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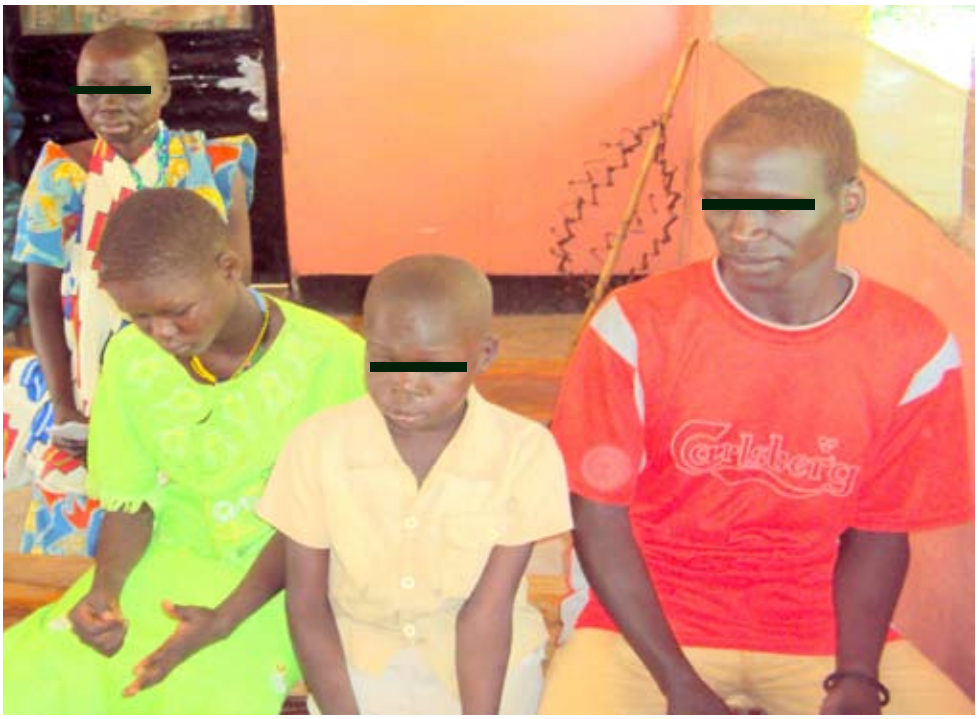

Two children with Nodding syndrome and their father at Lui Hospital, South Sudan. (credit: Thomas Akim).

Investigation into the NODDING SYNDROME in Witto Payam

Malaria in South Sudan

KAP of caretakers of malnourished children

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The Southern Sudan Medical Journal is a quarterly publication intended for Healthcare Professionals, both those working in the Southern Sudan and those in other parts of the world seeking information on health in the Southern Sudan. It aims to offer education and information in all specialities, and to identify research that will inform the development of Health Services in the Southern Sudan. We plan to include reports of original research, critical/systematic reviews, case reports, clinical photographic materials, letters to the Editor, use of drugs, medical news of public interest, and nutrition and public health issues.

The Journal is published in mid-February, May, August and November and is free online at

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Nodding Syndrome: challenges for a bizarre condition

In September 2010 the Government of Southern Sudan's Ministry of Health sent a team to Kediba, Witto Payam, Mundri East County in Western Equatoria State after reports that there was an increase in the number of cases of a bizarre disease known as 'nodding syndrome' in the area. The team found 96 cases registered in the internally displaced camp (1) (see the main article on page 3).

The syndrome has been known for long time in the communities of Western Equatoria State. What has been lacking is a consistent programme to address it. Children have been physically and mentally affected by the syndrome and communities are consumed with fear. The exact prevalence of nodding syndrome in Southern Sudan is unknown.

"Children have been physically and mentally affected by the syndrome and communities are consumed with fear"

Much effort is needed to understand the pathophysiology so that not only a cure can be found, but also prevention strategies can be put into place. Studies in Uganda and Tanzania have linked the syndrome to Onchocerca vovulus and abnormal EEG suggesting an epileptiform mechanism. (2) Low blood levels of vitamin B6 may also play a part in the syndrome development. (3)

A large study must be done in the affected areas. The Ministry of Health needs to work closely with the World Health Organization and the Centres for Disease Control in addressing this problem. Let the investigations in Witto Payam be a catalyst for more action. At least, the communities want to see that something is being done about this devastating condition.

Dr. Edward Eremugo Luka

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Two members of the Editorial Board have left in order to allow others to join. SSMJ thanks these members for their past assistance and looks forward to their continued support as reviewers or authors. We welcome Drs Charles Bakheit and David Tibbutt onto the Editorial Board.

Investigation into the Nodding syndrome in Witto Payam, Western Equatoria State, 2010

John Lagu Nyungura[a], Thomas Akim[b], Anthony Lako[c], Abe Gordon[d], Lily Lejeng[e], Gibson William[f]

Introduction

'Nodding syndrome' is found in South Sudan mainly in Western Equatoria where it was first reported in the 1980s (1) and where WHO did a series of assessments in 2001, 2004 and 2006 (2). It has also been described in Western Uganda (3) and Tanzania (4).

Nodding syndrome is a progressive condition characterised by head nodding (hence the name), mental retardation and stunted growth (4) affecting mainly children and young adults.

Some reports suggest that the episodes of nodding occur when the child begins to eat food or feels cold (1, 4). These episodes are said to be brief and disappear when the child stops eating or feels warm again.

Little is known about the prognosis of the nodding syndromebut it is thought to be a very debilitating physically and mentally. Attacks can cause children to collapse and injure themselves or die, for example, by falling into a fire.

Materials and Methods

In September 2010 a team led by the Ministry of Health, Government of Southern Sudan (MoH-GoSS) visited Witto Payam in Western Equatoria State in order to investigate reports from UNOCHA and some INGOs of nodding syndrome in Witto Internal Displaced People (IDP) camp. The objective of the team (composed of the authors of this report) was to provide the MoH-GoSS with workable recommendations on how to tackle the syndrome. The people now living in the IDP camp had been displaced from Diko, Tore and some surrounding villages by the Lord's Resistance Army. Most of the IDPs are originally from Witto Payam who moved and settled in Diko during the war time displacements. The team met community leaders, administrators and families at Witto IDP camp, Lui town and Jambo town.

At Witto IDP camp the community leader explained the purpose of our visit (the first government-led delegation to investigate the mysterious nodding phenomenon) to a large crowd of affected children and their parents. We then had a one-hour brainstorming session with a focus group of 25 parents, elders of the community and church leaders. We used unstructured questions and answers to find out:

- when the community noticed the first case of nodding syndrome
- events that happened, including eating habits, during the civil war and
- what the community thinks causes the disease.

We asked individuals to give a history of the syndrome and to suggest possible causes and ways the condition may be transmitted. We also interviewed five parents with affected children and we tried to elicit the nodding symptom in four children by asking their parents to feed them local foods.

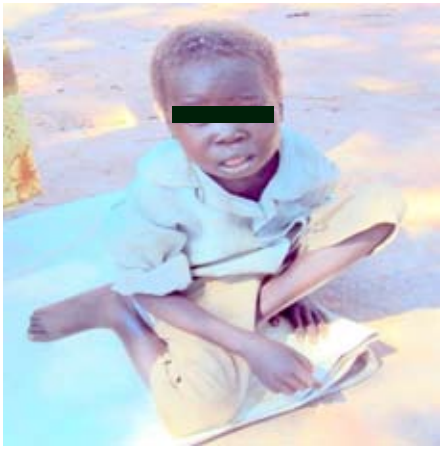

Figure 1. A 15-year old boy with stunting and mental retardation in Witto Payam (credit: Thomas Akim)

In Jambo town we repeated a similar brain storming session with a fifteen-member group and we interviewed

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the Clinical Officer at Jambo Health Centre.

At Lui Hospital we met the administrator and interviewed two doctors, the head nurse, the statistician and five cases at the outpatient department (OPD) and Medical Ward. Some of us briefly visited a nearby co-education primary school and collected a list of pupils reported to have the nodding syndrome.

Figures 1 and 2 show some affected children.

Figure 2. Mental retardation in the young adult in Witto Payam (credit: Thomas Akim)

Findings

We registered a total of 96 cases of nodding syndrome: 70 in Witto IDP camp and 26 at Jambo. Of the affected children:

- 46% were categorised as stunted according to our observation. However we do not know the prevalence of stunting in children without the syndrome.
- 52% of their parents said that "nodding is induced by the sight of food".

Figure 3 shows that:

- Most cases were aged between 5 and 20 years and therefore born between 1980 and 2005.
- The greatest number was in the 10 -15 year age group.

Figure 4 shows the increasing trend of nodding syndrome cases in Witto Payam and Jambo town over time.

