Past malaria epidemics in Sri Lanka – an analysis

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Abstract

In Sri Lanka malaria epidemics have been a common feature in the past, especially during the pre-insecticide era. These early periodical epidemics were due to adverse climatic conditions. Indoor residual spraying (IRS) with insecticides commenced in 1947 and was successful in causing a dramatic decline in the malaria incidence. An attempt at malaria eradication failed and was followed by resurgence eventually culminating in an island wide epidemic. The situation was controlled by recommencement of large scale IRS, however several epidemics, either generalized or regional, were witnessed during the subsequent period. The causes for these epidemics have been varied. Mechanisms to predict epidemics, early identification of epidemics, and to launch prompt remedial measures, are of primary importance.

Introduction

Malaria parasite is one of the most successful parasites known to mankind and has been responsible for some of the devastating epidemics in the past encountered by several countries in the world. Climatic factors such as changes in weather conditions and human behavioural factors including wars and mass migratory movements are among the common causes which were determined to be responsible for these events. During the last century malaria epidemics were abundant in Sri Lanka. The factors contributory in the causation of these epidemics were varied. “An epidemic is the results of the disturbance of a previously existing equilibrium of the ecological system comprising human, parasite and vector populations in a particular environmental niche. Depending on the resilience of the system, and whether or not the disturbance has changed some of its essential components, it will either return to its previous state of equilibrium after the end of the disturbance, or tend to find a new equilibrium, with or without going through a period of oscillation”¹. The form of an individual epidemic is primarily determined by the species of parasite, its inoculation rate and the proportion of susceptible in the human population.

Epidemiology of malaria in Sri Lanka

The disease has been prevalent in the country for several centuries. For purposes of malaria studies the country has been divided into the dry, intermediate, and wet zones, based on the total rainfall received during the south-west monsoon.

The disease was endemic in the entire dry zone and a greater part of the intermediate zone, with outbreaks or epidemics in the wet zone too, during excessively dry weather. The major factor which determined the distribution of malaria in Sri Lanka has been the extent to which conditions were favorable for the production of the principal vector of malaria, *Anopheles culicifacies* Giles. This essentially a dry zone species has been found to be extensively prevalent in the jungle covered plains and villages, with its prevalence decreasing with increasing altitude, and hardly found at altitude above 2500 feet. In the dry zone its prevalence increases during the rainy season and soon afterwards. The intermediate zone is invaded by the vector during times of drought when the rivers and streams undergo pooling creating preferred breeding places for this species². Although it is hardly prevalent in the wet zone records indicate that under conditions of a drought the vector densities can rise. In the past when the transmission levels were high the dry zone experienced malaria of a stable nature with little yearly fluctuations, whereas in the intermediate zone, malaria of an unstable nature was encountered, with the incidence rising during years of dry weather. Some important rivers starting from the central highlands and flowing through the intermediate and wet zones, especially,

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Deduru Oya, Maha Oya, Kelani Ganga and Kalu Ganga have played a major role in all the epidemics which were experienced during the pre-insecticide era, by the creation of numerous breeding sites for the vector during extremely dry weather, due to pooling. Accordingly the epidemics caused by deficient rains

were mostly confined to the intermediate and wet zones. The high altitude areas have been free of involvement in the epidemics because of the absence or scarcity of the vector.

Fig 1: Map of the climatic zones and some major rivers in the intermediate and the wet zone

After the commencement of large scale indoor residual spraying (IRS) in 1947, most parts of the country, even including dry zone areas, have experience unstable malaria. Consequently the intensity of transmission fluctuated markedly over the years, in space and time, long malaria-free periods resulting in loss of acquired immunity in the population. Latter condition has been responsible for the rapid spread of some past epidemics. Until the present low malaria status was reached two annual peaks were observed, the minor peak

corresponding to the south-west monsoonal rains and the major peak to the north-east monsoonal rains. Increased vector breeding in the ground pools of rain water was considered to be the causative factor. Over the last decade the endemicity of malaria in the country has declined to a very low level^{3,4}.

