

## **Haematological studies of lymphatic filariae, *Wuchereria bancrofti* affected patients in Arakkonam area, Tamil Nadu, India**

**S. Sarojini and P. Senthilkumaar**

*School of Enzymology and Environmental Toxicology, P.G. and Research Department of Zoology, Sir Theagaraya College, Chennai, Tamil Nadu*

### **ABSTRACT**

*Filariases are a group of vector – borne parasitic diseases of humans and other animals, caused by long, threadlike worms (hence the name “filaria” from Latin) that in their mature adult stages reside in the lymphatics or in connective tissue. Of the eight filarial parasites that commonly infect man three species account for most of the pathology associated with these infections: the lymphatic dwelling filariae; *Wuchereria bancrofti* and *Brugia malayi* and the skin dwelling *Onchocerca volvulus*. A study was undertaken to compare certain haematological parameters viz. Hb content, total blood cell count (RBC and WBC), WBC differential count, platelet count, E.S.R, P.C.V, M.C.V, M.C.H and M.C.H.C between normal persons and filaria affected patients coming for check-up in Primary Health Centres in Arakkonam to find out any specific variations.*

**Key words:** Filariasis, *Wuchereria bancrofti*, *Culex quinquefasciatus*, lymphangitis, hydroele

### **INTRODUCTION**

*Culex quinquefasciatus* is responsible for transmitting the filarial nematode, *Wuchereria bancrofti* (Tropical Africa and Southeast Asia), Chikungunya virus (CHIKV) (Africa, India, and Asia), and Rift Valley fever virus (RVF) (Africa) [1]. *Wuchereria bancrofti* is a filarial nematode that can cause lymphatic filariasis. Currently, worldwide there are approximately 120 million cases of lymphatic filariasis [2]. The mosquito picks up the microfilaria from an infected vertebrate. The nematode develops inside the mosquito, and is passed on to another vertebrate [1]. In India, the most common form of this disease is caused by *Wuchereria bancrofti* and is known as Bancroftian filariasis accounting for 9% of the cases. This form of filariasis is widely distributed both in urban and rural areas. The other form of a disease is caused by *Brugia malayi* and is commonly termed as Brugian (Malayan) filariasis. This is restricted to few rural pockets in the country and the largest endemic tract presently exists along the central part of Kerala. The other localized foci are in Assam, Orissa, Madhya Pradesh and West Bengal. In India, next to malaria, filariasis is an important public health problem among the mosquito borne diseases. It is a chronic disease with bad effects on social, economic and physical aspects. The chronic manifestations are irreversible and acute attack causes temporary disability.

The factors affecting the MDA programme in Kerala state were investigated [3]. In Kerala, MDA (Mass Drug Administration) was studied as a pilot project in Alappuzha and Kozhikode District from 2000-04 and the first round of MDA was launched in Kerala covering eleven endemic districts, in March 2005. On evaluation, the drug distribution coverage, compliance, etc. were found to be not satisfactory and a need to elicit the factors for poor performance of MDA is felt essential. The study has brought out the observations to the notice of the authorities so that appropriate remedial measures are incorporated. [4] found out an unidentified filarial species and its impact on fitness in wild populations of the black-footed ferret (*Mustela nigripes*). Disease can threaten the restoration of

endangered species directly by substantially decreasing host survival or indirectly through incremental decreases in survival and reproduction. The microfilariae of the filarial nematode *Litomosoides sigmodontis* exacerbate the course of lipopolysaccharide-induced sepsis in mice [5]. Helminths facilitate their own survival by actively modulating the immune systems of their hosts. The impacts that different life cycle stages of the rodent filaria *Litomosoides sigmodontis* have on the inflammatory responses of mice injected with sublethal doses of lipopolysaccharide (LPS) were also observed.

[6] reported a case of live intravitreal adult Brugian filaria, where the parasite was successfully removed by pars plana vitrectomy. Identification of the worm was done by light microscopy and confirmed by immuno chromatographic test. The age profiles of infection and specific antibody intensities in two communities with different transmission levels in East Africa to examine the contribution of humoral responses to human immunity to the vector-borne helminth *Wuchereria bancrofti* were compared [7]. The worm intensities were higher and exhibited a nonlinear age pattern in a high-transmission community, Masaika, in contrast to the low but linearly increasing age infection profile observed for a low-transmission community, Kingwede. [8] concluded that filariasis is vector-transmitted parasitoses, exclusively tropical, except for dirofilariosis. Their impact differs according to the type of filaria and the induced immune response. The diagnosis was made based on the presence of dermatological or lymphatic manifestations, acute or chronic, associated with usually extended stays in an endemic country.

