

Case report

TYPHOID AFTERMATH: PRESENTING AS VASCULITIS, NEURORETINITIS AND MACULAR NEUROSENSORY DETACHMENT

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ABSTRACT

Systemic immune-mediated reactions are known to occur following typhoid illness. Vasculitis, neuroretinitis and macular neurosensory detachment are amongst the rarely documented aftermath of typhoid fever. A 32-year-old male came with complaints of decreased vision in both eyes with history of typhoid fever (treated adequately 4 weeks prior and declared cured 2 weeks prior to ocular manifestations) who was found to have vasculitis, neuroretinitis and neurosensory detachment at macula. His BCVA was 1/60 in right eye and 6/12 in left eye. The inflammation completely resolved and there was marked improvement in the visual acuity, 6/12 in right eye and 6/6 in left eye after treatment with oral steroids. Immune-mediated vasculitis and neuroretinitis, the ocular aftermath of typhoid fever responded well to oral steroids

Keywords: Neuroretinitis, Immune-mediated neuroretinitis

INTRODUCTION

Salmonella typhi causes typhoid infection. It can give gastroenteritis rise to fever. and septicemia. Salmonella rarely affects ocular tissue^[1]. This may be direct infection or due to an immunemediated mechanism. Ocular complications of typhoid fever include iritis, choroiditis, retinal hemorrhage, panophthalmitis and endophthalmitis as reported by Hersing and Duke-Elders. After complete resolution of typhoid fever, there are documented of endophthalmitis^[2,3]. Here evidences we are patient presented who reporting a with neuroretinitis and had typhoid fever 4weeks prior to presentation

CASE REPORT

A 32-year-old Indian male presented to Dr Agarwal's Eye Hospital, Salem with sudden, painless diminution of vision in both the eyes (right eye > left eye) for 2 weeks. He gave a past history of typhoid fever 4

weeks ago. Lab investigations during the fever showed positive Widal test with high 'O' antigen (1:160) and `H' antigen (1:40) titres, while `AH' and 'BH' antigens were non-reactive. At the onset fever, treatment was initiated with oral ofloxacin 400 mg twice daily for 14 days; following which, the fever resolved. He started experiencing diminution of vision 2 weeks after the completion of treatment. At presentation, his best corrected visual acuity was 1/60, in the right eye and 6/12 in the left eye. On examination with slit lamp, anterior segments of both eyes were within normal limits except for relative afferent pupillary defect in the right eye. Colour vision was defective in right eye. On fundus examination media was clear, mild disc pallor was noted. Vasculitis with multiple areas of deep neuroretinitis was seen. Macular neurosensory detachment was evident on OCT scans (Figure 1). The left eye (Figure 1) had clear media, normal disc

and macular oedema, and one patch of neuroretinitis. Lab tests for HIV.

TB, syphilis, connective tissue disorders, rheumatoid arthritis and SLE were negative. Diagnoses of immune mediated vasculitis, neuroretinitis and macular neurosensory detachment secondary to typhoid infection were made. After consulting with a physician he was started on prednisolone 60mg/kg/day. Steroids were tapered over 2 months with regular monitoring of health status and ocular response. He had markedly recovered after 2month of treatment & his visual acuity was 6/12 in right eye and 6/6 in left eye. At 6 months follow-up (Figure 2) all the retinal lesions had completely resolved with pigmentary changes in the macula and mild pallor of the optic disc in the right eye. OCT at 6 months revealed retinal pigment epithelium changes and thinning of inner retinal layers over the lesions, in addition to complete resolution of macular neurosensory detachment (Figure2).





DISCUSSION

This is a case of immune-mediated retinitis secondary to typhoid fever. There can be a viral etiology which cannot be neglected. The lesions in this patient were posterior to the equator, did not show centrifugal or circumferential extension, and did not affect the arterioles, not associated with aqueous flare or cells or vitritis. Taking into consideration the time of onset of ocular complaints and onset of fever, clinical improvement with oral steroids without the use of antiviral medications, the most likely diagnosis was immune mediated vasculitis, neuroretinitis and macular neurosensory detachment as a aftermath of typhoid infection. There is not much regarding this type of pathology secondary to typhoid fever in the literature ^[4-6]. Reports of similar cases^[5] assume that the origin of this kind of pathology is due to retinal infiltration. Pathogenesis of immune-mediated vasculitis secondary to typhoid infection could be because of immunologic effects which can give rise to an immune response, affecting self-antigens by reacting with them due to homology or molecular mimicry giving rise to autoimmunity^[7]. Immunemediated vasculitis is a clinical diagnosis most often when there is past history of infection few weeks or days prior to the onset of ocular manifestations. Lab work up can help to find out the exact etiology many a times. Malaria, dengue fever, Chickungunya fever and other viral infections may lead to immunemediated vasculitis. Some non-infectious etiologies can be Behcet's syndrome and intraocular lymphoma may also manifest as immune-mediated vasculitis^[8]. Due to paucity of published literature on such rare clinical scenarios, management remains controversial. Mild cases may resolve spontaneously without treatment, but severe cases may be treated with oral corticosteroids, if these are not contraindicated. In this case, treatment with oral steroids was initiated due to inflammation of the vessels, macula and disc which had caused profound diminution of vision.

CONCLUSION

Though these cases are rare, an ophthalmologist may encounter such cases which are immune-mediated neuoretinitis, non-infectious neuroretinitis, vasculitis with or without macular neurosensory detachment after complete resolution of typhoid fever that may be effectively treated with a course of oral steroids **Conflict of interest:** The authors declare that they have no competing interests (financial or non-financial).

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