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Tuberculous optochiasmatic arachnoiditis and optochiasmatic tuberculoma in Malaysia

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Abstract

Background & Objectives: Arachnoiditis which involves the optic chiasm and optic nervecan rarely occurs in the patients with tuberculous meningitis (TBM). The primary objective of this study was to determine the incidence, assess the clinical and neuroimaging findings, and associations, understand its pathogenesis of these patients, and determine its prognosis. *Methods:* The patients admitted with TBM in the neurology wards of two tertiary care hospitals from 2009 to 2017 in Kuala Lumpur, Malaysia were screened. The patients with OCA and optochiasmatic tuberculoma were included in this study. We assessed the clinical, cerebrospinal fluid (CSF), imaging findings of the study subjects and compared with other patients without OCA or optochiasmatic tuberculoma. *Results:* Eighty-eight patients with TBM were seen during the study period. Seven (8.0%) had OCA and one (1.1%) had optochiasmatic tuberculoma. Five out of seven (71.4%) patients with OCA were newly diagnosed cases of TBM. The other two (28.6%) had involvement while on treatment with antituberculous treatment (paradoxical manifestation). The mean age of the patients with OCA was 27.3 ± 11.7. All the OCA patients had leptomeningeal enhancement at other sites. All had hydrocephalus and cerebral infarcts on brain neuroimaging. Three (42.9%) patients had cerebral tuberculoma at sites other than suprasellar and optic chiasm areas. On univariate analysis, the presence of OCA and optochiasmatic tuberculoma was associated with raised CSF opening pressure (p=0.014), younger age (p=0.024), cerebral infarcts (p=0.018) and hydrocephalus (p= 0.046). There was no statistically significant association on logistic regression. Only one (14.3%) patient had visual impairment.
Conclusion: OCA and optochiasmatic tuberculoma were seen in 9% of a cohort of Malaysian TBM patients. They were more likely to be younger, have raised CSF opening pressure, cerebral infarcts and hydrocephalus, suggesting the association with a more severe exudative disease.

Keywords: Tuberculous, meningitis, optochiasmatic, arachnoiditis, tuberculoma

INTRODUCTION

Tuberculous meningitis (TBM) is a central nervous system (CNS) infection which occurs commonly in the developing and underdeveloped countries.[1-3] TBM leads to significant morbidity and mortality.[1,4] TBM is complicated by cerebral infarction, vasculitis, exudates, tuberculoma and hydrocephalus.[5] Inflammatory exudates commonly affect the basal aspect of the brain, which includes prepontine fissure, interpeduncular fossa, suprasellar cistern and optic chiasm, as well as the Sylvian fissures.[4,6]

It has been said that optochiasmatic arachnoiditis (OCA) and optochiasmal tuberculoma develop when a large amount of inflammatory exudates accumulate in the optic chiasm and suprasellar cistern regions[7], and often result in vision impairment.[7-9]

Vision impairment is a rare but severe complication of tuberculous meningitis (TBM).[7-9] Visual loss has been reported in 35% of the patients with TBM in a previous study by Gourie-Devi.[10,11] In other studies, vision impairment affected 25–72% the patients with TBM.[4,7-8,12-15] Visual impairment contributes significantly to disabilities.[12-15]

Visual impairment in TBM may be due to compression, ischaemia or both.[16] The visual

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loss may be attributed by ischaemia of optic chiasm and optic nerve secondary to gradual compression.[9,17] Vasculitis involving the vasa vasorum supplying these nerves can also result in impairment of vision.[9,17] In addition, visual impairment, particularly visual acuity of worse than or equal to 6/18 was a predictor of mortality and severe disability at six months.[8]

Optochiasmatic tuberculoma is observed in approximately 18% of the patients at the time of the diagnosis of TBM.[8,17] Furthermore, optochiasmatic tuberculoma may rarely develop during treatment.[3,18-19] The site of involvement of optochiasmatic tuberculoma is the anterior optic pathway which includes the optic chiasm and optic nerve.[3,18-19]

The primary objective of this study was to determine the incidence of OCA and optochiasmatic tuberculoma, to evaluate the clinical and neuroimaging findings of these patients, thus to understand better its pathogenesis.

