AN ANALYSIS OF OUTCOME IN A NEWER TECHNIQUE OF FILARIAL LEG DEBULKING

A SHORT TERM FOLLOWUP STUDY

Dissertation submitted for

MCh DEGREE EXAMINATION

(PLASTIC AND RECONSTRUCTIVE SURGERY)

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CERTIFICATE

This is to certify that **DR.V.T.S.ARUL SIVAKUMAR,** postgraduate (2005-2008) in the Department of Plastic and Reconstructive surgery,Thanjavur Medical College and Hospital, Thanjavur, has done this dissertation on **AN ANALYSIS OF OUTCOME IN A NEWER TECHNIQUE OF FILARIAL LEG DEBULKING** under my guidance and supervision in partial fulfillment of the regulation laid down by the Tamilnadu DR M.G.R. Medical University, Chennai.India, for MCh (Plastic and Reconstructive surgery) degree examination held on August 2008.

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CONTENTS

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INTRODUCTION

Definition :

Filariasis is a disease group affecting humans that is caused by nematode parasites of the order Filariidae, commonly called filariae.

Filarial parasites may be classified according to the habitat of the adult worms in the vertebral host.

The lymphatic group includes Wuchereria bancrofti, Brugia malayi, and Brugia timori.

Lymphoedema has been defined as swelling of the soft tissues caused by abnormal quantity of lymph.

This definition can be further expanded to include the after effects of this accumulation i.e., proliferation of connective tissue, a variable degree of round cell infiltration, pigmentation and fibrotic thickening of the dilated lymphatics.

In long standing cases this results in a firm enlarged limb with hardened skin and papillomatosis justifying the name ` ELEPHANTIASIS' in severer cases.

In India, the commonest cause of lymphoedema of the limbs is due to Filariasis a disease that is endemic in many parts of our country.

Filariasis in India is caused by the helminths Wuchereria bancrofti and Brugia malayi

Acute infection is caused by the microfilariae, which are the larval forms and are transmitted by mosquitoes of various species.

Chronic filarial lymphoedema is caused by the adult worms which deposit in, and block the lymphatics.

The magnitude of this problem can be seen from the fact that nearly 200 million (20% of the population)live in the endemic zone and more than 19 million, actually suffer from the disease.

The WHO has identified lymphatic filariasis as the second leading cause of permanent and long-term disability in the world after leprosy.

AIM OF THE STUDY

To analyse the outcome in a newer technique of Filarial leg Debulking surgery in a short term follow up.

CLINICAL FEATURES OF THE DISEASE

Depends on the stage of infestation of the disease.

- **Stage of invasion**

  Microfilarial larvae develops producing esonophilia.

  Lymphadenopathy

- **Stage of symptomless carrier state**

  Detected by night blood smear examinations.

- **Stage of Acute manifestations**

  Filarial fever

  Lymphangitis

  Lymphadenitis

  Reversible lymph edema of various parts of the body and

  Epididymo orchitis in males.

- **Stage of Chronic manifestations**

Clinical features of limb edema which is reversible, Elephantiasis of legs,arm,genitals,hydrocele and chyluria.

Acute manifestations of lymphatic filariasis usually are referred to as adenolymphangitis (ADL).

ADL is characterized by episodic attacks of fever associated with inflammation of the inguinal lymph nodes, testis, spermatic cord, lymphedema, or a combination of these.

Skin exfoliation of the affected body part usually occurs with resolution of an episode.

Repeated episodes of inflammation and lymphedema lead to lymphatic damage; chronic swelling; and elephantiasis of the legs, arms, scrotum, vulva, and breasts.

Hydrocele is the most common manifestation of chronic *W bancrofti* infection in males in endemic areas but is rare with *B malayi* and *B timori* infection.

Chyluria also may be present in chronically infected persons

CAUSES OF LYMPHEDEMA

The following classification has been proposed by Foldi (1971).

- **PRIMARY FORMS**

(lymphangiography : Malformation of lymph vessels)

1. Congenital a) Familial (Milroys disease)

b) Non familial.

2. Lymphoedema praecox (appears before age 35 but after age 15)

a) Familial

b) Non-familial

3. Lymphoedema tarda (appears after age 35).

- **SECONDARY FORMS**

(lymphangiography : Dilated, tortuous lymphatics)

1. Neoplastic lymphangitis
2. Obstructive lymphangitis (infection, filariasis, trichophytosis, tuberculosis, etc.)
3. Degenerative lymphangiopathy

4. Lymph node disease

5. Post traumatic lymphangiopathy

6. Surgical resection (block dissection)

7. Roentgen or Radium therapy

8. Causalgia

9. Paralysis

BASIC SCIENCES

ANATOMY

The lymphatic system consists of

(1) lymph capillaries where lymph is formed and collected

(2) lymph vessels which carry the lymph to the regional lymph nodes and then into the

(3) larger collecting channels, emptying the lymph into the venous system in the neck via the thoracic duct and the right lymphatic trunk.

MICRO ANATOMY –Silent features

Lymph capillaries consist of a single layer of epithelial cells.

They do not have any basement membrane and are surrounded by and loosely adherent to collagen tissue (Rusznayak, Foldi and Szabo, 1967).

The intercellular junction of the capillary wall has minute clefts with overlapping of the cells to act as flap valves. A small proportion of these can open widely to accommodate fluid as

required being held apart by collagen microfibrils which open up very widely.

Essentially the structure is the same as blood capillaries but here the holes are more profuse -from 1% to 6% of the capillary surface area.

The lymph capillaries can distend two to three times of their size and also constrict to make their lumen invisible (Zhadnov, 1952).

The lymph vessels have unidirectional valves which allow lymph to flow in a centripetal direction.

These valves are located about a centimetre apart to make the lymph vessels appear like a string of pearls.

The lymphatics of the skin are distributed with a profusion equalling that of the blood capillary system. They form networks which are located in three distinct layers.

1. *Superficial plexus*, situated in the upper third of the dermis and composed of small vessels of uniform calibre which are devoid of valves,
2. *Intermediate plexus* vessels of varying calibre, also devoid of valves. Lastly,

3. *Deep dermal plexus* of vessels varying in size at the junction of the dermis with the subcutaneous tissue.

The epidermis is devoid of lymphatics.

Very little is known about the capillary lymphatic system of the subcutaneous tissue. A plexus situated on the deep fascia plane is sometimes described (Crockett, 1965) but not every one agrees with it.

In the subcutaneous tissue, the valved collecting channels run obliquely or vertically within connective tissue strands linked by horizontal arcades.

These empty into the main lymphatic trunks close to the deep fascia (Thompson, 1967).

Muscles in the body have no lymphatic supply, but the lymph trunks have been found next to the perimysiam internum i.e. around the blood vessels (Rusznayak, Foldi and Szabo, 1967).

It is believed that there are no communications between the superficial and deep lymphatics. Lewis (1975), reported that the only communication between the superficial and deep system is at the postero-lateral aspect of the foot.

More recently this fact has been utilized for doing contrast studies of the deeper lymphatics. However communications

between the superficial and deep lymphatics have been found in pathological studies (Wallace et al, 1964, Jackson, 1966).

Lymphatico-venous Anastomosis

The lymph vessels communicate with the venous system only via the thoracic duct and right lymphatic trunk in the neck. However Rusznayak, Foldi and Szabo (1967), presented data to show that the lymph produced in normal circumstances is much more than the amount which drains out via these channels.

Also, ligation of the thoracic duct does not stop dye from reaching the venous system (Drinker & Yoffrey, 1941).

Lymphatico-venous anastomoses have been described by various authors and hotly denied by others.

Thus after critical evaluation of the existing data, Rusznayak, Foldi and Szabo, (1967) concluded that there were no lymphatico-venous anastomosis other than in the neck in normal circumstances.

However, some degree of lymph might diffuse into the blood vessels at the lymph node level.

Lymphatic-venous anastomoses have, however been described in pathological states associated with lymphatic

obstruction (Wallace, 1964 & Gough, 1966).

Whether they arise de-novo or by opening of existing channels is not known.

LYMPHATIC DRAINAGE OF THE LOWER EXTREMITY

Two distinct set of superficial lymph trunks are developed and these are related to the superficial great and short saphenous veins.

The great saphenous lymphatics are readily cannulated over the dorsum of the foot and drain into the superficial inguinal nodes (Thompson, 1967). The thigh, however drains via separate channels from the leg (Ngu, 1964) and some of the thigh lymphatics bypass the superficial inguinal nodes to enter in the lower most lateral Iliac nodes (Jackson, 1967).

The lesser saphenous lymphatics, if cannulated in the posterior calf, flow laterally around the knee and lower thigh to drain the superficial inguinal nodes (Larson et al. 1966). If they are cannulated behind the lateral malleolus, the flow is via the postero-lateral side of the calf into the popliteal lymph node and thence to the deep lymphatics of the thigh (Malek et al, 1959).

PATHOPHYSIOLOGY

Lymph Formation

The accepted theory of the formation of lymph was proposed by Starling in 1896 who postulated that blood capillary endothelium is a semi permeable membrane.

Hydrodynamic pressure induces ultra filtration in the arterial limb of the capillaries. This slowly diminishes in the subsequent section to become lower than the colloid osmotic pressure of the plasma protein.

Since very little protein diffuses out, the pericapillary tissues are practically free of them so that the colloid osmotic pressure acts as a force of absorption in the venous limb of the capillary.

Thus filtration ceases and the protein free interstitial fluid is absorbed back. However, protein cannot be absorbed by this manner. These and the corpuscular elements are picked up and transported via the lymphatics, which finally empty into the veins.

Under normal circumstances, force of filtration and reabsorption are equal, but if the former is elevated or the latter is

decreased it results in a pathological collection of fluid in the interstitial space and increased lymph flow. **Lymph Absorption**

The precise mechanism by which lymphatics are able to drain the protein rich interstitial fluid is not known. Initially it was postulated that the intra lymphatic pressure was lower than the interstitial fluid pressure and this becomes increasingly negative as the lymphatics travel proximally (McMaster, 1947; Rusznayak, Foldi and Szabo, 1967).

However, Casley Smith (1976), theorised that lymph absorption is due to the higher intra capillary colloid pressure.

Lymph Flow

This is dependent on (Rusznayak, Foldi & Szabo, 1967).

1. Rate of production.
2. Movement passive and active.
3. Negative intra-thoracic pressure.
4. Cisterna Chyli, which acts as a passive heart.
5. Contraction of smooth muscle of the wall of the lymphatics.
6. Arterial pulsations in the deep lymphatics. (Persons and McMaster, 1938).

Mislin (1976) suggested that the cell at the lymphatic junction called `lymphangion' by him was capable of contraction to help the above mechanism.

Olszewski (1980) has suggested that lymphatic flow is determined, at least in normal lymphatics through a lymphatic pulse carrying upwards.

The normal drainage time of lymph from the foot to the thoracic duct is 5 minutes (Lewis, 1975).

LYMPH IN INFLAMMATION

Permeability of the blood capillaries is increased at the site of inflammation. This along with an increased hydrostatic pressure allows large amount of protein rich fluid to diffuse out. So in inflamed edematous tissue, the lymph capillaries and small lymphatics become strongly dilated and filled with protein rich fluid (Drinker and Yoffrey, 1941).

