

ADIEU ! GUINEA WORM

Pages with reference to book, From 83 To 88

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HISTORY

Guinea Worm Disease or Dracunculiasis (Dracontiasis) is among one of the most ancient scourges of mankind that has tormented its victims since time immemorial. It has been referred to as the 'Fiery Serpent' by Moses in the old testament, when it plagued the inhabitants settled along the shores of Red Sea at that time¹ - It has since been known to many Egyptian, Greek and Roman scholars. Galen named it Dracontiasis and Al Rhazes (865—920) showed for the first time that swelling caused in the disease is due to a parasite, the Guinea Worm. Later, Abu All al—Hussain ibne Abdullah ibne Sina (Avicenna), (980—1037), gave the first detailed clinical description of an illness that he called Madina sickness, as it was common in that region and recommended various treatment procedures and drugs² - Explorers of the 15th and 16th century A.D. into different parts of Africa have described the disease. Linnaeus in his publication 'Systema Naturae in 1758 gave the organism its scientific name *Dracunculus medinensis*. A young Russian researcher named Alexksi Pavlovic Fedchenko (1844—1873) discovered the role of cyclops as an intermediate host in 1869², and thus recognized for the first time the involvement of an intermediate vector host in the life cycle of a parasitic infection in humans.

EPIDEMIOLOGY

Dracunculiasis is primarily a disease of poverty, and affects remote rural communities that lack adequate drinking water supplies and are dependent on rain water. Once rampant in most of the Middle East, Africa, Central and South Asia, the disease is now restricted only to 19 countries of the world (17 countries in Africa, India and Pakistan in Asia). It was introduced into several Latin American countries and the Caribbean Islands along with the slave trade, but apparently all the New World foci are now extinct. The disease is most severe in West African countries of Benin, Burkina Faso, Ghana, Mali, Mauritania, Niger, Nigeria, Senegal, Togo and Upper Volta³. Serious disease problems also exist in areas of East Africa such as Ethiopia, Sudan and Uganda. In India, the disease has been eradicated from southern region of Tamil Nadu, however, it still affects many districts in the two north western states of Andhra Pradesh and Rajasthan (this state shares a long border with Pakistan). The number of cases that occur annually cannot be determined as few patients ever report at medical facilities. In one study in Rajasthan, none of the 985 patients seen, ever visited a health center⁴. Estimates however range from 10—48 million. The prevalence rates in some regions are low while in others these may exceed well over 50 percent. In Pakistan, the disease is restricted only to parts of five districts i.e. Bannu and Dera Ismail Khan in North West Frontier Province, Dera Ghazi Khan in Punjab and Tharparkar and Sanghar⁵ (Thar region) in the Sind province. Transmission of disease is seasonal corresponding with the rainy season. Prevalence rates of the disease, are low, ranging close to 2 %. Over 85 % of cases in Pakistan occur between June and October peaking in July and August. In the district of Bannu, the source of disease transmission are mainly the cisterns built in court yards where the rain water is drained and collected⁶. In Dera Ismail Khan and neighbouring Dera Ghazi Khan, the rain water is collected in large open ponds and in Sind, rain water is collected in Tarais which are depressions or pond like structures in the Thar Desert. The inhabitants for most of the year, use water from deep wells in which water is not only scarce, but is also brackish. Therefore when Tarais are filled following rains, these become a preferred source of drinking water⁷. Adults of working age groups (20—45 years) are the usual

victims of the disease. Children under the age of four and old people are seldom seen to have active infection. In communities where females are solely responsible for collecting water for domestic purposes, they play a significant role in disease transmission.

