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Tuberculosis affects one-third of the world population [1]. Extrapulmonary tuberculosis (TB) involving the pleura, lymphatics, bone, genitourinary system, meninges, or skin occurs in 15% of TB patients [2]. The incidence of ocular TB ranges from 1.4% to 5.74% [3]; it may occur in association with either pulmonary tuberculosis or in isolation, with no clinical or laboratory evidence of pulmonary infection [4]. The number of extrapulmonary tuberculosis cases has increased in recent times in immunocompromised individuals with AIDS and tuberculosis (2.8–11.4%) [3, 5].

The organism *M. tuberculosis* (MTB) is an obligate aerobic slow-growing, nonspore-forming, nonmotile bacteria. Humans are the only natural host, and infection is usually by airborne aerosol and enters into susceptible hosts through the lung and results in a latent infection in individuals with normal immune systems [3]. In 5% of newly infected persons, the pulmonary process progresses. Rarely lymphohematogenous spread of bacilli in large numbers may lead to miliary TB or other extrapulmonary manifestations [4].

Spread to the eye and other extrapulmonary sites usually occurs from hematogenous or adjacent spread of viable bacilli or as a local phe-

nomenon of hypersensitivity to circulating tuberculo-proteins [6]. The bacilli tend to localize in tissues that have high regional oxygen tension which includes the apices of the lungs, kidneys, bones, meninges, eye, and choroid [3]. Within the eye the preferred sites include the choroid and ciliary body where the oxygen tension is higher in comparison to other ocular structures [7].

Ocular TB is often misdiagnosed as retinoblastoma, squamous cell carcinoma, xanthogranuloma, or pseudotumor. Corneal or corneoscleral perforations may also occur. Its diagnosis is challenging in the absence of pulmonary disease [6].

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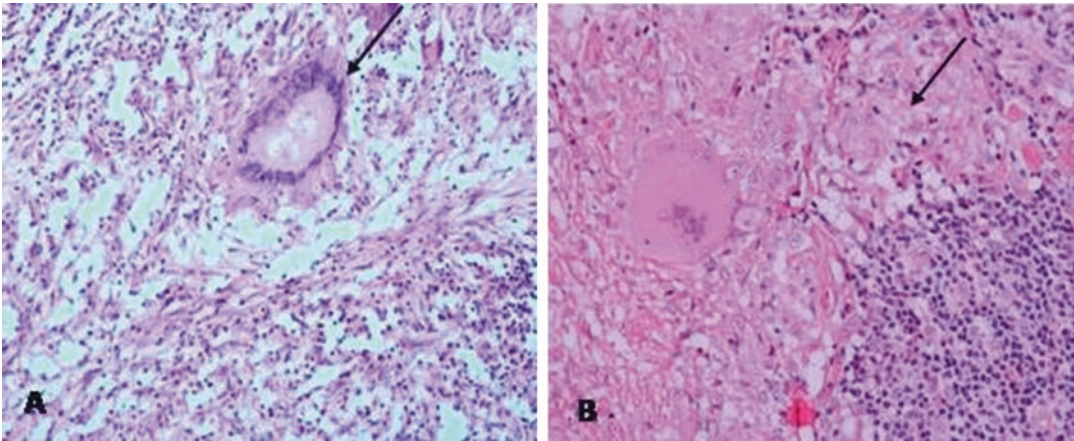
## Pathology of Ocular Tuberculosis

*M. tuberculosis* infection is usually chronic and insidious when it affects the eye and adnexa. It is usually a hematogenous spread of the organism. The three forms of disease include mycobacterial invasion of ocular tissues, hypersensitivity to antigen of MTB with viable mycobacteria, and hypersensitivity in the absence of viable bacteria [1].

