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Outcome of tuberculous meningitis in children: the first comprehensive retrospective cohort study in Indonesia

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SUMMARY

BACKGROUND: Tuberculous meningitis (TBM) is the most severe form of extra-pulmonary tuberculosis.

OBJECTIVE: To assess hearing, visual, motor function, neurological and mental development outcomes in paediatric TBM.

METHODS: A retrospective cohort study was conducted among 139 children with TBM registered and treated at the Department of Child Health, Dr Hasan Sadikin Hospital, Bandung, Indonesia, from January 2007 to December 2010. Hearing and visual function, appearance of optic disc, motor function, and neurological and mental development were evaluated.

RESULTS: Of a final 128 patients (10 died during hospitalisation, 1 was excluded), 34 (26.5%) died after hospital discharge, the addresses of 58 patients could not be found and 7 parents refused to participate. The remaining 29 patients (16 males, 13 females) were available for evaluation; the mean age was 44 months (range 7–162). Hearing loss and visual impairment were identified in respectively 11/28 and 10/25 patients. Most patients had motor disorders. Delayed neurological and mental development was observed in nearly three quarters of patients, 11 of whom had normal or borderline intelligence quotient.

CONCLUSIONS: TBM causes high mortality and sequelae involving hearing and visual impairment, and neurological and mental development.

KEY WORDS: outcome; TBM; children

ALTHOUGH CHILDHOOD TUBERCULOSIS (TB) contributes approximately 10–20% of all TB cases,[1] rising to 40% in some endemic countries,[2] data on childhood TB in endemic area are rare and inaccurately reported.[3,4] Children are at higher risk of developing severe extra-pulmonary TB, especially meningitis and miliary TB.[5] Of all forms of TB, tuberculosis meningitis (TBM) has been reported to have the highest rates of morbidity and mortality.[6] Limited-resource countries such as Indonesia still encounter many problems in the accurate diagnosis and early detection of childhood TB and TBM. In fact, poor outcome is directly associated with delayed diagnosis and poor treatment adherence.[7]

TBM prevalence among children in some countries is as follows: 214 cases in 8 years (1988–1996) in Turkey,[8] 23 cases in 20 years (1977–1997) in the United Kingdom,[9] 16 cases in 16 years (1982–1998) in Greece,[5] 20 cases in 10 years in California, USA,[10] 554 cases in 20 years in South Africa.[11] Half of the survivors had permanent neurological sequelae, varying from cranial nerve paralysis, ophthalmoplegy, seizure, psychiatric disorders and ataxia to paresthesia, blindness, deafness and mental retardation.[5,8,12] No study covering all aspects of TBM sequelae, including hearing, vision, motor development, neurological and mental disorders, has been published in Indonesia to date.

PATIENTS AND METHODS

Study design and setting

This was a retrospective cohort study among TBM patients admitted between January 2007 and December 2010 to Dr Hasan Sadikin Hospital, Bandung, Indonesia. Each patient's TBM record was reviewed and rechecked against the patient's hospital records for clinical features, results of investigations, treatment and outcome. We prospectively followed the 128 TBM patients post-hospitalisation (Figure).

Patient recruitment

Documented hospital-based TBM records of 139 TBM children were obtained. The parents and/or guardians of the patients were invited to bring the children for further examinations, i.e., physical,

