THE JOURNEY OF 123 YEARS FILARIASIS IN INDONESIA

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Abstract

Lymphatic filariasis had attacked the people of Indonesia for 123 years. This disease was first reported in Indonesia by Haga and van Eecke in 1889 in Jakarta. Filariasis is caused by filarial worms live in the blood and lymph vessels. Not only a problem in Indonesia, filariasis also becomes health problem in the world. The majority type of filariasis worm in the world is *Brugria malayi*. *B. malayi* was first discovered in 1927 in Bireu, North Sumatra, Indonesia. Recently, filariasis reported to be endemic in 81 countries of the world. The Government of Indonesia in collaboration with WHO has been aggressively dealing with this disease by conducting filariasis elimination program since 1975. As the result, in 1983 filariasis reported no longer be a threaten diseases in Indonesia. Nevertheless, in 1990, filariasis patients with elephantiasis reported in West Java, Indonesia. Finger Blood Analysis stated that there is reemerging diseases. In 2009 reported 31 out of 33 provinces in Indonesia have filariasis endemic area.

The difficulties of eradicating filariasis in Indonesia are caused by many factors. Various problems arising in the course of which the handling of filariasis vaccination to prevent the spread of this disease has not been found, almost all kinds of potential mosquito vectors of this disease, there is still lack of knowledge and awareness in protecting the environment so as not to become a brood of mosquitoes, there are types of filariasis hospes reservoir could potentially have a role in transmission of filariasis, namely cats, monkeys, dogs, and squirrels. Another problem is related to the treatment of mass associated with the fear of side effects. This is certainly an impact on the potential spread of filariasis and the reemerging disease.

WHO launched the movement with a target to eradicate filariasis is a disease-free world by 2020. But whether this can be achieved especially in Indonesia by analyzing the course of the disease in Indonesia? This needs to be a concern and we shared a good reflection of the policy holders and the general public.

Keywords: filariasis, journey, reemerging, Indonesia.

THE JOURNEY OF 123 YEARS FILARIASIS IN INDONESIA

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A. History of Filariasis in Indonesia.

Filariasis is not new in Indonesia. Filariasis in Indonesia was first reported by Haga and van Eecke in 1889 in Jakarta, namely the discovery of filariasis patients scrotum (testicles). At the same time Jakarta known endemic lymphatic filariasis caused by Wuchereria bancrofti. There are three types of worms that cause filariasis in Indonesia Wuchereria bancrofti, Brugia malayi and Brugia timori. Filarial species B. malayi was first discovered in the world in Bireun, North Sumatra by Lichtenstein and Brug (1927) based on the morphology of microfilariae from peripheral blood in patients with filariasis there. Pinhao (1961) and David and Edeson (1964.1965) has found that similar to the microfilariae microfilariae B.malayi (later known as B. timori) in humans in Portuguese Timor. Meanwhile, microfilariae are found in West Timor, Flores and Alor (Helen F McGarry, Leigh D, Plant and Taylor, MJ, 2005; Agoes, 1982; and Manabu Sasa, 1979; Faiz, Purnomo et al, 1977).
After the endemicity of filariasis reported in 1860, performed data collection, screening, and an intensive program to eradicate filariasis in endemic filariasis enclave 1860 includes 21 of the 27 provinces throughout Indonesia. The results achieved are satisfactory, based on finger blood survey (SDJ) is to check the presence of microfilariae in peripheral blood of patients, the prevalence of the disease was substantially reduced from 13.3% to 3.29% in 1987. However filariasis back into health problems since 1990 when the discovery of cases of chronic filariasis patients with swelling of the feet. New cases of both chronic and chronic back has not been reported in Indonesia. Laboratory results of the survey in 2002 based on finger blood survey, the average rate of microfilariae (Mf rate) 3.1%, meaning about 6 million people are infected with filarial worms, and about 100 million people have a high risk for contracting for mosquito penularnya widespread. Data in 2006 based on finger blood surveys indicate as many as 321 districts / cities spread across 26 provinces, including West Java stated as the location is endemic. The prevalence of filariasis continues to be reported until the year 2009 recorded 31 of the 33 provinces in Indonesia have endemic area of filariasis. Based on data from the Ministry of Health, up to October 2009 patients with chronic filariasis is spread in 386 districts / cities in Indonesia. The results of the national mapping known prevalence of microfilariae by 19%, meaning that approximately 40 million people in its body containing the microfilariae and can be a source of transmission through different types of mosquitoes to the 125 million people living in the surrounding area. (DG and PL MOH, 2010; Onggowaluyo, Ismid IS, Sungkar, S. 1999; Cohen and Small, 1998; Ilyas, 1990; Sudomo, 1990; Oemijati, 1990).
The disease is considered to be scary because in the chronic phase can lead to permanent disabilities such as enlargement of the legs, breasts, arms, and genital enlargement can occur, because the network of blood vessels around the body's organs were damaged. When the course of the disease continues, causing a large swelling, the operation was less than helpful and even more so cause of disability. WHO has set a global agreement to combat filarial in 2020 (The Global Goal of Elimination of Lymphatic Filariasis as a Public Health problem by the year 2020).