Of the 96 documented cases:

- 96% had been treated at Lui Hospital by a Medical Officer or at Jambo PHCC by a Clinical Officer. The drugs mainly used were anticonvulsants: carbamazepine and phenobarbitone - that had been issued monthly and provided by MoH-GoSS.
- 74% were the first, second, third or fourth borne of the mother.
- In some families more than three children were reported to be affected.

Four of the affected children were given food under our observation but we did not see any 'nodding phenomenon'.

Information from the community

There appeared to be two types of presentations of the syndrome:

1. The community reported that, "the nodding symptom usually begins with the sight of food" and over time changes to generalised tonic–clonic convulsions (i.e. partial seizures with secondary generalization).

2. Some patients were said to initially present with generalised seizures characterised by sudden loss of consciousness, tonic-clonic convulsions, rolling back of eyes, salivation, loss of sphincter control, confusion and finally a deep sleep (generalized seizures). The parents reported that the condition is preceded by an aura that could suggest a focal origin of an epileptic discharge.

The community could not provide a credible theory for the emergence of the syndrome. Some elders speculated that it could have resulted from relief food that might have been contaminated, childhood vaccinations or large or small flies which had invaded the areas of wartime displacement. They had observed that people became blind when bitten by these flies.

Some community members could not suggest any plausible theory while some thought it could have supernatural causes. Others thought the disease was caused by the settling amongst, and subsequent intermarriage between, the local community and other war-displaced Southern Sudan communities. Notably, traditional healers who had been consulted by the community had not succeeded in treating the syndrome.

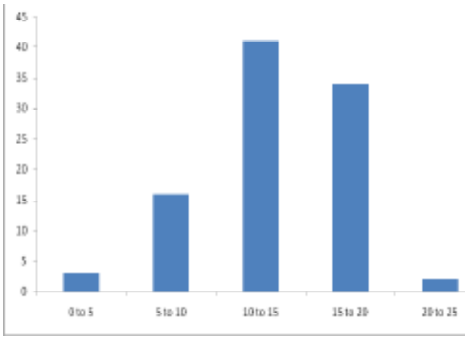

Figure 3. Distribution of numbers of affected persons in Witto Payam by age group (years)

Management

At present the syndrome is treated with common anti-convulsants although prognosis is said to be poor. According to health workers, children who start treatment early and on a regular basis have seizures less frequently. Therefore they have normal growth and no observable complications whereas their cohorts on irregular or no treatment have more complications.

In fact the drugs are in short supply. Some parents of the affected children bought medicines from a private pharmacy but said they were ineffective. This could have resulted from drug intolerance or under-dosing. Many health workers are not familiar with the syndrome and do not know how to treat and care for patients - indicating the need for more training.

As a remedy, the community in Witto Payam had tried to socially isolate their affected children (i.e. drinking, eating and sleeping separately) but said the syndrome continued to occur in healthy children. The school administration was in a dilemma as to whether or not they were required to have separate classes for affected children. If one pupil in the class showed symptoms, by the end of the year about seven other children were reported to have the same condition. This raises the question of whether this is a communicable disease.

Discussion

The team considered the following questions.

1. Is nodding syndrome a form of epilepsy?

Observations and reports of the nodding episodes both from this study and from others in Tanzania and Uganda lead us to speculate that the nodding syndrome may be a particular form of epilepsy found mainly, perhaps only, in this area of Africa. The study in Tanzania, which did MRI scans and EEG recordings (4), concluded that head nodding is "possibly a new epilepsy disorder in sub-Saharan Africa". A previous study in Lui indicated that EEG results were consistent with a specific encephalopathy, which progresses in well-defined stages, and nodding represents the onset of symptoms and the ictal events common to all stages of epilepsy (5). However until this condition can be further investigated by a clinical neurologist and by doing more EEGs it is difficult to come to definite conclusions.

Children with 'nodding' are said to have mental retardation, and nodding is often seen as a habit or 'comfort' activity in children with learning disability, autism, etc. The link between nodding and mental retardation needs to be clarified.

That the nodding syndrome may be a type of epilepsy is strengthened by the fact that the health workers reported that children who start treatment with anti-convulsants early and on regular basis have reduced frequency of the seizures compared to affected children on irregular or no treatment.

Some parents reported that nodding was triggered by the sight of food and this has been reported previously (1, 4). However it did not occur when we watched affected children being offered food. So this observation needs more investigation.

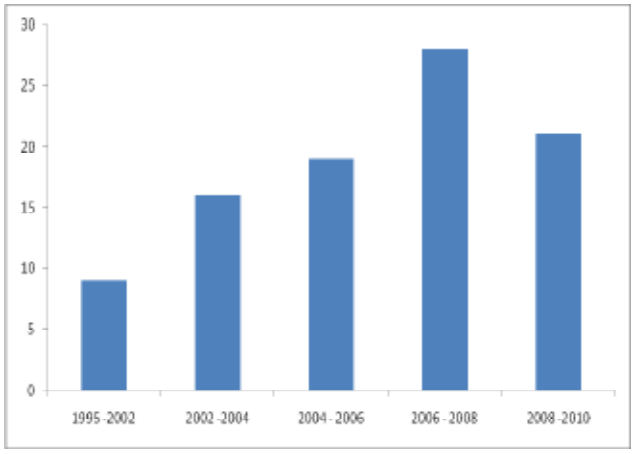

Figure 4. Trend in prevalence of nodding syndrome between 1995 - 2010

2. What are the causes of nodding syndrome?

The team considered whether the condition could be caused by:

1. Ingesting chemicals from biological and chemical weapons previously used in the area. Had these affected the parents so that the syndrome was genetically passed to their children? Although some families had more than one child with the syndrome this seems unlikely as the condition has been described in Tanzania and Uganda where there have been no wars.

2. Eating seeds covered in toxic chemicals that were provided by relief agencies and meant for planting – as suggested by the community. However toxicological investigations on relief foods, such as lentils and sorghum, gave insignificant results in a case control study done in Lui (5).

3. Infection by a parasitic worm, Onchocerca volvulus, which is carried by a black fly and which causes river blindness. Most children in South Sudan suffering from nodding syndrome live close to the Yei River and

93% of the victims carry the parasite (6). A link between river blindness and other cases of epilepsy (7) as well as retarded growth (8) had been proposed previously, although the evidence for this link is inconclusive (9). In the Tanzanian study MRI lesions were associated with positive skin polymerase chain reaction (PCR) for O. volvulus despite negative PCR of the cerebrospinal fluid (4).

According to the majority of the people we interviewed, the nodding syndrome was first noticed around 1997 in IDP camps near Tore, a village near Yei, and on the banks of Yei River. However, some community members claimed that, even before displacement took place in the bushes of Kediba County, cases were already present in the Jambo area (or in Witto Payam) around 1992.

The team considered that the involvement of a vector was not supported as the syndrome affected only children and young adults born around 1989 to 2005. Also it seems to occur in specific areas despite there being the same ecosystem in most of Western Equatoria State and part of Central Equatoria State (Yei County).

Before the role of Onchocerca volvulus can be ruled out further investigations are needed to find out whether children with the nodding syndrome have a greater prevalence of Onchocerca volvulus than non-affected children.

Conclusions

Based on the team's observations at Witto Payam and a review of the literature it is tentatively speculated that nodding syndrome may be a particular type of epilepsy, found mainly in certain areas of Eastern Africa, which begins with focal symptoms and later progresses to generalised convulsions. However its causes remain obscure.

The present study relied mainly on data from interviews with local people, and the team recommends that further studies be carried out to investigate this syndrome (including its causes, clinical presentation and prevalence) in more detail. These should include laboratory investigations, brain scans and EEGs, use objective research methods (e.g. case controls) and involve experienced clinicians.

Note: After returning from Witto Payam we found that more than 500 cases of nodding syndrome were being treated in Usratuna PHCC in Juba. The PHCC record review indicated that the cases were coming from the wider community of Southern Sudan.

Recommendations

The Ministry of Health should:

- provide adequate medicine for the symptomatic treatment of affected persons
- urgently conduct research into the possible causes of the nodding syndrome to allow better management of the patients with the illness and to help undertake preventive measures.

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We thank the Editorial team of SSMJ for helping to edit this article, and Edward Luka for additional references.

Malaria in South Sudan 1: introduction and pathophysiology

Charles Ochero Cornelio[a] and Oromo Francis Seriano[b]

This is the first in a series of articles on malaria. It is intended for everyone in South Sudan who diagnoses and treats malaria, and advises on how to prevent it. This article gives an overview of the epidemiology of malaria, the parasite's lifecycle and the pathophysiology of the disease. There is more information in items listed at the end of the article. Also in this issue of the journal are two articles on the diagnosis of malaria. Treatment and prevention will be covered in future issues.

