Short Communication

Chloroquine-resistant *Plasmodium falciparum* in Sokoto, North Western Nigeria

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Three patients, 30, 2 and one and a half years, were diagnosed as having *falciparum* malaria and were placed on chloroquine therapy which failed. They were then placed on quinine therapy that then cleared the parasitaemia. This case report seeks to draw the attention of the presence of possible chloroquine-resistant *falciparum* malaria in Sokoto, North Western Nigeria.

Key words: Chloroquine-resistant malaria, *Plasmodium falciparum*, haemoglobin level, packed cell volume.

INTRODUCTION

Malaria is a disease of immense public importance. Available data suggest that there are 200-300 million cases, with more than two million deaths each year (Krogtad et al., 1990) out of which one million is from the African continent alone (Daniel and Molta, 1989). Chloroquine, a 4-aminoquinolone, has been the mainstay of malaria chemotherapy for almost 50 years now. This is because of its rapid onset of action, low toxicity and, almost low expense. It has remained the cheapest and most widely used antimalarial drug in West Africa (Adubofour, 1992) and in Nigeria (Daniel and Molta, 1989). A WHO scientifc group has drawn the attention of the emergence of chloroquine-resistant Plasmodium falciparum in Sub -saharan Africa (WHO 1994). Since then, chloroquine-resistant P. falciparum was reported from the Cameroun (Sansonetti et al. 1985) and the Republic of Benin (Le Bras et al., 1986) and resistance to the drug have spread continously across West Africa. The question of chloroquine-resistant malaria in Nigeria was just a question of time. In Nigeria chloroquineresistant malaria was first reported from the South Western part of the country, in Ibadan in 1987 (Solako et al., 1987) and later in the North Central parts; from Jos and from Zaria (Lege-oguntoye et al., 1989) in 1989. Report of chloroquine resistance from the Eastern part of the country has also been documented (Ezedinachi et al., 1992). We are however not sure of any reports from

the North Western part. This case report is to draw the attention of possible resistant *falciparum* malaria in Sokoto, Nigeria.

CASE REPORT

- 1. A 30-year-old man came into the out-patient department of Sokoto Specialist hospital, complained of generalised body aches, weakness and intermittent fever. Clinical diagnosis of malaria was made, chloroquine injection (320 mg) was administered and was repeated six hours later. A repeat dose of the injection was administered on the second day. The fever persisted and a further chloroquine injection was administered along with intravenous saline solution of 1.5 litres over two hours. Laboratory investigations showed haemoglobin (Hb) level of 12.8 g/l, packed cell volume (pcv), 39 % and blood film stained with Leishman stain revealed features characteristics of P. falciparum. With no significant improvement on the fourth day, a new drug regiment of quinine tablets (300 mg twice daily for three days) was prescribed. The fever was gone by the fifth day and further blood film revealed no malarial parasites.
- **2.** Two infants ages 25 and 18 months respectively were brought into the emergency paediatric unit of Sokoto Teaching Hospital with high fever and they were restless. A Giemsa malarial parasite (mp) test revealed the presence of *P. falciparum* malaria parasites. They were placed on chloroquine injection 5 mg/kg according to their

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respective body weights and the injection was repeated after six hours. As there was no change in their conditions and a further (mp) test was positive for malarial parasites. They were then placed on intravenous quinine (10 mg/kg) diluted in dextrose solution. The infants were up and about by the following day and the parasitaemia was cleared.

DISCUSSION

Drug resistance in malaria has been defined (WHO 1963) as "the ability of a parasite strain to survive and/or multiply despite the administration and absorption of a drug in equal doses or higher than those recommended but within limits of tolerance of the subject". There three levels of chloroquine resistance (R) as defined by WHO 1963:

*RI; following treatment, parasitaemia clears but recrudescence occurs.

***RII;** following treatment, there is reduction in parasitaemia but no clearance.

*RIII; following treatment, there is no reduction of parasitaemia.

It is known that chloroquine resistant strain of the parasite infilterates and becomes established among chloroquine-susceptible strain population and it takes a year or more before it is detected by the conspicous failure in chloroquine treatment in clinics and hospitals.

The three cases reported in this paper are few, but there may be much more. At an early stage, both chloroquine-resistant and susceptible strains may be mixed such that malarial infection may still be treated with the usual chloroquine therapy, Sokoto may be at this stage. There is therefore the need to undertake a survey, using the *in vivo* and *in vitro* methods to determine the prevalence and extent chloroquine resistance.

The emergence and spread of *P. falciparum* resistant to chloroquine is clinically undesirable and it comes with it a lot of challenges. These challenges include the need to know more about the pathogenesis of the infection, its epidemiology, clinical manifestations, social and economic impact. Such challenges can only be met with trained manpower, improved and well-developed sensitive diagnostic methods to identify the resistant strains.

The acquisition of resistance by the malarial parasite is a result of an evolutionary selection under pressure (Wellems et al., 1990; Foote et al., 1990) and it is believed to be a stable strait in the parasite once it is acquired (Cowman and Foote, 1990). Other factors also come into play which contribute to the failure of chloroquine therapy, which include abuse/mis-use of drugs, proliferation of fake drugs, self medication and patient's non-compliance to chloroquine therapy. These

factors should be thoroughly investigated and the public should be educated about the dangers of drug resistance.

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