



UNILATERAL OPTIC NEURITIS FOLLOWING TYPHOID FEVER

Ophthalmology

Mousumi Malakar

Kiran Bhanot*

*Corresponding Author

ABSTRACT

Background: Post typhoid fever immune related reactions affecting the eye is a rare finding which can have various presentations in which optic neuropathy is rare sequelae.

Case presentation: we present a case of 60 year old female who presented with left eye sudden painless loss of vision 4 weeks after typhoid fever which was diagnosed significantly high Widal titres and treated successfully. Her BCVA was 6/6 in right eye and 3/60 in left eye. Fundus examination showed disc edema with blurred disc margin. Significant improvement in BCVA in left eye was observed after treatment with oral steroid. Diagnosis of post typhoid immune mediated optic neuritis was made with good resolution following treatment.

Conclusions: Immune mediated optic neuritis is a rare sequelae to typhoid infection, Even though a less documented entity in literature, an ophthalmologist may encounter such cases which can be successfully treated with systemic steroids.

KEYWORDS

typhoid fever, optic neuritis, steroid

INTRODUCTION

Typhoid or enteric fever is a systemic disease which is characterized by fever and abdominal pain caused due to dissemination of Salmonella typhi or paratyphi. Ocular manifestations of typhoid fever are rare and includes conjunctival congestion, uveitis, episcleritis, neuroretinitis, retinitis, and dacryoadenitis.¹

Posterior segment involvement can be seen in form of retinitis, optic disc involvement and subsequent macular involvement. Neuroretinitis is a particular form of optic neuropathy characterised by acute unilateral visual loss in the setting of optic disc swelling and hard exudate arranged in a star figure around the fovea.² From the ocular standpoint conjunctivitis, metastatic uveitis, optic neuritis, retinal haemorrhages, accommodation paralysis, tenonitis, orbital cellulitis and even panophthalmitis have been mentioned as the complications of the disease (Agarwal, 1963; Ballantyne and Michaelson, 1963; Duke Elder, 1964; Duke Elder, 1967; Schupfer, 1967; Sorsby, 1968)

Here we are presenting a case of optic neuritis developing after typhoid fever successfully treated with systemic steroids.

CASE REPORT

A 60 year old female presented to our hospital 1 week after experiencing diminution of vision in the left eye. She gave a history of typhoid fever 4 weeks prior to presentation for which Widal test was performed to confirm diagnosis. The test results showed significant titres for 'O' antigen (1:160) and 'H' antigen (1:180). He was subsequently started on oral Ofloxacin 400 mg twice daily for 2 weeks following which fever subsided. There was no known history of diabetes mellitus or hypertension. On ocular examination her best corrected visual acuity was 6/6 in the right eye and 6/60 in left eye. On examination with slit lamp Anterior segment findings were unremarkable with IOP being within normal range for both eyes except early lenticular changes in left eye. Fundus examination of left eye showed hyperemic disc with blur disc margins and tortuous blood vessels suggestive of optic neuritis (Fig. 1a). Right eye had normal disc and normal macula. MR Imaging reveals no significant intraorbital abnormality.

Lab test like complete blood count with ESR was within normal limit. HIV, VDRL, RA factor were negative. After Physician consultation treatment with oral steroid was initiated (prednisolone 1g/kg body weight). Steroid were tapered over 2 months along with monitoring of systemic and ocular health. Patient came for follow up every week for 2 months. Every visit vision and fundus photo was documented (fig2a& fig2b). After 2 months of initiating treatment there was an improvement in the BCVA in left eye to 6/18 which was maintained on further visits.



Fig 1a



Fig 2a



Fig 2b

DISCUSSION

Typhoid fever is an acute infectious disease capable of producing toxic and degenerative changes in the tissues throughout the body. Therefore involvement of eye is not surprising in this diseases³

Patients can present with Neuroretinitis like picture with optic disc edema and macular hard exudates⁴. The macular star becomes prominent over first 3 weeks with neuroretinitis resolving over 6-8 weeks⁵. Leakage from the optic nerve head can lead to retinal swelling, exudation and edema, whereas retinal venous occlusion due to

vasculitis results in intraretinal haemorrhage, cotton wool spots and retinal and optic nerve head edema⁶. It was postulated that microbial pathogens may be responsible for immune mediated ocular and systemic pathology through postinfectious immunological effects.

These may be due to molecular mimicry eliciting an immune response that cross react with self antigens. Even though active infection is an unusual cause of retinal vasculitis, it is possible that many idiopathic and systemic disease associated cases are precipitated by previous encounters with microbes bearing DNA sequence homologous to retinal and vascular autoantigens.⁷

Similar case reports by Relhan et al.⁸ and Laul et al.⁹ showed immune mediated response post typhoid fever presenting with neuroretinitis, vasculitis and macular detachment. Successful treatment with steroids was seen in them.

There is not much regarding this type of pathology secondary to typhoid fever in the literature. Reports of similar cases assume that the origin of this kind of pathology is due to retinal infiltration.

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.

Immune mediated optic neuritis is a rare sequelae to typhoid infection, Even though a less documented entity in literature, an ophthalmologist may encounter such cases which can be successfully treated with systemic steroids.

REFERENCES

1. Vishwanath S, Badami K, Sriprakash KS, Sujatha BL, Shashidhar SD, Shilpa YD. Post-fever retinitis: a single center experience from south India. *International ophthalmology*. 2014;34(4):851-7.
2. Maitland CG, Miller NR. Neuroretinitis. *Arch Ophthalmol*. 1984;102(8):1146-50.
3. S. Kujur et al. Varied ocular complications of enteric fever: A case series. *International journal of basic and applied medical sciences*; 2016 Vol 6(2)86-88
4. Jacobs DA, Guercio JR, Balcer LJ. Inflammatory optic neuropathies and neuroretinitis. In: Yanoff M, Duker JS, editors. *Ophthalmology*. 4th ed. London: Elsevier Saunders; 2014. p. 879–83.
5. Houghton OM, Brown GC, Brown MM. Coexistent optic nerve and macular abnormalities. In: Yanoff M, Duker JS, editors. *Ophthalmology*. 4th ed. London: Elsevier Saunders; 2014. p. 632–7.
6. Hughes EH, Dick AD. The pathology and pathogenesis of retinal vasculitis. *Neuropathol Appl Neurobiol*. 2003;29:325–40.
7. prabhushanker et al. *Int J Retin Vitreol* (2017) 3:11
8. Relhan N, Pathengay A, Albini T, Priya K, Jalali S, Flynn HW, et al. A case of vasculitis, retinitis and macular neurosensory detachment presenting post typhoid fever. *J Ophthalmic Inflamm Infect*. 2014;4:23.
9. Laul R, Atif Ali MIR, Shafi S. Typhoid aftermath: presenting as vasculitis, neuroretinitis and macular neurosensory detachment. *Int J Med Res Health Sci*. 2015;4:737–9.