Introduction

Approximately half of the world's population is at risk of malaria and most cases occur in sub-Saharan Africa (1) where 20% of childhood deaths result from this disease. African children have between 1.6 and 5.4 episodes of fever caused by malaria each year (1). In 2008, there were 247 million cases of malaria and nearly one million deaths.. Malaria is an important cause of prenatal anaemia and of preventable low birth weight.

Malaria is a major health problem in South Sudan. The peak period of transmission is during the rainy season - mainly April to October (2). P. falciparum is the dominant species of parasite and responsible for more than 90% of the cases (and for all cases of cerebral malaria) in South Sudan.

Factors affecting susceptibility, symptoms and the progress of malaria

- The host's genetic makeup. For example, the association between sickle cell trait and protection from malaria is well known. As more information emerges from the genetic analyses of malarial disease more associations are being discovered (3).
- The variable virulence of different strains of the malaria parasite. This may account for the wide variation in clinical symptoms.
- The number and frequency of mosquito bites that transmit the parasite and hence induce a (semi-) immune status.
- Other effects on the immune status of the host including HIV infection.

Recent studies (3) indicate that there are clinical and pathophysiological differences in severe malaria in populations of different ages, geographical locations and genotypes.

Immunity to malaria increases after each malaria attack. The following groups are reported to have partial immunity to malaria:

- Newborns - who are protected due to foetal haemoglobin (HbF).
- Those with glucose-6-phosphate dehydrogenase (G6PD) deficiency.
- Children with sickle cell trait who have low parasite rates and fatality rates.
- Those with thalassaemias who get some protection due to HbF
- West Africans because they lack the Duffy antigen needed for the P. vivax attachment; so they have immunity against the P. vivax species.

The role of maternal immunity in newborn susceptibility

The most recent evidence indicates that maternal immunity to malaria does not strengthen the neonate's immunity but instead predisposes the child to severe attacks of malaria later in life (4).

Groups at risk

Within South Sudan the groups at most risk of severe malaria are:

- Young children who have not yet developed their own protective immunity against the most severe forms of the disease (and HbF is reduced). Most of the deaths from malaria are those of young children.
- Semi-immune pregnant women. Pregnant women have a decreased level of immunity making them more susceptible to malaria. The placenta is a good breeding area for plasmodium due to the presence of the adhesion molecules. The hormonal changes during pregnancy make parasite penetration easy.

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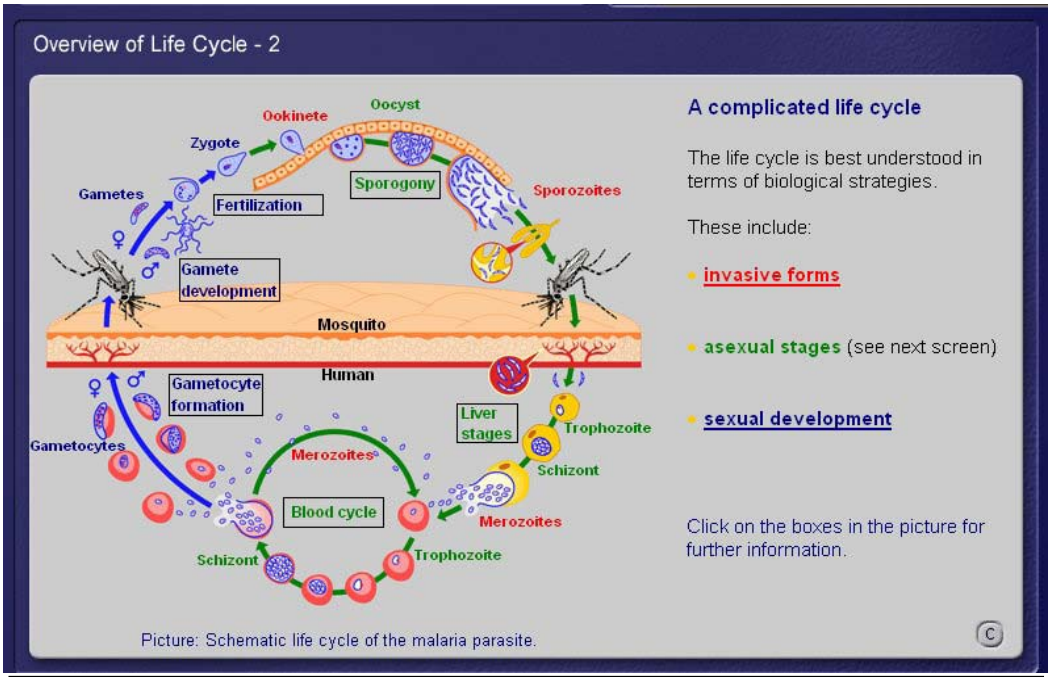

Figure 1: Life cycle of the malaria parasite (One of a series of slides in the Wellcome Trust's CD-ROM 'Malaria: Parasite Biology Tutorial' reproduced with permission from the Trustee of the Wellcome Trust www.wellcome.ac.uk (© Wellcome Trust).

Malaria can result in serious maternal and foetal health risks including anaemia, miscarriage (especially during the first and second pregnancies), abortion, stillbirths and a low birth weight baby. In highly endemic countries like South Sudan, many women have developed some immunity; consequently re-infection with malaria during pregnancy may be asymptomatic, therefore masking the need for treatment.

- Semi-immune HIV-infected pregnant women are at increased risk of malaria during all pregnancies. Women with malaria infection of the placenta also have a higher risk of passing HIV infection to their babies.
- People with immunodeficiency (for example, HIV/AIDS).
- Visitors/Immigrants from non-endemic areas are at high risk of malaria and its consequences because they lack immunity. This includes South Sudanese who have lived in non-endemic areas (and therefore lost their immunity) and return home.

Transmission

Malaria is caused by the Plasmodium parasite whose life cycle is in the Anopheles mosquito and humans. It is almost always transmitted to humans by female anopheles mosquitoes that bite mainly at night. The female mosquito needs a blood meal in order for her eggs to develop. The male feeds only on plants. Four main species of plasmodium cause malaria in humans:

- P. falciparum (causes the most severe illnesses and deaths, and is the most common species in South Sudan.)
- P. vivax (causes mild disease)
- P. ovale (causes mild disease)
- P. malariae (causes a mild disease).

There are occasional rare infections with P. knowlesi and P. simium. It should be noted that both P. vivax and P. ovale can cause a dormant liver stage (hypnozoites) that may lead to manifestations of malaria plus a positive film after months or even years.

Malaria can also be transmitted through:

- blood transfusion
- the placenta
- parenteral accidents.

Life cycle and pathophysiology of the malaria parasite

The life cycle of the malaria parasite (see Figure 1) is divided into:

1. The sporogonic cycle in the mosquito. The mosquito acquires gametocytes when it bites an infected person. These fertilise in the gut and eventually migrate as sporozoites to the saliva.
2. The erythrocytic cycle inside human red blood cells (RBC).
3. The exo-erythrocytic cycle outside RBC.

The erythrocytic cycle of P. falciparum

Pre-erythrocytic cycle

When the mosquito bites, sporozoites are injected with the saliva into the blood stream.

Within 30 minutes they invade the liver cells and multiply there for 7-10 days forming thousands of merozoites.

The pre-erythrocytic stage of infection produces minimal histopathologial changes, cannot be seen under the microscope and there are no detectable symptoms or functional disturbances in the host. (Other species have longer incubation periods with P. malariae being the longest - about 40 days at most. P. vivax and P. ovale species become hypnozoites from which relapses may occur months or even years later).

Erythrocytic cycle

After the pre-erythrocytic cycle, the merozoites burst out of the liver and invade the red blood cells (erythrocytes). Here they develop through ring forms to trophozoites and finally to multi-segmented schizonts.

The exo-erythrocytic cycle of P. falciparum

The infected RBCs rupture after about 12-16 days (for falciparum) but takes longer (up to 40 days) in species like P. malariae. Merozoites and gametocytes are released into the blood stream causing clinical signs. The released merozoites infect more RBCs (thus continuing the cycle of infection). The gametocytes also invade RBCs and are ready to be swallowed by the mosquito when it bites again.

The fever, febrile paroxysysms, headache and a variety of other aches and pains, prostrations plus the familiar and consistent (flu-like) symptoms of an acute attack are probably due to the cytokines released from macrophages and RBCs when the schizonts rupture.

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Further information

- Wellcome Trust. Malaria website at http://malaria.wellcome.ac.uk
- Wellcome Trust. Parasite Life cycle at http://malaria.wellcome.ac.uk/interactive/parasitelifecycle/interactive.html

Thanks to David Attwood for help in preparing this article.

Case History Quiz

'Face of baby'

'Stomach and thighs of baby'

Here are two photographs of a baby boy, aged 11 months, who was born in Nyala, Darfur. The mother is married to her direct/first cousin. This is her 6th child – two of them were born with the same condition – a boy died at age 3 months and a girl at age 27 months. Her brother had two children with the same condition – both died.