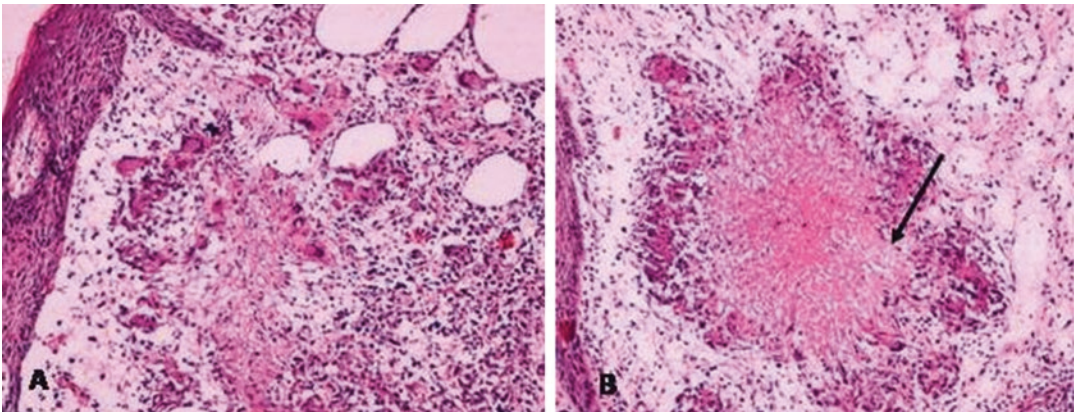
Characteristic histopathological features in ocular tuberculosis include granulomatous inflammation involving the sclera, cornea, conjunctiva, iris, and ciliary body with central caseous necrosis and occasional or no acid-fast bacilli (Fig. 2.1a, b). The granulomas are composed of abundant epithelioid histiocytes, occasional giant cells of Langerhans type, and lymphomononuclear

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**Fig. 2.1** (a) Granulomatous inflammation with giant cell reaction (arrow) (H&E  $\times 100$ ). (b) Higher magnification to show epithelioid cells (arrow) and lymphomononuclear surrounding the Langerhans giant cell (H&E  $\times 200$ )



**Fig. 2.2** (a) Subconjunctival necrotizing granuloma with giant cell reaction in a case of suspected eyelid tuberculosis (H&E  $\times 100$ ). (b) High-power view to show necrosis (arrow) in the center of granuloma (H&E  $\times 200$ )

cells. Since the granulomas in immunocompetent individuals contain occasional bacteria, the staining may not reveal the presence of organisms. In such cases, the bacterial DNA may be detected by polymerase chain reaction (PCR).

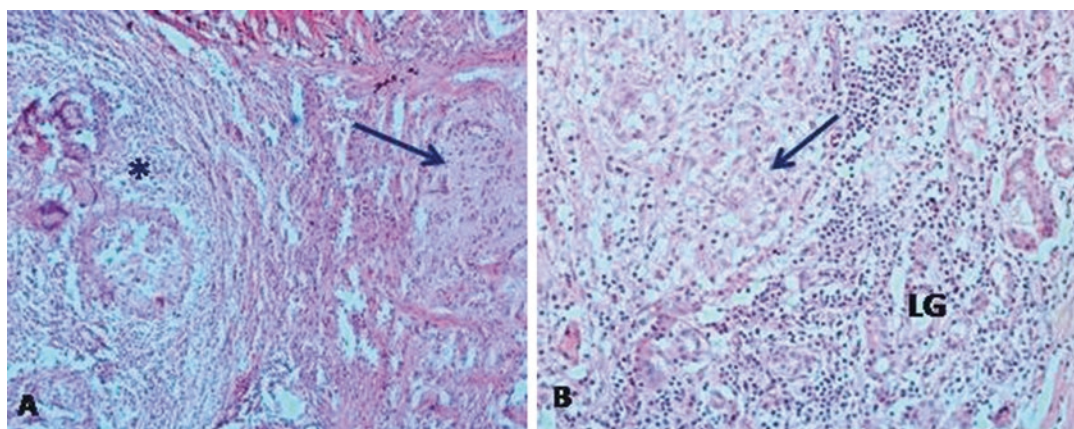
Ocular tuberculosis is a unique form of extrapulmonary tuberculosis which can present with several clinical manifestations based on the virulence of the organism and immune status of the individual. Both ocular and orbital tuberculosis are usually unilateral [8]. The most common clinical presentation is posterior uveitis followed by anterior uveitis, pan uveitis, and intermediate uveitis. Although granulomatous uveitis is common,

it may be nongranulomatous. The diagnosis of intraocular TB is difficult prior to enucleation.

