THE PRESENTATION AND CLINICAL SEVERITY GRADING IN MANAGEMENT OF CHRONIC VENOUS DISEASE AT KENYATTA NATIONAL HOSPITAL.

A DISSERTATION SUBMITTED IN PART FULFILMENT FOR THE DEGREE OF MASTERS OF MEDICINE IN SURGERY, UNIVERSITY OF NAIROBI.

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DECLARATION

This dissertation is my original work and has not been presented for a degree in any other University

Signed---------- Date---21/2/09---

DR. ANTHONY MUTHEE,

MBChB. (U.O.N),

Postgraduate Student in Surgery,

University Of Nairobi,

With my approval as a university supervisor,

Signed----------Date---22/2/2009---

MR. T.M. OMULO,

MBChB , MMed (U.O.N),

Consultant Cardiothoracic Surgeon,

Lecturer University of Nairobi,

Department of surgery,

P.O.BOX 19676,

Nairobi.

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DEDICATION

I dedicate this work to my parents the late Mr. Michael Muoki and Mrs. Helen Wangu Muoki my first teachers. My wife Mary, children Anita and Colin for bearing with my long absence during the study period.

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ABBREVIATIONS:

1. ABI or ABPI –ankle brachial pressure index.
2. BMI-body mass index (weight in kg/height in metres squared).
3. CVD- chronic venous disease.
4. CVI-chronic venous insufficiency.
5. CEAP – clinical, etiological, anatomical and pathophysiological classification.
6. DVI- deep venous insufficiency.
7. DVT- deep venous thrombosis.
8. KNH- Kenyatta national hospital.
9. LASV-lateral accessory saphenous vein.
10. LTFU- Lost to follow up.
11. LSV-Long saphenous vein.
12. MASV-medial accessory saphenous vein.
13. PAI – inhibitor plasmin activator(I &II)
14. PV-perforator vein(s).
15. SEPS—subfascial endoscopic perforator surgery.
16. SFJ-sapheno-femoral junction.
17. SPJ- sapheno-popliteal junction.
18. SSV-short saphenous vein.
19. US or U/S-ultrasound scan.
20. U.O.N-University of Nairobi.
21. VCSS-venous clinical severity score.
22. VDS-venous disability score.
23. VSDS-Venous Segmental Disease Score.

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SUMMARY

Chronic venous disease (CVD) describes a range of disorders usually of the lower limbs characterized by venous hypertension. Mild and moderate forms manifest as telangiectasia, reticular veins or varicose veins while severe forms present with skin changes or venous ulcers. These disorders are fairly common in the adult population. Symptoms of CVD vary and so do reasons for patients to report to health institutions. Lack of uniformity in diagnosis and classification of CVD has contributed to variability in reporting. CEAP (clinical, etiological, anatomical and pathophysiological) classification offers an effective and reproducible method of grading the disease. Epidemiological studies put prevalence rates for CVD at 10 to 35 percent. African studies in the 1970's showed low prevalence rates of between 0.12% and 7.7%. At Kenyatta National Hospital, a study on varicose veins by Cheruiyot (1995) showed an average of 28 patients treated annually.

There has been an apparent rise in numbers of patients with venous disease seen at KNH.

Management of CVD has moved from the domain of general surgeons to the cardiovascular surgeons. Despite this being a common problem, little has been reported from this hospital and the region.

In the current study, we looked at the pattern of presentation and management of chronic venous disease at Kenyatta National Hospital.

This is a prospective descriptive hospital based study.

Permission was obtained from the hospital's Ethics and Research committee. All patients with clinical features of CVD seen in the hospital, who met the inclusion criteria and gave consent during the study period, were enrolled. The patients were interviewed and examined by the researcher together with his assistant(s). Follow up examinations were carried out on the patients. Questionnaires were used to collect data which was then analyzed using SPSS computer program.

During the period January to August 2008 one hundred and forty patients were recruited into the study. Sixty four had bilateral disease giving a total of 204 legs. Over eighty percent were from Nairobi and the neighbouring provinces (Central, Eastern). Eighty percent lived in urban centers and 20% were from rural areas. Fifty three percent were between 30 and 50 years. Females were 75 in number and males were 65(1.15: 1.00). Our patients had a mean weight of 74 kg, a mean height of 167cm and a mean BMI of 26.8. Females had a relatively high BMI ($28kg/m^2$) compared to males ($25kg/m^2$). The commonest presenting complaint

was varicose veins followed by pain and oedema. The mean duration for symptoms was nine years. On average patients took 34 months before presenting to cardiovascular surgical clinic after the initial diagnosis. All patients were evaluated clinically and by radiological examination. The baseline CEAP clinical class and venous clinical severity score were recorded. Thirty two patients (22.9%) underwent surgery (ligation and stripping of truncal varices, subfascial ligation of perforators, or avulsion of isolated varices) and 99(71%) were managed conservatively. Nine patients (6.4%) did not start any form of treatment. Surgery was associated with greater improvement in symptom severity score compared to conservative treatment. Thirty one patients (22.1%) interrupted their treatment due to different reasons.

CVD cases are on the rise in our hospital. Majority of patients are from urban areas pointing to the possibility of CVD manifestation being linked to lifestyle. CEAP classification and VCSS are useful tools in grading venous disease. Introduction of dedicated venous ulcer clinics would help in handling the big number of patients.

INTRODUCTION

Definition

Chronic venous disease describes disorders of the venous system which largely affect the lower limbs as a result of venous hypertension. Chronic venous insufficiency (CVI) commonly refers to venous disease of greater severity and is associated with skin changes or ulceration (**CEAP** Clinical classes 4-6) [1]. A degree of valvular failure or insufficiency is present in all stages of chronic venous disease. The term CVI is used by a few to refer to all stages of this disease [2]. Varicose veins are elongated, dilated and tortuous veins. These may be associated with superficial or deep vein insufficiency.

ANATOMY AND PHYSIOLOGY

Veins have **valves** which direct blood flow towards the heart. The vena cava and its tributaries lack valves (exception; gonadal veins). Integrity of the vein wall prevents dilatation and consequent valvular incompetence. Endothelial lining provides a non thrombogenic surface. Some endothelial cells produce plasminogen activator. Damage as a result of stasis, hypoxia or leukocyte derived cytokines enhance thrombogenesis [4].
Peripheral veins are grouped into three. **Superficial** veins are superficial to the deep fascia, and **deep** veins lie deep to it. **Communicating** veins or **perforators** join the two systems of veins (*see figure* 1.) Venous sinuses within muscles are part of the pumping mechanism.
Venous pressure and flow depends on three factors;

(a) Arterial pressure across the capillary bed,

(b) Muscular pump pressure and,

(c) Gravitational force.

VENOUS PUMPS

During walking or muscle contraction, the deep veins and sinuses are compressed within muscle compartments. Valves direct blood flow cephalad. The foot, calf and thigh muscles constitute the peripheral pump. Venous pressure falls during exercise (~20mmHg) and rises moderately at rest (~90mmHg). Damage to joints, muscles or valves leads to stasis and

therefore no reduction in pressure. Obstruction in the deep system causes venous hypertension during exercises [4].

VEIN SYSTEMS

Superficial veins act as conduits collecting blood from the surface and draining via perforators into the deep system. The two main superficial system of veins in the lower limbs are; the **long saphenous vein (LSV),** and the **short saphenous vein (SSV).** Long or great saphenous vein drains the inner aspect of the leg to the superficial femoral vein (SFV). The short or small saphenous vein drains the back of the calf to the popliteal vein.

The anatomy of veins of the lower limbs is quite varied compared to arteries. Mullarky in 1965 reported 27 venous anomalies in 14 out of 65 cadaver dissections [5].

LSV is formed by the confluence of **medial marginal vein** and **internal malleolar vein.** The vein runs upwards a few centimetres medial and parallel to medial border of the tibia. The position of this vein is fairly constant at the ankle. In about 5% of people the common trunk of LSV is not formed at the land mark point in the ankle. The **posterior arch vein of Leonardo** is a frequent tributary on the posteromedial aspect. This tributary is linked to the deep system by at least 2 ankle perforators (**Linton**). It also receives a fan shaped group of small veins from the heel region. These present as the ankle flare or corona phlebectatica in case of perforator incompetence. **Superficial anterior vein** joins the LSV at the same level as the posterior arch vein. Its origin is 8-10cm below the inferomedial margin of the patella. This is often the site of the first varices. There are two long tributaries in the thigh, **anterolateral vein** and **posteromedial vein (lateral and medial accessory saphenous veins** respectively). **Lateral accessory SV** runs from the lateral knee and joins the LSV at the groin. At times this tributary bypasses the LSV to empty into veins on the abdominal wall. **Posteromedial vein (medial accessory SV)** joins LSV at the same level as the lateral tributary. Sometimes it bypasses this junction to join a gluteal vein. Ligation of the accessory vessels instead of the LSV is one of the causes of recurrence of varicose veins.

The oval fossa is 2-3cm below the border between the middle and medial thirds on the inguinal ligament. It is 1-2cm medial to the femoral artery pulsation.

At the fossa 4 tributaries join the LSV; **superficial circumflex iliac, superficial epigastric, superficial and deep external pudendal veins**. There may be other smaller tributaries to the greater saphenous at the fossa. In 20% of all legs there are less than five veins while in 65% there are 5-7 tributaries. About 15% of legs have more than seven tributaries [5].

Duplication of the great saphenous vein occurs in about 2-3% of all legs [5]. In other cases one or more of regular tributaries empty into the femoral vein. Commonly the superficial circumflex iliac vein (15%). Incision at the fossa should be at least 10 cm long and the saphenous vein ligated high or flush with the femoral vein.

The **short saphenous vein (SSV)** drains the lateral aspect of the foot and passes upward between the lateral malleolus and tendoachilles. A perforator joins it at a non-specific point between the malleolar tip and 8cm above it. SSV drains into the popliteal vein 3-7cm above the knee fold. It may empty into a tributary of the long saphenous vein or the deep femoral vein (about a third of cases). A number of connections exist between the SSV and the LSV [5, 6].

Perforators

There are over sixty sites of communication in addition to the main drainage points. Direct perforators connect directly to the deep veins. The indirect ones communicate with the deep system via venous sinusoids inside muscles. Direct perforators are found at fairly fixed locations. Valves in these vessels direct flow from superficial to deep system. Foot perforators however miss valves and can allow flow in either direction. Obstruction of the deep veins is associated with venous ankle flare (corona phlebectatica).

Perforators between the LSV and the deep veins include; one just below the medial malleolus and another 10cm above it. About the mid-calf there is one site of communication, others just distal to the knee and in the distal thigh. Lower perforators may join the posterior arch vein before it drains into the LSV. Ankle perforators are named **May** or **Kuster**. Medial ankle perforators (**Linton** or **Cockett)** just above the ankle connect posterior arch vein with posterior tibial vein. Another important perforator is at the upper part of the calf between the two heads of gastrocnemius (10cm below the knee fold – **Boyd** or **Gastrocnemius** perforator). In the thigh, there is a group of 3 to 4 veins that run in the Hunter's canal. A stripper passed proximally in the LSV may pass into femoral vein through one of these perforators. The **Dodd's** perforator joins the medial vein of thigh to the femoral vein.

Deep venous system corresponds with the arterial system. Each artery is accompanied by a vein or two bearing similar names [6, 7].