A study on morbidity pattern and time trends of filaria cases was carried out in a PHC of Dakshin Kannada District during November 2000 to January 2001 [9]. The age and sex distribution of all 416 filaria cases recorded revealed that 235 (56.49%) were females and maximum concentration of 263 (63.21%) was in the age group of 16-45 years. The seasonal distribution revealed that rainy season accounted the highest number of cases 197 (47.35%). The prevalence was highest, 1.96/1000 population in the year 1993 and lowest, 0.71/1000 population in the year 1995.

A 47-year-old Indian presented with an inguinal mass clinically suspicious as a tumor. Histological examination of the excised mass demonstrated tissue reaction to degenerating intravascular adult filarial worms. The worms have been identified as lymphatic filariae, most probably *Wuchereria bancrofti*. The case report underscored the need to maintain suspicion of genitourinary filarial lesion in non-endemic areas and describes atypical vascular lesions induced by lymphatic filariae [10].

## MATERIALS AND METHODS

Since the outbreak of Filariasis seems to be more in Arakkonam, Vellore District, Tamil Nadu, Arakkonam area was selected for the present study. The blood samples were collected from filarial affected volunteers in and around Arakkonam area. The age group of patients selected for the present study was between 20 and 60 years. The samples were subjected to estimate the haematological parameters viz, Haemoglobin, Total Count of Erythrocytes, Total Count of Leucocytes, Differential Count of Leucocytes, Platelet Count (PC), Erythrocyte Sedimentation Rate (E.S.R), Haematocrit Value (Packed Cell Volume), Mean Corpuscular Volume (M.C.V), Mean Corpuscular Haemoglobin (M.C.H) and Mean Corpuscular Haemoglobin Concentration (M.C.H.C) [11]. The results obtained were subjected to statistical analysis.

## RESULTS AND DISCUSSION

The vector for lymphatic filariae, *Culex quinquefasciatus* (Fig: 1) and lymphatic filariae, *Wuchereria bancrofti* (Fig: 2) and four stages of filariasis (Fig: 3-6) were represented. Haematological parameters viz. Hb content, total blood cell count (RBC and WBC), WBC differential count, platelet count, E.S.R, P.C.V, M.C.V and M.C.H.C were studied in 20 filariasis affected patients; 10 males (Table:1) and 10 females (Table:2).



Fig: 1 *Culex quinquefasciatus*



Fig: 2 *Wuchereria bancrofti*



Fig: 3 Filariasis (Stage – I)



Fig: 4 Filariasis (Stage – II)



Fig: 5 Filariasis (Stage – III)



Fig: 6 Filariasis (Stage – IV)

Reduction in haemoglobin content, total count of RBCs, lymphocytes, basophils, monocytes, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration was observed in filariasis affected patients when compared with normal patients. There was a considerable increase in total WBC count, neutrophils, eosinophils, platelet count, ESR and mean corpuscular value in the affected patients.

Table: 1 Haematological parameters in filariasis affected male patients

EP	Hb mg/ml	RBC-TC mi/cu.mm	WBC-TC /cu.mm	WBC-DC-N %	WBC-DC-L %	WBC-DC-E %	WBC-DC-B %	WBC-DC-M %	PC L/cu.mm	ESR mm/hr	PVC %	MCV cu.mic	MCH pg	MCHC %
C	15.25	5.3	7500	52	41	4	1	2	2.75	10	46	86	29	34
T1	10.3	3.4	15800	81	14	5	0	0	2.9	68	31	91	28	33
T2	13.2	4.4	13600	76	20	4	0	0	2.32	59	40	90	26	32.8
T3	13.4	4.6	12900	74	22	4	0	0	3.43	38	41	93	24	32
T4	13.3	4.4	11700	65	22	13	0	0	2.3	28	40	91	26	30
T5	11.3	3.8	14200	70	26	4	0	0	3.78	96	34	89	27	32
T6	12.5	4.5	10700	56	36	8	0	0	2.9	37	41	90	28	30
T7	13.2	4.6	10300	74	21	5	0	0	3.24	30	41	89	24	31
T8	14.3	4.7	13900	70	16	14	0	0	2.27	46	46	91	27	30.4
T9	12.8	4.2	10600	63	29	8	0	0	2.96	23	38	90	28	30.7
T10	16.2	4.1	9700	61	33	6	0	0	2.96	17	49	90	25	31
SD	1.6463	0.408	2414.95	8.903	7.078	3.62	0.301	0.603	0.478	25.05	5.22	1.758	1.69	1.323
SE	0.502	0.1694	641.84	2.428	2.239	1.169	0.00	0.00	0.1589	7.618	1.63	0.371	0.00	0.133