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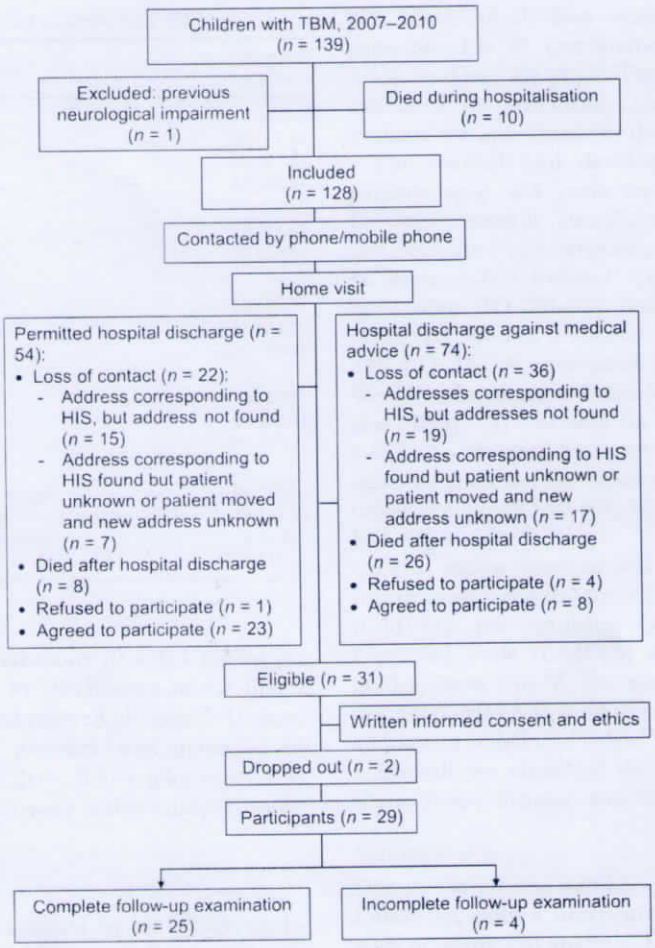

Figure Flow chart of participant recruitment. HIS = hospital information system; TBM = tuberculous meningitis.

hearing, vision, intelligence and motor function tests, after providing written informed consent.

Diagnostic criteria

TBM was diagnosed based on clinical appearance and cerebrospinal fluid (CSF) profile (macroscopically clear, pleocytosis with increased lymphocytes >5/mm^3, elevated protein >40 mg/dl and low CSF/serum glucose ratio <0.6). In addition, ⩾2 of the following criteria had to be present: 1) recent poor weight gain, 2) household contact with sputum-positive acid-fast bacilli (AFB); 3) cranial computed tomography (CT) scan compatible with TBM, 4) chest radiography compatible with TB; 5) positive Mantoux test, 6) AFB-positive clinical specimens (CSF, gastric aspirate or sputum), 7) other body fluids culture-positive for *Mycobacterium tuberculosis*, and 8) clinical response to anti-tuberculosis treatment. The Mantoux test was performed using purified protein derivative RT 23 2 tuberculin unit (TU) solution; an induration diameter of ⩾10 mm (⩾5 mm in severely malnourished patients) was deemed positive. Cranial CT was not performed in all patients.

Clinical staging of TBM

Disease severity was classified as follows: stage I, patients with non-specific symptoms such as fever, anorexia, headache or vomiting, and with no definite neurological manifestations (Glasgow Coma Scale [GCS] 15, with no focal neurological signs); stage II, patients with drowsiness, disorientation and signs of meningeal irritation and/or evidence of increased intracranial pressure (GCS 11–14 or 15, with focal neurological signs); stage III, patients who were unconscious, with paralysis and signs indicating severe intracranial hypertension (GCS < 11).[13]

Treatment

Initial treatment regimens for all patients included daily isoniazid (10–15 mg/kg) and rifampicin (15–20 mg/kg) for 9–12 months, with pyrazinamide (25–35 mg/kg) and streptomycin (20–25 mg/kg) during the

Table 1 Characteristics of 139 children with TBM

Characteristic	Children *n* (%)
Age, months	
<12	30 (21.6)
12–59	66 (47.5)
60–168	43 (30.9)
Sex	
Female	73 (52.5)
Male	66 (47.5)
Malnutrition	
Mild	28 (20.1)
Moderate	32 (23.0)
Severe	27 (19.4)
Stage TBM	
Stage I	9 (6.5)
Stage II	59 (42.4)
Stage III	71 (51.1)
Family income	
Below West Java minimum wage	98 (70.5)
Appropriate to West Java minimum wage	16 (11.5)
Above West Java minimum wage	4 (2.9)
No information available	21 (15.1)

TBM = tuberculous meningitis.

first 2 months. Dexamethasone (0.3–0.5 mg/kg per day) was administered to all children in the first 2 months of treatment, and tapered off over 7–10 days. Increased intracranial pressure was managed by administering mannitol 20% 0.5–1 g/kg every 8 h. Several children underwent ventriculoperitoneal shunt.