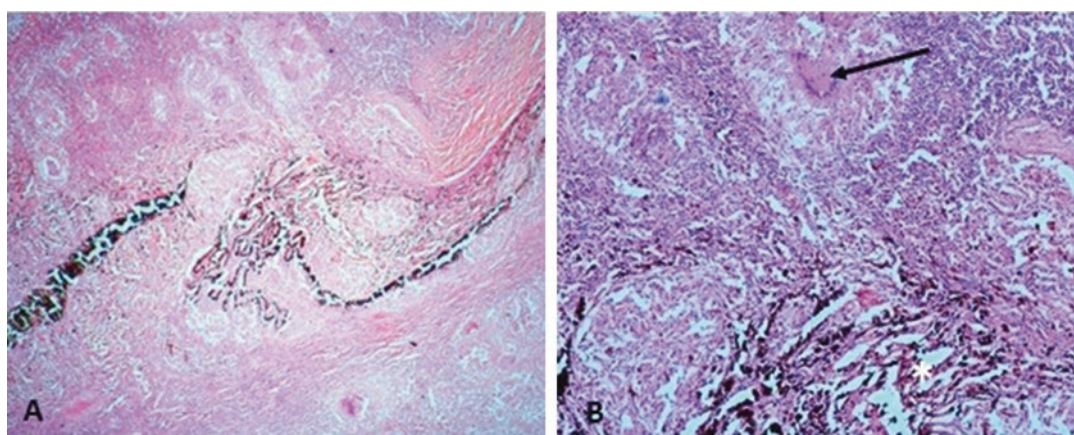
The eyelid involvement is very rare and is usually secondary to orbital TB and may appear as a small nodule simulating a chalazion or as a draining sinus (Fig. 2.2a, b). Rarely primary conjunctival and eyelid tuberculous granuloma may occur [8–11].

Orbital or lacrimal gland (Fig. 2.3a, b) and lacrimal sac granuloma may occur secondary to infection with *M. tuberculosis* [12, 13]. These may be associated with preauricular lymphadenopathy. Children can present as preseptal cellulitis with a fistula or as abducens nerve palsy [14].





**Fig. 2.3** (a) Granulomatous inflammation (*asterisk*) in lacrimal gland (*arrow* shows lacrimal gland acini) (H&E  $\times 100$ ). (b) Higher magnification shows epithelioid cell granuloma (*arrow*) and adjoining lacrimal gland (LG) (H&E  $\times 200$ )



**Fig. 2.4** (a) Tuberculous granuloma of the ciliary body with acute panophthalmitis. The vitreous shows necrotizing inflammatory reaction (H&E  $\times 40$ ). (b) Higher magni-

fication to show granuloma with giant cell (*arrow*) and destroyed ciliary processes (*asterisk*) (H&E  $\times 200$ )

