environmental water sources, and from sterile aqueous solutions used in treatment or in diagnostic procedures.[4]

We report a nosocomial outbreak of *A xylosoxidans* related to the administration of an intravenous computed tomography contrast solution to five immunocompetent patients. In the first two hours after the perfusion of this contrast solution all the patients developed high fever with chills, frontal headache, vomiting and hypertension. Four patients had no evidence of focal infection but one had clinical and radiological evidence of lobar pneumonia. Two days after hospital admission four patients developed perioral vesicles. From blood cultures of three patients we isolated Gram negative non-fermenting rods, identified as *A xylosoxidans* by colony morphology and biochemical tests.[2] The patient with lobar pneumonia received antibiotics on admission (ampicillin and tobramycin).

When the results of blood cultures were available all the patients were treated with azlocillin (3 every 4 hours); the fever disappeared after 24 hours of treatment, and the clinical evolution of all patients was favourable.

In 1971 Yabuchi and Ohyama reported the isolation of *A xylosoxidans* from ear discharge of seven patients with chronic otitis media.[5] The pathogenic importance was difficult to establish because it was mixed with other micro-organisms. *A xylosoxidans* is a rare cause of bacteremia, and in the hospital environment it must be regarded as an opportunistic micro-organism that may infect immunosuppressed patients and those receiving antibiotics.[3] Nosocomial outbreaks that occurred in this group of patients are usually associated with an aqueous source (distilled water, deionised water, dyalisis fluids, tracer solutions, contrast solutions chlorhexidine solution), and the infection causes substantial mortality.

Data on antimicrobial susceptibility of *A xylosoxidans* are limited, but most strains are susceptible to carbenicillin and trimethroprim-sulfamethoxazole, and are resistant to penicillin, ampicillin, most cephalosporins, and the aminoglycosides. Susceptibility to chloramphenicol, colistin, and tetracycline is variable, so it is recommended to make an antibiogram of all isolated strains.

When a strain of *A xylosoxidans* is isolated from superficial lesions it is difficult to establish if it has a pathogenic role, but if the isolation is from blood and in a group of patients with normal immune status, the importance is greater. In our nosocomial outbreak the only risk factor common to all the patients was the contrast solution so we believe that the clinical symptoms of the patients were related exclusively to the contamination of the contrast solution with a strain of *A xylosoxidans*.

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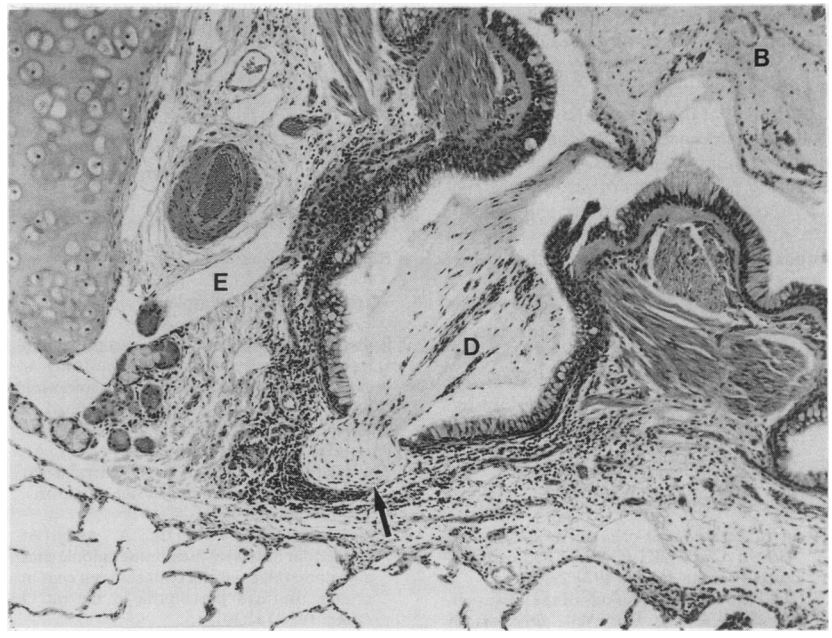
Bronchial diverticulitis: complication of bronchial asthma

The characteristic histopathological features which have been documented in bronchial asthma are smooth muscle hypertrophy, basement membrane thickening, mucus plugging, goblet cell metaplasia, epithelial cell desquamation and peribronchial inflammatory cell infiltration.[1,2] In this report of two cases we describe an additional histopathological feature which is of clinical relevance—bronchial diverticulitis.

