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Tuberculous meningitis in children: problem to be addressed effectively with thorough contact tracing

Received: 17 July 1999 and in revised form: 10 December 1999 and 24 January 2000
Accepted: 31 January 2000

Abstract Worldwide, tuberculosis is the most common cause of death from a single infectious agent in children. There has been an increase in its incidence in Europe in recent years. Early diagnosis of tuberculous meningitis in children is extremely difficult because of its nonspecific early presentation and it is universally fatal if not recognised and treated. Young children are at increased risk of acquiring tuberculosis and it is more likely to be disseminated in neonates. In summarising the cases of three children with tuberculous meningitis admitted to our centre, we highlight the importance of diagnosis and contact tracing of the source case.

Conclusion Diagnosis of tuberculous meningitis requires a high index of suspicion, thorough contact tracing, and appropriate investigations with early treatment as the key to reducing morbidity and mortality.

Key words Tuberculosis · Tuberculous meningitis · Contact tracing · Early diagnosis

Abbreviations AAFB acid-alcohol-fast bacilli · BCG bacillus Calmette Guérin · IVH intraventricular haemorrhage · TB tuberculosis · TBM tuberculous meningitis · TU tuberculin unit

Introduction

In addition to the burden of tuberculosis (TB) in developing countries there is evidence of an increase in the incidence of TB in developed countries [6, 8, 9]. Tuberculous meningitis (TBM) is universally fatal if not treated and has high morbidity and mortality, if not recognised early [4, 8]. The risk of progression of primary TB to TBM is higher in children than adults and is said to complicate 0.3% of untreated primary infections in children [1, 6]. Failure to diagnose TB in adults may have serious consequences for childhood contacts. A high index of suspicion must be maintained to avoid delay in appropriate diagnosis of TBM [3]. We present three recent cases of children with TBM and discuss the contact tracing and public health issues raised.

Case reports

Case 1

A previously asymptomatic, 2-year-old Caucasian girl was admitted to hospital with a 3-week history of pyrexia. During this period, she had been seen by the family physician four times and at the paediatric admission unit once. She had three courses of antibiotics and had been diagnosed as having an upper respiratory tract infection and possible urinary tract infection. Both her parents were employed and her maternal aunt often looked after her. Neither the parents nor aunt worked or lived in a socially deprived area. There were no obvious risk factors for TB evident at that time. She had not had Bacillus Calmette Guérin (BCG) vaccination at birth as she was not in a high-risk group. Clinical examination was normal and in particular she had no neurological signs. Chest X-ray showed right hilar lymphadenopathy and miliary mottling (Fig. 1). In view of the X-ray findings and nonspecific systemic features, a lumbar puncture was performed and CSF showed pleocytosis (213 white
investigations were performed. The chest X-ray showed left hilar lymphadenopathy and right lower lobe pneumonitis. There is generalised miliary shadowing of the lungs.

cells/µl) with predominantly lymphocytes (90%), protein 0.4 g/l, and hypoglycorrhoea (CSF glucose 1.1 mmol/l; blood glucose 4.2 mmol/l). Gram staining of CSF showed no organisms and no acid-alcohol-fast bacilli (AAFB) were seen. Cranial CT scan was not performed at this stage. She was started on anti-tuberculous treatment, isoniazid, rifampicin, pyrazinamide and steroid (prednisolone 2 mg/kg per day, for 6 weeks). Pyrazinamide was given for 2 months. She made good initial recovery but 3 months later was readmitted with pyrexia, lethargy and minimal left ptosis. Cranial CT scan showed a ring enhancing tuberculoma in the left basal ganglia. This was treated medically, with reintroduction of pyrazinamide for 2 months (isoniazid, rifampicin, pyrazinamide) and prednisolone (2 mg/kg per day) for a month, with clinical improvement. Anti-tuberculous treatment was continued for 1 year with good compliance and normal neuro-developmental outcome.

Contact tracing

The 4-year-old sister was diagnosed as having primary TB with hilar lymphadenopathy and right lower lobe pneumonitis on chest X-ray and a positive 10 tuberculin units (TU) Mantoux test. The maternal aunt had had a chronic productive cough for approximately 1 year. She had consulted her general practitioner on several occasions. A chest X-ray taken a year before was reported as showing “old TB” but otherwise normal. This finding was not communicated to the parents. Following the diagnosis in the child, the aunt was admitted to hospital where a further chest X-ray showed hilar lymphadenopathy and right lower lobe pneumonitis on chest X-ray and a positive 10 TU Mantoux test. Following the diagnosis in the child, the aunt was started on chemoprophylaxis with isoniazid after a normal chest X-ray with a positive 10 TU Mantoux test.

