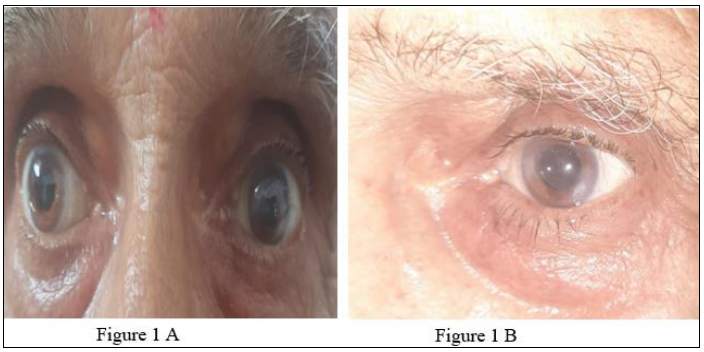

Fig 1: (A & B), Pseudophakia both eyes, with macular corneal opacity left eye

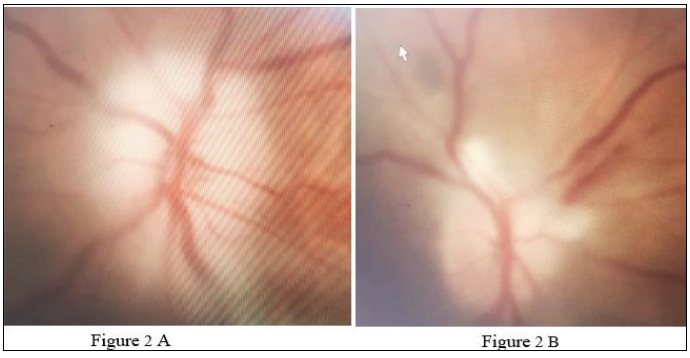

Fig 2: (A & B), Pallid disc oedema bilaterally with hemorrhage near disc in left eye

Financial Disclosure(s)

The author(s) have no proprietary or commercial interest in any materials discussed in this article.

Funding/Support

The author (s) don't have any government and non-government support.

Conflicts of interest

There are no conflicts of interest.

Other Acknowledgments

I am thankful to the department of Ophthalmology of our institution for the diagnosis and management of this patient.

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How to Cite This Article
Mohammad SK, Gupta S. Giant cell arteritis with bilateral vision loss: A case report. International Journal of Medical Ophthalmology. 2022;4(2):34-36.