METHODS

This was a cohort study on the patients with TBM with prospective follow-up and prospective inclusion of new cases.

All the patients admitted with TBM to the neurology wards of University Malaya Medical Centre and Kuala Lumpur General Hospital from 2009 to 2017, were screened and recruited if they fulfilled the inclusion criteria. University Malaya Medical Centre and Kuala Lumpur General Hospital are two large tertiary referral hospitals in Kuala Lumpur, the capital of Malaysia. The study was approved by the Institutional Review Board of University Malaya Medical Centre the Ministry of Health, Malaysia. All patients or their legally acceptable representatives provided written informed consent for participation.

The patients were recruited if they fulfill the diagnostic criteria of TBM. The diagnosis was based on the modification of Thwaites criteria.[5] All the "definite", "probable" and "possible" TBM patients were recruited.

TBM was defined as "definite" when the cerebrospinal fluid (CSF) mycobacterial culture, acid-fast bacilli direct (AFB) smear, polymerase chain reaction (PCR) or histopathological results were positive.[5] Tuberculous meningitis was categorised as "probable" when the patients had ≥ 1 of the following features: active pulmonary tuberculosis (PTB), acid-fast bacilli in specimens other than CSF or extrapulmonary tuberculosis, such as spinal tuberculosis (TB).[5] Tuberculous meningitis was defined as "possible" when the patients had ≥ 4 of the following features: CSF pleocytosis, lymphocyte predominance in the CSF, CSF glucose/plasma glucose <0.5, turbid CSF, a period of illness lasting for > 5 days, altered conscious level, focal neurological signs, absence of cryptococcal infection or response to antituberculous therapy (ATT).[5,12] The patients with TBM who did not undergo computed tomography (CT) scan of brain within one month of hospitalization were excluded from the study.

The demographic data as well as the data on clinical features, such as the duration of the disease and the clinical presenting features on admission was collected. We also documented the data on the CSF opening pressure, protein, glucose, white cell count (lymphocyte and neutrophil differential count), tuberculous (TB) PCR, AFB smear, mycobacterial culture and sensitivity to antituberculous drugs. The information on extrameningeal TB, such as PTB, spinal TB, TB lymphadenitis and splenic TB was also recorded, as well as the status of human immunodeficiency viral illness (HIV).

The stage of TBM on admission was assessed using the British Medical Council criteria.[5] The TBM patients in stage 1 were fully conscious and rational with Glasgow Coma Scale (GCS) of 15, with meningeal signs but no focal neurological deficit.[5] The patients in stage 2 had GCS score of 11–14 or 15 with focal neurological signs.[5] The patients in stage 3 had GCS score of 10 and less.[5] The clinical progress was evaluated regularly. Advanced stage was defined as stages 2 and 3 of illness.

The patients were given first line ATT. The ATT comprised of an intensive phase consisting of four drugs (ethambutol, isoniazid, rifampicin and pyrazinamide) followed by a maintenance phase of two drugs (isoniazid, rifampicin). The total duration of ATT was 12-18 months according to the British Infection Society guidelines.[20] Steroids were given to the patients with severe TBM.

The Modified Rankin scale (mRS) was used to evaluate the clinical outcome at six months. The mRS ranged from 0 to 6.[21] Good outcome and poor outcome were defined as mRS scores of 0-2 and 3-6 respectively.[21]

The findings of the computed tomography (CT) scan of the brain performed on admission and were recorded. Magnetic resonance imaging (MRI) of brain was with a 3.0-Tesla Signa HDx MR system (GE healthcare). The sequences included T2 weighted (T2W) image, T2 fluid-attenuated inversion recovery (T2 FLAIR) image, T1 weighted (T1W) image without and with

gadolinium contrast enhancement, diffusion-weighted image (DWI), apparent diffusion coefficient (ADC) and magnetic resonance angiography (MRA). Serial CT scan of brain and MRI of brain studies were performed.

