

PREVALENCE OF MALARIA EPIDEMIC IN AFRICA

Prof. J.Vijayalakshmi*

MUVANDIMWE Emmanuel**

ABSTRACT

Malaria is both preventable and curable. Malaria epidemic display the full explosive power of vector-borne infections erupting with suddenness and intensity that can overwhelm vulnerable communities. The disease is life threatening and needs diagnosis and treatment which can be difficult in remote areas that lack clinics, medicine and trained health care providers. The objectives of this study are to examine the extent of prevalence of malaria in Africa and to identify the causes of malaria.

Key word: *Malaria epidemic, transmission, Immunity, vulnerability.*

* Professor in Population Studies, Annamalai University

** PhD. Scholar in Population Studies, Annamalai University

Introduction

Malaria epidemics display the full explosive power of vector-borne infections, erupting with a suddenness and intensity that can overwhelm vulnerable communities. The very instability of malaria epidemics exacerbates their clinical threat. Long, inter-epidemic periods of low transmission allow immunity to wane in populations. Thus, in areas where malaria is transmitted less stably, the risk for severe disease in children is greater, and older children and adults are more likely to have cerebral manifestations that are often fatal. This exacerbated risk to older children and adults is characteristic of malaria infections in unstable transmission areas and may produce different socioeconomic consequences than stable transmission.

Malaria is both preventable and treatable. Yet more than 220 million cases of malaria are estimated to occur each year, and approximately 785,000 people die from the disease annually. Half of the world's population some 3.3 billion people living in 109 countries are at risk of malaria (Roll Back Malaria 2008)¹. Worldwide, malaria is the fifth-leading cause of death from infectious diseases (after respiratory infections, HIV/AIDS, diarrheal diseases, and tuberculosis). The disease is life threatening and needs early, accurate diagnosis and treatment, which can be difficult in remote areas that lack clinics, trained health care providers, technical assistance, or medicine.

Malaria has been a perennial cause of human suffering and mortality for millennia. The Greeks were the first to write about it, and Egyptians hieroglyphs also made a mention of it. It exists throughout much of "...tropical and sub-tropical regions of Africa, Asia, and South and Central America."² A World Health Organization report routinely puts the number of yearly infections at about 300 million and malaria mortality at 2 million a year. Over 400 species of the malarial parasites (*Plasmodium spp.*) are said to exist. Many infect a wide variety of cold-and-warm-blooded animals - only four routinely infect humans. Malaria is transmitted from one person to another by the bite of an infected female *Anopheles spp.* mosquito. It follows then, that ecological alterations favoring the spread of these insects also facilitate the spread of the infection wherever malaria occurs.

Changes in the environment of the mosquito habitat, such as those taking place in Ethiopia, whether natural or man-made, "... rearranges the ecological landscape in which these vectors breed". Every *Anopheles spp.* occupies a specific "...ecological niche that is genetically determined". Changes in temperature, humidity, altitude, population density of humans, and deforestation are just a few ecological factors that play essential roles in the transmission of malaria.²

Temperature and humidity have a direct effect on the longevity of the mosquito. Each species can thrive at an optimal level as a result of ecological adaptation. The spread of malaria requires that conditions are favorable for the survival of both the mosquito and the parasite. Temperatures from approximately 21°-32°C and a relative humidity of at least 60% are most conducive for maintenance of transmission.²

The potential for dams to alleviate poverty significantly contributes to the enhancement of human health, and simultaneously increases the likelihood of human infection due to schistosomiasis, malaria, dysentery, and river blindness. During the construction of dams and canals, excavation pits provide temporary breeding sites for mosquitos."² A study in Tigray investigating the possible impacts of small dams on malaria transmission found an unmistakable link. The rate of infection among children near dams was seven times greater than in communities with no dams. The study, thus, concluded that " microdams close to villages have the potential to increase the incidence of malaria substantially among children living nearby".³

Objectives: The objectives of the present study are to examine the extent of prevalence of malaria in Africa and to identify the causes of malaria.

Observation: In the present study relevant discussions have been summarized on the basis of objectives under following heads.

Prevalence of malaria epidemic

Malaria epidemics tend to occur where endemicity is relatively low, reflecting the infrequency of transmission. The prevalence of enlarged spleens in chronically infected 2–9-year-old children provides a classic indicator of endemicity, with epidemics more likely to occur in mesoendemic (spleen rate 11–50%) or hypoendemic areas (10%). Using these same percentages, an alternative classification of endemicity uses the proportion of blood smears found to contain malaria parasites. While epidemics may occur in areas of greater endemicity, a low prevalence of chronic infection provides a useful indication of the relative intensity and stability of transmission and of the vulnerability of a population to malaria epidemics.