Follow-up

Hearing function was assessed by an otolaryngologist-neuro-otology/audiology consultant with the Brainstem Auditory Evoked Response (BAER) method using an EP 15 Interacoustic machine (Interacoustics, Middelfart, Denmark). Degree of hearing loss was determined based on the Pure Tone Average (PTA), and classified as follows: normal, if PTA ≤15 decibels (db); slight, 16–25 db; mild, 26–40 db; moderate, 41–55 db; moderately severe, 56–70; severe, 71–90 db; profound, >90 db.[14]

Visual examination consisted of a visual acuity test, an eye movement test, and the examination of the anterior and posterior segments of the eyeball performed by a neuro-ophthalmologist and a paediatric ophthalmologist. Visual acuity was tested using Cardiff Acuity Cards or the Snellen Chart. The result was classified as normal, moderate and severe (low vision) and blindness, according to World Health Organization classification.[15] Eye movement was examined to evaluate the function of the oculomotor, trochlear and abducens nerves. The anterior segment of the eyeball was evaluated using a slit lamp or a loop/magnifier 3D and a penlight. The posterior segment of the eyeball/fundus examination was performed using direct and indirect ophthalmoscope (Welch Allyn™ type 11500 USA PAT, Welch Allyn, Skaneateles Falls, NY, USA). All children received one drop of cyclopentolate 1% in combination with phenylephrine 2.5% to dilate the pupil. The posterior segment was examined for specifically choroid tubercle and to evaluate the appearance of the optic disc.

The mental and developmental outcomes of patients aged ≤8 years were measured using the Griffiths General Developmental Quotient by a certified neuro-paediatrician. Children were grouped as normal (intelligence quotient [IQ] 90–110), borderline (IQ 70–89), mild mental retardation (MR) (IQ 50–69), moderate MR (IQ 35–49), severe MR (IQ 25–34), profound MR (IQ < 25).[16] The Wechsler Intelligence Scale for Children-Third Edition (WISC-III) was used for children aged >8 years and was performed by a certified paediatric psychologist. Patients were grouped as average (IQ 91–110), below average (IQ 80–90), borderline (IQ 66–79) and MR (IQ < 65).[16]

Gross motor function was measured by a paediatrician using Growth Motor Functional Measurement (GMFM); the resulting Gross Motor Function Classified Scale (GMFCS) was classified into five grades, from I to V. The sequelae were grouped as normal (GMFCS I), mild to moderate (GMFCS II–III) and severe (GMFCS IV–V).[17]

Overall we classified the outcome based on IQ score, vision, hearing, and motor ability.

Statistical analysis

Due to the limited number of patients available for follow-up, only a descriptive study was conducted with no statistical analysis. Data were entered into a Microsoft Excel (MicroSoft, Redmond, WA, USA) spreadsheet and presented as frequencies.

RESULTS

During the study period, 139 patients were recorded in our TBM registry (Table 1). Of these, 128 fulfilled the inclusion criteria; 10 patients died during hospitalisation and 1 was excluded. Most of the patients were taken home against medical advice. Thirty-four (26.5%) children were reported to have died after hospital discharge, and the home addresses of 58 (45.3%) children could not be found. The parents and/or children (36 patients) were invited to Dr Hasan Sadikin Hospital for examination. Two parents opted out of the study because they moved out of the Bandung area (Figure).

Demographic and clinical characteristics

All of the 139 patients were eligible for government medical insurance for poor families. The mean age of the patients was 44 months (range 7–162); 52% were females. Eight of the 139 children (57%) had previously received a bacille Calmette-Guérin vaccination. Nine (6.5%) patients presented with Stage I

Table 2 Clinical characteristics of 29 children with TBM at hospital admission

Characteristic	Children *n* (%)
Stage TBM	
Stage I	1 (3.4)
Stage II	14 (48.3)
Stage III	14 (48.3)
Intracranial hypertension	
Yes	1 (3.4)
No	28 (96.6)
Status epilepticus	
Yes	3 (10.3)
No	26 (89.7)
Contact with adult TB patient	
Yes, AFB-positive	8 (27.6)
Yes, AFB unknown	20 (70.0)
Unknown	1 (3.4)
History of BCG immunisation	
Yes	21 (72.4)
No	7 (24.2)
Unknown	1 (3.4)
Mantoux test	
Positive	6 (20.7)
Negative	23 (79.3)

TBM = tuberculous meningitis; TB = tuberculosis; AFB = acid-fast bacilli; BCG = bacille Calmette-Guerin.

disease, 59 (42.4%) with Stage II and 71 (51.1%) with Stage III.