The findings of leptomeningeal enhancement at the optic chiasm and suprasellar area (optochiasmatic arachnoiditis) were recorded. The involvement of the other areas with leptomeningeal enhancement, such as Sylvian fissure, prepontine fissure, interpeduncular fossa and cortical areas, was also documented. The radiological findings of cerebral tuberculoma at the optochiasmatic region and other locations were noted and documented. The presence of hydrocephalus and cerebral infarcts, was also recorded.

The visual impairment was categorized according to the standard World Health Organization (WHO) definition for visual impairment.[22] On admission, the pupillary light reflex and funduscopic exanimation were assessed. In the patients in stage 3 of illness, absence of menace reflex and absence of aversion to bright light was classified as "blindness" according to WHO definition for visual impairment.[22] The vision function was classified into four categories, according to the International Classification of Diseases -10 (Update and Revision 2006): 1-normal vision, 2-moderate vision impairment, 3-severe vision impairment, and 4-blindness.[22]

Statistical analysis

All descriptive statistics were done using Statistical Package for Social Sciences, SPSS (Version 21.0, SPSS Inc., Chicago, USA). Chi-square test or Fisher's test was used for categorical data. Continuous variables were analysed with Student's t test. Logistic regression was performed to analyse for the independent clinical and neuroimaging determinants of OCA and optochiasmatic tuberculoma. A p value of <0.05 was taken as statistically significant.

RESULTS

Demography characteristics of the TBM patients

Eighty-eight patients with TBM were recruited and included in the study. 52 (59.1%) patients were males.The mean age of the patients with TBM was 36.4±13.7 (range 16-75). The patients consisted of 35 (39.8%) Malays, 16 (18.2%) Chinese, 21 (23.9%) Indians, and 16 (18.0%) foreigners. The foreigner patients comprised of 9 (10.2%) Indonesians, 4 (4.5%) Myammarese, one each (1.1%) of Filipino, Somalian, and Bangladeshi.

Demography, clinical, CSF characteristics and neuroimaging findings of the patients with OCA

Table 1 shows the demography characteristics of the patients with OCA and optochiasmatic tuberculoma. Eight (9.1%) patients had OCA and optochiasmatic tuberculoma. Seven (8.0%) patients had OCA, whereas one (1.1%) patient had optochiasmatic tuberculoma. All of the patients were HIV negative.

Optochiasmatic arachnoiditis

Out of the 7 patients with OCA, 4 (57.1%) patients had leptomeningeal enhancement at the optic chiasm, whereas 3 (42.9%) patient had suprasellar enhancement. One (25%) patient who presented with enhancement at the optic chiasm, also had enhancement at both the optic nerves, seventh cranial nerve and eighth cranial nerve. Five out of 7 (71.4%) patients with OCA were newly diagnosed cases of TBM. Two out of 7 (28.6%) patients with OCA developed this complication while on ATT (paradoxical manifestation). The 2 patients developed paradoxical manifestation after three months of ATT. Both patients were given ATT and steroids.

Only 1 (14.3%) patient had visual impairment (symptomatic OCA). He had blindness according to WHO classification. Two (28.6%) patients had sixth cranial nerve palsy and 2 (28.6%) patients had third cranial nerve palsy.

All of the patients (100%) also had leptomeningeal enhancement in the other sites of the brain. All the 7 (100%) patients had leptomeningeal enhancement at the Sylvian fissure. Six (85.7%) patients had enhancement at the prepontine cistern and at the interpeduncular fossa. Four (57.1%) patients had enhancement at the quadrigeminal cistern. Two (28.6%) patients had enhancement at the temporal and parietal areas respectively. One (14.3%) patient had enhancement at the ambient cistern.

All the patients (100%) had hydrocephalus and cerebral infarcts on brain neuroimaging. Three (42.9%) patients had cerebral tuberculoma at sites other than suprasellar and optic chiasm areas.

Altogether, 4 out of 7 patients (57.1%) with OCA had extrameningeal TB. Four (57.1%) patients had concomitant pulmonary tuberculosis (PTB). One (14.3%) patient had tuberculous disease of the spine, consisting of psoas abscess and osteomyelitis.