The low immunity of people residing in malaria epidemic prone areas exacerbates their risk of experiencing acute disease. Immunity to severe malaria generally requires only a few infections at any level of endemicity.⁴ However, the long interval between infections and the spatial variability of transmission in areas of unstable endemicity fail to provide frequent enough challenge to sustain much disease-modulating immunity. As a result, serious clinical consequences become common during outbreaks. Where malaria is transmitted stably, the probability of dying from an untreated case of malaria is approximately 2–3%.⁵ Where unstable transmission fails to sustain immunity in the population, case fatality rates up to 10 times greater can occur during epidemics.^{14–16} High case fatality rates characterize epidemic malaria.

The nature of morbidity can also be affected by the stability of transmission. As transmission intensity decreases, the cumulative risk for experiencing a severe disease episode during childhood increases.¹ Severe malaria becomes less likely as children grow older, but when severe malaria does occur, 8–15-year-old children (60.6%) are more likely to develop life-threatening cerebral manifestations than those who are 4–7 years old (28.2%) or younger (11.3%). Thus, cerebral malaria is more likely to develop from malaria infections in epidemic-prone regions, which may in part account for the high case fatality rates noted during epidemics.

The stability of transmission also affects the clinical expression of malaria in pregnancy. Premature deliveries were more common in epidemic-prone regions, while intrauterine birth retardation predominated where transmission was stable.⁶ As occurs in endemic

regions, pregnancy increases the likelihood of severe malaria. Severe infections were three times more likely in pregnant women.⁷ During the 1998 epidemic in Rwanda, pregnant women were 2–5 times more likely to be admitted to the hospital for malaria than other adults. Maternal and fetal mortality were also greater during the first pregnancy in areas where malaria transmission was less stable. Similar clinical manifestations as well as low birth weights can also be seen in multigravidae.⁸

While infants and young children are most likely to have severe illness and die in areas of high endemicity, this vulnerability extends to older children and adults in epidemic-prone areas. Where transmission is relatively stable and intense, such as coastal Kenya, the mean and median ages of patients diagnosed with malaria at clinics is generally less than five years old. Where transmission is most intense, the average child presenting at clinics with malaria is less than two years old.⁹

During the 1991 and 1998 malaria epidemics in Ethiopia, however, patients diagnosed with clinical malaria averaged greater than 10 years old (Ministry of Health, Ethiopia, unpublished data). Where transmission is unstable, incidence rates arranged by age tend to follow the same age structure as the general population. In Ethiopia, for example, 67.2% of the population is at least 10 years old.¹⁰ During the 1998–1999 malaria epidemic in the southern Rift Valley of Ethiopia, approximately 55% of the malaria cases reported by clinics occurred in people \geq 10 years old (Ministry of Health, Ethiopia, unpublished data). During an earlier epidemic that followed the extended 1987–1988 drought, malaria cases were even more prevalent in the older demographic, with people 10 years old accounting for approximately 76% of clinically reported malaria cases (Ministry of Health, Ethiopia, unpublished data). Unstable malaria transmission extends vulnerability to clinical disease into older age groups.

Malaria is a life-threatening caused by Plasmodium parasite infection. Malaria is the most deadly, and it predominates in Africa¹¹. In 2010, there were 219 million malaria cases leading to approximately 660,000 malaria deaths, mostly among African children.

- .An estimated 90 percent of all malaria deaths occur in Africa of which the majority are children under five years (91%) of age.

- About 80 percent of malaria cases occur in 17 countries.
- 80 percent of malaria deaths occur in 14 African countries.
- Together, the Democratic Republic of the Congo and Nigeria account for over 40 percent of the estimated total of malaria deaths globally.

Causes of malaria

“Classic” epidemics occur in areas where environmental conditions are marginal for mosquito vector and malaria parasite development, such as highlands or semi-arid regions. In such areas, conditions more permissive for malaria transmission appear during climatic anomalies. Epidemics can also occur when people who have had little exposure to infection move into endemic regions as refugees, in resettlement programs or to take advantage of economic opportunities.¹² Outbreaks can also be caused by any changes that enhance the development or survival of vector mosquitoes including changes in the landscape, breakdowns in intervention programs, or insecticide resistance. Instability induced by abrupt change is a common element of the determinants of malaria epidemics.

Malaria epidemics have often been linked with famines, the precise relationship between malnutrition and vulnerability to malaria infection remains somewhat hazy. In the Sudan,¹³ children with a poorer nutritional status based on age and weight were more likely to experience clinical malaria. However, other studies suggest that nutritional stress is protective against malaria infection.¹⁴ Still others have failed to detect an association between nutritional status and malaria susceptibility. Various associations between malarial susceptibility and micronutrients, such as zinc, have also been reported, but with similarly conflicting results. In Swaziland, meteorologic conditions expected to precipitate epidemics (e.g., heavy rainfall), generally have not done so unless the human population was also stressed nutritionally. This pattern is seen elsewhere in the world (e.g., Ethiopia, India) and may explain in part why malaria epidemics commonly follow in the aftermaths of droughts severe enough to destroy agricultural production and trigger famines.

