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Editorial

Giant Cell Arteritis: A Clinical Challenge

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Giant cell arteritis (GCA) is a systemic disease, characterized by granulomatous inflammation of the large and medium sized arteries. It almost exclusively affects older than 50 years and is part of the spectrum including polymyalgia rheumatica. The incidence of GCA increases with the age. Women are affected more commonly than men. It can lead to profound permanent vision loss, if not detected early due to mainly arteritic anterior ischaemic optic neuropathy. The vision loss can occur within hours to days. If left untreated, around 50% of patients with GCA and vision loss in one eye will lose vision in the opposite eye within 7 days. Treatment is required even if the patient loses vision in the both the eyes, as GCA is a systemic disease.

It can affect any branch of external carotid artery and the clinical features will depend on it. Clinical features of GCA may include headache, tenderness of the temporal artery or the scalp, jaw or tongue claudication, ocular pain, ear pain, neck pain. Headache is most common symptom of GCA, and it is very non-specific type. So, any elderly patient with pain in the distribution of external carotid artery should be considered as GCA, until otherwise proved. Jaw or tongue claudication is very specific for GCA. However, there are many other signs and symptoms associated with GCA like, posterior ischemic optic neuropathy, central retinal artery occlusion, cilioretinal artery occlusion, pyrexia of unknown origin, transient vision loss, transient diplopia, cranial nerve palsies. Constitutional symptoms as anorexia, weight loss are also common [1]. So, there is wide spectrum of manifestations. However, approximately 25% of the patients with vision loss from GCA do not have any systemic manifestations; known as occult GCA. Patients with occult GCA need high index of suspicion. Diagnosis is mainly clinical.

If GCA involves temporal artery, it is also known as temporal arteritis. In such a case, the temporal artery becomes thickened, tender and there is decreased or absent pulse. The temporal artery biopsy (TAB) can be done for diagnosing GCA. TAB should be performed within 10-14 days of start of corticosteroids. The sample should be taken first from the side and site of temporal artery tenderness. Always take at least 2 cm sample of the temporal artery for pathological examination, because the lesion of GCA shows skip lesions. The histopathology will show monocular inflammation with the giant cells, loss of internal elastic lamina. It is important to know that, presence of giant cells is not required for the diagnosis. Results of the unilateral involved TAB can be falsely negative (approximately 9-13% cases) due to inadequate sample leading to only skip area sample, long use of steroids before sampling. In case of negative report of the biopsy, and still there is high index of the suspicion, go for contralateral TAB.

GCA patients will show raised C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) even up to Westergren 70-100 mm/hour or greater. However, there can be normal values also in some cases, and a normal CRP and ESR do not rule out GCA [2,3]. There is associated thrombocytosis.

Vascular imaging of the chest and abdomen should also be done to rule out associated aortitis, aortic dissection and other large vessel involvement [4,5].

GCA is a very aggressive disease. It needs quick diagnosis and prompt management with high dose of the steroids. We should not wait for the diagnosis to be made by biopsy and then only start treatment. The protocol adopted should be to treat and diagnose, instead of to diagnose and then treat.

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