Table 1: **Baseline demographic characteristics and cerebrospinal (CSF) results of the patients with OCA and optochiasmatic tuberculoma**

	Patients with OCA and optotuberculoma (n=8)	Patients with OCA (n=7)	Patient with optotuberculoma (n=1)	p-value
Age (mean±SD)	26.0 ± 11.4	27.3 ± 11.7	17	0.44
Gender (n, %)				
Male	3(37.5%)	3(42.9%)	0	1.00
Female	5(52.5%)	4(57.1%)	1(100%)	
Ethnic group (n, %)				
Malay	2 (25%)	1(14.3%)	1(100%)	1.00
Chinese	1(12.5%)	1(14.3%)	0	
Indian	2(25%)	2(28.6%)	0	
Indonesian	2(25%)	2(28.6%)	0	
Myammarese	1(12.5%)	1(14.3%)	0	
Stage of illness (n,%)				
Stage 1	3(37.5%)	2(28.6%)	1(100%)	0.50
Stage 2	4(50%)	4(57.1%)	0	
Stage 3	1(12.5%)	1(14.3%)	0	
CSF opening pressure, cm H2O (mean±SD)	36.5±14.0	35.1±14.5	46	0.51
CSF protein,g/L (mean±SD)	1.8±0.5	1.7±0.5	2.1	0.45
CSF glucose, mmol/L (mean±SD)	1.2±0.6	1.2±0.7	1.8	0.39
CSF white blood cells, cells/ml(mean±SD)	151.3±	173.0±182.2	0	0.41

Opto=optochiasmatic, OCA=optochiasmatic arachnoiditis

Optochiasmatic tuberculoma

Only one (12.5%) patient had tuberculoma at the optic chiasm area. A 17 year old woman developed optochiasmatic tuberculoma five months after the diagnosis of TBM. However, she was not compliant to ATT in the first five months after the diagnosis of TBM. She had visual impairment (symptomatic OCA). She had moderate blindness according to WHO classification.

Radiologically, she had leptomeningeal enhancement at the temporal and frontal areas bilaterally, prepontine fissure and interpeduncular fossa. She had hydrocephalus and cerebral infarct. Other than optic chiasm, she also had tuberculoma in the other parts of the brain, as well as PTB. She did not have surgical decompression for the tuberculoma. The MRI brain images of the optochiasmatic tuberculoma are illustrated in Figures 1a-1b and 2a-2b.

Clinical and neuroimaging findings between the patients with and without OCA/optochiasmatic tuberculoma

Table 2a shows univariate analysis of the clinical and neuroimaging features among the patients with and without OCA/optochiasmatic tuberculoma. On student's T-test, the presence of OCA and optochiasmatic tuberculoma was significantly associated with higher CSF opening pressure (p=0.014). The patients with OCA/optochiasmatic tuberculoma were also younger (mean age 26.0 ± 11.4 vs 37.4 ±13.6, p=0.024).

On univariate analysis, the presence of OCA and optochiasmatic tuberculoma was also significantly associated with cerebral infarcts (p=0.018) and hydrocephalus (p=0.046). The logistic regression results on the clinical and neuroimaging predictors of OCA/tuberculoma are illustrated in Table 2b. There was no statistically significant predictor on logistic regression.

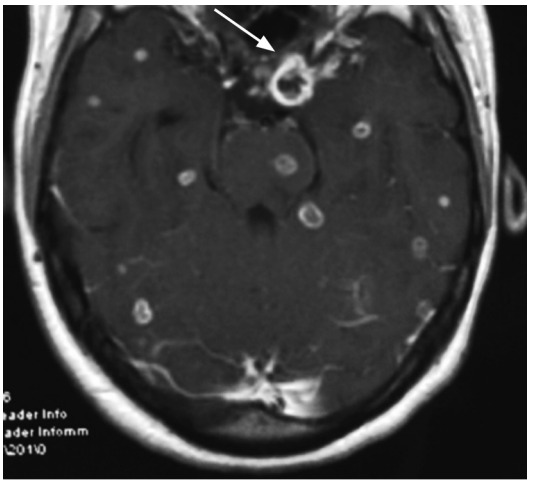

Figure 1a: MRI of brain (axial view) showing optochiasmatic tuberculoma (arrow) andmultiple tuberculoma at both cerebral hemispheres

