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Case Report

From transient ischemic attack to peripheral vascular disease: a multifactorial case requiring steroid therapy

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ABSTRACT

A 70-year-old male from Himachal Pradesh, India, initially presented with two episodes of transient loss of consciousness and persistent temporal headache. He was diagnosed with transient ischemic attack (TIA) and temporal arteritis, which responded well to steroid therapy. One year later, he experienced weight loss and developed axillary lymphadenopathy (LAD), with histopathological and CBNAAT confirmation of extrapulmonary tuberculosis (TB). He underwent a six-month course of anti-tubercular treatment (ATT). However, six months' post-treatment, he developed vascular claudication of the left lower limb and severe left iliac region pain, which dramatically responded to steroid therapy. Computed tomography (CT) angiography revealed peripheral vascular disease (PVD), and CT enterography showed abdominal LAD, leading to the re-initiation of ATT alongside steroids. This case underscores the importance of steroids in managing complex, multifactorial conditions and highlights the necessity of a multidisciplinary approach for optimal patient outcomes.

Keywords: Temporal arteritis, Tuberculosis, Peripheral vascular disease, Steroids, Multisystem involvement

INTRODUCTION

Temporal (giant cell) arteritis is an inflammatory condition affecting small- and medium-sized blood vessels, primarily outside the brain. This disease can lead to serious complications such as reduced blood flow to the eyes, inflammation of the aorta that may progress to dissection, and impaired circulation to the limbs. Given the potential for severe organ damage, especially vision loss, it is considered a medical emergency. Symptoms can be vague, but a tender temporal artery is a key clinical finding that warrants a biopsy for confirmation. However, treatment with high-dose corticosteroids should begin immediately if the condition is suspected, rather than waiting for biopsy results. Once diagnosed, steroids should be tapered gradually over a period of at least a year.[1]

Temporal arteritis and tuberculosis (TB) are distinct clinical entities with different etiopathogeneses. Diagnosing temporal arteritis and tuberculosis (TB) in elderly patients can be particularly challenging due to their overlapping symptoms. Both conditions can present with headaches, visual disturbances, systemic symptoms, and elevated inflammatory markers, making differentiation difficult. TB diagnosis is further complicated by its broad and nonspecific clinical features, contributing to a significant proportion of fever of unknown origin (FUO) cases in older adults. Steroids play a pivotal role in managing inflammatory conditions like arteritis, while anti-tubercular treatment (ATT) remains the cornerstone for TB treatment.[2]

This case highlights the importance of recognizing recurrent vasculopathy in the setting of TB and emphasizes

the need for a comprehensive, multidisciplinary management approach.

CASE REPORT

Patient profile

A 70-year-old male from Himachal Pradesh, India, presented with multiple health complications at DR. RPGMC Kangra at Tanda over a span of two years, leading to a complex diagnostic and therapeutic course.

The patient initially presented with two episodes of loss of consciousness, accompanied by a severe headache localized to the temporal region and associated eye pain. A non-contrast computed tomography (CT) (NCCT) scan of the head revealed no abnormalities; however, laboratory investigations showed elevated inflammatory markers, including a C-reactive protein (CRP) level of 7 mg/l and an erythrocyte sedimentation rate (ESR) of 80 mm/hour. Based on the clinical presentation and laboratory findings, a diagnosis of transient ischemic attack (TIA) with temporal arteritis was made. The patient was started on high-dose corticosteroid therapy with prednisone at a dose of 60 mg per day, leading to significant improvement, with complete resolution of the headache within two days.

Six months later, the patient developed an enlarged right axillary lymph node, raising concerns about a possible underlying pathology. Initial investigations, including fine-needle aspiration cytology (FNAC) and a chest X-ray, were normal; however, persistent pus formation prompted further evaluation. An axillary biopsy and cartridge-based nucleic acid amplification test (CBNAAT) confirmed a diagnosis of tuberculosis, and the patient was subsequently started on ATT.

One year after the initial presentation, the patient reported left leg pain with an absent palpable pulse, suggesting peripheral vascular disease (PVD). A diagnosis of PVD was made based on clinical assessment, and cilostazol was prescribed for symptom management; however, the medication had to be discontinued due to the onset of tachycardia.

Subsequently, the patient developed acute pain in the right iliac fossa, radiating to the scrotum and back, which was unresponsive to standard analgesics. In the emergency setting, intravenous dexamethasone (4 mg) and hydrocortisone (100 mg) were administered, resulting in immediate pain relief.

Further diagnostic imaging, including an abdominal ultrasound, revealed a necrotic lymph node (Figure 1), and CT enterography was suggestive of abdominal tuberculosis. The patient was started on high-dose steroids for one month, followed by a taper to low-dose steroids in combination with ATT for long-term management.

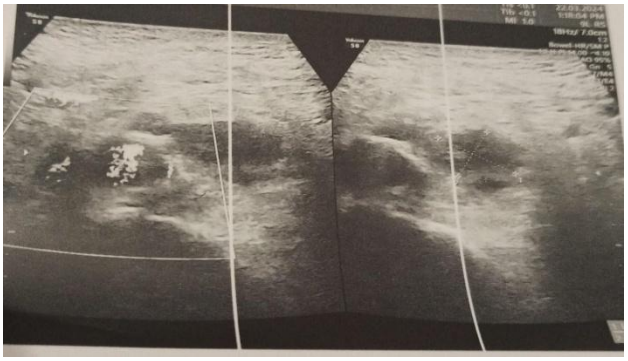

Figure 1: USG abdomen showing intra-abdominal necrotic lymph node.