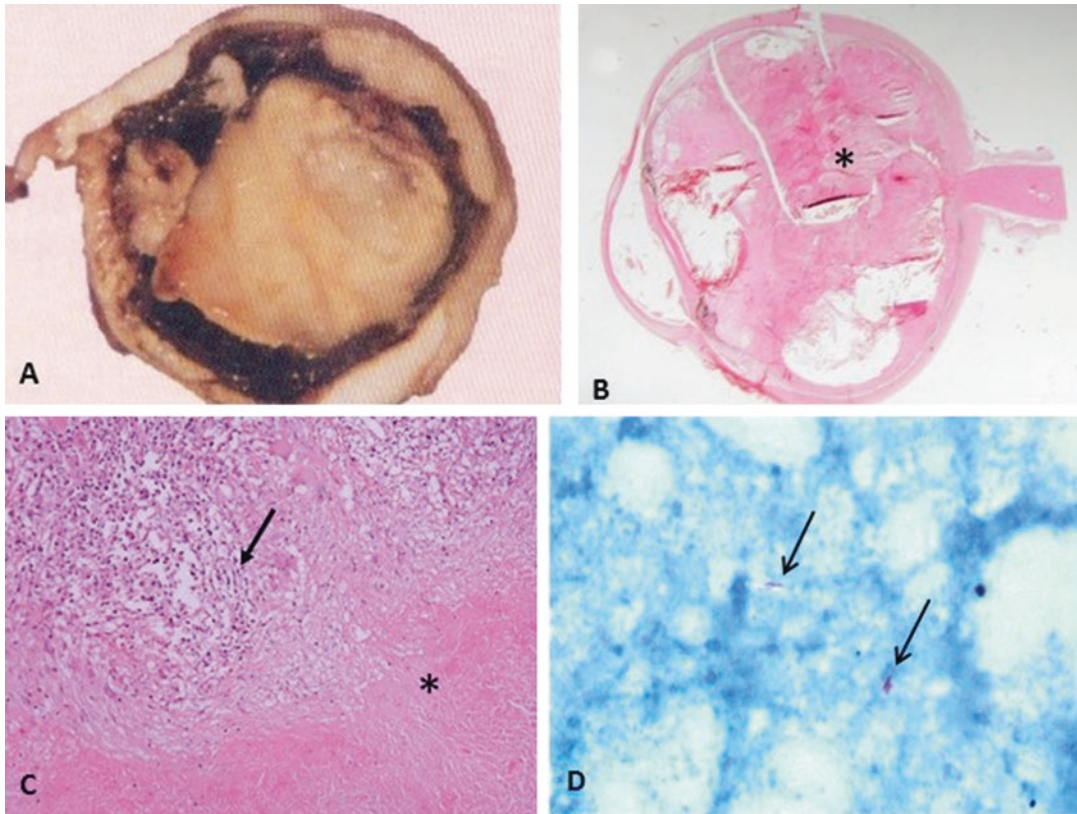
*Interstitial keratitis and phlyctenular keratoconjunctivitis* represent a localized immunologic (hypersensitivity) response to the antigens of mycobacteria. Tuberculous scleritis presents as anterior scleritis mostly in the form of focal elevated nodules which may undergo necrosis and result in scleromalacia. Many of the cases of anterior segment are not associated with systemic manifestations of TB and appear localized to the eye [3, 10, 15].

*Scleritis*, both necrotizing and non-necrotizing, diffuse or nodular, may be associated with TB [16]. The diagnosis is often presumptive and

rarely confirmed by histopathology or PCR following enucleation.

*Anterior uveitis* presents with insidious granulomatous uveitis which may be unilateral or bilateral. Iris lesions in tuberculosis appear as nodular areas at the pupillary margin, over the surface, or in the angle and are made up of epithelioid cells, giant cells, and lymphocytes with extensive caseation. Cyclitis is seen frequently and may cause caseating granulomas (Fig. 2.4a, b) [10, 17].

*Posterior segment* involvement is more common and may include features of endophthalmitis or panophthalmitis simulating intraocular tumors



**Fig. 2.5** (a) Gross specimen of enucleated eye ball shows yellowish white mass in vitreous cavity. (b) Low-power view to show the entire vitreous cavity replaced by necrotizing inflammatory mass (*asterisk*). (c) Tubercular endophthalmitis. Low-power view of necrosis (*asterisk*) with

adjacent chronic granulomatous inflammatory infiltrate (*arrow*) (H&E  $\times 200$ ). (d) Ziehl-Neelsen acid-fast stain demonstrates acid-fast bacilli (*arrow*) in necrotic tissue ( $\times 1000$ )

