INTRODUCTION
Tuberculosis is known to have been one of the oldest of human diseases. It is as old as human hatred and hypocrisy. It has been appropriately described as "the captain of all the men of death" by John Bunyan (17th century). Few diseases have had such extensive or such intensive impact on the sober consciousness of mankind as tuberculosis. In no other disease has there been such a spectacular reversal of dread and danger as when, for the first time, truly effective remedies emerged on the chemotherapeutic horizon some four decades ago. In another couple of years there was a steep and striking decline in the attack rate. In countries which had the knowledge, the money and the men, tuberculosis was soon listed for ignominious retirement, though alas most of the poorer countries lagged, and still lag, tragically behind. India is one of them. The size and extent of the problems of tuberculosis in children seems to be directly linked to the prevalence of pulmonary tuberculosis among adults in community.

More than 200 years after the first description by SIR ROBERT WHYTT in 1768 tuberculous meningitis continues to be a serious problem. Of all the manifestations of tuberculosis, tuberculous meningitis is the most dreaded complication and a common cause of prolonged morbidity and mortality among children. Not with standing the availability
of potent and specific drugs, fatality rate of tuberculous meningitis still remains high as compared to other forms of tuberculosis in children.

With the advent of antitubercular treatment life, in a child of dreaded disease like tuberculous meningitis, may be spared, but innumerable crippling sequelae are seen. One of the important sequelae is in the form of BLINDNESS — total or partial leaving the patient as a walking and talking HEALTHY CADAVER.

Ocular changes form an important group of clinical manifestations of tuberculous meningitis. Moreover in those who survive, the disease might leave its permanent and dreadful hallmark in the form of partial or total blindness, within the past years cases of these ocular manifestations have been reported in the medical literature, but these complications are still disastrous, as once the sight is lost the patient is handicapped in all activities. Keeping in mind all these facts, early detection of ocular complications and their management in tuberculous meningitis still required a review.

Tuberculous meningitis may produce a variety of changes in the form of intra or extra ocular manifestations having a sinister significance. These ocular changes may either manifest as papillitis, papilledema or optic atrophy or there may be partial ocular pareses.
In the light of these dreaded ocular complications of tuberculous meningitis, the present study was undertaken to find out the prevalence of ocular manifestations of tuberculous meningitis in paediatric patients of Bundelkhand region, to make an early diagnosis and to prevent the dreading sequelae of BLINDNESS by early detection of ocular complications and their management.
REVIEW OF LITERATURE
HISTORY
Tuberculosis and tuberculous meningitis

Tuberculosis, one of the major public health problems in the developing countries of the world today has made its impact felt throughout the ages. The ancient writings of Indian medicine reveal that tuberculosis has been existing in India for more than 2000 years Park and Park (1976) have mentioned that a detailed description of tuberculosis has been found in epics written by past masters like Hippocrates, Charak and Susruta but the cause of this disease has been steeped in ignorance and superstition. The authors have also referred the disease "Captain of the man of death" and "the great white plague". Authors further quote Hippocrates (400 BC) the father of medicine as calling it "phthis" which means "to dry up". Robert Koch disclosed to the world on March 2, the tubercle bacillus.

Robert Whyth (1768) published the first clinical description of tuberculous meningitis in his monogram on "Dropsy in the brain" in children. Meindl (1982) described in his article that 150 years ago, the term TBM was not used. The term used most frequently for children was acute hydrocephalus. Two centuries ago knowledge on the growth and development and health of children was quite rudimentary. In the 1803's the condition known as acute hydrocephalus and
chronic hydrocephalus were viewed as two forms of the tuberculous meningitis. The decisive motion to abandon the use of the term hydrocephalus was made in 1836 by Green. He introduced the term tuberculous meningitis (TBM). Ghai (1977) and Satya Gupta (1978) have commented that tuberculous meningitis is the most dreaded complication of tuberculosis and is the most common cause of death in infants and children suffering from tuberculosis.

**Ocular manifestations**

Duke Elder & Scott (Neuroophthalmology Vol. XII, 1971) have mentioned the work in the field of ocular manifestations of TBM, carried out by the workers in past. The work described in past is as follows -

The condition was produced experimentally by Deutschmann (1881), who injected pus from a tuberculous joint into the cranial cavity of rabbits and produced a clinical picture of papillitis and choroidal tubercles, necropsy showed a wide spread tuberculous meningitis, a perineuritis where in the dural and pial sheaths were studied with tubercles, and a spreading peripheral neuritis. Chiari (1877), Sattler (1878), Sisaric (1921) and Igerscheimer (1925) recorded an analogous process in clinical cases.