Epidemics in the pre-insecticide era

Several epidemics, either localized or generalized, have been recorded in the wet zone and the intermediate zone. In the wet zone basins of Gin Ganga, Kalu Ganga, Attanagalu Oya, Kelani Ganga, upper and middle reaches of Maha Oya, and Mahaweli Ganga were involved in these epidemics, in different combinations. In the Intermediate zone the basins of Deduru Oya, Mahaweli Ganga and Nilwala Ganga were involved, again in different combinations. The major epidemics occurred at intervals of four to six years, or at longer intervals. Failure of either the south-west or the north-east monsoons which resulted in reduced flow through the rivers in the intermediate and wet zones, resulting in pooling, was closely related to the genesis of the epidemics⁵. These periodic epidemic cycles which could reflect a similar periodicity of abnormal meteorological conditions such as droughts, heavy rains and flood, have been described as paraquinquennial cycle¹. Bouma and van der Kaay have described a relationship between the pre-insecticide era epidemics in Sri Lanka, and the El Niño Southern Oscillation, stating that the epidemics were significantly more prevalent during El Niño years, when the south-west monsoon tends to fail⁶. A parameter of the oscillation is the sea surface temperature anomalies in Eastern Equatorial Pacific. However, in a study done by Zubair, et.al, shows a relationship between El Niño and the epidemics in Sri Lanka from 1870 to 1927 has been confirmed, but suggests a breakdown between El Niño and the epidemics after 1928, likely to be due to an epochal change in the El Niño-rainfall relationship in Sri Lanka around the 1930's⁷. Introduction of insecticides as a causative factor for this breakdown of the relationship is unlikely since large scale IRS commenced only in 1947.

The 1934-35 epidemic

The above epidemic was the most devastating of all Malaria epidemics experienced by the country, and was described by Gill as the greatest pestilence in the recorded history of the country⁸. It is mentioned in all accounts describing severe malaria epidemics in the world the past has seen, and generated much interest among Malariologists world over.

Climatic conditions preceding the epidemic

A drought which started in April 1934, involving the country in general, worsened during the months of July, August and September. The rainfall during these three months in the area covered by the epidemic was lowest ever recorded for the July – September period, at nine out of the thirteen stations tabulated. There were heavy rains in October, and at the request of the Director of Medical and Sanitary Services, the Superintendent of the Anti Malaria Campaign prepared a tentative forecast of likely places which could experience an increased incidence of malaria and therefore action has to be taken whenever necessary. The respective Provincial Surgeons were accordingly informed. The predictions were accurate; however the magnitude of the epidemic that was to be encountered was never anticipated. The north-east monsoon of 1934-35 too was a failure and there were no inter-monsoonal rains⁹. To quote Brohier "... even Minneriya tank which in the memory of living man had never been known to be empty, ran almost completely dry"¹⁰.

The beginning of the epidemic

Alawwa and surrounding villages on the north bank of Maha Oya (Kurunegala district) were the first to record an unusual increase of malaria cases. By early October the epidemic had spread to the middle catchment area of the river, and by end of November to the entire river basin. An explosive outbreak occurred during the last week of October and the first week of November, covering an area of about 5800 sq.miles, with the involvement of basins of Maha Oya, Deduru Oya, Kelani Ganga and the upper reaches of the Mahaweli Ganga. It is reported that almost the entire population of the greater part of this area, numbering about 3.1 million, fell victims to malaria. This population had been already weakened by the famine which resulted from the failure of the paddy crop⁸. The peak of the epidemic as far as morbidity was concerned was in middle of December 1934, but was in mid January 1935 with regard to mortality. The epidemic started waning from April 1935 onwards; however during the months of May and June a minor increase occurred in both morbidity and mortality⁹.

Table 1: Rainfall during July to September 1934 (Epidemic Area)

Stations	No. of years of observation	July-Sept averagerainfall (inches)	1934 July-Sept rainfall(inches)	Percentage of average rainfall
Magalawewa	63	6.16	0.10	2
Horakele	66	8.34	3.00	36
Kurunegala Survey	48	12.98	1.99	15
Matale	62	14.23	3.70	26
Ambanpitiya (Kegalle)	63	23.55	5.04	21
Kandy	65	19.17	5.15	27
Henaratgoda	44	18.73	4.42	24
Avissawella P.W.D	64	33.65	7.74	23
Padupola	64	79.44	26.6	33
New Valley	64	38.55	15.79	41
Kalutara	64	19.69	8.60	44
Geekiyanakanda	61	34.58	14.35	41
Ratnapura	66	39.73	15.75	40

Source: Briercliffe, R. The Ceylon Malaria Epidemic 1934-35 (9)

The Maha Oya Basin

Out of the river basins affected Maha Oya basin was the most affected. At the peak of the epidemic the weekly attendance rose to over 76,000 per week (171 per 1000 of estimated population of the area) compared to a weekly attendance rate of 21 per 1000 during

Deduru Oya basin

The epidemic emerged in the last week of October, Mawatagama being the first area in which increased attendance at dispensary due to malaria was observed.

Kelani Ganga basin

Most severely affected was the upper catchment area of the river. A moderately severe outbreak occurred in Watawala area near the source of the river. Pindeniya, Karawanella, Avissawella nad Waga were badly affected areas.

Kalu Ganga

The upper basin was more severely affected than the lower. The worst affected centres were Kuruwita, Pelmadulla, Ratnapura and Horana.