EP – Experimental parameters; Hb – Haemoglobin; RBC-TC – Red Blood Corpuscles-Total Count; WBC-TC – White Blood Corpuscles-Total Count; WBC-DC – White Blood Corpuscles-Differential Count; N – Neutrophil; L – Lymphocyte; E – Eosinophil; B – Basophil; M – Monocyte; PC – Platelet Count; ESR – Erythrocyte Sedimentation Rate; PVC – Packed Cell Volume; MCV – Mean Corpuscular Volume; MCH – Mean Corpuscular Haemoglobin; MCHC – Mean Corpuscular Haemoglobin Concentration; SD – Standard Deviation; SE – Standard Error

Table: 2 Haematological parameters in filariasis affected female patients

EP	Hb mg/ml	RBC-TC mi/cu.mm	WBC- TC /cu.mm	WBC- DC-N %	WBC- DC-L %	WBC- DC-E %	WBC- DC-B %	WBC- DC-M %	PC L/cu.mm	ESR mm/hr	PVC %	MCV cu.mic	MCH pg	MCHC %
C	13.75	4.8	7500	53	40	4	1	2	3.4	12.5	43	84	28	35
T1	12.5	4.5	14200	82	14	4	0	0	3.0	20	41	89	30	33
T2	12.8	4.3	14200	76	19	5	0	0	2.43	41	38	88	30	33
T3	12.7	4.2	11800	73	16	10	0	1	3.2	57	38	90	30	32
T4	12.6	4.2	8700	70	20	10	0	0	2.74	46	36	88	31	33
T5	11.8	3.96	9800	75	17	8	0	0	3.26	57	35	89	30	32
T6	12.8	4.1	9400	75	18	7	0	0	2.24	46	37	90	31	32
T7	12.8	4.2	134200	80	15	5	0	0	2.3	25	38	90	30	33
T8	10.3	3.4	8600	79	14	7	0	0	2.17	28	31	91	30	32
T9	12.2	3.9	12800	81	15	4	0	0	2.12	50	34	87	31	33
T10	11.6	3.8	12400	78	16	6	0	0	2.51	32	34	89	31	33
SD	0.8868	0.3678	2432.59	22.89	7.38	2.24	0.30	0.62	0.47	15.10	3.37	1.911	0.87	0.87
SE	0.252	0.0972	696.59	2.306	2.287	1.753	0.00	0.00	0.171	4.197	0.89	0.378	0.00	0.133

EP – Experimental parameters; Hb – Haemoglobin; RBC-TC – Red Blood Corpuscles-Total Count; WBC-TC – White Blood Corpuscles-Total Count; WBC-DC – White Blood Corpuscles-Differential Count; N – Neutrophil; L – Lymphocyte; E – Eosinophil; B – Basophil; M – Monocyte; PC – Platelet Count; ESR – Erythrocyte Sedimentation Rate; PCV – Packed Cell Volume; MCV – Mean Corpuscular Volume; MCH – Mean Corpuscular Haemoglobin; MCHC – Mean Corpuscular Haemoglobin Concentration; SD – Standard Deviation; SE – Standard Error

Filaria parasites develop in two hosts. Man is the Primary host (definitive) where as mosquito acts as the Secondary hosts (Intermediate). Development of the parasite in man takes a long time (5-18 months) but in mosquitoes it takes 10-14 days only. The adult worms (thread like, 4-10 cms long) are lodged in the lymphatic system of man. The female and male worms mate within the human body and the fertilized female liberates thousands of larvae, known as microfilariae (mf). During day time microfilariae remain concentrated in the capillaries and blood vessels of internal organs especially lungs. These are released into the blood stream and circulated in the peripheral blood at night periodically. Further development of mf takes place in the body of the mosquito vector.

