Tuberculosis

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7.1 Introduction

Tuberculosis is a major public health problem worldwide and has been recognized as one of the common causes of infectious uveitis in developing countries [1]. Intraocular tuberculosis (TB) is one of the rare forms of extrapulmonary TB. It usually occurs without concomitant pulmonary or other systemic TB. Any part of the eye can get affected by *Mycobacterium tuberculosis* (MTB). It has protean manifestations, and the diagnosis is often presumed by a set of clinical signs and corroborative laboratory evidence.

7.2 Epidemiology

Reports from different regions of the world indicate that the prevalence of ocular TB ranges from 0.39 % (South India) to 10.5 % (Saudi Arabia) [2–10]. In developing countries, the prevalence of intraocular TB among uveitis patients is about 10 % [11] and less than 1 % in the USA [12]. Different regions within the same country have also shown variations [13]. While it is reported to be 0.39 % in South India [2], a study from North India has shown prevalence of 9.86 % [3]. Recently, ocular TB has been found to be more common in Los Angeles (six patients with probable or definite TB out of 142 consecutive uveitis patients) [14] than in Chicago (14 patients with ocular TB out of 3606 uveitis patients in a 16-year period) [15]. Recently, there is a growing evidence of ocular TB from countries with low or intermediate burden of TB [15–21].

7.3 Pathophysiology

In extrapulmonary TB, the tubercle bacilli, after inhalation into the lungs, are believed to disseminate into the distant organs via hematogenous or lymphatic route. The active infection of extrapulmonary tissues may occur during primary infection or upon reactivation of the latent infection. As the intraocular tissues remain difficult to be biopsied, the exact mechanism of intraocular TB remains unclear. However, a significant understanding of the pathogenesis has been recently provided in a few experimental studies. Rao et al. offered an excellent model of ocular TB to address its pathogenesis. They exposed the guinea pig lungs to MTB via an aerosol delivery of the organisms that led to its hematogenous dissemination [22]. All animals developed pulmonary lesions, with dissemination to the spleen. Of six animals receiving no antitubercular therapy (ATT), ocular lesions developed in 42 % eyes. The granulomatous reaction seen on histological analysis of lungs and ocular tissues was similar to that seen in humans with TB. In the second group of four guinea pigs receiving ATT 14 days after

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infection, none showed granulomatous inflammation, suggesting the role of systemic anti-TB drugs in preventing ocular TB. The similarity between guinea pig model of TB and humans with TB was also reflected by development of pulmonary TB in all animals and ocular TB in some of them.

Vascular endothelial growth factor (VEGF) has been used as a biomarker for active TB disease. In another animal model of ocular TB following aerosol delivery of MTB in guinea pigs, Thayil et al. demonstrated microbiological, histological, and clinical features of intraocular TB infection [23]. The retinal pigment epithelium (RPE) and photoreceptors demonstrated VEGF expression, and choroidal granulomas showed reduced oxygen tension. The authors hypothesized that VEGF upregulation in lungs and RPE occurred through inflammatory mediators of MTB infection and/or local inflammation causing tissue hypoxia. Their results suggested a hematogenous route of intraocular infection rather than direct inoculation.

The presence of MTB genome within the retinal pigment epithelium (RPE) cells was first demonstrated by Rao et al. in an enucleated globe with panuveitis [24]. The authors suggested a preferential localization of the MTB within the RPE, providing the site for reactivation that may manifest clinically as choroiditis.

Nazari et al. investigated the mechanism of MTB phagocytosis and its growth in the RPE compared to macrophages [25]. They suggested that MTB is readily phagocytized by the RPE in a manner similar to macrophages, but the viability of RPE is not affected by the intracellular MTB. They control the bacillary growth (better than the macrophages) and, hence, can act as reservoirs for intraocular MTB infection.

7.4 Clinical Features

7.4.1 Systemic Disease

Ocular involvement in patients with active pulmonary TB is extremely rare (1.4–6.8 %) [26–28]. Majority of ocular TB cases occur as isolated disease, with very few associated with extraocular TB. In a study on consecutive patients with a diagnosis of ocular TB at a center in Italy, 45 patients had isolated ocular TB, and 17 had ocular TB with extraocular TB [29]. In Spain, 18 % of patients with culture-proven systemic TB had intraocular TB [30]. We found a very low rate (3 %) of systemic TB in our series of patients with presumed intraocular TB [31].

