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OCULAR TUBERCULOSIS PRESENTATION IN NORTH INDIA AND ITS CURRENT PERSPECTIVES.

Ophthalmology

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ABSTRACT

Ocular tuberculosis is a form of extra pulmonary infection, with a myriad form of presentations the most common of which is infectious uveitis and panuveitis. It's quite difficult to come to a definite diagnosis masking its true prevalence. This was a prospective case series study based in a tertiary care hospital. Study was done at the Department of Ophthalmology, Vardhaman Mahavir Medical College and Safdarjung Hospital New Delhi, between January 2018 to February 2019. Total of 50 patients with active uveitis and a positive mantoux test were recruited for this study. Demographic data was collected, all the patients were started on antitubercular drug therapy and treatment response monitored in regular follow-up at 2 weeks, 4 weeks and 12 weeks in terms of improvement of visual acuity and subsidence of inflammation of the eye. The mean age was 39.75 years. The male to female ratio was 1.3:1. The incidence was higher in patients with history of contact with tuberculosis. The most common complaint was defective vision (64%) followed by floaters. Recurrence was seen in 12%. The most common presentation was choroiditis (37%) followed by choroidal tubercles (23%). The mean best corrected visual acuity (BCVA) at presentation was 0.82 which improved to 0.12 at 12 weeks follow-up.

Ocular tuberculosis is a great mimicker of intraocular inflammation and must be initially considered as the cause especially in endemic areas like South-East Asia and ATT should be started early along with steroids to reduce long term ocular morbidity.

KEYWORDS

Ocular Tuberculosis, Uveitis, Logmar Scale, Visual Acuity

INTRODUCTION

The incidence of ocular involvement which has been recorded varies considerably and it depends on the population being examined and the criteria which has been used for diagnosis. Tuberculosis affects the lungs in 80% of the patients, with 20% occurring in other organs including the eye[1].

Ocular tuberculosis is usually not associated with clinical evidence of pulmonary tuberculosis, and upto 60% of extra-pulmonary tuberculosis patients may not present with pulmonary disease[2]. In most of the cases of tuberculous uveitis the diagnosis is only presumptive. It is a great mimicker of various types of uveitis and is kept in the differential diagnosis of any type of intraocular inflammation[3]. Tuberculous uveitis is a vision-threatening disease that inevitably leads to blindness if not properly diagnosed and treated. The aim of this study is to illustrate the various types of tubercular uveitis, their incidence, clinical features and how they affect visual acuity and their management.

Tuberculosis (TB), a multisystem infectious disease caused by Mycobacterium tuberculosis (MTB). Mycobacterium tuberculosis is a slow-growing (with a doubling time of 15-20 hours while most bacteria have 1hour or less), obligate aerobe, facultative intracellular, non-sporing, non-motile, acid-fast bacilli. Acid-fastness is ascribed to the presence of unsaponifiable wax, mycolic acid, which gives integrity to the cell, protecting against desiccation and is a key virulence factor[4]. TB bacilli are resistant to dry environment and remain viable in expectorated sputum for several weeks and is an important factor in the transmission of infection. Humans are the only natural reservoir and infection is mainly by airborne respiratory aerosol.

There are several different mechanisms through which the eye can become infected with tuberculosis:

1. The most common form of ocular involvement is from hematogenous spread. The uveal tract (iris, ciliary body, and choroid) is the coat of eyeball most frequently involved, presumably because of its high vascular content.
2. Primary exogenous infection of the eye is unusual but can occur in the lids or conjunctiva. The cornea, sclera and lacrimal sac may be affected less commonly.
3. Direct extension from surrounding tissues or by contamination with the patient's own sputum may lead to secondary infection of the eye.
4. Other forms of ocular tuberculosis, such as phlyctenular disease and Eales' disease, are mostly the result of hypersensitivity reaction[5].

Tuberculosis (TB) is caused by Mycobacterium tuberculosis, and can affect multiple organs throughout the body, including the eye. The term “ocular TB” the term which is used to describe an infection caused by the M. tuberculosis species affecting any part of the eye (intraocular, superficial, or adnexal), with or without systemic involvement. Secondary ocular TB is defined as ocular involvement as a result of spread by hematogenous route from a distant site or direct invasion by contiguous spread from surrounding structures, like the sinuses or the cranial cavity. More than 9 million new cases of TB are diagnosed each year with 95% being in in developing countries around one-third of world's population is latently infected with TB. The WHO statistics for 2015 gave an estimated incidence of 2.2 million cases of TB in India out of a global incidence of 9.6 million[6]. The estimated TB prevalence in India for the year 2015 was about 2.5 million cases[7]. The prevalence of ocular tuberculosis is highly variable, given the absence of definitive diagnostic guidelines. It ranges from 0.39 to 9.86% in South and North India respectively. India has reported the largest number of tuberculous uveitis cases in the world. In India the current prevalence of ocular truberculosis is 0.4-9.8%[8].

