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A Rare case report: Giant Cell Arteritis presenting as Trismus

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ABSTRACT:Giant cell arteritis (GCA), also referred to as Temporal arteritis is an inflammation of medium and large sized arteries. It commonly involves one or more branches of carotid artery particularly the temporal artery. Commonly involves elderly > 50 years of age.[1] Early diagnosis and treatment are essential to avoid serious complications. This article reports a rare case of GCA presenting as trismus.The occurrence of relative acute jaw opening reduction in an elderly patient with headache and weight loss should alert for diagnosis of GCA and should be investigated thoroughly including a temporal artery biopsy and consideration of steroid use if not contraindicated to prevent devastating complications of visual loss.
KEYWORDS: Giant cell Arteritis, jaw claudication, Trismus.

I. INTRODUCTION

Giant cell arteritis, also referred to as Temporal arteritis is an inflammation of medium and large sized arteries. It commonly involves one or more branches of carotid artery particularly the temporal artery. Commonly involves elderly > 50 years of age.[1]

However, it is a systemic disease that can involve arteries in multiple locations particularly the aorta and its branches.

It is a Pan arteritis with inflammatory mononuclear cell infiltrate within the vessel wall with frequent giant cell formation.[2]

GCA may result in permanent visual loss due to ischemia of the optic nerve as a result of vascular occlusion. Therefore, GCA is considered as a medical emergency. GCA has been associated with other life-threatening complications including Myocardial infarction, renal insufficiency, intestinal infarction, aortic aneurysm, Stroke, transient ischemic attack, Pulmonary embolism. Hence Early diagnosis and prompt treatment are essential to prevent serious complications.[3]

II. CASE PRESENTATION

Mr. A, a 50-year-old male, resident of Goa, a known Diabetic and hypertensive was admitted in Goa Medical College with restriction in mouth opening for 1 month.

It had a gradual onset when he complained of multiple aches and pains in the chest, shoulders and lower limbs. Along with this, patient started noticing a loss in weight and then progressive difficulty in chewing and opening the mouth for 15 days prior to admission. In the last 7 days he had an added complaint of left eye visual blurring when he attempted to chew.

Patient also gave history of odynophagia while swallowing.

However, there was no history of fever or instability of the jaw. Patient denied any history of headache or scalp tenderness.

There was no history of oral ulcerations, dry mouth, foreign body sensation in the eyes or any history suggestive of Raynaud's phenomenon.

On physical examination, vitals were found to be within normal limits.

Mouth opening was restricted to 2.1 cm (unassisted).Mandibular opening pattern was straight.

Oral hygiene was notably poor.

Bilateral temporal arteries were thickened and tender.

All other peripheral pulsations were normal.

Neurological, cardiovascular, respiratory examination were within normal limits**.**

His laboratory tests revealed the following
ALP- 309, SGOT- 90, SGPT- 112
ESR- 62 (raised)
Quantitative CRP- 192mg/L
Platelet count – 5.5 lakhs/mcl
Hb- 9.7 g%
Total CPK- 20 mcg/L (normal)
Anti-Nuclear Antibody- Negative
Rheumatoid factor- Negative
Anti-CCP- Negative
HbA1c- 9.8%
Fundus- within normal limits.
Visual Fields- Within normal limits.
MRI brain and spine- Normal

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Glaucoma and Ischemic Optic Neuropathy were ruled out.

Raised liver enzymes
Raised ESR
Raised CRP
Thrombocytosis
Anaemia

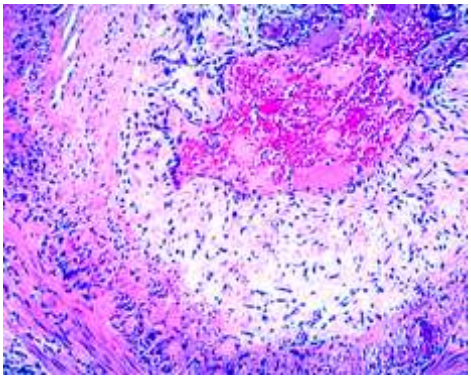
Based on patients age, clinical presentation, lack of history of temporo- mandibular joint disorders and laboratory tests including raised ESR and CRP, a provisional diagnosis of GCA was made. Patient was further referred for a temporal artery biopsy.