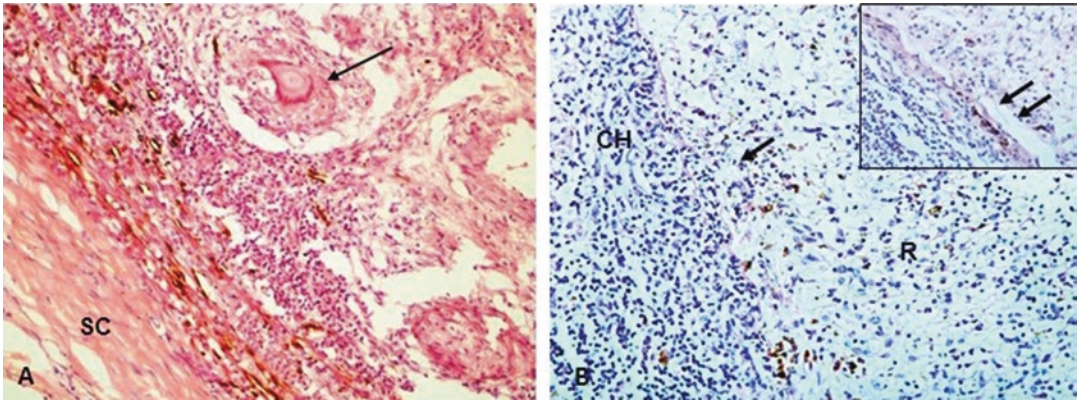
including retinoblastoma (Fig. 2.5a–d) [18]. The retina and choroid are frequent targets with multifocal choroiditis being most common. Solitary choroidal tuberculoma (Fig. 2.6a) may occur in immunocompetent patients and in patients with disseminated tuberculosis [19–21]. Multifocal choroidal tubercles may occur anywhere in the posterior segment with retinal involvement. Vitritis is frequently associated with large choroidal tuberculomas. In the choroid these tuberculomas involve all the choroid layers including choriocapillaris (Fig. 2.6b). These are usually surrounded by choroidal vessels which get obliterated. The RPE initially is normal but can get disrupted in later stages. The granulomas are typical; however, the necrotic areas contain few bacilli.

Tuberculosis may present with several manifestations including retinal vasculitis and serpiginous choroiditis. Retinal vasculitis may occur in the absence of choroiditis or retinitis. This form of phlebitis in patients with healed TB may represent immune-mediated reaction to tuberculoproteins. The inflammation may spread anteriorly to involve the anterior chamber angle, limbus, and cornea resulting in globe perforation.

### Pathogenesis of Ocular Tuberculosis

Pathogenesis of extrapulmonary tuberculosis can be extended to ocular tuberculosis. Due to absence or rare isolation of MTB from ocular samples, the role of immune-mediated and direct





**Fig. 2.6** (a) Choroidal granuloma (arrow) with lymphomononuclear infiltrate. Adjoining sclera (SC) (H&E ×200). (b) Tubercular retinitis. The retina (R) and choroid

(CH) are replaced by intense chronic inflammatory cell infiltrate (H&E ×200). Inset shows RPE cells (double arrow) and Bruch's membrane (arrow) (H&E ×400)

bacterial-mediated inflammation is debated [22]. The mechanisms involved in pathogenesis include:

- (i) Bacterial dissemination from the site of primary infection
- (ii) Bacterial localization in ocular tissues
- (iii) Bacterial reactivation and inflammation in these tissues

MTB is an obligate aerobic intracellular organism which invades tissues rich in oxygen. It enters the body through the respiratory system and spreads via the lymphatics or blood to other parts of the body [23]. Usually the bacteria are destroyed by alveolar macrophages or however they may grow destroying the alveolar macrophages resulting in initial nidus of developing tubercle [5].