Saenger (1879) and Luce (1903) described involvement of cranial nerves in tuberculous meningitis Urbanek (1927) and Frenimesocules (1938) reported that in tuberculous meningitis the sixth nerve is particularly liable to be involved and may be the first sign of the disease.
Incidence

The first national sample survey carried out by Indian Council of Medical Research (1958) showed the prevalence of tuberculosis to be about 7 per 1000 in children 5 to 14 years of age. Of these 0.3 per 1000 were infectious. Subsequent surveys conducted by Pamara (1973) in Delhi and another one at the National tuberculosis institute, Bangalore in the year 1974, have shown a significant and continuous decline in the disease in the children during the last 10 years or so.

Sheth (1961) reported that TBM constituted 4 to 8% of total admissions in pediatric wards. Udani (1961) reported that TBM was the second cause of admissions in preschool children and accounted for 22.1% of total admission in this age group. Bharucha et al (1967) in a 4 year old study conducted at the K.E.M. Hospital, Bombay from 1961-64, reported the incidence of tuberculous meningitis to be 4 percent of all the pediatric admissions of a large teaching hospital. Bhakoo & Gupta (1969) reported the incidence of CNS tuberculosis as 1.8% from a study conducted at Chandigarh. Rao (1972) reported that in different studies conducted in different parts of India, the incidence of TBM was 1-4% of the total in patient admissions.
FACTORs INFLUENCING MORTALITY AND MORBIDITY IN TERMS OF NEUROLOGICAL DAMAGE

In the Indian literature, Manchanda and Lal (1966) reported the maximum mortality (71.9%) and Kapur (1969) reported minimum mortality (17%) in children suffering from TM. Between these two variable results have been reported by various other workers in the field.

Dikshit and Singh (1976) studied one hundred children to find out the various factors that influence mortality and morbidity in terms of neurological damage. It was observed that mortality and morbidity was highest if the child was less than 3 years of age, in IIIrd stage of disease (MRC classification), malnourished, belonged to low socioeconomic group, BCG test negative and suffered from pertussis or measles as a preceding illness or presented as gastroenteritis. In their study poor nutrition cases have more neurological damaged 59% when compared with cases of fair nutrition 29% upto 3 years of age 75% cases had optic atrophy and 63% had mental retardation.

Gupta and Chopra (1981) commented that various factors influencing mortality and morbidity in terms of neurological damage are primarily dependent on the stage of the disease in which the treatment is started. Highest mortality as well as neurological damage occur in the IIIrd stage of the disease. The high mortality and neurological damage in children of low socioeconomic status is probably
due to malnutrition, overcrowded living conditions, ignorance, illiteracy, poverty and failure to get prompt and adequate therapy. Antecedent infections like measles and pertussis have an adverse influence probably because of the depressed immunological status and rapid dissemination of infection.

**PHYSICAL CHARACTERISTICS (CLINICAL FEATURES):**

TEM is usually insidious in onset but may be fulminant if a caseous lesion discharges directly into the subarachnoid space. Heinz & Thomas (1979) grouped the clinical manifestations of TEM into 3 stages.

Stage I (General, nonspecific symptomatology).

Stage II (Appearance of definite neurologic signs), and

Stage III (Coma).

**Stage I** - This is characterised by irritability disinterest in play, idly staring in space, fever, headache, vomiting, anorexia and constipation.

**Stage II** - This is characterised by convulsions, signs of meningeal irritation, cranial nerve palsy and features of bulging anterior fontanelle or crack pot sound over head. Fundus examination may reveal papilloedema and optic disc may show primary or secondary optic atrophy.