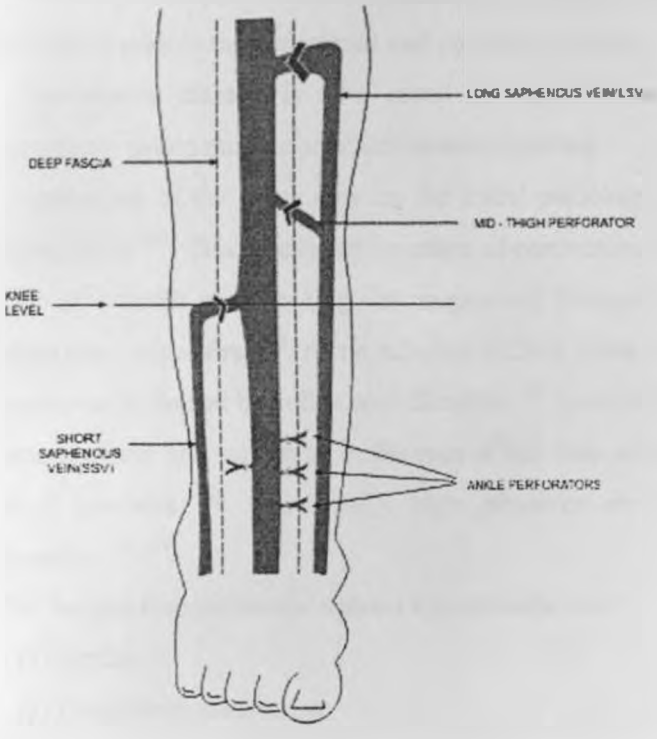


Figure 1-Venous systems(adapted from 'The pathology and surgery of the veins of the lower limb' by Dodd, H and Cockett, Longman group Ltd. 2nd edition[3])

ETIOLOGY AND PATHOLOGY OF CHRONIC VENOUS INSUFFICIENCY:

Primary chronic venous insufficiency is due to valvular failure of unknown aetiology that affects mainly the superficial and perforator systems.

Secondary disease is as a result of valvular failure after deep venous thrombosis, pressure by pelvic masses or arteriovenous shunting.

Dilatation of the veins may be the initial pathology and valvular incompetence being a consequence [8]. This is evident in cases of connective tissue disorders or hormone induced atony of smooth muscles (e.g. in pregnancy). Congenital aplasia or hypoplasia of venous valves may occur first [5]. Some scholars believe valve failure in superficial veins is the first occurrence followed by reflux and dilatation [9]. Less frequently extramural compression may cause outflow obstruction as in the case of left iliac vein compression by right iliac artery or pelvic tumours [10]. Chronically high pressures are associated with skin changes and ulceration [7, 11].

The factors responsible for venous hypertension are;

(1) Reflux,

(2) Outflow obstruction or,

(3) Failure of the calf muscle pump in obesity or leg immobility.

Smooth muscle in parts of the vein exposed to high pressures hypertrophy while low pressure areas show atrophy. The muscle layer is progressively replaced by collagenous connective tissue. Eventually the sheath of collagen is replaced by broad strips of hyaline material. These sclerotic changes may involve any of the three layers of the venous wall. Superficial leg veins have well developed bundles of longitudinal muscle fibres in the adventitia. This layer is involved early in fibrosis. Parts of the intima under high pressures develop thickened plaques of soft mesenchymal tissue (may be absent in varicosis) [12]. As wall fibrosis becomes severe the valves begin to thicken and to have deposits of collagen. Thickened valves are stiff and incompetent.

Risk factors in CVD

1. Hereditary defects in the vein wall [5, 11].

2. Age. Incidence rises with age and peaks at 60-80 years [13, 14, 15].

3. Sedentary lifestyle with reduced peripheral pump action and occupations which involve standing or sitting down for long hours [16, 17].

4. Increased height in males [16].

5. History of previous vein surgery [16]

6. Systolic hypertension was associated with higher risk in the Framingham study[17].

8. Trauma to the lower limbs. Nearly 30% of lower limb fractures develop venous thrombosis (DVT) and post thrombotic syndrome [18].

10. History of DVT [18].

11. Female sex and pregnancy [16, 19]. In a study by Sumner et al (1981), CVD was found in 10-20% of pregnant women. They found that the diameter of competent and incompetent veins increases during pregnancy and recedes postpartum. Mashiah et al (1999) demonstrated increased Estrogen receptors in dilated vein segments [19, 20].

12. Obesity; in a study by Gudmundur D, Bo Eklof et al. BMI>25 was associated with higher incidence of CVD and skin changes [21].

13. Compressive pelvic tumours [22].

Venous ulcers:

About ninety percent of chronic leg ulcers result from **venous insufficiency**. About 4-5 % are due to **arterial insufficiency.** Both arterial and venous diseases coexist in **4-5%** while **other causes** of leg ulcers account for **1-2%** only [23]. The cause of ulceration is ambulatory venous hypertension. The mechanism by which venous hypertension causes this, is not well understood. A number of theories have been proposed:

1. **Fibrin cuff theory**

A fibrin cuff around capillaries in CVI acts as a barrier to oxygen and nutrient diffusion. Elevated plasma fibrinogen levels plus increased capillary permeability lead to high fibrinogen levels in lymph. Impaired fibrinolysis has been reported in various studies. The cause for this appears to be an increase in the inhibitor plasminogen activator-1(PAI-1). PAI-1 is produced by endothelium and smooth muscle cells of the microvasculature. The release is triggered by mechanical stress from venous hypertension, or hypoxia. The levels are markedly elevated in lipodermatosclerosis. This rise in PAI-1 is strongly related to rheological and fibrinolytic disorders of CVI [24, 25, 26, 27].

2. **White cell rheology (plugging)**

Venous hypertension reduces capillary blood flow thereby leaving lines of trapped leukocytes. These white cells attach to endothelium and become activated. The result is release of free radicals, proteolytic enzymes and cytokines. These lead to tissue damage and

ulceration. Immunohistochemical studies show that capillaries of the papillary plexus are surrounded by an infiltrate of inflammatory cells (macrophages, T-lymphocytes) and a fibrin cuff[28,29].

3. **Macromolecules theory**

Leakage of fibrinogen and alpha-2 macroglobulins into the dermis secondary to venous hypertension and endothelial injury, in effect trap growth factors and other proteins. This trapping denies the affected region raw materials for integrity and repair of tissues [23, 30].

4. **Microvascular ischaemia**

In chronic venous insufficiency, there is a reduction in the number of skin capillaries. Venous ulceration is probably as a result of ischaemia [31, 32].

Skin changes in CVD may be explained by the observation of leukocyte depletion in blood returning from dependent feet (40-60 minutes). This is largely due to adhesion to the endothelium as well as migration through the wall in small vessels [33].

LITERATURE REVIEW:

CVD is a common condition but is generally ignored in terms of health priority. In a cross-sectional study of a random sample of 1566 subjects aged 18-64years in Edinburgh, telangiectasia and reticular veins were found in 80% of males and 85% of females, varicose veins in 40% males and 16% females. Ankle edema was found in 7% males and 16% females. Active or healed leg ulcers occurred in about 1% of the group [16, 26]. In the Framingham study, incidence per year for chronic venous insufficiency (CVI) was 2.6% in women and 1.9% in men [17].

Epidemiological studies put the prevalence of CVI at 10-35 per cent [34]. The Basle study (1967) involved 4376 workers of Basle chemical industries who were examined for peripheral arterial, venous and coronary heart diseases. CVI was found in 22% of the population (19% males and 25% females)[5]. Prevalence in African studies ranged from 0.12% in Ugandan adults to 7.7% of adult females in Transkei [4, 35, 36]. In a study on varicose veins in Africans by Richardson JB and Michael Dixon, 1259 Tanzanians in a provincial town (Moshi) were interviewed. Only 5.5% had varicose veins (6.1% males, 5% females). No relationship was found between varicose veins and age or the number of children. Pregnancy contributed but was not the principal factor. Diet and constipation seemed to be a factor [37]. A more recent study by Cheruiyot [38] showed a small number of patients at our referral hospital (KNH). In this seven year retrospective study, 252 lower limbs in 193 patients were evaluated. Twenty eight patients were treated for varicose veins annually. Over half (57%) of the group were between 21 and 40 years. Males were more than females with a ratio of 1.61: 1. Those with parity of 5 and above made up 54.1% of the females group [38].

In the San Diego population study (1994-1998), prevalence increased with age, non-Hispanic whites were affected more and trophic skin changes were seen in 6.2% of the population above 40 years [14]. Superficial reflux and visible signs were commoner in females while deep vein insufficiency was more prevalent in males. The long term effects are many and the cost to the patient and to the national health budget is enormous. In United States, the cost of managing venous ulcers is greater than $ 1 billion yearly [39]. In the UK, the annual cost is above $720 million [40].

Different studies show different prevalence rates. This is due to true geographical variation in the incidence and also differences in diagnosis and classification of chronic venous diseases [34].

CLINICAL FEATURES

Symptoms of chronic venous disease as reported in the literature include; Aching of the legs, heaviness, tingling, itchiness, numbness, restlessness or bursting pain , pigmentation, swelling, dilatation of superficial veins, skin changes or ulceration[4,40,42].

In a study at Kenyatta National hospital (1995), the commonest symptom was varicose veins (46%), followed by pain (20%), leg swelling (14%), ulceration (9%) and itchiness (5%) [38]. Signs elicited may include; telangiectasia, reticular or varicose veins, oedema, skin changes (dark brown discoloration, eczema) or frank ulceration. In the Edinburgh vein study telangiectasia and reticular veins were found in over 80% of the adult population. Varicose veins occurred in 16-40%, oedema in 7-16%, while active and healed ulcers were found in one percent [16]. In San Diego the prevalence of oedema was high at 48.2% in those with trophic skin changes. Trophic changes were found in 6.2% of those above 40 years [14]. A defect at the sapheno-femoral junction (SFJ) presents with varicosis in the upper thigh which progresses distally. Lipodermatosclerosis is noticeable in long standing cases. A positive fluid thrill and cough impulse may be elicited by placing the hand on prominent varices or at the saphenofemoral junction. A continuous palpable thrill is a feature of arteriovenous malformation. Difference in temperature may be noted when comparing the two legs or the foot and the upper leg [4, 7, 40, 42]. In a review by John J Bergan et al clinical signs were classified into two; Major signs -Varicose veins and venous ulcers.

Minor signs - Oedema, venous eczema, hyper pigmentation, atrophie blanche or lipodermatosclerosis [1].

Patients may present first with complications which include;

1. Bleeding (spontaneous or after trauma).
2. Superficial thrombophlebitis- sterile inflammation of vein wall due to local thrombosis.
3. Venous hypertensive skin changes (a) lipodermatosclerosis (fibrosis of subcutaneous tissues). (b) Skin pigmentation due to haemosiderin deposits. (c) Ulceration or white areas of scarring (atrophie blanche).
   (d) Eczema [4, 15].

Venous ulcers characteristically occur on the gaiter area (over the medial malleolus). They are single or multiple and may involve the whole circumference. Their margins are irregular, have a flat base and steep borders. The bed is shallow with granulation tissue and some

fibrinous material. Necrosis or exposure of tendons is rare except in association with arterial insufficiency [4, 34, 42].

Symptoms of CVD are mild compared to arterial disease. The two conditions may coexist in 20-25% of chronic leg ulcers [34].

In the San Diego population study, visible disease (varicose veins, trophic changes) and functional disease (superficial or deep reflux) were concordant in 92% and discordant in 8% of the limbs studied [114].

Prevalence of trophic changes in the same study was 8% in males and 5% in females. 25.9% of limbs had skin changes with no functional abnormality by Duplex U/S.

Deep reflux is associated with increased clinical severity. DVT is rare among the Chinese; mixed superficial and deep reflux was seen in 70-80 percent in a study by Chiwai Ting and others [68]. Superficial reflux was thought to cause deep vein incompetence by causing an overload of the deep system [68].