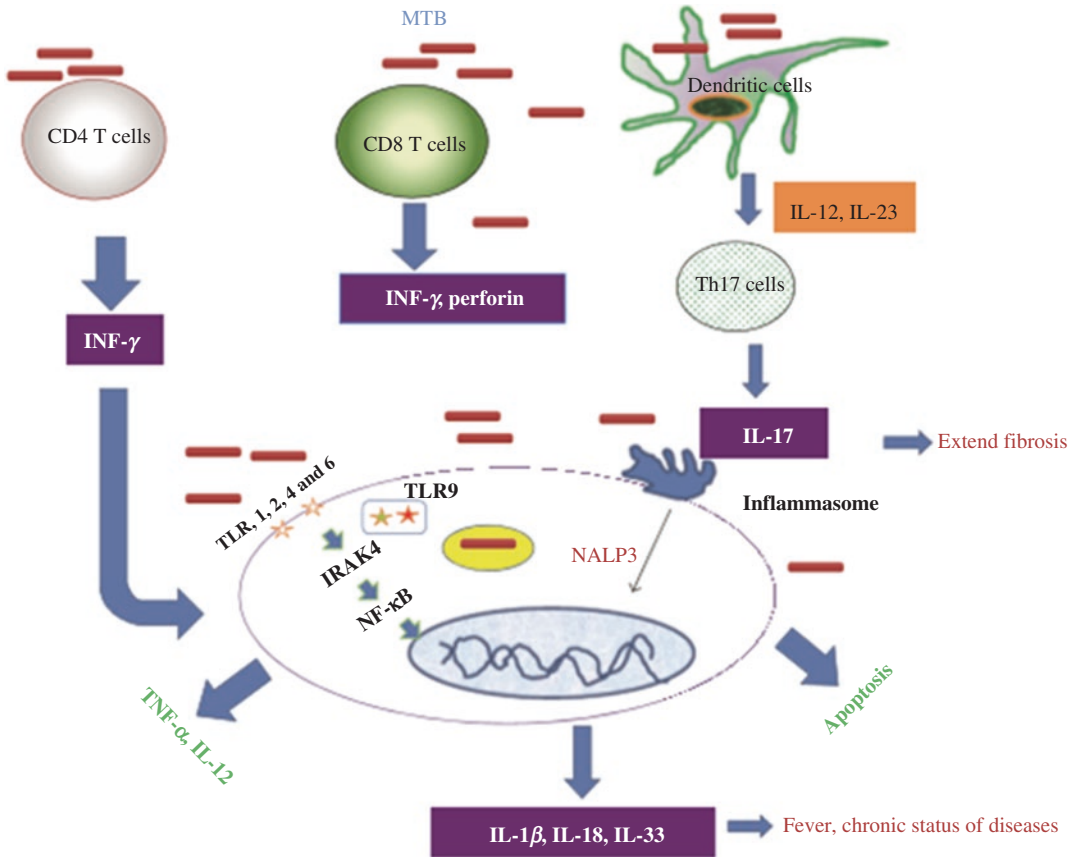
An initial growth of MTB results in a *delayed-type hypersensitivity response* which is characterized by the formation of small necrotic lesions with solid caseous centers in infected area. After starting delayed-type hypersensitivity (DTH) and tubercle formation, stimulation of macrophages by CD4+ T<sub>DTH</sub> cells enables the macrophages to kill bacilli inside of the tubercle lesions [23]. The growth of the bacteria becomes limited, and the number of viable bacteria becomes stationary [5].

If the cell-mediated immune response is poor, bacilli start re-multiplying in nonactivated and partly activated macrophages. The T cells are primed followed by initiation of adaptive immu-

nity which takes 5–7 days. During this latent period, macrophages carrying *M. tuberculosis* or even free bacteria may disseminate to the eyes or other organs. Once localized to the ocular tissues, *M. tuberculosis* may remain latent for long periods without apparent clinical disease [5, 24, 25]. The organism preferentially infects macrophages and other reticuloendothelial cells. Choroid is the most common site in the eye [1]. The RPE is the most suited among various ocular cell types to harbor MTB within the eye. It has alveolar macrophage-like properties like phagocytosis and expression of TLRs (Toll-like receptors) and complement receptors [22]. Although retina and uvea are involved by inflammation in tubercular pan uveitis, MTB localizes preferentially in RPE [26]. These organisms sequestered within RPE may cause recurrences.

