Malaria Status amongst Some Patients at an Elite Hospital in Abuja, Nigeria

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Abstract: The malaria status amongst some patients at an elite hospital in Abuja, Nigeria was investigated from May 2000 to December 2010. Of the 80 947 patients blood samples analyzed with Giemsa staining methodology, 26 593 (32.9%) were positive for malaria parasites. Of the positive cases encountered 13 (0.05%) were *Plasmodium malariae*; 2 mixed infections of *P. falciparum* and *P. vivax*; one mixed infection of *P. falciparum* and *P. malariae*; while the rest (99.9%) were *P. falciparum*. A case of mixed infection of *P. falciparum* and microfilariae of *Mansonella perstans* was also encountered. Of the 26 593 positive samples, 506 (1.9%) had parasite density of >5000 parasites/µl of blood; 970 (3.6%) had between 500 – 5000 parasites/µl of blood; 3744 (14.1%) had between 50 – 500 parasites/µl of blood; while 21 373 (80.4%) had between 5 – 50 parasites/µl of blood. The parasite detection limit of Giemsa stained thick blood film microscopy is 5 parasites/µl of blood as against that of Malaria Rapid Diagnostic Tests (M-RDTs) that have a detection limit of 397 – 500 parasites/µl of blood. The implication of the continued use of these malaria rapid diagnostic test methodologies includes underdiagnosis, misdiagnosis of malaria and mismanagement of non-malarial fever, which wastes limited resources, erodes confidence in the health care system, and contributes to drug resistance. In conclusion, there is the need to incorporate the laboratory component into the RBM strategy by enhancing parasitological diagnosis by way of providing microscopic diagnostic tolls at all levels of health care – primary (rural settings), secondary and tertiary.

Keywords: Malaria status, *Plasmodium*, parasitological diagnosis, parasite density, parasite detection limit.

INTRODUCTION

Malaria is the most important of all the tropical diseases in terms of morbidity and mortality on a global scale. Worldwide, some 2 billion individuals are at risk; 100 million develop overt clinical disease and 1.5 – 2.7 million die every year. Nearly 85% of the cases and 90% of carriers (many asymptomatic) are found in tropical Africa, where in some countries 20 – 30% of deaths in childhood are attributed to the disease (Greenwood *et al.*, 1987; Defo, 1995). One or more of four species of intracellular parasite, *Plasmodium falciparum*, *P. vivax*, *P. ovale* and *P. malariae* cause malaria in humans. These species differ in geographical distribution, microscopic appearance, clinical features (periodicity of infection, potential for severe disease, and ability to cause relapses) and potential for development of resistance to antimalarial drugs (CDC, 2004). A fifth species, the zoonotic species of *P. knowlesi*, prevalent in Southeast Asia, causes malaria in macaques (Collins, 2012), these are mostly of limited public health importance (Collins and Barnwell, 2009) - but can also cause severe infections in humans.

WHO stated that, globally millions of deaths attributable to malaria are still being recorded. And that the disease constitutes a huge epidemiologic burden in Africa and continues to cripple the economic development in the region. The World Health Organization has estimated that in 2010, there were 219 million documented cases of malaria. That year, between 660 000 and 1.2 million people died from the disease (roughly 2000–3000 per day) (Nayyar *et al.*, 2012), the
majority of cases (65%) occur in children under 15 years old (Oliver, 2012; Murray et al, 2012), many of whom were children in Africa. In Nigeria, the disease is responsible for 60% outpatient visits to health facilities, 30% childhood death, 25% of death in children under one year and 11% maternal death (FMOH, 2008).

The financial loss due to malaria annually is estimated to be about ₦132 billion in form of treatment costs, prevention, loss of man-hours etc; yet, it is a treatable and completely evitable disease (Leighton et al, 1993). The economic impact includes costs of health care, working days lost due to sickness, days lost in education, decreased productivity due to brain damage from cerebral malaria, and loss of investment and tourism (Greenwood et al, 2005). The disease has a heavy burden in some countries, where it may be responsible for 30 – 50% of hospital admissions, up to 50% of outpatient visits, and up to 40% of public health spending (Roll Back Malaria, WHO).