The incidence of ocular involvement has been reported varies considerably, depending upon the criteria used for diagnosis and the population sampled. In 1890, two cases of tuberculous iritis were reported by Terson[9] in a population of 30,000 patients with ocular disease. In patients with known systemic tuberculosis, the incidence of ocular involvement is, as expected, much higher[10,11]. In India the incidence of ocular tuberculosis is reported as 1.39% in patients with pulmonary and extra-pulmonary tuberculosis[12,13]. Immunocompetent individuals with latent tuberculosis infection (LTBI) run a 5-10% lifetime risk of developing systemic reactivation when their immune system is compromised, majority developing TB disease within five years of initial infection. High risk groups include immigrants from endemic areas, health care professionals, indigent and immunocompromised patients (HIV infected, chronic disease, immunomodulator therapy).

The aim of this study is to show the different clinical manifestations and treatment of a presumed case of tubercular uveitis.

MATERIAL AND METHODS

This was a prospective type of study carried out in a tertiary care hospital. The study was done in the period between January 2018 to February 2019. Ethical clearance was taken from the institutional ethical committee.

50 patients were recruited for the study who gave consent and fulfilled

the inclusion criteria. Patients between 15-70 years, with positive Mantoux test i.e. 10mm or more induration at 48-72 hours with or without specific lesion in chest radiograph, with evidence of active uveitis and positive contact history or known pulmonary or extrapulmonary tuberculosis were included in the study. Exclusion criteria – patient denying consent,, patients with infective uveitis other than tubercular uveitis and patient diagnosed with non-infectious uveitis.

Patients presenting to Uvea and Retina services and who fulfilled the above inclusion and exclusion criteria were registered, and after obtaining consent for participation in the study, were included. A detailed history regarding the symptoms - duration, course and recurrence, contact history and previous history of any form of tuberculosis treated or untreated with anti-tubercular therapy was noted.

Systemic examination included evaluation of cervical lymph nodes and respiratory system. Ocular examination included best corrected visual acuity (BCVA) (using Snellen's chart and converting to LogMar scale for statistical purpose), intra-ocular pressure (using Goldmann Applanation Tonometer), examination of anterior segment (using slit-lamp) and posterior segment (using +90D biomicroscopy). Fundus Fluorescein Angiography was done when required. Laboratory investigations included hemoglobin, total blood count, differential blood count, erythrocyte sedimentation rate, blood sugar, HIV-ELISA, VDRL, HLA-B27, Toxocara ELISA, TORCH serology tests, Tuberculin skin test and chest radiograph. Patients were referred to pulmonologist and were started on anti-tubercular therapy (ATT) along with steroids either in oral, topical, periocular or in all forms.

TREATMENT AND FOLLOW-UP

All patients, following chest physician opinion, were started on anti-tuberculosis therapy and corticosteroids. Choice of route of corticosteroids was made depending on the diagnosis. In patients with only posterior uveitis oral prednisolone 1mg/kg body weight was started initially and tapered according to response. In patients with intermediate uveitis periocular steroid injection of triamcinolone acetonide was given. In patients with anterior uveitis topical 1% prednisolone acetate suspension was advocated, along with cycloplegic 2% homatropine eye drops. Patients were followed up in 2 weeks, 4 weeks and 12 weeks duration. Response to treatment was assessed using improvement in BCVA and decrease in intraocular inflammation. Complications such as secondary glaucoma, complicated cataract, vitreous opacities, worsening of inflammation, choroidal neovascular membrane and vitreous hemorrhage were looked for.

STATISTICAL ANALYSIS:

The patients' data were entered in excel spread sheet and statistical analysis was done using SPSS 16.0 package. Statistical analysis included descriptive statistics, where mean and 95% confidence interval were calculated for continuous variables whereas percentages were calculated for categorical variables. A p value <0.001 was considered as statistically significant. Paired t-test was used to compare the continuous variable (mean visual outcome) before and after treatment at 2 weeks and 3 months.

RESULTS

In our study the maximum number of patients were seen in the age group of 35-44 years (36%). The minimum age of the patient was 18 years and maximum age was 67 years with a mean age of 39.75 years.