The following was inferred from the laboratory tests:

Temporal artery Biopsy was done under Local anaesthesia.

Sections showed a medium calibre artery with thickening of wall. There was internal hyperplasia and myxoid degeneration. Tunica media showed smooth muscle hyperplasia and dystrophic calcification. Adventitia showed fibrosis and capillary proliferation. All three layers showed lymphoplasmacytic infiltration.
All these features were consistent with the diagnosis of **Temporal Arteritis.**

Fig 1: Examination of **Biopsy** sample of temporal artery showing histopathological picture of GCA. There is narrowing of lumen of vessel wall and cellular infiltration of vessel wall along with intimal hyperplasia.



Treatment And Outcome

Patient was diagnosed as a case of Temporal Arteritis and was started on high dose Steroids (injection methylprednisolone 1g OD for 5 days)
Patient showed a dramatic response to steroids.
Jaw opening showed a drastic improvement from 2.1 cm on the day of admission to 4.5 cm on Day 5 of steroid therapy.
Patient was continued on steroid at 1mg/kg/day for 4 weeks and then gradually tapered.
4 months post treatment patient is doing well and is asymptomatic on prednisolone 15mg per day which will be tapered gradually.

III. DISCUSSION AND REVIEW OF LITERATURE

Giant Cell Arteritis is diagnosed based on a clinical criterion given by the American College of Rheumatology which is as follows:[4]

- Age at onset of disease > 50 years
- Localized headache (of new onset or of a new type)
- Abnormality of the temporal artery, specifically tenderness to palpation or decreased pulsation, unrelated to arteriosclerosis of cervical arteries
- Erythrocyte sedimentation rate > 50 mm/h
- Abnormal results on biopsy of temporal artery: artery showing vasculitis characterized by a predominance of monocular cell infiltration or granulomatous inflammation, usually with multinucleated giant cells.

3 out of the 5 features should be present for a diagnosis of giant cell arteritis with high sensitivity and specificity.
In this case report, the age of onset, elevated ESR and abnormal Temporal artery biopsy confirmed the diagnosis of GCA.

Giant Cell Arteritis (GCA) presents with common complaints which include nonspecific signs such as fatigue, fever, headache, muscle and girdle pains.[5]
These nonspecific symptoms in combination with the age of the patient and results of a few tests (raised ESR, Raised ALP, Thrombocytosis) leads to tentative diagnosis of GCA. Diagnosis is confirmed by positive biopsy of temporal artery.

Jaw claudication is a very common symptom in patients with Temporal Arteritis occurring in up to 40% patients.[6,7]

Trismus has been reported very rarely in the literature in conjunction with GCA.It may be an underestimationas these complaints are underreported by the patients or it may be due to lack of physician's awareness as this complaint may be easily confused with a more common complaint of Claudication.[5]

It is seen in Literature that patients with GCA and Trismus have a more aggressive form of disease manifested by high rate of eye involvement and markedly shorter duration of symptoms until diagnosis (4 weeks in comparison to 12 weeks in GCA).
This implies that Trismus Sign reflects a more aggressive form of GCA.[5]
In addition to trismus, the patient described here had worsening arthralgias which improved with steroid therapy. This may be linked to flare of symptoms of Polymyalgia Rheumatica, a form of arthritis of proximal large joints which may be associated with GCA.

It should be mandatory for all physicians including dental and ENT surgeons to be aware that both claudication and trismus can be a potential symptom of GCA; a disease which can have devastating consequences like blindness if not diagnosed and treated on time.

GCA can be an elusive diagnosis as it has diverse clinical manifestations.

This case highlights the importance of considering the diagnosis of GCA in a patient presenting with trismus. Cases of trismus misdiagnosed as temporo-mandibular joint disorder have been reported in literature.[8]

IV. CONCLUSION

We conclude that the occurrence of relative acute jaw opening reduction in an elderly patient with headache and weight loss should alert for diagnosis of GCA It should be investigated thoroughly including a temporal artery biopsy and consideration of steroid use if not contraindicated to prevent devastating complications of visual loss.

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