In the later stages, a fibrinous network is formed in the inflammatory area which prevents entrance of fluid into the lymphatic channels. There is associated functional spasm (Foldi et al, 1950) of the lymph vessels.

Organic occlusion may result from thrombi in the lymphatic

circulation (Menkin 1930,1931) leading to lymphatic fixation. Collagen deposition occurs in and around the lymphatics. Thus lymphoedema patients have much thicker lymph vessel walls than normal.(Rada et al,1983; Rada & Tudose,1986)

LYMPHOEDEMA MECHANISM

From Starlings' theory it is clear that insufficiency of lymph flow will give rise to edema.

Rusznayak, Foldi and Szabo (1967) categorised the causes of lymphoedema as following.

I Mechanical insufficiency.

i) **Organic (anatomical causes)**

a) Occlusion of lymphatics.

b) Extirpation of lymphatics or lymph nodes.

ii) **Functional (where flow is decreased)**

a) Haemodynamic insufficiency

b) Lymphangiospasm

c) Akinetic insufficiency

d) Valvular insufficiency

II Dynamic insufficiency (i.e. when excessive lymph is formed e.g. Cardiac or Renal edema)

III Insufficiency of absorption

a) Change of protein ?

b) Change of interstitial space ?

c) Change of lymph capillaries ?

When lymph flow is blocked, as also in inflammation the residual protein is immediately precipitated into a fibrinous network (Zimmerman and Takats 1931).

Fibroblasts wonder into the area leading to a thickening of subcutaneous tissue, multiplication of collagenous elastic fibres and thus result in non pitting edema in late cases.

However, in early cases of filariasis, attacks of lymphangitis are more due to the worm than secondary streptococcal infection (see lymphoedema in filariasis below) (Pani, 1991, Dandapat 1985).

In later stages the worm is mostly dead and very few lymphatics remain to allows reinfection. At this stage the above mechanism promotes perpetuation of the disease.
In late case, following recurrent inflammatory attacks, the skin also

becomes thickened and may show papillomatosis, exercrecences, verrucal changes, recurrent ulceration and hyperpigmentation, finally ending in a non-pitting fibrotic solid oedema.

LYMPHOEDEMA IN FILARIASIS

The adults worms in cases of filariasis localise in the lymphatics and causes blockage of the lymphatic trunks to result in lymphoedema (Lewis 1975).

Repeated lymphangitis related to release of microfilaria is however common. Macrophage action as well as release of complement and other inflammatory protein result in blockage of a wider group of lymphatics.

In endemic areas, massive infection as well as reinfection by the Filaria worm are responsible for lymphoedema.

According to Dey & Dey (1980), lymphoedema in filariasis is due to associated secondary streptococcal infection and absorption of allergen from the worm.

The symptoms of lymphangitis are due to

1. Mechanical irritation.
2. Liberation of toxic fluid by the female worm during parturition.
3. The Toxins paralyses Lymphatic peristalsis.
4. Absorption of toxic products or allergen by the disintegration of the adult worm.
5. Secondary streptococcal infection.

Thus the essential feature is blockage of all the lymphatics over a distance of a few centimetres at a certain level or even all along the limb. Regeneration can only occur if the block is over a very short length.

FINALLY ELEPHANTIASIS IS DUE TO

1. Repeated infection of multiple lymph channels.
2. Endo lymphangitis of the proximal portion obstructing lymph flow.
3. Dilatation of the distal part of the lymphatics and lymph stasis.
4. Infection and.
5. Inability to establish adequate collaterals through hypertrophied tissues.

DIAGNOSIS

Usually the diagnosis is made from the following

1. History
2. Clinical features and findings
3. Lymphangiography
4. Fluorescent microlymph angiography
5. Lymphoscintigraphy
6. CT Scan
7. Study of lymph fluid
8. Night blood smear for microfilariae by Millipore technique
9. Tests to rule out TB,LGV
10. Cytoimmunochemistry- Detection of filarial antigen and antibody
11. Serum immunoglobulins.
12. ACT – CARD TEST
13. Urine examination and microscopy
14. Radio-iodine human serum albumin (rihsa) uptake studies

Detection of microfilariae:

The traditional diagnostic method is to demonstrate microfilariae in the peripheral blood or skin.

Detection of microfilariae in blood :

The microfilariae of all species that cause lymphatic filariasis and the microfilariae of *L loa, M ozzardi,* and *M perstans* are detected in blood.

Capillary finger-prick or venous blood is used for thick blood films. Venous blood also can be concentrated or passed through a Nuclepore filter before being examined microscopically. The species of infection then can be determined by the microscopic appearance.

Microfilaria may be periodic in appearance in the peripheral circulation, and the blood should be examined at different intervals over a 24-hour period to achieve the best chances of detection.

Provocation of nocturnally periodic microfilariae may be achieved with a daytime dose of DEC at 1-2 mg/kg.

Microfilariae also may be observed in chylous urine and hydrocele fluid.

Microfilariae may be absent in patients with ADL or late chronic lymphatic disease.

Detection of filarial antigen:

The presence of circulating filarial antigen in the peripheral blood, with or without microfilariae, now is considered diagnostic of patent filarial infection and is used to monitor the effectiveness of therapy. Commercial kits are available to test venous blood.

Complete blood cell count:

Eosinophilia is marked in all forms of patent filarial infection.

Serum immunoglobulins:

Elevated serum IgE and IgG4 may be observed in patients with active filarial disease.

Histologic Findings:

Affected lymph nodes fibrose.

Lymphatics stenose and obstruct with the creation of collateral channels.

The skin of individuals with elephantiasis is affected with hyperkeratosis, acanthosis, lymph and fatty tissue, loss of elastin fibers, and fibrosis.

Lymphangiography

A radio-opaque dye Iotrolan has been used to delineate lymphatics radiologically after subcutaneous injection.

Present day techniques use ultra-fluid Lipiodol, a fatty based substance as contrast injected via a special slow injector pump (Thompson, 1967).

In secondary lymphoedema the changes are of dilatation leading on to dermal back flow, varicosity, tortuosity and obliquity

The initial changes in lymphangiogreaphy are of secondary lymphoedema but at a later stage they may get fibrosed and decrease in number.

Thus Kanetkar (1966) classified these changes into four groups depending on severity of the disease.

Grades I and II showed extensive proliferation, varicosities and collaterals which decreased with severity

, In later grades the lymph vessels decreased in number but still exhibition dermal back flow.

Thus an elephantiatic limb (gr. IV) showed very few lymph channels.

Occasionally a figure of 8 shadow may be visualised, suggestive of an adult worm lying coiled up in the lymph node of lymphatics.

DIFFERENTIAL DIAGNOSIS

1. Edema due to pregnancy
2. Edema due to malignancy
3. Venous edema
4. Lipedema
5. Edema due to lymphadenopathy
6. Lymphangiomas
7. Iliac vessel compression syndrome.

MANAGEMENT

This depends on the grade of the disease.

Common to all the grades:

Leg rest and elevation

Removal of foci of infection

Long acting Penicillin (Benzathine)—12 L IU IM once in 15 days

Diethyl carbamacine—100mg qd daily for 2 days once in 15 days.

Antihistamines- 1 od for for 2 days once in 15 days.

Gradewise management schedule:

GRADE I : Pressure therapy of intermittent pressure of 30-50 mmHg.

Manual lymphatic drainage

Elastic support

Nodo-venous shunt (*optional)*

GRADE II : Pressure therapy of intermittent pressure of 30-50 mmHg.

Manual lymphatic drainage

Elastic support

Nodo-venous shunt

GRADE III : Pressure therapy of intermittent pressure of 30-50 mmHg.

Manual lymphatic drainage

Elastic support

Nodo-venous shunt

Debulking surgery

GRADE IV : Manual lymphatic drainage

Elastic support

Nodo-venous shunt

Debulking surgery

Sculpturing

SSG for raw areas

MEDICAL THERAPY

1. Foot care and prevention of infection and ulceration is important.
2. Ace stocking, massage and elevation.
3. In cases getting repeated attacks, one could justify a long term penicillin prophylaxis similar to the prophylaxis of Rheumatic fever.

   Manual massage euphemistically called Combined Physical Therapy (CPT)

4. ANTI-PHLOGISTIN DRUGS

   Coumarin and the Benzopyrone group of drugs

5. INTERMITTENT PNEUMATIC EXTERNAL COMPRESSION :

   IPEC increases interstitial fluid pressure so that water is forced hydrostatically across the capillary bed into the venous system. The resulting increase in venous outflow increases further clearance of water and colloid. Existing lymphatic and collateral flow is also augmented in a similar fashion.

6. HEAT TREATMENT

Currently microwave therapy is being used.

Temperatures of around 39-40 degrees are generated in the muscle and subcutaneous tissue.

Elastic dressing is helpful as a conjunct. (Okhuma, 1992).

7. DRUGS

1. Ivermectin: (anthelmintic)

150-200 mcg/kg/d PO as single dose; repeat q2-3mo

Used alone or in combination with DEC.

Exerts its antiparasitic action by acting as a potent agonist at GABA receptors and potentiating the inhibitory signals sent to motor neurons, which paralyses the parasite.

Because GABA is confined to the CNS in humans and because ivermectin does not cross the BBB, the drug has no paralytic action in humans.

Recent studies have validated the use of single-dose regimens of ivermectin and DEC or albendazole for large-scale control and elimination programs and to reduce *W bancrofti* microfilaremia, antigenemia, and clinical manifestations.

2. *Diethylcarbamazine :* 6 mg/kg PO qd for 12 d to 3 wk

Microfilaricide.

The precise mechanism is not understood.

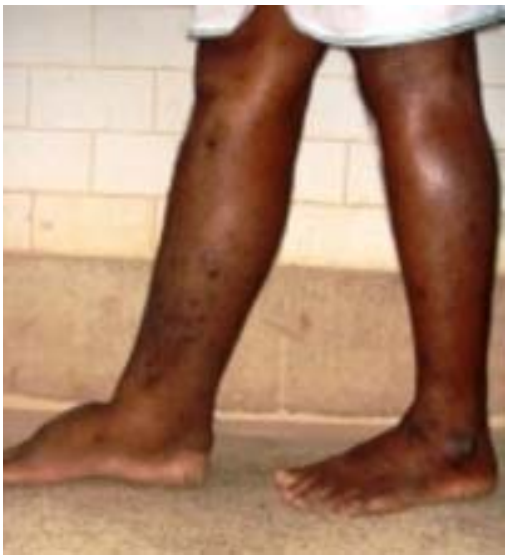
Shown to induce immobilization of microfilariae by decreasing muscle activity because of hyperpolarization effects.

Alteration of surface membrane also occurs, with enhanced destruction by the host's immune system.