DIAGNOSIS

The visual appearance of the lower extremities is useful but not always a reliable guide to the peripheral venous condition. Clinical signs of venous disease are common to many other disorders of the lower limb. Imaging tests are necessary to rule out deep venous obstruction, assess paths of reflux, and to guide in the treatment plan. The Trendelenburg test is traditionally part of the physical examination and may be helpful in making the differential diagnosis. Three quarters of patients with CVD and ulceration can be diagnosed by clinical methods alone. A quarter of cases are of mixed characteristics [34, 41]. The ankle-brachial pressure index (ABI) is used to detect those with arterial insufficiency. Normal ABI is greater or equal to one. ABI<0.90 signifies peripheral arterial disease. If less than 0.7 compressive bandages may be harmful. In the elderly or diabetics a falsely high ABI may be recorded due to atherosclerosis. Transcutaneous oxygen measurement is used to evaluate arterial flow in these patients [41]. Colour duplex U/S is diagnostic in almost all cases. It is accurate, reproducible and non-invasive. It provides anatomic and functional information on both arteries and veins. Photo and air plethysmography are used in assessment of venous haemodynamics of the whole limb. Venography is useful when planning for surgery particularly repeats or for complex malformations. Plain X-ray imaging is used if bone involvement is likely. Ulcers older than three months require biopsy to rule out malignancy or atypical infections [34, 41, 42].

CLASSIFICATION AND GRADING OF CVD

An Ad Hoc committee of the American Venous Forum produced a consensus document for grading and classification of chronic venous disease. The **CEAP** classification (**Porter & Moneta**1995) is based on **C**linical signs, **E**tiology, **A**natomic distribution and **P**athophysiologic condition. Clinical part of CEAP classification is the most important in day to day patient care and communication among health professionals. Disease severity can be assessed using the CEAP disability score or the CEAP clinical score [42, 43].

Venous Clinical Severity Score (VCSS) is an analogue of CEAP clinical score. It is an effective tool in assessing disease severity before, during and after treatment. In an evaluation of validity and reliability of VCSS as a measure of disease severity, Meissner et al found little inter observer differences and good correlation with CEAP clinical classification [44].

An observational study to validate three components of a new venous severity scoring system(VCSS, Venous Segmental Disease Score(VSDS), CEAP clinical score) by Kakkos et al, all the 3 components showed a clear linear association with CEAP clinical score. A good correlation was observed among all 3 severity scores particularly between CEAP clinical class and VCSS ($p=0.001$). VCSS correlated with CEAP clinical class and also VDS ($p<0.001$)

There was however weak correlation between VSDS and VCSS, VDS.

Percent change for VCSS was significantly greater than corresponding change in CEAP clinical class. They concluded that VCSS and CEAP Clinical Score were equally sensitive and significantly better for measuring changes in response to superficial venous surgery than the CEAP clinical class [45].

IMAGING TESTS:

Doppler U\S scan.

Bidirectional Doppler can detect augmented flow through the veins. Hand held probe is used to show patency, increased flow and reflux. Hand Held Doppler (HHD) can also be used in measurement of ABI in patients suspected to have arterial disease [42,47]. Training of surgeons on how to use HHD may help compliment clinical examination in localizing the points of reflux [47].

Duplex scan

This method utilizes B-mode imaging for veins and arteries together with Doppler. Colour imaging is used to demonstrate patency, the state of the valves and evidence of previous thrombosis/scarring in the deep veins. Scanning is done with the patient standing and facing the examiner, or in 15 degrees reverse Trendelenburg position. Valsalva manoeuvre is used to demonstrate reflux. Reflux of more than 0.5 seconds is considered significant. Duplex scan is preferred in many centres because it offers good anatomical definition and is non invasive [42, 47]. It is currently regarded as the gold standard in CVD imaging [34, 42]. This mode of investigation has been used in a number of large scale epidemiological studies to diagnose and correlate clinical findings with functional disease [14, 16].

Venography

Venography is an invasive procedure whose role has been overtaken by Duplex U/S. It was previously regarded as the "gold standard" to test the accuracy of new investigation methods. In ascending Venography a non-ionic contrast medium is injected into a dorsal foot vein with a tourniquet tied around the ankle. Deep veins and their patency, perforator veins and the presence of reflux can be demonstrated. Ascending phlebography is useful in detecting venous obstruction. Descending phlebography is used to demonstrate reflux in both deep and superficial systems [42, 46].

Varicography

Contrast medium is injected into a varix. This shows anatomic connections of the varix and can define pattern in venous anomalies [42].

Plethysmography

Measurement of the leg volume is used to demonstrate the amount and speed of blood flow. Selective occlusion is then used to map out the diseased vessel. Three modes exist;

photoplethysmography, strain-gauge and air plethysmography. These methods are used in haemodynamic studies of the entire limb [34, 42].

Ambulatory venous pressure

This is an invasive procedure. It involves measuring venous pressure with a needle inside a dorsal vein of the foot. Recordings are made at rest and during standard exercises (10 tiptoe movements or knee bends). Exercises are then repeated with an inflated cuff at the ankle. The test is useful in haemodynamic studies [42].

Magnetic Resonance Venography or spiral CT scan can be used to define complex malformations. These are expensive tests and are not used routinely [47].

In a previous study venography was the most commonly used radiological test in Kenyatta National hospital followed by Doppler U/S [38].

TREATMENT:

The goals of treatment in a patient with chronic venous disease are;

1. Reduction of oedema
2. Minimizing pain
3. Reversal of skin changes
4. Ulcer healing and
5. Prevention of ulcer recurrence.

These can be achieved through bed rest, limb elevation (30minutes 3-4 times a day) and compression therapy. Injection sclerotherapy and surgery are used to ablate dilated veins and reduce recurrence. Drugs have been used though sparingly to reduce the effect of venous hypertension. The mode selected amongst other considerations depends on the size of varices, extent of the disease and symptomatology. Exercise and weight control helps in reduction of venous stasis. Patients whose careers involve standing or sitting for long hours may need to change their occupation [4, 34, 47].

Graduated compression is the cornerstone of ulcer treatment. Adherence to this mode of treatment is associated with improved ulcer healing rate and continued use prevents recurrence. Optimal pressures to stop capillary exudation at the ankle are 35-40 mmHg [34, 48]. Care should be exercised in patients with chronic heart failure or arterial insufficiency. Compression bandages are either elastic or inelastic. Rigid bandage like the Unna boot (zinc oxide impregnated paste) offer limited pressure at rest but high pressure with muscle contraction (high working pressures). Elastic bandages accommodate volume change providing both resting and working pressures.

Multilayer bandages give higher and sustained pressures than single layer. Multilayer bandages are more expensive than single layer but they promote faster wound healing. Samson R.H and Showalter D.P (1996) reported recurrence in 79% of non-compliant patients and only 4% in those who continued to use compression therapy. One of the main causes of noncompliance in that study was the high cost of compressive stockings [48, 49, 50].

In another study by Mayberry JC, Moneta GL et al recurrence at 36 months was 100% for non adherent patients as compared to 15% in those who used compression treatment [51]. Multilayered bandages were able to maintain higher pressures and faster healing than single layer [52]. Barwell and colleagues demonstrated that the rates of ulcer healing at 6 months

were similar for compression plus surgery and compression alone. However recurrence rate after surgery was lower [53].

Graduated external compression is used to minimize or reverse skin changes. Medical hosiery or stockings with graduated pressures are classified into three;

Class	Pressure	Indications
I	14-17mmHg	Superficial or early varices.
II	18-24mmHg	Medium severity varices, ulcers, mild oedema.
III	25-35mmHg	Gross varices, post thrombotic syndrome [54].

In patients with active ulceration bandages are preferred to stockings due to the ease of application.

The pressure developed beneath any bandage is determined by; (i) tension in the fabric, (ii) radius of the limb and (iii) number of layers applied. A bandage with a 50% overlap produces two layers of fabric, with pressure twice that produced by a single layer.

Sub-bandage pressure may be calculated using a formula derived from the Laplace equation as follows:

$$P = (TN \times 4630) / CW$$

P= Pressure (in mmHg)

T= bandage tension (in kgf)

C = circumference of the limb (in cm)

W = bandage width (in cm)

N = number of layers applied [54]

A bandage applied with constant tension to a limb of normal proportions will produce highest pressure at the ankle [54].

Comparison of four layer bandage system with the traditional adhesive plaster showed that the former produces higher pressures at the ankle (42.5mmHg) than adhesive plaster (29.8mmHg). Pressure was maintained for a week with the four layers, while in the latter, it dropped to 10.4 mmHg in a day [48]. Compression should be maintained until the ulcer heals.

Subsequently patients should wear graded compression stockings to prevent recurrence [55]. Assessment of arterial adequacy by feeling for distal pulses or measuring ankle-brachial-pressure index (ABI) is essential before compressive therapy.

A review of 22 trials through Cochrane library database, compression therapy was associated with accelerated ulcer healing. Elastic bandages were more effective than the non elastic ones [52]. Tension beneath bandages varies between practitioners. New types like Setopress come with geometrical designs printed at intervals along their length. These designs change shape when the bandage is stretched to predetermined level of tension. Setopress has 2 series of rectangles (green, brown). Green rectangle becomes square at 70% extension and the brown ones changes at a 100% extension. The level of extension gives predetermined pressure depending on the limb circumference [54].

Bandage Classification.

Bandages are broadly classified into 3 types; Type 1 (lightweight conforming bandages) are low pressure bandages that conform well to a limb or joint. Type 2(short stretch bandages) of the crepe variety are used in prevention of oedema, management of mild sprains, and sometimes in management of venous leg ulcers. Type 3(compression bandages) are used for deliberate application of pressure in controlling oedema. They are sub classified into 4; (i) Light compression (20mmHg) for superficial varices like those associated with pregnancy. Varices in pregnancy usually recover post partum and compression therapy is all that is needed. (ii) Moderate compression (30mmHg) for varicosis of pregnancy, moderately severe varices, prevention and treatment of ulcers.(iii) High compression (40mmHg) treatment of gross varices, postthrombotic venous insufficiency and management of leg ulcers and gross oedema.(iv) Extra-high performance compression(=>50mmHg). This maintains high pressures in the large or oedematous limb for extended periods [54].

DRUGS.

(1)**Oxpentifylline**, a methylxanthine derivative works via 3 modes; antifibrinolytic activity, reduction in leukocyte adherence to endothelium and antithrombotic activity. Studies show there is a higher cure rate of venous ulcers in patients on this drug plus compression compared to a placebo[56,57].(2) **Diosmin450/hesperidin50(Daflon500)** is a phlebotropic drug thought to act by reducing distensibility and venous stasis[58]. (3) **Acetylsalicylic acid (Aspirin)** given at 300mg a day together with compression bandage, improves the rate of ulcer healing [59, 60]. (4) **Stanozolol** is an androgenic steroid with fibrinolytic properties. It reduces pain and induration around the ulcer but does not speed healing [61]. (5) **Calcium Dobesilate** acts by reducing capillary hyper permeability induced by histamine or bradykinin. Increases red cell membrane flexibility and reduces platelet aggregation [62]. It is effective in improving some CVI symptoms e.g. pain and swelling.

GROWTH FACTORS

Granulocyte-macrophage colony stimulating factor improves healing of venous and other types of leg ulcers. Intralesional injections (painful) or topical applications have been used with higher healing rates [63]. **Keratinocyte growth factor 2** is a member of fibroblast growth factor family. It stimulates normal keratinocyte proliferation. Promotes reepithelization and granulation tissue formation [64].Tissue engineered **Human fibroblast-derived Dermal Substitute (HDS)** such as Derma graft (Smith & Nephew) accelerate wound healing. These are effective in all types of chronic leg ulcer and more so rheumatic ulcers [65, 66].