LIFE CYCLE

Human infection occurs when water containing infected cyclops (those having infective stage larvae) is ingested. Cyclops are killed by gastric juices in the stomach and the larvae pass quickly through into the duodenum. In experimental animals, the larvae are seen to penetrate out of the duodenal wall in 10 to 13 hours post ingestion, and migrate via mesentery to the abdominal and thoracic muscles by about 15th day. As they develop further, they begin to migrate towards the connective tissues of axillary and inguinal region, where they mature into adult worms. The size of the worms at this stage remains small. Mating occurs at around three months post infection, and thereafter the males (size 1 to 4 cm) usually move into deeper tissues and perish after 6 months. Sometimes they are encysted and calcify. By eight months of post infection, the gravid female is filled up with eggs, which turn into 1st stage larvae by about 10 months. At this time the worm begins its migration usually towards extremities and is ready to emerge from the body between 10 to 14 months after infection. The worm emerges through a blister which ruptures when it comes in contact with water. Numerous first stage larvae are expelled into the water in a milky white stream. Estimates of the larvae contained in the uterus of a single worm range from 1.4 to 3 million. Not all of these larvae are released at once and it has been shown that about half million larvae are released on first immersion in water. The anterior end of the worm then becomes flaccid and dries up. When the host enters the water again, more larvae are expelled through the broken end, though in greatly reduced number for upto a period of 1 to 2 weeks⁸. The first stage larvae (640 micron x 13 micron) remain active in the ponds for about one week. Following ingestion by the cyclops, the larvae penetrate the cyclop gut wall and reach its haemocoel within one to six hours. They molt twice inside the cyclops and reach the infective 3rd stage in 14 days⁹. Temperatures above 24°C and below 19°C inhibit the growth of larvae which are then incapable of reaching the infective stage. It has also been observed that the cyclops containing infected larvae are sluggish in their movements and tend to sink to the bottom of the ponds as compared to the non-infected ones. There is some evidence to suggest that the life span of the infected cyclops is also shortened^{9,10}.

CLINICAL PICTURE AND TREATMENT

The worm shows no clinical signs until it reaches a point where it is ready to discharge its larvae. In over 90 % of cases, it emerges in the lower extremities although occasionally it may exit from other sites (especially the back, in water carriers). Infection with a single worm is usual, but multiple worm infections are not uncommon. There is no immunity and reinfection is frequent even in already infected individuals. The worm prior to its emergence is sometimes visible and palpable in the subcutaneous tissues. The adult female worm (measuring between 70 to 90 centimeters) secretes a toxic substance at the site of its emergence that causes a local inflammatory lesion. An indurated papule is formed which enlarges and turns into a vesicle within 24 to 48 hours. Severe pain and burning is noticed at the site of emergence. This soon ruptures leaving a superficial ulcer through which the head of the worm is visible. In some cases there may be generalized pruritus sometimes accompanied by urticaria. There may also be nausea, vomiting, diarrhoea and asthma like symptoms with dyspnoea in some cases. The severity of general symptoms varies greatly among individuals and from one geographical location to another. These however, generally subside by the time the local lesion has ruptured and worm has emerged. After the blister has ruptured, the worm body slowly emerges or is extracted out a few centimeters a day over several weeks. In the absence of secondary infection, the

ulcer heals completely after the worm is expelled out in 4 to 6 weeks. The patient during this time finds difficulty in walking and is often compelled to quit work. Secondary infection however is the rule, and this may lead to serious complications. Formation of large abscesses involving deep structures including tendon, periosteum and bone occasionally result in severe septicaemia. Joint lesions caused either by direct involvement or perhaps by some toxins liberated by the worm are also seen. These lesions vary from painful red swellings to advanced pyogenic infections which often result in fibrosing ankylosis and deformity of involved joints. Majority of such lesions are seen in anide and knees. Increased incidence of tetanus has been associated with the disease and in a study in Upper Volta, 7% of persons dying of tetanus had been infected through Guinea worm ulcer. Rare complications include abscess in the pericardium causing constrictive pericarditis, paraplegia as well as quadriplegia resulting from extra dural abscess. Scrotal, vesicle and renal lesions are among other rare complications. A. number of female worms never reach the surface. These become encysted and are either absorbed or are calcified. 29% of uninfected villagers from an endemic area in India, showed calcified worms in roentgenograms¹¹. There is no known cure for the disease and each emerging worm is laboriously roiled out a few centimeters a day on a small stick. Although an ancient practice, if performed with proper antiseptic care and precautions, this still remains an effective method for worm extraction. Since the late 1960s, three compounds have reported to have some effects on the emerging adult worm. These are Niridazole (25mg /kg body wt, daily for 10 days), Thiabendazole (50mg / kg body wt, daily for 7 days) and Metronidazole (400 mg daily, for 10 to 20 days). These drugs produce symptomatic relief of pain and pruritus and hasten the expulsion of worms to a certain degree. They have however, no effect on the pre-emerging worms, and it appears that these compound act primarily against host reaction, as their anti inflammatory properties reduce the intensity of tissue reaction around the worm cuticle. None of these compounds are of any value in the prevention of disease. In Rhesus monkeys local treatment with hydrocortisone containing antibiotics eased greatly the expulsion of the worms¹². Surgical removal of worms under local anaesthesia is frequently used in some parts. This however, can be hazardous procedure if the worm is embedded in the deeper tissues or around tendons.