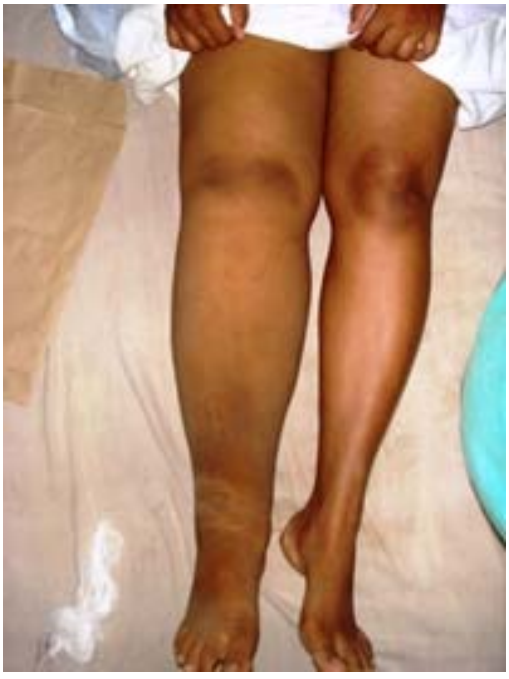
Evidence exists that DEC may enhance adhesion of granulocytes via antibody-dependent and independent mechanisms.

Hypotheses also include interference by microfilarial intracellular processing and transport of specific macromolecules by DEC.

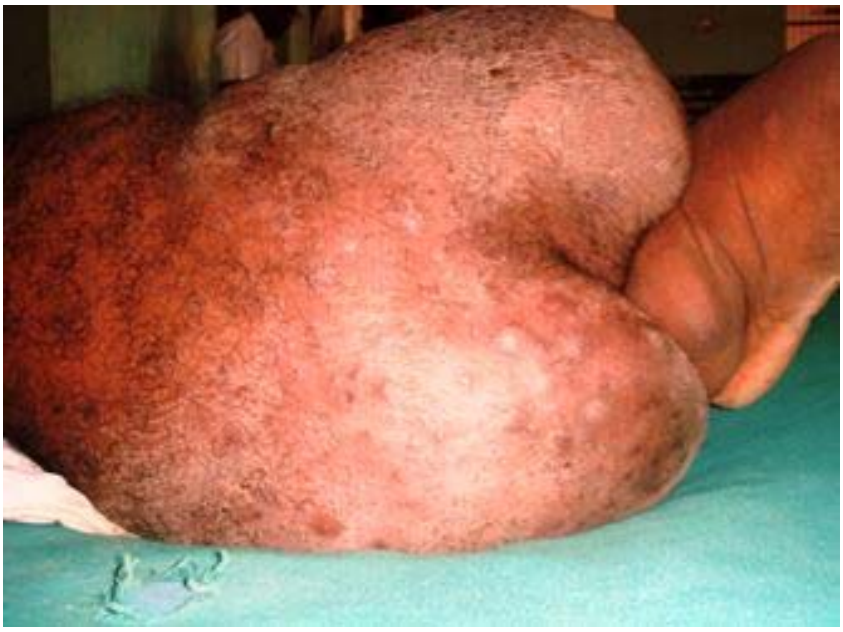
3. Flubendazole :100 mg PO bid for 3 d



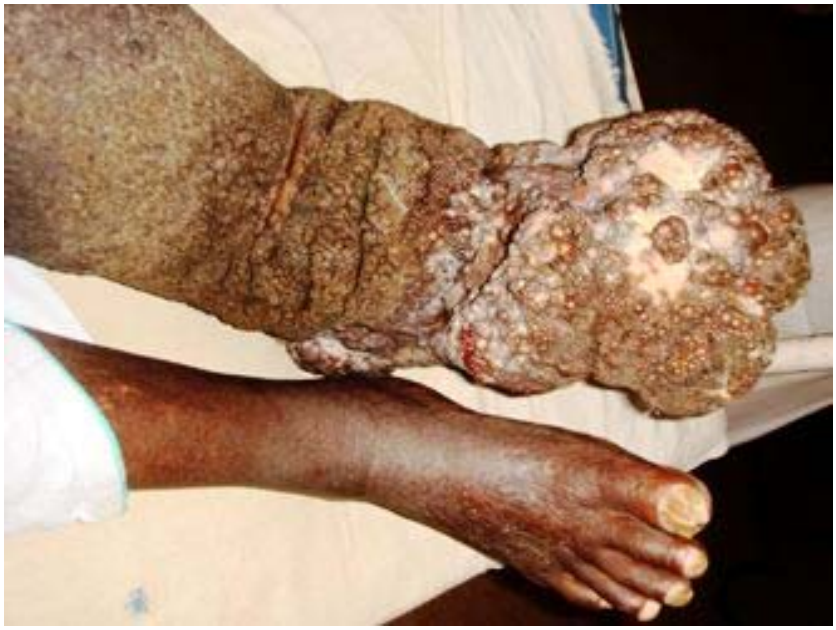
GRADE II LEG



GRADE III LEG



GRADE IV LEG



GRADE IV WITH MARKED SKIN CHANGES

FILARIAL COT

SEQUENTIAL COMPRESSION PUMP

SURGICAL MANAGEMENT

To improve lymphatic transport capacity :

Reconstruction of new lymph vessels with threads and tubes (Handles 1908)

To increase drainage:

1. Drainage through strips of fascia or through Tensor Fascia Lata (Martorell 1958)

2. Drainage through omental flaps ((Dicks k 1935)

3. Drainage through skin flaps (Rosanow 1912)

4. Drainage through Entero mesenteric bridging surgery (Kinmoth 1932)

5. Drainage through Lymphatico –Venous anastomosis

    i. Lympho-Nodovenous anastomosis (Olzeweski 1966,Jamal 1981)

    ii. Anastomosis between lymph collectors and vein (Degni1974)

6. Debulking in foot and legs- Helps in formation of lympho-venous shunts

Procedures to reduce the load of lymph :

a. *Resection of epifascial lymphedematous tissue*

b. *Reduction of epifascial lymph space*

c. *Ligation of arterial blood supply to limb.*

REVIEW OF LITERATURE

Standard surgical procedures :

The standard debulking procedures that are done are,

1. CHARLES PROCEDURE (1911) :

He excised all the skin, subcutaneous upto the deep fascia and applied skin grafts.

The main problem of this operation is frequent break down of the graft with ulceration; weeping, ugly exercresences, pantalooning of the distal portions, as well as recurrence.

2. **LANZ (1909)** :

He excised all the deep fascia, and even trephined the bone to improve lymph drainage of his patients.

3. **KONDOLEON :**

He excised strips of deep fascia to debulk the limb and closed the defect primarily.

4. **SISTRUNK (1917) :**

This is a modification of above procedure.

It involes excision of skin,subcutaneous tissue and deep fascia fro iliac crest to malleous.

The drawbacks are there was no significant reduction and further it could not be applied to huge legs.

5. **HOMANS (1938) :**

He excised the subcutaneous tissue after raising flaps to close them primarily. This procedure is used even now. The main drawbacks are only strips of 2.5cm could be excised which results in minimal reduction in huge legs.

6. **THOMPSON (1962)** :

This involves transposing a flap of dermis with the epidermis shaved off under the deep fascia.

So all the above procedures had their own limitation. Moreover, most of the patients who admit for surgery here have either grade III or IV in which the above procedures did not suit well.

So, we modified little the Homans procedure in that it could be used for huge legs to improve the functional outcome of the patient with pleasing aesthetic effect.

BACK GROUND OF STUDY

Since Thanjavur is one of the endemic areas of filarial infection, filarial lymphedema is one of the major problem we see in our department.

The filarial clinic is exclusively conducted every Monday to treat an average of 3500 patients every year.

Census of filarial patients treated at our centre during the period of study

	2005	2006	2007	2008
Total cases	5450	4863	4332	-
New patients	425	256	311	55
Old patients	5025	4607	4005	321
Surgeries	142	88	57	17
Major	115	79	49	12
minor	27	9	8	5

Depending on the grades of edema the patients are subjected to

1. Prophylactic antibiotic usually Penicillin :

   (Allergens are given either Doxycycline / ciprofloxacin recently)

2. Intermittent Pneumatic compression therapy (for Inpatients)

(Elastic stoking for out patients and post op cases)

3. Nodovenous shunts
4. Sculpturing.
5. Debulking.

MATERIALS AND METHODS

This study was conducted in The department of Plastic surgery and reconstructive surgery Thanjavur medical college during the period 2005 - 2008.

Those patients who had grade III and IV lymphedema and gave willingness for debulking surgery were admitted in the filarial wards and included in the study.

A total of 40 patients from Filarial lymphedema grade III and IV were takenup for study which includes 27 males and 13females.

Pts in age group of 17-71 were included in the study

Inclusion criteria:

Pts with grade iii and iv were included in study.

Exclusion criterias:

Pts in grade I and II.

Pts with comorbid conditions unfit for surgery

Pts with active lymphorroea, ulcerations, raw areas and secondary skin infections .

All the patients were subjected to regular Penicillin prophylaxis as per standard regimen.

Measurements at standard landmarks were recorded in both normal and diseased limb.

All these patients were given regular Pnematic compression pre operatively.

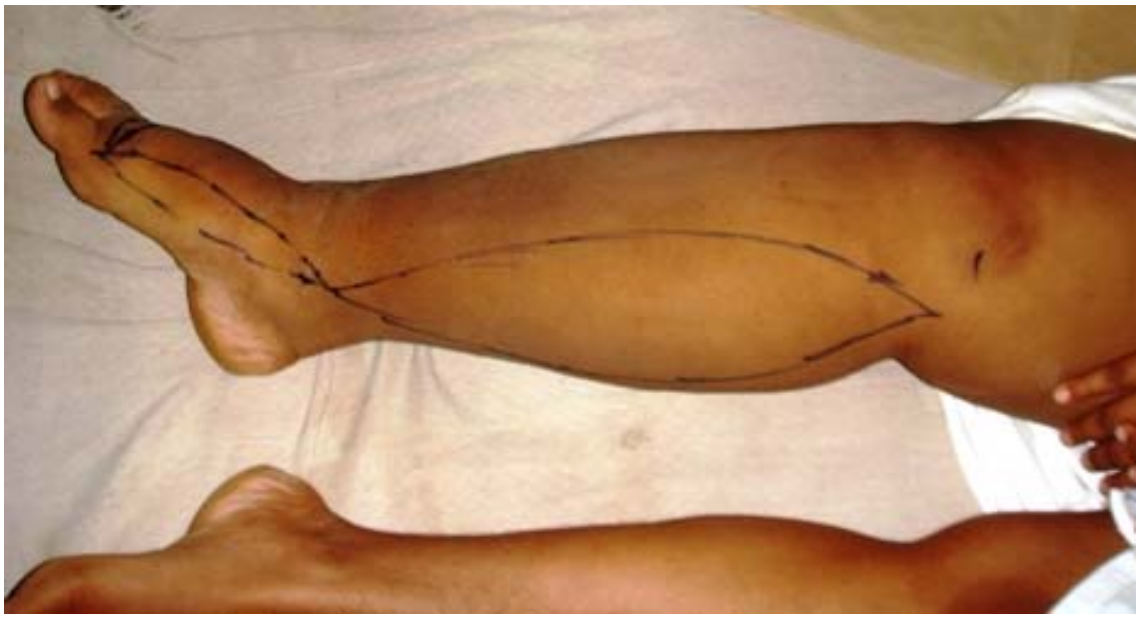
First the patients were operated for nodovenous shunts if not done earlier under local anaesthesia and was advised strict bed rest along with pneumatic compression for a period of week and then takenup for debulking surgery

Those patients who had already undergone nodovenous shunts were subjected to physical therapy for a week and takenup for debulking surgery .