2. Filariasis endemicity in the World

Filariasis (filariasis) is caused by filarial worms live in the blood and lymph vessels. More than 1 billion people in the world has been reported to suffer from this disease and more than 120 million people worldwide have been infected with this disease. Refers to data about the high incidence of filariasis in the world, filariasis endemic areas is widespread in tropical and subtropical regions around the world including Asia, Africa, China, the Pacific and most Americans (Bockarie, Taylor, and Gyopong, 2009; WHO, 2002; Cohen , JE. and Small, C; 1998).

In Indonesia, have been reported cases of filariasis is endemic in many regions. Data in 2009 showed 31 of 33 provinces in Indonesia have endemic villages including West Java province. The disease is transmitted by mosquitoes cucukan. Until now there are 23 known species of the genus Anopheles mosquito, Culex, Mansonia, and Aedes Armigeres widespread and can act as a vector-borne disease filariasis in Indonesia (MOH DG and PL, RI, 2010).

3. The cause of filariasis

The cause of filariasis is a worm that belongs to the phylum Nematoda, Filaroidea superfamily, Family Filariidae (Leiper, 1911). Threadlike filarial worms mature white and living in the tract and lymph nodes (lymphatic systems). The main types of worms cause lymphatic filariasis in humans are Wuchereria bancrofti, Brugia malayi and Brugia timori. W.bancrofti adult worm length between 72-105 mm (females) and 28-42 mm (male), while Brugia malayi between 50-62 mm (females) and between 20-28 mm (male). Of the three types of worms B. malayi filariasis as the cause of the majority. B. reported malayi is endemic in 81 countries in the world.

4. Life Cycle of Filaria Worm

Growth and development of filarial worms occurred in two phases, namely phase in vector mosquitoes and in humans. Mosquitoes act as vectors of biological and hospes intermediate. The mosquito is required for growth and transmission of worms.

When the mosquito sucks human blood, microfilariae ingested and go into the digestive tract of mosquitoes. Microfilariae then took the veil and penetrate the mosquito stomach into the thoracic muscles where the microfilariae will grow, change skins, and develop into infective larvae L1, L2, and the latter being a three-stage infective larvae (L3). The time of entry of microfilariae to be the L3 stage takes 7-21 days. L3 will gather in the glands of the mosquito and then enter salivatorius proboscis (mouth awl) mosquitoes.

When mosquitoes mencucuk human skin, L3 will enter into the human body through the skin cucukan hole. Larvae migrate to nearby lymph nodes next to the adult worms in about 6 months. After becoming an adult, female worms copulation occurs and male worms. One adult female worms can produce up to 10,000 microfilariae per day. W.bancrofti adult worm length between 72-105 mm (females) and 28-42 mm (male), while Brugia malayi between 50-62 mm (females) and between 20-28 mm (male). Adult worms can live 5-10 years and cause various problems due to damage to the lymph vessels and immune system response is generated. (CDC, 2009; Bain and Babayan, 2003; Onggowaluyo et al, 1999)

Figure 2.4. Life cycle and the occurrence of Filaria Worm Lymphoedema Quoted from Albiez, 1985.