Mahaweli Ganga basin

Worst affected places were Kandy, Katugastota, Eriyagama, Kadugannawa.

Severity district-wise

Kegalle and Kurunegala districts were the worst affected, followed by Chilaw (according to demarcation of the districts at the time) District. Other affected districts were Matale, Kandy, Nuwara Eliya (small portion) and Ratnapura, Kalutara, Colombo and Negombo (according to demarcation of the districts at the time).

Total Morbidity

The estimate was that probably 1.5 million persons within the epidemic area suffered from malaria⁹.

Mortality during the epidemic

At the time throughout the country registration of deaths was compulsory, however in rural areas which contributed to the great majority of deaths in the island, the registration was done by the Headmen of the districts, without being medically certified. Therefore the accuracy of the stated cause of death in the rural populations lacked complete accuracy. However it was considered that the excess mortality

2 The Epidemic Curve of the 1934/35 epidemic

in the epidemic area, over that during normal months, may be safely assumed to be due to malaria⁹.

Mortality outside the epidemic area

In most districts outside the epidemic area there had been much more sickness than usual during the epidemic period, with an excess mortality of 10451 over the expected number during the 6 months Nov. 34 to April. 35. Anuradhapura and Mannar districts recorded a mortality rate of over 80 per 1000, and the districts of Mulativu, Trincomalie and Puttalam too had a high mortality rate. It is reported that the districts of Anuradhapura and Mannar were sparsely populated at the time and the population was chronically ill with malaria. The economic depression of 1934, accompanied by the crop failure due to drought, had affected this population in a very adverse manner.

Age-wise distribution of mortality

Young children were most affected. An infant mortality rate of 355 per 1000 live births was recorded in the epidemic area, during the period Oct 1934 to March 1935. In the non-epidemic area the infant mortality rate for the same period had been 228 per 1000 live births. However Anuradhapura and Puttalam districts had recorded high rates of 613 and 512.3 per 1000 live births respectively, for this period. The death rate for children under 5 years

during the first quarter of 1935, when the mortality was at worst, was 278 per 1000⁹.

The parasite formula

Reports from 9 laboratories revealed the following figure for the period Nov 34 – Mar 35. The highest no of *P. falciparum* cases for the period was from Kandy district

Table 3: The Parasite Formula during the period Nov 34 – Mar 35

Species	No. positive	percentage
<i>P. vivax</i>	7679	62.2 %
<i>P. falciparum</i>	4524	36.7 %
<i>P. malariae</i>	131	1.1 %
	12334	100 %

Source: Briercliffe, R: The Ceylon Malaria Epidemic 1934-35⁹

The vector factor

At the time the only proven vector was *An. culicifacies*, its preferred breeding places being shallow, stagnant pools of clear sun-lit water. The vector is normally found in high densities only in the dry zone, however if there is drying up of rivers and streams in the intermediate and the wet zones the vector densities can rise significantly². The failure of the south-west monsoonal rains (April -September)

Table 2: The number of Excess Deaths recorded during the Epidemic

District	Population 1931 census	no. of expected deaths* Nov 1934-Apr 1935	no. of actual deaths Nov 1934-Apr 1935	excess no. of deaths
Colombo	875,488	8941	15063	6122
Negombo	205,761	1740	4301	2561
Kalutara	363,785	3344	4411	1067
Kandy	587,916	6018	16570	10552
Matale	129,697	1660	5972	4312
Kurunegala	397,239	5254	24737	19483
Chilaw	114,640	1030	2506	1476
Ratnapura	263,801	2744	5962	3218
Kegalle	314,567	2611	12965	10354
Total	3,252,894	33411	92556	59145

***Expected no. of deaths = average of 4 previous years**

Source: Briercliffe, R The Ceylon Malaria Epidemic 1934-35 (9)

The peak mortality occurred in January 1935 except in Kegalle district in which peak mortality

and north east monsoonal rains (November- March), with rains only in October 1934, resulted in the creation of numerous vector breeding sites in the wet and the intermediate zones. The larval surveys carried out during the epidemic revealed excess densities of vector larvae in the drying up river beds in the epidemic area (Table.4). Indoor resting vectors caught in the dwellings in the epidemic area revealed high infection rates.

During the period November 1934 to May 1935 a total of 5,063 Anophelines had been collected from the epidemic area, mainly from localities within the catchment areas of Maha Oya, Deduru Oya, Kelani ganga and Mahaweli Ganga. 88.5 % of the Anophelines collected had been *An. culicifacies*, 8.7 % *An. subpictus*, the remaining species being *An. varuna*, *An. jamesi*, *An. hyrcanus*, *An. tessellatus* and *An. maculatus*. The highest infection rate was observed in Dec 1934, with an infection rate ranging from 10.8 % (Deduru Oya basin) to 14.0 % (Maha Oya basin).