PREVENTION AND CONTROL

Provision of Safe Drinking Water

Since ingestion of contaminated drinking water is the only mode of transmission, provision and use of drinking water, not contaminated with infected cyclops interrupts transmission and will result in the disappearance of disease in 1 to 2 years. Provision of piped water in a town in Nigeria reduced the incidence of disease from 60% to zero in 2 years. In Ivory Coast a well drilling programme in the 1970 reduced the prevalence of dracunculiasis from 30% to one percent. In Pakistan provision of piped water by the Public Health Engineering Department has eliminated the disease from some previously known infected foci of high endemicity. It must be emphasized here that provision of safe drinking water to the affected communities alone, does not always result in disease eradication. Proper use of such water must be simultaneously ensured. Several instances are recorded in which the villagers continued to use traditional contaminated sources despite having safe drinking water supplies, because these were simply easier to access, better in taste or because of broken pumps¹³.

CHEMICAL TREATMENT OF CONTAMINATED WATER SOURCES

Provision of permanent safe drinking water in many areas has multiple constraints such as expenses involved, length of time needed for construction and so forth. Nevertheless, transmission of dracunculiasis can be interrupted in endemic villages by periodic chemical treatment of drinking water

sources to kill the cyclops. In areas such as Pakistan where transmission of disease is seasonal, monthly application of an organophosphorus compound, Abate (Temephos) during the 3 to 4 month transmission season is adequate. The chemical is applied at a concentration of 1 part per million. At this concentration it kills cyclops species but remains harmless to vegetation and fish. It also has a wide margin of safety for ingestion by humans. The chemical itself is tasteless, colourless and odorless in drinking water. It is available in formulations of 1% or 2% preparations in sand granules or as 50% emulsion in oil. The latter leaves mild odor and taste in the treated sources, but this soon disappears in a few hours. This insecticide is being used widely in Malaria Control Programme as well as Onchocerciasis Control Programme by a number of W.H.O projects in many countries.

HEALTH EDUCATION

Health Education is an effective tool in combating dracunculiasis. This however requires a certain degree of community mobilization and cooperation involving both infected and noninfected members. These measures essentially include boiling or filtering of drinking water and prevention of infected individuals from entering into drinking water reservoirs. Because firewood and other sources of fuel are expensive and in short supply, filtering is a recommended measure to prevent ingestion of contaminated water, as this involves a simple low cost technology. A 100 micrometer mesh monofilament fabric has been shown to effectively remove cyclops from drinking water and resist clogging as well. This is being extensively used in endemic villages in Pakistan, where it is readily accepted and easily lasts at least one transmission season, if handled with care. Meticulous and thoroughly planned health education programmes in certain communities have brought about significant changes in health behaviour, and have resulted in drastic reduction in the incidence of dracunculiasis within 2 years in such communities¹⁴.

