Tuberculous meningitis with tuberculomata presenting as postpartum pyrexia of unknown origin

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INTRODUCTION

Tuberculosis is uncommon during pregnancy with an incidence of between 12.4 and 94.8 per 100 000 deliveries (Margono et al, 1994). Pregnancy is not a risk factor for reactivation of dormant tuberculous infection following adequate treatment (de March, 1975). Untreated tuberculosis during pregnancy is associated with a higher rate of preeclampsia, vaginal haemorrhage and miscarriage. When appropriate treatment is started early, the prognosis is excellent for both mother and baby (Snider, 1984).

Extrapulmonary tuberculosis constitutes 5–10% of tuberculous infections with similar frequency in both pregnant and non-pregnant patients (Snider, 1984). Tuberculous infections of the CNS typically cause diffuse meningitis or meningoencephalitis, and focal lesions as single or multiple tuberculomata are occasionally seen (Mak et al, 1998). Unlike other tuberculous infections, tuberculous meningitis complicating pregnancy has a high mortality and morbidity (Kingdom and Kennedy, 1989). This article presents an unusual case of tuberculous meningitis with tuberculomata presenting as postpartum pyrexia of unknown origin and the patient's baby having developed congenital disseminated tuberculosis.

CASE REPORT

A 35-year-old Chinese woman had primary infertility as a result of bilateral fallopian tube blockage. She had had uterine fibroids resected 3 years previously. Her precious pregnancy via in-vitro fertilization was complicated by premature rupture of amniotic membrane at 18 weeks' gestation. Two doses of betamethasone 12 mg were given at 29 weeks' gestation, and fetal bradycardia was noted on the next day. A baby girl was delivered by emergency caesarean section. The patient had a high swinging fever following the delivery. Extensive investigations for postpartum pyrexia were normal or negative. She was given courses of broad-spectrum antibiotics for 4 weeks before she developed mental dullness and right partial ptosis. On the next day, she became disorientated with left hemiplegia. Examination also revealed left-sided neglect, dysphasia, conjugate gaze deviation to the right, and left homonymous hemianopia. Computed tomography (CT) of the brain revealed a left temporal hypodense area (*Figure 1a*). Lumbar puncture revealed the following CSF findings: opening pressure of 34 cmH_2O, protein level of 3.8 g/litre, glucose level of 2.1 mM (serum 6.4 mM), cell count of 30 x 10^6/litre (56% lymphocytes, 40% neutrophils, 4% monocytes), and red cell count of 11 x 10^6/litre. Other CSF tests were negative.

Intravenous penicillin G and cefotaxime were started for partially-treated bacterial meningitis. Amikacin was added and penicillin G was replaced with ampicillin on the next day for possible listeriosis. As there was no improvement, empirical antituberculous chemotherapy (isoniazid, rifampicin, pyrazinamide and ethambutol) were added on the following day. Two days later, *Mycobacterium tuberculosis* DNA was detected in the CSF. Magnetic resonance imaging of the brain revealed recent infarctions (*Figures 1b* and *c*). After adding dexamethasone, her fever subsided with gradual improvement over the next 3 months. CSF culture for *M. tuberculosis* was positive. CT of the brain performed after 3 months of antituberculous therapy showed multiple tuberculomata around the basal subarachnoid space (*Figure 1d*) and old infarcts. Upon completion of her antituberculous chemotherapy for a total of 1 year, she recovered with residual left hemiparesis and mild expressive dysphasia. Granulomatous inflammation with Langerhans giant cells was found upon reviewing the histological slides of her uterine fibroid, endometrical sampling and tubal tissue obtained 3 years ago; Ziehl-Neelsen staining was positive. Both the umbilical cord and placenta revealed granulomatous inflammation on histological examination. Her baby girl had fever, pulmonary infiltrates and hepatosplenomegaly. Ziehl-Neelsen staining was positive from the gastric aspirate, lymph node biopsy, and skin biopsy. Antituberculous chemotherapy was commenced at 2 weeks of birth with complete recovery from her congenital miliary tuberculosis.

DISCUSSION

Tuberculosis is endemic in Hong Kong, and the local incidence of tuberculous meningitis is high (Mak et al, 1998). Delayed diagnosis of tuberculous meningitis in pregnant women is common, and the prognosis is poor when the diagnosis is made after delivery (Kennedy and Fallon, 1979). Genital tract tuberculosis is an important cause of infertility, as the fallopian tubes are invariably affected (Vallejo and Starke, 1992). This patient had subclinical tuberculous endometritis and salpingitis as the cause of her infertility, but the subclinical tuberculous infection was not recognized and treated before her in-vitro fertilization.