The microfilaria (mf) ingested by mosquitoes along with the blood, sheds its body cover and migrates to the thoracic muscle of the mosquito, where it undergoes two moultings L1 stages or 1st stage; which is short, thick and sausage form. L2 stage or 2nd stage; which is long with slow movement. L3 stage or 3rd stage; after second moulting, the parasite looses its cuticle and it known as L3 - or infective stage larva, (L3) migrates to the proboscis (mouth parts) of the mosquito. When the infective feeds on man, these larvae are deposited on the skin near the site of the bite, few of them succeed in penetrating the wounds. The infective stage larvae develop into adult worm within human body.

In humans, the microfilariae show a characteristic periodicity in the course of the circadian or 24 hour cycle. They live mainly in the pulmonary capillaries, from where a proportion of them escape into the peripheral blood and can be detected during the hours of their periodicity. The appearance of mf in the peripheral blood of man synchronizes with the biting period of the vector mosquito and depends upon the sleeping habits of man. Filariasis the disease of 'lymphatic system', a system comprising of lymphatics and lymph gland through which tissue fluids known as 'lymph' passes from different parts of the body to the heart. When filarial adult worms are lodged in lymphatics and in lymph glands, they obstruct mechanically the flow of lymph and also produce inflammatory and allergic reactions. The disease phases are obstruction, inflammation (affected part becomes red, warm, painful and makes movement difficult) and allergy.

Lymphatic filariasis is characterized by a wide spectrum of clinical manifestations with signs and symptoms often differing from one endemic area to another. The clinical course of filariasis can be divided into: (a) The asymptomatic stage: This is characterized by the presence of microfilariae in the peripheral blood, although there are no clinical manifestations of filariasis (b) The Acute stage: The acute clinical manifestations are characterized by fever with chills and rigours, headache, bodypain and sweating. The acute manifestations are: (i) Lymphadenitis: Painful, enlarged lymph gland at groin (Inguinal), armpit (Axilla), above elbow (Epitrochlear), behind Knee joint and on thigh (Femoral) (ii) Lymphangitis: Acute inflammation of lymph channels, resulting in reddish streaks on skin over the lymphatics. These start from a lymph node usually in the groin or armpit and progress away from the node. This is usually associated with fever and enlargement of lymph nodes (iii) Funiculitis: This is an acute, painful inflammation of spermatic cord. This is associated with fever and inflammation of testis (Orchitis) and painful glands in the groin (iv) Epididymo-orchitis: This is an acute painful condition involving the testis and epididymis. This is usually associated with fever, funiculitis and lymph node enlargement in the groin (v) Tropical pulmonary Eosinophilia (TPE) : Individuals complaining of difficulty in breathing associated with or without wheeze an living in filarial endemic areas should be suspected for TPE and (c) The Chronic stage: The chronic signs of filariasis do not usually develop before the age of 15 years and only a small proportion of the infected community is affected. During the chronic stage, microfilariae are usually absent from the blood. The chronic

manifestations are: (i) Hydrocele: Repeated attacks of epididymo-orchitis and funiculities results in accumulation of clear fluid in the covering of testis. This results in unilateral or bilateral scrotal swelling, which is usually not painful (ii) Chyluria: This is the condition where the patient complains of passing milky white urine (known as chyle), caused by admixture of lymph with urine due to the rupture of lymphatics into the urinary system (iii) Lymphoedema: These are swelling usually affecting the limbs caused by accumulation of fluid due to blockage of lymph flow. This condition progresses from initial fluid accumulation to irreversible swelling called elephantiasis. There are three grades of lymphoedema depending upon the duration of illness, reversibility of swelling and skin changes. Longevity during the blood feeding stage of mosquito is an important consideration in determining vector potential. In general, the longer the life span of a species, the better its chances of acquiring and transmitting pathogen.

None of the immuno-diagnostic tests currently available is able to define accurately the presence of infection, either because of lack of specificity or because of inability to discriminate between present and past infection. However, the absence of antifilarial antibodies in a patient residing in a non-filarial endemic area excludes the possibility of a filarial aetiology.