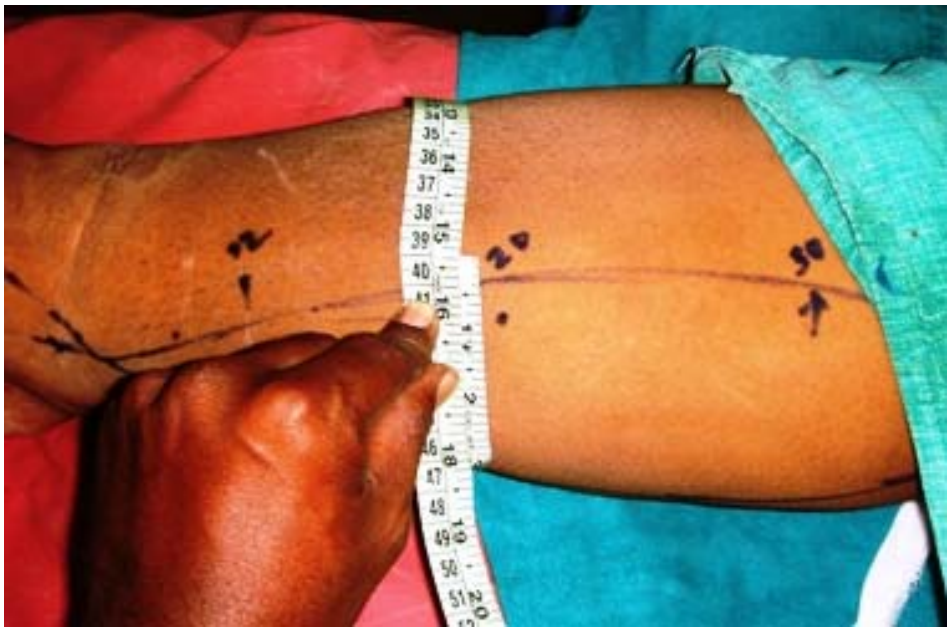
Pts were given elastic stockings post operatively and discharged after suture removal.

A final recordings of measurements at standard sites were done before discharge.

All patients were followed up in filarial op twice a month during their visit for prophylactic antibiotic.