Reactivation of extrapulmonary tuberculosis occurred during this patient's pregnancy, since her baby developed congenital tuberculosis. Although congenital tuberculosis from endometrial spread is rare (Cantwell et al, 1994), there is histological evidence of tuberculosis affecting the umbilical cord and placenta in this patient. Haematogenous spread from maternal circulation to fetal circulation is also possible. Use of systemic corticos-

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teroids in this patient may have contributed to the flare up of tuberculosis in both the patient and her baby. Reactivation may occur in patients with a previous history of tuberculosis who are given systemic corticosteroids (Mak et al, 1998).

CONCLUSIONS

Tuberculosis, including tuberculous meningitis, should be considered in postpartum pyrexia of unknown origin in endemic regions. Genital tract tuberculosis must be carefully looked for and treated before in-vitro fertilization is undertaken. Early diagnosis and use of antituberculous medications plus corticosteroids are crucial in minimizing mortality and morbidity while maximizing recovery. HM

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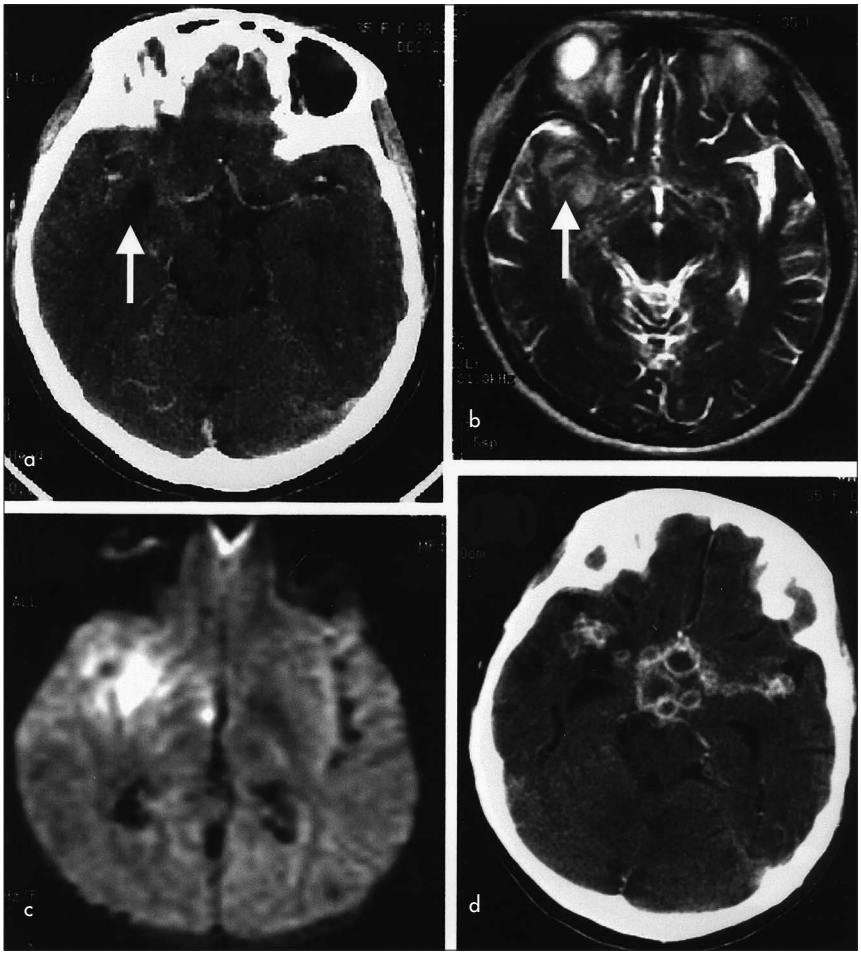

Figure 1. a. Contrast computed tomography brain scan showing a small right temporal lobe infarct (arrow) without contrast enhancement. b. Magnetic resonance imaging showing T2-weighted hyperintense lesion (arrow) over the right anterior temporal lobe. c. Diffusion weighted magnetic resonance imaging showing acute right temporal infarct. d. Contrast computed tomography brain scan showing multiple ring-enhancing tuberculomata.