Before clinical symptoms arise swelling varies per patient and the perceived loss incurred. There are only felt mild aches, some fever, sore area folds, muscles seemed withdrawn, others complained about peeling skin feels hot and accompanied by pain that makes people with limited motion. If not handled properly, the trip will be a chronic filariasis disease characterized by swelling (lymphoedema). The swelling started from the death of adult worms. Therefore, a new swelling occurred after 5-10 post-entry stage filarial worms microfilariae by vector mosquitoes cucukan. At the time of the adult worms die, the worms will be wrapped by connective tissue as a reaction of the patient's body, causing granulation will clog the lymphatic channels. Blockages cause the lymph fluid can not rise again and cause swelling of the distal (bottom) occlusion. Lymph channel wall is located at the distal occlusion over time will have hiperselular and matrix solidifies and hardens ekstraselulernya causing damage to valves in lymph vessels and muscle contraction around to be less effective in the conduct of lymph flow. In this condition, the swelling is settled.

5. The animals that role in the transmission of filariasis
Filariasis is transmitted by mosquito vectors. Almost all the mosquitoes in Indonesia could potentially transmit the disease. Vector B. malayi in Indonesia vary in different regions. In Sumatra, the vector B. Periodic malayi are Mansonia species mainly borbagai Ma. bonneae / dives and Ma. uniformis. In addition to Mansonia, the type of mosquitoes that potentially can act as a vector is of the genus Anopheles, namely An. paditaeniatus and An. Nigerrimus. Type of vector B. malayi subperiodik in Sumatra is particularly Ma. uniformis, Ma. indiana and Ma. bonneae / dives. In Kalimantan, the vector B. Periodic malayi are Mansonia spp. especially Ma. uniformis that breed in freshwater marshes close to the forest and rubber plantation. In Sulawesi, the vector B. Periodic malayi is Anopheles barbirostris and An. nigerrirnus than Ma. uniformis, Ma. indiana and Ma. bonneae / dives. The main vector is An in Central Sulawesi. barbirostris which breed in rice fields in Sulawesi Mansonia breeding in the same place with Anopheles, but they are also found in a swamp in Maluku vector B. Ma predicted periodic malayi. uniformis and An. bancrofti. In West Java, especially in rural and coastal areas, B. malayi is spread by mosquitoes Ma. Indiana. (Atmosoedjono et al, 1976; heat et al, 1984; Ministry of Health, 2003). The mosquitoes were created Kholik Allah long before the first human born on this earth even before Jurassic age (dinosaurs). Mosquito fossils discovered show that the lives of thousands of mosquitoes are there millions of years ago on this earth and still exists today. This suggests that humans can not be arrogant to claim to eradicate mosquitoes because it is not possible to eradicate the mosquitoes disappear in the earth's surface, although its size is very small compared to humans. Thus a human can do is control the mosquitoes that do not play a role in disease transmission to humans. Some ways you can do is to kill the mosquito, preventing cucukan mosquitoes to humans, and prevent the breeding of mosquitoes in various places brood to protect the environment.

Based on clinical symptoms and morphological examination of peripheral blood using the technique in the examination under the microscope, filariasis in Indonesia is dominated by W. bancrofti and B. malayi. W. bancrofti has no hospes reservoir, while Brugia malayi filarial species can live and breed in animals. (Ilyas, 1990, Sandhosam, 1963; Palmieri, 1985) There are several types of animals that can act as a reservoir for B.malayi hospes including Presbytis monkey species obscura, P. Melalophos, P.cristata, long-tailed macaques irus type Maccaca, M.nemestrina, some type of cat as Felis domestica, F. bengalesnis, paradourus hermaphrodites, and Manis javanica. (Sandhosam, 1963). Of various animals that have the potential as a reservoir hospes, cats are the easiest animals found in humans.