The possible organ infections associated with human filariasis, helminthiasis and malaria in Oguta Local Government Area of Imo State, South-Eastern Nigeria was investigated [12]. Blood, urine and stool samples were collected in appropriate containers from 200 male and female respondents aged 31 – 85 years. Parasitological studies were carried out on blood samples for malaria and/or microfilariae parasites while stool samples were tested for the presence of some intestinal parasites. The study showed a prevalence of intestinal protozoa (*Entamoeba histolytica*), *Wuchereria bancrofti*, the intestinal helminthes *Ascaris lumbricoides* and Hookworms. Biochemical parameters of liver integrity were also studied across the various infection cohorts among the respondents. Results obtained show that these parasitic infections depressed the hematological parameters relative to 'normal' respondents. These findings correlate with the present study.

Elimination of lymphatic filariasis (LF) in the Pacific Island Countries and Territories (PICT) has been defined as <0.1% circulating filarial antigen (CFA) prevalence in children born after the implementation of successful mass drug administrations (MDAs). This research assessed the feasibility of CFA and antibody testing in three countries; Tonga, Vanuatu, and Samoa. Transmission is interrupted in Vanuatu and Tonga as evidenced by no CFA positive children and a low antibody prevalence and titre. Transmission is ongoing in Samoa with microfilaraemic (Mf) and CFA positive children and a high antibody prevalence and titre. Furthermore, areas of transmission were identified with Mf positive adults, but no CFA positive children. These areas had high antibody prevalence in children. In conclusion, CFA testing in children alone was not useful for identifying areas of residual endemicity in Samoa. Thus, it would be beneficial to include antibody serology in the PICT surveillance strategy [13].

Lymphatic filariasis is a neglected disease still prominent in low-resource settings and is very disabling when it progresses to chronic pathology caused by lymphedema. Until now, studies on the contribution of Tregs to lymphocyte hyporesponsiveness in human filariasis have focused on frequency and phenotypic characteristics of these cells. [14] observed at the functional consequence of the presence of Tregs in filaria-specific immune responses during different stages of human lymphatic filariasis. Proliferation of not only T cells, but also B cells, was decreased in patients with microfilaraemia compared to uninfected individuals and chronic pathology (lymphedema) patients. The suppressed lymphocyte proliferative responses were increased after *in vitro* removal of Tregs in the microfilaria-positive group only, indicating the presence of filaria-specific functional Tregs in microfilaraemic patients which are not as active in subjects with chronic pathology or without infection. Th2 cytokine responses were specifically enhanced in microfilaraemics as well after Treg depletion, suggesting Treg-associated suppression of filaria-specific Th2 responses. Taken together, filaria-specific Treg contribute to immune modulation during microfilaraemia and might need to be considered in therapeutic strategies to prevent chronic pathology induced by filarial infection.

The study investigates the relationship between microfilaria density, antigenemia and clinical signs of Bancroftian filariasis in Epe creek communities, June 2009 – July 2010 was investigated [15]. Prevalence rates of microfilaria (MF) and circulating filarial antigen (CFA) in 1803 consenting individuals were 7.0% and 11.3% respectively. Male, (MF: 7.5%, CFA: 11.5%) were more infected than female (MF: 6.5%, CFA: 11.0%) ( $P > 0.05$ ). The microfilaria density and circulating filarial antigen decreased with age and confirms the hypothesis of immunological tolerance during high transmission. More microfilaria density were recorded in age bracket 10 – 19 yrs old while higher circulating filarial antigen was recorded among the age bracket 20 – 29 years old ( $P < 0.05$ ). Clinical signs of filariasis decreased with age; more clinical signs occurred among age bracket of 40 – 60 years. Febrile attack, chyluria, hydrocoele and elephantiasis of the leg and breast were commonly observed. 7.4% of the subjects that were microfilaraemic and 4.2% amicrofilaraemic individuals were also tested positive for circulating

filial antigen. Therefore, to appraise the filarial endemic status in human population, both circulating filarial antigen and microfilaria density should be determined in order to eliminate the chances of underestimating the infection.