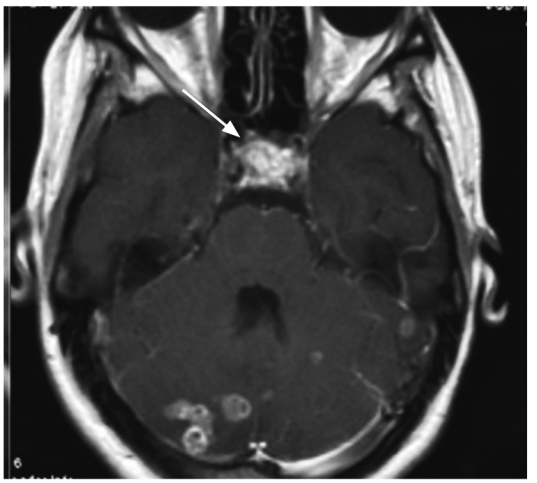

Figure 1b: MRI of brain (axial view) showing optochiasmatic tuberculoma (arrow) and multiple tuberculoma at right cerebellum

Functional outcome at 6 months

The mRS scores at six months for the patients were: 0 in one (12.5%) patient, 3 in one (12.5%) patient, 5 in one (25%) patient and 6 in 4 (50%) patients. All of the patients had mRS 3-6 (poor outcome).

There was no statistical significant association between poor functional outcome with hydrocephalus, advanced stage of illness, visual impairment and raised CSF protein (>1 g/L) on univariate analysis.

DISCUSSION

In this study, 8% of the TBM patients developed OCA, and this was lower than the study by Sinha *et al*. (21.8%).[22] Our study also demonstrated that 87.5% of the chiasmal lesions in the TBM patients were OCA, with 12.5% of the lesions were optochiasmatic tuberculoma. This is similar to the study by Feld *et al*., where 86.7% of the lesions located in the chiasmal region were OCA and 13.3% were tuberculoma.[23]

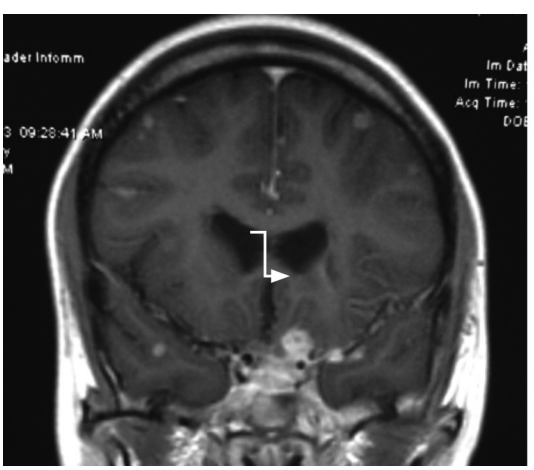

Figure 2a: MRI of brain (coronal view) showing optochiasmatic tuberculoma (arrow)

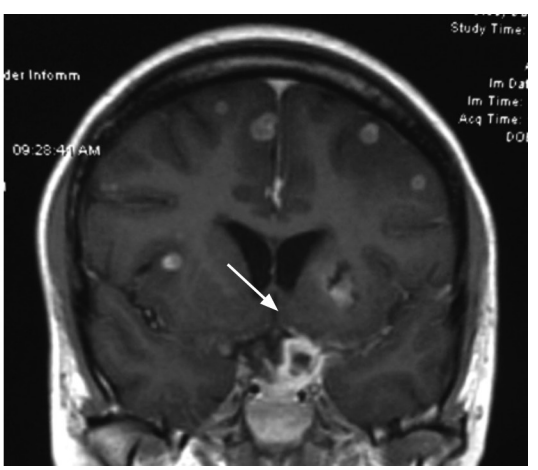

Figure 2b: MRI of brain (coronal view) showing optochiasmatic tuberculoma (arrow)

Table 2a: Univariate analysis of the clinical and neuroimaging differences between the patients with OCA/tuberculoma and without OCA/tuberculoma