Treatment outcomes

Of the 29 TBM children followed up, 16 were males and 13 were females. Twenty-eight children presented with Stage II or III TBM in the same proportion, one child had increased intracranial pressure, and three children had status epilepticus (Table 2).

The mean follow-up period for the 29 children included in the study was 21 (11–48) months post hospitalisation. Twenty-five children completed the follow-up examination; 4 parents refused the visual acuity and ophthalmoscope examination (2 adolescent TBM children had given birth, 1 was not cooperative and 1 was not present until the end of the study). Malnutrition was detected in 7/29 TBM children. Permanent neurological sequelae were observed in 24, motor disorders in 16, hearing loss in 13/28 and delayed neurological and mental development in 23. Of 25 patients examined, low vision/blindness was observed in 10 (Table 3).

Of 28 patients, 16 had severe sequelae, while 8 had mild to moderate sequelae. Children with severe sequelae had Stage II and III TBM, while the one patient with Stage I TBM had no sequelae. The majority of the TBM patients had more than one sequela (Tables 2 and 3). Three TBM children dropped out from school due to decreased intellectual ability, and two married and had children. TBM children with mild sequelae were able to receive education appropriate to their intellectual capabilities and to perform daily activities independently. Five study patients with normal and mild sequelae were able to attend regular school, but their performance at school was not confirmed by documented school reports or teacher's appraisal.

Table 3 Treatment outcomes by TBM stage

Outcome	TBM stage I	II	III
Hearing threshold, db (n = 28)			
Normal: 0–15			
Borderline: 16–25	1	9	7
Mild: 26–40	0	3	3
Moderate: 41–55	0	0	1
Moderate-severe: 56–70	0	1	2
Severe: 71–90	0	1	0
Profound: >90	0	0	0
Ophthalmological manifestation (n = 24)			
Visual acuity			
Normal	1	7	7
Low vision	0	1	0
Blindness	0	4	4
Ophthalmic nerve paralysis			
Yes	0	2	1
No	1	12	13
Optic disc atrophy			
Yes	0	3	1
No	1	9	13
Motor function (n = 29)			
GMFCS I	1	7	5
GMFCS II	0	3	3
GMFCS III	0	1	1
GMFCS IV	0	0	0
GMFCS V	0	3	5
Neurological and mental development performance, IQ (n = 28)			
Normal	0	4	2
Borderline	1	4	2
Mild mental retardation	0	1	2
Moderate mental retardation	0	3	0
Severe mental retardation	0	1	5
Profound	0	1	2

TBM = tuberculous meningitis; GMFCS = Gross Motor Function Classified Scale; IQ = intelligence quotient.

DISCUSSION

According to the hospital registry, 139 cases of TBM were diagnosed in the 4 years from January 2007 to December 2010. This is 18 times higher than in industrialised countries[9] and 1.3 times more than in Turkey and South Africa.[8,11] Ten patients died during hospitalisation and one was excluded from the study due to previous neurological abnormality; a final 128 subjects met the inclusion criteria. We contacted the subjects by phone and visited them at home; 58 cases were lost to follow-up and 34 died after hospital discharge (Figure). Possible reasons for the loss of contact were the specific geographic area of Jawa Barat, lack of parental education, low socio-economic status, residence in an informal settlement and lack of permanent housing. Homes were difficult to access due to lack of phones, distance and unreported changes of address. Deaths were also not reported.