7.4.2 Ocular Disease

Men and women are equally affected in any age. There is a lack of consensus on clinical diagnostic criteria for intraocular TB [32, 33]. The clinical spectrum of intraocular TB is highly variable and may mimic features of other uveitides. While granulomatous anterior uveitis is common in intraocular TB, the presence of mutton-fat keratic precipitates needs exclusion of other known causes such as viral uveitis, sarcoidosis, Vogt-Koyanagi-Harada (VKH) disease, sympathetic ophthalmia, or syphilis.

7.4.2.1 Ocular Surface Disease

The primary eyelid TB is a rare condition, and it occurs usually secondarily to orbital involvement. It can manifest as a chronic painless swelling, discharging sinus, chalazion, or an atypical lid swelling after blepharoplasty [34–37]. Orbital TB may involve the lacrimal gland as dacryoadenitis [38, 39], soft tissue as tuberculoma or cold abscess with or without bony involvement, and periosteum as classic periostitis or may spread from paranasal sinuses [40–42]. It may masquerade as an orbital malignancy in the presence of proptosis [43].

Conjunctival involvement in TB may manifest as chronic conjunctivitis (ulceration, epibulbar mass, papillary lesion) [44], allergic conjunctivitis in a child [45], chronic conjunctivitis with neighboring cutaneous TB [46, 47], or a tuberculoma [48]. Corneal involvement may manifest as phlyctenular keratoconjunctivitis [49] and chronic red eye [50]. Involvement of sclera may occur as nodular episcleritis [51], sclerouveitis

masquerading as ocular tumor [52], or posterior scleritis [53]. Infective scleritis in immunosuppressed patients may occur as a result of reactivation of latent MTB [54].

7.4.2.2 Anterior Uveitis

Anterior uveitis in intraocular TB is usually granulomatous with mutton-fat keratic precipitates (Fig. 7.1) [55–62]. It may be unilateral or bilateral. Iris nodules are less frequent, which are seen on pupillary border or iris surface. Chronic recurrent inflammation produces posterior synechiae, which are usually broad based, and has been found to be strong clinical predictors of TB uveitis, as compared to other etiologies [16, 62, 63]. Other rare features may include acute anterior uveitis [64] and hypopyon [65, 66].

7.4.2.3 Intermediate Uveitis

It presents with vitritis, snowballs, peripheral retinal phlebitis, and peripheral vascular sheathing. Cystoid macular edema is the cause of visual loss. In a study from high-endemic setting, TB was found the commonest etiology in a series of intermediate uveitis patients [67]. It may sometimes present as chronic, low-grade vitritis with phlebitis. In Singapore, a country with intermediate TB burden, significant vitritis and phlebitis were more commonly associated with latent TB infection [16].

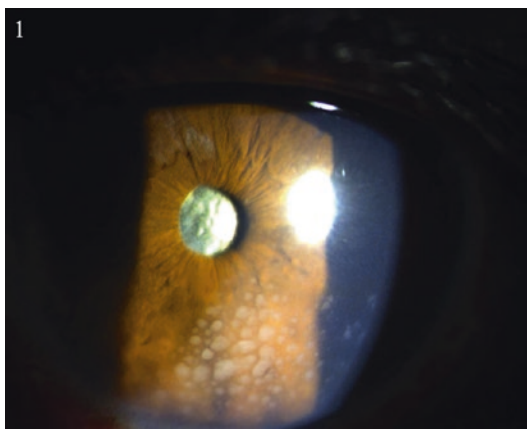

Fig. 7.1 Slit lamp photograph of the left eye of a 45-year-old female with tubercular anterior uveitis showing mutton-fat keratic precipitates

7.4.2.4 Posterior Uveitis

Choroidal TB

This is the commonest form of uveitis associated with TB [12, 31, 62, 68–70]. Choroidal tubercles were the earliest sign described in ocular TB in children with miliary tuberculosis [71]. They may be solitary or multiple in numbers and are usually diagnostic of disseminated TB, indicating a hematogenous spread of tubercle bacilli [72–78]. On fundus fluorescein angiography (FFA), they show an initial hypofluorescence followed by a late hyperfluorescence, with a peritubercular hyperfluorescence, suggesting active focal infection and inflammation [79]. They heal with atrophic scars and variable pigmentation. Optical coherence tomography (OCT) shows a raised RPE-choriocapillaris complex in the initial active stage with normal overlying retina and flattening of this region with scarring underneath [80]. While tubercles are small in size (0.2 mm–3 mm) and larger in numbers, a tuberculoma is larger in size (4 mm–14 mm) and appears predominantly in the posterior pole as a subretinal granuloma with surrounding exudative retinal detachment [81–84]. Caseation results from rapid bacillary growth within the granuloma and can be seen histopathologically in these abscesses [85]. A subretinal abscess may have overlying hemorrhages and develop retinal angiomatosis proliferans [86]. Larger tuberculomas may masquerade as ocular tumors [87]. The OCT shows retinal elevation with subretinal fluid that resolves with ATT and oral corticosteroids [88].