Lymphatic Filariasis (LF) is targeted for elimination by the Global Programme for the Elimination of Lymphatic Filariasis (GPELF). The strategy adopted is based on the density dependent phenomenon of Facilitation, which hypothesizes that in an area where the vector species transmitting *Wuchereria bancrofti* are *Anopheles* mosquitoes, it is feasible to eliminate LF using Mass Drug Administration (MDA) because of the inability of *Anopheles* species to transmit low-density microfilaraemia. Even though earlier studies have shown *Anopheles species* can exhibit the process of Facilitation in West Africa, observations point towards the process of Limitation in certain areas, in which case vector control is recommended. [16] studied on *Anopheles* species in West Africa and have also shown genetic differentiation, cryptic taxa and speciation, insecticide resistance and the existence of molecular and chromosomal forms, all of which could influence the vectorial capacity of the mosquitoes and ultimately the elimination goal. The study has outlined the uniqueness of LF vectors in West Africa and the challenges it poses to the 2020 elimination goal, based on the current MDA strategies.

To evaluate predictors of success in a LF control program, [17] conducted an ecological study during a pre-existing MDA program. We studied 27 villages in Lihir Island, Papua New Guinea, from two areas with different infection rates before MDA. Surveys were undertaken to collect information on variables potentially having an influence on the outcome of the program, including epidemiological (baseline prevalence of infection, immigration rate), entomological (vector density) and operational (treatment coverage, vector control strategies) variables. The success in a village was defined using variables related to the infection (circulating filarial antigenemia prevalence <1%) and transmission (antigenemia prevalence <1 in 1000 children born since start of MDA). 8709 people were involved in the MDA program and average coverage rates were around 70%. The overall prevalence of filariasis fell from an initial 17.91% to 3.76% at round 5 ( $p < 0.001$ ). Viewed on a village by village basis, 12/27 (44%) villages achieved success. In multivariate analysis, low baseline prevalence was the only factor predicting both success in reducing infection rates (OR 19, 26; CI 95% 1, 12 to 331, 82) and success in preventing new infections (OR 27, 44; CI 95% 1, 05 to 719, 6). Low vector density and the use of an optimal vector control strategy were also associated with success in reducing infection rates, but this did not reach statistical significance.

Bancroftian filariasis, caused by the filarial parasitic nematode *Wuchereria bancrofti*, affects about 120 million people in the tropics and subtropics. [18] assessed the potential vector competence of *Cx. quinquefasciatus* and studied the temporal distribution and age structure in Benin City, Nigeria. The study was conducted between March and September 2006. Adult females of *Cx. quinquefasciatus* were collected, dissected and microscopically examined for filarial stages. No filarial parasite was detected. The bulk of the population was recorded between 0600 to 0700hrs (42.0%) and 0700hrs to 0800hrs (35.1%). The abundance of *Cx. quinquefasciatus* adults and larvae at all sites were not significantly ( $P > 0.05$ ) different. The parous stage of *Cx. quinquefasciatus* was significantly ( $P < 0.05$ ) higher than the nulliparous stage. The findings of this study and opined that the persistent occurrence of mf, and breeding habitat diversification by *Cx. quinquefasciatus* poses a serious epidemiological concern to the inhabitants of Benin City, Nigeria.

Childhood helminth infections can reduce the risk and severity of allergies and autoimmune diseases, by means of immunomodulation, and a decrease in helminth infections could potentially account for the increased prevalence of these diseases in the western world (hygiene hypothesis). [19] hypothesized that the same immunomodulatory effect can have an impact on metabolic diseases like obesity, diabetes, hypertension and atherosclerosis, wherein inflammation plays a crucial role (extended hygiene hypothesis). To test this hypothesis, we examined the prevalence of lymphatic filariasis (LF) among diabetic, pre-diabetic and non-diabetic subjects were examined who were part of the CURES (Chennai Urban Rural Epidemiology Study) study. In accordance with immunomodulation, reduced prevalence of LF among diabetic subjects compared to non-diabetic and pre-diabetic subjects were found. This was associated with decreased filarial antigen load and anti-filarial antibody levels. The association remained significant even after adjusting for socioeconomic status, age and gender. Interestingly, within the diabetic subjects, those who were filarial positive had reduced levels of pro-inflammatory markers (TNF- $\alpha$ , IL-6 and GM-CSF) compared to those who were filarial negative. In light of these findings, the decreasing incidence of filarial infection due to mass drug administration could potentially have an unexpected adverse impact on the prevalence of diabetes in India.