TB choroidal granulomas in guinea pigs have shown evidence of tissue hypoxia with VEGF upregulation in RPE. However, AFB are rarely identified [27]. In most adults who are not HIV positive, ocular TB develops from postprimary reactivated lesion [5].

Although MTB evades killing if a good CMI develops, the bacteria are destroyed in the phagosomes on fusion with lysosomes, thereby exposing bacteria to acid pH, reactive nitrogen species, and lysosomal enzymes [26]. *M. tuberculosis* evokes an inflammatory response from the host to control the infection which may cause extensive



**Fig. 2.7** Immunopathogenesis of tuberculosis. MTB causes immune cells T4 cells, T8 cells, DCs, and Th17 cells to be activated. Th1 cytokines are released by the T cells. Macrophages are activated by IFN- $\gamma$ , MTB, IL-12, and TNF $\alpha$ , and they also undergo apoptosis – a major host

protection strategy against MTB infection (From Mortaz et al. [23], which is published under a Creative Commons Attribution License <https://creativecommons.org/licenses/by/3.0/>)

tissue damage [1]. The progression of the lesion may get halted at subclinical stage.

The rare isolation of mycobacteria has led to the development of an alternative hypothesis in pathogenesis of intraocular TB. Garip et al. [28] have hypothesized that antigen mimicry between tubercular and retinal antigens could be a cause of uveitis in latent TB patients. Supporting features of this hypothesis include demonstration of significantly increased IL-6, rather than TNF- $\alpha$ , IL-12, and interferon gamma that characterize TB [22].

### Immunopathogenesis of Ocular Tuberculosis (Fig. 2.7)

Initial immune response to *M. tuberculosis* results in the development of cell-mediated immunity and also hypersensitivity to mycobacterial antigens. Macrophages, T cells (CD4 and CD8), cytokines IFN  $\gamma$ , IL-12, TNF  $\alpha$ , and IL-6 are most important in immune response to MTB.

The immune response to active MTB is initiated with bacterial recognition by macrophage

and dendritic cells through TLRs. Activated macrophages via TLRs stimulate production of IL-12 and TNF- $\alpha$ . IL-12 causes a Th1-cell-mediated adaptive immune response which recruits CD4+ cells further and is responsible for controlling the infection [1]. Mycobacteria have a unique ability to delay the initiation of T-cell-mediated adaptive immune response by 2–3 weeks. This is due to the failure of dendritic cells to migrate to regional lymph nodes [22]. The immune response to MTB results in response from T cells, neutrophils, B cells, and NK cells. The mycobacterial antigens are presented by the macrophages and dendritic cells via MHC class II molecules on their surface to CD4 Th1 cells [2, 22].

