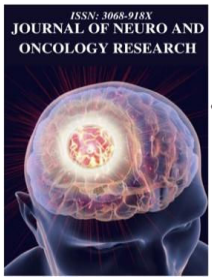

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Case Report

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Giant Cell Arteritis with a Rare Manifestation of Internal Carotid Artery and Middle Cerebral Artery Stenosis and Cerebral Infarct: A Case Study

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Abstract

Giant Cell Arteritis (GCA) is a granulomatous vasculitis of large and medium arteries, most frequently affecting branches of the external carotid artery and rarely presenting with ischemic stroke.

To describe a rare case of giant cell arteritis identified incidentally in a carotid endarterectomy specimen performed for symptomatic internal carotid and middle cerebral artery stenosis leading to cerebral infarction.

A 58-year-old male presented with sudden loss of consciousness, slurred speech and right hemiparesis. MRI revealed an acute left gangliocapsular infarct and MRA demonstrated absent flow in the left ICA/MCA. Carotid Doppler showed ~50% stenosis due to an eccentric plaque. The patient underwent left carotid endarterectomy and histopathology unexpectedly showed giant cell arteritis superimposed on a hard atherosclerotic plaque. Corticosteroid therapy was initiated after histopathological confirmation.

Routine histopathological examination of CEA specimens can reveal rare but clinically important causes of ischemic stroke, such as GCA, thereby altering long-term management.

The patient demonstrated neurological improvement following surgical intervention and corticosteroid therapy, highlighting the importance of routine histopathological examination of carotid plaques in atypical cerebrovascular presentations.

Keywords: Giant Cell Arteritis; Internal Carotid Artery Stenosis; Middle Cerebral Artery; Carotid Endarterectomy; Cerebral Infarction; Large-Vessel Vasculitis; Histopathology

Introduction

Giant Cell Arteritis (GCA) is a granulomatous vasculitis of medium and large-sized arteries characterized by immune-mediated injury to the arterial wall [1-4]. The disease exhibits reactive vascular remodeling, including endothelial activation, medial destruction and marked intimal hyperplasia, leading to luminal narrowing and ischemia. These changes occur in a segmental distribution ("skip lesions"), reflecting discontinuous inflammatory involvement. The resulting vascular reactive regions represent a non-neoplastic, inflammation-driven response rather than a true proliferative vascular lesion and account for the variable diagnostic yield of temporal artery biopsy. Giant Cell Arteritis (GCA) is chronic granulomatous arteritis that affects medium- to large-sized arteries and is classified as large-vessel vasculitis. Giant Cell Arteritis (GCA) is the most common primary vasculitis in adults over 50 years, classically affecting branches of the external carotid artery such as the superficial temporal artery [1-9]. The typical presentation includes headache, scalp tenderness, jaw claudication, visual symptoms and elevated inflammatory markers [1,11,12]. We describe a case of a Cerebral (Left Gangliocapsular Region) Infarct in the case of Internal Carotid Artery (ICA) and Middle Cerebral Artery (MCA) Stenosis, who underwent a Carotid Endarterectomy as a therapeutic intervention. Stroke is an uncommon but serious complication of GCA, occurring in only 3-7% of cases [2-4]. When stroke occurs, the vertebrobasilar circulation is more commonly involved [4,10]. Involvement of the extracranial Internal Carotid Artery (ICA) is rare and the discovery of GCA within a Carotid Endarterectomy (CEA) specimen is

even more unusual [11].

Case Presentation

History

A 58-year-old male presented with a history of loss of consciousness 2 days before admission to our centre. He regained consciousness over a few hours, following which he was found to have a slurred speech and weakness of the right upper and lower limbs. After initial evaluation and treatment, the patient was shifted to our centre for further management.

Clinical Findings on Admission

- Vitals: Pulse - 90/min, BP -180/100 mmHg, Respiratory Rate - 20/min, SpO_2 - 99%
- Sensorium: Drowsy, Arousable
- Speech: Slurred
- Right Facial Weakness (Upper Motor Neuron type)
- Power (MRC Grades): Right Upper Limb: Grade 0, Right Lower Limb: Grade 3, Left Upper Limb: Grade 5, Left Lower Limb: Grade 5
- No headache, scalp tenderness, vision loss, jaw claudication, fever or constitutional symptoms

Laboratory Investigations

All routine laboratory investigations were done pre-operatively and post-operatively and were normal. Erythrocyte Sedimentation Rate and C-Reactive Protein sent after diagnosis of Giant Cell Arteritis, were found to be slightly elevated (Table 1).