	OCA/tuberc present (n=8)	OCA/tuberc absent (n=80)	p-value
Female (n, %)	5(62.5%)	31 (38.8%)	0.26
Age in years (mean±SD)	26.0± 11.4	37.4± 13.6	***0.024***
CSF opening pressure, cm H2O (mean±SD)	36.50± 14.00	22.59 ± 14.13	***0.014***
CSF protein,g/L (mean±SD)	1.78± 0.46	3.85 ± 5.63	0.31
CSF glucose, mmol/L (mean±SD)	1.24± 0.64	2.03 ± 1.25	0.083
CSF white blood cells, cells/ml (mean±SD)	173.00 ± 182.23	128.09± 215.17	0.60
Cerebral infarcts (n, %)	8 (100%)	37 (54.4%)	***0.018***
Cerebral tuberculoma (n, %)	4 (50%)	26 (38.2%)	0.71
Hydrocephalus (n, %)	8 (100%)	42 (62.7%)	***0.046***
Extrameningeal involvement (n, %)	5 (62.5%)	44 (63.8%)	1.00

Tuberc=tuberculoma

In the present study, only 2 out of 88 patients with TBM (2.3%) presented with visual impairment secondary to OCA and optochiasmal tuberculoma. In comparison, visual impairment occurred in 25% of the patients with TBM due to OCA and optochiasmatic tuberculoma in the study by Sinha *et al*.[8] and 35% in a study by Miettinen *et al*.[24]

The current study also showed that one out of seven 14.3% of the patients with OCA (14.3%) had visual impairment (symptomatic OCA), similar to the reported percentage by Aaron *et al*. (14%).[25] In the study by Sinha *et al*., 41% of the patients with OCA had visual impairment.[8]

The patients in our study presented relatively early as 71.4% were newly diagnosed. In contrast, in the study by Aaron *et al*, only 22% of the OCA patients were patients who were newly diagnosed with TBM.[25]

In addition, 28.6% of our study patients had OCA as paradoxical manifestation even though they were administered ATT and steroids. In comparison, Aaron *et al*. reported that 78% of the patients with OCA had paradoxical manifestation while on ATT.[25] Only 3% of their study patients were receiving steroids.[25]

We found the presence of OCA and optochiasmatic tuberculoma to be associated with younger age, high CSF opening pressure, cerebral infarcts and hydrocephalus. The patients with OCA and optochiasmatic tuberculoma were more likely to have cerebral infarcts, possibly due to the inflammatory exudates. We have earlier observed that profound exudates at the optic

Table 2b: Logistic regression of theindependent clinical and neuroimaging predictors of OCA/ tuberculoma

	β	p value	Odds ratio	95% CI
Age	-0.078	0.18	0.93	0.83-1.04
CSF opening pressure	0.06	0.16	1.06	0.98-1.15
Cerebral infarcts	-20.06	1.00	0.00	0.00
Hydrocephalus	-19.53	1.00	0.00	0.00

chiasm and suprasellar regions as evidenced radiologically by leptomeningeal enhancement was more likely to be associated with cerebral infarcts as demonstrated.[26]

Similarly, raised CSF opening pressure and hydrocephalus are also reflective of more exudates at the base of the brain including the optic area. In previous studies, hydrocephalus was frequently found in the patients with optochiasmatic tuberculoma.[7,17,25] The patients with tuberculous OCA had vision impairment which improved after insertion of ventriculoatrial or ventriculoperitoneal shunt.[27-29] This was likely to be due to a decrease in local pressure or compressive effect on the optochiasmal area from hydrocephalus and exudates.[27-29]

Our patients with OCA and optochiasmatic tuberculoma were significantly younger compared to the patients without OCA/ optochiasmatic tuberculoma, similar to previous studies.[7-8,19,25,30] The significantly higher prevalence of OCA among the younger patients may be due to a more robust immune response and exudative disease.[25]

