

Neonatal tuberculosis

Sir,

We were interested to read the short report by Bate, Sinclair, and Robinson as it reminded us of a similar case, previously unreported, seen by us some years ago.¹

A baby girl was delivered by forceps at 36 weeks' gestation to a 19 year old married primigravida, who was a Spanish hotel waitress. After a normal pregnancy she was an emergency admission with what seemed initially to be a pyogenic meningitis but was subsequently shown to be tuberculous meningitis, although culture for acid fast bacilli yielded negative results. Her uncle had almost certainly died from pulmonary tuberculosis.

The baby was separated from her mother at birth in good condition but became jaundiced, with a maximum serum bilirubin concentration of 190 $\mu\text{mol/l}$ on the fourth day. She failed to thrive, developing a series of staphylococcal infections followed on the 22nd day by a fever of 38°C (rectal), pulse 140–172/min, irregular respirations averaging 48/min, fine crepitations in the right lower zone, and moderate hepatosplenomegaly. A chest x ray film (Figure) showed extensive coarse miliary mottling due to miliary tuberculosis. Scanty acid fast bacilli were identified on the Ziehl-Nielsen film from gastric washings, and subsequently *Mycobacterium tuberculosis* was grown on culture. Treatment was started with rifampicin 10 mg/kg/day, isoniazid 20 mg/day, and prednisone 2 mg/kg/day, and she began to gain weight slowly and was well on discharge aged 5 months, soon afterwards emigrating with her mother to Caracas, Venezuela, where she was thriving at 13 months.

Our case shows features of the 'aspiration' type of congenital tuberculosis, as opposed to the 'haematogenous' transplacental type,² with infected amniotic fluid entering the fetal lungs, probably intrapartum,³ and is unusual in that both mother and baby survived.

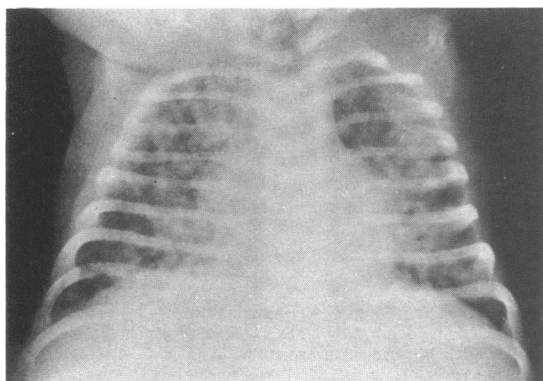


Figure Chest x ray of the case aged 1 month, showing extensive bilateral miliary mottling due to miliary tuberculosis.

References

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- ³ Corner BD, Brown NJ. Congenital tuberculosis: report of a case with necropsy findings in mother and child. *Thorax* 1955;**10**: 99–103.

E S MUCKLOW and R J HALLETT
St Mary's Hospital,
Newport,
Isle of Wight PO30 5TG,
and St Mary's General Hospital,
Portsmouth PO30 6AD

Height measurements at the onset of acute lymphoblastic leukaemia

Sir,

I read the article by Bessho with interest.¹ The author could not find significant height differences between children with acute lymphocytic leukaemia and healthy controls matched for age and sex. This indeed is remarkable, for most other investigators have claimed the opposite, leukaemic children being taller than controls. Yet I am not convinced for the following reasons.

It is known that severe illnesses result in a decrease in growth velocity. This has been investigated also in the case of acute lymphoblastic leukaemia by Berglund *et al.*² The authors observed a decrease in growth before the start of treatment and suggested that the disease might cause the growth failure. Most interestingly, they not only compared height with a reference group at the clinical onset of the disease but also one year before diagnosis. Thus I am sceptical indeed whether single measurements of body height at the instant of the diagnosis of acute lymphoblastic leukaemia in fact represent valid information on growth. Could pre-existing tall stature have vanished by the time of the diagnosis?

Though the role of growth hormone as cited by Bessho is indeed questionable, there is overwhelming evidence for the involvement of polypeptide growth factors not only in the regulation of normal growth but also in growth factor initiated pathways in the aetiology of cancer.³

With respect to this, child growth before the onset of acute lymphoblastic leukaemia is an interesting variable. Bessho's patient group is very large and obviously well matched with its controls. I would like to know more about the patients' growth, not just mean height at one point of their development.

References

- ¹ Bessho F. Height at diagnosis in acute lymphoblastic leukaemia. *Arch Dis Child* 1986;**61**:296–8.