Erythrocyte Sedimentation Rate	20 mm at 1 hour
C-reactive Protein	15.1 mg/L

Table 1: Laboratory investigations.

Histopathology

Multiple haematoxylin and eosin stained sections of the submitted tissue were studied. They revealed the bits to be composed of arterial wall. The wall showed a plaque compose of lots of lipid material with a large area of fibrotic material calcification. There was no area of haemorrhage in the plaque. The overlying intimal cap was thick with non-ulcerated intimal lining. The intima showed a mild infiltrate composed of lymphocytes with a few neutrophils. The wall of the vessel beneath the plaque showed a dense lymphoid infiltrate with some neutrophils. This infiltrate was admixed with multiple foreign body type of giant cells. There was no evidence of any other specific pathology and the histopathological impression was: giant cell arteritis with hard atherosclerotic plaque. These features were consistent with giant cell arteritis superimposed on atherosclerotic disease (Fig. 1) [1,6].

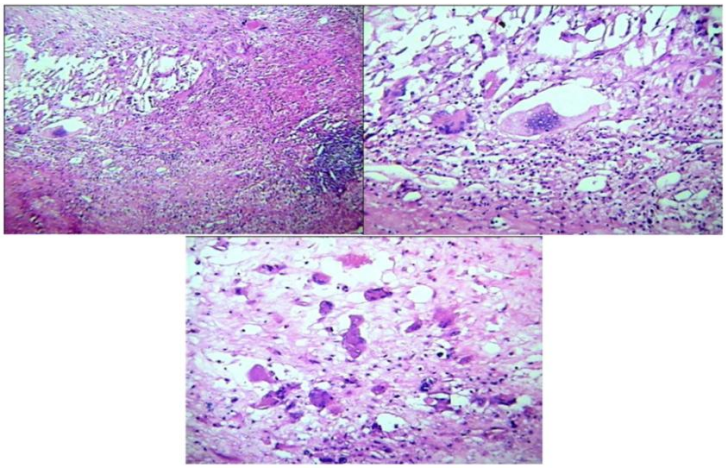

Figure 1: Histopathological examination: haematoxylin and eosin-stained sections.

Radiological Findings

The patient had reported to us with an MRI done at another centre which showed an acute infarct involving the left basal ganglia, left internal capsule and left corona radiata. An MR-Angiography done showed a complete absence of flow signal in the entire intracranial and extracranial portion of the left Internal Carotid Artery (ICA) as well as left Middle Carotid Artery (MCA) and an absent flow signal in the entire length of the left vertebral artery, suggestive of occlusion of these arteries. It also noted a severe hypoplasia of the A1 segment of the left Anterior Cerebral Artery (ACA). A colour Doppler study of the neck vessels was done to further evaluate these arteries; and it showed a 18 x 1.3 mm eccentric echogenic plaque in the left carotid bulb, extending into the proximal left ICA causing around 50% stenosis at the origin of the internal carotid artery. Slow flow was noted at the mid left internal carotid artery suggestive of a distal occlusion. It also showed a 11 x 2.8 mm eccentric echogenic plaque in the right proximal internal carotid artery causing 45% stenosis. No flow was seen in the left vertebral artery suggestive of its complete occlusion. A screening 2D Echocardiogram was done to rule out cardiac vegetations/clots; it was reported normal.

Therapeutic Intervention

The patient was started on dual antiplatelet therapy (ASPIRIN + CLOPIDOGREL), along with a statin (ATORVASTATIN) and fibrate (FENOFIBRATE) for dyslipidaemia. Anti-epileptic drug, LEVETIRACETAM, was also given; along with antihypertensive drugs and other symptomatic treatment. In view of the left internal carotid artery stenosis; left carotid endarterectomy was done with written, informed consent. EEG monitoring during this procedure revealed no abnormalities throughout the procedure. After detection of giant cell arteritis, corticosteroid therapy was initiated with oral methylprednisolone (Fig. 2).