Thinking aspects of zoonotic B. malayi began in 1957 when the successful transmission of the infection experiment B. malayi from man to cat by Edeson and Wharton. Thought Other findings are supported by colleagues about the natural infection B. malayi in cats in 1960 in Malaysia. Furthermore many studies reported that indicate the presence of a natural infection in cats by Brugia species as reported in Thailand, Malaysia, India, and South Kalimantan. Type of filaria have been reported to breed in the cat's body is Brugia malayi and Brugia pahangi. Both species are morphologically similar filaria making it very difficult to distinguish from peripheral blood by staining methods, but in line with the development of engineering technology with PCR examination of these two species can be distinguished. B. pahangi a filarial species commonly found in cats. Inoculation B.pahangi from cats to humans has been done to the volunteers in Malaysia. Based on observations during the 6 weeks was reported B. pahangi can live on the human body. (Sandhosam, 1963).

In Indonesia, the detection of filariasis research in cats has been done by Palmieri et al (1985) in South Kalimantan with the allegations contained in the transmission of filariasis types B.pahangi cats to humans. B. pahangi has a morphology similar to B. malayi. This study uses the technique of microfilariae in peripheral blood staining and detection of acid phosphate activity in cats and humans. But with these techniques have not been able to prove the existence of transmission from cats to humans.

In 1984, Lim and Sudomo managed to find B. naturally malayi in cats and monkeys in the South and North Bengkulu Bengkulu. (Sudomo, 1990). In the WHO report mentions in West Kalimantan found an infected cat and B.pahangi W.kalimantani. In the countries of Asia, there are five species of filaria mammals that have properties similar to the human filarial W.kalimantani, B.pahangi, B.buckleyi, C.ceylonensis, and B.tupaiae. In 1995 a study conducted by Phantana indicate the presence of infection in cats by B.malayi Narathiwat Province, Thailand and some of the next year in 2001 reported infections in cats by B.pahangi same province. (Kanjanopas et all, 2001)

A study in Petaling Jaya (2008) obtained the result that 50% of cats infected with B. pahangi. B.pahangi infections reported in humans using PCR in the year 2008 in the Klang Valley Malaysia located adjacent to the Petaling Jaya. This event is very interesting because many observers of filariasis in line with the progress of investigation techniques, this is the first evidence that filarial infection cat type B. pahangi can occur naturally in humans (Fong et all, 2008).

Information that describes a specific relationship between the environment, is of key importance in the epidemiology of diseases transmitted by animals. Handling of potentially zoonotic filariasis endemicity can be done by cutting the chain of transmission between human-mosquito-human, human-mosquito-hospes reservoir, and hospes-mosquito-human reservoir.

6. Symptoms of Filariasis

Clinical symptoms of filariasis caused by adult worms in the lymphatic system and the reaction in the form of occult filariasis hiperresponsif. In the course of the disease, filariasis begins with acute recurrent lymphangitis and ends with the onset of chronic obstruction of the lymphatic system. Journey when ordered from the incubation period can be divided into: (CDC, 2009; Figueredo-silva et al, 2002; Cook, 1996; Kurniawan, 1994)

1) The prepaten
Prepaten period, the period between the entry of infective larvae to the range between 3 ¬ mikrofilaremia -7 months. Only a part of the population in endemic areas who become mikrofilaremia, and from the mikrofilaremia even this is not all went on to show clinical symptoms.

2) The incubation period
The incubation period, the period between the entry of infective larvae to the occurrence of clinical symptoms ranges between 8 ¬ -16 months.