Multifocal serpiginoid choroiditis (MSC, previously called serpiginous-like choroiditis) is highly specific of tubercular uveitis [62]. It may be unilateral or bilateral and frequently affects young healthy males. Vitritis is often present. The lesions are multifocal and noncontiguous to optic disc and spread in a serpiginous pattern. They usually involve both the posterior pole and peripheral fundus and respond very well to ATT and oral corticosteroids (Fig. 7.2) [89, 90]. Despite initial aggressive inflammation involving the macula, the fovea is spared, and the patients maintain a good final visual acuity. On

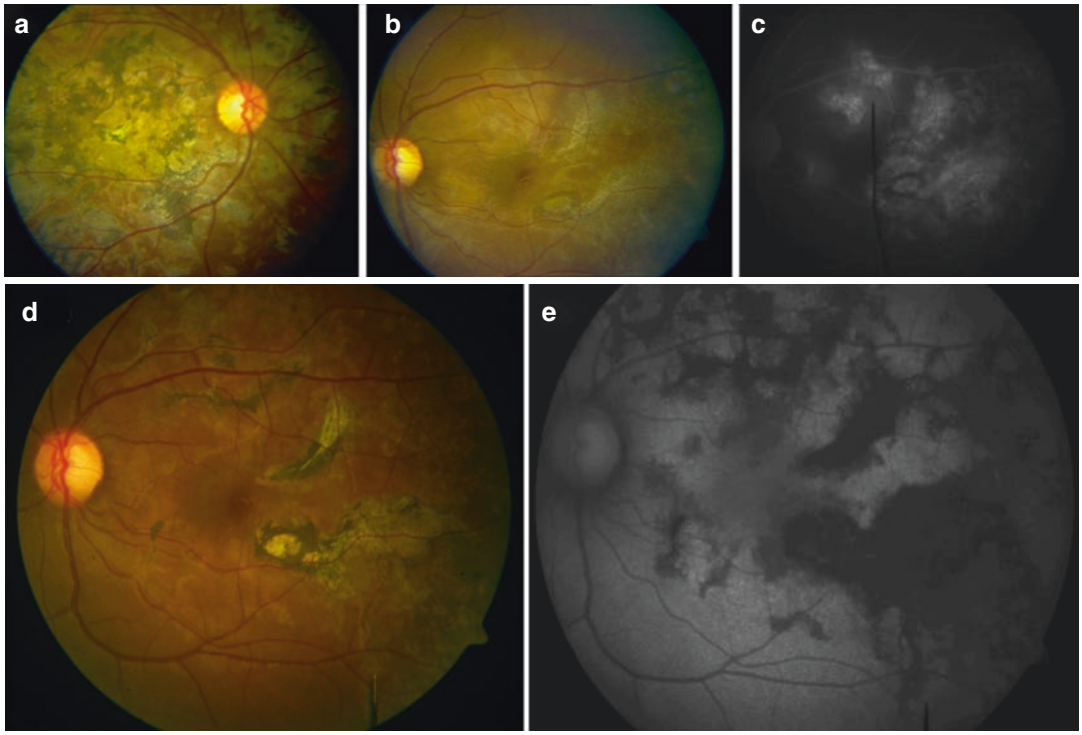

Fig. 7.2 Fundus photographs of a 31-year-old female with healed lesions of multifocal serpiginoid choroiditis in the right eye (**a**) and active lesions in the left eye (**b**) that showed hyperfluorescence in the late phase of fluorescein angiography (**c**). The QuantiFERON-TB Gold (QFT) test was positive. Following treatment with oral corticosteroids and antitubercular therapy, the left eye showed healed lesions at 21 months (**d**) that were hypoautofluorescent (**e**)