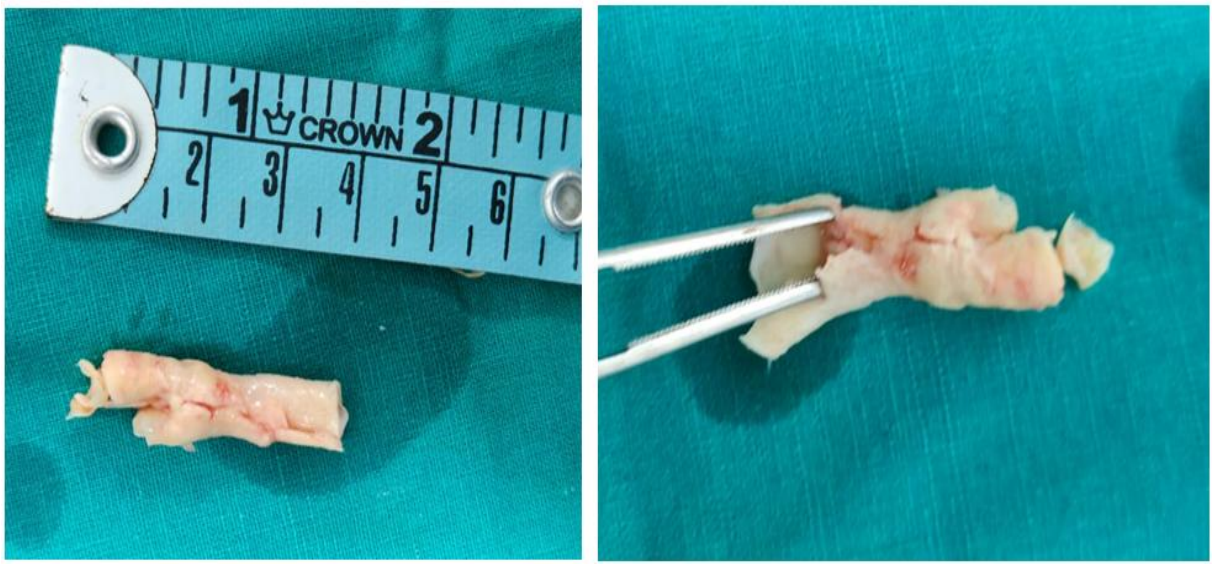

Figure 2: Gross specimen.

Course During Indoor Stay

With written, informed consent, surgical intervention via a left carotid endarterectomy was done as a measure to prevent further infarcts. Regular physiotherapy was given. Gradual improvement was noted in the patient. His sensorium improved; he became more alert. Power in his right upper and lower limbs also improved. Speech became more comprehensible. The patient had difficulty in swallowing despite having a gag reflex on both sides; for which swallowing exercises were taught. The patient was able to take oral feeds thereafter. On post-operative day 12, the surgical wound was examined and found to be healthy and sutures were removed. As the patient was neurologically stable, he was discharged with further advice for physiotherapy and rehabilitation; and instructions to follow up regularly for further evaluation and management.

Condition at Discharge

The patient was vitally stable at the time of discharge. He was conscious, both his pupils were equal in size (3 mm) and reactive to light. He had a slurred speech and had a weakness of the right side of the face (upper motor neuron type); improvement was noted in these, as compared to admission. He was paretic in his right upper limb (MRC Grade I) and the right lower limb (MRC Grade III). The power in his left upper and lower limb was intact (MRC Grade V).

Follow Up

The patient returned for a routine follow-up 1 month after discharge. He was conscious, with an improvement in his speech, as well power in his right upper limb (Grade III from Grade I) and right lower limb (Grade IV from Grade III, MRC). The power in his left upper and lower limb was intact (MRC Grade V).