### Cellular Immunity to MTB

- *Macrophages and monocytes*: MTB is a facultative intracellular parasite in macrophages. Entry into the macrophage is gained by phagocytic receptors like complement toll-like and mannose receptors followed by delivery of bacteria to phagolysosome. Nonpathogenic MTB get degraded by acidification of the phagosomes by hydrolases at low pH. Virulent MTB restrict fusion of vacuole with lysosome resulting in blockage of phagosome maturation which assures intracellular survival and replication of MTB. In macrophages activated by IFN- $\gamma$ , phagosome maturation is blocked. Within the phagolysosome MTB is deprived of essential nutrients and exposed to antimicrobial peptides and reactive oxygen or nitrogen intermediates. Macrophages may also undergo TNF- $\alpha$ -mediated apoptosis. Apoptosis contributes to host defense by removing the MTB growth by direct antimicrobial effects [23]. In resting macrophages MTB blocks phagosome maturation which assures intracellular survival and replication. Helper T cells recruit and activate new monocytes and macrophages [29–31].
- *CD4 Th-1 cell* is the most well-studied cell in the pathogenesis of tuberculosis and is very important in the formation of granulomas. CD4+ T cells are helper T cells which secrete various interleukins (IFN- $\gamma$  and IL-2) responsible for activating macrophages for protection against mycobacteria. IFN- $\gamma$  specifically activates macrophages and stimulates them to kill mycobacteria [23, 32, 33].
- *Dendritic cell response*: Dendritic cells are very important antigen-presenting cells of the innate immune system with the ability to stimulate memory T lymphocytes. After phagocytosis by alveolar macrophages and resident dendritic cells, dendritic cells are able to move to the local lymph nodes and present the antigen to the T cells. This is very important to generate a cell-mediated immune response. On exposure of dendritic cells (DCs) to MTB, IL-12p70 and IL-23 are produced which are critical in TB pathogenesis (Fig. 2.7).
- *The role of IL-23 and IL-17* has emerged recently. IL-23 is essential for expression of both Th17 and IL-17 response to human mycobacterial infection. IL-23 and IL-17 act in a complex manner to control inflammation caused by TB (Fig. 2.7). During TB infection IL-17 can mediate accumulation of both neutrophils and mononuclear cells.
- *Gamma/delta T cells* also play an important role by lysing macrophages with mycobacteria and by producing IFN- $\gamma$  in the initial stages of immune response [34].
- *The CD8+ T cells* can recognize antigens on non-phagocytic cells as epithelial cells.
- *NK cells and B cells* play a role in immunity against TB. NK cells are important in host immune response to MTB. NK cells have the ability to lyse host cells infected with MTB.
- MTB also induces activation of *regulatory T cells (T<sub>reg</sub>)* which have inhibitory and anti-inflammatory functions.
- Another important CD4+ cell is Th17 cells which are proinflammatory cells which mediate immunity against extracellular bacteria and fungi especially at mucosal surfaces [2].
- *Polymorphonuclear cells*: Neutrophils reach the site of MTB infection promptly where they phagocytose bacilli effectively.
- *Humoral immunity*: IFN- $\gamma$  an inflammatory cytokine stimulates the antimicrobial activity of macrophages and regulates antigen presentation

through MHC class II molecules by upregulating mRNA and protein expression [23].

MHC type II-restricted CD-4<sup>+</sup> T cells, MHC class I CD-8<sup>+</sup> T cells, and macrophages are important in the protective immunity against MTB. Decrease in the number or function of these cells results in reactivation of the infection.

Immunity to MTB is associated with Th1 activity through TNF- $\alpha$ , IFN- $\gamma$ , and IL-12 release. TNF- $\alpha$  neutralization reactivates the disease, and genetic defects of receptors for IFN- $\gamma$  or IL-12 increase susceptibility to MTB. Immune response starts with pattern recognition of microbial structures called pathogen-associated molecular patterns (PAMPs). Dectin and TLR4 are important in IL-17 induction by TB.

## Recent Advances in the Diagnosis of Ocular TB

Diagnosis and treatment of active TB are important for preventing blindness [35]. A definitive diagnosis of ocular tuberculosis is made when there is granulomatous eye involvement with the presence of AFB (microscopy or culture) or PCR-based detection of genomic DNA [22, 25]. Only 60% cases with histopathology findings suggestive of ocular tuberculosis may have positive Tuberculin skin test (TST), and 57% may have normal chest x-rays [36].

Demonstration of AFB by microscopy or culture, although standard, is prolonged and cumbersome and may not be positive in low yield of organisms. PCR from intraocular fluids including aqueous, vitreous, subretinal fluid, or rarely chorioretinal biopsy or IS6110 or other conserved sequences in MTB genome although sensitive and extremely useful for early diagnosis of intraocular TB is not recommended due to false positivity [3].

IFN- $\gamma$  release assay which measures IFN- $\gamma$  production by T cells in response to MTB antigen is more specific and rapid than TST. However, it lacks specificity to distinguish between latent and active TB.

Quantitative PCR (qPCR) has been evaluated and found to be useful in making a diagnosis sug-

gestive of active ocular tuberculosis in combination with clinical symptoms [37].

**Compliance with Ethical Requirements** Seema Sen declares no conflict of interest. No human or animal studies were carried out by the author for this chapter.

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