The causes for the epidemic

Briercliffe, the Director of Medical and Sanitary Services at the time, in his report on the epidemic⁹, stresses the importance of the connection of the prolonged drought which prevailed. Col. Gill, a

Malariologist who was commissioned as an expert adviser to report on the epidemic, too was of the view that the prolonged drought resulting in increased vector breeding was a plausible explanation to the causation, however expressed the opinion that an unknown factor (X factor) probably related to the parasite, too played a role in the causation of periodic epidemics in the country⁸.

Macdonald however stated that abrupt changes in transmission factors alone could explain the epidemic curves, and it was not necessary to consider any other causative factor¹¹. He found a similarity between the synthetic curve he constructed using a mathematical model and utilizing certain entomological parameters, and the actual epidemic curve of the Deduru Oya basin drawn. He concluded that episodes in the 1934/35 epidemic were possible if the anopheline density multiplied by 5.3 times in a background of very low transmission if the conditions were conducive to moderate longevity of the vector.

Although Gill thought that the epidemic had an explosive beginning Wijerama¹², a physician serving in General Hospital, Colombo, throughout the epidemic period, analysing the clinical picture and the blood smears of 729 patients of malaria admitted

Table 4: Prevalence of An. culicifacies in river basins (Dec 34-Feb 35)

River basin	No. of actual Breeding Places of Anopheline examined	No. of Anopheline larvae identified	Breeding Places of An. Culicifacies (%)	An. culicifacies larve as % of Total Anophelines larvae
i. Deduru oya				
Main rivers	48	6183	87.5	41.8
Associated streams	24	702	37.4	19.1
Sand pools	26	4825	92.3	78.2
Rock pools	17	891	100.0	66.2
Total	115	12601	79.8	56.1
ii. Maha Oya				
Main rivers	134	7152	65.5	24.3
Associated streams	48	1191	31.3	17.3
Sand pools	51	1787	90.2	67.6
Rock pools	48	1110	56.4	50.3
Total	281	11240	63.1	33.0
iii. Kelani Ganga				
Main rivers	19	1592	63.2	26.6
Associated streams	22	473	4.5	0.4
Sand pools	68	1526	56.0	41.5
Rock pools	11	18	9.1	5.6
Total	120	3609	43.3	29.4
iv. Mahaweli ganga				
Main rivers	27	996	35.8	9.8
Associated streams	88	2931	47.7	7.6
Sand pools	155	6154	85.3	38.2
Rock pools	79	2681	83.5	54.5
Total	349	12762	71.6	32.4

Source: Briercliffe, R The Ceylon Malaria Epidemic 1934-35 ⁹

to GHC between September and December 1934, was of the view that the onset was heralded by a series of primary attacks and was gradual. Dr. Wijerama based his view on the clinical manifestations, characteristics of the fever charts, low spleen index, low gametocyte index, and the presence of heavy asexual parasitaemia in the blood smears. Gill's view was that the sudden onset of the epidemic was partly due to relapses, based on the argument that had it been mainly due to new infections, there should have been a sharp rise in the mortality among children under 10 years of age, within a week of its onset at

end of October 1934. On the contrary the mortality among this age group had been scarcely raised until the first week in December 1934.

Population movements too have been incriminated as a factor in the spread of this epidemic. Due to the prevailing economic depression and scarcity of employment ill nourishment among peasants was common, and since 1929 hundreds of people from the wet zone, including especially Kegalla and Kurunegala districts and many other coconut and rubber areas, have migrated annually to the malarious dry zone. However within short periods

they returned due to sickness, and they would have been an important source of introduction of the parasite into the wet zone¹³.

1967/69 epidemic

This epidemic too drew much attention among Malariologists, as an example of resurgence of malaria following a point of near elimination status within a formerly malarious country. It spread throughout the island in areas which used to experience endemic malaria.