In this study, there were 21 male and 29 female patients. The female out-numbered the male with a ratio of 1.3:1. The incidence of ocular tuberculosis was higher in patients with exposure to or contact with tubercular patients (70%) followed by patients with previous extra-pulmonary tuberculosis (12%). 10% patients had miliary tuberculosis at the time of diagnosis of ocular tuberculosis. 8% patients were previously treated for pulmonary tuberculosis. Right eye was more frequently affected (38%) than the left eye (30%) and bilateral involvement was found in only 32% of the patients.

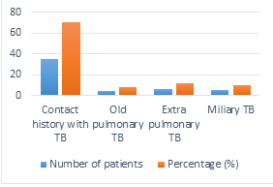

Figure 1: Risk of Ocular Tuberculosis

The most common complaint of the patients was defective vision (94%) followed by floaters in 28% of the patients.

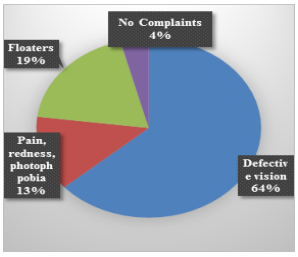

Figure 2: Frequency of various complaints in the study population.

In the present study, tubercular uveitis presented most commonly as posterior uveitis (54%) and least commonly as panuveitis (6%). Among the posterior uveitic cases, choroiditis was most common (37%) followed by choroidal tubercles (23%).

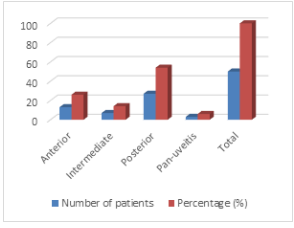

Figure 3: Different types of uveitis in the study population.

The recurrence rate was 12% mostly seen in posterior uveitic cases. Only two patients with anterior uveitis had a recurrence. No recurrence was seen in 38% cases , most of whom had completed their full course of antituberculous treatment along with a course of systemic steroids.

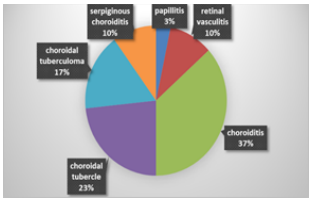

Figure 4: Types of posterior uveitis in the affected patients

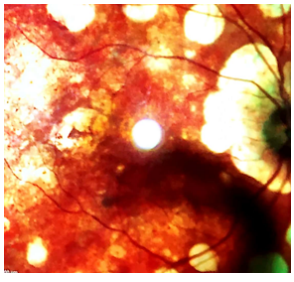

Figure 5: Inactive Choroiditis (atrophic chorioretinal patches) in right eye

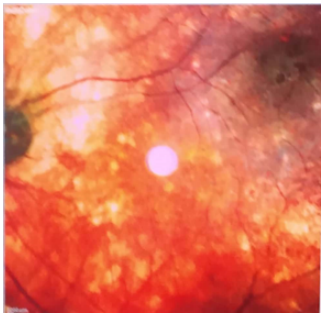

Figure 6: Choroidal tubercles seen in left eye of same patient

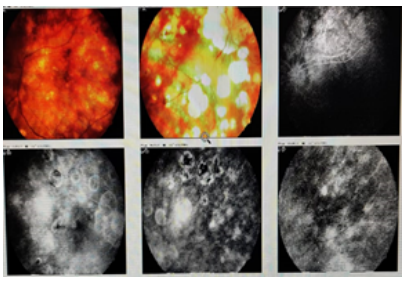

Figure 7: Fluorescein angiography of the same patient showing reactivation of choroiditis in left eye.

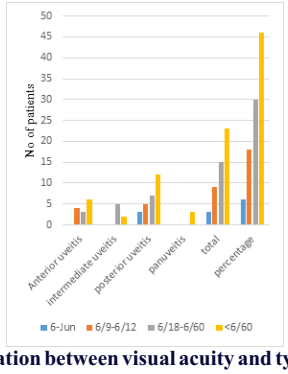

Figure 8: Correlation between visual acuity and type of uveitis

Poorer visual acuity (<6/60) at presentation was noted mostly in posterior uveitic cases and those presenting with anterior uveitis followed by panuveitic cases. On follow-up, improvement in visual acuity was noted following anti-tubercular treatment. In intermediate uveitis, the vision of the patients was not much affected.

