Nigerian Journal of Clinical Practice
Sept. 2009 Vol. 12 (3):319-320

FATAL MASSIVE HAEMOPTYSIS IN AN AFRICAN CHILD

***M A Alhaji, **H Ahmed, ***HAA Umdagas**
*Departments of *Paediatrics, University of Maiduguri Teaching Hospital Maiduguri, Borno State, Department of **Paediatrics Federal Medical Centre, Azare, Bauchi State, ***Department of Radiology, Ahmadu Bello University Zaria, Kaduna State Nigeria.*

Key Words: Massive haemoptysis in an African child. *(Accepted 7 March 2008)*

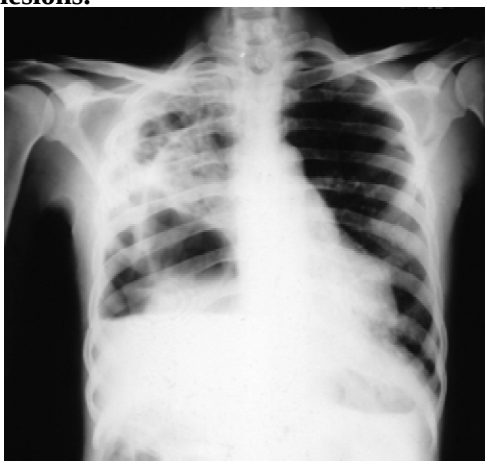
INTRODUCTION

Haemoptysis is rare in children,[1-4] yet is one of the most frightening manifestations of cardio-pulmonary disease. Haemoptysis is defined as coughing up blood, which could be mild, less than 150ml / day, large, between 150 and 400ml / day and massive, more than 400mls / day.[5] Infectious diseases such as tuberculosis is an uncommon but a known cause of haemoptysis in children in developing countries.[4] Massive haemoptysis is rare but associated with fatal outcome[6] and a high mortality,[7] even in best of centres. Consequently, no consensus about its investigation and management has emerged, with the result that much time may be lost in inappropriate investigations and treatment may be misdirected.[5].

CASE REPORT

An 8 year old girl presented with a six-month history of productive cough of copious foul smelling sputum associated with progressive weight loss of three months and difficulty in breathing of two weeks duration. The child had not received BCG vaccination but there was no history of contact with persons with chronic cough. Examination revealed a chronically ill, acyanosed and afebrile child. There was no significant peripheral lymphadenopathy, pedal oedema or digital clubbing. She was wasted and stunted with weight and height of 63% and 65% of expected for age, respectively. The significant physical findings were in the respiratory system with tracheal deviation to the right, dull percussion notes and bronchial breath sounds over the right upper zone with bilateral widespread crackles. There was a soft, non tender, non pulsatile hepatomegaly of 5cm below the right costal margin in the mid-clavicular line and a firm non-tender splenomegaly of 5cm. Other systems were essentially normal. An initial diagnosis of bronchopneumonia with collapse consolidation of right apical lobe, possibly secondary to pulmonary tuberculosis was made with a differential diagnosis of bronchiectasis.The postero-anterior chest radiograph showed tracheal deviation to the right with evidence of collapse consolidation on the ipsilateral side with multiple cavitatory lesions (Fig 1). The heart and the rib cage were normal but the patient was wasted. Sputum was positive on two of the three occasions for acid / alcohol fast bacilli (AAFB) by Ziehl Neelson (ZN) staining. Retroviral screening was negative. Full blood count showed leucocytosis with neutrophilia.Stool parasitology did not reveal any ova or cysts.The patient was commenced on anti-tuberculosis medications (Isoniazid, Rifampicin, Pyrazinamide and Streptomycin). Contact tracing was not vigorously pursued. Clinical improvement was sustained with abating cough, less dyspnoea, and steady weight gain over the four weeks of in-patient management.The patient had two episodes of massive haemoptysis during admission and had to be resuscitated with blood transfusion. Surgical review was sought but conservative management was advised. She was discharged home and was seen twice on follow-up visits. There was clinical improvement on the anti-TB drugs but unfortunately she died at home six weeks after discharge following massive haemoptysis and was brought in dead (BID) to the hospital.

