HIV and tuberculosis

To the Editor: In this edition of the *SAMJ* we report that the prevalence of HIV infection among adults diagnosed with tuberculosis in the Hlabisa health district of KwaZulu-Natal was 36% (95% confidence interval (CI) 31 - 42%) in late 1993.[1] To determine temporal trends in HIV prevalence among this key indicator group we repeated the survey earlier this year. HIV testing was done at the Edendale Hospital laboratory as described.[1]

During the month of April 1995 all adults diagnosed with tuberculosis were offered voluntary HIV testing and counselling; 63 patients were so diagnosed, and 41 were tested for HIV infection. Seven patients declined testing, 1 died shortly after admission before being counselled and was not tested, and the other 14 were not tested owing to procedural errors on the ward. The mean age of the patients was 35 years and the male/female ratio was 1.6:1. The patients who were not tested were similar to those tested (age and sex), and while disappointed that not all eligible patients were tested, we do not believe that undue bias was introduced by this (essentially random) failure.

Of the 41 patients tested, 24 were HIV-positive (58.5%, 95% CI 43.1 - 72.8). The prevalence in women was 73.5% and that in men 50%, confirming the higher rates in women we reported previously.[1] The prevalence of HIV infection in adults with tuberculosis has risen from 36% to 58.5% ($P = 0.006$) in less than 2 years — this in a part of the country where the prevalence of HIV infection in women attending antenatal clinics is currently around 14% (in preparation) and is doubling every 16 months.

There has been an almost threefold increase in the tuberculosis caseload at this hospital over the last 4 years, patients are much sicker with HIV-related diseases, and the mortality rate is increased. We have seen tuberculosis become a quite different disease with differing epidemiology, differing clinical picture and progress, and difficulties with diagnosis. We believe that this means that a somewhat different and particularly vigorous approach to tuberculosis control is needed.[2] While we can — and must — learn from the lessons of other sub-Saharan countries[3] we must develop our own novel solutions to this 'new' disease of HIV-related tuberculosis. At Hlabisa, despite our increased workload, we are maintaining completion-of-treatment rates of around 80%.[4] Furthermore, we believe that patients with tuberculosis should be given high-quality HIV education and be offered pretest counselling and HIV testing.

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Cardiobacterium hominis *as a cause of bacterial endocarditis*

To the Editor: Recently, a 32-year-old man presented at Pelonomi Hospital with the clinical features of bacterial endocarditis. Positive BACTEC blood cultures showed short slender Gram-negative bacilli, some with bulbous ends and a tendency to retain Gram-positive staining areas within the bacillus. The bacilli were arranged in rosettes. This appearance is typical of *Cardiobacterium hominis* and biochemical tests confirmed the identification.

C. hominis is part of the commensal oropharyngeal flora[1-3] of man and should be considered a cause of endocarditis when cultures remain negative despite prolonged incubation.[4] The growth may take up to a fortnight to become detectable.

Since this organism was identified as an aetiological agent of endocarditis 30 years ago, only 50 or so cases have been reported in the literature. This patient was discharged well, after 6 weeks of intravenous gentamicin therapy.

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Briewe

Letters to the Editor

Shigella sonnei *meningitis*

To the Editor: A 10-month-old boy was referred to Groote Schuur Hospital with a 3-day history of fever, vomiting and watery stools. Physical examination revealed a lethargic patient. There was no indication of any underlying disease and he appeared well-nourished. The hydrational state was assessed clinically to be 5% depleted and intravenous rehydration therapy was commenced. On day 2, blood was cultured and a lumbar puncture performed in response to persistent fever, diarrhoea and vomiting. No stool culture was requested. Microscopic analysis of the cerebrospinal fluid (CSF) showed 22 polymorphs, 47 lymphocytes and no red blood cells. No globulin was detected and the protein level was 0.2 g/l. After 1 day of incubation *Shigella sonnei* was identified in both the blood and CSF specimens by API