The study mortality rate was 32%; other studies

have reported rates of 13–41%.[8,9,18] A study in England reported 170 cases in 8 years, with 41% mortality and 23% physical or mental sequelae.[18] A 2-year follow-up of paediatric TBM patients in India reported a mortality rate of 22%; among the survivors, 47% were neurologically deficient.[19] Our study showed a high mortality rate and a high rate of neurological complications. Our data show that in the past 60 years *M. tuberculosis* has posed serious problems to public health and that there have been no significant changes in mortality or morbidity during this time. The mean age of the children in this study was 4.3 years. Preventive measures should therefore target children aged <5 years.[20]

Most of the patients presented with severe disease requiring admission to the intensive care unit and surgical treatment. The lack of facilities at Dr Hasan Sadikin Hospital and the limited coverage provided by government medical insurance caused many patient families to ignore medical advice and take their children home before proper management could be given.

The high morbidity and mortality, as well as the high number of patients lost to follow-up, showed that children with TBM did not receive sufficient attention, due to inadequate health information systems in developing countries and the lack of importance given to childhood TB by the public health authorities.[4]

Delayed diagnosis and initiation of anti-tuberculosis treatment were confirmed by the fact that 128/139 (92%) patients presented with stage II or III TBM at hospital admission. Missed opportunities for early diagnosis, late presentation and inaccessibility of medical care are the most common causes of delayed diagnosis in childhood TBM.[21] The delayed treatment of TBM usually results in progression to stage II and III disease, with high rates of morbidity and mortality.

A definitive diagnosis with proof of the presence of mycobacteria in the central nervous system is not always possible.[22] In 2010, a uniform case definition for use in clinical research was proposed;[23] however, this case definition was not used in our study, as most of our patients had been admitted before it came into effect. On retrospective review, however, all of our patients were categorised as possible TBM.

Corticosteroids were used to reduce mortality, but their use did not significantly alter the long-term sequelae of TBM as it did not reduce brain oedema, cytokine production or subsequent brainstem encephalopathy. Corticosteroid use improved survival and functional outcomes, but did not improve motor deficit, blindness or deafness.[24–26]

Diagnosis of visual disturbances and unilateral or bilateral abnormalities on BAER due to TBM was multifactorial, and included cortical blindness, the involvement of the cranial nerve due to basal exudates, toxic effects, vascular involvement and intracerebral haemorrhage (ICH). The diversity of these abnormalities and lack of a specific pattern may be due to the diversity of pathophysiological mechanisms in TBM, including hydrocephalus, infarction, tuberculoma and varying degrees of ICH.[27] Loss of vision occurred in 10/25 (40%) patients. Other studies have reported visual disturbances in 14–16% of patients.[9,11] Hearing loss was identified in 11/28 (39%) patients; other studies have reported hearing loss in 7–30% of TBM patients and in 37% of patients with severe sequelae.[9–11] Abnormal BAER scores were found in 56.3% of the children with TBM and correlated with GCS on admission and discharge, stage of meningitis, intracranial hypertension, seizure activity and poor outcome.[27]

The GMFM test was specially designed and validated for measuring gross motor function in children with cerebral palsy (CP).[28] It can be used to measure gross motor function in all types of CP, including diplegia, hemiplegia, tetraplegia, dyskinesia and ataxia subjects.[29] We therefore assumed that GMFM was applicable to our cohort of TBM patients. Motor disorders were found in 16/29 (55%) patients; other studies reported rates of 27–43%.[11] Adequate, regular physiotherapy should be performed among all TBM patients with motor disturbances to diminish motor sequelae.

Delayed neurological and mental development was observed in 22/28 (78%) patients. Other studies have reported delayed neurological and mental development in 13–77% of TBM patients and in all patients (100%) with severe sequelae.[10] Our study found that 12/28 (42%) patients belonged to the normal and borderline categories and were able to attend regular school appropriate to their level of intelligence, but were unable to achieve higher education.

VanWell et al.'s similar retrospective cohort study in 2009 was conducted among TBM patients over 20 years between January 1985 and April 2005, with a larger sample size than our study. Our TBM registry was started in 2007, and our study focused on treatment outcomes in TBM patients. The mean duration of follow-up in the study by VanWell et al. was 6 months vs. 21 months in our study. However, vanWell et al.'s study had no loss to follow-up.[11]