**Stage III** - In this stage there is unresponsive opisthotonus. The most common neurologic sequelae are developmental retardation, cranial nerve palsy, hydrocephalus, optic atrophy, deafness, paralysis, continuing stupor or coma, convulsions and pituitary disturbances.
CLINICO-PATHOLOGICAL ASPECT

Rich & Mcrordon (1933) suggested that after the initial bacillemia, granuloma formation (sometimes referred to as Rich foci) occurs in the meninges and for reasons that are unknown, these granulomas break down at a later date in susceptible hosts and tubercle bacilli spills into the subarachnoid space producing meningitis. Macgregor & Green (1937) reported that 78 patients out of 88 cases studied had granulomas in the brain, spinal cord, meninges or in combination of these. Tandon (1973) commented that meningeal exudates give rise to meningeal signs, cranial nerve palsies and hydrocephalies. Lesions in the brain parenchyma causes alteration of sensorium, seizures, hypothalamic symptoms and brain stem disturbances. Arteritis causes vascular obstruction and focal neurologic deficit. Allergy and hypersensitivity cause oedema of the brain.

Gupta & Chopra (1981) described that TB is never a primary manifestation but always occurs as a result of secondary haematogenous dissemination from the site of primary extra cranial tuberculosis lesion which is frequently in the lung. It usually occurs with in the first 6-12 months after the primary infection. As a result of hematogenous dissemination the tubercle bacilli are lodged principally at leptomeninges and brain parenchyma.
Dastur et al (1983) studied 100 autopsied cases of TBM including 78 children. They have shown different mechanisms to produce brain damage. These pathogenic factors are the meningitis itself, the infiltration of the brain by meningeal exudate causing borderzone encephalitis, the involvement of large & small vessels by the meningeal exudate resulting ischaemia and infarction; the internal hydrocephalus ensuing from the blockage of C.S.F. pathways, most commonly the basal cisterns by exudates; the presence of intraventricular exudate constituting open-dymitis and subependymitis; the development of large or small brain tuberculomas and the occurrence of brain oedema.

**Pathogenesis of ocular involvement** -

Boyd (1945) described that interstitial optic neuritis may result secondary to tuberculous meningitis leading to optic atrophy. Taylor et al (1955) commented that optic atrophy results from end arteritis obliterans involving the arteries supplying the optic nerve.

Mooney (1956) observed that the pathologic process in reversible oedema is external hydrocephalus whereas in progressive papilloedema the tuberculous arachnoiditis causes papilloedema by interfering with the circulation of C.S.F. The pale disc results from strangulation of pial vessels going to optic nerve & Chiasma.
Walsh (1957) described that widely dilated pupils became narrowed by lumbar puncture but return to original size soon after. It has been argued that release of pressure by lumbar puncture relieves the cerebral irritation and thus the pupils contract, but with refilling of the ventricles, the pressure rises again and pupils dilate.

Bhatnagar & Srivastava (1961) reported that paralysis of ocular muscles results from infiltration of III and VI nerve at the base of brain or it may result from raised intracranial tension. The aetiology of retrobulbar neuritis is by involvement of the region of optic Chiasma and progression of inflammatory process forward along the optic nerve. Optic atrophy may result from thick inflammatory exudate at the base of the brain surrounding the optic Chiasma and leading to vascular occlusion and fibrosis. It may also result from endarteritis obliterans, organisation of tubercles along the course of blood vessels, due to raised intracranial pressure leading to papilloedema and post neuitic atrophy, extension of surrounding neuroretinitis or by interstitial optic neuritis. The cause of papilloedema is attributed to the result of blockage of the inter meningeal spaces around the optic nerves by the inflammatory process.

Desai & Ankelsaria (1967) observed that the pathological basis of papillitis is opticocochiasmatic arachnoiditis. It is due to further extension of meningitis along with the sheath of the optic nerve. The pale disc is suggestive of reduction in the blood supply whereas bitemporal pallor suggests a compression type of lesion at the level of the Chiasma.
Verma et al (1981) described that pupillary involvement in TBH may be due to the nerve involvement or due to pressure on the brain stem by dilation of third ventricle (Richman's theory). He further suggested that primary optic atrophy may be due to internal hydrocephalus of third ventricle causing pressure on optic chiasma which in turn causes primary optic atrophy.