Other risk factor for OCA and optochiasmatic tuberculoma previously noted was raised CSF protein level.[19-20] Sinha *et al*. reported that cranial nerve paralysis, raised CSF protein level and OCA were the predictors of deterioration of vision.[11,12] Raised CSF protein and cranial nerve palsies could all reflect a more severe meningeal inflammation and exudative disease.[17,25]

Early and prompt recognition of OCA and optochiasmatic tuberculoma is important because of the important therapeutic and outcome implications.[4,7,31] In 52% of the patients with OCA, the vision did not deteriorate further with treatment.[25] However, only 17% of the patients showed improvement with treatment. The prognosis of optochiasmatic tuberculoma is poor, particularly when there is a delayed diagnosis and inadequate treatment.[2,32]

Gradual onset and progressive visual impairment in patients with TBM should raise a suspicion of optochiasmatic tuberculoma[2-3,32-33], calling for urgent MRI of the brain with gadolinium.[3,7] Prompt treatment with steroids can be instituted for a longer period of time to reduce the inflammation.[3,7,17,19,31,34] ATT can also be continued for a longer duration.[3,19] Urgent surgical decompression may be considered when there is failure of medical therapy for tuberculoma.[3,19,34]

There were several limitations in this study. Firstly, the small sample size. Secondly, the study was conducted in a tertiary hospital, and therefore the findings may not be generalised to the general population in the community. The current study also failed to identify prognostic or predictive factor after multivariate analysis, probably due to the limited number of patients. The fact that optic chiasmal involvement is associated with factors associated with more exudative disease suggests that future study should include immune response markers.

In conclusion, the patients with OCA and optochiasmatic tuberculoma were more likely to be associated with age, CSF opening pressure, cerebral infarcts and hydrocephalus, all associated with more exudative disease. Early recognition and treatment of OCA may prevent severe visual impairment.

DISCLOSURE

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REFERENCES

1. Donald PR, Schoeman JF. Tuberculous meningitis. *N Engl J Med*2004; 351(17):1719-20.
2. Kalkan A, Serhatlioglu S, Ozden M, *et al*. Paradoxically developed optochiasmatic tuberculoma and tuberculous lymphadenitis: a case report with 18-month follow up by MRI. *South Med J* 2006; 99(4):388-92.
3. Wani NA, Khan N, Kosar T, Qayum A. Optochiasmatic tuberculomas: a vision-threatening paradoxical response in tuberculous meningitis. *Turk Neurosurg* 2012; 22(2):246-9.
4. Chee RI, Dinkin MJ. Tuberculous optochiasmatic arachnoiditis and vision loss. *Neurology* 2016; 87(17):1845.
5. Thwaites GE, Nguyen DB, Nguyen HD, et al. Dexamethasone for the treatment of tuberculous meningitis in adolescents and adults. *N Engl J Med* 2004; 51:1741-51.
6. Misra UK, Kalita J, Maurya PK. Stroke in tuberculous meningitis. *J Neurol Sci* 2011; 303(1-2):22-30.
7. Garg RK, Paliwal V, Malhotra HS. Tuberculous optochiasmatic arachnoiditis: a devastating form oftuberculous meningitis. *Expert Rev Anti Infect Ther* 2011; 9(9):719-29.
8. Sinha MK, Garg RK, Anuradha HK, *et al*. Vision impairment in tuberculous meningitis: predictors and prognosis. *J NeurolSci* 2010; 290(1-2):27-32.
9. Scott RM, Sonntag VK, Wilcox LM, Adelman LS, Rockel TH. Visual loss from optochiasmatic arachnoiditis after tuberculous meningitis. *J Neurosurg* 1977; 46(4):524-6.
10. Gourie-Devi M. Optochiasmatic arachnoiditis