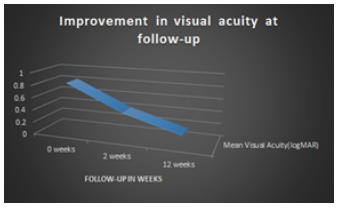

Figure 9: Trend of improvement in visual acuity with treatment.

There was a clear cut improvement in best corrected visual acuity (BCVA) with treatment at every follow-up visit. The mean BCVA (logMAR) at initial presentation in this study population was 0.82 which improved to 0.41 at 2 weeks and 0.15 at 12 weeks follow-up.

DISCUSSION

The intraocular tuberculosis is a great mimicker of various uveitis entities. It should be considered in the differential diagnosis of any type of intraocular inflammation. In ocular tuberculosis, inflammation may involve one or both the eyes, sometimes one eye getting involved months or years before the other. Uveitis in intraocular tuberculosis may present as anterior, intermediate, posterior, or panuveitis(no comma)[12,13].

Tubercular anterior uveitis, which is classically a chronic granulomatous disease presents with an insidious onset. The ocular manifestations seen are mostly those which are observed in other granulomatous disease such as mutton-fat keratic precipitates (collections of inflammatory cells and macrophages) on the posterior aspect of the cornea , Koeppes and Busaccas nodules on the iris and cells and flare in the aqueous[5,14]. In our study 26% cases presented with anterior uveitis which was treated with topical prednisolone acetate (1%) and homatropine. Nongranulomatous uveitis can also be seen in tuberculosis, which usually manifests as small white keratic precipitates present on the corneal endothelium and in absence of iris nodules[15]. Anterior uveitis is often accompanied by vitritis, and in course of time is complicated by the development of posterior synechiae and cataract[5,16]. In patients with HIV, the clinical manifestations and severity of tubercular uveitis depends on the CD4+ count.

In intermediate uveitis cells are seen in the vitreous along with formation of snowballs, and in severe cases snow banking with peripheral retinal vasculitis occurs in the pars plana region, sometimes with spilling of cells into the anterior segment causing a spill-over anterior uveitis. Intermediate uveitis can be unilateral or asymmetrically bilateral. Relapsing chronic forms should lead to a high degree of suspicion for tubercular etiology. In chronic cases, decrease in vision may be due to cystoid macular edema or epiretinal membrane formation and can also lead to secondary vitreous hemorrhage from traction predominantly in long standing cases.

The most common presentation of intraocular TB is posterior uveitis as seen in our study with choroiditis present in 37% of the cases. The lesions predominantly present in the choroid as focal, multifocal or serpiginous choroiditis. Solitary or multiple choroidal nodules (tubercles), choroidal granuloma (tuberculoma), neuroretinitis, subretinal abscess can also be seen in posterior tubercular uveitic cases as seen in our study. Endophthalmitis, panophthalmitis and ischemic retinal vasculitis may lead to proliferative vascular retinopathy with recurrent viterous haemorrahge, rubeosis iridis and neovascular glaucoma.[3,18-20]. In a study conducted by Gupta A and Gupta V[21] , in Indian population, comprising of 158 patients with presumed intraocular TB, 66 (42%) had posterior uveitis, 57 (36%) anterior uveitis, 18 (11%) panuveitis, and the remaining 17 (11%) had intermediate uveitis. In this study, posterior uveitis was seen in 54%, anterior uveitis in 26% followed by intermediate uveitis in 14% and panuveitis in 6% of cases.

Choroidal tuberculoma are large granulomatous infiltrative lesions of the choroid by the tubercle bacilli, presenting as sub-retinal mass varying in size from 4 – 14 mm. Clinically they appear as creamy yellow elevated lesions accompanied by minimal vitritis[22,23]. Studies have shown that choroidal granuloma have hypoxic center secreting vascular endothelial growth factor (VEGF), thus to the formation of retino-choroidal anastomosis[24] , a feature which favours the diagnosis but not pathognomonic of tuberculous etiology. Flourescein angiography in choroidal tuberculoma shows early hypofluorescence due to necrotic lesion with late hyperfluorescence due to leakage. Post treatment with ATT and steroids they heal leaving a chorioretinal atrophic scar. Choroidal granuloma should be followed up with FFA and OCT because they may develop choroidal neovascularization even after treatment.