DIAGNOSIS

The diagnosis of TBH is based on certain supportive and diagnostic criteria. The supportive tests primarily include a positive tuberculin skin test and an X-ray chest showing a primary focus. Lincoln (1947) and Lincoln & Sowell (1963) studied the incidence of positive montoux test in cases of tuberculous meningitis. In both the studies authors found that montoux test was positive in about 85% of cases of tuberculous meningitis. The diagnosis criteria includes a CSF examination. Heinz and Thomas (1979) have reported that white blood cells in CSF are usually fewer than 350/cubic mm and consists mainly of mononuclear cells, but in few cases the fluid showed upto 1000 cells, with a predominence of polymorphonuclear cells. Wright (1953) showed that by the time the disease was well established the sugar was usually below 45mg/100 mg and thought in the early stages protein concentration was normal or slightly elevated but with time it is increased to 300 mg/100 ml or more. Gierson and Marx (1955) studied the significance of chloride estimation in the diagnosis of TBH. These authors reported that the chloride content almost always falls below 115 meq. Thayer (1967) observed that isolation of tubercle bacilli from the CSF varies with different techniques.
% by ordinary centrifuge method, 38.8% by precipitation of deposit from CSF by alcohol and 79% by floatation hydrocarbon technique.

Gupta & Chopara (1981) in their review article mentioned minimum diagnostic criteria for TBM. They reported that the CSF examination revealing raised proteins with low sugar and pleocytosis predominantly of lymphocytes along with the history and clinical picture confirms the diagnosis of TBM. They also mentioned tests, viz. Nitroblue Tetrazolium test, Iromide partition test, Lactate Dehydrogenase Isoenzymes and cerebrospinal fluid protein-ogram, which have been used and found useful in TBM.

OPHTHALMIC MANIFESTATIONS

Tooke (1915) was perhaps the first worker to have done an extensive study on cases of tuberculous meningitis with regard to the various ophthalmological manifestation. He studied 70 cases of TBM and observed that the fundoscopic findings were mainly optic neuritis in 43% of cases, while only one case had presence of papilloedema.

Illingworth and Wright (1948) in their study of tuberculous meningitis and miliary tuberculous reported the presence of choroid tubercles. They divided these cases in three groups. In first group composed of 18 cases of TBM without miliary tuberculosis. In second group there were 28 patients of TBM with miliary tuberculosis while in third group 14 patients of miliary tuberculosis without TBM were
studied. The author observed the presence of choroid tubercles in the three groups to the extent of 15.5%, 64% and 50% respectively.

Mager (1949) has drawn attention to the optic nerve involvement in his study of tuberculous meningitis. He reported optic nerve involvement in 33% of cases. He commented that swelling of the papilla in the initial stages of the disease as being of no importance with regard to diagnosis or prognosis while complication may be expected in the presence of choked disc.

Isente Ivan (1950) studied 300 cases of tuberculous meningitis. He reported in his study a high incidence of ocular involvement in the form of chorioretinitis and disc changes in 95% cases. Dolcet et al (1950) studied 63 cases of tuberculous meningitis. The workers also reported optic nerve involvement in majority of their studied cases.

Arreth & Landarow (1953) carried out yet another study in 100 cases of TM to evaluate the various ophthalmological changes. He observed that out of the 100 children studied 45 cases exhibited chorioretinal lesions, 41 showed alterations of the optic nerve head in the form of 30 cases of active papilloedema, 8 cases of passive papilloedema and 3 cases of optic atrophy. The authors however failed to report any choroid tubercle in their study.

Mooney (1956) in his extensive study of 15 months, studied 65 cases of tuberculous meningitis for
various ocular sequelae of the 65 patients examined 6 died from meningitis. Out of these 65 cases 47 cases (72%) had ocular lesions, some of which were multiple. The author observed papilloedema in 26% of cases, pale discs in 20% and retrobulbar neuritis in 3.1% cases. Ocular palsies were noted in 15.5% cases where as choroid tubercles were reported in 25% of cases.

Laha & Dev (1956) studied 32 cases of tuberculous meningitis for various clinical manifestations of disease. They observed optic nerve involvement in 18.8% of their cases mainly in the form of optic atrophy.

Lincoln and Cordillo (1960) studied 241 cases of tuberculous meningitis and evaluated mainly the extra ocular changes in their study. They divided the cases into the two groups, according to whether they were treated (74 cases) or untreated (167 cases). The authors reported that in both the groups facial palsy was present in 7 cases, while ptosis was observed in 7 untreated cases as compared to only one case in the treated group. Similarly the authors observed a higher incidence of strabismus in untreated (16 cases) than the treated group (10 cases).