INJECTION SCLEROTHERAPY

This method is used in treatment of small varices (<3mm) where the main superficial trunks are competent or have been ligated (ineffective for bigger vessels). It acts by destroying the endothelium of an empty vein. Compression is applied immediately after injection and maintained for 3 – 6 weeks. The vein so treated heals by fibrosis. The drug that is commonly used in the UK is sodium tetradecyl sulphate (STD). In our region ethanolamine oleate has been in use. Other agents include polidocanol, hypertonic saline and chromated glycine. Complications of sclerotherapy include skin pigmentation, ulceration (extra-luminal injection) thrombophlebitis, and deep vein thrombosis (rarely) [53]. In our previous study [38], sclerotherapy was frequently used and the outcome was equivalent to that of surgery. After

one year the results were good for sclerotherapy 82% of the time and the success rate for surgery was 92%.

SURGERY

Patency of the deep system is a prerequisite to superficial vein surgery. Venography comes in handy particularly in recurrences or in patients with venous malformations. Operative treatment is associated with a higher cure rate than injection/sclerotherapy or compression /elevation. Barwell and colleagues [ESCHAR study] demonstrated that the rates of ulcer healing at 6 months were similar for compression plus surgery and compression alone (65%). However recurrence rates after surgery were lower (12% versus 28%) [53].

Superficial vein surgery

This is beneficial in those with superficial venous incompetence [67]. It involves surgical removal of varicose veins of the main saphenous trunks and their tributaries. Patency of the deep veins must be confirmed by Duplex ultrasonography or Venography before removal of superficial trunks. Surgery involves ligation at the point of reflux and stripping of the varices. Removal of saphenous trunks may be associated with damage to accompanying nerves (i.e. saphenous, sural nerves). The great saphenous vein should be stripped down to the mid-calf only. Localizing the varices and the perforators clinically and by ultrasound is necessary to avoid injury to arteries and nerves [4]. In a study at Mary Queen hospital, Hong Kong deep reflux improved after superficial vein surgery. The proportion of DVI (deep venous insufficiency) at more than one point decreased from 70% to 44%. The venous filling index decreased and the ejection fraction increased significantly [68].

Superficial variceal avulsion

Traditionally done through small incisions and avulsion carried out using small artery forceps. This has been replaced by small hooks through 1-2mm incisions. The varices are teased out, ligated and excised.

Superficial vein surgery alone was found to reduce ulcer recurrence at 3years from 44% to 26%. Superficial vein surgery together with compression bandaging decreased 3year recurrence to 9% compared to 38% with compression alone[67]

Subfascial ligation by open method

Small incisions are made over marked areas of perforator incompetence. The communicating vessel is identified where it joins the superficial trunk. Through an opening in the deep fascia, the perforator is double ligated and incised. Long incisions may complicate with sepsis or scarring [68].

Subfascial endoscopic perforator vein surgery(SEPS)

An endoscope is used to find and ligate perforators under the deep fascia. North American SEPS registry preliminary results (1997) showed an average healing time of 42 days, four times faster wound healing, less morbidity and a recurrence rate of only 3 per cent [67]. In a study by Chiwai Ting et al there was improvement in deep reflux after SEPS and superficial vein surgery SEPS and superficial vein surgery could be the optimum operative treatment for advanced CVI [68].

Endoluminal venous obliteration by radiofrequency heating

Endovenous obliteration with RF-resistive heating is an endoscopic procedure where a radiofrequency generator is programmed to maintain the temperature of the vessel wall at 85°C. A catheter inside the vein lumen is pulled back slowly (~ 3 cm / min) to maintain the impedance between 95 and 125 ohms and the temperature at 85°C. Studies indicate that endovenous obliteration may offer an advantage over conventional stripping operation in terms of reduced postoperative pain, shorter sick leaves and faster return to normal activities. Endovenous obliteration is more expensive. Larger studies are required to determine the precise role of this procedure in the treatment of primary LSV insufficiency [47].

Skin grafting

Skin grafting is necessary for large slow healing ulcers. Pinch grafts and split thickness grafts have been used. Factors that improve healing include, adherence to compression

therapy and elevation. Those that inhibit it are local fibrin deficiency (reduced graft adhesion) and microthrombi in dermal vessels (ischaemia).

Skin substitutes have been used with promising results. Allogeneic cultured bilayer of human skin with epidermal and dermal components (Graftskin) is approved in USA. Studies done showed faster healing than compression alone [65].

VENOUS RECONSTRUCTIVE SURGERY:

This is rarely carried out due to lack of appropriate prosthesis for valve reconstruction. It is however reserved for patients with deep vein insufficiency or occlusions who fail to resolve with other methods of treatment. Presence of a block is confirmed by direct venous pressure measurement in supine position and with exercises. **Raju test** is a measure of venous pressure in supine position in the hand and the foot. The foot pressure should be equal to or no more than 5mmHg above the hand pressure.

Venous Blockage:

Bypass procedures with vein or prosthetic material for the big vessels can be carried out. **Palma operation** involves mobilizing long saphenous vein of the normal side, tunneling it suprapubically and joining it to the femoral vein distal to the block. **May-Husni procedure** is used in superficial femoral vein blockage. The long saphenous vein is joined to the popliteal vein on the same side.

Venous Incompetence:

Two operations are carried out though in few specialized centres. **Kistner procedure** involves plication of the valve leaflets. It is a difficult operation to perform and may complicate with thrombosis. The other option is a **vein autograft**. Axillary vein has been used successfully in patients who have damaged deep veins [4, 7].

Complications of surgery

1. Haemorrhage – usually minimal within the first 12hours and stops with pressure.
2. Wound infection (1%)
3. Healing fibrosis – firmness under the scar or in the line of stripping.
4. Neuropraxia occurs in 5-10% and affects saphenous nerve after LSV stripping or the sural nerve after SSV stripping.
5. Recurrence ~20% within 5 years.
6. Thrombosis [4, 7]

STUDY JUSTIFICATION

There has been an apparent increase in numbers of patients with venous disease being seen at KNH both as inpatient and outpatients.

Patients with CVD and its attendant complications often tend to be neglected. Patients with leg ulcers go through a long process of treatment before definitive diagnosis is made.

In earlier years these patients were managed in the general surgical units. However in recent years the trend has been to refer them to cardiovascular surgeons. Despite CVD being a common problem, very little has been reported from this hospital. I was only able to trace one retrospective study on this subject, a 1995 dissertation. In that study outcome was assessed using the Hobb's criteria. Part of this study was to assess the usefulness of the new CEAP classification and the Venous Clinical Severity Score (VCSS) in measuring our disease pattern. This being a common problem, with the recent change in referral for management, and the general paucity of data on this disease in our setup, this study will perhaps make some useful contribution.

OBJECTIVES OF THE STUDY

Main objective:

To look at the clinical presentation, severity grades and management of chronic venous disease (CVD) at the Kenyatta National Hospital.

Specific objectives:

1. Determine the clinical presentation of chronic venous disease as seen at Kenyatta National Hospital.
2. Assess the clinical and research utility of CEAP classification and Venous Clinical Severity Score in our group of patients.
3. To determine the investigation trends in evaluation of CVD at KNH.
4. Outline the current management trends at Kenyatta National Hospital.

METHODOLOGY:

This was a prospective descriptive study to evaluate patients with chronic venous disease at Kenyatta National Hospital. All patients attended to as inpatients and outpatients in this hospital between January and August 2008 were recruited. The researcher interviewed patients admitted to the cardiovascular and other wards. Outpatients clinics covered included the cardiothoracic/cardiovascular, plastic, orthopaedic, general surgical and haematology. A set of questionnaires were used by the researcher or his assistant. Patients were examined and findings recorded in the questionnaire. Background information about patient's age, gender, occupation, posture at work, exercises and history of previous treatment was obtained. Examination for signs of venous disease and other co-morbidities was then carried out. Patients' weight, height, blood pressure and ankle pressures were taken. Mapping out of the anatomical area of disease and grading it by the CEAP clinical classification and Venous Clinical Severity Score (VCSS) was done for all patients. Ankle-Brachial Pressure Index (ABI) was recorded for all patients to rule out concurrent arterial disease. A Hand Held Doppler (Summit) with an 8MHz transducer was used to record ankle pressures. The attending clinician was informed if the ABI was low (<0.9).

Ulcer sizes were estimated by measuring the length and breadth.

Follow up interviews and examinations were carried out at the cardiothoracic/vascular clinic or ward after two weeks, one month, two months and three months of treatment. However, if the patient's clinic appointment was far, a telephone call was made to have at least one review after 3 months of treatment.

Inclusion criteria

1. All patients attending Kenyatta National hospital with clinical signs of chronic venous disease during the study period.
2. Patients who consented to participate in the study.

Exclusion criteria

1. Those who refused or could not validly give consent for the study.
2. Patients with other severe co morbid states such as congestive heart failure, severe arterial insufficiency or advanced cancer.

Ethical consideration

Approval was applied for and obtained from the Ethics and Research Committee of Kenyatta National Hospital. All participating patients/guardians signed an informed consent after they understood the purpose of the study.

Data collected was treated with utmost care and patient confidentiality observed.

Data analysis

Information obtained was entered in a computer database (Microsoft excel) and analyzed using SPSS version 13 computer software. A statistician assisted in data analysis.

Sample size:

The sample size for this study was derived using the formula;

$$n = \frac{Z^2 p (1-p)}{d^2}$$

Z=1.96 critical value for 95% confidence interval.

d =standard deviation=5%

p =prevalence (10% or 0.10).

Patients who come to hospital are mainly those with severe venous insufficiency or very gross varices. We took CVI prevalence of 10-35% as seen in various studies (3, 5, 6, 38).

$$n = \frac{(1.96)^2 \times 0.10(1-0.10)}{(0.05)^2}$$

n = 138.

Study limitations

1. The duration of follow up of patients in our study was short as this is part of a structured Masters of Medicine program.
2. Some patients could not afford certain imaging tests and treatment during the period of the study.
3. Some patients missed recruitment into the study because they were seen in the outpatient clinics and their hospital stay was brief.

RESULTS

Demographics

One hundred and forty four patients were interviewed between January and August 2008. Two of them could not give a valid consent and 2 were excluded due to severe co-morbidities. One hundred and forty fitted the inclusion criteria. There were 75 females and 65 males (1.15:1.00). The youngest 2 were a male and a female aged 17 years and the oldest was a 90year old man. Three patients (2.1%) in the group had congenital venous malformations. Primary venous insufficiency was found in 101 patients (79.5%) while secondary disease due to DVT occurred in 26(18.4%). In the congenital malformations group two had Parker-Weber syndrome with ulceration and hemi hypertrophy and the third patient had Trauney syndrome.

Table 1: Age groups, sex, and the frequency distribution

	Male		Female		Total	
Age groups(yrs)	n	%	n	%	n	%
17- 20	4	6.3%	2	2.7%	6	4.3%
21- 30	7	10.9%	9	12.0%	16	11.5%
31- 40	15	23.4%	26	34.7%	41	29.5%
41- 50	11	17.2%	21	28.0%	32	23.0%
51- 60	10	15.6%	12	16.0%	22	15.8%
61- 70	15	23.4%	4	5.3%	19	13.7%
71- 80	1	1.6%	1	1.3%	2	1.4%
81- 90	1	1.6%	0	.0%	1	.7%
Total	65	46.4%	75	53.6%	140	100%

Figure 2(a). Age groups (yrs) and the frequency (%).