3) Clinical symptoms of acute
Clinical symptoms are acute lymphangitis (Acute Filarial lymphangitis / AFL) with the heat, fever, and malaise. AFL is usually unilateral. Patients with acute clinical symptoms and mikrofilaremia amikrofilaremia.
Lymphangitis are infection of the lymph vessels that drain an inflammatory locus. Lymphangitis is found in forms such as red streaks are located subcutaneously pain along the affected lymphatic vessels. Dilated lymph vessels are filled with neutrophils and histiocytes. This inflammation extends into the perilimfatik tissue and can progress to cellulitis or abscess. Lymphangitis is usually accompanied by regional lymphadenopathy. Lymphadenitis most frequently on the inguinal glands, often occurs after working hard. Sometimes lymphadenitis with retrograde lymphangitis. Lymph vessels become hard and painful and frequent lymphedema in the ankle and foot. Patients are unable to work for several days. Attacks can occur 1 ¬ 2 X / year to several times per month. Affected lymph nodes may be an abscess, bacterial or fungal infections, break down, forming ulcers and left a scar that typically after a few months. (Atmadja, 1999). (Wejesinghe, 2008; Dreyer, 1999; Pani, 1995).

4) Symptoms of chronic
Chronic symptoms occur about 4-10 ¬ years after the first acute attack. Microfilariae are rarely found at this stage, while adenolimfangitis can still occur. This leads to symptoms of chronic disability that interferes with the activity of the patient and family burden. Figures Disability Adjusted Life-Years (DALY) due to filariasis reach 5549 (WHO, 2001). Symptoms of chronic lymphoedema of the lower extremities are common and pose like an elephant's foot form so-called elephantiasis (elephantiasis). Lymphoedema often affect the occurrence of venous hypertension. (Vacas and Ryan, 2003). Malayi filariasis elephantiasis occurs in the lower leg below the knee and forearm, while the size of the enlarged extremity of not more than 2 times the original size. Elephantiasis caused by W. bancrofti can occur below and above the knee, the size of the enlargement of the extremities can achieve three times the original size. Bancrofti filariasis may occur on kiluria. Clinical symptoms experienced by patients is a liquid urine or urine is white as milk. Liquids such as milk is caused by the leakage of lymph channels in the renal pelvic area, so that the lymph fluid into the urethra. (MOH, 2006).
In the case of elephantiasis may occur skin disease called ADLA (acute-Lymphangio Dermato-adenitis). ADLA caused by secondary infection due to chronic obstruction of

lymph flow to support a secondary infection (Dreyer et al, 1999). Another manifestation of filariasis is kllinik hiperesponsif reaction of the onset of Tropical pulmonary eosinophilia (TPE). Patients usually show a picture similar to symptoms of shortness of bronchial asthma, and the x-ray shows nodular or diffuse adalnya lesions in the lungs. On laboratory examination occurred hipereosinofili increased to 20-90%, increase in serum IgE and filaria-Meyers histologic entity Kouwenaar the lymph nodules, lung, spleen, and liver. At TPE, Mf is often not found in peripheral blood. (Cook, 2000; Atmadja, 1999)

7. Filariasis Diagnosis enforcement
Diagnosis based on clinical filariasis, parasitologik, and epidemiological.
a) Diagnosis Clinic
Enforced through the anamnesis and clinical examination. Clinical diagnosis is important in determining the acute and chronic morbidity (Acute and Chronic Disease Rate). In the circumstances amikrofilaremia, clinical symptoms supported the diagnosis of filariasis in the symptoms and experiences retrograde lymphadenitis, lymphadenitis recurrent and chronic symptoms.

b) Diagnosis Parasitologik
Diagnosis of filariasis is parasitologik enforced if found microfilariae in the blood of the finger print examination (SDJ) with a time of blood sampling in accordance periodisitasnya. Diagnosis can also be confirmed by serological examination.

c) Diagnosis epidemiological
Filariasis endemicity of an area is determined by determining the rate mikrofilarial (Mf rate), Acute Disease Rate (ADR) and Chronic Disease Rate (CDR) by examining at least 10% of the population. Practical approach to determine the filariasis-endemic areas can be done through the examination of at least 500 people SDJ, examined 500 people who are at least 5 people in a positive Mf (10% positive). Another practical method by finding people with elephantiasis. Research conducted by the WHO stated that the discovery of a patient with elephantiasis of the 1000 population, can be estimated there are 10 clinical patients with acute and 100 are mikrofilaremik.