The correct and timely diagnosis of malaria is critically important because the infection may rapidly develop into a life-threatening form of the disease, requiring urgent medical attention. The diagnosis of malaria can be conventionally subdivided into clinical, parasitological, biochemical, serological and molecular biological detection. The most important factor in the clinical diagnosis of malaria is a high index of suspicion (Doherty et al, 1995). Malaria can mimic many diseases and the differential diagnosis is an extensive one. Diagnosis based on clinical symptoms alone is not reliable. Treatment of malaria infection based on clinical symptoms (presumptive treatment) can lead to the following:

1. Unnecessary expenditure;
2. Inappropriate use of antimalarial drugs (antimalarial drug misuse);
3. A delay in establishing a correct diagnosis;
4. A delay in the treatment of a patient;
5. Reliance on presumptive treatment can facilitate the development of antimalarial drug resistance due to “Selective Pressure”. In 1991, Oliver et al; reported that the use of presumptive treatment for malaria had the potential for facilitating resistance by greatly increasing the number of people who are treated unnecessarily but will still be exerting selective pressure on the circulating parasite population. In some areas and at some other times of the year, the number of patients been treated for malaria can be very large.

Despite the wide range of serological, immunological and molecular techniques currently available, the only certain means of diagnosing all species of human malaria parasite is the detection of the *Plasmodium* species by microscopic examination of the blood. Microscopy remains the crucial methodology for malaria diagnosis. When performed in optimal conditions, the technique has a remarkable sensitivity (it can detect a parasitaemia as low as 0.0001%), it is specific and enables the identification of the parasite at the species level, and it is quantitative, reasonably easy to perform and cheap (Hommel, 2002).

Instead of identifying the parasite itself, immunological methods provide the means for detecting either the parasite antigens or the host antibodies directed against the parasite. The detection of antigens may be an acceptable alternative to parasite detection, particularly if the assay is robust, inexpensive, easy to use in field conditions and does not require a microscope, but the detection of antibodies merely provides information on past malaria experience and is of limited use for individual diagnosis.

Serological methods have been used since the early 1960s, when indirect fluorescent antibody tests (IFAT) and indirect haemagglutination assays (IHA) were described. Because such tests detect antimalarial antibodies, they cannot distinguish between current or past infection and they are therefore of limited value as a guide to the treatment or management of the disease.

In contrast to serology, a positive antigen detection assay should detect a current infection. The ideal target antigen should not persist after parasitaemia disappears, should be abundant in the blood (or other body fluids such as urine) to maximize sensitivity and should be malaria specific without cross-reactions with other microorganisms. Experimental tests for detecting malarial antigens are based on either an antigen-capture or an antigen-competition format and often use ELISA or the radioimmunoassay (RIA) methodology. The best antigen detection assays described have a maximum sensitivity of 0.01- 0.001% parasitaemia and are 5-10 times inferior to good quality microscopy (Mackey et al, 1982; Fortier et al, 1987; Taylor and Voller, 1993).

The detection of parasite lactase dehydrogenase (pLDH) originally developed as a way to monitor in vitro drug susceptibility assays has the potential of being useful for the detection of *Plasmodium* parasitaemia (Knoblock and Henk, 1995). The principle of the assay is that pLDH has different biochemical characteristics from human LDH and may therefore be differentially measured using a simple colorimetric assay. Such assays are not species specific, can detect a parasitaemia as low as 0.1% - a level of parasitaemia just below the threshold of 10,000 parasites/µl (Piper et al, 1995). In 2005, VanderJagt et al, reported that OptiMAL assay has a parasite count detection limit of 400 – 10 000 parasites /µl of blood.
Quality Control

Satisfactory diagnosis of malaria by light microscopy requires the availability of a functioning, well maintained microscope, an adequate source of illumination and an operator experienced and competent in the preparation and staining of blood films, as well as in the recognition and identification of the characteristic stages of malaria parasites. Substandard equipment, which is irregularly maintained, can seriously affect the effectiveness of microscopy. To retain competence, it is necessary that good supervision and regular re-training take place. Operational fatigue is also a limiting factor and it has been suggested that the examination of 50 thick films daily is the absolute maximum for any microscopists, and that no more than 20 films should be examined without a break for at least 30 minutes of non-microscope activity (Milne et al., 1994). In actual practice, about 30 blood films should be examined because both the thick and thin films are viewed for each patient. Standard practice requires that the thick film should be examined for at least 5 minutes (corresponding to approximately 100 microscopic fields under oil immersion). The aim of this research work is geared towards establishing the malaria status in Abuja. The objective on the other hand is to enhance proper and prompt diagnosis for effective treatment of malaria.