healing, the scars show significant pigmentation. It bears significant differences from the classic serpiginous choroiditis (SC), which affects elderly patients that has juxtapapillary, large, solitary lesions with minimal or no vitritis [91, 92]. The classic SC shows relentless progression and recurrent episodes despite corticosteroids and immunosuppressive agents and causes significant visual morbidity. In MSC, anterior segment inflammation and retinal vasculitis are rare [89, 93]. On FFA, the active lesions exhibit hypofluorescence in an early phase and progressively become hyperfluorescent in late phases, and the healed lesions (scars) show window defects [89, 94]. On indocyanine green angiography (ICGA), the lesions appear hypofluorescent in early, intermediate, and late phases [89, 94, 95]. The lesions show a characteristic pattern on fundus autofluorescence (FAF) imaging as they evolve from an acute stage to the healed stage, and this modality can be reliably used for clinical monitoring of the patients [94, 96]. The multimodal imaging with OCT reveals outer retinal morphological changes in the form of RPE-photoreceptor disruption in acute stages, followed by their atrophy as the lesions heal [97].

Retinal Vasculitis

It affects males more commonly than the females. It usually occurs without any systemic association. Previously called Eales' disease, its association with MTB has been reported by several studies [98–100]. It may be unilateral or bilateral and focal or diffuse, involving veins more commonly than the arteries. Perivascular infiltrates are seen as cuffing and are frequently associated with retinal hemorrhages. Vitritis is almost always present [101]. Snowball opacities, neuroretinitis, cystoid macular edema (CME), and

branch retinal vein/artery occlusion are commonly associated with tubercular retinal vasculitis. The FFA shows staining and leakage from the vessel walls (which may be focal or diffuse), CME, optic disc staining, and typical occlusive nature of the vasculitis in the form of peripheral capillary nonperfusion (Fig. 7.3). Rosen et al. found ischemic retinal vasculitis as the most common form of uveitis in their series with a marked tendency to neovascularization [50]. Vitreous hemorrhage, neovascularization of the optic disc or elsewhere in the retina, and tractional retinal detachment result from untreated occlusive disease. Advanced cases may present with combined retinal detachment and iris or angle neovascularization.

It is frequently accompanied by choroiditis, which may be healed or active. The presence of retinal vasculitis with perivascular choroiditis scars has been found to be highly specific for TB in endemic countries [62, 102]. Central retinal vein occlusion (ischemic) and frosted branch angiitis are rare presentations [103, 104]. Active retinal vasculitis responds well to oral corticosteroids and ATT [31]. Laser photocoagulation and vitrectomy are required for neovascular sequelae.

Endophthalmitis and Panophthalmitis

These are atypical presentations in ocular TB and may mimic as ocular tumors [65, 66, 87, 105–108]. Diagnosis in these cases is confirmed by microbiological/histopathological evidence of MTB.

Optic Nerve Involvement

Involvement of optic nerve in ocular TB can result from direct infection or a hypersensitivity reaction and can present as optic disc tubercle, neuroretinitis, optic neuritis, retrobulbar neuritis, and papillitis [109–111].

7.5 Diagnosis

In the absence of a gold standard laboratory test demonstrating MTB in intraocular tissues, the diagnosis of intraocular TB presents a unique challenge and is restricted to a presumptive diagnosis by the clinical signs and corroborative evidence. Further, the lack of uniform diagnostic criteria adds to difficulties in diagnosing intraocular TB. Recently, Gupta et al. proposed a new classification system, based on clinical signs and diagnostic tests, for diagnosing intraocular TB as confirmed, probable, or possible intraocular TB [70]. According to this classification, a patient with a clinical sign(s) suggestive of ocular TB and microbiological confirmation of MTB from ocular fluids/tissues is diagnosed to have "confirmed ocular TB." A patient with suggestive clinical sign(s), exposure to TB/immunological evidence of TB, along with clinical/radiological/microbiological evidence of TB infection in extraocular sites would be diagnosed as "probable ocular TB." "Possible ocular TB" is diagnosed in the presence of a clinical sign of ocular TB, either with exposure to TB/immunological evidence of TBs or with clinical/radiological evidence of extraocular TB.