Lymphatic filariasis is a vector borne parasitic disease causing long term disability. The Global Programme to Eliminate Lymphatic Filariasis aims to achieve its objective through two strategies; Mass Drug Administration (MDA) to interrupt transmission and Morbidity Management (MM) to manage disability for those already affected. MDA is going on in full swing in endemic areas; but MM is lagging behind. An exploratory study was conducted in

Pondicherry through focus group discussions to find out whether there are delivery issues if any, in the MM programme and get suggestions from end users. The results showed that MM has not received the same attention as MDA and there are shortcomings in the delivery mechanism of the programme [20].

Now, in the present investigation, only the haematological studies were concentrated. Further work is necessary for the observation of changes in the blood cells and blood cell organelles. To conclude based on previous studies and our investigation the Mass Drug Administration (MDA) is recommended in the areas affected by mf.

#### REFERENCES

- [1] Foster W.A, Walker E.D, *In* Mullen G, Durden L. (Eds.). Medical and Veterinary Entomology, Academic Press, New York, NY. **2002**, 245-249.
- [2] World Health Organization, Lymphatic filariasis, **2000**.
- [3] Showkath Ali M.K, Rajendran R, Regu K, Mohanan M.K, Dhariwal A.C, Lal S, *J Commun Dis*, **2007**, 39(1), 51-56.
- [4] Wisely S.M, Howard J, Williams S.A, Bain O., Santymire R.M, Bardsley K.D, Williams E.S, *J Wildl Dis*, **2008**, 44(1), 53-64.
- [5] Hubner M.P, Kalaydjiev B, Soboslay P.T, Lengeling A, Schulz-Key H, Mitre E, Hoffmann W.H, *Infect Immun*, **2008**, 76(4), 1668-1677.
- [6] Rao N.G, Mahapatra S.K, Pattnayak S, Pattnaik K, *Indian J Ophthalmol*, **2008**, 56(1), 76-80.
- [7] Jaoko W.G, Michael E, Meyrowitsch D.W, Estambale B.B, Malecela M.N, Simonsen P.E, *Infect Immun*, **2007**, 75(12), 5651-5662.
- [8] Carme B, *Rev Prat*, **2007**, 57(2), 157-165
- [9] Ravikiran E, Sajjan B.S, Vijaya K, Kumar S, Ramkrishna V.A, Dev A.V, Jegan D.P, *Indian J Public Health*, **2005**, 49(2), 100-101.
- [10] Abdel-Hameed A.A, Dura W.T, Alkhalife, I.S, *Saudi Med J*, **2004**, 25(8), 1106-1108.
- [11] John Bernard Henry M.D, *Clinical Diagnosis and Management by Laboratory Methods* 17<sup>th</sup> Ed. W.B. Saunders
- [12] Ojiako O.A, Onyeze G.O.C, *African Journal of Biochemistry Research*, **2009**, 3(4), 114-119.
- [13] Joseph H, Maiava F, Naseri T, Taleo F, Ake M, Capuano C, Wayne Melrose W, *Journal of Tropical Medicine*, **2011**, Article ID 492023, 8 pages.
- [14] Wammes L.J, Hamid F, Wiria A.E, Wibowo H, Sartono E, *PLoS Negl Trop Dis*, **2012**, 6(5), e1655.
- [15] Ebenezer A, Amadi E.C, Agi P, *International Research Journal of Microbiology*, **2011**, 2(9), 370-374.
- [16] Dziedzom K de Souza, Koudou B, Kelly-Hope L.A, Wilson M.D, Bockarie M.J, Boakye D.A, *Parasites & Vectors*, **2012**, 5, 259.
- [17] Oriol M, Raymond P, Russell H, Lysaght G, Nedley L, Mellie S, Quique B, *PLoS. Negl. Trop. Dis*, 2011, 5(8), e1286.
- [18] Aigbodion F.I, Uyi O.O, Akintelu O.H, Salau L.A, *European Journal of Experimental Biology, Pelagia Research Library*, **2011**, 1(4), 173-180.
- [19] Aravindhavan V, Mohan V, Surendar J, Muralidhara Rao M, Pavankumar N, et al. (2010) *PLoS Negl Trop Dis*, 4(6), e707.
- [20] Krishnakumari A, Yuvaraj J, Das L.K, *The Scientific World Journal*, **2012**, Article ID 372618, 6 pages.

Advert