Choroidal tubercle can present as unilateral or bilateral and single or multiple lesions frequently away from the posterior pole and as part of disseminated disease. They appear as orange-yellow lesion with indistinct margins and are not accompanied by inflammation. Patients are usually asymptomatic, since the lesions does not involve the macula. In our study also 23% cases showed presence of choroidal tubercules mostly seen away from the macular area and showed leakage on fluorescein angiography. The lesions are histologically similar to tubercular granuloma presenting anywhere else in the body. After ATT, the lesions faded completely leaving a flat hypopigmented chorioretinal scar. Paton had described the appearance of choroidal tubercles 4 days after admission, which were not observed on earlier examinations, suggesting the need for repeated fundus examinations[25].

Tubercular retinal vasculitis is due to hypersensitivity reaction to the tubercular bacilli, and presents more commonly as periphlebitis[26]. They can present with or without a chorioretinal lesion. The presence of an active or healed choroiditis patch is highly suggestive of tubercular etiology. Other causes such as Systemic Lupus Erythematosus, Sarcoidosis, Behcet's disease should be excluded even in areas endemic for tuberculosis. Tuberculous retinal vasculitis can present in three forms: as retinal vasculitis with overlying choroiditis, isolated retinal vasculitis or Eales's disease. It can cause neovascularization, non-resolving vitreous haemorrhage, epimacular membrane and tractional retinal detachment.

Miliary tuberculosis with hematogenous spread of tubercle bacilli infects all layers of the eye leading to panophthalmitis. They are seen in severely immunocompromised patients such as HIV infection[27]and present acutely with corneal infiltration and intense anterior chamber reaction with hypopyon, along with post segment manifestation. Occasionally, a large choroidal tuberculoma can burst into the vitreous producing endophthalmitis and can involve sclera leading to panophthalmitis.

A study conducted by Bansal et al[28] found the maximum number of uveitic cases in the age group of 34-45 years, which was similar to our

study (36%). According to Ang M et. al (2012) and Sanghavi et al (2011)study showed a slight female preponderance which was similar to our study (38%). In our study posterior uveitis was the most common presentation as seen in studies conducted by other researchers like Gupta et al.

In another study done over 50 patients presenting with multifocal choroiditis, treated with anti-tubercular therapy (ATT) and without simultaneous use of oral corticosteroids. All patients treated had a favourable response. There was no recurrence recorded.This response to therapy can be attributed to direct microbial invasion which lead to choroiditis.[3] .

Management of tubercular uveitis depends on the diagnosis of a tubercular etiology. Failure to start the patient on anti-tubercular therapy with immunosuppressive drugs not only causes recurrence of the disease but at times can be life-threatening causing a disseminated tuberculous disease. Ophthalmologists mainly face challenges in the diagnosis mainly because of lack of definite diagnostic guidelines, varied manifestation of the disease and limited ocular samples. The US FDA has recommended 9 months course of ATT in the treatment of extra-pulmonary tuberculosis[29]. The commonly used drugs are Isoniazid, Rifampicin, Pyrazinamide and Ethambutol. An intensive course of 2 months followed by maintenance course of 4-7 months duration is advocated. If there is no improvement after 2 months of intensive treatment, the patient has to be reassessed and alternate diagnosis or resistant forms of bacilli are to be suspected.

The main aim in tubercular uveitis is the reduction of inflammation. Steroids are given through different routes of administration, namely, topical, periocular and oral. Anti-tubercular drugs should be administered along with steroids since they reduce the antigenic microbial load and thus reduce the chances of recurrence. The use of oral steroids in patients with presumed tubercular uveitis is clearly a confounding issue[30]. Patients treated only with systemic corticosteroids showed worsening or recurrence of inflammation. Several studies reported a favorable response to ATT when administered concomitantly with systemic corticosteroids in patients with presumed tubercular uveitis[19,20]. Oral prednisolone used in posterior uveitis reduces macular edema[31]. It may be desirable diagnostically to delay the steroid treatment in order to assess the response to ATT. This must be balanced against the risk of loss of sight[32]. Secondary cataract in eyes with TB uveitis can be safely managed, after controlling of the inflammation, by phacoemulsification cataract surgery with intraocular lens implantation.

CONCLUSION

Tubercular etiology as a cause of uveitis should be kept in mind when patients present with clinical signs such as granulomatous uveitis, broad synechiae, tuberculoma, tubercles but what if a patient present with chronic and relapsing intermediate, non-granulomatous anterior uveitis or retinal vasculitis. India being an endemic country and control of tuberculosis being far from reach, and also with emergence of MDR-TB and with unwavering incidence due to poor socio-economic conditions, overcrowding and illiteracy a suspicion of tuberculous etiology is always warranted.

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