Events preceding the epidemic

The attempted malaria eradication (The term eradication now refers to the permanent reduction to zero of the worldwide incidence of infection caused by human malaria parasites as a result of deliberate efforts) campaign commenced in December 1958, with the attack phase, targeting complete interruption of malaria transmission in the country by the end of a 5 year period¹⁴. Initial results were quite promising there being only 17 malaria cases detected in 1963, out of which only 6 cases found to be of indigenous origin. IRS was discontinued in April 1964, however within the last quarter of the year active foci of *P. falciparum* and *P. malariae* appeared. During the subsequent 3 years more foci started appearing in several of formerly malarious districts, with the situation taking a turn to the worse within the period July to September 1967 during which 800 positive cases were detected. By now *P. vivax* became the dominant parasite, a species hardly detected during the preceding 3 years. Authorities were of the view that this reflected a hidden reservoir of infection in the population and the existence of a long lasting strain of *P. vivax* in the country. *P. vivax* outbreaks were detected mainly in new development projects and chena settlements (plots of slash-and-burn cultivations) in Kurunegala, Matale, Anuradhapura and Polonnaruwa districts, and were considered as the beginning of the resurgence which led to the epidemic which erupted in February 1968. The invasion of certain centres of population aggregation, viz, gemming centres in Elahera, development projects in Rajangana and Wilgamuwa, and the pilgrim centre at Kataragama, by the infection, was considered the triggering factor of the epidemic¹⁵⁻¹⁸. The major centre of the epidemic was identified as adjacent portions of Kurunegala,

Matale, Polonnaruwa, and Anuradhapura districts. Kataragama area was identified as a minor centre¹⁷. Population movements played a key role in the dissemination of infection from these two centres. At the time Elahera was a very popular gem-mining centre drawing people from many parts of the country, and there were much to and fro movements of people between Elahera and their places of origin. An extensive area on the banks of Kalu Ganga was studded with gem pits, and the dry weather prevailing at the time was quite conducive to malaria vector breeding. The role played by gem-pits in the breeding malaria vectors has been established¹⁹. Kataragama is a highly venerated religious centre visited around the year by large numbers of people from throughout the country, and once malaria infection is introduced, transmission within the area and dissemination throughout the country would have been inevitable. From these two main centres the infection spread to many other centres of population aggregation, with the eventual spread of the epidemic to the entire traditionally malarious parts of the country.

Extent and the course of the epidemic

The epidemic enveloped nearly three fifths of the country which had a population of over 5 million. It was estimated that over 1.5 million people contracted the disease during the epidemic period. The entire dry zone and a large part of the intermediate zone were involved, while localized outbreaks appeared in the wet zone. The epidemic displayed peaks in February 1968, June/July 1968, Dec 1968/Jan 1969, June/July 1969, and a final peak in Dec 1969/Jan 1970 which surpassed all four previous peaks. A downward trend then followed. The traditional peak did not occur in Dec 1970/Jan 1971 (fig.3).

During the period October 1967 and September 1970, the number of microscopically positive cases detected by the anti malaria campaign and the general health services of the country, was 1,407,957. It was estimated that the actual number of malaria cases would have been much more if the patients treated on clinical grounds without parasitological confirmation were to be added.

The parasite factor

The epidemic was unique in that 99.9 % of the infections were *P. vivax*. It is highly probable that the

Figure 3: Epidemic wave of the 1967/69 epidemic

peaks which followed the first peak were due to relapses of *P.vivax* infections, especially considering the fact that for practical reasons the regimen of primaquine given for the elimination of hypnozoites of *P. vivax*, had to be changed from the recommended 14 day-course to a 5-day course, from January 1968. *P. vivax* from tropical regions are characterized by multiple relapses with short latent periods (3-5 weeks), compared to *P. vivax* strains from temperate regions which usually have long incubation and latent periods (5-10 months)²⁰. The present drug regimen recommended by the anti malaria campaign for vivax malaria infections includes primaquine for 14 days provided there are no contraindications including pregnancy, infancy, and G-6-PD deficiency²¹. Detection of a vivax focus, after an absence of any detected cases of *P.vivax* for a period of over 2 years was most significant, and led to some speculation whether the focus was of simian origin. However, the investigations conducted by the Parasitology Unit of the Faculty of Medicine, University of Ceylon, revealed that it was not so²². The Anti Malaria Campaign authorities were of the view that this phenomenon suggested the existence of a hidden parasite reservoir in the country population, and also the presence in the country, of a *P.vivax* strain with a long life span¹⁶.

Vector factor

Insecticide susceptibility tests carried out prior to 1969 revealed susceptibility of *An. culicifacies* to DDT, the insecticide which was in use for IRS^{16,17}. However in 1969, tests conducted in 4 localities under IRS, revealed that the vector had developed resistance towards DDT, or was in an intermediate stage of developing resistance. Development of DDT resistance in *An. culicifacies* was confirmed during 1969¹⁸.

High Basic Reproduction Rate

Sivagnanasundaram, who calculated the Basic Reproduction Rates for the Grama Niladhari areas in the Hiripitiya Vigilance Unit covering 311 villages (1967 population 67,096) in Kurunegala district, for the epidemic period, found the rates to be high in the region of 50 to 200²³. Basic reproduction rate in malaria denotes the number of secondary cases one malaria case generates on average over the course of his/her infectious period, in an entirely susceptible population.