7.6 Indirect Evidence

Besides the clinical signs, corroborative evidence is provided by a positive tuberculin skin test (TST), or a positive interferon gamma release assays (IGRA), or radiological findings suggesting old or active TB on a chest X ray, or evidence of manifest TB elsewhere, exclusion of other causes of uveitis, and a positive response to ATT. The TST has been used since long for diagnosing and treating ocular TB due to its low cost and wide availability. The association of latent TB (as diagnosed by a positive TST) and uveitis has been shown in an endemic setting in the form of a favorable therapeutic response to ATT [31]. It, however, has its own limitations such as inability to distinguish between latent and manifest TB or between tuberculous and nontuberculous mycobacteria, false-positive (due to prior BCG vaccination or infection by atypical mycobacteria) or false-negative (immunocompromised state such as HIV infection) results, errors in conducting or interpreting the result, and need for a double visit by the patient. In a series of definite

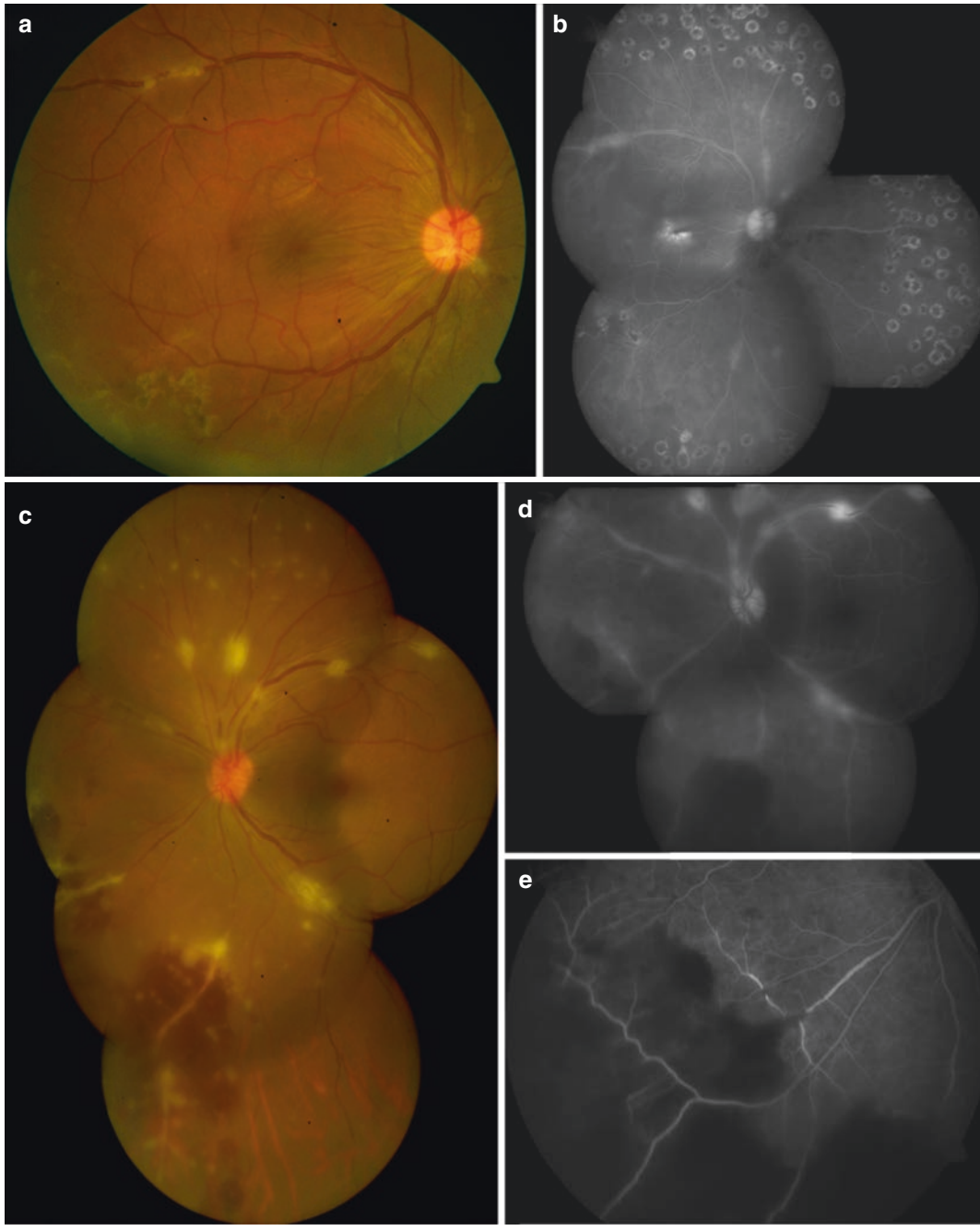

Fig. 7.3 Right eye fundus photograph (**a**) and fluorescein angiogram (**b**) of a 22-year-old male (having undergone pars plana vitrectomy and laser photocoagulation for vitreous hemorrhage in the past) with active retinal vasculitis and cystoid macular edema. The left eye also had active retinal vasculitis (**c**, **d**) with peripheral areas of capillary non-perfusion seen on fluorescein angiography (**e**). The tuberculin skin test was 17 × 18 mm. He received oral corticosteroids and antitubercular therapy, along with laser photocoagulation in left eye. At 16 months, both the eyes were quiescent (**f**, **g**)