MATERIALS AND METHODS

Study Area

The Federal Capital Territory (FCT), Abuja is the study area. Abuja is located between latitude 8.25 and 9.20 North of the Equator and longitude 6.45 and 7.39 East of the Greenwich Meridian. The Federal Capital Territory has a total landmass of about 8,000 sq kms; located geographically at the centre of the country. The current population size of the FCT according to the 2006 census figures stands at 1,405,201 (National Bureau of Statistics, 2006). Two seasons are experienced in FCT. These are the wet (rainy) season and the dry season. The rainy season lasts between April to October (with a mean total rainfall of 62.8 – 262.7 mm; August being the highest). The dry season is between November and March (with a mean total rainfall of 1.2 to 11.3 mm). Abuja has a daily temperature range between 20.4 – 34.7°C with an average of 27.2°C/81°F in January; and between 21.9 – 29.1°C with an average of 25.6°C/78°F in July (WWIS, 2004).

Study population

The population studied consisted of 80,947 patients (comprising of 17,234 adult males, 39,424 adult females, 13,435 male children – day of birth to 15 years and 10,854 female children – day of birth to 15 years) whose blood samples were presented at the Parasitology Laboratory of National Hospital, Abuja for malaria parasite diagnosis. The study was carried out from May 2000 to December 2010.

Parasitological Techniques

Thick and thin blood films were made on clean grease-free slides and stained appropriately using Giemsa’s staining method. Giemsa stain is the most commonly used of the Romanowsky stains and is the best for routine diagnosis because of its applicability to both thick and thin smears, its stability on storage and its constant and reproducible staining quality over a wide range of temperatures. The stained films were then examined microscopically using 100x objective to count the parasites.

Parasites were counted by estimating the parasite numbers/µl of blood from the thick film. This was carried out by multiplying the average number of parasites per thick film field (100x objective) by 500. Between 10 -100 fields (depending on parasite density) were examined to determine the average number of trophozoites per thick film field. Ten fields are sufficient when the parasite density is high.

The factor of 500 was proposed by Greenwood and Armstrong (1991). They calculated that 5 – 8 µl is the volume of blood required to make a satisfactory thick film and that the volume of blood in one thick film field (100x objective) of a well-prepared thick film is about 0.002 µl. Therefore the number of parasites per thick film field multiplied by 500 gives the estimated number of parasites/µl of blood. This method, Greenwood and Armstrong (1991) found to be more accurate and quicker than counting the parasites against 100 white blood cells in a thick film using WHO method as used by Molineaux and Gramiccia (1980). In 2002, Ikeh et al, used this technique in their study at Jos.

For designation of the relative parasite count on a thick film, a simple code of from one to four crosses or the plus sign scheme is used to report parasite numbers:
• + (1+) = 1 – 10 parasites per 100 thick film fields
• ++ (2+) = 11 – 100 parasites per 100 thick film fields
• +++ (3+) = 1 – 10 parasites per one thick film field
• ++++ (4+) = > 10 parasites per one thick film field

RESULTS

Of the 80,947 patients blood samples analyzed for malaria parasite diagnosis, 26,593 (32.9%) were positive for malaria (table 1). Of the positive cases encountered 13 (0.05%) were Plasmodium malariae; 2 mixed infections of Plasmodium falciparum and P. vivax; one mixed infection of P. falciparum and P. malariae; while the rest (99.9%) were P. falciparum. A case of mixed infection of P. falciparum and microfilariae of Mansonella perstans was also encountered.