Figure 1: **Chest X-ray of an 8 year old girl showing right sided lung collapse and multiple cavitory lesions.**



DISCUSSION

In Paediatric practice, haemoptysis is an uncommon but potentially serious condition. The risk of primary tuberculous infection developing into active disease in children is between 5% to 15% during the first decade of life after primary infection and depends on several factors such as the host's defences, the age of the child, as the risk is highest during the first 2 years of age as well as the intensity of contact of the child with the

Correspondence: Dr M A Alhaji
E-mail:m_alhaji@hotmail.com

source of infection.[1] poor host's defence system as in immunodeficiency state and / other risks factors facilitates the development of secondary / reactivation tuberculosis. The rarity of secondary / reactivation tuberculosis in children with extensive caseation and liquefaction resulting in cavitations and haemoptysis are documented.[1,2] Pulmonary infection was the leading cause of haemoptysis in children, but *Mycobacterium tuberculosis* is an uncommon causative agent.[4] Congenital heart diseases, trauma, foreign bodies in the airways and other infectious diseases such as pneumonias, aspergilloma or complications such as bronchiectasis are also known causes of haemoptysis world wide.[5,8] However, cystic fibrosis (CF) is the commonest etiologic factor in Europe.[5] Haematemesis may be misinterpreted as haemoptysis, particularly when accurate history is not possible in children. Therefore, bleeding from the upper aero-digestive tract should be studied meticulously using endoscopy.[4] This facility was not available in our centre.Massive haemoptysis in patients with tuberculosis is reported infrequently and almost always in association with aspergilloma or cavitatory disease[5] as in the index patient.The management of massive haemoptysis has passed through various stages in history, ranging form medical to surgical intervention. Patients with haemoptysis of any amount would require aggressive diagnostic evaluation, including bronchoscopic examination. This may help define the site of bleeding and thus permit rapid surgical intervention if haemoptysis persists and increases in quantity.[8] Other investigations that are useful in evaluating a patient with massive haemoptysis include chest radiograph, spiral computerized-scan, fibreoptic bronchoscopy and bronchial arterial angiography, in addition to the baseline full blood count, clotting profile, sputum microscopy, culture and sensitivity and Ziehl-Neelson (ZN) staining. In our setting most of the requisite facilities are not available, making it difficult to localize the bleeding site. That notwithstanding, the bleeding site may not be localized in up to about one third of the patients.[5] We were therefore left with conservative / medical management. Surgical options include lobectomy; pneumonectomy or bronchial artery embolization with absorbable materials.[9] The clinical outcome for massive haemoptysis reflects the generalized nature of a destructive disease process involving both lungs and limited respiratory reserve. Surgery is associated with a high risk of morbidity and mortalities, and should be performed only in selected patients. Postoperative morbidities include prolonged ventilatory support, bronchopleural fistula, empyema and myocardial infarction.

CONCLUSION

Mycobacterium tuberculosis is one of the causative agents of pulmonary infection in children in our environment but rarely associated with haemoptysis. Bronchoscopy and other diagnostic tools for evaluating the site of haemoptysis are scarce in most Resource-poor countries; hence conservative management is the rule. The rarity of haemoptysis in paediatrics practice with its fatal outcome necessitated this report and the need for providing basic facilities for its treatment cannot be over emphasized.

ACKNOWLEDGEMENT

The authors are grateful to colleagues and nursing staff who were involved in the management of the patient. We also thank the management of the Hospital for granting approval for this case report. The criticisms and useful suggestions of colleagues were useful and appreciated.

REFERENCES

1. **Osinusi K.** Tuberculosis in childhood. Update course in Paediatrics, West African College of Physicians, Faculty of Paediatrics, 2002; 1-19.

2. **Aderele WI.** Pulmonary tuberculosis in childhood. Postgraduate Doctor (Africa) 1981; 3: 146-52.

3. **Hendrickse RG.** Tuberculosis: Clinical presentation. In Hendrickse RG, Barr DGD and Mathews TS 1st Ed Paediatrics in the Tropics. London Blackwell (Publishers) 1991: 661-685.

4. **Wong KS, Wang CR, Lin TY.** Haemoptysis in children. Chang Gung Med J 1998; 21:57-62.

5. **Coss-Bu JA, Sachdeva RC, Bricker JT, Harrison GM, Jefferson. LS.** Haemoptysis: A 10 year retrospective study. Pediatrics 1997; 100: 7-10.

6. **Van Kralingen KW, Van Kralin**gen-Heijboer Ac, Zimmerman M, Postmus PE.Management of haemoptysis in a Third World city hospital: a retrospective study. Tuber Lung Dis 1995; 76:344-8.

7. **Patel U, Pattison CW, Raphael M.** Management of massive haemoptysis. Br J Hosp Med1994; 52: 76-8.

8. **Abal AT, Nair PC, Cherian J.** Haemoptysis: aetiology, evaluation and outcome a prospective study in a third-world country. Respir Med. 2001; 95(7): 548-52.

9. **Middleton JR, Sen P, Lange M, Salaki J, Kapila R, Louria DB.** Death producing haemoptysis. Chest 1977; 72: 601-4.

10. **Lee TW, Wan S, Choy DK, Chan M Arifi A, Yim A P.** Management of massive haemoptysis: a single institution experience. Ann Thorac Cardiovasc Surg. 2000; 4: 232-5.