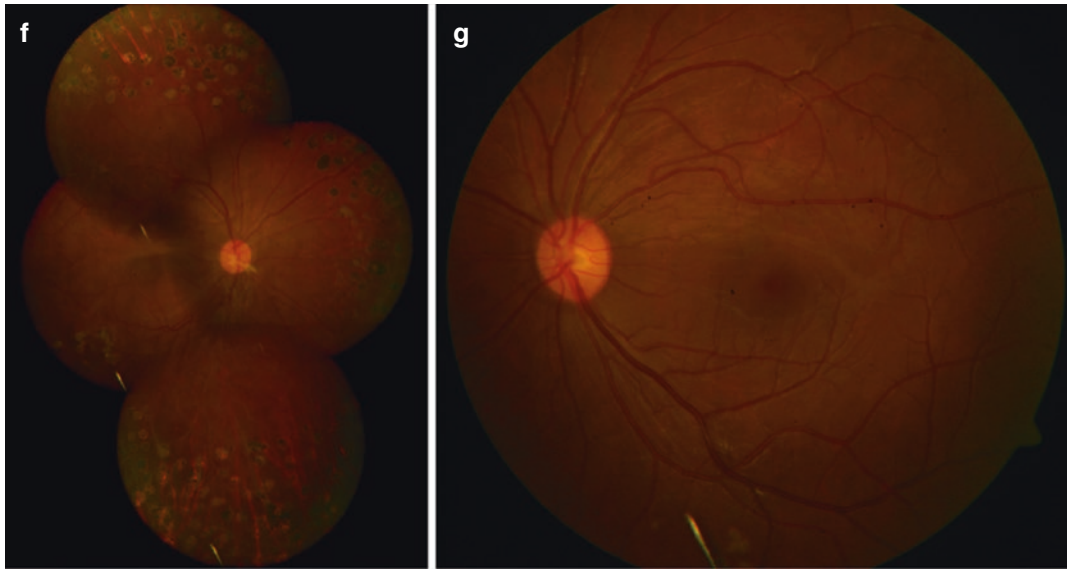

Fig. 7.3 (continued)

ocular TB cases, 40 % patients did not have a positive TST [112].

The more recent IGRAs have improved the specificity of a previous exposure to MTB, as they are not influenced by BCG vaccination or nontuberculous mycobacteria. Both TST and IGRAs indirectly detect an immune response to recent or prior exposure to MTB. The genomic region of MTB complex encodes several antigens that elicit a severe immunogenic response involving helper T cells, which can be measured in vitro through quantification of interferon gamma or interferon gamma-producing T cells by the IGRAs. But they also, like the TST, lack the ability to differentiate latent from manifest TB. QuantiFERON-TB Gold (QFT) In-Tube (Cellestis Inc., Carnegie, Australia) has been proved only slightly superior to TST in diagnosing TB uveitis [112]. On the other hand, Gineys et al. have used its cutoff value as a measure to identify cases of TB-related ocular inflammation that can benefit from ATT [113]. In a more recent prospective study on patients with TB-associated uveitis, T-SPOT.TB (Oxford Immunotec, Oxford, United Kingdom) test was found less sensitive but more specific than TST in populations with high prevalence of TB-associated uveitis, and hence, the authors concluded that TST should be the first-choice test in this population, while in low TB-prevalence populations, T-SPOT.TB test should be preferred to TST [114].

A recent survey among specialists dealing with different forms of TB (uveitis experts, pulmonologists, and rheumatologists) in India reported that the use of QFT Gold test in clinical practice was limited by its increased cost and limited data from India related to interpretation of the result [115].