Figure 1 visually shows that the monthly infectivity rate is from 30.6 – 35.7%.

The seasonal parasitaemia (% positivity) is stated in table 2. The dry season (November - March) had a % positivity of 32.3% as against the 33.3% recorded for the rainy (wet) season (April - October).

Table 3 and figure 2 indicate the parasitaemia levels while using Chloroquine from 2000 – 2005 as against the use of Artemisinin Combination Therapy (ACT) from 2006 - 2010. During the Chloroquine years, 2.8% of patients had parasitaemia > 5000 parasites/µl of blood as against the 0.9% patients that were recorded for the ACT years. 5.3 % of patients during the Chloroquine years had between 500 – 5000 parasites/µl of blood as against the 1.7% of patients during the ACT years. A wider difference rate was noticed for patients that had between 50 – 500 parasites/µl of blood; while 18.8 % were recorded for the Chloroquine years, 8.8% were for the ACT years. For patients that had between 5 – 50 parasites/µl of blood, the reverse was the case as a higher occurrence rate of 88.6% was recorded during the ACT years as against the 73.1 % for the Chloroquine years. Total % positivity during the Chloroquine years was 35.8% as against the 30.0% recorded during the ACT years.

Of the 17,234 adult male blood samples examined, 5,817 (33.8%) were positive; 12,108 (30.7%) of the 39,424 adult female blood samples examined were positive; 4,845 (36.1%) of the 13,435 male children were positive; while 3,823 (35.2%) of the 10,854 female children blood samples were positive for malaria (table 4).

Table 1. Monthly Variation of Parasitaemia (Monthly Infectivity Rate)

<table>
<thead>
<tr>
<th>Month</th>
<th>Total No. Examined</th>
<th>Negatives</th>
<th>Total Positives</th>
<th>% Positivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>5623</td>
<td>3883</td>
<td>1740</td>
<td>30.9</td>
</tr>
<tr>
<td>February</td>
<td>6036</td>
<td>3898</td>
<td>2138</td>
<td>35.4</td>
</tr>
<tr>
<td>March</td>
<td>6464</td>
<td>4411</td>
<td>2053</td>
<td>31.8</td>
</tr>
<tr>
<td>April</td>
<td>5577</td>
<td>3791</td>
<td>1786</td>
<td>32.0</td>
</tr>
<tr>
<td>May</td>
<td>6376</td>
<td>4427</td>
<td>1949</td>
<td>30.6</td>
</tr>
<tr>
<td>June</td>
<td>7258</td>
<td>4842</td>
<td>2416</td>
<td>33.3</td>
</tr>
<tr>
<td>July</td>
<td>7272</td>
<td>4927</td>
<td>2345</td>
<td>32.3</td>
</tr>
<tr>
<td>August</td>
<td>7790</td>
<td>5214</td>
<td>2576</td>
<td>33.1</td>
</tr>
<tr>
<td>September</td>
<td>7266</td>
<td>4716</td>
<td>2550</td>
<td>35.1</td>
</tr>
<tr>
<td>October</td>
<td>7432</td>
<td>4781</td>
<td>2651</td>
<td>35.7</td>
</tr>
<tr>
<td>November</td>
<td>7516</td>
<td>5128</td>
<td>2388</td>
<td>31.8</td>
</tr>
<tr>
<td>December</td>
<td>6337</td>
<td>4336</td>
<td>2001</td>
<td>31.6</td>
</tr>
<tr>
<td>Total</td>
<td>80,947</td>
<td>54,354</td>
<td>26,593</td>
<td>32.9</td>
</tr>
</tbody>
</table>

An annually % positivity range of 30.9 – 35.7% was encountered. The month of May had the lower limit of monthly infectivity rate of 30.6% while October had the upper limit of 35.7%; both months within the rainy (wet) season period.

