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MALARIA

David Parkinson

The Solomon Islands is spread over a fairly wide area of sea. It is divided into Provinces, (Western Province and Ysabel Province) to the West and Makira and Temotu to the East. Most of the malaria in the Solomon Islands, in the east, is very low endemicity and in the west is also of very low endemicity. The places we have to worry about are the islands in the Central Group, Malaita, Guadalcanal and the Floridas. Where you are right now and where you intend to dive.

Malaria has a long history in the Solomon Islands. It goes back as far as Mendana and his group of men who came here in 1568. Many of these people died, mostly of intermittent fever which was later on thought likely to have been malaria. Mendana returned later to Santa Cruz where, once again, his men fell ill with the dreaded intermittent fever and many died. The descriptions closely resemble those of malaria. There is little record of malaria in our history books after that, possibly because the western world lost sight of the Solomon Islands and it was not until the 19th century, when the first missionaries, gold miners and entrepreneurs visited the Solomons that we began to hear again of the dreaded fevers. These people came to the Solomon Islands and tried to settle but found it was difficult, mainly because they became ill with the fever and many of them suffered for many weeks. Some of them became immune to the disease and stayed, others developed blackwater fever and died and this was about all we heard.

In the early 20th century, we had very little literature and it was not until 1913 when hospital statistics began to be collected that we began to learn a little of what was happening with the fevers in the Solomon Islands.

Between World Wars I and II, there were many admissions to hospital. By that time, there was a means to find out what the cause of the fevers and sickness was. There was a tremendous amount of malaria found in Guadalcanal and in Tulagi. There was not so much found in the West or in other Provinces. In Guadalcanal, in fact, most of the northern side was unpopulated or very lightly populated because people got malaria when they tried to settle and died. This was the indigenous population, as well as the expatriate population.

During World War II, the greatest causes of illness and morbidity were malaria and dysentery but malaria took first place and, in 1942, during the campaign on Guadalcanal, the number of malaria cases was 722 per thousand. In some cases there were up to 3,000 cases per 1,000 per month during some months of that particular year. In 1943, there was a slight improvement due to the use of stringent methods of malaria control which were introduced to the US, British and Australian Armies. This was the result of some very astute work at Cairns and on the Atherton Tablelands by the Land Headquarters Medical Research Unit of the Australian Army.

By 1944, the number of malaria casualties was reducing because of the intensive malaria control measures that were being undertaken on the north coast of Guadalcanal. However, in 1945, when all the hostilities had ceased and the northern pan of Guadalcanal was being used both as a rest and recreation area and as a source of fresh vegetables for the rest of the Pacific, the incidence of malaria could not be reduced below 20 per thousand.

After the second World War, many surveys were undertaken of the various medical problems in the British Solomon Islands Protectorate, as it was then known, including malaria which caused high morbidity and mortality amongst all age groups and, in particular, amongst children. A comparison of north Guadalcanal and similar areas in Papua New Guinea showed infant mortality rates of the order of 250 per thousand per year, mostly due to malaria or malaria related causes.

As a result, the Medical Department decided to establish a pilot project to find some means of controlling the disease, mostly here in Guadalcanal, on Savo Island and some other island groups. The methods that were used were based on the spraying of DDT or a residual insecticide inside every household in the Solomon Islands. This required a large manpower force organised along military lines which could carry around sufficient spray cans and spray the houses. It might seem an easy task but, in actual fact, it was very difficult and required caution in the amount of insecticide put on the walls of each house. However, it was done and when the pilot projects were concluded, the results were similar to those in Papua New Guinea.

Malaria prevalence rates came down from the order of 84% to around 10% in a matter of two years. The studies that I did with Dr Schofield and others in the Wam area in Papua New Guinea showed that infant mortality rates in the first five years of such programs came down from 250 per 1,000, to 80 per 1,000 per year and in the next five years to 40 per 1,000 per year.

The pilot projects were so successful that it was decided to try and conduct a country-wide program in the Solomon Islands. The people who undertook it, did so with a great deal of dedication and, within five years, the incidence of malaria was reduced to very low levels, particularly in the Western and Eastern Provinces but never down to such low levels across the central part of the country. Here it only reached the figures that we saw at the end of the second World War of 20 per 1,000 per year. Despite this it was decided during the mid-1970s to stop spraying in the East and West of the country. This was done at a time when the

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next long term cycle of malaria was due to begin. Unfortunately, the mass drug administration programs which were also conducted in the central groups of islands, were also stopped at that time. As a result, the cases of malaria started to increase rapidly. There were 3,000 cases in 1974 but the number of cases rose rapidly so that in 1983, only 9 years later, there were 84,000 cases.