Mortality during the epidemic

During the epidemic only 58 deaths reported were considered as probable deaths due to malaria^{17,18}. This extremely low number of deaths was quite remarkable compared to the number of malaria patients recorded during the epidemic, the likely reasons being the fact that more than 99 % of the patients had vivax malaria, and treatment was readily

available for the patients both from the medical institutions and from field workers of the anti malaria campaign.

The problem of acute intravenous haemolysis following primaquine in G-6-PD deficient patients

The association between administration of primaquine and the occurrence of IVH in G-6-PD individuals was revealed for the first time in Sri Lanka by Abeyaratne during the epidemic, in early 1968, who observed that several children who were treated with antimalaria drugs at the General Hospital, Anuradhapura, passed blood in the urine²⁴. At the time primaquine was extensively used, Anuradhapura district having the highest district-wise malaria incidence at the time. During a subsequent survey by Abeyaratne the overall prevalence of G-6-PD deficiency in the district was found to be 6.35 %²⁵. During a survey carried out by the anti malaria campaign in the eighties, overall prevalence in the country excluding the eight districts in the north and eastern provinces, was found to be less than 3 %²⁶. In the north-central province survey, Abeyaratne found a much higher prevalence among the inhabitants of old villages (up to 20.9%), results clearly showing that the incidence of the enzyme deficiency was much higher in the populations in ancient villages, as compared to that among the populations living in recently colonized areas, supporting the theory that the distribution of the deficient populations has a relation to past exposure to falciparum malaria. An association between the incidence of G-6-PD deficiency in populations, and their exposure to falciparum malaria in the past has been established, with the postulation that this deficiency in the red cells confers some degree of immunity against falciparum malaria²⁷.

Spatial distribution

A remarkable feature was that unlike in most of the previous epidemics (including the massive 1934/35 epidemic), the epidemic engulfed the entire traditionally malarious parts of the country. The most plausible explanation is that the populations living in the traditional malarious areas have had an unusually long period of malaria-free years, following the successful malaria control which had been achieved after the introduction of DDT

spraying. In fact most of the children, certainly those under 5 years of age, probably would not have been exposed to malaria at all, in contrast to the past when malaria epidemics were recurring every 3 to 5 years or so. Therefore it is very reasonable to conclude that the lack of any acquired immunity against malaria in the population at the time, was a main contributory factor for the rapid and extensive dissemination of the infection during the 1967/69 epidemic. Previous epidemics had been generally confined to the intermediate and wet zones of the country, which were either areas with unstable type malaria or traditionally non-malarious areas.

Causes for the epidemic

This epidemic has been cited as a classic example of resurgence of malaria resulting from progressive return of endemicity in highly endemic areas due to failure to sustain the success of control measures which reduced or interrupted malaria transmission¹. In such instances the incidence will gradually increase, often from dispersed foci, like what happened during this epidemic. Development of vector resistance towards the insecticide used for IRS, deteriorating quality of IRS, or complete interruption of IRS may cause such resurgence. Bruce-Chwatt, commenting on the set back faced by the Eradication programme of Sri Lanka, stated as causes, the premature cessation of total coverage with spraying, pooling of epidemiological data in such a way that persistent smaller foci were not spotted in time, and the much higher vectorial capacity of the Sri Lankan strain of *An. Culicifacies*²⁸. The vectorial capacity is the daily rate at which future inoculations arise from a currently infective case, provided that all females mosquitoes biting that case become infected (Garret-Jones). Black describes that in the consolidation phase of a malaria eradication programme, the vector density will definitely reach a critical level during the second and third years of the phase due to the lack of insecticide coverage²⁹. Gabaldon was of the view that in highly endemic zones in the tropics, or in areas with a great transmission potential and continuously exposed to imported malaria cases, IRS should not cease with the commencement of the consolidation phase in an eradication programme³⁰. He recommends maintenance of spraying during the 3 – year period after the last indigenous case, and is of the opinion

that active case during this period is used only as an evaluation activity and not for the elimination of residual cases. The WHO Team who came to assist the national malaria authorities, at the invitation of the government, identified in their report, four clear causes which they concluded played a dominant role, viz., persistence of pre-epidemic foci of infection, abnormal climatic conditions, population instability, and a population susceptible to malaria³¹.

Pre-epidemic foci of infection

Judging from the sequence of events connected to the appearance of vivax foci during the early consolidation phase, it might be quite reasonable to conclude that even after the cessation of attack phase, low-grade active transmission had persisted in areas which had high malariogenic potential in the past. Following the focal outbreaks in the chena settlements in Kurunegala and Matale Health Divisions, in early 1967, anti malaria campaign investigations revealed that there were several persons coming from the wet and intermediate zones, into these chena settlements. The possibility of these migrants introducing malaria to the chena settlements was considered because of the chances that low-grade malaria transmission could have been easily missed in the wet and the intermediate zones, as there was no effective case detection machinery to cover these climatic zones¹⁷. Deficiencies in the surveillance mechanism would have caused the persistence of these foci, the consequences of which manifest themselves when residual insecticide cover was withdrawn.