Case 3

A 6-week-old Asian baby boy was admitted with a prolonged generalised seizure following a 24 h history of vomiting. There was no history of fever. Antenatal, natal and post-natal histories were unremarkable. There was no history suggestive of accidental or non-accidental injury prior to this illness. His mother had been diagnosed as having smear-positive recrudescent pulmonary TB 3 weeks before and had been started on anti-tuberculous treatment. The family was visited for initial contact tracing by the TB nurse on the morning of admission when the child was reported to be well. He had received BCG vaccination after birth. On admission, the baby was grey and shocked and resuscitated with intravenous fluids. His weight was on the 10th percentile and equal to his birth weight. There was no organomegaly. The chest X-ray showed hilar

lymphadenopathy and the CSF showed pleocytosis (248 white cells/µl, all lymphocytes), protein 0.4 g/l, hypoglycorrhoea (CSF glucose 0.6 mmol/l with blood glucose of 4.9 mmol/l). AAFB were seen in the CSF and confirmed as *M. tuberculosis* on culture. He was started on anti-tuberculous treatment including streptomycin, isoniazid, rifampicin, pyrazinamide and prednisolone (2 mg/kg per day). Two weeks later he was readmitted as he had been unwell for 2 days with vomiting, lethargy, irritability and on examination was ataxic with head circumference at 97th percentile and normal fundoscopy. These features suggested acute hydrocephalus confirmed on cranial MRI scan (Fig. 2) which necessitated a ventriculo-peritoneal shunt. Streptomycin was given for 2 weeks, prednisolone for 6 weeks, and pyrazinamide for 2 months. He had 1 year of anti-tuberculous treatment (isoniazid, rifampicin). He remained ataxic for several months but subsequently made a full neurological recovery.

The mother was diagnosed as having smear-positive pulmonary TB. She had consulted her general practitioner several times over the previous 6 months and on one occasion had suggested the diagnosis of TB based on her general reading about chronic cough. No chest X-ray had been carried out until the week that her son became ill. The sister aged 4 years was diagnosed as having primary TB with hilar lymphadenopathy and a positive 10 TU Mantoux test. The 3-year-old sister was started on chemoprophylaxis with pyrazinamide after a normal chest X-ray with a positive 10 TU Mantoux test.

Contact tracing

The 4-year-old sister was diagnosed as having primary TB with hilar lymphadenopathy and right lower lobe pneumonitis on chest X-ray and a positive 10 tuberculin units (TU) Mantoux test. The maternal aunt had had a chronic productive cough for approximately 1 year. She had consulted her general practitioner on several occasions. A chest X-ray taken a year before was reported as showing “old TB” but otherwise normal. This finding was not communicated to the parents. Following the diagnosis in the child, the aunt was admitted to hospital where a further chest X-ray showed hilar lymphadenopathy and right lower lobe pneumonitis on chest X-ray and a positive 10 TU Mantoux test. Following the diagnosis in the child, the aunt was started on chemoprophylaxis with isoniazid after a normal chest X-ray with a positive 10 TU Mantoux test.

Case 2

An 18-month-old Caucasian boy was admitted to hospital with a 2-week history of low grade, continuous fever with evening rise, sweating and non-specific symptoms (sleepy, miserable, not interested in playing and eating) with head circumference along the 50th percentile for his age. He had not been immunised with BCG. He was diagnosed as having a viral illness and was admitted to hospital overnight. The following day, the mother informed ward staff that she had to attend the adult chest clinic for the results of a chest X-ray and sputum smear. She had had a chronic cough for 6 months and had been referred the previous week for a chest X-ray. The possibility of TB in the child was realised and further investigations were performed. The chest X-ray showed left hilar shadowing of the lungs and mid zone parenchymal changes. There is generalised miliary shadowing of the lungs.

Fig. 1 Case 1. The chest X-ray showing a right paratracheal opacity and mid zone parenchymal changes. There is generalised miliary shadowing of the lungs.

Fig. 2 Case 2. MRI scan showing hydrocephalus and extensive arachnoiditis around the base of the brain.