What happened between 1974 and 1983? As I mentioned, the first thing that happened was that spraying was stopped in the West and East. This allowed the anopheles mosquito, which transmits malaria, to multiply in great numbers. In addition, there was increased economic development around that time. Population movement increased, particularly between the West of the country and the island of Guadalcanal and between the Florida Islands and Guadalcanal. These were the main areas affected with case numbers and case loads growing rapidly. In addition to this, chloroquine resistant strains of falciparum malaria were introduced from Papua New Guinea. This occurred in 1978 and, gradually spread across the country, from West to East. It was quite remarkable because in 1980 during i*n vitro* sensitivity tests in the field we found that there was no chloroquine resistant falciparum malaria in Guadalcanal, nor was there any in Malaita but quite a lot was found in the Western Province. We had to carry around a generator weighing about 175 kg and had to put it across a little canoe to take it from village to village hoping that someone would overturn the canoe so the Army would provide us with a new, lighter generator. We carried out these tests in the field and found there was quite a lot of chloroquine resistant falciparum malaria in the West, but none in the central group of islands of Malaita, Floridas and Guadalcanal in 1980. As there was an epidemic of malaria at that time, by 1981 there was plenty of chloroquine resistant falciparum malaria throughout the central group of islands. So, within one year, there was a complete change in the pattern of sensitive and resistant strains and the parasite species ratio.

When malaria parasite species were first looked at in the Solomon Islands there was a predominance of Pl*asmodium vivax*, which does not kill but makes people very sick. By the time chloroquine resistant falciparum malaria had appeared, the species ratio had been inverted and there was a predominance of *Plasmodium falciparum* which exists today.

Dr William Osler was probably the first person to elucidate the principles of malaria eradication around 1906. These were (a) to protect people from bites by infected mosquitoes, (b) to reduce the number of mosquitoes, and (c) to radically treat patients. This is exactly what we are doing today but with slightly different technical means of doing it.

When we refer to drug resistant malaria, this does not mean an absolute degree to resistance. The common parasite which is resistant to the drugs we use in malaria treatment is *Plasmodium falciparum*.. Unfortunately, it is the killer parasite and quite a nasty parasite; it creeps up insidiously and it makes people very sick, frequently causing death or severely debilitating them. So severely that in areas where you have a predominance of chronic malaria, most people have a haemoglobin level of about 70%, therefore an oxygen carrying capacity of around 70% and all the repercussions of that.

Here in the Solomon Islands at the moment, about 50% of falciparum malaria is resistant to chloroquine, this being the most commonly used drug in treatment of malaria. About 50% is sensitive so that is not a bad level. Of the 50% of resistant strains, 95% are resistant at the R1 level or at the level of least resistance. About 2% or about 3% are at R3 level but we rarely see these cases. They are fairly easy to treat once you get them.

How do you recognise chloroquine resistant falciparum malaria? When a person first falls ill with malaria, they are treated with chloroquine for 3 days. After treatment, the patient recovers both clinically and parasitologically. In fact, the parasites clear completely from the blood. If the parasite is sensitive, there will be no further recurrence at all and no relapse because falciparum malaria does not have a secondary tissue phase. Once it is treated and it has gone, it has gone. However, with the chloroquine resistant strains there are a few parasites which remain in the bloodstream at sub-patent level, so they cannot be recognised by looking at the blood, they are just there. The malaria recrudesces from between 7 days and a month after the first treatment and the patient will come in again complaining of the usual prodromal symptoms of malaise, flu-like illness, joint pains, etc., which will progress on to a rigor and which will eventually become synchronised and will re-occur about every 48 hours. At that stage, having treated the case, knowing that the patient has come back with another rigor, another attack of malaria between 7 and 28 days after the first, you suspect that he probably has an R1 type of chloroquine resistant falciparum malaria. If it is the R2 type, there is a slightly different picture. Initially, after the treatment with chloroquine, the patient responds fairly well but, the parasitemia only comes down to about a quarter of the original level and then begins to climb again and the patient's symptoms return. With R3 type, there is either no improvement or the patient just gets progressively worse.