Case reports

A 26 year old woman died 12 hours after hospital admission with acute severe asthma, complicated by subcutaneous emphysema and bilateral pneumothorax which followed mechanical ventilation. Haematoxylin and eosin stained sections of the lungs which had been inflation fixed with 10% buffered formaldehyde before cutting, showed multiple mucosal diverticula-like outpouchings from the large airways. There was pronounced inflammatory cell infiltration around these diverticula, which were lined by goblet cells and ciliated respiratory epithelium. Examin-

Figure *Photomicrograph of a bronchus (B) from 26 year old woman who died of severe asthma, showing a diverticulum (D) which has ruptured (→), and adjacent interstitial emphysema (E).*



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ation of serial sections showed that one diverticulum had ruptured (figure). This may account for the interstitial emphysema seen histologically and may provide an explanation for the clinical features.

A 13 year old girl developed a precipitous asthmatic attack from which she died a few minutes after the onset of breathlessness. The histological findings of the lungs were again those of interstitial emphysema and numerous bronchial diverticula. Around most of the diverticula there was a pronounced inflammatory cell infiltrate, composed predominantly of eosinophils, plasma cells, and lymphocytes.

Dunnill has suggested that diverticula present in asthmatic airways represent the mouths of mucous gland ducts.[3] We propose that although they originate at the site of origin of these ducts, their evolution is comparable with that of diverticula of the intestine, gall bladder, and urinary bladder. The diverticula develop at points of least resistance in the muscular wall; in the colon this occurs at sites where vessels enter the muscle coat, but in the bronchial wall the weak point is likely to occur at the mouths of the mucous glands.

The features of these disease processes show a striking similarity in that they are all outpouchings of mucosa between muscle. In each, the lining epithelium may ulcerate, and pronounced inflammatory cell infiltration may occur in the mucosa, submucosa and peridiverticular tissues.

Consistent with diverticula at other sites, the cause of the bronchial diverticula is likely to be primarily mechanical, resulting from raised intraluminal pressure and changed smooth muscle contraction.[4] Furthermore, we propose that the bronchial diverticula are of clinical relevance, for not only may the associated inflammatory changes be important in the pathogenesis of airflow obstruction in asthma, but if a diverticulum ruptures, interstitial emphysema may result and complicate the exacerbation of asthma.

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Other correspondence

Multiple slit membranes and proteinuria

I read with interest the article by Harrison, Jenkins and Dick,[1] which recorded the presence of multiple slit membranes having a "step ladder" appearance in renal biopsies before and after transplantation from a patient with focal and segmental glomerulosclerosis.

I have found identical multiple slit membranes in rat kidneys after induction of simple protein overload proteinuria.[2] One reason for their apparent rarity may relate to the fact that they seemed to occur only in glomeruli showing intermediate levels of structural damage and that they are best shown after enhancement of staining by the use of tannic acid. I am convinced that they are the same structures as those described by Ryan, Rodewald, and Karnowsky.[3]

As protein overload proteinuria is not immunologically mediated and unlikely to be a toxic or basement membrane charge effect,[4] it is highly debatable whether the slit membranes are in any way directly related to the initial causation of the proteinuria.

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Immunological abnormalities in myelodysplastic syndrome

Economopaulos *et al*[1] and Multi *et al*[2] have both recently drawn attention to the occurrence of immunological abnormalities in patients with myelodysplastic syndromes (MDS). Among the abnormalities reported were hypogammaglobulinaemia, hypergammaglobulinaemia, monoclonal gammopathy, and tissue autoantibodies. A new finding by the Bournemouth group was of a positive direct antiglobulin test (DAT) in eight of 98 patients.

We have studied 37 patients with various types of MDS presenting to us between July 1985 and October 1987. Eight (21%) had a positive DAT (six IgG, one C_3, one IgG plus C_3). Three of the eight had refractory anaemia (RA), three chronic myelomonocytic leukaemia (CMML), and two RA with excess of blasts (RAEB).

The high prevalence of positive DATs in MDS is not easily explained. A general increase in immunoglobulin production has been described in CMML,[3 4] possibly caused by the release of B cell growth factors from activated monocytes. Alternatively, the Bournemouth group[5] have suggested that an initial oncogenic event selects a clone of stem cells which retains the capacity to differentiate into both myeloid and lymphoid cells, both lineages being marked by functional abnormalities. Whatever the pathophysiological basis of this finding, a positive DAT is of considerable practical importance in a group of patients requiring frequent blood transfusion.

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