Khatua (1961) studied 231 cases for extensive clinical study of tuberculous meningitis. He observed pupillary abnormalities in majority of his studied cases. The incidence of pupillary abnormalities was 76% in his study group.
Bhatnagar & Srivastava (1961) carried out an study to evaluate the various ocular changes in tuberculous meningitis in children. The clinical material comprised of 30 patients, who were admitted in the children medical wards of Hamidia Hospital, Bhopal from March 1960 to February 1961. A detailed clinical examination was done and a thorough ophthalmic check up carried out on admission and repeated at weekly intervals. The various ocular manifestations included pupillary changes in 50.0% cases in the form of dilated & fixed pupil, 16.6%, moderately dilated with sluggish reaction 20% and an isocoria 13.3%, conjugate deviation of eye balls 13.3%; changes in eye grounds in 50.0% of the cases. Optic nerve changes were found in 43.3% of the patients of whom 13.3% had optic atrophy and 16.6% exhibited papilloedema. Choroid tubercles and choroiditis were manifested in 3.3% patients each. Ocular muscle palsies were observed as 16.6% III nerve palsy and 6.6% VI nerve palsy. In their study 33.5% patients had no ocular lesions.

Dutta (1962) studied 2462 children for 'pattern of eye disease in children'. In his studied cases he reported 28 cases of optic atrophy. The common cause attributed to optic atrophy in his study was tuberculous meningitis.

Gupta & Webb (1962) studied tuberculous meningitis in 117 children of south Indian zone to evaluate the clinical manifestations of the disease. They observed pupillary abnormalities in 35% of their cases. The cranial nerve
palsies involving third, sixth and seventh were observed in 15.4% cases.

Miller (1962) studied 38 patients of tuberculous meningitis. He reported 2.7% cranial nerve palsies while analysing the various sequelae of the disease.

Misra and Gupta (1962) carried out another study for ocular complications of tuberculous meningitis they studied 40 patients of TM for various ocular involvement. They reported chorioretinitis in 7.5% cases. The optic nerve involvement was 57.5% in the form of papillitis 35%, papilloedema 7.5%, retrobulbar neuritis 5%, and optic atrophy 29.5%. 20% cases of papillitis, 5% of papilloedema and 15% optic atrophy recovered. The ocular palsies were observed in 30% cases. The oculomotor nerve involvement was present in 12.5% of cases of whom 5% was totally and 7.5% was partially involved. The abducent and facial nerves were involved in 17.5% and 7.5% cases respectively. The workers also observed pupillary abnormalities in 25% cases of which 10% cases were having dilated and fixed pupil, 7.5% sluggish and 7.5% anisocoria (1962).

Bharucha & Talwarkar (1962) in their study for treatment of tuberculous meningitis observed pupillary abnormalities in 24.99% of cases in the form of dilated and fixed pupil.

Verma and Agarwal (1966) carried out an extensive study for ocular manifestations of tuberculous
mенингитис in children. The cases were below age of 16 years and were selected from the patients of S.R.N. Hospital and R.L.M. Medical College, Allahabad. They included 65 cases for the study group. The optic nerve changes observed were papillitis 12.3%, papilloedema 7.6% and optic atrophy 9.2%. The pupillary abnormalities reported were 24.6% in the form of dilated and fixed pupil. The cranial nerves involved in the study were facial in 10.7% cases, abducent in 9.2% cases and oculomotor in 30.1% cases. The workers also observed miliary tubercle of choroid in 1.5% cases.

Banchanda & Lal (1960) studied 249 complicated cases, of 184 out of 742 children suffering from tuberculosis in V.J. hospital Amritsar. They carried out an extensive study for various problems of tuberculous meningitis. The cranial nerve involvement in their cases was having a very low incidence. They observed only 1 case of facial palsy. Optic atrophy was reported only in 6 cases of the total studied cases of TB.