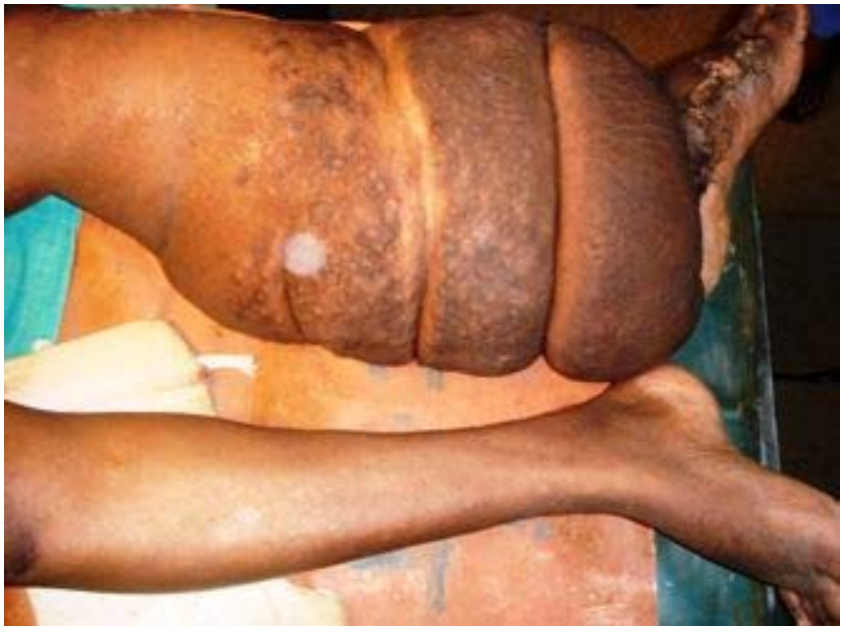


INCISION FOR FOOT AND LEG

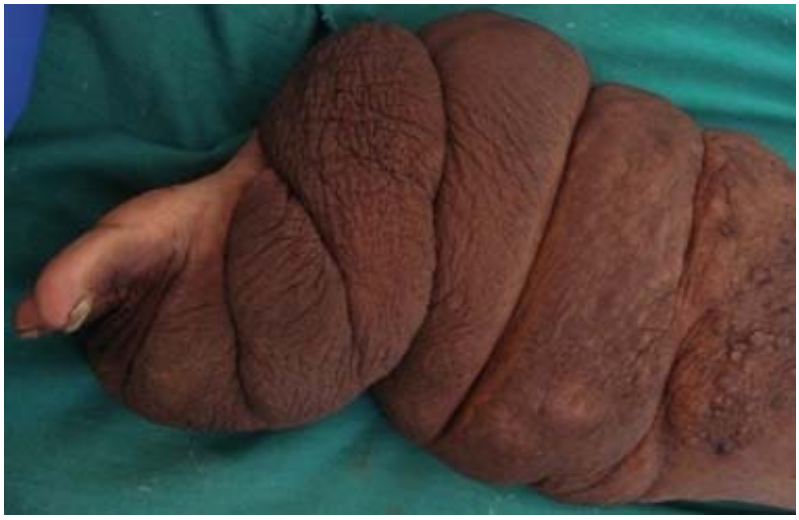


MEASUREMENT AT STANDARD SITE – 20 cm

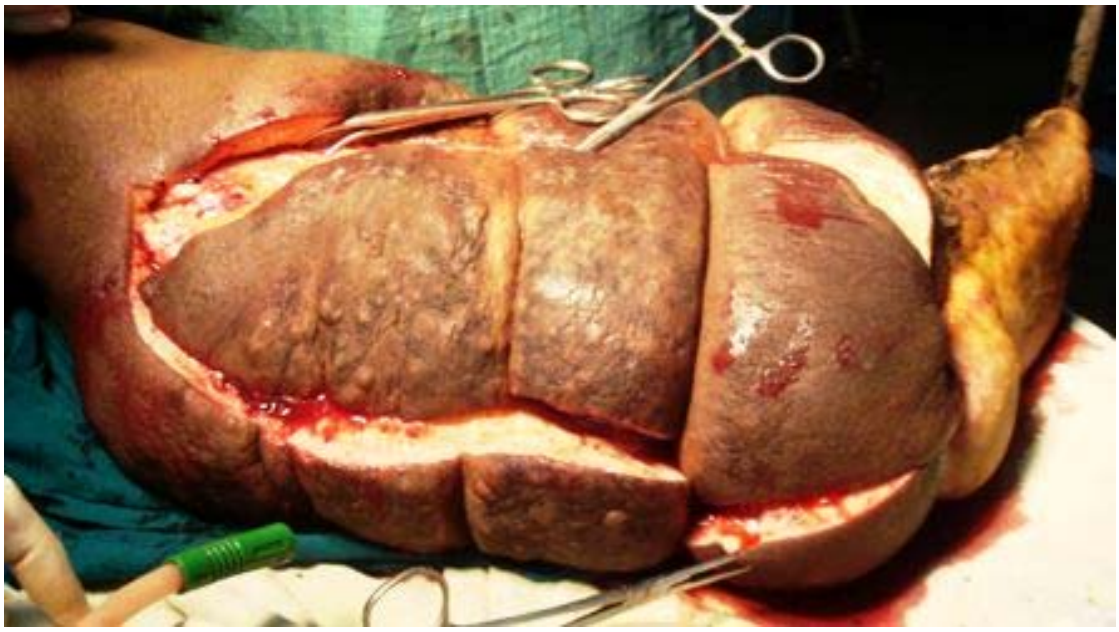
PROCEDURE ILLUSTRATION



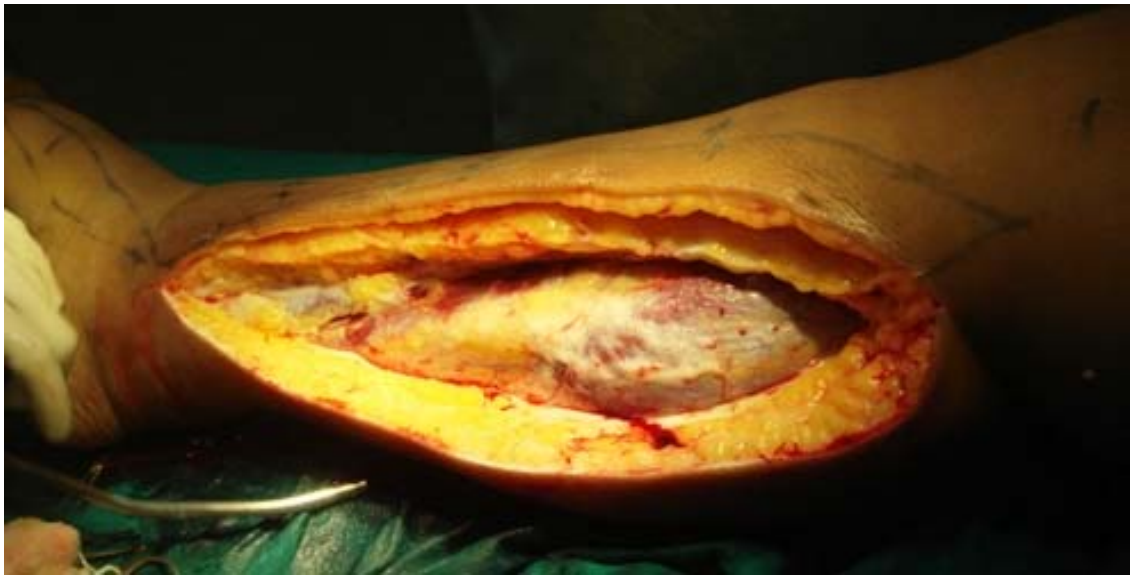
PRE OPERATIVE



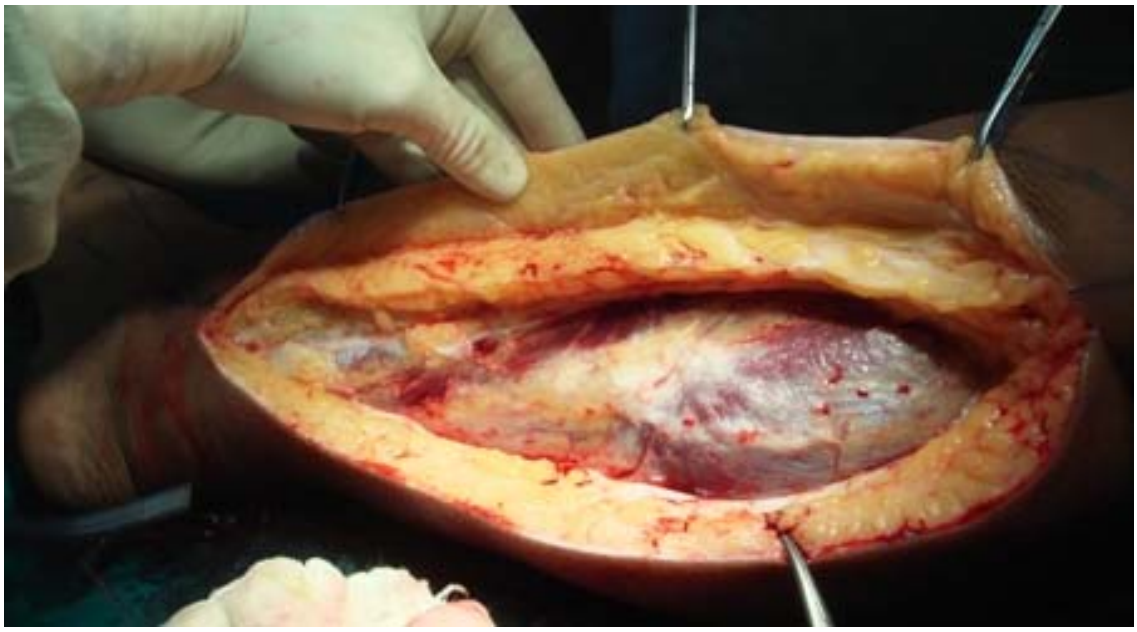
SUPPLE SKIN FACILITATING REDUCTION FOLLOWING SHUNT



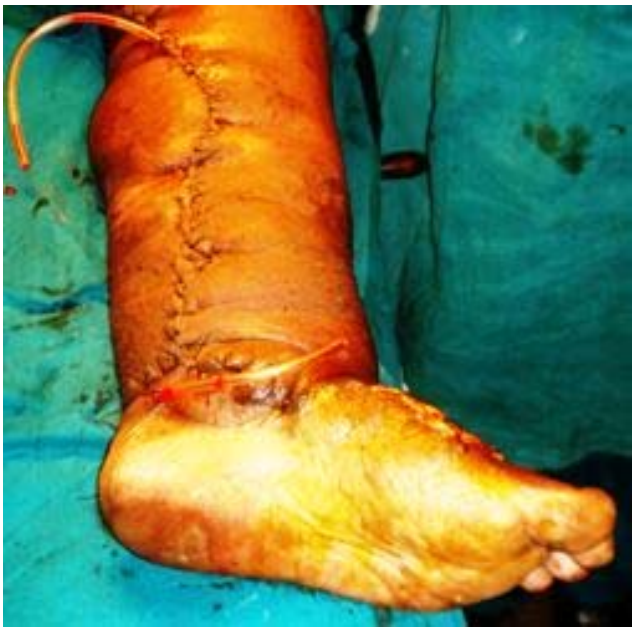
EXCISION OF A LARGER FRAGMENT



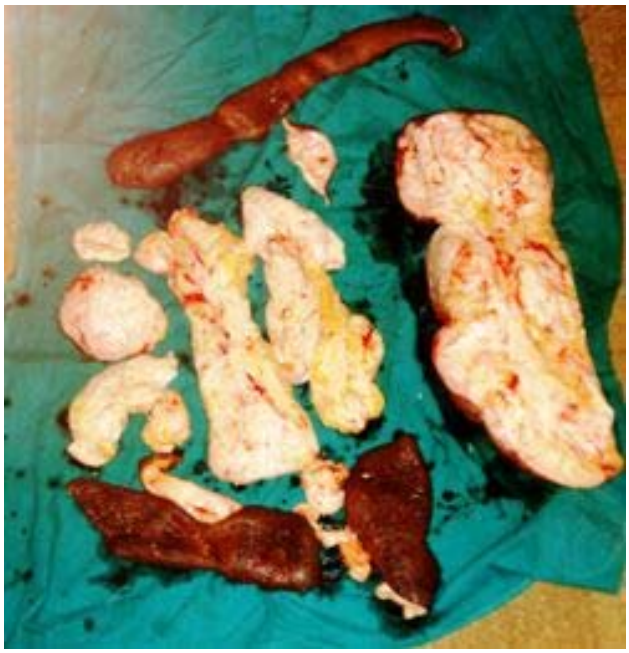
EXCISION INCLUDING DEEP FASCIA



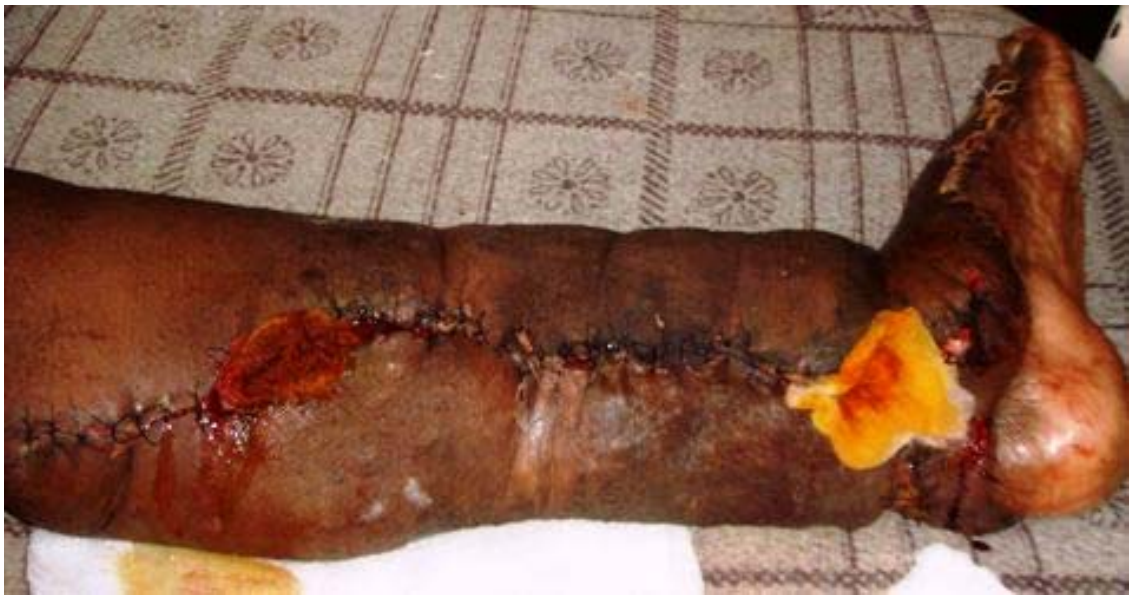
DEBULKING THE FLAPS



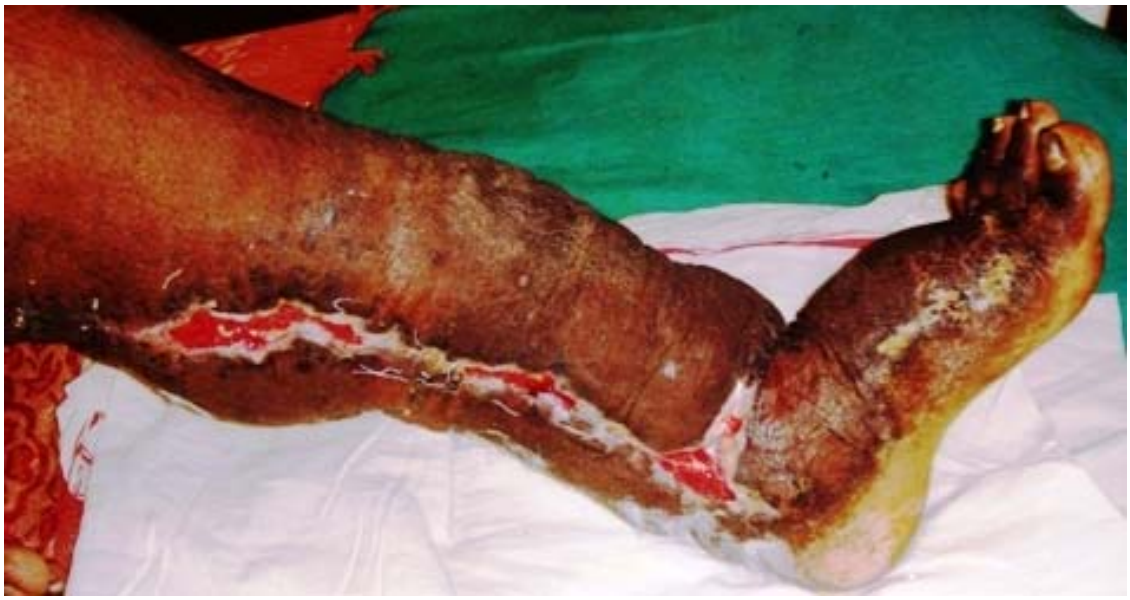
TENSIONLESS SUTURING WITH DRAINAGE TUBE



SPECIMEN



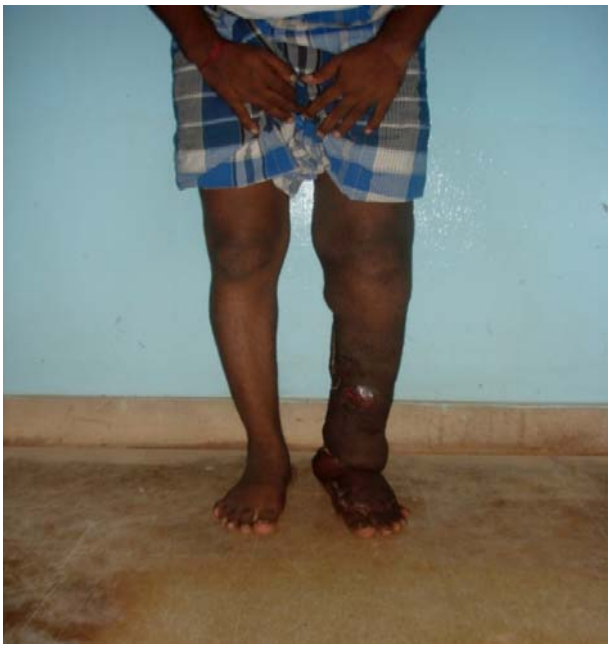
POST OPERATIVE PERIOD WITH EDGE NECROSIS



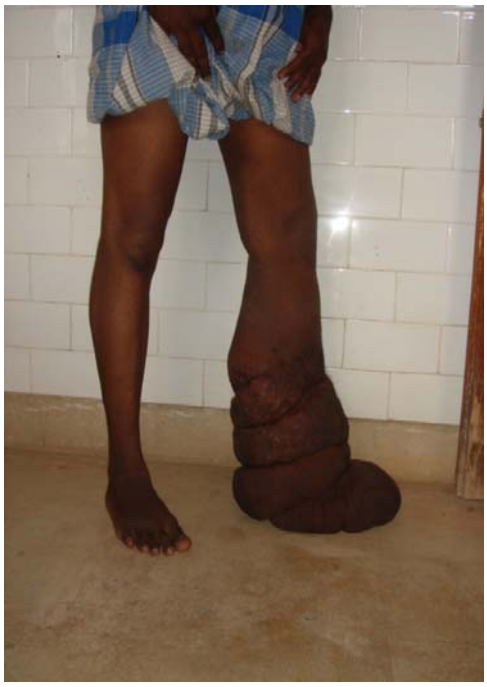
FLAP NECROSIS UNDER CONSERVATIVE TREATMENT

(*ALLOWED TO HEAL BY SECONDARY INTENTION*)

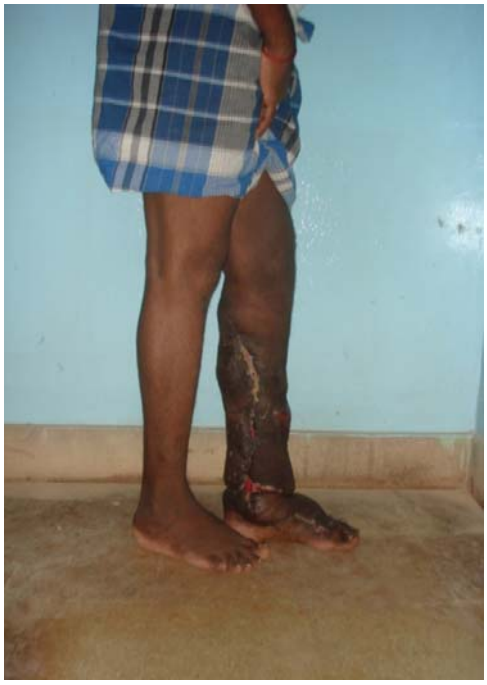
PRE REDUCTION SIZE -ANTERIOR VIEW



POST REDUCTION SIZE- ANTERIOR VIEW



PRE REDUCTION SIZE -MEDIAL VIEW



PROCEDURE

Our procedure of debulking is based on the principles of Homans procedure.