2.1.6. Filariasis Elimination Program
Since 1975, filariasis eradication program carried out includes the enclave of 1860, covering 21 of the 27 provinces throughout Indonesia. The results achieved are satisfactory, the prevalence of the disease was substantially reduced from 13.3% to 3.29% in 1987. However filariasis back into health problems since 1990. New cases reported in Indonesia and back in a few countries in the world. The disease is of concern to the WHO established the Global Agreement to eradicate filariasis by 2020 (The Global Goal of Elimination of Lymphatic Filariasis as a Public Health problem by the year 2020).

8. Vaccination and Treatment of Filariasis
Until now there has been found vaccination to prevent filariasis. Treatment can be done to filariasis is a treatment for acute-phase using anti-filarial drugs.
Filariasis eradication activities in Indonesia in general include screening, treatment, mosquito control, and counseling. (Ilyas, 1990; Kurniawan, 1994). Filariasis Elimination Program aims to protect the public from the threat of filariasis transmission in order to avoid the attack and release from suffering from the disease.

On the operational objectives is described by means of indicators Microfilaria Rate (in%) and Acute Disease Rate (in%), namely:
a. M-f lowers rate to less than 1%;
b. ADR decrease (Figures Acute Pain) up to 0%.
In addition to the special purpose other than the elimination program is to decrease the intensity of filarial infection, no increase in new cases of chronic, and chronic cases the patient can seek help themselves to limit the degree of disability.
Mosquito control is done by 3M plus program is shut down water reservoirs, burying places that can be used as a brood of mosquitoes and drain the tub plus the use of repellent and mosquito nets to prevent mosquito cucukan. Apart from that mosquito control is also performed with the appeal to keep the environment clean.
Drugs used in filariasis eradication program is Carbamazine Diethyl citrate (DEC). DEC is a derivate piperazin with the chemical formula C10H21N3O (N, N-diethyl-4-methylpiperazine-1-carboxamide) is used as the Drug of Choice for filariasis caused by W. bancrofti, B.malayi, B. timori, and Loa loa-. DEC was found in 1947. DEC marketed in the form of citrate salts. This medicine is used in the Global Programme for Elimination of Lymphatic Filariasis the accordance with the program of WHO.
DEC has been used in the treatment of filariasis since the end of the second world war in 1947. DEC does not have a direct lethal effect against microfilariae but by changing the surface structure of the larva so easily removed from the body tissues and make them more easily destroyed by the host defense system. Preparations of these drugs reach peak levels in the blood after 3-4 hours after consumption. DEC research on how to keep a lot of work to be done in 2003-2005 by studying their effect on the sheath of microfilariae W. bancrofti, Peixoto and his colleagues suggested that DEC causes flaking mf sheath without damaging the cuticle. On further research, made it known that DEC work targets arachidonat acid (Arachidonate 5-lipozygenase) and cyclooxygenase pathway (cytochrome c oxidase subunit 1) is located on the sheath of microfilariae (Imming et all, 2006). DEC effectively kills microfilariae W. bancrofti mf rate and decrease in peripheral blood. Farid et al (2005) in his research suggests that DEC kills microfilariae W. Bancrofti are Culex pipiens mosquito sucked.
There are problems that arise in the use of DEC is the difficulty of determining the appropriate dosage according to body weight in mass treatment. Another problem is that the side effects of treatment with DEC is often complained. Side effects can occur as a reaction to the DEC or immunological reaction against the dead worms. DEC side effects of headache, fatigue, joint pain, chills, anorexia, nausea, and vomiting. (Imming et all, 2006; Hoda et all, 2005; Helen et all, 2005). Side effects are consistent with the magnitude of the dose used Apart from side effects of DEC in person, the body can also provide immunologic reaction against worms that die on treatment with DEC. This reaction is characterized by elevated cytokines (IFN / TNF) related to the large number of microfilaria, clinical spectrum, and species of the cause of filariasis. Reaction caused by B. malayi are usually heavier than the W. bancrofti. The more the number of microfilaria in the body of the dead, the greater the induced immunological reactions (Mitchinter, 2000; Neil and Kazura, 1979).
DEC can kill filarial stages that play a role in the transmission of the microfilariae transmission. But DEC little effect in killing adult worms that play a role in causing clinical symptoms. For the various studies have been done to combine the DEC with other treatment methods to improve the effectiveness of treatment and lower doses of DEC that reduce side effects. (WHO, 2002) Several types of drugs have been studied as Mebendazol and Ivermectin but it raises other issues in terms of cost. Currently DEC mass treatment in developing countries including Indonesia, supplying, incorporated with the soil treatment