Table 2. Seasonal Variation of Parasitaemia

<table>
<thead>
<tr>
<th>Season</th>
<th>Total No Examined</th>
<th>Total Negatives</th>
<th>Total Positives</th>
<th>% Positivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry Season (Nov.-March)</td>
<td>31,976</td>
<td>21,656</td>
<td>10,320</td>
<td>32.3</td>
</tr>
<tr>
<td>Rainy Season (April-Oct)</td>
<td>48,971</td>
<td>32,698</td>
<td>16,273</td>
<td>33.3</td>
</tr>
</tbody>
</table>

There is no seasonal variation of parasitaemia as indicated in table 2. During the dry season (November - March), a % positivity of 32.3% was recorded as against the 33.3% recorded for the rainy (wet) season from April – October.
Table 3. Comparison of Chloroquine Years (2000 - 2005) & ACT Years (2006 - 2010)

<table>
<thead>
<tr>
<th>Period</th>
<th>4+</th>
<th>3+</th>
<th>2+</th>
<th>1+</th>
<th>Total Positives</th>
<th>Total No Examined</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000-2005</td>
<td>397</td>
<td>756</td>
<td>2653</td>
<td>10 332</td>
<td>14 140</td>
<td>39 447</td>
</tr>
<tr>
<td>(Chloroquine)</td>
<td>(2.8%)</td>
<td>(5.3%)</td>
<td>(18.8%)</td>
<td>(73.1%)</td>
<td>(35.8%)</td>
<td></td>
</tr>
<tr>
<td>2006-2010</td>
<td>109</td>
<td>214</td>
<td>1091</td>
<td>11 041</td>
<td>12 453</td>
<td>41 500</td>
</tr>
<tr>
<td>(ACT Years)</td>
<td>(0.9%)</td>
<td>(1.7%)</td>
<td>(8.8%)</td>
<td>(88.6%)</td>
<td>(30%)</td>
<td></td>
</tr>
<tr>
<td>2000-2010</td>
<td>506</td>
<td>970</td>
<td>3744</td>
<td>21 373</td>
<td>26 593</td>
<td>80 947</td>
</tr>
<tr>
<td>(1.9%)</td>
<td>(3.6%)</td>
<td>(14.1%)</td>
<td>(80.4%)</td>
<td>(32.9%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Key:
4+ - > 5000 parasites/µl of blood
3+ - 500 – 5000 parasites/µl of blood
2+ - 50 – 500 parasites/µl of blood
1+ - 5 – 50 parasites/µl of blood

Table 4. Prevalence of Malaria Parasitaemia in Relation to Age and Gender

<table>
<thead>
<tr>
<th>Age Group/Gender</th>
<th>No. Positive</th>
<th>No. Positive</th>
<th>% Positivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult male</td>
<td>17 234</td>
<td>5 817</td>
<td>33.8</td>
</tr>
<tr>
<td>Adult female</td>
<td>39 424</td>
<td>12 108</td>
<td>30.7</td>
</tr>
<tr>
<td>Children male</td>
<td>13 435</td>
<td>4 845</td>
<td>36.1</td>
</tr>
<tr>
<td>Children female</td>
<td>10 854</td>
<td>3 823</td>
<td>35.2</td>
</tr>
</tbody>
</table>

Figure 1. visually show that the monthly infectivity rate is from 30.6 – 35.7%.

DISCUSSION

Malaria, being a major public health problem and cause of suffering and premature death in tropical and subtropical countries, requires a drastic attention which would encompass provision of facilities (including human capacity) geared towards accurate and proper diagnosis with the availability of efficacious drugs for treatment.

In this study, the level of malaria infectivity is 32.9%; with a monthly variation of 30.6 – 35.7% parasitaemia (table 1 and figure 1). By this data (table 4), Abuja is a mesoendemic region having a stable transmission being based on the
WHO classification of endemicity revised by Metselaar and Van Thiel (1959). Of the 26,593 positive samples, 506 (1.9%) had parasite density of > 5000 parasites/µl of blood (4+); 970 (3.6%) had between 500–5000 parasites/µl of blood (3+); 3,744 (14.1%) had parasite density of between 50–500 parasites/µl of blood (2+), while 21,373 (80.4%) had parasite density of between 5–50 parasites/µl of blood (1+).