Procedure is done either in GA / Spinal anaesthesia

The limb is lifted for 10-15 minutes and Esmarch tourniquet applied in the upper thigh.

The parts are cleaned with Betadine and surgical incision site with spirit in addition to betadine.

Incisions :

For leg:

A large elliptical incision extending from the medial aspect of knee to medial malleolus

The width of the incision varies according to size of the limb.

For foot:

1. An elliptical incision starting from the distal end of the previous leg incision upto base of the great toe.
2. Another elliptical incision starting from the distal end of the previous incision and extending upto base of the fifth toe on the dorsum of foot.

For digits :

An elliptical incision oriented vertically.

The tissue included in the elliptical incision is excised including the fascia.

The medial and posterior flaps are raised upto 3/5 of the limb circumference.

The flaps are raised in such a manner that the thickness of the flap is maintained at 1 cm with little thicker at its base.

Hemostasis is achieved and verified by removing the tourniquet.

The flaps are trimmed in such a way that gives a tensionless suture line.

The flaps are sutured following insertion of the Vaccum Drain separately for leg and foot.

A tight bandage is applied.

Postoperatively pt is nursed in limb raised position in filarial cot with strict bed rest for a week.

The DT is removed usually on the 4^{th} postoperative day.

Patient is discharged usually between 10-12 days after surgery.

METHODS

The patients were evaluated using measurements of limb circumference at standard sites preoperatively and postoperatively.

Limb circumference measurements :

SITE I :

10 cm from tip of the great toe proximally over dorsum of foot.

SITE II :

12 cm proximal to the dorsoplantar skin junction of the foot in medial side.

SITE III :

20 cm proximal to the dorsoplantar skin junction of the foot in medial side.

SITE IV :

30 cm proximal to the dorsoplantar skin junction of the foot in medial side.

These measurements are recorded both in preoperative period (day before surgery) and postoperative period (at the time of discharge).

The amount of reduction is calculated from the difference in the above two readings.

The percentage of reduction is calculated from this difference to the preoperative reading.

The measurement at III rd site at 20 cms is taken as standard measurement site in leg for comparative study.

Stastical analysis was done using **paired samples-T test procedure, Descriptive and Analytical statistics.**

OBSERVATION

General charecters of the study group:

40 patients of grade III and IV filarial lymphedema were included in the study.

Among these 27 were males and 13 were female.

TABLE I : DISTRIBUTION OF SEX

SEX	FREQUENCY	PERCENT
MALE	27	68%
FEMALE	13	32%
TOTAL	40	1OO%

18 patients belonged to grade III and 22 patients belonged to grade IV.

31 patients underwent nodovenous shunt procedure.

All patients wre subjected to antibiotic and physical therapy esp pneumatic compression.

All patients were given foot care advice.

The patients who were undergoing shunt sugery were taken up for surgery the next week.

TABLE II : DISTRIBUTION OF AGE

AGE	FREQUENCY	PERCENT
< 20	**6**	**15%**
21 – 30	**6**	**15%**
31 – 40	**9**	**22%**
41 – 50	**14**	**35%**
51 - 60	**4**	**10%**
>61	**1**	**3%**
TOTAL	**40**	**100%**

The most comman age group was 41 – 50 years with 14 patients (35%).

