CLINICAL IMAGES

Proximal oesophageal strictures in a child with HIV disease

William van der Merwe, Richard Pitcher, Ekkehard W Zöllner

The patient and her twin brother were born preterm by caesarean section. Her birth weight was 2 100 g. She had perinatal exposure to HIV, and was therefore bottle-fed with term formula milk. She remained well until the age of 11 months when, weighing 6 880 g, she required hospital admission for a right upper lobe pneumonia, which responded well to oral amoxicillin. No causative organism was isolated. There was no evidence of active tuberculous infection on gastric washings or Mantoux test. She was discharged from hospital well, on a mixed diet of Pellargon 250 ml five times daily and solids.

At age 20 months, at a weight of 7 500 g, she required admission for 3 days to evaluate poor feeding and failure to thrive. She had clinical herpes stomatitis which received no specific treatment and began resolving during her hospital stay. An enzyme-linked immunosorbent assay (ELISA) confirmed the presence of HIV infection. She was discharged home on a mixed diet, with dietary supplements, for follow-up at the nutritional clinic.

A month later she was admitted again. Her weight had dropped to 6 800 g and she had signs of kwashiorkor, attributed to inadequate diet, persistent diarrhoea and progressive HIV disease. There were clinical features of oral candidiasis and herpes gingivostomatitis, treated with nystatin and oral aciclovir respectively. For the first 10 days of this 3-week hospital stay she was fed by nasogastric tube. After its removal she sometimes vomited feeds immediately after eating. This was attributed to ongoing diarrhoeal disease, which had resolved before her discharge at a weight of 7 090 g.

Over the next 2 weeks her mother brought her to the outpatient department three times because she would only take fluids and was vomiting all solids. Severe oral candidiasis was noted and oral fluconazole commenced, with a presumptive diagnosis of oesophageal candidiasis. She was readmitted on her third outpatient visit. Following failed attempts to pass a nasogastric tube she was referred to the gastroenterology unit at Red Cross Children's Hospital, with a presumptive diagnosis of oesophageal stricture.

A barium meal examination (Fig. 1) documented three tight strictures in the cervical oesophagus, a morphologically normal thoracic oesophagus and no evidence of gastro-oesophageal reflux. The proximal stricture could not be negotiated with an appropriate paediatric Olympus endoscope and did not allow passage of the smallest Rusch dilator. A feeding gastrostomy was performed and retrograde dilatation attempted without

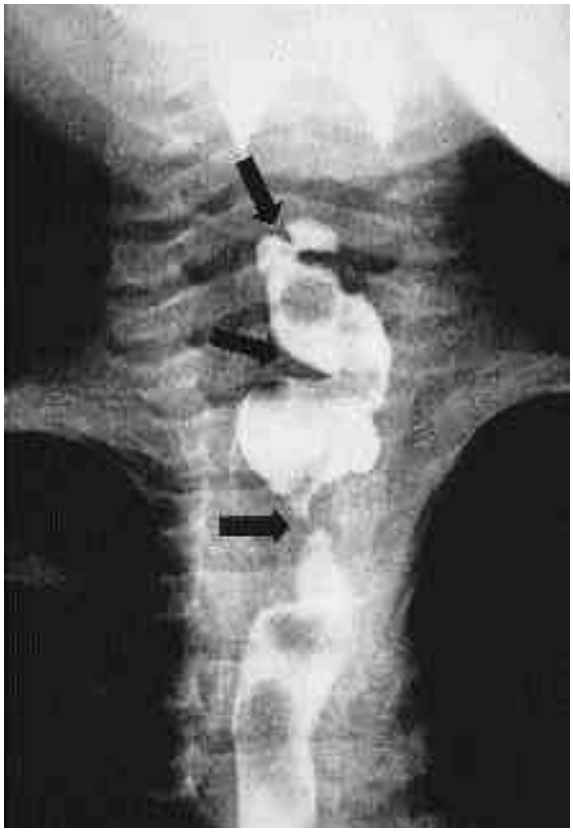

Fig. 1. Barium swallow: AP projection demonstrating three tight strictures in the cervical oesophagus (arrows).

William van der Merwe is Senior Registrar in the Gastro-intestinal Unit at Red Cross War Memorial Children's Hospital and the School of Child and Adolescent Health of the University of Cape Town, Richard Pitcher is Head of Paediatric Radiology at Red Cross Hospital and the School of Child and Adolescent Health, and Ekkehard Zöllner is Senior Specialist and Community Paediatrician at Conradie and Red Cross Hospitals and the School of Child and Adolescent Health.

success. Biopsy specimens of the proximal stricture showed a normal squamous mucosa with chronic inflammatory cells in the submucosa. No viral inclusions, micro-organisms or granulomas were noted.