There is no difference in seasonal variation in parasitaemia between the dry season (November - March) – 32.3% and the wet (rainy) season (April - October) – 33.2% (table 2). There is no significant difference in the prevalence of malaria parasitaemia in relation to age and gender as stated in table 4. The adult males had 33.8% positivity against the 30.7% of the adult females; the male children had 36.1% positive cases while the female children had 35.2% positives.

In the treatment of malaria, resistance of Plasmodium species became a global problem and concern. WHO stated that resistance to antimalarial drugs was identified in two of the Plasmodium species. Most resistant strains of P. falciparum have developed due to inadequate drug doses mainly as a result of unregulated drug distribution and prescription, lack of adequate drugs, poor quality of drugs, and incorrect taking of antimalarials by patients. When insufficient drug is taken to kill all the parasites, the stronger parasites survive and multiply (Chessbrough, 1998). In 1995, WHO reported that the problem of drug resistance could be attributed primarily to increased selection pressures on P. falciparum in particular, due to indiscriminate and incomplete drug use for self treatment.

Several mechanisms can account for changes in drug sensitivity in the malaria parasites, for example, physiological adaptations due to non genetic changes, selection of previously existing drug resistant malarial parasites from a mixed population under drug pressure, spontaneous mutation, mutation of extra-nuclear genes, or the existence of plasmid-like factors. Selection of mutants by the drugs themselves appears an important mechanism (WHO, 1987; Wernsdorfer, 1994). Additionally, reliance on presumptive treatment can facilitate the development of antimalarial drug resistance. In 2003, Abdullahi et al. reported a 100% chloroquine resistance in Sokoto; Sowunmi et al, (2004) reported 81.4% chloroquine resistant strains from Ibadan; while Peletiri et al, (2010) reported 88.9% chloroquine resistance from Abuja. Their report also stated a 27.8% resistance to quinine.

Based on the increasing rate of resistance of Plasmodium species to various antimalarial drugs showed by way of treatment failures, the Artemisinin-Combination Therapy (ACT) regimens was introduced in 2005 in Nigeria. So, how well has the ACT fared since its incorporation into the reviewed National Antimalarial Treatment Policy?

From our data as stated in table 3, there is just a reduction in parasite density in cases of >5000 parasites/µl of blood, between 500 – 5000 parasites/µl of blood, and between 50 – 500 parasites/µl of blood in the Artemisinin Combination Therapy (ACT) years. There is no much difference in total positives between the chloroquine years (35.8%) to that of the
ACT years (30%). This may portend a grave danger ahead of us in our fight against the menace of malaria; vis-a-vis the proper treatment of and eventual eradication of malaria from our population.

In areas of high endemicity, a parasitaemia above 5000 – 10000 parasites/µl of blood is usually suggested as a guideline, hence used as the critical value (clinical threshold). Coosemans et al. (1994) proposed the sensible ‘rule of thumb’ that a 100% field positivity on a thick film may be used as a good morbidity indicator. However, because *P. falciparum* matures in internal organs (‘sequestration’), a severe form of malaria may occur at a time when the parasites in the peripheral blood are scanty or even absent (Silamut and White, 1993). It is therefore necessary to examine serial blood films at intervals of 6-12 hours to confirm the diagnosis (WHO, 2000).

Our attention has been drawn to the continued call and use of Malaria Rapid Diagnostic strips/cassettes for the diagnosis of malaria in Nigeria. While the microscopy of a thick blood film has a remarkable sensitivity capable of detecting parasitaemia as low as 0.0001% (5 parasites/µl of blood, or 1-10 parasites/100 thick film fields - 1+), the best antigen detection assays described have a maximum sensitivity of 0.01-0.001% (5000, 500 parasites/µl of blood - 3+, 2+) parasitaemia and are 5-10 times inferior to good quality microscopy (Mackey et al., 1982; Fortier et al., 1987; Taylor and Voller, 1993). The detection of parasite lactase dehydrogenase (pLDH) assays are not species specific, can detect a parasitaemia as low as 0.1% (>5000 parasites/µl of blood - 4+) - a level of parasitaemia just below the threshold of 10 000 parasites/µl of blood (Piper et al., 1995).