Questions

Q1. What is wrong with this baby? What signs of disease can you identify?

Q2. What is the diagnosis?

Q3. What further questions should the doctor ask?

Q4. What causes this condition?

Q5. What treatment should the doctor give?

Q6. Do you think the child is likely to get better?

Have you seen children with this condition? If so, what treatment/advice did you give? What was the outcome?

Answers are on page 16

A case from Darfur presented by Dr Massimo Serventi

Malaria in South Sudan 2: clinical features and diagnosis

David Attwood[a]

The previous article, 'Introduction and patho-physiology', reviewed the mechanism of transmission of malaria, the types of parasite and the life cycle of the malarial parasite. In South Sudan, 90% of malaria is caused by Plasmodium falciparum (P. falciparum). This article focuses on the clinical features and diagnosis of P. falciparum but for completeness will also discuss the other main species of malarial parasites.

The incubation periods of the different types of malaria are shown below:

Type of malaria incubation period

- P. falciparum 12-14 days
- P. vivax 15 days
- P. ovale 15 days
- P. malariae 18 days

However, it must be remembered that P. vivax and P. ovale may present weeks or months after the initial illness due to reactivation of hypnozoites in the liver (1, 2). Furthermore, longer incubation periods may be likely in individuals who are semi-immune or in those taking ineffective anti-malarial prophylaxes. Thus asking about type of anti-malarial taken and compliance is extremely important.

Clinical features

Symptoms

The clinical features may be preceded by non-specific symptoms such as

- malaise
- arthralgia (joint pain)
- myalgia (muscle pain)
- diarrhoea and
- nausea.

The classical presentation of malaria is:

1. The 'cold stage' associated with rigors (shaking).

2. The 'hot stage' where the patient becomes febrile, often exceeding 40°C associated with nausea and vomiting.

3. The 'sweating' stage where the temperature returns to normal (3).

The fever is referred to as a 'swinging fever' and the duration between fevers may point to a certain type of malaria:

- P. ovale/P. vivax 38-42 hours between fevers ('tertian fever')
- P. malariae 62-66 hours between fevers ('quartan fever) (3).

This clinical presentation is due to red blood cell rupture and subsequent merozoite release into the circulation. However in P. falciparum the timing of fevers tends to be less periodic. Other features include

- headache
- abdominal pain and on rare occasions may suggest an "acute abdomen",
- vomiting (3)
- a dry cough. If this occurs then typhoid must be considered in the differential diagnosis.

Signs

Signs of malaria include:

- conjunctival pallor (a sign of anaemia)
- mild jaundice which is caused by haemolysis. P. falciparum may be associated with severe jaundice and is caused by liver damage
- splenomegaly (the spleen is palpable). It takes only a few days for the spleen to enlarge in an acute attack of malaria. However, there are many causes of anaemia, jaundice and splenomegaly
- hepatomegaly (liver enlargement).

Other more general signs are those that may suggest sepsis; and may include tachycardia (fast heart rate) with a bounding pulse, and tachypnoea (fast breathing rate) especially in children.

Chronic malaria

The persistence of malaria in the blood, especially in people who live in subtropical regions, leads to 'chronic

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malaria.' Symptoms may include attacks of 'acute malaria' interspersed with anaemia, weight loss or other infections (e.g. gastroenteritis). Chronic malaria can improve with the patient developing some immunity to the parasite or there may be other complications such as:

- Hyper-reactive malarial splenomegaly. This takes the form of massive splenomegaly causing anaemia, pancytopenia (low levels of red blood cells, white blood cells and platelets), secondary infection, fever and jaundice.
- Quartan malarial nephropathy. This is caused by P. malariae infection and malarial antigens are found in the renal glomerular basement membranes. It presents as a nephrotic syndrome which is a triad of:
- low albumin levels (hypoalbuminaemia) (>3.5g/day) secondary to protein leaking from the kidney into the urine
- high fat levels (hyperlipidaemia)
- oedema.

Complicated malaria

This is characterised by high levels of parasitaemia (≥5 to 10 percent of RBCs affected) (4) and is mostly due to P. falciparum, although patients with complicated infection due to P. vivax have been reported. Those at greatest risk of severe disease are:

- non-immune individuals
- immunocompromised individuals
- children 6 to 36 months old
- pregnant women (1).

The pathogenesis of complicated malaria is due to capillary infarcts, leakage and organ dysfunction. Organ dysfunction may appear as:

- altered consciousness with or without seizures
- respiratory distress or acute respiratory distress syndrome (ARDS)
- circulatory collapse
- metabolic acidosis
- renal failure
- haemoglobinuria (when haemoglobin escapes into the urine turning it dark brown and giving the name 'black water fever')
- hepatic (liver) failure
- coagulopathy (blood clotting abnormalities) with or without disseminated intravascular coagulation
- severe anaemia or massive intravascular haemolysis
- hypoglycaemia.

Poor prognostic markers include:

- impaired consciousness (the deeper the coma the graver the prognosis)
- convulsions >3 in 24 hours
- respiratory distress
- substantial bleeding
- shock
- renal impairment (creat >265 umol/L)
- acidosis (bicarbonate <15 mmol/L)
- jaundice
- raised lactate >5 mmol/L
- hypoglycaemia
- parasitaemia >500,000 parasites/mm3 or >10,000 mature trophozoites and schizonts/mm3
- >5% neutrophils contain malaria pigment.

The combination of renal failure and jaundice carries a particularly grave prognosis (5).

Symptoms of malaria in children

Symptoms of malaria present differently in children and they may display:

- convulsions
- confusion and neurological impairment progressing to coma
- hypoglycaemia
- metabolic acidosis
- severe anaemia.

Jaundice, renal failure and lung complications are less frequently observed than in adults.

Malaria during pregnancy

Pregnant women are a high-risk population. However previous exposure (which can give partial immunity) may mean that patients remain asymptomatic, despite high concentrations of parasites in the placental microcirculation. Pregnant women with no previous exposure are prone to severe infection and are vulnerable to the complications of malaria (6).

Malaria in primigravid (first pregnancy) and secundigravid (second pregnancy) women puts them at higher risk of:

- foetal distress, premature labour and stillbirth
- low birth weight (average reduction 170 g in P. falciparum) (6).

HIV co-infection reduces birth weight further and increases morbidity and mortality associated with the malaria infection. Mothers with HIV may be immunosuppressed and therefore have higher parasite densities and develop more severe clinical disease. Post- partum they are at higher risk of anaemia (7).

Differential diagnosis

Viral illnesses can present with a variety of features including malaise, headache, myalgia and abdominal discomfort. Tachypnoea may indicate an acute respiratory tract infection in a child. A severe dry cough should alert the clinician to the possibility of typhoid or respiratory tract infection especially bacterial pneumonia, tuberculosis (TB) and Pneumocystis jeroveci (PCP) (formerly called P. carinii). However, the cough of PCP and TB is likely to have a longer history than that of malaria or typhoid. The headache, fever and vomiting associated with malaria can be misdiagnosed as meningitis or even an atypical pneumonia. However meningism (photophobia - avoiding bright lights- and neck stiffness) is not seen in malaria. Malaria is not associated with a rash, unless disseminated intravascular coagulation (DIC) ensues and anyone with a fever and a rash should have viral haemorrhagic fever or leptospirosis considered in their differential diagnosis.

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Every fever is not malaria - a message from Kenya

In Kenya we are trying to focus on confirming the diagnosis of malaria using microscopy or rapid test diagnostic kits (RDTs) rather than just treating a presumed clinical diagnosis. Many health staff in dispensaries and health centres still believe that every fever is malaria and that malaria tops the list of diseases even in non-endemic area. This belief is strong particularly among those who have had training in IMCI (Integrated Management of Childhood Illness). One result of this belief is that many patients are given artemether-lumefantrine treatment (AL) unnecessarily with the additional risk that other causes of fever go untreated. I wonder if it is the same in South Sudan?

So the new malaria strategy in Kenya puts emphasis on laboratory confirmation of the cause of a fever to make sure that it really is malaria. We still have problems related to the availability of microscopes and well-trained laboratory technicians – and RDTs are not available everywhere. However, we are addressing these challenges and running workshops to try to change attitudes. In this way we hope that more staff will look for the real causes of fever and not just rush to treat malaria.

Our strategy also focuses on malaria surveillance. We need to convince health staff that malaria prevalence in Kenya is going down due to the scaling up of interventions like LLINs (Long Lasting Insecticide Impregnated Nets), indoor residual spraying, advocacy, community and social mobilisation and AL treatment.

Based on an email sent to the HIFA2015 email forum by Beatrice Muraguri (Health Information Officer, Ministry of Health, Nairobi, Kenya. bemura68@yahoo.com) and published with her permission.