and neurotuberculosis: prognostic indicators and therapeutic strategies. *Neurol India* 2010; 58(5):714-5.
11. Miettinen P, Wasz-Hocket O. Late ophthalmological sequelae of tuberculous meningitis. *Acta Pediatr* 1962; 51:43-52.
12. Misra UK, Kalita J, Srivastava M, Mandal SK. Prognosis of tuberculous meningitis: amultivariate analysis. *J Neurol Sci* 1996; 137:57-61.
13. Kalra V, Ghose S. Optic nerve involvement in tuberculous meningitis. *Indian Pediatr* 1985; 22:783-5.
14. Amitava AK, Alam S, Hussain R. Neuro-ophthalmic features in pediatric tubercular meningoencephalitis. *J Pediatr Ophthalmol Strabismus* 2001; 38:229-34.
15. Mooney AJ. Some ocular sequelae of tuberculous meningitis. A preliminary survey. *Am J Ophthalmol* 1956; 41:753-8.
16. Monga PK, Dhaliwal U. Paradoxical reaction in tuberculous meningitis resulting in involvement of optic radiation. *Indian J Ophthalmol* 2009; 57:139-41.
17. Sinha MK, Garg RK, Anuradha HK, Agarwal A, Parihar A, Mandhani PA. Paradoxical vision loss associated with optochiasmatic tuberculoma in tuberculous meningitis: a report of 8 patients. *J Infect* 2010; 60(6):458-66.
18. Silverman IE, Liu GT, Bilaniuk LT, Volpe NJ, Galetta SL. Tuberculous meningitis with blindness and perichiasmal involvement on MRI. *Pediatr Neurol* 1995; 12:65-7.
19. Joseph M, Mendonca TM, Vasu U, Nithyanandam S, Mathew T. Paradoxical growth of presumed optochiasmatic tuberculomas following medical therapy. *JAMA Ophthalmol* 2013; 131(11):1463-7.
20. Thwaites G, Fisher M, Hemingway C, Scott G, Solomon T, Innes J. British Infection Society guidelines for the diagnosis and treatment of tuberculosis of the central nervous system in adults and children. *J Infect* 2009; 59(3):167-87.
21. Wilson JT, Hareendran A, Grant M, *et al*. Improving the assessment of outcomes in strokes. *Stroke* 2002; 33:2243-6.
22. *Vision impairment and blindness*. *World Health Organisation*. Updated October 2017. www.who.int/mediacentre/factsheets/fs282/en/.
23. Feld R, Sicard J: Sur deuxcas de tubercule du chiasma. *Rev Neurol* 1947; 79:664-6.
24. Miettinen P, Wasz-Hocket O. Late ophthalmological sequelae of tuberculous meningitis. *Acta Pediatr* 1962; 51:43-52.
25. Aaron S, Alexander M, Mathew V, *et al*. Tuberculous optochiasmatic arachnoiditis. *Neurol India* 2010; 58:732-5.
26. Tai MLS, Viswanathan S, Rahmat K, *et al*. Cerebral infarction pattern in tuberculous meningitis. *Sci Rep* 2016; 6:38802.
27. Miettinen P, Wasz-Hocket O. Late ophthalmological sequelae of tuberculous meningitis. *Acta Pediatr* 1962; 51:43-52.
28. Block J. Visual impairment in "low pressure" hydrocephalus. *J Neurol Neurosurg Psychiatry* 1971;34:107 (Abstract).
29. Bhagwati SN. Ventriculoatrial shunt in tuberculous meningitis with hydrocephalus. *J Neurosurg* 1971; 35(3):309-13.
30. Verma R, Patil TB, Lalla R. Tuberculous optochiasmatic arachnoiditis. *J Glob Infect Dis* 2014; 6(2):89.
31. Lima MA, Aversa A, Maranhão-Filho P, *et al*. Optochiasmatic tuberculoma as the sole manifestation of late recurrent tuberculosis. *Rev Inst Med Trop Sao Paulo* 2012; 54(4):229-30.
32. Akhaddar A, El Massani MY. C'hakir N, *el al*. Optochiasmatic tuberculoma: complication of tuberculous meningitis: report of a case and review of the literature. *J Neuroradiol* 2001; 28:137-42.
33. Sharma K. Pradhan S, Varma A. *et al*. Irreversible blindness due to multiple tuberculomas in the suprasellar cistem. *J Neuro-Ophthalmol* 2003:23:211-2.
34. Poon WS, Ahuja A, Li AK. Optochiasmatic tuberculoma causing progressive visual failure: When has medical treatment failed? *Postgrad Med J* 1993; 69(808):147-9.