Abnormal climatic conditions

On comparison of the total rainfall offsets for the country during the period of September to June, of 1934/35 and 1967/68, based on data from 17 representative stations in the country, it was concluded that the rainfall pattern in the intermediate and the dry zone for 1934/35 and 1967/68 corresponded very closely. The dry period in 1967/68 was mid December 67 to end of February 1968, as compared to the dry period in 1934/35, which was from mid October to 1934 to end of January 1935. They found the rainfall pattern in all districts which had a high intensity of malaria during the epidemic was similar, especially between December 1967 and May 1968, rainfall being abnormally low in February 1968.

Commenting on the relationship between rainfall and malaria, Gill stated that in Ceylon, epidemics in the wet zone are the result of drought, whereas in the dry zone excessive rain is the determining factor for epidemics⁸. However the WHO team was of the view that the relationship is more complex, and that “in the river basins there will be a range of rainfall between a level just sufficient to cause formation of pools in the dry bed and an upper level which just fails to maintain flow over the whole channel. Within these levels *An. culicifacies* production can be expected. This range will differ according to peculiarities of the upper catchment areas and lower reaches of river valleys”. Why then during the 1934/35 epidemic, extent of the involvement was limited to the wet and the intermediate zones, if the rainfall pattern was similar? It is explained by the fact that herd immunity against malaria, resulting from continuous exposure to malaria in the pre-DDT era, protected the dry zone population during the 34/35 epidemic.

Population instability

“Diffusibility” has been defined by Gabaldon as the number of localities infected from a single focus, which are the functions of vector and its ecological conditions and of man and his social habits respectively, parasite playing the same role in both cases. The WHO team was of the opinion that the population movements that took place in the country, had resulted in enhanced diffusibility, and was an important factor in the spread of the epidemic. Both long-range movements and short-range movements were evident in the sixties. Discovery of gems at Elahera, and the Food Production Drive which had been launched, contributed to increased long-range movements which also included visits of pilgrims to the traditional religious centres, most of which are in the dry zone. Short-range movements comprised travel associated with youth camps and land development schemes.

A population susceptible to malaria

Following the introduction of DDT spraying programme in 1946 the malaria incidence in the formerly malarious areas was a drastically reduced, the decline being further reinforced by the launching of the eradication programme in late 1958. During the period 1958 to 1966 the annual parasite incidence was less than 0.01 per 1000. The adult population living in formerly malarious areas would have more

or less lost whatever acquired immunity they had developed. Perhaps the children living in these areas had never encountered a single episode of malaria. Therefore the increasing malaria transmission at the commencement of the epidemic was faced by a population highly susceptible to malaria.

Epidemic in 1974-75

During the period 1974-75 the malaria incidence increased in several districts, along with an alarming increase in the *P. falciparum* incidence in many of the affected districts. No drug resistant *P. falciparum* strains were prevalent during the period, however DDT resistance in *An. culicifacies* which was first detected in a limited manner in 1969 had by now spreading widely throughout the country. High *falciparum* malaria transmission prompted the anti malaria campaign to replace DDT with malathion for IRS, initially in the affected areas. High levels of widespread vector resistance against DDT, eventually resulted in DDT being replaced with malathion in the entire IRS programme, in 1977.

Epidemic in 1982-84

During the period 1982 to 1984 a countrywide epidemic occurred recording more than 275,000 cases. At first it was limited to the dry zone but in 1983 the intermediate zone too was involved. A suggestion of emerging malathion resistance in *An. culicifacies* was detected in 1982³², however it was not of operational significance, but bio-assay tests in some areas revealed poor quality IRS. The limited number of drug resistance studies proved that *P. falciparum* was sensitive to chloroquine³³. The cause of the epidemic was tracked down to operational failures of the IRS programme as a result of several factors³⁴. The climatic factors too could have been contributory, the rain fall over the whole country being deficient between the period January to October 1983 except during September.