Malaria in South Sudan 3: laboratory diagnosis

Charles Ochero Cornelio[a]

Introduction

The previous article described the clinical features and diagnosis of malaria. However, for a definite diagnosis, the malaria parasite must be seen in a blood film. In this article we cover laboratory tests used to diagnose malaria (1, 2). These include:

- Microscopy including thick and thin blood films (best method for diagnosing malaria)
- Serodiagnosis such as the rapid diagnostic tests (RDTs) and Enzyme-Linked ImmunoSorbent Assay (ELISA).
- Other tests such as Polymerase Chain Reaction (PCR).

How to do the laboratory tests

What type of blood to collect

- Capillary blood (finger or heel prick)
- EDTA anti-coagulated venous blood
- Maternal placental blood.

When to collect the blood sample

- As soon as possible if malaria is suspected. However, it may be necessary to collect blood on several occasions to detect the parasite
- During peaks of fever
- Before the patient receives antimalarial drugs.

Note: Always ask the patient if he/she has taken any antimalarial drug.

Direct diagnosis of malaria

The blood film method for the laboratory diagnosis of malaria remains the gold standard in diagnosing malaria, i.e. blood film examination under the microscope. There are two kinds of blood films: thick and thin. The thick film is used for quick identification and quantification of parasites and the thin film is used for differentiation of parasite species.

You can prepare thick and thin blood films on separate slides or on the same slide. Common stains used are Field's stain, Giemsa stain and Leishman stain.

Thick blood film

This is suitable for the rapid detection of malaria parasites particularly when they are few.

Advantages:

- More sensitive by 30 times than thin films because:
  - * the blood is concentrated allowing a greater volume of blood to be examined and
  - * malaria parasites are concentrated as the RBCs are lysed
- Detects low parasitaemia.
- Can answer the question "Does the patient have malaria?" but only in experienced hands.

Disadvantages:

- Cannot differentiate between species of plasmodia.
- Parasites in the lysed cells are distorted
- Are more difficult to read so laboratories that have limited experience may prefer thin smears.

Thin blood film

This is the diagnostic tool most widely used to identify the parasite species.

Advantages:

- Confirms the plasmodium species
- Greatly assists in the identification of mixed

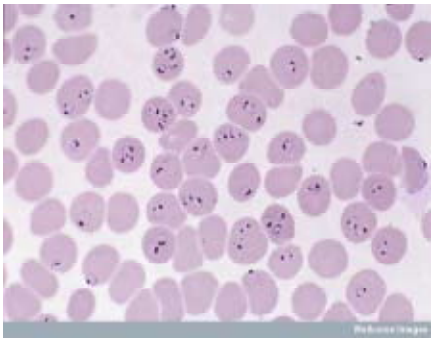

Figure 1: Ring stage P. falciparum. Note the multiplicity of rings within the red blood cells (© Wellcome Images. W0042190).

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infections

- Valuable in assessing whether a patient with falciparum malaria is responding to treatment in areas where drug resistance is suspected
- Provides an opportunity to investigate anaemia and white blood cell abnormalities in the absence of malaria parasites

Disadvantages:

- Less sensitive than a thick film especially where there is a low parasitaemia. This may delay diagnosis if, after a prolonged search, no parasites are found.

An explanation of the direct diagnosis

To establish the diagnosis of malaria, a blood film must be prepared from fresh blood obtained by pricking the finger or a heel (in case of baby/child).

The thin film is fixed in methanol before staining; the thick film is stained unfixed. Many hospitals in South Sudan have a Wright-Giemsa stain available, which is acceptable. However, Wright stain alone will not reliably show Plasmodium parasites. For the best results, the smear should be stained with a 3% Giemsa solution (pH of 7.2) for 30 - 45 minutes.

For rapid diagnosis, make thick and thin smears on separate slides. Air dry the thin film, fix it with methyl alcohol, and immediately stain it. If no parasites are found on the thin film, wait until the thick film is dry and examine it for organisms that might not have been detected on the thin film.

In P. falciparum infections, estimate the parasite density by counting the percentage of red blood cells infected - not the number of parasites - under an oil immersion lens on a thin film. Plasmodium parasites are always intracellular and they demonstrate, if stained correctly, blue cytoplasm with a red chromatin dot.

Common problems in reading malaria smears are caused by:

- platelets overlying a red blood cell
- concern about missing a positive slide
- misreading artefacts as parasites.
- poor staining
- partial haemolysis of red cells.

If smears are persistently negative, an alternative diagnosis should be considered.

A single negative blood film does not exclude malaria.

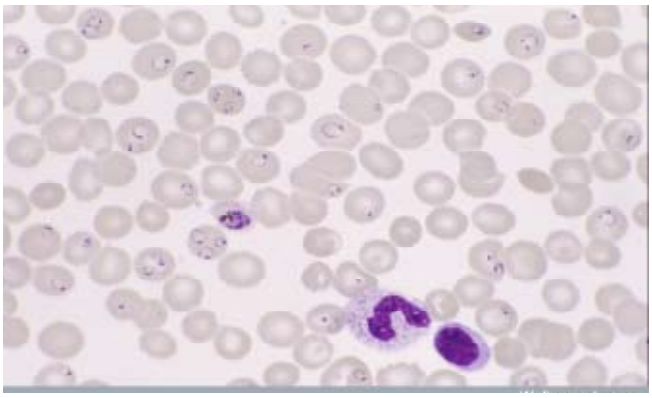

Figure 1: Thin film showing P. falciparum ring stage and a schizont in the centre of the slide.
(© Wellcome Images. W0042199)

A person suspected of having malaria but whose blood films do not show the presence of parasites should have blood films repeated approximately every 12--24 hours for 3 consecutive days. Only when all are negative, we can say that a febrile patient does not have malaria providing:

- we trust the competency of the laboratory technician and the facilities used and
- we can exclude other sources of errors (e.g. a patient received an anti malarial at home before coming to the health facility. This is a very common pitfall).

Figures 1, 2 and 3 show parts of the life cycle of the malaria parasite. See a full explanation in "Malaria in South Sudan: 1. Introduction and pathophysiology" in this issue of the journal.

Indirect diagnosis of malaria

Serodiagnosis of malaria

These are tests carried out by finding an antibody to the malarial parasite in the patient's serum. That is to say, a specific antigen (of a plasmodium species) is mixed with a patient's serum and, if reaction occurs, then it is positive but these kinds of tests have their own pros and cons. They are no use in the diagnosis of acute malaria because:

- They do not differentiate species
- They are tests that detect antibodies and an antibody can remain positive for years after a malarial infection.

Then when do we use these tests?

- To exclude malaria in a patient suffering from recurrent bouts of fever but presenting at the moment with no fever

- In surveys measuring the extent of population exposure to malaria. The commonest test is the Immunochromatographic Test (ICT). The ICT is the type of Rapid Diagnostic Test (RDT) available in South Sudan. The ELISA (Enzyme-Linked ImmunoSorbent Assay), another form of test used for detecting specific proteins of the plasmodia species, is not found in South Sudan.

Other new methods

- Quantitative Buffy Coat (QBC) is used when there is a low parasite count. A capillary tube is used to concentrate the blood and parasites are detected according to the specific gravity of the RBCs.
- Polymerase Chain Reaction (PCR) is used for detecting the parasitic DNA (mostly for detecting the resistant strains) and is a very sensitive method.

These modern methods are only useful in research studies and have never replaced the thick and thin blood films for routine clinical diagnosis of malaria.

Note: In South Sudan, only blood films and the antibody detecting test (ICT) are presently used.

Other laboratory investigations to accompany the blood film

- Haematological parameters (haemoglobin and haematocrit) to detect how far the RBCs are affected.
- Blood glucose – to detect how far the parasite has caused hypoglycaemia through stimulating glucogenolysis (which is a bad prognostic factor) and also before starting quinine which causes hypoglycaemia as a common side-effect.
- Coagulation studies (malaria affects the coagulation status)
- Screening for G6PD Deficiency (to avoid the antimalarials like primaquine that precipitates haemolysis)
- Renal function tests (malaria affects the kidneys either directly or by hypovolaemia caused by vomiting, diarrhoea and fever)
- Urinalysis (to detect haemolysis).

Laboratory diagnosis of malaria in children[3]

In all children suspected of severe malaria, check:

- thick blood film (and thin blood film if species identification required)
- haematocrit.

In children with altered consciousness and/or convulsions, check blood glucose.

Children with positive blood films and the following findings have severe malaria:

- severe anaemia (haematocrit <15%; haemoglobin <5 g/dl)
- hypoglycaemia (blood glucose <2.5 mmol/litre or <45 mg/dl).

In suspected cerebral malaria (i.e. children with unrousable coma for no obvious cause), perform a lumbar puncture to exclude bacterial meningitis - provided there are no contra-indications to lumbar puncture.