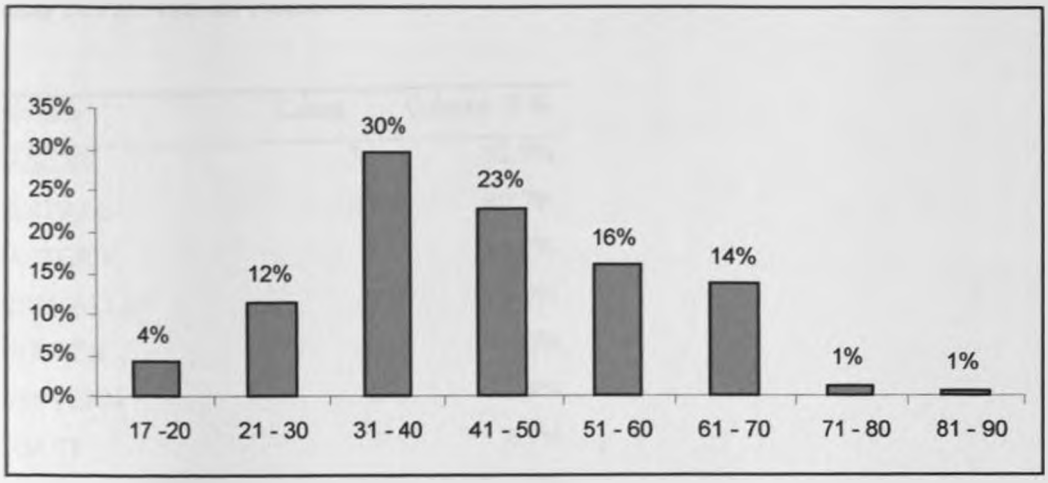
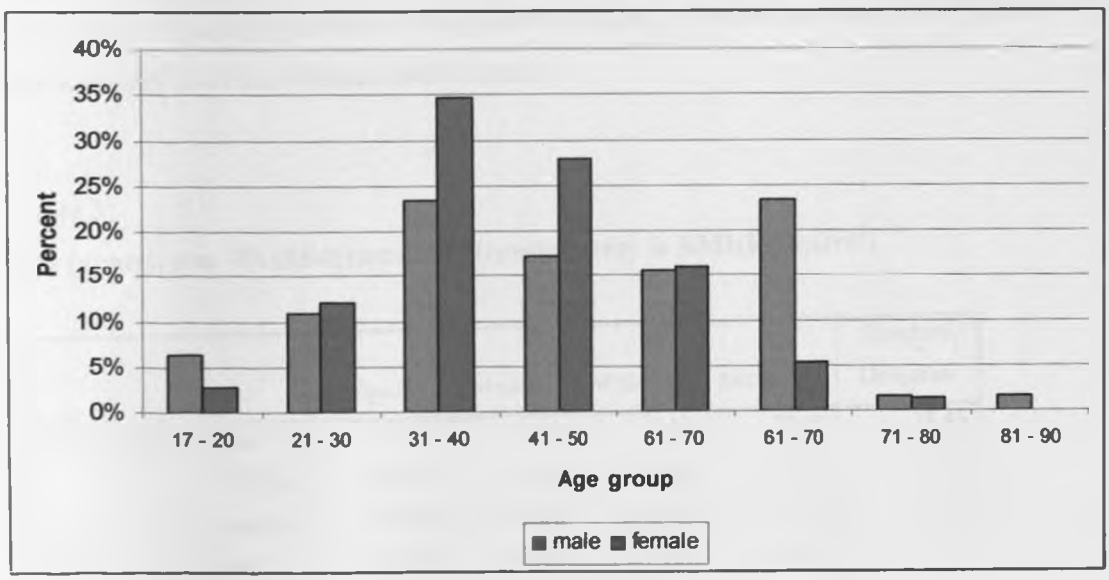


Figure 2(b). Age groups (years) distribution (males and females).



Majority of our clients were in the group aged 30-50 years (52.5%). A slight increase was noted in males above sixty years.

Table 2: Province of residence

Province	Count	Column N %
NAIROBI	74	52.9%
CENTRAL	43	30.7%
EASTERN	8	5.7%
RIFTVALLEY	8	5.7%
NYANZA	4	2.9%
WESTERN	2	1.4%
COAST	1	0.7%
NORTH EASTERN	0	0%

Over 80% of patients hailed from Nairobi the capital city of Kenya and the neighbouring provinces of Central and Eastern. One hundred and twelve of our sample population (80%) lived in urban areas while only 28(20%) resided in the rural areas. Not a single patient was from the North eastern province. Two patients residing in Nairobi and one from Thika were non Kenyans (Danish, Eritrean, and Sudanese).

Table 3:

Age (years), sex, Wt (kilograms), Ht (centimeters) & BMI(kg/metre^2)

			Mean	Median	Minimum	Maximum	Standard Deviation
sex	Male	Age	46.52	45.00	17.00	90.00	16.43
		Weight	74.84	73.70	47.00	127.00	15.54
		Height	172.21	172.00	153.00	187.00	7.25
		BMI	25.18	24.46	17.24	36.32	4.62
	Female	Age	41.65	41.00	17.00	73.00	11.22
		Weight	73.69	72.00	48.50	104.00	12.54
		Height	161.92	161.00	148.00	178.00	6.72
		BMI	28.20	27.82	18.95	41.09	5.09

Means for both male & female: age =44 yrs, height =166.7cm, weight =74.2 kg and BMI =26.8 kg/m^2

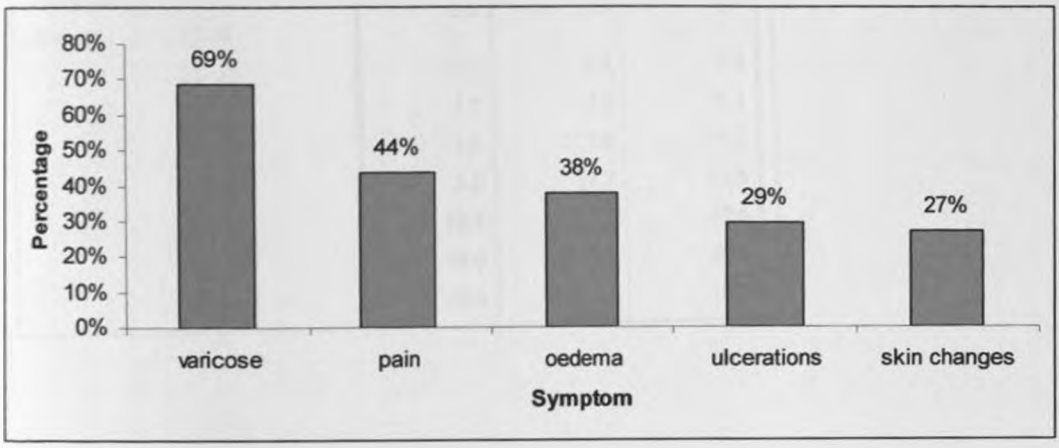
The mean age was 44 years (46yrs for men and 42 yrs in women). The average weights were comparable (males 75kg, females 74kg). Females were of shorter height but had a significantly higher BMI (females 28.2, males 25.2 kg/m^2). Seventy four percent of our patients were above 160cm in height.

Table 4: Occupation and the frequency

Occupation.	Count	%
Business people	24	17.0%
Farmers	18	12.9%
Teachers	15	10.7%
Secretary/clerks	12	8.6%
Industrial technicians/Engineers	11	7.9%
Cleaners/laundry workers	9	6.4%
Waiters/cooks	8	5.7%
Housewives	8	5.7%
Security people	7	5.0%
Students	6	4.3%
Nurses	5	3.6%
Messengers	3	2.1%
Butchers	3	2.1%
Drivers	3	2.1%
Tailors	2	1.4%
Sales people	2	1.4%
Pharmacists	2	1.4%
Laboratory technicians	2	1.4%

On interview, some of these patients were in careers that involve standing or sitting for long hours. Business people, farmers, teachers and secretaries dominated our group (49.2%).

Figure 3: Frequency of presenting complaints



Sixty four patients had bilateral disease making a total of 204 legs. One hundred and four had disease on the left and one hundred on the right (LT: RT = 1.04: 1.0). The commonest symptom was varicose veins in 140 limbs (68.6%), followed by pain in 89 legs (43.6%). Oedema in 77 (37.8%) was third in frequency, while ulceration with 60 (29.4%), skin changes 55 (27.0%) and itchiness in 7 (3.4%) limbs followed in that order.

Table 5: Age groups and the mean duration of symptoms (yrs).

		Male	female	Total
Age groups (years)	17-20	3.6	9 0	5.4
	21- 30	5.3	9.7	7 8
	31- 40	3.7	7.5	6.1
	41- 50	3.2	7 8	6.2
	51- 60	8.3	14.7	11.8
	61- 70	20.2	7.5	17 6
	71- 80	48.0	3.0	25.5
	81- 90	10.0		10 0

The mean duration of symptoms was 9.0 years. The median was 6 years and the mode was 2 years. Exclusion of those with durations above 20 years as outliers gave a mean of 6 yrs. Females younger than 60 years had longer duration of symptoms compared to males.

Table 6: Symptoms and duration in categories:

Duration(years) of symptoms.grouped	Male		Female		Total	
	Count	%	Count	Row N %	Count	%
0.0 - 2.0	24	49%	25	51%	49	35%
2.1 - 5.0	11	55%	9	45%	20	14%
5.1 - 9.0	10	56%	8	44%	18	13%
9.1 - 15.0	10	36%	18	64%	28	20%
>15.0	10	40%	15	60%	25	18%
Total	65	46%	75	54%	140	100%

Sixty five percent of our patients had symptoms for longer than 2 years while 38% had them for longer than 9 years.

The mean duration before presentation to cardiovascular surgical unit after diagnosis was 34 months (2 yrs 10 months). Primary health institutions referred at an average duration of 44 months, secondary institutions at 35 months and those from tertiary institutions came to the clinic after 23 months. There was no significant statistical difference among the groups ($p=0.210$).

Examination findings and severity grading

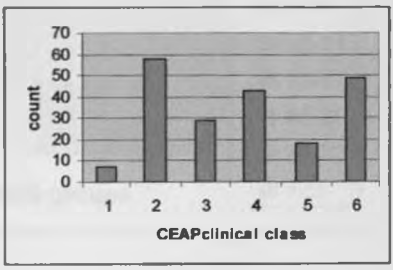
CEAP clinical classification based on the worst symptom (204 legs)

(See appendix III for details)

Table 7:

CEAP clinical class	count	Percentage
0	0	0%
1	7	3%
2	58	28%
3	29	14%
4	43	21%
5	18	9%
6	49	24%

Figure 4.



On examination, fifty eight (28%) legs were found to have varicose veins. The second largest group of forty nine (24%) had ulcers. Skin changes (eczema, hyper pigmentation, lipodermatosclerosis, atrophie blanche) were noted in forty three (21%) limbs. Oedema was observed in twenty nine (14%) limbs, eighteen (9%) had scars of healed ulcers and only seven (3%) had telangiectasia.

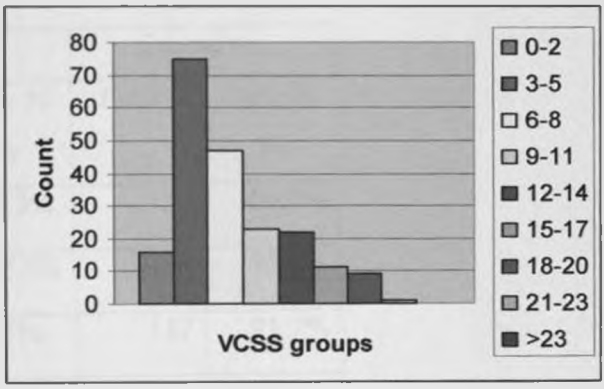
Initial Venous Clinical Severity Score (204 limbs)

(See appendix II for details)

Table 8:

VCSS groups	count	percent
0 – 2	16	7.8%
3 – 5	75	36.8%
6 – 8	47	23.0%
9 –11	23	11.3%
12-14	22	10.8%
15-17	11	5.4%
18-20	9	4.4%
21-23	1	0.5%
>23	0	0%

Figure 5.



Sixty percent of all limbs had VCS Scores of below 8 (mild symptoms). Twenty two percent were in the moderately severe group of 9 to 14.

Table 9: Clinical examination:

Brodie-Trendelenburg test for 204 lower limbs.