Discussion

Giant Cell Arteritis (GCA) is not commonly detected at our centre. Although GCA frequently affects cranial arteries, ischemic stroke is rare [2-4]. Posterior circulation strokes predominate due to vertebrobasilar involvement [4,10]. ICA-territory stroke as the presenting feature of GCA is much less common [8]. However, it is noted that diagnosis of GCA is critical because delay can lead to major problems to the patient such as irreversible loss of vision, depending on the intracranial artery involved [2]. At our centre, after we perform Carotid Endarterectomies, the plaque extracted from the carotid artery is sent for Histopathological Examination as part of routine protocol. This is to look for diseases such as vasculitis, so that treatment might be initiated promptly. However, the patients and/or their attendants often opt out of this. This might have led to missed diagnosis of GCA over the years, the present case thus heralding a change in institute protocol. Several authors have reported incidental GCA in CEA specimens, highlighting the value of routine HPE in atypical or disproportionate carotid stenosis [17].

Chronic Granulomatous Inflammation
↓
Persistent Immune Cell Activation
(Macrophages, T-cells, cytokines)
↓
Chronic Vascular Wall Injury
↓
Intimal Hyperplasia
(↑ Smooth muscle proliferation
and extracellular matrix deposition)
↓
Luminal Narrowing
(Reduced vessel diameter)
↓
Altered Blood Flow and Endothelial Dysfunction
↓
Thrombosis
(Platelet aggregation and clot formation)
↓
Acceleration of Atherosclerosis
(Increased plaque formation and instability)

These mechanisms explain how vasculitis may coexist with typical atherosclerotic stenosis, as seen in this patient. Biopsy of the temporal artery is the principal means of making the diagnosis; however, even during the active period of GCA, temporal artery biopsy can give negative results because of the segmental pathological changes in the temporal arterie [3]. Stroke is a potentially severe complication of Giant Cell Arteritis, although it is rarely attributed to it; the prevalence of GCA in a study of over 4000 patients with stroke was 0.15% [3]. A study in 2022 by Hashami, et al., reports a similar case wherein the patient had presented with acute ischemic infarction and internal carotid artery dissection, with a high ESR level [2]. Presenting complaints of the patient were blurred vision and ptosis in the right eye, left hemiparesis and frozen movements. A temporal artery biopsy was done to confirm their suspicion of giant cell arteritis and steroid therapy was given. While inflammatory markers were reduced and improvement in headaches, there was no significant improvement in the symptoms of the patients. Hence, it is to be considered that both these diseases should be evaluated comprehensively; not exclusively of each other. For this, we propose the following to be included in the routine evaluation of a patient of stroke/giant cell arteritis.

1. Detailed Neurological History and Examination, with emphasis on:

- Jaw/Head/Neck/Lim Claudication
- Amaurosis Fugax
- Scalp Tenderness

2. Laboratory Investigations
- Lipid Profile
- C-Reactive Protein
- Erythrocyte Sedimentation Rate
3. Radiological Investigations
- Colour Doppler Study of the Neck Vessels
- MRI of the Brain with Angiography of the Cerebral and Neck Vessels
- Screening 2-D Echo

A study by Alsolaimani, Roaa, et al., shows arrest of disease progression after initiation of therapy, in cases of giant cell arteritis with stroke, with significant improvement and even complete remission in 1 case [7]. We propose that the following therapeutic strategies be considered in the management of the disease(s), with the aim of reducing ischemic complications, controlling vascular inflammation and addressing significant arterial stenosis:

1. Dual antiplatelet therapy, to reduce the risk of thrombotic events associated with vascular inflammation and luminal narrowing
2. Statin therapy, for lipid lowering and potential pleiotropic anti-inflammatory and endothelial-stabilizing effects
3. Glucocorticoid therapy, as the cornerstone of treatment to suppress vascular inflammation and prevent disease progression
4. Invasive interventions to address critical arterial stenosis, including carotid endarterectomy or balloon angioplasty, in selected patients with hemodynamically significant lesions

Conclusion

This case illustrates a rare presentation of giant cell arteritis discovered incidentally in a carotid plaque removed during endarterectomy for symptomatic ICA stenosis. The findings reinforce the importance of routine histopathological evaluation of CEA specimens and remind clinicians to consider vasculitic etiologies in atypical carotid pathology. Early identification of GCA enables timely initiation of corticosteroids, reducing the risk of recurrent vascular events and systemic complications.