Contraindications to lumbar puncture

- Suspected increased intra cranial pressure especially if demonstrated by fundoscopy (i.e. papilloedema)
- Any suspected intracranial space occupying lesion e.g. brain abscess, tumour
- Coagulopathy
- Local infection at the lumbar puncturing site
- Midline shift in the Computer Tomography (not found in Southern Sudan).

Note: There are many contraindications divided into absolute and relative but these are the most common and important ones.

If you cannot exclude bacterial meningitis, give treatment for this also. If you suspect severe malaria on clinical findings and the blood film is negative, repeat the blood smear. Review the child clinically (history and examination) and review/consider the differential diagnosis, i.e. diseases causing febrile convulsions.

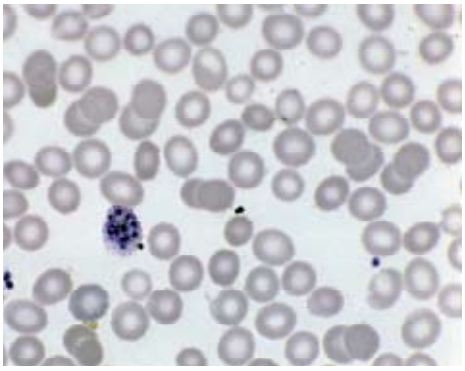

Figure 1: P. ovale schizont. Affected cell is oval, enlarged, irregular border and contain less than 12 merozoites
(© Wellcome Images. W0042150).

Laboratory prognostic indices

Laboratory indicators of poor prognosis in falciparum malaria include:

- Heavy parasitaemia i.e. when >5% of RBCs are parasitized
- Presence of mature trophozoites, malaria pigment and schizonts
- Peripheral leukocytosis of >12000/ml
- Low CSF lactate level
- Low antithrombin III levels
- Serum creatinine of >265umol/l
- Blood urea nitrogen >21.4 mmol/l
- Packed cell volume (PCV) of less than 20%
- Haemoglobin of less than 7.0g/dl
- Blood glucose of less than 2.2mmol/l
- Raised serum aminotransferases.

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Further information

- Wellcome Trust. Malaria website at

  http://malaria.wellcome.ac.uk

- WHO. See the following sites: 'Diagnosis of malaria' at http://www.who.int/malaria/diagnosis_treatment/diagnosis/en/index.html and 'Malaria' at http://www.who.int/topics/malaria/en

Thanks to David Attwood for help in preparing this article and to the Wellcome Trust for allowing us to use their images.

Answers to Case History Quiz from Page 9.

A1. General condition is poor; the child is crying and looks as if he is suffering and in pain; a taught/tight membrane resembling parchment covers his skin. There is ectropion, eversion of lips, flattening of the nose and pus secretion from the eyes and base of the nose.

A2. Harlequin ichthyosis. This is characterised by the taught/tight membrane covering the entire body surface. The involvement of eyes (ectropion), mouth (fish-like deformity – eclabium) and distorted flat ears are typical features. Differential diagnosis: Collodion baby - which has a similar presentation of a taught membrane, but is less severe and lacks the additional facial features. The membrane in this condition typically breaks up and peels off in the 1st two weeks of life. Conditions associated with this include lamellar ichtyosis, ichthyosiform erythroderma and Netherton's syndrome.

A3. This is a clinical diagnosis. Questions that can help in making the diagnosis are: a) Is the couple consanguineous (blood relatives)? b) Does the couple have another child with ichthyosis? c) Does the family have a history of severe skin disorders?

A4. It is an autosomal recessive disease, transmitted by both parents. In this case they are direct/first cousins.

A5. Give: a) Systemic antibiotics. The use of prophylactic antibiotics is debatable, but probably indicated if the baby cannot be nursed in a sterile environment. b) IV fluid. This is very important as affected infants are often unable to feed.c) Systemic retinoids. Acitretin 1mg/kg/day and isotretinoin 0.5mg/kg/day both enhance/improve survival and reduce morbidity. Water is lost through the skin. So bathe the baby twice a day and use sodium chloride compresses followed by bland emollients to soften hard skin. Do not use salicylic acid preparations. Use eye lubricant ointments rather than eye drops.

A6. No, this child seems very seriously affected. Possible causes of death are sepsis, dehydration and malnutrition.

Thanks to Chris Bower for helping to provide answers for the quiz.

SSMJ is proud to support the continuing development of the health care system in South Sudan as the country moves towards Independence. Please share with us your experiences during this period, particularly those related to the challenges caused by the large number of 'returnees'. Email admin@southernsudanmedicaljournal.com

Knowlege, attitudes and practises of caretakers of malnourished children in Aweil East and North Counties, South Sudan

Cyprian Ouma[a]

Introduction

Malnutrition is a chronic public health problem in Aweil East and North counties with an estimated prevalence of between 15% and 25%.

Underlying contributing factors include: political instability, poor infrastructures, droughts and floods resulting in low crop yields, poverty and limited awareness of good nutrition and health practices.

At the time of the survey there were six decentralised centres feeding severely and moderately malnourished children below 5 years. As well as feeding activities, nutrition and health education was given in order to improve health and nutrition awareness.

The objective of this survey was to assess knowledge, attitudes and practices of mothers (or caretakers) of children admitted in the feeding programme.

Methodology

The survey was conducted with the help of Tearfund in Aweil East and North counties in northern Bahr-el-Ghazal region in September 2006. This was two months after the feeding programme started when there were 1472 beneficiaries. The respondents were mothers/caretakers of:

- children aged under 2 years who were in the feeding programme
- breastfeeding children aged less than 6 months.

We selected a sample of 50 children from the 1472 beneficiaries using a systematic simple random sampling method (because the number of the children in each feeding centre was known). We interviewed their mothers/caretakers using a questionnaire to obtain information about their knowledge, attitudes and practices on various nutrition and health topics.

Results and discussion

Infant and young child feeding practices

Table 1 shows the responses on child feeding practices from the 50 respondents. Most (94%) had breastfed their babies within one hour of birth and 6% gave cow milk immediately. 82% breastfed on demand especially during daylight, and 69% breastfed 2-3 times at night.

Based on a 24-hour recall all the children aged above 6 months had eaten some kind of food in the previous 24 hours of which

- 18% took food and milk once a day.
- 58% ate food and 24% drank milk twice a day
- 24% ate food and 28% drank milk 3 to 4 times a day.
- 32% did not take milk at the time of the survey.

No mother used infant formula/powdered milk; 13 were unaware of oral dehydration salt solution (ORS). No child was taking ORS, supplements or medicines.

In response to the question "When do you think it is best to give foods other than breast milk to the children"

58% of the respondents said they gave cow milk before 6 months of age and 40% started at or before the age of 3 months. 24% started complementary feeding after the age of 6 months (these were mainly mothers who had no access to cow milk).

Knowledge/Attitudes about food rations (Unimix, BP-5 and Plumpynut) and immunisation

All the mothers appreciated the food ration given to their children. They thought it improved the children's health and the children enjoyed the ration more than the local foods at home.

Most mothers had good knowledge and attitudes towards immunisation and said it protected against diseases such as polio and measles. Two mothers did not like immunisations, especially the polio vaccine, because they said it made children sick; one mother said that the child's father was against immunisation because it was against their culture.

Knowledge about the causes of malaria, diarrhoea and malnutrition

When asked what causes malaria, diarrhoea and malnutrition mothers gave the following responses:

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Table 1. Number and percentages of responses on child feeding practices

Practice	Yes		No	
	N	%	N	%
Breastfed baby less than an hour after delivery	47	94.0	3	6.0
Child was given cows milk immediately after birth	3	6.0	47	94.0
Child was breastfeeding at the time of the survey	39	78.0	11	22.0
For the 39 breastfeeding children:				
Child fed on demand at night	12	30.7	27	69.3
Child fed on demand during daylight	32	82.1	7	17.9
Mother had heard of or seen ORS	37	74.0	13	26.0
24 hours prior to the survey child had taken:				
Plain water	49	98.0	01	2.0
Sweetened or flavoured water	1	2.0	49	98.0
Tea or infusions	13	26.0	37	74.0
Infant formula/powdered milk	0	0.0	50	100
Fresh milk	34	68.0	16	32.0
Other liquids e.g. soup	47	94	3	6.0
Mushy or solid foods like cereals, porridge, stews	50	100	0	0.0
ORS	0	0.0	50	100
Vitamin, mineral supplement, medicine	0	0.0	50	100

- Malaria: 28 (56%) said mosquito bite; the rest did not know (9) or said hunger (7), exposure to cold (3), impure food and water (2), fatigue (2) or rain (1).
- Diarrhoea: 41 (82%) said dirty water or food; 8 did not know and one said teething.
- Malnutrition: 29 (58%) said diseases, 31 (62%) said lack of food; 16 did not know and 2 said 'evil eye'.

