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Letters to the Editor

Leucocytic alkaline phosphatase activity, marker of evolution in leprosy?

Lepromatous leprosy is an interesting disease regarding macrophage function and the host's failure to control the disease.[1] The hypothesis that impaired non-specific defences might have a role in leprosy has become increasingly plausible, with speculation that macrophages are unable to present leprous antigens to the immune system. As impaired phagocytosis has been implicated, we undertook a survey of the whole phagocyte population. We chose to study leucocytic alkaline phosphatase (LAP) activity because of the similarity between lepromatous leprosy and mucocutaneous candidiasis: this latter disease, characterised by the persistence of *Candida* in spite of high titres of anticandida antibodies had been reported as showing a substantial decrease in LAP activity.[2]

Blood specimens collected on lithium heparinate were obtained in the French West Indies from 31 patients with leprosy aged between 4 and 73, and 11 healthy controls. All 31 patients had major cutaneous or neurological lesions. They were classified according to Ridley and Jopling's criteria[3] as follows: 14 cases of lepromatous leprosy (eight receiving treatment and six not yet treated); nine with tuberculosis (seven had been treated); three borderline cases under treatment; and five with indeterminate leprosy (not yet treated). Healthy controls were of the same race and social background as the patients.

The blood smears were fixed in formalin (10% in methanol) and stored at 4°C, and processed within three days for LAP activity (technique adapted from Kaplow).[4] The slides were examined microscopically for a positive reaction by semiquantitative evaluation; the formation of a brown intracytoplasmic precipitate was scored from 0 to 4.[4] The final result was expressed as the mean scores obtained from the cytological examination of 100 polymophonuclear cells.

Table *Mean LAP score for each type of leprosy and for controls*

Samples	*(n=)*	*Mean (SEM) t=0·05 Sm*	
Tuberculoid			
Treated	7	44·8 (9·8)*	p<0·001
Untreated	2	55·5 ND	
Total	9	47·2 (11·4)*	p<0·001
Lepromatous			
Treated	8	16·5 (11·4)*	p<0·001
Untreated	6	26·1 (20·9)*	p<0·001
Total	14	20·6 (9·3)*	p<0·001
Borderline			
Treated	3	31 ND	
Indeterminate	5	48·2 ND	
Controls	11	109·1 (12·5)	

*p<0·001

The distribution of the different values showed a significant difference ($p < 0.001$) between patients with leprosy (mean (SEM) score 33·8 (7·3)) and healthy controls (109·8 (12·5)). As there was no significant difference between patients who had been treated and those who had not, they were grouped in the same category as the 14 with lepromatous leprosy and the nine with tuberculosis. These two groups each showed significantly different values compared with healthy controls ($p < 0.001$). Furthermore, there was a significant difference ($p < 0.001$) between the group with tuberculosis (47·2 (11·4)) and the group with lepromatous leprosy (20·6 (9·3)). Although the borderline group is too small to permit statistical analysis, it is interesting to note that its mean score (31) places borderline cases betweeen the scores for tuberculosis and lepromatous leprosy. Finally, the LAP score of those with indeterminate leprosy matches a value distribution which spans all the leprosy types.

This work shows a correlation between LAP activity and leprosy with a progressive decrease of the score from tuberculosis to lepromatous leprosy. As the role of LAP is unknown,[5] we cannot speculate whether it is a cause or consequence of infection. The role of granulocytes in protection against leprosy is dubious, and changes in granulocyte enzyme activity are likely to be epiphenomena, but we think that the LAP score might have a predictive value for the evolution of indeterminate leprosy. In this respect it is interesting to note that the lowest score we obtained was from a patient initially diagnosed with indeterminate leprosy that proved later to be lepromatous. This test is cheap and easy to perform, and it might be interesting to evaluate LAP score in those with indeterminate leprosy as well as in the follow up of those with borderline leprosy.

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Troublesome Romanowsky stain deposits

These deposits may be removed from blood films by the following simple method. A sheet of cellulose tissue is placed on the slide overlapping one end. Two or three drops of a mixture of xylene, three parts, and 74 OP ethanol, one part, are run on to the tissue so that it adheres to the slide. The tissue is then drawn off the slide by pulling in the plane of the film. In this way the whole film is wetted and gently wiped by the tissue. The xylene appears to temper the solvent action of the ethanol so that staining of the blood film is unaffected even by repeated treatments.

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Nosocomial outbreak of *Achromobacter xylosoxidans* associated with a diagnostic contrast solution

Achromobacter xylosoxidans is a Gram negative rod, strictly aerobic, oxidase and catalase positive, motile by peritrichous flagella and which does not attach to carbohydrates. Taxonomically, it is recognised by the Centers for Disease Control as a genus[1] and is subdivided into two species, *Achromobacter xylosoxidans* and an unnamed species designated Vd. *Bergey's Manual of Systematic Bacteriology* still does not accept *Achromobacter* as a genus name and it is listed as *Alcaligenes denitrificans* biotipe *xylosoxidans*.[2] The natural habitat of this organism remains unknown, but it has been isolated from many clinical sources (cerebrospinal fluid, blood, urine, pleural and peritoneal fluid, faeces, wounds, pharynx, and sputa),[3] various hospital and

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).*