Radiological evidence is usually sought in the chest X ray, as lungs are the primary sites of TB infection. Any evidence of present or previous TB on chest X ray increases the probability of uveitis being tubercular in origin, but this is rare as majority of intraocular TB cases occur in the absence of pulmonary TB. The reliability on chest X ray findings reduces further as some patients with primary TB may have a normal chest X ray [116]. Such patients need CT scan of the chest as a useful alternative [117]. In a series of definite ocular TB cases, 57 % had negative chest radiograph results [118]. Although usually inconclusive, chest X rays still form an integral part of the baseline laboratory workup of a patient with suspected TB uveitis.

When the above conventional radiological tests are negative, positron emission tomography-computed tomography (PET-CT) is useful in establishing TB as the cause of uveitis by demonstrating uptake of fluorodeoxyglucose (FDG) in metabolically active TB lesions [119, 120]. In patients with presumed ocular TB, Doycheva et al. demonstrated positive PET-CT findings in extraocular sites (mediastinal or hilar lymph nodes) in 45 % patients, which were diagnosed and treated accordingly [119]. While some patients may not demonstrate any systemic uptake at all, others may show an increased FDG uptake in various extraocular sites (pulmonary, extrapulmonary, or disseminated) suggesting a more widespread disease than presumed by the ophthalmologist [121]. There is, however, an insufficient evidence to suggest the use of PET-CT as a routine imaging modality in tubercular uveitis due to its high cost and limited reports of its use in uveitis diagnosis and management.

7.7 Direct Evidence

Demonstration of MTB in intraocular specimens (fluid or tissue) provides a direct and definitive evidence of intraocular TB. Smear positivity for acid-fast bacilli is extremely rare from ocular samples, due to low yield of fluid volume (aqueous/vitreous) as well as paucibacillary nature of intraocular TB [112]. Laborious and delayed culture reports often show no growth from ocular samples. Moreover, the risk of damage to ocular structures while sampling intraocular tissue/fluid adds limitations to performing histopathological diagnosis. Destructive interventions like evisceration or whole globe enucleation may require to be undertaken in cases of ocular TB masquerading as purulent ocular infections or tumors [85, 87] or those showing progressive worsening despite systemic corticosteroid treatment [24]. Although histopathological evidence forms the gold standard for diagnosing intraocular TB, it is never used as a first-line investigative tool.

Polymerase chain reaction (PCR) has long been reported in tubercular uveitis [101, 122–124]. While conventional PCR showed a low sensitivity and high specificity, multi-targeted PCR has emerged as a novel method by detecting different MTB genomes in intraocular samples and has shown an improved sensitivity [125]. Quantitative PCR (qPCR) is a fast method with minimum risk of cross contamination, which additionally quantifies the bacterial load in the tested sample [24, 126]. However, these tests require laboratories with good research facilities and, hence, remain limited only to resourceful settings. Further, poor correlation between qPCR and AFB results has been reported by Wroblewski et al., in which two out of three qPCR-positive patients did not show AFB in tissue sections [118]. One patient with positive AFB results had negative PCR results. Negative PCR results cannot exclude the diagnosis due to low sensitivity. Also, while performing these tests, the limited role of PCR technology in ocular TB should be kept in mind as suggested in paucibacillary form of cutaneous TB [127].

7.8 Newer Diagnostic Tools and Drug Resistance

The diagnostic armamentarium of intraocular TB has seen significant recent advances. While drug resistance has been a major health problem in pulmonary and extrapulmonary TB, it has been recently detected in intraocular TB [128–130]. The Xpert MTB/RIF assay (Cepheid, Sunnyvale, California) was approved by the WHO in 2010 for diagnosis of pulmonary TB [131]. It detects MTB DNA and simultaneously tests its susceptibility to rifampicin (RIF). The other advantages include quick results and elimination of cross contamination. High sensitivities and specificities have been reported from pulmonary and extrapulmonary samples [132–134]. The line probe assay [GenoType MTBDRplus (Hain Lifescience, GmbH, Nehren, Germany)] simultaneously detects MTB DNA as well as RIF and isoniazid (INH) resistance and produces results within about 5 h [135, 136]. In our experience, while both tests had low sensitivities (40 % and 60 %, respectively) for detecting MTB

genome in a series of patients with MSC, they detected multidrug-resistant (MDR) tubercular uveitis in patients showing poor response to conventional four-drug ATT [130]. Early detection of MDR in intraocular TB is of immense relevance to prevent ocular morbidity, particularly in cases showing poor initial response, paradoxical worsening, or with atypical presentation. However, these rapid molecular tests have cost issues and need skill and specialized infrastructure, making their suitability restricted to tertiary care centers.