Conclusion

Malaria epidemics impose substantial burdens on the populations. Although the average number of people infected in epidemic-prone regions may be relatively few compared with where transmission is stable, the impact per case can be far greater due to higher rates of severe disease and mortality. Further, more productive members of the community bear the full brunt of malaria epidemics, amplifying their effects on families and society. The disruptive nature of large-scale outbreaks even interferes with the ability to record them. As clinical cases surge, health care workers may be forced to choose between patient care and timely record-keeping.

Thus, as malaria epidemics peak, cases may be vastly under reported. Malaria outbreaks are epidemic in character when case rates surpass normal expectations such that they catch communities unprepared, disrupting the continuity of community life and depleting the resources and exhausting the personnel of health systems attempting to provide treatment. Thus, the ability to cope with changes in malaria transmission ultimately determines whether a community perceives a shift in malaria transmission as an epidemic. Building the capacity to cope with such events should be a priority of any anti-malaria intervention program.

Suggestion

Governments should prioritize the maintenance of strong malaria control despite the competing health priorities that they face. By ensuring the viability of their malaria control programs, these countries will be able to sustain high coverage of malaria control tools, continue to avert malaria cases and deaths, and generate far-reaching economic benefits into the future.

Antivector interventions before and during epidemics should focus on indoor residual spraying because this method can most rapidly reduce the clear and present danger of infectious mosquitoes,¹⁵ as well as reducing the longevity of those that might otherwise become infectious.¹⁶ To maximize the effectiveness of limited resources, villages and neighborhoods should be prioritized for spraying according to current information on where most cases are occurring or based on previous experience in epidemic-prone areas.

References

1. Roll Back Malaria Partnership, 2008, *The Global Malaria Action Plan—for a Malaria-Free World*.
2. http://www.medicalecology.org/diseases/malaria/print_malaria.htm#sect
3. <http://bmj.bmjournals.com/cgi/content/full/319/7211/663>
4. Gupta S, Snow RW, Donnelly CA, Marsh K, Newbold C, 1999. Immunity to non-cerebral severe malaria is acquired after one or two infections. *Nat Med*, 5, 340–343.
5. Alles .H.K, Mendis .K.N and Carter .R, 1998, Malaria mortality rates in south Asia and in Africa: implications for malaria control. *Parasitol Today* ,14, 369–375.
6. Sullivan .A.D, Nyirenda .T, Cullinan T, Taylor T, Harlow S.D, James S.A, Meshnick SR, 1999, Malaria infection in pregnancy: intrauterine growth retardation and preterm delivery in Malawi. *J Infect Dis* ,179,1580–1583.
7. Luxemburger .C,Ricci .F, Nosten .F,.Raimond .D,Sather S, and White N.J,1997, The epidemiology of severe malaria in an area of low transimission in Thailand .*Trans R Soc Trop Med Hyg* ,10, 795-803.
8. Nosten D, der Kuile .F, Maelenkirri L.M, Becludt .B. and White NJ, 1991, Malaria during pregnancy in an area of unstable endemicity. *Trans R Soc Trop Med Hyg* ,85: 424–429.
9. Snow .R.W, Omumbo .J.A, Lowe B, Molyneux .C.S., Obiero .J.O., Palmer A, Weber .M.W., Pinder .M, Nahlen .B., Obonyo .C., Newbold .C., Gupta .S., Marsh .K., 1997, Relation between severe malaria morbidity in children and level of *Plasmodium falciparum* transmission in Africa. *Lancet*,349: 1650–1654.
10. Ethiopian Central Statistical Authority,Office of Population &Housing Census Commission,1999.,1994, Population and Housing Census of Ethiopian, Results at Country Level. VolumeII.(Analytical Report).Addis Abeba: Central statistical Authority.
11. Adhanom .T.D.W, Witten .H.K, Getachew .A, Seboxa .T: Malaria. In The Epidemiology and Ecology of Health and Disease in Ethiopia 1st edition. Edited by Berhane Y, Hailemariam D, Kloos H. Ababa Addis, Ethiopia: Shama PLC, 2006:556–576.

12. Woube .M., 1997, Geographical distribution and dramatic increases in incidences of malaria: consequences of the resettlement scheme in Gambela, SW Ethiopia. *Indian J Malariol* ,34: 140–163.
13. El Samani .F.Z., Willett .W.C., Ware .J.H., 1987, Nutritional and sociodemographic risk indicators of malaria in children under five: a cross-sectional study in a Sudanese rural community. *J Trop Med Hyg*, 90: 69–78.
14. Ahmad .S.H., Moonis .R., Shahab .T., Khan .H.M., Jilani .T., 1985, Effect of nutritional status on total parasite count in malaria. *Indian J Pediat* ,52: 57–61.
15. Smith A, 1959. Effect of residual house spraying in the plains in anopheline density in huts on the Pare mountains. *Nature*, 183: 198–199.
16. Randriantsimaniry .D., 1995, Vector control in the epidemics of the Madagascar highlands. *Sante* ,5: 392–396.