Epidemic in 1986-87

In 1986 there was a sharp rise in the countrywide malaria incidence, associated with an alarming increase in the percentage of *falciparum* malaria. 687,599 microscopically positive cases were detected 1987. The *falciparum* percentage which was only 2.5 % of the total cases in 1984, reached 26.8 % in 1987³⁵. Again, it was the traditionally

hyperendemic areas in the dry zone which were affected. The decentralization of microscopists from the Regional Laboratories of the anti malaria campaign to medical institutions carried out in 1986 resulted in more parasitologically diagnosed cases being recorded³⁶. Chloroquine-resistance in local *falciparum* strains, first detected in 1984³⁷, was gradually extending to all malaria transmission areas in the country. However, the degree and the extent of spread of the resistance at the time, did not warrant the change of the first-line drug regimen administered to *P. falciparum* patients, but a second-line drug regimen was recommended to patients detected as having chloroquine-resistance. The recrudescence of infection in chloroquine-resistant *P. falciparum* patients resulted in increased *P. falciparum* percentage. The susceptibility status of *An. culicifacies* against malathion remained high. Ecological changes caused by the irrigation development in the Mahaweli Development areas resulted in the increase of vector densities and an increased role by the secondary vectors of malaria in Sri Lanka, *An. subpictus* and *An. Annularis*^{38,39}. The large scale migration of non-immune persons to malaria-endemic areas in the dry zone, under the scheme too was responsible for increased transmission. Wijesundere reported that the parasite rates among those living in the new colonization areas of system 'B' of the Mahaweli Scheme, were found to be much higher than in those living in the areas such as Polonnaruwa town and Hingurakgoda which were colonized several decades ago⁴⁰. She attributed the high infection rates among these new settlers who came from traditionally non-malarious areas (and hence non-immune), as the most significant cause of the epidemic situation in Polonnaruwa which prevailed at the time. The poor security situation in many parts of the country was responsible for frequent interruptions in the IRS programme, which was also found to be of poor quality. A high refusal rate by householders towards spraying of the houses, especially spraying in a complete manner, was a regular feature and led to the demoralization of the spraying teams.

Epidemic in the Northern and the Eastern province (1991 – 2001)

From 1991 onwards the malaria incidence in the districts of the above two provinces started rising,

reaching a climax in 1998/99 (Fig.4). This situation resulted from an escalation of the conflict situation which had prevailed in the two provinces for over two decades. Detection and treatment of malaria patients suffered due to the inability of the medical institutions to function properly. Patients too found it difficult to access the functioning medical institutions because of poor public transport facilities and the poor security situation. Malaria control operations, essentially field-based, were quite frequently interrupted, sometimes for long periods. Dispatch of essential items such as antimalarial drugs and insecticides for IRS, was severely disrupted. The temporary cessation of the conflict situation which took place in early 2002 offered the anti malaria campaign to resume malaria control activities with a resulting decline in the malaria incidence in the two provinces.

Discussion

Analysis of the causation of the past malaria epidemics reveal a multitude of factors which have been responsible at different times. In the pre-insecticide era natural factors, especially climatic, played the major role in the genesis of the epidemics, however the spread within the country was limited by the herd immunity against malaria the population in highly endemic areas had. This was quite evident during the virulent epidemic of 1934/35 which was limited mostly to the intermediate zone and the wet zone of the country. During the post-insecticide era a major epidemics was due to resurgence of malaria resulting from failure at sustaining the achievements gained, several factors being causative in the genesis and the spread of the epidemic. During the subsequent epidemics both vector resistance to insecticides and drug resistance in *P. falciparum* have played a part.

Fig.4 The prevalence of malaria and the deaths attributed to malaria in the Northern & Eastern provinces 1990 - 2002

Operational failures, by themselves, have resulted in epidemics, whereas interruptions in parasite control and vector control activities, consequent to poor security situation posed by the conflict situation have caused an epidemic in the affected regions.

An epidemic may not be preventable, especially if due to adverse climatic conditions or due to a long drawn out conflict situation. However much advances have been made in the field of prediction of epidemics caused by climatic changes. Prediction of an epidemic, epidemic preparedness and the ability for early responses to an epidemic are essential activities. The Intergovernmental Panel on Climate Change (IPCC) has projected an approximate average global warming of 2-5 °C within the twenty-first century which would be accompanied by an increase in the frequency of extreme and anomalous weather events such as heat-waves, floods and droughts. It has been predicted that these projected changes may have significant impacts on the timing and severity of infectious disease outbreaks⁴¹. It could be expected that some of these projected changes may have a significant impact on the malariogenic potential within a geographical area. Advanced Epidemic Warning Systems in malaria have been developed based on analysis of past morbidity data and climate and weather pattern. New technology in the prediction of climate and weather pattern, Geographical Information Systems (GIS), and Remote Sensing, have been widely used in the long-range prediction of vector-borne diseases. Early identification of an epidemic, and epidemic preparedness are of utmost value in the reduction of morbidity and mortality that could ensue. Considering the technical factors that may contribute to an epidemic, continuous monitoring of the susceptibility of patients to the antimalarial drugs used in a programme and the vector susceptibility to insecticides in use or kept ready for use if warranted, are of primary importance.

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