7.9 Management

There are no specific guidelines on the treatment protocol of intraocular TB [33]. There is a wide heterogeneity among uveitis specialists worldwide in the approach to diagnosis and management of ocular TB [32]. As an empirical treatment, ATT has been shown to be highly effective in reducing recurrence of uveitis when given with anti-inflammatory therapy in patients with presumed tubercular uveitis in endemic as well as low-endemic countries [31, 137, 138]. While corticosteroids are administered along with ATT and tapered as per the clinician's discretion depending upon the clinical response, ATT is administered for a prolonged duration in ocular TB, as recommended for any extrapulmonary site that is slow to respond to therapy [139]. Duration of more than minimum 9 months has been associated with an 11-fold reduction of recurrence of uveitis in a retrospective study [138]. Exclusion of other systemic disease or history of exposure to TB in non-endemic regions have been suggested as important factors in considering ATT in patients with relevant clinical presentation [138, 140]. Despite these suggestions, a simple algorithm still remains to be proposed for treating ocular TB. The conventional four-drug ATT comprises of isoniazid, rifampicin, ethambutol, and pyrazinamide for initial 2 months, followed by isoniazid and rifampicin for another 9–10 months, along with pyridoxine supplementation. Since most patients benefit from empiric ATT when started timely, an underdiagnosis would cause visual morbidity in an otherwise treatable uveitic entity. On the other hand, a judicious combination of clinical presentation and laboratory results is required to avoid overtreatment, as the ATT is expensive and potentially toxic. Besides potential drug toxicities, ATT may cause paradoxical response that is well known in pulmonary and extrapulmonary TB. Worsening of inflammation or the development of new lesions has been well documented in the eye after initiating ATT for ocular TB [141–145]. Although addition or rise of corticosteroids resolves this phenomenon, its occurrence in the eye may complicate judgment by raising several concerns such as poor compliance, drug resistance, disease relapse, or a nontubercular etiology.

In cases where rifampicin resistance is detected, the diagnosis is revised to MDR ocular TB, and the treatment comprises of levofloxacin 750 mg/day, ethionamide 750 mg/day, cycloserine 750 mg/day, streptomycin injection 1000 mg/day (intramuscular), and pyrazinamide 1500 mg/day for initial 5 months, followed by levofloxacin 750 mg/day, ethionamide 750 mg/day, and cycloserine 750 mg/day for another 18 months, under the supervision of a hepatologist with regular monitoring of liver and renal function tests.

7.10 Prognosis

A timely diagnosis of ocular TB and initiation of ATT with corticosteroids is associated with a favorable outcome in terms of reduced rate of recurrences [31, 137, 138].

Conclusion

The diagnosis of intraocular TB is challenged by a number of factors such as a wide variation in clinical manifestations, the absence of concurrent systemic TB, low sensitivity and specificity of laboratory tests, paucibacillary nature of ocular TB, and difficulty in obtaining adequate intraocular sample for histopathological diagnosis. The issues related to ATT such as lack of treatment guidelines,

long duration of therapy, side effects, paradoxical worsening, recurrence of inflammation, and emergence of drug resistance further complicate the course in management of intraocular TB.

Core Messages

- Intraocular TB has a wide clinical spectrum worldwide. However, broad-based posterior synechiae, retinal vasculitis, and multifocal serpiginoid choroiditis have been recognized as highly specific for intraocular TB in an endemic country.
- Intraocular TB is being increasingly reported from non-endemic countries, too, predominantly in recent migrants from endemic areas.
- Recent experimental studies have shown significant advancements in the understanding of the pathogenesis of intraocular TB.
- A newer classification system has been proposed for intraocular TB (with "confirmed," "probable," and "possible" as three diagnostic groups), which needs to be validated in future studies.
- Specific clinical signs, immunological evidence of TB infection, documented exposure to TB, radiological evidence of TB infection, clinical evidence of extraocular TB, and microbiological/histopathological evidence of MTB are the criteria recommended for classifying intraocular TB.
- Antitubercular therapy with corticosteroids is highly effective in majority of cases.
- A high index of suspicion for multidrug resistant intraocular TB should be kept in mind for cases not responding to conventional therapy.
- Patients should be strictly monitored for any potential drug toxicities by an internist.

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