	Positive		Negative	
	Count	Row N %	Count	Row N %
Perforators reflux	70	34.3%	134	65.7%
SFJ reflux	56	27.5%	148	72.5%
SPJ reflux	17	8.3%	187	91.7%

Fifty six (27.5%) of all the limbs had saphenofemoral junction incompetence by the Brodie-Trendelenburg test, seventeen (8.3%) showed saphenopopliteal incompetence while seventy (34.3%) had perforator veins reflux. Five limbs had a positive Perthes test.

Pearson correlation coefficient between clinical tests and radiological imaging was low (-0.417, -0.327, -0.178, 0.043).

Twenty two percent (22.6%) of men had systolic BP above 140mmHg compared to 10.7% of women. A steady rise in the average ABI was noted in advancing age groups. Seven patients had an ABI less than 0.9. These seven were precluded from using compression therapy and sent for arteriography to determine further management.

Possible aetiological/risk factors:

Fifty one (36.4%) patients had trauma to the lower limbs. A third of this group (11.4%) sustained a fracture or neurovascular injury. Nine patients with DVT (6.4%) had previ0ous trauma. The correlation between trauma and DVT was low.

Females had a higher rate of DVT (23%) than males (14%). A third of the females used hormonal contraceptives. No correlation was found with history of DVT.

Table 10: DVT and CEAP clinical classes

	DVT	NO DVT
CEAP class 1-3	12(30.8%)	82(49.7%)
CEAP class 4-6	27(69.2%)	83(50.3%)
Total	39(100%)	165(100%)

P value=0.03

Patients with history of DVT tended to have severer disease by CEAP class than those without.

Sixty one (81.3%) female patients had a parity of 4 or less. Thirteen (17.3%) were nulliparous. Two of our patients were pregnant at the time of examination and both had primary disease.

Table 11: Posture at work, exercises, and prolonged immobilization.

			History of DVT	
			No	Yes
Posture at work	sitting	Count	31	8
	standing	Count	49	9
	mobile	Count	33	9
Exercises	No	Count	66	13
	Yes	Count	48	13
Prolonged immobilization	No	Count	106	23
	Yes	Count	8	3

Fifty eight patients (41.5%) were in careers that involve standing most of the time (teachers, business people, farmers and security workers) and 39(27.5%) spent the bigger part of the day sitting. Fifty six percent (56.4%) were not involved in any form of exercise. Prolonged bed rest was reported by 11(7.9%) of the whole group. Analysis with Pearson chi-square did not show statistical significance.

RADIOLOGICAL EXAMINATIONS

Twenty one (15%) patients had Duplex U/S before presenting in the cardiovascular clinic (KNH). Twelve (8.6%) had Venography performed before. Eighty one venographies and 65 Duplex scans were performed after the initial visit.

Table 12: Limbs examined by Doppler U/S scans (n=65)

Pathology	Count	Row N %
SFJ reflux	4	6.2%
SPJ reflux	2	3.1%
Perforators reflux	20	30.8%
DVT	9	13.9%
Deep reflux	8	12.3%
Superficial varicoses	5	7.7%
Superficial thrombosis	2	3.1%
Normal	15	23.1%

Eight scans(8) were bilateral. In twenty cases (30.8%) perforator incompetence was reported. Fifteen Duplex scans (23.1%) showed no abnormality. There were nine (13.9%) cases of DVT and 8 (12.3%) of deep venous reflux. Superficial varicoses was noted in five (7.7%), two cases (6.2%) of SFJ incompetence, two (3.1%) of SPJ reflux and two (3.1%) with superficial thrombosis. In eight cases a follow up venography was ordered for additional information.

Table 13: Limbs examined by venography (n=81)

	Count	Row N %
SFJ reflux	4	4.9%
SPJ reflux	0	0%
Perforators reflux	70	86.4%
DVT	4	4.9%
Superficial thrombosis	1	1.2%
Superficial varicosis	1	1.2%
Normal	1	1.2%

Twenty one patients had bilateral examinations. Seventy (86.4%) of the limbs examined showed perforator incompetence. SFJ reflux was noted in only four (4.9%) patients. Four examinations (4.9%) revealed presence of DVT. None (0%) of the limbs showed SPJ incompetence. Superficial thrombosis was reported in one; superficial varicoses in one and another one reported no abnormality (3.6%).

TREATMENT

Forty eight percent (48.1%) of patients were diagnosed in tertiary health institutions (National teaching and referral). Another big proportion (44.4%) was diagnosed in primary institutions (Clinics and District hospitals). Clinical diagnosis was made in primary and secondary institutions (Provincial) 51.9% of the time.

There is a change in trend of management of CVD in our hospital. Previously this was under general surgeons, but currently almost all patients with signs of venous insufficiency are referred to cardiovascular surgeons. A small proportion is seen in the haematology clinic with post thrombotic sequel. In this study 93% of patients were recruited from the cardiovascular surgical department. Five percent were from other surgical units and 2% were in medical departments. The actual proportion was not obtained because we did not see all patients that went through the other units.

The 140 patients who participated were recruited from the following departments;

1. Cardiothoracic clinic -111 (79.3%)
2. Ward 4B (cardiothoracic) -19 (13.6%)
3. Orthopaedic wards -4 (2.9%)
4. Ward 4D (plastic) -3 (2.1%)
5. Haematology clinic -2 (1.4%)
6. Medical ward -1 (0.7%)

Table 14: Treatment decision on first consultation

	n-140	%
Compression	89	63.6%
Elevation	89	63.6%
Drugs	29	20.7%
Sclerotherapy	1	.7%
Operation	43	30.7%
Wound care	42	30.0%

Table 15: Treatment administered.

	Total count (n-140)	%
Compression	88	62.9%
Elevation	93	66.4%
Medication	39	27.9%
-antibiotics	3	2.1%
-venotensive drugs	18	12.9%
-anticoagulation	19	13.6%
Injection sclerotherapy	0	0%
Operation	32	22.9%
Wound care	42	30.0%

Majority (66.2%) of our patients were started on conservative treatment. Forty three were scheduled for operative management but only thirty two (74.4%) underwent surgery during our study period. Majority of the remainder group were waiting to get admissions to cardiothoracic ward. Compressive treatment was given 99% of the time. Elevation also had good compliance rate. The only patient for sclerotherapy missed due to lack of a sclerosing agent. This method of treatment was rarely prescribed. Eight (17%) patients with ulcers underwent skin grafting and the rest were assigned conservative treatment. Vein surgery was considered only after ulcer healing.

Table 16: Surgical methods.

Type of surgery	Frequency
Perforator ligation	23(34%)
LSV ligation/stripping	21(31%)
Avulsion/phlebectomy	11(16%)
Skin grafting	9(13%)
SSV ligation/stripping	4(5%)
Total	68(100%)

Fifty nine venous operations and nine cases of skin grafting were performed. Perforator ligation was the commonest procedure (34%) followed by LSV stripping (31%). Some patients had more than one procedure.

OUTCOMES OF TREATMENT

Ninety nine patients (99) were managed conservatively while 32 underwent venous surgery. Nine patients did not start any form of treatment due to various reasons. Overall there was improvement in VCSS and CEAP clinical class after 3 months. Mean change in CEAP class was 0.43 points and VCSS changed by an average of 3.0 points. Ulcer sizes also reduced markedly with a majority of patients experiencing complete healing. Average ulcer size reduction was $25cm^2$. The mean change in VCSS remained 3.0 points for 32 operated patients compared to 2.0 points in the conservative group (p value=0.058). Percentage changes in VCSS was significantly higher in the operation group (40% vs. 21% $p<0.0001$)

Table 17: Types of surgery and the change in VCSS score.

	VCSS change right		VCSS change left		Total	
	N	Mean	N	Mean	N	Mean
LSV ligation	7	3	8	4	15	3.5
SSV ligation	1	7	4	2	5	4.5
Ligation subfascial	17	3	7	4	24	3.5
avulsion	3	7	5	5	8	6
Skin grafting before vein surgery	4	10	16	2	20	6

Avulsion and skin grafting had the biggest change in VCSS. This is due to the high scores given to ulcers and varicose veins.

Table 18: Percentage VCSS change in 3 months of treatment.

		Type of treatment	
		Non-operative	Operative
Percentage change in VCSS	Mean	21	40
	Minimum	0	0
	Maximum	69	100
	Standard Deviation	20	26

P<0.0001

Majority of patients showed improvement in symptom scores after 3 months of treatment. There was a significantly higher change in VCSS for surgical patients compared to the conservative group.

Table 19: Mean change in CEAP Clinical class (Individual limbs)

		Non-operative	Operative	P value
CEAP change left.	Count	99	32	0.002
	Mean	.34	.88	
	Standard Deviation	.65	1.01	
CEAP change right.	Count	99	32	0.007
	Mean	.25	.79	
	Standard Deviation	.63	1.25	

The average shift in CEAP clinical class was 0.43 for all groups. The operative group had greater improvement in clinical classes (P value 0.002 and 0.007).

Table 20: Ulcer size change in 3 months:

	Ulcer size 0weeks	Ulcer size 3 months	Ulcer size change	% Ulcer size change
Count	40	40	40	40
Mean	60	35	25	24
Standard Deviation	90	62	61	163
Median	10	5	2	50
Minimum	1	0	-48	-900
Maximum	375	280	310	100

Nine legs were grafted, one small ulcer ($1cm^2$) healed in two weeks without specific therapy.

Twenty limbs (40%) with ulcers healed, nine (18%) with grafting and eleven (22%) by secondary intention. Five ulcers increased in size (10%). One patient developed a new ulcer (2%). The remaining 24(48%) limbs with ulcers all showed a decrease in size.

The mean ulcer healing time was three months and on average ulcer patients resumed work after 3 months of treatment.

Table 21: Complications in the course of treatment

Complications	Count
BLEEDING (n=140)	3 (1.5%)
SURGICAL WOUND INFECTION (n=32)	5 (15.6%)
ULCER SIZE INCREASE (n=50)	6 (12%)
PAIN, ITCHINESS, NUMBNESS(n=140)	4 (2%)
UPPER GI BLEEDING(n=19)	1 (3.9%)

Complications occurred in twenty two patients in either treatment arm. Those that were noted included surgical wound infection [5(15.6%)], ulcer enlargement [6(12 %)], bleeding from the ulcer or ruptured veins [[3(1.5 %)], pain itchiness or numbness [4(2 %)] and upper GI bleeding [1(3.9%)] in a patient on warfarin.

Table 22. Recurrences and duration after previous treatment

	Mean duration in years		
Treatment/ Recurrence	Varicose veins	Ulcers	Other symptoms
Compression	2.97	1.82	1.28
Elevation	2.66	2.43	2.46
Drugs	0	0	0
Sclerotherapy	2.00	2.00	0
Operation	3.28	2.78	2.88
Wound care	2.06	1.72	1.00

Duration before recurrence was longer for the surgically treated patients.

Table 23: Treatment not started or stopped prematurely.

REASONS TREATMENT NOT FOLLOWED:		
SURGERY	Investigations	13(9.3%)
19(13.6%)	Cost of surgery	1(0.7%)
	Pregnancy	1(0.7%)
	Ward booking	1(0.7%)
	Patient preference	2(1.4%)
	LTFU	1(0.7%)
COMPRESSION.	Investigations	7(5.0%)
20(14.3%)	Cost of stockings	3(2.1%)
	Difficulty in application	4(2.9%)
	Appearances	2(1.4%)
	Other illnesses	2(1.4%)
	No prescription	1(0.7%)
	LTFU	1(0.7%)

Fourteen percent could not have surgery due to cost related issues. Seven percent did not use compression therapy as prescribed due to similar reasons. Other reasons for non compliance were difficulty wearing the stockings, concern about appearance and patient preference.