Diarrhoea: prevalence, health-seeking behaviour and feeding

Respondents were asked:

- "Has your child had diarrhoea in the last 24 hours?" Three (6%) answered yes.
- "Has your child had diarrhoea during the last month?" 9 (18%) answered yes.

Of the 12 children who had had diarrhoea, 9 were taken to a health facility for treatment, 2 to a feeding centre and one was not taken for treatment.

Figure 1 shows that when children had diarrhoea most (>50%) ate, drank and breastfed as normal but that 33% ate less, 25% drank less and 8% breastfed less. Reducing food and fluid intake is an inappropriate way of managing diarrhoea and should be remedied through nutrition and health education.

Practices related to water and hygiene

When asked what they did to make water safe, only 2 (4%) of respondents said they boiled their water, 21 (42%) said they filtered it and 27 (54%) did not treat water at all. Most people said they used treated water for drinking. However, a few also filtered water for bathing to prevent guinea worm infestation. The population receives cloth filters from the Carter Centre in accordance with the eradication of guinea worm policy in the area.

When asked when they wash their hands, only 6 (12%) of respondents said after using the latrine/defecating, 17 (34%) said before preparing food, 41 (82%) said before eating food and 36 (72%) said after eating food (see Figure 2).

When asked when they thought it was necessary to wash

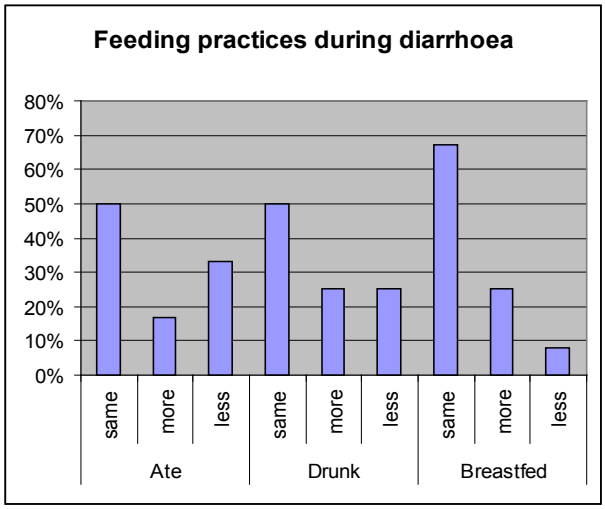

Figure 1. Changes in feeding practices during diarrhoea.

hands with soap, 38% of respondents said after using the latrine/defecating, 8% said before preparing food, 36% said before eating food and the majority (48%) said after eating food. However, only 14 (28%) of respondents said they actually used water and soap to wash hands, and only 6 (12%) after using a latrine; the rest used water alone. Only 9 (18%) used soap to wash dirty utensils.

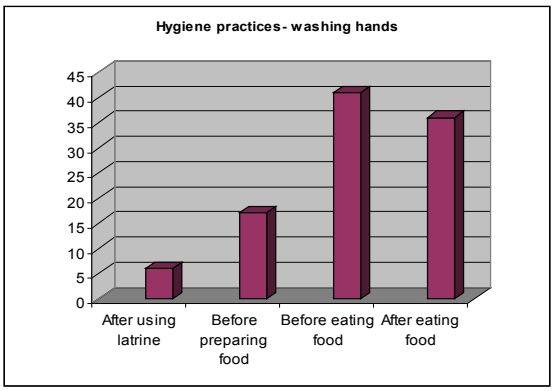

Figure 2. Number of respondents saying when they washed hands.

Conclusions

- There were good breastfeeding practices in the community.
- Complementary feeding practises were not very good:
- the majority of children were fed food and milk two or less times a day
- the age at which complementary foods were introduced ranged from one to 12 months; therefore some children received these foods too early and some too late.
- Almost all mothers accepted that immunisation protected against childhood diseases.
- All the mothers thought the food ration improved their children's nutritional status.
- For diarrhoea most respondents' health seeking behaviour was good, but for many its nutritional management was relatively poor. Many either reduced or did not increase fluid or food intake.
- More respondents knew the cause of diarrhoea compared to causes of malaria and malnutrition. However, few had good hygiene practices (hand washing and boiling drinking water) that help to prevent diarrhoea.
- Most people used water without soap for washing their hands and utensils. However the survey did not investigate whether people could afford soap or if they normally bought it.
- As well as attitudes and behaviour, factors like availability and accessibility/affordability of products such as food and soap might have contributed to the low levels of putting knowledge into practice.

Recommendations

1. There is need to continue with nutrition and health awareness in the feeding centres as well as in the community through continuous health and nutrition education.
2. The nutrition extension workers should continue to visit the families of the beneficiaries to ensure that mothers and other caretakers put the knowledge acquired into practice.
3. Distribution of soap in the feeding programme remains necessary in order to improve hygiene practices.
4. There is a need to further study complementary feeding practices and to intervene appropriately.

Acknowledgement: thanks to Tearfund, the staff of Aweil East and North counties and the respondents.

MULTI-INDICATOR NUTRITION, HEALTH, WASH AND MORTALITY CLUSTER SURVEY: TWIC COUNTY, WARRAP STATE. 2010

Each year GOAL in South Sudan conducts a Multiple Indicator Cluster Survey (MICS) at their field sites in Twic County (Warrap State), Agok (Abyei) and Sobat (Ulang and Baliet Counties, Upper Nile State). These give comprehensive and representative data on many indicators including: nutritional status; mortality; morbidity in the last two weeks; immunisation coverage; child feeding practices; household food security; water; sanitation & hygiene; maternal and general health; malaria prevention; HIV/AIDS, and education.

Selected young child-related findings from the recently published MICS survey from Twic County are:

- According to the WHO Growth Standards a quarter of young children were malnourished (GAM =~25%) and about 6% were severely malnourished.
- Under-5 mortality rate for the 3 months prior to the survey was 1.05/10,000.
- Percent of households where children under 5 years had slept under LLITN the previous night (of households with children under 5 years) was 53%.

Reference: Jemal Seid. 'Findings of a Multi-Indicator Nutrition, Health, WASH (Water and Sanitation Hygiene) and Mortality Cluster Survey. ' March 2010. GOAL. South Sudan. To obtain a copy of this and other MICS reports send an email to acdprog@goalsouthsudan.org

Redevelopment of the Martha PHCC Eye Clinic, Yei, South Sudan

Poppy Spens[a]

The redevelopment of the Martha PHCC Eye Clinic took place during 2010. Figure 1 shows what the building looked like at the start of the year. It had been constructed as two wards for the former eye clinic, and was to have been operated by an NGO. However, it was never completed and for several years the empty building was used by youths for drug taking and graffiti, and to generally cause a nuisance to those who lived nearby.

In 2009 the building was handed over to the Diocese of Yei, which owned the site, and plans were made to develop it as a new eye clinic, with full facilities and an operating theatre. As the building was large, it was decided that the remainder would be refurbished to create a much-needed paediatric ward for 10 children.

Figure 1: The original building.

In the early part of 2010, the Martha PHCC project manager worked with a local contractor to specify what refurbishments were required. These included the complete renovation and upgrading of the building to include running water and solar electricity.

Work began at the end of March and was completed by the end of June at a cost of nearly US$64,000. This was fully funded by a UK charity called The Brickworks. As Figure 2 shows, the building has been finished to a high standard. You can see the solar panels on the roof and the water harvesting in the foreground. Elsewhere on the site, the pit latrines have been refurbished, three new washrooms have been constructed and a kitchen shelter has been built so that families can cater for their in-patient relatives.

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Figure 2: The refurbished building.

The paediatric ward was the first part of the building to be opened and the eye clinic was opened on November 1st. We are very grateful to Dark and Light, Light for the World, ECS Diocese of Yei and the Brickworks who together have enabled us to set it up. We have an ophthalmic clinical officer, Mr Abui Simon, who recently trained in Juba with the Christian Blind Mission. We thank this Mission and a church in Ireland for assisting Abui to complete his training.

The opening of the clinic coincided with a visit from Dr Trevor Buchanan (ophthalmologist from Ireland) and Mrs Pam Whitehead (optometrist from Southampton). These two visitors helped Abui Simon to assemble equipment and start the clinic (see Figure 3).

The eye clinic has been busy from the start. Using the Martha PHCC mobile clinic, outreaches have taken place on several Saturdays to identify and treat people with eye disease in villages. We are drawing up a list of patients waiting for cataract surgery and hope that the first cataract operations will take place in February 2011.