Discussion

Half of all patients infected with HIV can expect to develop oesophageal symptoms during their illness.[1] Despite this prevalence, the nature of oesophageal disease in this clinical setting remains poorly understood and documentation in children is limited.[2] Our knowledge is largely extrapolated from adults. Bonacini and co-workers[3] produced a landmark study showing that although two-thirds of oesophageal symptoms in HIV disease reflect opportunistic infections by *Candida albicans*, cytomegalovirus (CMV) or herpes simplex virus (HSV), often in combination, the remainder of symptoms were idiopathic.

Wilcox[4] has provided the most definitive work on stricture formation, showing this to be rare, occurring in only 8% of cases. He showed that although *CANDIDA* is the most prevalent pathogen, it alone is not implicated in stricture formation and that while CMV or HSV were implicated in 60% of strictures, the remaining 40% were idiopathic.

Thus, in HIV oesophagitis, dual opportunistic infections are common, strictures are rare and a significant minority of cases remain idiopathic on current diagnostic criteria.

In considering the cause(s) of multiple proximal stricture formation in our young patient, we could find no comparable case in the medical literature. In the absence of definitive virological studies and faced with nonspecific histology, any diagnosis is presumptive.

However, based on the available literature, candidiasis as the sole cause of these strictures can be excluded, while its role as a dual pathogen seems well founded.

A viral aetiology is a real consideration, with HSV arguably the likely organism. The child had herpes stomatitis documented clinically on two occasions. The association between herpetic oropharyngeal lesions and oesophagitis is well recognised[5] and a presumptive diagnosis of herpes oesophagitis with subsequent mid-oesophageal stricture formation in an immunocompetent child has its precedent in the report by Heydenrych *et al.*[6] However, one cannot completely exclude unrecognised CMV infection as a cause, either alone or in association with HSV, considering Wilcox's figures showing CMV to be the commonest cause of both ulcerative oesophagitis and oesophageal stricture formation in adults with AIDS.[4]

There is nothing to suggest that these strictures are unrelated to the patient's HIV disease. There is no history of corrosive ingestion. The absence of gastro-oesophageal reflux and hiatal hernia on barium study, together with the number and location of the strictures make Barrett's oesophagus unlikely. The patient's white cell count and differential count were normal, thus eosinophilic oesophagitis was not considered. There were no clinical stigmata of pemphigoid or epidermolysis bullosa.

Conclusion

Oesophageal disease in patients with HIV disease is common. The majority of symptoms will reflect opportunistic infections by *Candida albicans*, HSV or CMV, allowing empirical therapy and rapid resolution of symptoms. Failure of symptoms to respond to antifungal and antiviral agents should prompt referral for further assessment. In such cases endoscopy may be warranted for a definitive diagnosis, allowing specific treatment and prevention of complications including stricture formation. The challenge of diagnosing dysphagia in young children will only be met by a high index of suspicion in any patient with persistent vomiting.

1. Bonacini M, Young T, Laine L. The causes of esophageal symptoms in human immunodeficiency virus infection. A prospective study of 110 patients. *Arch Intern Med* 1991; **151:** 1567-1572.
2. Chiou CC, Groll AH, Gonzalez CE, *et al.* Esophageal candidiasis in paediatric acquired immunodeficiency syndrome: clinical manifestations and risk factors. *Pediatr Infect Dis J* 2000; **19:** 729-734.
3. Bonacini M, Young T, Laine L. Histopathology of human immunodeficiency virus associated esophageal disease. *Am J Gastroenterol* 1993; **44:** 549-551.
4. Wilcox CM. Esophageal strictures complicating ulcerative esophagitis in patients with AIDS. *Am J Gastroenterol* 1999; **94:** 339-343.
5. Springer DJ, DaCosta LR, Beck IT. A syndrome of acute self-limiting ulcerative esophagitis in young adults probably due to herpes simplex virus. *Dig Dis Sci* 1979; **24:** 535-544.
6. Heydenrych JJ, Keet AD, Mare JB, Becker WB. Herpesvirus hominis oesophagitis and esophageal stricture. *S Afr Med J* 1980; **58:** 176-178.