CT enterography

Report

There is e/o heterogeneously enhancing, centrally necrotic lymph nodal mass seen in para-aortic region (3.5×2.6 cm) at L4 level and along the left external iliac artery (3.2×3.5 cm) with surrounding fat stranding. There is e/o pulled up cecum. Rest of the gut loops are sub-optimally distended however appears grossly normal. Illeal loops are sub optimally distended however IC junction appears unremarkable. Liver measures 14.2 cm, normal in size, shape, CT attenuation and contrast enhancement. Gall bladder is distended and appears unremarkable. GB wall is normal. PV is normal. CBD is normal. Pancreas normal in size and CT attenuation. MPD is not dilated. Spleen measures 11.5×3.8×12 cm enlarged in size with splenic index of 526, CT attenuation and contras enhancement. B/L kidneys are normal in size, outline and post contrast enhancement, B/L PCS are compact, B/L ureters are normal. Urinary bladder is distended grossly unremarkable. Prostate measures 3.8×3.4×3.7 cm with volume up to 20 cc. No ascites and peritoneal thickening seen. There is minimal b/l pleural effusion. Scanned spine shows degenerative changes in the form of anterior and posterior marginal osteophyt. There is e/o calcified and soft plaques in abdominal aorta and its branches.

Impression

F/u/c/o tuberculosis with present scan showing heterogeneously enhancing, centrally necrotic lymph nodal mass in para-aortic region and along left external iliac artery (3.2×3.5 cm) with surrounding fat stranding and pulled up cecum as described above abdominal tuberculosis, and adv clinical correlation.

DISCUSSION

This case highlights the complexity of diagnosing and managing overlapping vascular and infectious diseases, particularly in elderly patients from endemic regions like Himachal Pradesh, India. The patient's initial presentation with transient ischemic attacks (TIAs) and a temporal

headache, coupled with a favourable response to corticosteroids, strongly pointed toward temporal arteritis. It is a large-vessel vasculitis predominantly affecting individuals over the age of 50, often presenting with headache, jaw claudication, visual disturbances, and elevated inflammatory markers.[3]

The later development of axillary lymphadenopathy (LAD) and histopathologically confirmed extrapulmonary tuberculosis (EPTB) raises the possibility of concurrent or sequential disease processes. TB, especially in its extrapulmonary form, can mimic systemic vasculitis both clinically and radiologically, complicating diagnosis.[4]

The emergence of EPTB in a patient already on immunosuppressive therapy underscores the risk of opportunistic infections associated with corticosteroid use, especially in TB-endemic regions.[5]

The recurrence of symptoms after completion of ATT—this time with left lower limb claudication and iliac pain—and a dramatic response to steroids suggest a relapse or persistence of a vasculitis. CT angiography revealing PVD and CT enterography indicating abdominal lymphadenopathy again raise the possibility of immune-mediated vasculopathy exacerbated or unmasked by TB or its treatment.[6]

This clinical trajectory required re-initiation of ATT alongside corticosteroids, reflecting the delicate balance between infection control and immunosuppression. The patient's repeated improvement on steroid therapy supports the role of inflammation-driven vascular pathology, but the reactivation of TB remains a critical concern. Therefore, multidisciplinary care, involving infectious disease specialists, rheumatologists, and vascular surgeons, is vital in managing such multifactorial presentations.

CONCLUSION

This case underscores the diagnostic and therapeutic challenges in elderly patients presenting with overlapping symptoms of vasculitis and tuberculosis. In TB-endemic regions, the risk of misdiagnosis is heightened due to the protean manifestations of TB and the similarities it shares with autoimmune vasculitis. The patient's repeated responsiveness to steroids illustrates the pivotal role of inflammation in symptom generation, even in the presence of infection. It also highlights the importance of a multidisciplinary approach to differentiate and co-manage infectious and autoimmune aetiologies. Physicians should maintain a high index of suspicion for TB reactivation in patients receiving immunosuppressive therapy and be prepared for integrated treatment strategies that address both infection and inflammation.

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REFERENCES

1. Lee AW, Chen C, Cugati S. Temporal arteritis. Neurol Clin Pract. 2014;4(2):106-13.
2. Zhang Y, Wang D, Yin Y, Wang Y, Fan H, Zhang W, et al. Tuberculosis infection in Chinese patients with giant cell arteritis. Sci Rep. 2019;9(1):14364.
3. Weyand CM, Goronzy JJ. Giant-cell arteritis and polymyalgia rheumatica. N Engl J Med. 2014;371(1):50-7.
4. Sharma SK, Mohan A. Extrapulmonary tuberculosis. Indian J Med Res. 2004;120(4):316.
5. Jick SS, Lieberman ES, Rahman MU, Choi HK. Glucocorticoid use, other associated factors, and the risk of tuberculosis. J Rheumatol. 2006;55(1):19-26.
6. Mahr A, Guillevin L, Poissonnet M, Aymé S. Prevalences of polyarteritis nodosa, microscopic polyangiitis, Wegener's granulomatosis, and Churg-Strauss syndrome in a French urban multiethnic population in 2000: a capture–recapture estimate. Arthritis Care Res. 2004;51(1):92-9.

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