DISCUSSION

This was a hospital based descriptive prospective study. One hundred and Forty patients were recruited over a period of 7 months. This translates to 20 patients in a month or 5 patients per week. The number is higher than what was recorded in a previous retrospective study by Cheruiyot [38]. In that study 28 patients were treated annually giving an average of 2 patients a month. The focus in that study was on varicose veins. Possibly, some patients with CVI who did not manifest with varicose veins were missed out.

This ten fold increase in numbers may also be explained by the fact that there is a general rise in the level of awareness and patients are seeking health care sooner than before.

Eighty Percent of our patients lived in urban areas. A similar proportion came from Nairobi and the neighbouring Districts. Not a single patient was from the North Eastern province of Kenya. Failure to register any patient from that region may be related to the high cost of travel and treatment. On the other hand, many people from the North Eastern province do live in Nairobi.

If indeed there is a true rise in incidence, lifestyle change associated with urbanization may be one of the likely factors. Forty one percent of our patients were in careers that involve standing most of the time (business people, farmers, teachers, security workers and hotel workers). Twenty seven percent spent most of the day seated. Careers associated with this category included secretaries, housewives and drivers. Only 43.6% were involved in some form of exercise. In the Edinburgh vein study standing at work was associated with more severe disease in females [16].

In our study the mean BMI for women was 28 (overweight) and in the males it was close to the normal (25.2). In the same Edinburgh study, persons with high BMI had severer disease than those with normal BMI. They also found that in males, increased height and straining at stool were related to superficial reflux. An association with the number of pregnancies was less marked after correcting for age [16].

In our study over 63% were 160cm in height or taller. Males were generally taller than the females.

Eighty one percent of the female patients had a parity of 4 or less. Seventeen percent were nulliparous. This is different from the previous study where 54% had a parity of 5 or more [38].

Patients with history of DVT presented more in the higher grades of CVD (CEAP class 4 – 6). Studies have shown that patients with deep vein reflux tend to have severer disease than

the ones with superficial disease. Not all patients with deep venous insufficiency have had DVT [68].

The commonest presenting symptom was varicose veins (69%) followed by pain (44%) and oedema (38%). Ulceration (29%), skin changes (27%) and itchiness (7%) were less frequent. Among the physical findings varicose veins was the commonest sign followed by ulceration. Sixty two patients (43.3%) gave history of previous or active ulcer in the leg. Forty seven (33.6%) patients had active ulcers at the time of examination. Three were bilateral giving a total of 50 legs (24.5%). This is similar to findings in the previous study where varicose veins was the leading symptom (46%) followed by pain (20%) and swelling (14%). However in the current study we saw more of secondary disease (18.4%) than in the former study (3.6%). There were also more females (1.15:1.00) in the current study [38].

Duration of symptoms was long for most patients. Sixty five percent had symptoms for more than 2 years. The mean duration was 9 yrs and 38% of patients exceeded this duration. In the previous study, the same long duration of symptoms was noted. Ninety six percent had symptoms for more than one year while 22% had durations longer than 9 years [38].
The mean duration before review in the cardiovascular clinic for the current study was 34 months. Those diagnosed in primary health institutions took longer than other groups but this was not statistically significant. This delay in patients seeking medical attention may be due to the relatively mild symptoms associated with CVD. In addition difficulty accessing health services and lack of investigative tools at the primary health institution contributed to the delay. Only 15% of our patients were referred with a Doppler scan result.

The pattern of disease presentation was consistent with the chronicity of symptoms. Fifty five percent of limbs examined were in CEAP classes 4-6. Varicose veins, skin changes and ulceration were the three commonest manifestations. After treatment there was a shift to the left mainly to classes 1, 4 and 5. Using CEAP classes, it was not possible to move down beyond class 5 from class 6 in the short term. In a study by Rutherford et al CEAP classes were found to be relatively static and poor in measuring response to treatment [70].

Severity scores by VCSS showed that majority of patients had mild symptoms initially (VCSS<9 in 67.6%). Big changes in scores were seen particularly after surgery. In a study by Rutherford et al VCSS was found to be better in evaluating change after treatment than CEAP classes [70]. Percentage changes were comparable for the two methods. Meissner et al found good correlation and little inter observer difference [44]. Kakkos and others also found good correlation between VCSS, VDS and the CEAP clinical classes. However the Venous Segmental Disease Score (VSDS) showed a weak correlation [45].

ABI was recorded in all the limbs (280). There was a steady rise in ABI associated with increasing age. This is an expected finding as the prevalence of atherosclerosis rises with age. Seven patients (5%) had low ABI (<0.9). These patients were advised not to use compressive therapy. They were then sent for arteriography and started on treatment for the concurrent arterial insufficiency.

Venography was the most commonly used mode of investigation in our study. Eighty one venograms and 65 Duplex studies were done. The pick up rate for perforator reflux was lower with Duplex compared to venography in this study. In diagnosis of deep reflux Duplex was superior to venography. In Cheruiyot's study venography was the main radiological imaging though in a small proportion of patients (39 limbs [15.5%]). Various studies recommend Duplex U/S as the main imaging test. It is accurate in trained hands, reproducible and non invasive. Venography is recommended for recurrences or complex malformations [14, 16, 42, 47]. Perforator reflux was the commonest pathological defect elicited by clinical tourniquet test (34.3%), venography (86.4 %) and Duplex (30.8%) imaging. It would appear in this study venography had higher sensitivity than Duplex U/S in diagnosis of perforator incompetence. This is in contrast to Cheruyoit's study findings where LSV incompetence was the most frequent pathology (63.8%). However for the 39 limbs imaged with venography, 29(74.4%) showed incompetent perforators [38]. Perforator incompetence is commonly associated with deep vein insufficiency (DVI).

Operative management was recommended in 30.7% of our patients. Only 22.9% received this form of treatment. This is low considering the benefits in symptoms score after surgery. In a study on cost effectiveness of surgery versus conservative, Ratcliffe J. et al found that surgery offers a modest health benefit compared to conservative treatment. However, surgery was a little more expensive [69]. In the ESCHAR study [53] surgery and conservative treatment worked the same in promoting ulcer healing. However, recurrences were fewer with surgery. The type of surgery offered to our group of patients included, ligation and stripping of truncal varices (35%), perforator ligation (34%), and Phlebectomy (16%). Skin grafting was performed in 9 patients (13%). Sclerotherapy was not administered during this study. The only patient who was to have it missed due to lack of a sclerosant. This contrasted markedly with the previous study [38] where sclerotherapy was the commonest mode of treatment.

In our study 20 (40%) leg ulcers eventually healed out of which 9 (18%) healed after skin grafting and 11 (22%) on dressings alone. Twenty four (48%) limbs showed ulcer size decrease. Five (10%) limbs had ulcer enlargement and one patient (2%) developed a new ulcer. The rate of compression therapy use was low in the whole group. Only 12 (19%) of the

62 patients with history of ulceration were advised to use compression before their first consultation. It has been reported elsewhere that the outcome of treatment for venous leg ulcers is generally poor. About 50% heal in 4 months, while 8% remain open at 5 years [13, 17]

The percentage VCSS change and CEAP class change were significantly higher in the operative group compared to the conservative group. Assessment by VCSS showed greater improvement after avulsion and skin grafting. This may be explained by the fact that this scoring system gives higher points to ulcer symptoms, and there is possibility that long term follow up may not show the same. Percent change for VCSS was significantly greater than corresponding change in CEAP clinical class. This is similar to findings of a study done by Kakkos et al to validate the new venous scoring systems [45]. Patients who were managed surgically generally had longer periods before recurrence of symptoms (ulcer, varicose veins and other symptoms) compared to the other methods (3.0 yrs for surgical patients, 2yrs for compressive therapy and 2.5 yrs for the elevation group). This is in agreement with the ESCHAR study where they also found fewer recurrences following surgery [53].

In 9.3% of cases, surgery was not scheduled due to lack of imaging investigations.
Five percent of our study population did not start compressive therapy as they awaited review with radiological results. Inability by patients to pay for these tests was mentioned as one of the causes for delay. Another reason for non compliance was inability to afford treatment (2.8%).Four patients (2.9%) had difficulty applying the stockings, 2(1.4%) were concerned about appearance and 2(1.4%) had other illnesses that precluded use of stockings. Two (1.4%) in the surgical group preferred conservative treatment. Other factors that may affect compliance to stockings use include; obesity, arthritis and advanced age [4, 39]. In a study by Samson RH and Showalter DP the high cost of compression therapy was the main cause of non compliance [50].

Conclusions

1. Chronic venous disease is now more common in patients presenting to our cardiovascular/cardiothoracic surgical unit. Sixty eight percent are below 50 years a relatively young age group compared to Western populations. Majority of our patients are from urban areas.

2. Most of our patients present after long durations of symptoms (average 9years). Varicose veins were the commonest symptom followed by pain and oedema. There is a rise in the number of patients with secondary disease compared to the previous study.

3. Clinical Etiological, Anatomical and Pathophysiological (CEAP) classification and the Venous Clinical Severity Score (VCSS) are useful grading tools in evaluating response to treatment.

4. Venography, though invasive, is still the most commonly used imaging test. Duplex scan was also widely used.

4. Majority of the patients were managed by conservative methods possibly due to mild symptoms. However the rate of usage of pressure stockings by patients with CVD is low. There are no special clinics for follow up of patients with CVD and ulcers.

5. Surgically managed patients experienced greater improvement in symptom severity scores than the conservative group. There was also a significant change in CEAP class. Recurrences were less frequent and occurred after a longer duration.

RECOMMENDATIONS:

1. Chronic venous disease patients should ideally be investigated at the primary health institution before referral to the cardiovascular surgeon. This would reduce the waiting time before surgical intervention.

2. CEAP and VCSS classifications should be adopted for routine use in evaluating chronic venous disease in this hospital as this would improve standardization and reporting. VCSS would be useful in assessing outcome of treatment.

3. Duplex US as a non invasive test should be encouraged in evaluation of CVD. The possibility of availing portable Hand Held Doppler for the surgeons who deal with this condition should be considered.

4. Dedicated venous disease and ulcer clinics should be set up to handle the big number of patients who require compressive therapy and specialized wound care.

5. Further epidemiological studies may be necessary to determine the true disease burden in the country. And possibly another study in this set up with a longer follow up period would help in evaluating long term outcome.

6. Introduction of newer forms of therapy (endoscopic ligation of perforators, laser therapy and radiofrequency heating) should be considered for Kenyatta National Hospital.

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APPENDIX I

QUESTIONAIRE

Serial number..... Date ..... Venue .....

PERSONAL DATA:

1. Name.....
2. Age.....
3. Sex.....
4. Unit number.....
5. Province.....
6. Occupation.....
7. Weight.....
8. Height.....
9. BMI (mass (kg)/Ht $(m)^2$).....
10. Alcohol intake Yes☐ No☐
11. Tobacco smoking Yes☐ No☐
12. Position/posture at work (a) sitting☐ (b)standing☐ (c) mobile☐
13. Exercises Yes☐ No☐ Type of exercise.....

CLINICAL PRESENTATION(Symptoms and duration):

	RT	LT	Duration (yrs)
Pain	☐	☐	☐
Varicose veins	☐	☐	☐
Skin changes	☐	☐	☐
Ulcerations	☐	☐	☐
Oedema	☐	☐	☐

Others specify_____

PAST MEDICAL HISTORY

1. Trauma to the lower limbs Yes☐ No☐

   If yes; (a) Superficial☐

   (b) Deep☐ (i) Fracture☐ (ii) Neurovascular injuries☐

2. History of deep venous thrombosis or sudden onset painful leg swelling Yes☐ No☐

3. Anticoagulation therapy Yes☐ No☐

4. Ulcers in the lower leg Yes☐ No☐

   (a) Duration ..... Days☐ Weeks☐ Months☐ Years☐

   (b) Mode of treatment Dressing Drugs☐ Compression☐

   Other_____

5. Surgery on the lower limbs Yes☐ No☐

   (a)Type of surgery Vein☐ Orthopaedics☐ Grafting☐

   Other_____

   (b)Postoperative complications.....