How does one treat it? The treatment of chloroquine resistant falciparum malaria is fairly simple. First of all, with the R1 resistant strains, the response to chloroquine is still very good, so on the third day when the parasitemia has gone down to a very low level, again usually sub-patent, you give Fansidar, a combination of pyrimethamine and sulphadoxine in a single dose and that is sufficient to mop up the remaining parasites. Why do we give it on the third

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day? Pyrimethamine and sulphadoxine are plasmodistatic drugs as opposed to the plasmodicidal drugs like chloroquine, quinine and amodiaquine. So, first of all, we have knocked down the parasites to a very low level by using plasmodicidal drugs and we only have a very few parasites remaining. These can then be mopped up, so to speak, by the slow acting Fansidar which also remains in the blood for some period of time having a half-life of approximately one week. This is the simple treatment of uncomplicated chloroquine resistant falciparum malaria.

If the person has an R2 or R3 resistant strain, then one would use quinine in the first instance. Quinine is a very old and well tried drug. It is very rapidly absorbed and it is plasmodicidal in its action and reduces the parasitemia very quickly, so that within 24 hours the parasites come down from the order of say, 100,000 per cu mm to about 1,000 per cu mm, then further down so that by the third day most of them have disappeared and are ready to be mopped up by the Fansidar. Quinine alone could be used, but if used alone, it would take 7 days of treatment. I do not know whether any of you have taken quinine but, after the third day, when you feel as though a railway train is rushing around in your head and there are bells ringing all over the place, you are having hallucinations and sweating like blazes and thinking it is worse than the malaria attack you have just had you do not feel like taking any more quinine. Therefore, quinine therapy should be supervised to ensure the drug is taken and dosage reduced where necessary to relieve these untoward side effects of the drug.

So much for the treatment of the uncomplicated case. The complicated cases are much more difficult to treat and would require a separate talk.

Malaria chemoprophylaxis. What should one do when going to an area where there is malaria? The first thing is to find out whether there is any chloroquine resistant falciparum malaria and find out which drugs are effective in prophylaxis. The second thing to do on arrival in the area is to remember hat the anopheles mosquitoes have a predilection for biting at night. They bite around the ankles mostly and if one is covered around the ankles they tend to try and bit further up but not so frequently. They also tend to stay around the rural areas and not so much in the urban areas so one will not find too many around the Mendana Hotel. If you stray into the bush at night, you might find some anopheles mosquitoes biting. The female anopheles is the one that bites because she is the only one that sucks blood. The male does not suck blood at all, so it is only the female that transmits malaria.

How do you protect yourself?. You should wear long trousers and socks and long sleeves. If you do not do that, I suggest that you use a repellent. These are the first principles of protecting yourself from mosquito bites. You should also sleep in screened quarters at night or under a net and if you do that, in most cases, you will be lucky enough not to be bitten by an infective mosquito. In addition to that, in areas where malaria is highly prevalent and where it is transmitted constantly throughout the year, it is wise to take some sort of chemoprophylaxis. Some people do not but they are tempting fate and I have treated too many of them and brought them back from death's doorstep to know that they should be taking chemoprophylaxis.

What does one take? Here in the Solomon Islands the parasite is still very sensitive to the very simple antimalarial proguanil, which is the least toxic of all the antimalarials and does not cause many side effects and certainly does not cause any nasty ones. Pyrimethamine cannot be used because there are pyrimethamine resistant strains which are highly resistant. Chloroquine, despite the fact there are chloroquine resistant parasites here, can still be used and it is very effective. Amodiaquine can also be used and belongs to the same chemical group as chloroquine, the 4-aminoquinolines, and is just as effective. Third line drugs will be discussed later. Proguanil has to be taken daily. For people who cannot remember to take daily tablets, it is pointless giving proguanil because it is excreted very rapidly and the blood levels would fall too rapidly to ensure protection. Chloroquine is taken weekly. Fansidar, a third line drug, is to be used only in treatment of chloroquine resistant strains in combination with chloroquine or quinine since it is plasmodistatic and, because of its mode of action, resistance develops very rapidly. As it is our only line of treatment for the drug resistant cases at the moment, we like to keep it in reserve for that purpose.

Dr David Parkinson's address is World Health Organisation, PO Box 22, Honiara, Solomon Islands.

CEREBRAL ARTERIAL GAS EMBOLISM OR CARBON MONOXIDE POISONING A CASE REPORT

John McKee

This is a case report about a 32 year-old diver, a Victorian who visits the south coast of New South Wales periodically for sports diving activities. He has had 7 years experience on hookah and on scuba equipment. Last year he was having another sporting dive off the far south coast of New South Wales.

On the day of his "accident" we have no idea of the profile of his first dive. We believe it was probably not more than 30 or 40 feet as he was diving on hookah equipment. We know the second dive profile and that was also using hookah, a dive to 20 feet for approximately 5 minutes when he was struggling with and trying to loosen an anchor