

1. INTRODUCTION

The ongoing development of drug resistance in malaria infections has necessitated updating chemoprophylaxis and treatment policies globally.

In South Africa, chloroquine resistance was demonstrated first in KwaZulu-Natal^{1,2} (KZN), and later in Mpumalanga³. This led to a change in drug policy from chloroquine to sulfadoxine-pyrimethamine (SP) as first line treatment for uncomplicated malaria in 1988 in KZN, and in 1997 in Mpumalanga and Limpopo (Northern) provinces.

The development of significant SP resistance in KZN resulted in a further policy change in 2001 to artemether lumefantrine as first line treatment for uncomplicated *Plasmodium falciparum* malaria infections⁴. In order to halt the pattern of continued drug resistance to sequential single drug therapy, combination chemotherapy, preferably using an artemisinin derivative, is the recommended way forward. Additional benefits include an improved therapeutic response, a potential decrease in malaria transmission and thereby greater cost effectiveness.

These guidelines have been compiled utilising available information, but adaptations may sometimes be necessary to suit local circumstances. Information on the incidence, distribution and degree of drug resistance in South Africa is incomplete. There is ongoing monitoring of therapeutic responses which may result in policy revision in the future.^{5,6}

2. OBJECTIVES

The objectives for the treatment of malaria are:

- the prevention of mortality
- the prevention of complications
- the reduction of morbidity
- the elimination of parasitaemia to minimise transmission
- to limit the emergence and spread of drug resistance

3. PARASITE SPECIES

In sub-Saharan Africa, over 90% of human malaria infections are due to *Plasmodium falciparum* (*P. falciparum*) while the rest of the infections are due to *Plasmodium ovale* (*P. ovale*), *Plasmodium vivax* (*P. vivax*) or *Plasmodium malariae* (*P. malariae*). Occasionally mixed infections occur. Infections due to *P. falciparum* may be severe and complicated.

These complications occur almost invariably as a result of delay in treating an uncomplicated attack, the use of ineffective therapy or underdosing with effective treatment.

4. RISK GROUPS

Almost all South Africans are non-immune, including residents in seasonally endemic malaria areas. Immunity may be acquired after long-term repeated infection by *P. falciparum* parasites, a situation that occurs in residents of high transmission areas such as parts of Mozambique, Malawi and Tanzania, amongst others.

High-risk groups for the development of severe *P. falciparum* malaria include: all non-immune travellers to malaria areas, residents (of all age groups) in the seasonally endemic areas of South Africa and other areas with relatively low endemicity and particularly pregnant women, young children, splenectomised and immunosuppressed individuals.

There is limited data on the interaction between HIV and malaria. There is evidence to support an increase in clinical attacks of malaria and higher parasitaemias in semi-immune

adults in Uganda who are HIV infected.⁷ Data suggests that non-immune adults co-infected with HIV in South Africa have a higher risk of severe malaria.⁸