SOCIAL AND ECONOMIC EFFECTS

Dracunculiasis is a non-fatal disease and even though there is no known treatment, infection is usually followed by spontaneous recovery. The disease however, causes a certain degree of disability and incapacitation lasting over a period of several months, more particularly so in cases who have more than one emerging worms. Peak incidence of disease sometimes coincides with that of planting and harvest season in certain communities. Therefore the losses in agriculture in such communities are of considerable magnitude. In a study in West Africa, it was shown that about a quarter of working population between the ages of 15 to 40 years are incapacitated for a period of 10 weeks each year, and 0.5 % of the sufferers become permanently disabled¹⁵. School attendance in many endemic areas drops as many students are unable to attend school either because they themselves are affected or they have to work in place of their disabled family members. Studies in Nigeria confirmed that absenteeism from school rose from 13% during the rest of the year, to 60% during peak of guinea worm season and two schools had to close down totally for a period of two weeks¹⁶. Student absenteeism during this period leads to failure in examinations, repetition of grades and termination of education. In Togo, it is estimated that the number of work days lost due to guinea worm disease may be as high as 40 million, and in India the estimates range between 10 to 15 million. In 1982, the World Bank estimated global loss attributable to guinea worm to be as high as one billion US dollars with an estimated annual loss of US \$ 56 to 277 million in wages alone. In Burkina Faso the annual losses from guinea worm disease are as high as 10% of its per capita income¹⁷.

CURRENT INTERNATIONAL EFFORTS' TO ERADICATE GUINEA WORM DISEASE

Barring a few countries, Guinea Worm Disease is considered a low public health problem even in

countries where it is endemic. The International Drinking Water Supply and Sanitation Decade has played a significant role in highlighting the current attention being focussed on this disease. It is the only disease that is exclusively transmitted by drinking contaminated water, and its elimination represents a yardstick to measure the success of the International Drinking Water Supply and Sanitation Decade. The UN Steering Committee for the Decade declared guinea worm eradication, a Decade subgoal. This was also endorsed in a resolution on the Decade by the 34th World Health Assembly in Geneva in 1981 and again in the 41st World Health Assembly in 1988. The first International Workshop on Opportunities for Control of Dracunculiasis was held in Washington, June 1982. This was cosponsored by US National Research Council, W.H.O and U.S.AID. This Workshop, in addition to the First African Regional conference in Niamey, Niger in 1986 and Second African Regional Conference in Ghana, Accra in March 1988, provided useful exchange of knowledge on guinea worm disease as well as opportunities for international collaboration and cooperation among international donor agencies and governments where the disease is endemic. Pakistan along with India were invited to participate in the 2nd African Regional Meeting at Accra, Ghana. Many endemic countries including Pakistan have prepared national plans of action aimed at eradication of dracunculiasis which are in various stages of implementation.

GUINEA WORM ERADICATION PROGRAMME IN PAKISTAN

This programme has been initiated by Global 2000 Inc, a non profit international organization in collaboration with the Federal Ministry of Health. The National Institute of Health, Islamabad, is the national coordinating body for eradication of guinea worm disease from the country. The programme in Pakistan is being financed by the Bank of Credit and Commerce International. In the first phase of the programme which was initiated in 1987, epidemiological surveys were carried out throughout the country. A total of 401 villages with an estimated population of 361,000 in 5 districts of the country reported having guinea worm disease in the preceding 3 year period (Table).

TABLE . Districts Reporting Guinea Worm Disease*.

District	Tot. No. of Pos. Vills	No. with > 10 Cases /Yr	No. with <10 Cases / Yr
Bannu	28	7	21
D.I.Khan	51	5	46
D.G. Khan	70	12	58
Tharparker	244	52	192
Sanghar	8	1	7
Total	401	72	324

***Based on history of guinea worm disease in 1985-1987 during national search, Apr - Jun 1987.**

In addition two pilot control projects were also initiated, one in NWFP and the other in Sind. The objective was to develop field test control measures for later use on a nationwide scale. Useful data on the acceptance, use and durability of nylon filters was obtained. Use of Abate in ponds and cisterns was evaluated and the response of the population was ascertained. Health education modules acceptable to

local population were developed. The second phase of the programme began with the onset of 1988 disease transmission season i.e. April '88. Intensive efforts to eliminate the disease have been initiated in all endemic villages of the country. These include epidemiological surveillance, provision of special nylon filters to all households in the endemic villages, monthly application of Abate (50% preparation in oil) in all rainwater ponds that are being used for drinking water purposes and health education. Supportive measures for guinea worm cases such as bandaging of ulcers and distribution of drugs like antibiotic ointments, analgesics etc. are also being carried out. The programme is being conducted in close collaboration with the provincial health authorities. The target date set for elimination of Guinea Worm disease from Pakistan is the year 1990. It may however be necessary to continue 'mopping up' operations for another 2 to 3 years before final eradication is achieved.

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