Our study has several limitations. An abnormally large number of patients were lost to follow-up. Thirty-four patient addresses were later found, but the patients were reported to have died after hospital discharge. This is not surprising; Deery et al. also found that only 35% of patients in a developing country could be successfully traced using a home tracing programme.[30] The small number of patients who completed the study means that the study findings are not fully representative. We had no data on hearing, vision, motor function or neurological condition before illness; this may have resulted in

bias. All TBM patients should undergo hearing, vision, motor function and mental and developmental assessment during hospitalisation or before hospital discharge, to collect baseline information, and should be followed up at an out-patient clinic to confirm the progression of sequelae and evaluate the treatment given, including physiotherapy.[10]

Treatment outcomes in paediatric TBM remain poor. A comprehensive approach for TBM control should be implemented and early and sustained interventions should be ensured to improve outcome and enhance quality of life. We believe that even the stage III TBM patients in our study could have had optimal growth and development according to their potential had they received proper management during and after hospitalisation.

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Written informed consent for the publication of this case report and any accompanying images was obtained from the parents of all patients.

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Conflicts of interest: none declared.

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RESUME

CONTEXTE : La méningite tuberculeuse (TBM) est la complication extrapulmonaire la plus grave de la tuberculose.

OBJECTIF : Evaluer les conséquences de la TBM pédiatrique en matière d'audition, de vision, de fonction motrice, de développement neurologique et mental.

MÉTHODE : Une étude rétrospective de cohorte a été réalisée sur 139 enfants atteints de TBM qui ont été enregistrés et traités dans le service de santé des enfants de l'hôpital Dr Hasan Sadikin, à Bandung, Indonésie, de janvier 2007 à décembre 2010. La fonction auditive, l'acuité visuelle, l'apparence de la papille optique, la fonction motrice et le développement neurologique et mental ont été examinés.

RÉSULTATS : Parmi les 128 patients (10 sont décédés pendant l'hospitalisation, 1 enfant a été exclu), 34 (26.5%) patients sont décédés après la sortie de l'hôpital, 58 patients n'ont pas pu être localisés et 7 parents ont refusé de participer. Le reste des 29 patients (16 garçons, 13 filles) étaient disponibles pour l'évaluation ; l'âge moyen était 44 mois (fourchette 7–162). Une perte auditive et une altération de la vue ont été identifiées chez 11/28 et 10/25 enfants, respectivement. La majorité des patients a eu un problème moteur. Un retard de développement neurologique et mental a été identifié chez près de trois quarts des enfants, dont 11 patients qui avaient un quotient d'intelligence normal ou limite.

CONCLUSION : La TBM est à l'origine d'une mortalité élevée et de séquelles en termes d'audition, de vision et de développement neurologique et mental.

RESUMEN

MARCO DE REFERENCIA: La meningitis tuberculosa (TBM) representa la complicación extrapulmonar más grave de la enfermedad.

OBJETIVO: Evaluar los desenlaces clínicos con respecto al desarrollo auditivo, visual, neurológico, mental y de la función motora de los niños con TBM.

METODOS: Se llevó a cabo un estudio retrospectivo de cohortes de 139 niños con diagnóstico de TBM, que se registraron y recibieron tratamiento en el departamento de pediatría del Hospital Dr Hasan Sadikin en Bandung, Indonesia, de enero del 2007 a diciembre del 2010. La función auditiva, la agudeza visual, la apariencia de la papila óptica, la función motora y el desarrollo neurológico y mental se evaluarón

RESULTADOS: De los 128 pacientes (10 fallecieron durante la hospitalización y se excluyó un paciente), 34 fallecieron después del alta hospitalaria (26,5%), no se pudo obtener la dirección de 58 pacientes y 7 padres rehusaron participar en el estudio. De los 29 pacientes restantes que se pudieron evaluar, 16 eran de sexo masculino y 13 de sexo femenino; la edad promedio fue 44 meses (entre 7 y 162 meses). Se detectó una pérdida auditiva en 11 de 28 pacientes y 10 de 25 pacientes exhibieron trastornos visuales. La mayoría de los pacientes presentó un trastorno motor. Se observó un retraso en el desarrollo neurológico y mental en cerca de tres cuartos de los pacientes y 11 presentaron un coeficiente intelectual normal o fronterizo.

CONCLUSION: La TBM provoca una alta mortalidad y secuelas que dan lugar a trastornos auditivos y visuales y afectan el desarrollo neurológico y mental de los niños.