Future Implications

This case emphasizes the need to consider large-vessel giant cell arteritis as a potential etiology in patients with ischemic stroke and carotid artery disease, particularly when clinical or radiological features are atypical for atherosclerosis alone. Routine histopathological examination of carotid endarterectomy specimens may uncover clinically significant inflammatory vasculopathies that directly influence long-term management. Increased awareness and multidisciplinary evaluation can facilitate earlier diagnosis and timely initiation of immunosuppressive therapy, potentially reducing the risk of recurrent cerebrovascular events and other systemic complications.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Data Availability Statement

The data may be made available from the corresponding author upon reasonable request.

Ethical Statement

The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations, and therefore, was exempt.

Informed Consent Statement

Written informed consent was obtained from the patient's parents/guardian for publication of this case report and accompanying images

Authors' Contributions

All authors contributed equally to this paper.

References

1. Salvarani C, Cantini F, Boiardi L. Polymyalgia rheumatica and giant-cell arteritis. N Engl J Med. 2002;347:261-71.
2. Hashami L, Haj Mohamad Ebrahim Ketabforoush A, Nirouei M. Giant cell arteritis with rare manifestations of stroke and internal carotid artery dissection: A case study. Clin Case Rep. 2022;10:e05597.
3. Wiszniewska M. Giant cell arteritis as a cause of first-ever stroke. Cerebrovasc Dis. 2007;24(2-3):226-30.
4. DaCosta M. Carotid endarterectomy. In: StatPearls. Treasure Island (FL): StatPearls Publishing. 2023.
5. Takatsuki K, Kojima Y, Ikeuchi Y, Kitayama J, Tanaka A, Inoue Y. Intracranial vascular stenosis in giant cell arteritis successfully treated by two balloon angioplasty procedures. Mod Rheumatol Case Rep. 2022;7:rxac080.
6. Conticini E. Giant cell arteritis presenting as a stroke in the internal carotid artery territory: A case-based review. Reumatologia. 2021;59(2):121-5.
7. Alsolaimani R. Severe intracranial involvement in giant cell arteritis: 5 cases and literature review. J Rheumatol. 2016;43(3):648-56.
8. Nesher G, Berkun Y, Mates M, Baras M, Rubinow A, Sonnenblick M. Risk factors for cranial and large-vessel complications in giant cell arteritis. Medicine (Baltimore). 2004;83(5):262-8.
9. Singh AG, Kermani TA, Crowson CS, Weyand CM, Matteson EL, Warrington KJ. Visual manifestations in giant cell arteritis: Trend over 5 decades in a population-based cohort. J Rheumatol. 2015;42(2):309-15.
10. de Boysson H, Liozon E, Larivière D, Samson M, Parienti JJ, Girot M, et al. Stroke in giant-cell arteritis: A population-based study. J Neurol Neurosurg Psychiatry. 2015;86(2):216-21.
11. Kermani TA, Warrington KJ, Crowson CS. Large-vessel involvement in giant cell arteritis: A population-based cohort study. Ann Rheum Dis. 2013;72(12):1989-94.
12. Maleszewski JJ, Younge BR, Fritzlen JT, Bower TC, Hunder GG, Goronzy J, et al. Clinical and pathological evolution of giant cell arteritis: a prospective cohort study. Arthritis Rheumatol. 2017;69(12):2350-60.
13. Sveen KA, Sundholm JK, Berntsen GK, Myklebust G, Diamantopoulos AP. Diagnostic delay in giant cell arteritis and the risk of vision loss: A population-based study. Rheumatology (Oxford). 2019;58(9):1653-61.
14. Mallik S, Nandish S, Das S, Khandelwal N, Lal V. Giant cell arteritis presenting as ischemic stroke: Report of three cases and review of literature. Neurol India. 2019;67(2):482-6.
15. Hunder GG. Giant cell arteritis and polymyalgia rheumatica. Med Clin North Am. 1997;81(1):195-219.
16. Sarkar S, Banerjee S, Banerjee TK. Ischemic stroke in giant cell arteritis: Angiographic features and review of literature. Neurol India. 2015;63(4):512-6.
17. Falkenberg M, Kral F, Lehmann P, Niesen WD, Weimar C, Weber R. Carotid endarterectomy specimen revealing giant cell arteritis: Case report and review of literature. Vascular. 2017;25(6):651-4.