The minimum age - 17 years

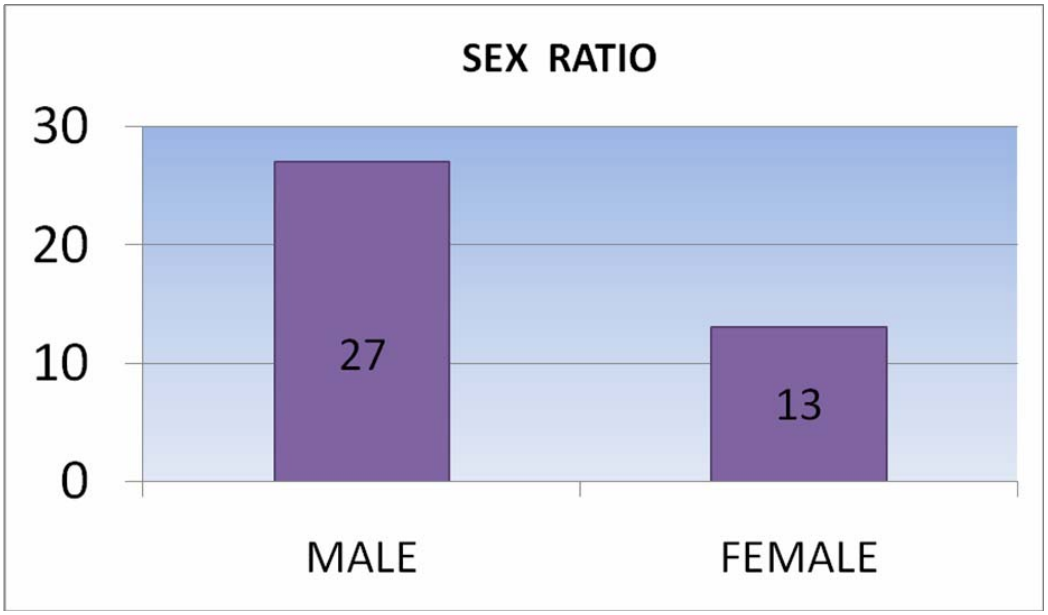
The maximum age - 71 years

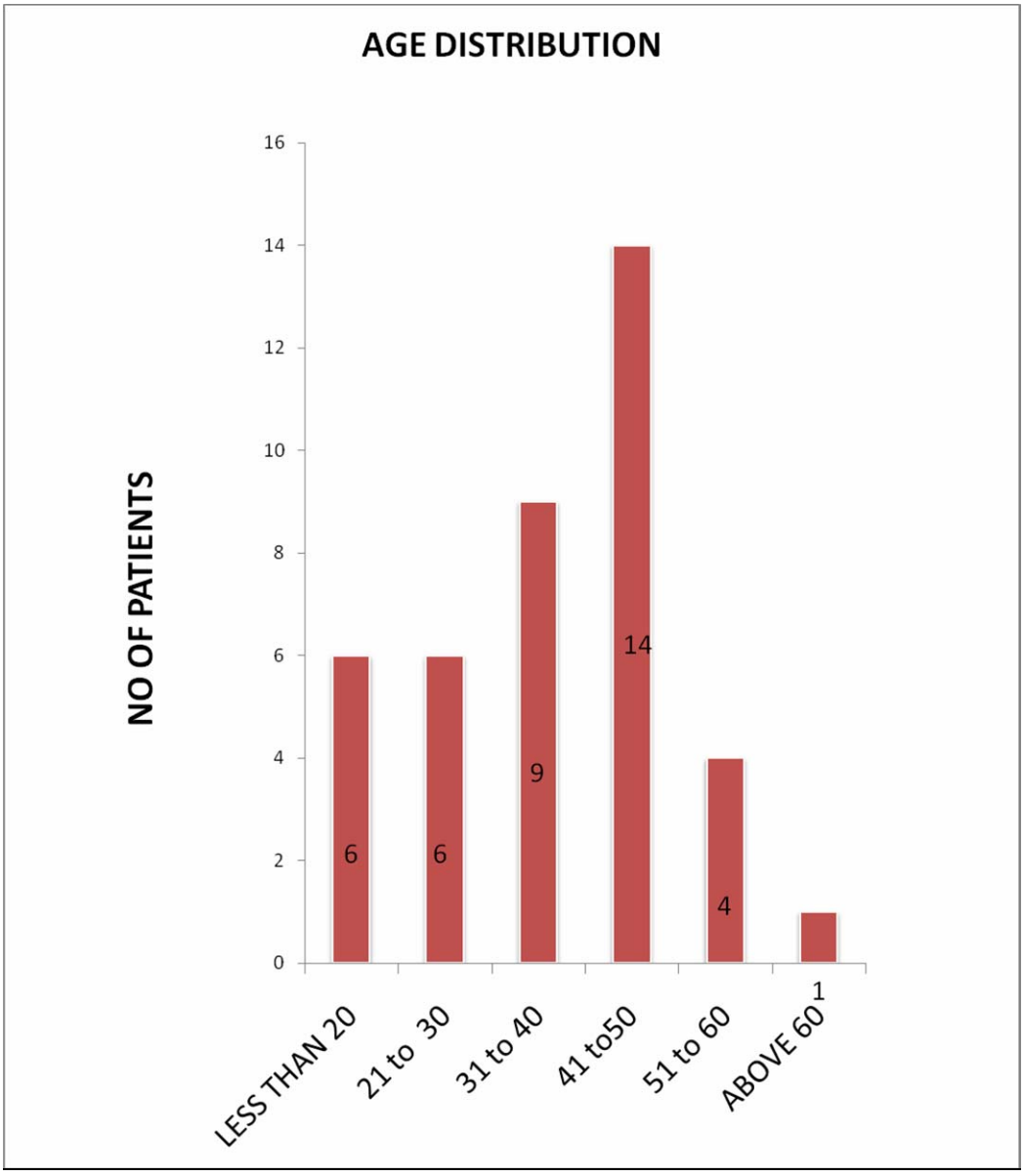
TABLE II : DISTRIBUTION OF GRADE OF FILARIAL LIMB

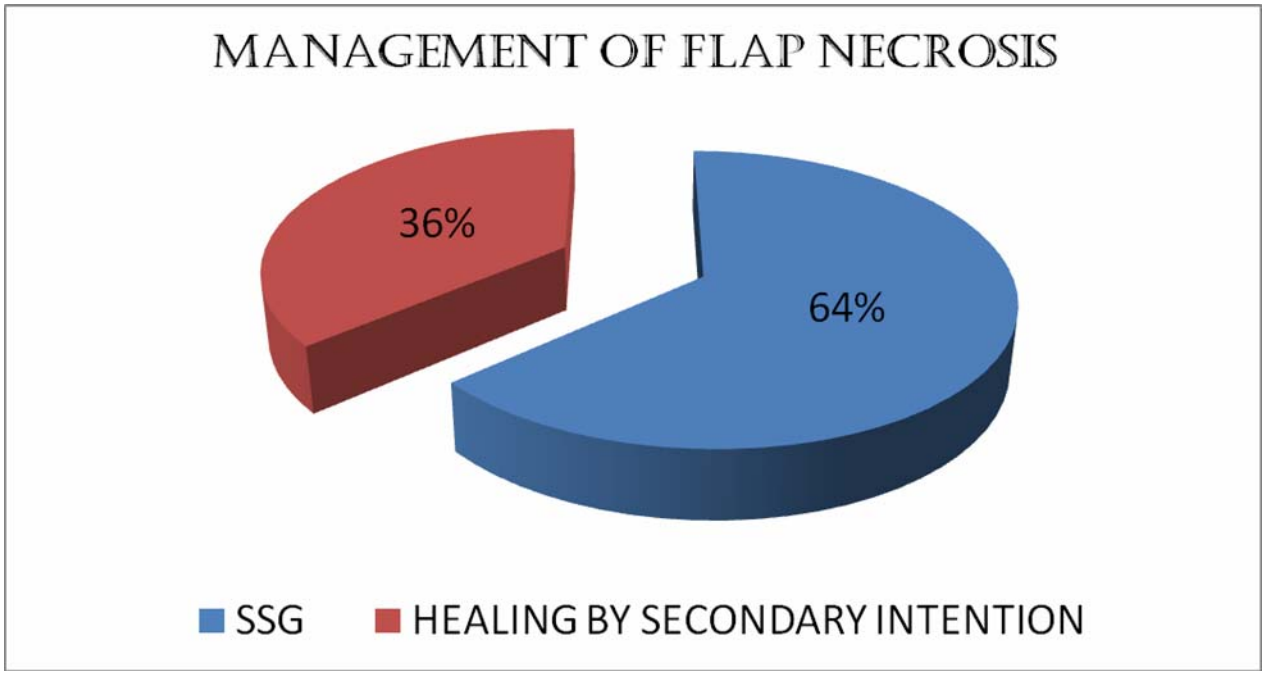
	FREQUENCY	PERCENT
GRADE III	18	45 %
GRADE IV	22	55%

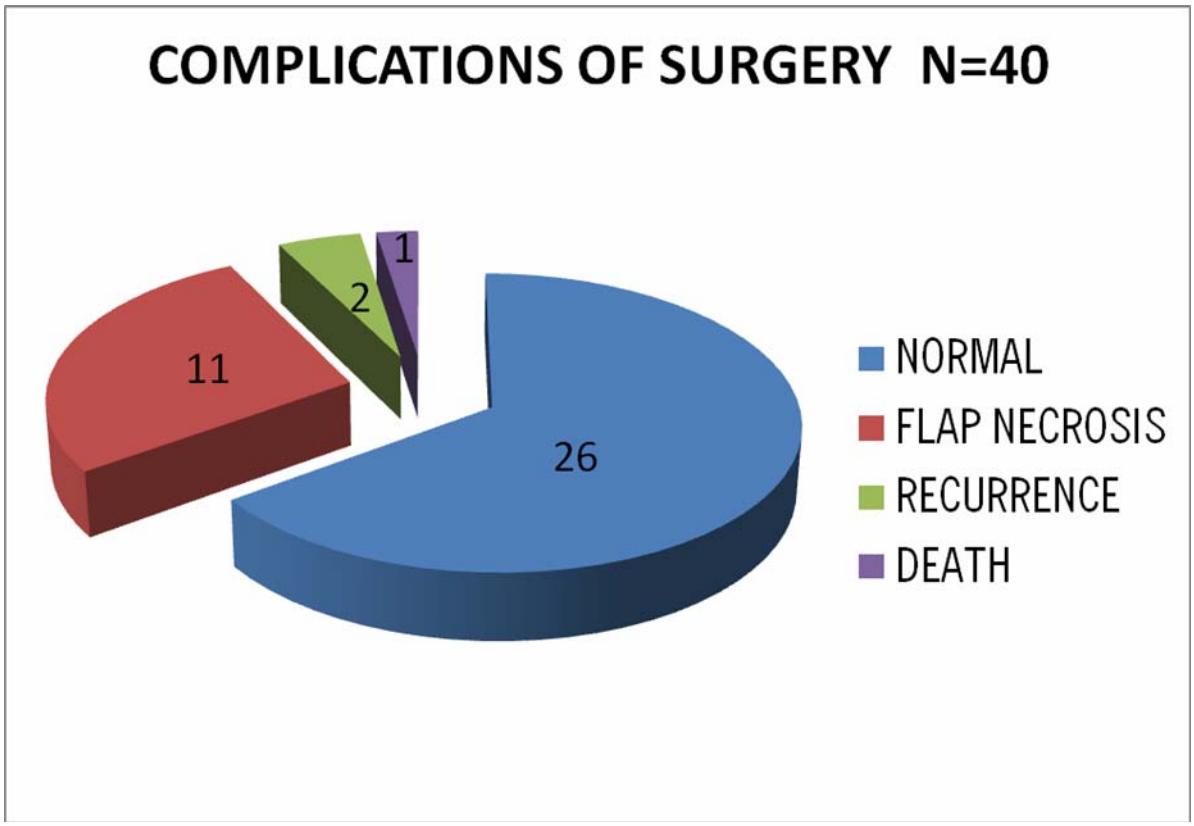
Number of patients in grade III - 18

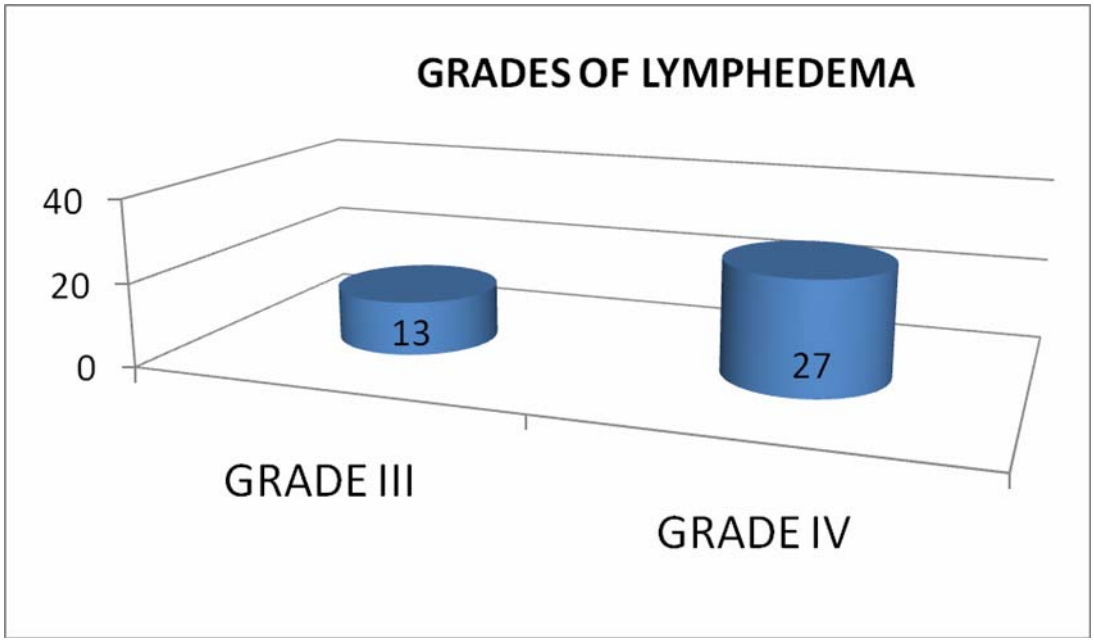
Number of grade IV patients - 22


SEX RATIO
30
20
10
0
27
13
MALE
FEMALE


AGE DISTRIBUTION
NO OF PATIENTS
16
14
12
10
8
6
4
2
0
6
6
9
14
4
1
LESS THAN 20
21 to 30
31 to 40
41 to50
51 to 60
ABOVE 60


MANAGEMENT OF FLAP NECROSIS
36%
64%
SSG
HEALING BY SECONDARY INTENTION


COMPLICATIONS OF SURGERY N=40
1
2
11
26
NORMAL
FLAP NECROSIS
RECURRENCE
DEATH


GRADES OF LYMPHEDEMA
40
20
0
13
27
GRADE III
GRADE IV

RESULTS OF STUDY

The patients who underwent reduction surgery had significant reduction in limb size.

The range of reduction was seen from 7 cm (15.2 % of preoperative limb size) to 29 cm (42% of preoperative limb size).

TABLE III : OUTCOME OF SURGERY

OUTCOME	FREQUENCY	PERCENT
SIGNIFICANT REDUCTION	26	65%
FLAP NECROSIS	11	27.5%
RECURRENCE	2	5%
DEATH	1	2%
	40	100%

The flap necrosis at the suture site occurred in **11** Patients

(27.5 %)

Of these the number of patients who underwent SSG fo resultant raw area is **7 (63.5 %)**

The number of patients whose raw area healed by secondary intention is **4** (**36.4 %)**

Recurrence is form of lymphedema is noted in **2** patients (**5 %**) in average of minimum 3 months to 2 years of follow up.

One patients **(2 %**) died in the postoperative period around second week due to Acute renal shutdown for which he was on dialysis.

TABLE IV : MANGEMENT OF COMPLICATIONS

	FREQUENCY	PERCENT
SSG	**7**	**63.5%**
SECONDARY INTENTIONAL HEALING	**4**	**36.4%**
TOTAL	**11**	**100%**

The mean standard deviation of pre op filarial limb size noted in study is **50.4 ± 11.84**

The mean post op limb size noted in study is **15.7 cm.**

The mean standard deviation of reduction noted in study is **35.37 ± 7.68.**

The significant 2 tailed result was < . 0005.

The mean hospital stay including management of complications was **34** days (23 – 45 days).

Descriptive Statistics

	N	Range	Minimum	Maximum	Mean	Std. Deviation
Normal	40	21.00	13.00	34.00	25.5250	5.0332
20 cm	40	49.00	30.00	79.00	50.4000	11.8469
Post reduction	40	30.00	22.00	52.00	35.3750	7.6793
Diff in CM	40	22.00	7.00	29.00	15.4250	5.7963
Diff in %	40	26.81	15.22	42.03	30.1419	6.3552
Valid N (listwise)	40					

T-Test

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	20 cm	50.4000	40	11.8469	1.8732
	Post reduction	35.3750	40	7.6793	1.2142

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	20 cm & Post reduction	40	.880	

Paired Samples Test

	PAIRED DIFFERENCE					t	df	Significance (2 Tailed)
	Mean	Std. Deviation	Std.Error Mean	99% confidence interval of the difference				
				Lower	Upper			
PAIR 1 20 Cm Post Reduction	15.0250	6.2583	0.9895	12.3455	17.7045	15.184	39	**<0.0005**

DISCUSSION

All the patients who underwent surgery had significant reduction in limb size compared to the pre surgical status.

Majority of the patients **(65%)** did not suffer complications with uneventful intra operative and post operative period .

All patients received atleast 1 unit of whole blood transfusion to a maximum of 3 units.

The flap necrosis was found to be commonest complication noted after the surgery as **27.5 %** of patients (11) who underwent surgery experienced it.

The necrotic flaps was managed by split thickness skin graft in 7 patients **(64 %)** and in the remaining 4 patients (**34%)** it was managed by secondary suturing in the next two weeks or allowed to heal by secondary intention.

The patients were followed up in the filarial clinic for a mean period from 3 months to 2 years and recurrence was noted in two patients in the form of lymphedema both of whom have not undertaken stocking.

