Editor’s Choice September 2011

This issue is a bumper one with the harvest of much interesting leprosy research covering the topics of clinical leprosy, microbiology, treatment and rehabilitation.

Whether clofazimine protects against ENL has been long debated. Balagon et al. (pp. 213–221) report an observational study from The Phillipines in which they looked at episodes of ENL in two cohorts of patients, one treated with 12 months WHO MDT (Rifampicin, Dapsone and Clofazimine) and one group treated with 24 months MDT. There was a high rate of ENL in the lepromatous leprosy patients; being treated with clofazimine did not reduce the occurrence of ENL but patients who received longer treatments had milder ENL over a shorter period. This suggests that clofazimine is not able to alter the underlying pathology of ENL but does have an anti-inflammatory action. However these two cohorts of patients were not contemporaneous, which weakens the conclusions.

In another study published in this issue Duraes et al. (pp. 304–309) reports on the use of azathioprine in a series of patients with chronic ENL. All patients needed steroids to prevent their ENL manifesting clinically and when azathioprine was added to their treatment their steroid requirement was reduced and their ENL episodes declined. Both studies highlight the need for prospective randomised studies on ENL using established scores and run over long time frames so that the effect of drugs on the clinical outcome can be measured.

In India significant numbers of children are still being detected with leprosy and Sonthalia et al. (pp. 259–269) report from a tertiary clinic in Delhi reports significant numbers of childhood patients. This is important because it indicates that transmission of leprosy is ongoing since these children have been infected recently. Children had all types of disease with 20% having bacteria on slit skin smear testing and 19% had reactions including some with ENL. This also indicates that there is an ongoing need for paediatricians to be trained in recognising leprosy and its complications. It also indicates that child friendly services and paediatric doses of drugs need to be provided. Two other papers from India, (Atre et al. (pp. 222–234) and Gautham et al. (pp. 286–295) also highlight the problems of late diagnosis and the resultant disease progression. Again sensitisation of patients and health workers is needed to reduce disability.

Burgos et al. (pp. 253–258) have studied established antibiotic drugs for leprosy using the mouse footpad model and show that both rifamycins and fluoroquinolones have activity against M. leprae and no laboratory evidence of antagonism between these compounds was found.

Shetty et al. (pp. 235–243) report on the patients with confirmed relapse who are being detected in Mumbai. Several patients had been treated with DDS mono-therapy or a short course of chemotherapy. This again highlights the importance of having treatment regimens that are long enough to kill mycobacteria whether active or dormant.

Relapses and reactions were detected by Rao et al. (pp. 244–252) in their long term follow up of a group of patients with PB leprosy. These patients are often ignored because they have simpler disease with a much lower risk of nerve damage. Rao et al. show that this group of patients can have lesions that increase in size after the end of treatment and that this can be caused by both reactions and relapse.

Gerson Penna has contributed a review highlighting the need for evidence to underpin decisions made in leprosy control. When changes occur in programme it is vital that these should be made on the basis of good evidence for the change and that post changes evidence should be collected to monitor the effect of changes.
The leprosy world was intrigued by a recent case report suggesting that a new Mycobacterial species might be cause The Lucio phenomenon. Gillis et al. (pp. 205–209) consider this proposal bringing together the clinical, pathological and molecular biological aspects of the claim. They find that there is not enough evidence to support the proposal of a new species and outline the work that needs to be done to establish a new species causing a clinically important variant of leprosy.

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