for worms are Albendazole and tularan plus paracetamol. A study says that the combination of DEC-Albendazole to kill microfilaria to an average of 85% (varying from 54% - 100%) and worms can suppress productivity up to 100%. For the fulfillment of filariasis mass treatment, the government issued Rp. 750,000 per person. (West Java Provincial Health Office, 2008). The views of the combined treatment efficacy, compliance and cost effectiveness is still controversial.
Combination of DEC-Albendazole has been tested through a variety of research is quite effective in controlling filariasis. Adverse effects on foreign direct result of the use of DEC that cause death in humans has not been reported. However, side effects caused by drug filariasis causes a reluctance to take medicine and non-compliance, which in turn inhibit the filariasis elimination program. In India reported that medication non-adherence due to side effects caused 30-40%. In Colombo, Sri Lanka, taking medication compliance rate was only 37.5%. (Wijesinghe et all, 2008). In West Java Province, on 10 November 2009 made the launching of mass treatment in Bandung regency. On the third day after the declaration was reported by more than 900 people complained of side effects such as dizziness, nausea, and vomiting, and 579 people treated at the hospital due to panic with these side effects (Kompas, 12 November 2009; Indonesia Headline, 14 November 2009). Evaluation results of mass treatment coverage in West Java is still far from achieving the target, from 2005 until 2009 in a row at 48.7%, 28.3%, 34.3%, 41.3%, 51.0%. (Dinkesprop Jabar, 2010).
In addition there is a problem in mass treatment of filariasis, there are problems in handling patients with chronic filariasis. Used in the treatment of filariasis eradication program that DEC can not deal with lymphedema that occurs in chronic filariasis. This drug can only be used to treat patients with a positive larvae / worms in the body but the child has not experienced swelling, so it is only to prevent to become chronic. When it entered the chronic stage in which there is swelling and hardening (fibrosis) condition of the patient can not be dealt with taking any anti-filariasis.

9. Cover
Filariasis has been reported to attack the people of Indonesia for 123 years. Types of filariasis caused by B. malayi (now endemic in 81 countries) and even found the first time in the world is in Bireun, North Sumatra Indonesia. Government of Indonesia in collaboration with WHO has been aggressively dealing with this disease by conducting filariasis elimination program from 1975 to 1983. But in 1990 occurred in 2009 and reemerging diseases reported 31 out of 33 provinces in Indonesia have endemic area of filariasis. Various problems arising in the course of handling filariasis from the discovery of vaccination yet, the difficulty of controlling the mosquito vector, yet the discovery of vaccines, there are animals around humans that could potentially have a role hospes reservoir in the transmission of filariasis such as cats, monkeys, dogs, and squirrels, and not achieving the target of mass treatment in endemic areas.
Given these issues, to ponder how to answer the question "Can Indonesia Free Filariasis by 2020 according to the WHO target?" Do not be repeated Filariasis re-emerging in the future? This certainly requires hard work and smart work from various parties, both from the central government, local government, various NGOs and community organizations, clinicians, academics, and community to work together to create an environment free of filariasis transmission.

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