Desai and Ankelsaria (1967) studied 47 cases of tuberculous meningitis with special reference to the ocular aspect. Out of these 37 were from the paediatric and 10 were from other medical wards. Optic nerve affection was seen in 74.46% cases, other cranial nerve involvement was in 25.53% cases and pupillary changes in 63.82% cases. They observed 6th nerve palsy in 14.09% cases out of them in followup study
1 case left against medical advice and others recovered. 3rd nerve palsy was seen in the form of ptosis out of them 50% recovered completely and 50% partially. Seventh nerve palsy was seen in 8.5% cases and all cases recovered completely. Posterior segment changes were found with greater frequency. Pale disc was observed in 12.76% cases out of them 50% remained same, 33.33% progressed to optic atrophy and 16.66% cases expired in follow up study. Papilloedema was observed in 10.63% cases out of them 50% cases cleared up, 25% progressed to optic atrophy and 25% expired. Papillitis was seen in 29.78% cases of them 69.2% cleared, 7.69% progressed to optic atrophy and 23% cases expired. Optic atrophy was seen in 14.89% cases out of which 85.7% cases remained same in follow up study and 14.2% expired. Retrobulbar neuritis was observed in 2.1% cases which all recovered. Bitemporal pallor was reported in 4.2% cases of them 50% cleared and 50% remained same. The workers were not able to report choroid tubercles in their study.

Thapar et al (1968) carried out a study to report ocular manifestations in tuberculous meningitis. They studied 100 cases of tuberculous meningitis from those admitted to the department of pediatrics S.N. Medical College, Agra in years 1965–66. Optic nerve involvement was present in 49%, pupillary abnormality in 12% and cranial nerve palsy in 17% cases. The fundus examination revealed papillitis 25%, papilloedema 13% and optic atrophy 11%. The pupillary abnor-
mality observed was in 12% cases in the form of dilated and fixed pupil. The various cranial nerve palsies were VIIth nerve in 8%, IIIrd in 4% & VIth in 5% cases. The workers also observed nystagmus in 3% cases.

Agarwal and Kumar (1969) in follow up study of treated cases of tuberculous meningitis studied 25 cases. The cases were followed up from 6 months to 3 years. They observed and incidence of 12% blindness. The optic nerve involvement was seen in 44% cases. 32% cases were having visual impairment. They also reported 7th cranial nerve palsy in 28.3% cases.

Rama Chandran et al (1970) studied 288 children of TB selected from 2554 cases of tuberculous patients below 12 years registered in the tuberculosis clinic of Raja Mirasad and Thanjavur Medical College Hospital, Thanjavur, Tamilnadu for the period from 1964 to 1969. They observed blindness in 46% cases as residual sequelae. Cranial nerves involvement was also observed. Facial nerve was involved in 27 cases where as IIIrd and VIth nerve involvement was reported in 1.3% cases each.

Saxena & Tomar (1970) carried out an extensive study of 100 patients of TB for ocular lesions especially. Various ocular manifestations were in 89% of studied cases, 11% being normal. Fundus findings were seen in 55% cases. These included papillitis in 56.4%, papilloedema in 12.7%.
optic atrophy in 29.1% and choroid tubercles in 1.8% cases. The workers observed dilated and fixed pupil in 53% cases cranial nerve palsy were seen in 47% cases out of which VIIth nerve was involved in 23.5% cases where as IIIrd and VIth nerves were involved in 11.7% cases each.

Lothe et al (1972) studied 91 patients of tuberculous meningitis with special reference to complications of the disease. They observed pupillary abnormalities in 80% cases. The cranial nerve palsy was seen in 32.2% cases. They reported optic atrophy in 67.7% cases where as loss of vision was observed in 8% cases.

Udani et al (1974) studied 500 cases of tuberculosis of central nervous system. In their study they also observed for involvement of cranial nerves. Involvement of 2nd nerve was observed in 14.2% cases where as third, Sixth and seventh nerves were involved in 5.4%, 10.8% and 29% cases.

Benakappa et al carried out an study of 50 cases of TB among children who were admitted to the paediatric ward of Vanivilas Hospital Bangalore. They noted fundus changes in 24% cases. Cranial nerve involvement was observed in the form of sixth cranial nerve (20%) and facial nerve (14%).

Smith (1975) made an attempt to study the ocular changes in tuberculous meningitis in children. He observed pupillary abnormalities in 45.2% cases. Various cranial nerve palsy involving 3rd, 6th and 7th were seen in 13% of his cases.
Tamaskar and Bhandari (1976) in their clinicopathological study of meningitis in infancy and childhood studied 50 cases. Out of 50 cases 29 cases were of tuberculous meningitis. The fundus changes were seen in 45% cases in the form of optic atrophy and papilloedema cranial nerve palsies were reported in 14.3% cases.