6. Other forms of surgery Yes☐ No☐

7. Prolonged immobilization/bed rest Yes☐ No☐

8. History of arterial hypertension Yes☐ No☐

9. Diagnosis of CVD made Yes☐ No☐

10. Diagnosis at institution: primary☐ secondary☐ tertiary☐

11. Duration before referral Days☐ Weeks☐ Months☐ Years☐

12. Tests done prior to referral Ultrasound☐ X-Ray☐ Venogram☐

13. Family history of venous disease Yes☐ No☐

OBSTESTRICS HISTORY IN FEMALES:

Parity..... Last delivery (years)..... Last menstrual period .....

14. Contraceptives: Oral☐ Injectible☐ Implant☐ None☐

15. Other forms of hormonal therapy: Yes☐ No☐

    Type.....

PHYSICAL EXAMINATION:

Patients Blood Pressure: Systolic..... Diastolic.....

Rt. Ankle pressure ABPI.....

Lt. Ankle pressure ABPI.....

CEAP Clinical (0--6 a or s) tick the class.

	RT	LT
(0) No visible or palpable signs of venous disease	☐	☐
(1) Telangiectasia and/or reticular veins	☐	☐
(2) Varicose veins	☐	☐
(3) Oedema	☐	☐
(4) Skin pigmentation, venous eczema, lipodermatosclerosis	☐	☐
(5) Skin changes as above with healed ulceration	☐	☐
(6) Skin changes with active ulceration	☐	☐

Other features of CVD.....

Veins affected	Limb		Region			
1. LSV	Left ☐	Right ☐	Thigh ☐	Calf ☐	Ankle ☐
2. SSV	Left ☐	Right ☐	Thigh ☐	Calf ☐	Ankle ☐
3. Perforators	Left ☐	Right ☐	Thigh ☐	Calf ☐	Ankle ☐

4. Others_____

Ulcer present Yes ☐ No ☐

Ulcer details: Right ☐ Left ☐ Both ☐

RT: Lateral ☐ Medial ☐ Circumferential ☐

LT: Lateral ☐ Medial ☐ Circumferential ☐

Size: Length.....cm, Width.....cm Approximate area.....cm^2

Dorsalis pedis pulse	present ☐	absent ☐
Chest examination	Normal ☐	Abnormal ☐
Abdominal examination	Normal ☐	Abnormal ☐

CLINICAL TESTS

1. Trendelenburg's test:

Left Leg

SFJ: positive ☐ negative ☐

SPJ: positive ☐ negative ☐

Perforators: positive ☐ negative ☐

Right Leg

SFJ: positive ☐ negative ☐

SPJ: positive ☐ negative ☐

Perforators: positive ☐ negative ☐

2. Perthes' test: positive ☐ negative ☐

If positive, Right ☐ Left ☐ Both ☐

IMAGING TESTS.

1. Doppler Ultrasound scan:

Left: SPJ Reflux ☐ SFJ Reflux ☐ Perforators Reflux ☐ DVT ☐

Right: SPJ Reflux ☐ SFJ Reflux ☐ Perforators Reflux ☐ DVT ☐

2. Duplex U/S:

Left: SPJ Reflux ☐ SFJ Reflux ☐ Perforators Reflux ☐ DVT ☐

Right: SPJ Reflux ☐ SFJ Reflux ☐ Perforators Reflux ☐ DVT ☐

3. Venography:

Left: SPJ Reflux ☐ SFJ Reflux ☐ Perforators Reflux ☐ DVT ☐

Right: SPJ Reflux ☐ SFJ Reflux ☐ Perforators Reflux ☐ DVT ☐

3. Others: Specify _____

Left: SPJ Reflux ☐ SFJ Reflux ☐ Perforators Reflux ☐ DVT ☐

Right: SPJ Reflux ☐ SFJ Reflux ☐ Perforators Reflux ☐ DVT ☐

TREATMENT:

Treatment decision at the first consultation;

Compressive bandage/Stockings _____

Elevation _____

Drugs _____

Sclerotherapy _____

Operation _____

Wound care _____ days/weeks

What are the reasons for delay?.....

.....

Treatment given and the duration.

Compressive bandage/Stockings _____ days/weeks/months/years

Elevation _____ days/weeks/months/years

Medication _____ days/weeks/months/years :

Antibiotics Venotensive drugsAnticoagulation

Injection/compression ____ days/weeks/months/years:

Type of Sclerosant _____

Operation _____ days/weeks/months/years

Wound care _____ days/weeks/months/years

OPERATIVE TREATMENT:

(a) **Ligation and stripping;**

(i) LSV Right ☐ Left ☐

(ii) SSV Right ☐ Left ☐

(b) **Ligation of communicating veins**

(i) Extrafascial ☐ (ii) Subfascial ☐

Number of perforators ligated Left side

Above knee ____ or not recorded ☐

Below knee ____ or not recorded ☐

Number of perforators ligated Right side;

Above knee ____ or not recorded ☐

Below knee ____ or not recorded ☐

(c)**Avulsion/phlebectomy** Right ☐ Left ☐

(d) **Valvuloplasty/ vein grafting** Right ☐ Left ☐

(e) **Skin grafting** (i) Before vein surgery Right☐ Left☐

(ii) After vein surgery Right☐ Left☐

(f) **Other operations** .....

OUTCOME OF TREATMENT

(a) Ulcer size(cm^2): 0wks ___ 2 wks ___ 1mo ___ 2mo ___ 3mo ___ >=4mo ___

(b) Healing of the ulcer___ days/weeks/months

(c) Resumption of work___ days/weeks/months

(d) Stoppage of treatment___ days/weeks/months

(e) VCSS Initial..... VCSS Post treatment.....Duration .....days/months/years

VCSS=venous clinical severity score (see page 70).

Complications.....

.....

.....

.....

.....

Recurrence

(i) Varices Yes ☐ No ☐

Treatment: Compressive bandage/Stockings _____ days/weeks

Elevation _____ days/weeks

Drugs _____ days/weeks

Sclerotherapy _____ days/weeks

Operation _____ days/weeks

Wound care _____ days/weeks

(ii) Ulcer Yes☐ No☐

Treatment: Compressive bandage/Stockings _____ days/weeks

Elevation _____ days/weeks

Drugs _____ days/weeks

Sclerotherapy _____ days/weeks

Operation _____ days/weeks

Wound care _____ days/weeks

(iii) Other symptoms Yes ☐ No ☐

Treatment: Compressive bandage/Stockings _____ days/weeks

Elevation _____ days/weeks

Drugs _____ days/weeks

Sclerotherapy _____ days/weeks

Operation _____ days/weeks

Wound care _____ days/weeks

Treatment not followed or stopped Yes ☐ No ☐

If treatment not followed, why.....

.....

.....

.....

.....

APPENDIX II

Venous Clinical Severity Score (VCSS).

Attribute	Absent = 0	Mild = 1	Moderate = 2	Severe = 3		
Pain	None	Occasional	Daily	Limit activities		
Varicose veins	None	Few, scattered	Multiple (LSV)	Extensive (LSV, SSV)		
Venous edema	None	Evening, ankle	Afternoon, leg	Morning, leg		
Pigmentation	None	Limited area	Wide (lower 1/3)	Wider (above 1/3)		
Inflammation	None	Cellulitis	Cellulitis	Cellulitis		
Induration	None	Focal (< 5 cm)	< lower 1/3	Entire lower 1/3		
Number of AC	0	1	2	3		
Duration of AC	None	< 3 months	3 months – 1 year	> 1 year		
Size of AC	None	< 2 cm diameter	2-6 cm diameter	> 6 cm diameter		
Comp therapy	Not used	Intermittent use	Most days	Continually		
LSV, long saphenous vein; SSV, short saphenous vein; AC, active ulceration; lower 1/3, lower 1/3 of the leg.						

A scale of 0 to 30 is used to grade disease severity before, during and after treatment[32,42].

APPENDIX III

CEAP CLASSIFICATION OF CHRONIC LOWER LIMB VENOUS DISEASE.

Mark	*Definition.*
C	Clinical signs (grade 0-6), supplemented by (s) for symptomatic and (a) for asymptomatic presentation.
E	Etiological classification (congenital, primary, secondary).
A	Anatomic distribution (Superficial, Deep, or Perforator, alone or in combination).
P	Pathophysiologic dysfunction (Reflux or Obstruction, alone or in combination).

Primary varicose veins (95%) are caused by increased pressure in superficial veins due to incompetence of valves between deep and superficial systems.

Secondary VV's (5%) are associated with superficial pressure rise due to a blockage and reflux within the deep system as in thrombosis or arteriovenous malformations (cong. or acquired)[15].

Classification by clinical signs

Class	characteristics
0.	No visible or palpable signs.
1.	Telangiectasia, reticular veins or malleolar flare.
2.	Varicose veins.
3.	Oedema without skin changes.
4.	Skin changes such as pigmentation, eczema or lipodermatosclerosis
5.	Skin features as above and signs of a healed ulcer.
6.	Skin changes plus active ulceration[125].

CEAP Etiological Classification(E)

Congenital - Ec

Primary -Ep undetermined cause.

Secondary -Es known cause

CEAP Anatomic classification(A)

Segment no.

Superficial veins (As)

1. Telangiectasia\reticular veins
2. GSV –above knee
3. GSV –below knee
4. SSV
5. Nonsaphenous

Deep veins (Ad)

6. Inferior vena cava.
7. Common Iliac.
8. Internal Iliac.
9. External Iliac.
10. Pelvic- gonadal, broad ligament, others.
11. Common Femoral Vein.
12. Deep Femoral.
13. Superficial Femoral.
14. Popliteal.
15. Crural-ant. , post. Tibial, Peroneal.
16. Muscular-gastrocnemial, soleal, others.

Perforators (Ap)

17. Thigh Perforators
18. Calf Perforators.

CEAP Pathological classification(P)

Reflux - PR

Obstruction - PO

Reflux and Obstruction - PR, O

Example of CEAP classification: A patient with symptomatic varicose veins involving the SSV territory with swelling, pain and lipodermatosclerosis. The patient has a normal deep system.

CEAP class; C2, 3,4s -Ep -As4, -Pr.

CEAP classification is widely used in research. It has been criticized for being complex. However, it is reproducible and not too difficult to learn.

In assessing disease severity or treatment outcomes CEAP Disability Score or Venous Clinical Severity Score are useful tools[42, 47].

CEAP Disability Score

0. Asymptomatic
1. Symptomatic can function without support device.
2. Can work 8hr day only with support device.
3. Unable to work even with support device.

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O Box 20723, Nairobi.
Tel: 726300-9
Fax: 725272
Telegrams: MEDSUP", Nairobi.
Email: KNHplan@Ken Healthnet.org

Ref: KNH-ERC/ 01/ 81

25th January 2008

Dr. Anthony Muthee
Dept. of Surgery
School of Medicine
University of Nairobi

Dear Dr. Muthee,

RESEARCH PROPOSAL: "CHRONIC VENOUS DISEASE OF THE LOWER EXTREMITY AT KENYATTA NATIONAL HOSPITAL" (P228/8/2007)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** your revised research proposal for the period 25th January 2008 – 24th January 2009.

You will be required to reques: for a renewal of the approval if you intend to continue with the study beyond the deadline given. Clearance for export of biological specimen must also be obtained from KNH-ERC for each batch.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely

PROF A N GUANTAI
SECRETARY, KNH-ERC

c.c. Prof. K.M.Bhatt, Chairperson, KNH-ERC
The Deputy Director CS, KNH
Dean, School of Medicine, UON
The Chairman, Dept of Surgery, UON
Supervisor, Mr. T. M. Omulo, Dept of Surgery, UON