One patient expired during dialysis for postoperative acute renal failure in the nephrology department.

CONCLUSION

Most of the current procedures done for debulking the filarial leg cannot be applied for huge legs.

Moreover patients suffer many complications like Flap necrosis (in common) and management of post reduction raw area.

The evaluation of patients undergoing our type of reduction showed significant reduction in size with fewer complications.

Smaller sized legs achieved significant reduction in single sitting whereas multiple sitting in one or two can achieve larger reduction even in very huge legs.

The functional and esthetic outcome is significantly better.

The complications of this procedure is easily managable.

Understanding the importance of thickness of flap and avoiding button hole has significantly reduced the necrosis of flap in later cases.

The functional outcome includes carrying daily activities, change of dress code, change in gait and respect in society.

BIBLIOGRAPHY

1. Berthwhistle A.P. & Gregg A.L. : Elephantiasis. *Br. J. Surg.* 16: 267, 1928.

2. Bihari I. & Meleg M. : Conservative management of lymphoedema of the limbs. *Orv. Hetil.* 132 : 1705, 1991.

3. Bosworth N. & Evert A. : Effect of streptococci on the persistence of Brugia malayi and on the production of elephantiasis in cats. *Int. J. parasit.* 5: 583, 1975.

4. Browse N. : Wolfe single procedure series 39. (REDUCING OPERATION FOR LYMPHOEDEMA OF THE LOWER LIMB). Wolfe Medical publications Ltd. (1986)

5. Calnan J.S & Pflugg I.I.: The normal anatomy of this lymphatic system in the human leg. *Br.J.Surg.* 58: 925, 1971.

6. Casley Smith J.R.:The medical treatment of lymphoedema. *Experientia* 15: 825, 1976.

7. Casley Smith J.R.: New trends in lymphoedema. Fine pore structure. *Experientia* 15: 818, 1976.

8. Casley Smith J.R., Jamal S. & Casley Smith J.R. : Reduction of filariatic lymphoedema and elephantiasis by 5,6 benzo-alpha-pyrone (coumarin), and the effects of diethylcarbamazine (DEC). *Ann. Trop. Med. Parasitol.* 87 : 257, 1992.

9. Clodius M., Foldi E., and Foldi M. : On non operative management of chronic lymphoedema. *Lymphology* 23: 2, 1990.

10. Clodius D.L., Piller N.B. & Casley Smith J.R. : The problems of lymphatic microsurgery in lymphoedema. *Lymphology* 14 : 69, 1981.

11. Clodius M., Foldi E., and Foldi M. : *Annals of Plastic Surgery* 22: 505-515, 1989.

12. Cordeiro A.K. and Baracat F.F.; Surgical classification of lymphoedema. Chapter in Progress in Lymphology. Ed. Mayall/

Witte. Plenum Press (1975).

13. Dandapat M.C., Mahapatro S.K., Mohanty S.S. and Patro S.K.: Management of the chronic manifestations of filariasis. *J.I.M.A.* 84: 210, 1986.

14. Dandapat M.C., Mukherjee L.M. & Patra S.K. : Evaluation of different surgical procedures in filarial lymphoedema of the lower extremity. *J. Ind. Med. Assoc.* 89 : 127, 1991.

15. Degni M. : Lymphatico-venous anastomosis. Chapter in Progress in lymphology. Ed. Mayall & White. Plenum Press (1977).

16. Gogia S.B. : Evaluation of Intermittent Pneumatic External Compression in Filarial lymphoedema of the extremeties. *THESIS For M.S.* (gen. surgery), All India Institute of Medical Sciences, New Delhi 1981.

17. Goldsmith H. : Omental transposition for the treatment of lymphoedema. *Rev. Surg.* 23: 303, 1966.

18. Homans J. : The treatment of elephantiasis of the legs; a preliminary report. *N. Eng. J. Med.* 215: 1099, 1936.

19. Jamal S. : From the proceedings of the 12th International Conference of Lymphatic pathology and Immunopathology in Filariasis. Thanjavur, India Nov.18-22, 1985.*Lymphology* 18: 148, 1985.

20. Jamal S. : Lymphovenous anastomosis in filarial lymphoedema. *Lymphology* 14 : 64, 1981.

21. Kromosodirdjo T.R. and Portono F. : Lymphangiography in Filariasis due to W. bancrofti in men. Chapter in Progress in Lymphology edited by Mayall/Witte, Plenum Press, (1977).

22. Krylov V.S., Milanov N.O., Abalmasov K.G., Sandrikov V.A. & Sadavnikov V.I. : Reconstructive surgery in treatment of lymphoedema of the extremities. *Int. Angiol.* 4 : 171, 1985.

23. Larson D.L., Coen C.R., Doyle J.E., Happerport.A.S., Kloen R. and

Lewis S.A. : Lymphoedema of the lower extremity. *Plast. Recons. Surgery.* 38: 293, 1966.

24. Lewis R. : Chapter on Lymphoedema in PLASTIC RECONSTRUCTIVE SURGERY; Vol. 7. 2nd edition. Ed. by CONVERSE (1975).

25. Madan N.C., Dhawan I.K., Gogia S.B. and Jai Narayan : A simplified treatment for lymphoedema. *Proc. VII Int. Conf. Plast. Surg.* Ontario, Canada :1644, 1983.

26. Miller J.A.: Surgical approach to lymphoedema. *Am.J.Surg.* 134 :191, 1977.

27. Nielobow Z. L. & Olszewski W.: Surgical relief of lymphoedema. *Abstr. World Med.* 1: 90, 1966.

28. Olszewski W.L. & Engeset A. : Intrinsic contractility of leg lymphatics in man. *Lymphology* 12 : 81, 1979.

29. Olszewski W.L. : Physiology and microsurgery for lymph vessels in man. *Lymphology* 14 : 44, 1981.

30. Olszewski J. : What is lymphology in 1991. *Lymphology* 25: 50, 1992.

31. Parks J.K. : Textbook of preventive and social medicine. Orient Longmans (1979).

32. Peer L.A., Shahgholi M., Walker J.C. & Ungars D.M. : Modified operation for lymphoedema of the extremities. *Plast. Reconstr. Surg.* 14 : 347, 1954

33. Sistrunk W.E. : Further experiences with Konodoleon operation for elephantiasis. *J. A. M. A.* 71 : 800 1918.

34. Thompson N. : Surgical treatment of chronic lymphoedema of the lower limb. *Br. Med. J* (2) : 1567, 1962.

35. Thompson N. : Surgical treatment of chronic lymphoedema of the extremities. *Surg. Clin. N. Am.* 47 : 445, 1967 .

36. Wolfel. R. A : Lymphaticovenous el R.A. lymphatico- venous communications. Am. J. Roentgen. 95 : 766, 1965.

CLINICAL FILARIAL RESEARCH UNIT.
FOLLOW - UP
THANJAVUR MEDICAL COLLEGE HOSPITAL, THANJAVUR - 4.

PROFORMA.

Name :
Address :
FIL No :
Age :
I.P. No :
Sex : M / F.
AD. No :
B.P. :
Dis On :
Diagnosis : Leg. Rt. Lt. Arm. Rt. Lt. Genital
Grade :
Duration :
Caries :
Fungus :
Adenitis :
Fever c Rigor :
Hydrocele :
Family History :
Married :
Children :
Others :

Swelling

Measurements :		Dt. Rt.	Lt.	Dt. Rt.	Lt.	Dt. Rt.	Lt.	Dt. Rt.	Lt.
Foot / Hand	: 10cm.								
Ankle / Wrist	: 12cm.								
Leg.	: 20cm.								
Thigh	: 60cm.								
Tonometry	:								
Volume	:								
Height	:								
Weight	:								

Operation : Rt. Lt. Date

Lymphangiogram :
Sculpturing :
N.V.S. done :
Skin Excision foot :
,, ,, leg :

S NO	GRADE	PT DETAILS	SIZE OF NORMAL LIMB AT 20 CM	PRE OP MEASUREMENT IN FILARIAL LEG AT 20 CM	POST OP SIZE OF LIMB	DIFFERENCE IN Cm	PERCENTAGE OF REDUCTION	COMPLICATIONS
1	III	NEELAMEGAM 50/M	28	40	32	8	20%	
2	III	MURUGESAN 45/M	26	40	32	8	20%	
3	III	SAMPATH 50/M	21	36	24	12	33%	
4	IV	MALARKODI 38/F	23	40	28.5	11.5	28%	
5	IV	RANGASAMY 58/M	30	56	36	20	35.70%	NECROSIS
6	III	NAINAMOHAMMED 24/M	44	38	25	13	34.20%	*EXPIRED*
7	IV	SAMPATH 40/M	27	54	40	14	25.90%	
8	IV	MURUGESAN 58/M	30	59	44	15	25.40%	
9	IV	KALIDASS 40/M	30	52	35	17	32.60%	
10	III	ANIL 17/M	19	31	22	9	29%	NECROSIS
11	IV	SELVI 15/F	13	40	25.5	14.5	36%	
12	III	SUSEELA 50/F	24	43	33	10	23.20%	
13	III	ROSAIYA 50/M	20	40	26	14	35.80%	
14	IV	VELAYUTHAM 24/M	30	63	45	18	28.50%	NECROSIS
15	IV	SHYAMALA 24/F	22	52	34	18	43.60%	NECROSIS
16	IV	FATHIMA 35/F	34	46	39	7	15.20%	
17	IV	GUNASEKARAN 35/M	22	40	30	10	25%	
18	IV	SATHYARAJ 19/M	17	36	25	11	30.50%	
19	IV	FAROOK 45/M	26	49	37	12	24.40%	
20	IV	MANOHAR 41/M	30	58	48	10	17.20%	
21	IV	MEGALA 31/F	26	79	52	27	34%	NECROSIS
22	IV	BALASUBRAMANI 52/M	22	60	35	25	41%	
23	IV	RAMALINGAM 71/M	32	47	33	14	29%	
24	III	FATHIMA 29/F	28	60	40	20	33.30%	
25	IV	AYEL KHAN 38/M	20	32	24	8	25%	NECROSIS
26	III	RAJENDIRAN 43/M	31	56	40	16	28.50%	
27	IV	VALLI 23/F	23	45	30	15	33.30%	
28	IV	SAKUNTHALA 46/F	30	57	47	10	17.50%	NECROSIS

29	IV	APPORVASAMY 40/M	24	63	40	23	36.50%	
30	IV	VIJI 40/M	27	70	42	28	40%	NECROSIS
31	III	ZAHEER 20/M	19	30	22	8	26.60%	
32	IV	SANGEETHA 44/F	28	46	32	14	30.40%	NECROSIS
33	IV	KUMARAVEL 55/M	22	52	39	13	25%	
34	III	VALARMATHI 43/F	26	39	29	10	25.60%	
35	IV	MARTIN 33/M	23	59	41	18	30.50%	
36	IV	MOHAN 40/M	34	69	45	24	34.70%	
37	IV	AMUDHAN 42/M	31	69	40	29	42%	NECROSIS
38	IV	SURTESH 20/M	20	48	30	18	37.50%	NECROSIS
39	III	RUBINI 18/F	28	54	35	19	35.10%	
40	III	VALLI 35/F	34	65	45	20	30.70%	