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Intermittent treatment of malaria with amodiaquine reduces malarial fever and anaemia in infants

Falciparum malaria remains a major cause of childhood mortality and morbidity in sub-Saharan Africa despite continuing control efforts. In areas where the transmission rate is high the incidence of malaria peaks in infants, and severe anaemia is one of the most common life-threatening complications. Effective interventions targeted at children between their 4th and 7th months of life and after the wet season could have substantial effects in reducing malarial morbidity.

The authors of this recent paper in *Lancet*[1] investigated the effect of presumptive intermittent treatment with amodiaquine and daily iron supplementation on malarial fevers and anaemia in infants. The study was carried out in a holo-endemic area of Tanzania where malaria is largely resistant to chloroquine and sulphadoxine/pyrimethamine.

The study took place in the Muheza district of north-eastern Tanzania. The area has a tropical climate with two rainy seasons, one long and one short. Malaria transmission peaks during and following the long rainy season.

The authors enrolled 291 infants aged 12 - 16 weeks who attended 3 clinics. Enrolment was timed to coincide with the greatest vulnerability to malaria. The infants were randomised to receive amodiaquine, iron supplementation, amodiaquine plus iron supplementation, or placebo. Malarial fevers and anaemia were monitored at bimonthly visits and by self-reporting to health centres.

The results of the study showed that the protective efficacy of intermittent amodiaquine in the prevention of malarial fevers and anaemia was 64.7% and 67.0%, respectively. This was similar in the group receiving amodiaquine plus

iron supplementation. Those infants who received only iron supplementation were partly protected against anaemia, but not against malarial fevers. There was no rebound morbidity at 4 months follow-up.

The conclusion was that presumptive intermittent treatment for malaria with amodiaquine reduces malarial fevers and anaemia in infants in an area where there is high resistance to other antimalarials. This could be of great benefit to public health in other highly endemic malarial areas.

1. Massaga J, *et al. Lancet* 2003; **361:** 1853-1860.

The growing burden of TB – global trends and interactions with the HIV epidemic

The increasing global burden of tuberculosis (TB) is linked to human immunodeficiency virus (HIV) infection.

Corbett *et al.*[1] reviewed data from notifications of TB cases, cohort treatment outcomes, surveys of *Mycobacterium tuberculosis* infection, and HIV prevalence in patients with TB and in other subgroups. Information was collated from published literature and databases held by the World Health Organisation (WHO), the Joint United Nations Programme on HIV/Acquired Immunodeficiency Syndrome (UNAIDS), the US Census Bureau, and the US Centers for Disease Control and Prevention.

There were an estimated 8.3 million (5th - 95th centiles, 7.3 - 9.2 million) new TB cases in 2000 (137/100 000 population; range 121/100 000 - 151/100 000). TB incidence rates were highest in the WHO African Region (290/100 000 per year; range 265/100 000 - 331/100 000), as was the annual rate of increase in the number of cases (6%). Nine per cent (7 - 12%) of all new TB cases in adults (aged 15 - 49 years) were attributable to HIV infection, but the proportion was much greater in the WHO African Region (31%) and in some industrialised countries, notably the USA (26%). There were an estimated 1.8 million (5th - 95th centiles, 1.6 - 2.2 million) deaths from TB, of which 12% (226 000) were attributable to HIV. TB was the cause of 11% of all adult AIDS deaths. The prevalence of *M. tuberculosis*-HIV co-infection in adults was 0.36% (11 million people). Co-infection prevalence rates equalled or exceeded 5% in 8 African countries. In South Africa alone there were 2 million co-infected adults.

The HIV pandemic presents a massive challenge to global TB control. The prevention of HIV and TB, the extension of WHO DOTS programmes, and a focused effort to control HIV-related TB in areas of high HIV prevalence are matters of great urgency.

1. Corbett E, *et al. Arch Intern Med* 2003; **163**: 1009-1021.

Diet changes successfully reduce cholesterol in HAART-treated patients

The most recent edition of *AIDS*[1] carries a report which shows that diet modification can successfully reduce cholesterol levels in HAART-treated HIV-positive patients. This small study in Australia involved 16 HIV-positive men with hypercholesterolaemia, 14 of whom were on a protease inhibitor regimen. Their average age was 45 years, and all had increased cholesterol levels, averaging 7.47 mmol/l. They had been referred for dietary counselling.

The patients were advised not to eat a low-fat diet, but rather to modify their fat intake by eating less saturated fat and more polyunsaturated and monounsaturated fats. They were also advised to maintain a high-fibre intake. The guidelines were similar to those offered to anyone with hypercholesterolaemia. None of the men modified their HAART regimen.

These dietary changes achieved an average fall in serum cholesterol from 7.47 to 6.48 mmol/l at the end of the study, although this was still above the recommended level of ≤ 5.2 mmol/l.

1. Batterham MJ, *et al. AIDS* 2003; **17**: 1414-1416.