Idriss & Sinco (1976) studied 43 children suffering from TB who were admitted to the American University Medical Centre of Beirut. They observed optic atrophy only in 9.3 cases. Various cranial nerve palsies were reported in the form of third nerve, sixth nerve and seventh nerve each in 9% cases.

Gupta (1976) studied 50 cases of tuberculous meningitis to evaluate the optic nerve involvement. Out of 50 cases, 25 cases were taken from children ward of civil hospital, Jhansi. Optic nerve involvement was observed in 88% cases. The optic nerve was involved in the form of temporal pallor in 20%, papilloedema in 12%, primary incomplete optic atrophy in 20% and primary complete optic atrophy in 36% cases. It was interesting to note that no case was reported having post neuritic atrophy. Pupillary abnormality was observed in the form of sluggish reaction in 32% cases.

Verma et al (1981) carried out an recent study regarding various, ocular manifestations of tuberculous meningitis and their prognostic value in children. A total
50 cases were selected for this study. Ocular involvement was observed in 70% of total cases. The workers observed conjunctivitis and corneal ulcer in 6% cases each in their study. They have reported pupillary abnormalities in 40% cases. 8 cases showed dilated & fixed pupil, 6 cases semi-dilated pupil, 4 cases sluggish pupil and 2 cases anisocoria. It was evident that mortality was highest in cases with dilated & fixed pupil i.e. 25%. Cranial nerve involvement was a common feature and in this series the ocular motor nerve involvement was seen in 12% of cases, 2 cases showed a complete third nerve involvement while 4 patients showed partial third nerve involvement. Complete involvement showed mortality 16.6% while there was no mortality in partial third nerve involvement. Abducent nerve was also involved in 12% cases 10% cases had unilateral involvement. The patients having abducent nerve palsy showed a mortality of 33.3%. Facial nerve was involved only in 2 cases. 31 cases had optic nerve involvement in this series. Primary optic atrophy was seen in 20% cases, whereas secondary optic atrophy was observed in 12% cases. It was interesting to note that there was no mortality in patients having primary optic atrophy. Temporal pallor was seen in 5 cases Papilloedema was seen in 4 cases, out of which 3 expired thus showing grave prognosis. 6 cases exhibited papillitis out of 6, 2 cases expired.
MANAGEMENT

Steroid:


Steroids in optic nerve involvement—Boyd (1945) described that early detection & judicious therapy instituted at the stage of optic neuritis could prevent occurrence of optic atrophy in most of the cases.

Bhatnagar & Srivastava (1961) observed that treatment of optic atrophy lies in its prevention. Many exudative agents and cortisones were tried for, workers in the field, in treatment of optic atrophy but all except cortisones failed to stand the test of time.
PEROSMOLAR AGENTS

Udani et al (1974) observed that Glycerol 10% in a dose of 1.5 to 2 gm/Kg. Orally or intravenously may help to reduce the oedema over a long period. Mannitol (100 to 200 ml) intravenously twice a day should be tried to reduce the oedema. Gupta & Chopra (1981) described that apart from cortisones, glycerol (0.5–1.5 gm/kg.) body weight) every 4–6 hourly intravenously or orally is useful for chronic treatment of raised intracranial tension. The most frequently used drug is, mannitol (20%, 1.5 – 2 gm/Kg. body weight) by intravenous route, in the management of oedema.

Vasodilators – Keith Lyle (May & Worths manual disease of eye, 3rd ed. 1963) described that vasodilators are useful in the dilation of occluded central artery of retina or its branches. In an inadequate supply to retina we can administer tolazoline hydrochloride 2 mg/ thrice daily orally. It can also be injected retro-ocularly.

RETROOCULAR INJECTIONS

Duke Elder (system of ophthalmology Vol. VII, 517, 1971) have given a description of retroocular treatment. In this volume he has mentioned that the technique was introduced by Weiss & was standardize by Lowenstein. In this technique a long fine needle is inserted into the lower lid at the inferior temporal angle of the orbit so as just to clear the orbital margin and is pushed inwards, medially and slightly
upwards some 3.5 cm in the direction of optic foramen. It is well to aspirate to eliminate the very unlikely point of the needle entering an abnormally large vein. Attempts have been made to introduce the medicaments into the posterior segment of the globe or to reach the optic nerve by this route. Further conditions in which such injections have been employed include the use of vaso-dilators in case of occlusion of the retinal artery and medicaments in optic atrophy.