We have quality controlled four of such rapid malaria diagnostic kits (OptiMal by DiaMed, Malaria pLDH Cassette by Unlimited Diagnostics, CareStart™ Malaria HRP-2 by DiaSys, and Paracheck pf) and found out that such kits gave negative results on microscopy slide positive cases of <500 parasites/µl of blood (2+). These rapid malaria test kits were given to us for performance evaluation by prospective suppliers.

In 2005, VanderJagt et al. reported in their study on Comparison of the OptiMAL rapid test and microscopy for detection of malaria in pregnant women in Nigeria; and that the OptiMAL assay detected less than half as many positive microscopy cases. Their report specifically stated that of the 20 microscopy positive cases, only four were positive with OptiMAL assay (i.e. 20% positivity rate). And that while the parasite count detection limit of microscopy was 40 parasites/µl of blood; that of the OptiMAL assay was 400 parasites/µl of blood (confirming the 10 times inferior result to good quality microscopy earlier reported by some researchers (Mackey et al., 1982; Fortier et al., 1987; Taylor and Voller, 1993). Though in their study, VanderJagt et al (2005) encountered three positive OptiMAL assays which were microscopy negative, we did not encounter such cases. VanderJagt and coworkers had recommended in principle that due to the simplicity of the OptiMAL assay, it should be suitable for use in developing countries, particularly in rural areas, as it eliminates the need for highly trained microscopist. A position, we do not share.

Our stand is based on the fact that if Ministry of Health can employ Medical Doctors, Pharmacists and Nurses alike and post them to these rural areas with necessary working tools; why can’t same government agency (ies) provide laboratory facilities and employ Medical Laboratory Scientists as well as Medical Laboratory Technicians (highly trained microscopists) to carry out microscopic examination of blood for malaria parasite diagnosis amongst other tests at these rural health facilities? Are the rural dwellers not entitled to quality health care? Should they be denied of the services of seasoned Medical Laboratory Scientists / highly trained microscopists? We should not be seen to be discriminatory to the rural poor. Remember, current medical practice rely on “evidence based”.

In their study at Benin, Tattfeng and Bawo (2008) reported that the detection rate of thick blood film technique was significantly higher than that of the ICT test kit. The report also stated that there was no significant difference in the detection rate of the thick film microscopy and ICT test techniques with parasitaemia >1000 parasites/µl of whole blood (3+). This report is in conformity with our own experience were such rapid kits only shows positive with microscopy slide positivity from 3+ (500-5000 parasites/µl of blood). We rely on the ‘gold standard’ – slide microscopy for malaria diagnosis.

Adesanmi et al (2011), reported in their study at Enugu that one of the rapid immunochromatographic test methodology - “Paracheck pf” had malaria parasites detection limit of 397 parasites/µl of blood. The question is what happens to patients having between 5 – 396 parasites/µl of blood (1+, 2+), a value that can easily be detected with a well stained blood film examined microscopically by a well trained microscopist. Though, in their conclusion, Adesanmi et al (2011) recommended the use of “Paracheck pf” in Nigerian children, but that negative results should be cautiously interpreted in infants because symptomatic malaria may occur in these children at parasite densities as low as 100 parasites/µl of blood. We see no basis for this recommendation and so totally disagree with it.

It should be noted that if rapid techniques are used for malaria diagnosis, apart from the fact that most malaria cases (94.5%) as in this study including those having 50 – 500 parasites/µl of blood 2+ (14.1%) and 5 – 50 parasites/µl of blood 1+ (80.4%) as seen in table ii above) would be missed, and other parasites usually encountered during diagnosis of haemoparasites would equally be missed. With a detection limit of 397 parasites/µl of blood reported by Adesanmi et al (2011) and 400 parasites/µl of blood reported by VanderJagt et al (2005), this means that of the 50 – 500 parasites density group (2+) in our study – 3744, only patients having between 397 – 500 parasites/µl of blood (20.8% - 779
patients) would give a positive test result. The remaining 79.2% (2971 patients) would give a negative result. So, using the limit of 397 parasites/µl of blood, 90.4% would be missed in our study (10% for those that had between 50 – 500 parasites/µl of blood 2+ and 80.4% for those that had between 5 – 50 parasites/µl of blood 1+). It therefore mean that, if rapid diagnostic test methodology was used in this study, only about 5.5 – 9.6% of positive cases would have been recorded as against the 32.9% encountered.