Photos from Poppy Spens

Figure 3: Abui Simon examining a patient

News and Resources

These resources are listed under:

- Chronic diseases
- HIV and other infections
- Maternal and child health

Chronic diseases

Lancet series on chronic diseases

The Lancet launched its third series on chronic diseases in November 2010, this time focusing on the "collective failure" in addressing the global state of chronic diseases. The articles look at what is preventing chronic diseases from receiving the global attention they need, as well as what systems, policies, and interventions (and their barriers) are needed to reduce the burden of chronic diseases in low- and middle-income countries. To access the entire series, including commentaries and articles go to http://www.lancet.com/series/chronic-diseases-and-development. To gain access to complete articles register at http://www.lancet.com/user/register

[seen on ProCor www.procor.org 12Nov2010. Procor is a global community promoting cardiovascular health. To get ProCor's free weekly email summary of the latest news, knowledge, and discussion about cardiovascular health visit the website or email subscribe-procor@list.procor.org]

Understanding cardiovascular diseases (CVD)

The Heart and Stroke Foundation of South Africa has produced a useful 2-page educational brochure for clients that includes a breakdown of all risk factors and warning signs of CVD. Topics include: heart attack, cholesterol, blood pressure, diabetes, exercise, nutrition, smoking and stress. Download from http://bit.ly/gE0PQP

HIV and other infections

TB kills 4,000 people with HIV each day

More needs to be done to identify and treat HIV in TB patients, and to prevent TB in people with HIV. The new TB control data from the WHO http://www.who.int/tb/publications/global_report/2010/en/index.html) show that the majority of people with HIV and TB are still not receiving antiretroviral therapy or isoniazid preventive therapy. Nevertheless the findings do show improvement in rates of HIV testing among TB patients. Across sub-Saharan Africa, 53% of TB cases were tested for HIV in 2009 compared to 38% in 2007, suggesting that efforts to promote integration of HIV and TB activities is beginning to have an impact. Forty-six per cent of TB patients tested for HIV had a positive result in 2009 in sub-Saharan Africa. However some important interventions that have the potential to improve the health of people with HIV and TB, or to prevent the development of TB in people with HIV, still have limited coverage. Cotrimoxazole as an adjunct to TB treatment reduces the risk of death in people with HIV.

To read the full article by Keith Alcorn download it from HATiP, issue 169 (10 December 2010 page 2) at http://www.aidsmap.com/hatip. To get regular email bulletins from HATiP go to http://www.aidsmap.com/resources/Email-bulletins and click on 'Email bulletins'.

New WHO guidelines: Tuberculosis prevention for people with HIV

People living with HIV can be protected from one of their deadliest threats – tuberculosis - with a regular, low-cost preventive medication according to new 2010 guidelines from WHO. Of the nearly two million AIDS-related deaths each year, a quarter of them are associated with tuberculosis.

Key recommendations are:

- All children and adults living with HIV, including pregnant women and those receiving antiretroviral treatment, should receive isoniazid prevention therapy.
- Isoniazid should be prescribed for six to 36 months, or as a life-long treatment in settings with high HIV and TB prevalence.
- People living with HIV who may have symptoms of TB should be further screened for active TB or other conditions so that they are able to access the appropriate treatments

Download the guidelines from http://www.who.int/hiv/en [From WHO Press release of 1 December 2010]

Effect of circumcision of HIV-negative men on transmission of human papillomavirus to HIV-negative women: a randomised trial in Rakai, Uganda

The findings indicate that male circumcision should be accepted as an efficacious intervention for reducing the prevalence and incidence of HPV infections in female partners. However, protection is only partial; the promotion of safe sex practices is also important.

Reference: Wawer MJ etal. The Lancet; 377(9761):209 -

218, 15 January 2011

doi:10.1016/S0140-6736(10)61967-8

Web Platform for support in infectious diseases and oncology

The iSA (International Support Action) Clinical Platform at www.isaplatform.com, is a global platform for the exchange of clinical cases. The aim is to provide expert diagnostic opinion to hospitals in Africa, which may not have the knowledge to manage certain complex cases in infectious diseases and oncology.

[Seen on HIFA 2015 10Nov10]

WHO leaflet summarising guidelines for rehydration of patients with cholera

Download at http://whqlibdoc.who.int/hq/2004/WHO_CDS_CSR_NCS_2003.7_Rev.1_eng.pdf

Maternal and child healths

Latest WHO publications on family planning

1. Medical Eligibility Criteria for Contraceptive Use. 4th edition http://www.who.int/reproductivehealth/publications/family_planning/9789241563888/en/index.html.

The recommendations have been updated and include immediate insertion of IUDs postpartum. IUDs are a safe and effective long term contraceptive, but there are a number of misunderstandings that have grown up around their use. These are acting as a barrier for women to have a safe long-term device to prevent unwanted pregnancy.

2. Family Planning Global Handbook for Providers - a publication for working with clients to support informed choice. http://www.who.int/reproductivehealth/publications/family_planning/9780978856304/en /

New hope for mother-to-child transmission of HIV during breastfeeding

A new study shows that giving a triple antiretroviral therapy (ART) during pregnancy, delivery and breastfeeding cuts the risk of mother-to-child transmission (MTCT) of HIV by 43% compared with the standard regimen of zudovidine and niverapine.

The Kesho Bora study – 'A better future' in Kiswahili – offers new hope for preventing HIV infection and death among infants in low-resource settings. The study was a randomised controlled trial in antiretroviral-naïve pregnant woman infected with HIV-I conducted in five sites in Burkina Faso, Kenya and South Africa. The findings have influenced the revised WHO guidelines on prevention of MTCT. WHO now recommends providing combination ART to all pregnant women with CD4 count at or below 350 cells per µL and to provide ARV prophylaxis (to either mother or child) for the entire duration of breastfeeding if the mother is not already on ART.

Reference: Recquet r & Ekouevi DK. Breastfeeding, triple ARV prophylaxis, and MTCT prevention. Lancet Infectious Diseases 14 January 2011 doi:10.1016/S1473-3099(10)70299-1

See also Impact of HAART during pregnancy and breast-feeding on MTCT and mother's health: the Kesho Bora study at http://bit.ly/gbUq3L

Pediatric HIV Treatment Toolkit

The Pediatric HIV Treatment Toolkit from Aidstar-One (from USAID) provides tools, resources, and training materials for managing pediatric HIV care and treatment services to meet the latest (2010) WHO and national treatment guidelines. It provides tools, resources, and training to assist in:

- Managing pediatric HIV care and treatment services
- Updating programs or sites to meet the latest WHO and national treatment guidelines

Download the toolkit from http://j.mp/g7zDh5

[From AFRO-NETS www.afronets.org – see below]

WHO CHARTS FOR EVERYONE CARING FOR CHILDREN IN HOSPITAL

We reproduce here Chart 5. How to give oxygen from 'Pocket Book of Hospital Care for Children - Guidelines for the Management of Common Illnesses with Limited Resources' WHO 2005. See the whole book at http://www.ichrc.org/. We published Charts 1, 2, 3 and 4 in previous issues of this journal (see vol 3 nos 1, 2, 3 and 4).

You can use these charts in different ways. For example, you can print them and display them in relevant wards or clinics (laminated if possible), or use them as a 'memory aid' in your pocket, as handouts or as training aids.

We thank the WHO for permission to reproduce these charts, and Dr O'Hare who gave us the idea of making the charts more widely available.

The SSMJ Editorial Team would like to thank everyone who helped to prepare this issue especially Charles Bakheit, Chris Bower, Richard Idro, Moses R. Kamya and Peter Newman.

CHART 5. How to give oxygen

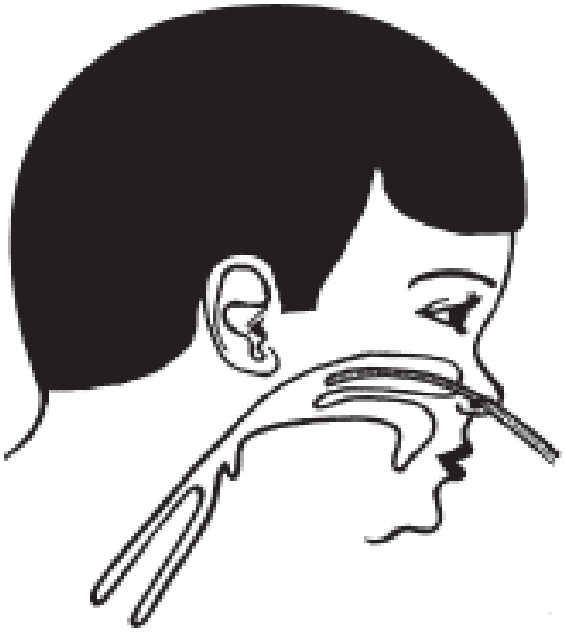
Give oxygen through nasal prongs or a nasal catheter

Nasal Prongs

- Place the prongs just inside the nostrils and secure with tape.

Nasal Catheter

- Use an 8 FG size tube
- Measure the distance from the side of the nostril to the inner eyebrow margin with the catheter
- Insert the catheter to this depth
- Secure with tape



Start oxygen flow at 1–2 litres/minute
(see pages 281–284)