The implication of the continued use of these malaria rapid diagnostic tests (M-RDTs) methodologies include underdiagnosis, misdiagnosis of malaria and mismanagement of non-malarial fever, which wastes limited resources, erodes confidence in the health care system, contributes to drug resistance and eventual administration of unnecessary antimalarial drugs aimed at mopping up all negative test results where patients may still present with symptoms. Remember that malaria can mimic many diseases and the differential diagnosis is an extensive one.

In this study, we encountered a case of mixed infection of *P. falciparum* and microfilariae of *Mansonella perstans*. While working in a private medical laboratory in Jos Plateau State of Nigeria from 1997 – 1999, I (corresponding author) encountered two patients that were sent for malaria diagnosis from a private clinic. One of the patients had a mixed infection of *P. falciparum* and *Trypanosoma* species; while the second patient had a negative malaria result but had trypanosomes in the blood. The clinicians that sent them were very grateful and the patients were properly treated accordingly.

CONCLUSION

Malaria as a preventable, diagnosable and treatable ailment demands that, all hands must be on deck.
1. The missing component of the Roll Back Malaria (RBM) strategy – Laboratory Diagnosis should be incorporated. The vision of the current five-year strategic plan (2009 – 2013) is to ensure that Malaria no longer becomes a major public health problem in Nigeria as illness and death from malaria gets significantly reduced. This is to be achieved by ensuring that families will have universal access to malaria prevention and treatment. This latter aspect should rather read “access to malaria prevention, laboratory diagnosis and treatment.
2. Community participation and health education strategies promoting awareness of malaria and the importance of control measures aimed at reducing the incidence of malaria in our environment should be encouraged through media advocacy and at various health facilities, schools and during town hall meetings.
3. The use of Malaria Rapid Diagnostic Tests (M-RDTs) methodologies in Nigeria for individual diagnosis of malaria should be discouraged if we must end the proliferation of resistant strains of *Plasmodium* species as well as win the war to reduce and eventual eradication of malaria in our locality.
4. We must enhance parasitological diagnosis by way of providing diagnostic tolls (both man-power and materials) at all levels of health care – primary (rural settings), secondary and tertiary.
5. Presumptive treatment of malaria should be highly discouraged while ensuring efficiency and productivity of microscopists who are experienced and competent in the preparation and staining of blood films, as well as in the recognition and identification of the characteristic stages of malaria parasites usually found in human blood by way of capacity building through continuous training.
6. Above all, the turn-around time for malaria parasitological diagnosis should be drastically reduced so that such patients get their results same day and those with positive results see their doctors again for appropriate treatment before going back home. If this is done, we would have taken care of the situation whereby some clinicians give antimalarial treatment to all patients sent to the laboratory for malaria parasite test. The usual instruction has been, make sure that your blood is collected for the test before commencing the administration of the antimalarial drugs; see me with your result on your next appointment. Therefore, patients should no longer wait at home until their situations get to the emergency level before seeking medical attention at the hospital.

Out of curiosity, we reviewed six months pattern of how blood samples for malaria parasite diagnosis were handled. Between July to December 2011, we processed 4 550 blood samples for diagnosis of malaria. Of this number, 619 (13.6%) of the results were unclaimed (not collected by the patients or their relatives). Of these unclaimed results, 446 (72.1%) had negative malaria parasite result while 173 (27.9%) were positive for malaria parasites. Most of these malaria parasite negative patients would have been given presumptive treatment for malaria.
7. In a bid to monitor effective treatment regimen amongst others, we strongly recommend that henceforth, malaria test results should indicate the actual malaria parasite count for each individual patient sample.
8. Finally, if we must win this war against malaria, all unnecessary politicking associated with the prevention, accurate diagnosis at all levels of healthcare and effective treatment must be put aside.
References


Roll Back Malaria WHO partnership. “Economic costs of malaria”. WHO.


