

#### **University of Huddersfield Repository**

Dunkley, Liza

Peripheral Oedema - Institute of Chiropodists and Podiatrists March AGM 2010

#### **Original Citation**

Dunkley, Liza (2010) Peripheral Oedema - Institute of Chiropodists and Podiatrists March AGM 2010. In: The Institute for Chiropodists and Podiatrists West Yorkshire Branch, March 2010, The University of Huddersfield. (Submitted)

This version is available at http://eprints.hud.ac.uk/id/eprint/13962/

The University Repository is a digital collection of the research output of the University, available on Open Access. Copyright and Moral Rights for the items on this site are retained by the individual author and/or other copyright owners. Users may access full items free of charge; copies of full text items generally can be reproduced, displayed or performed and given to third parties in any format or medium for personal research or study, educational or not-for-profit purposes without prior permission or charge, provided:

- The authors, title and full bibliographic details is credited in any copy;
- A hyperlink and/or URL is included for the original metadata page; and
- The content is not changed in any way.

For more information, including our policy and submission procedure, please contact the Repository Team at: E.mailbox@hud.ac.uk.

http://eprints.hud.ac.uk/



## Peripheral Oedema and Venous Disorders

### The Institute of Chiropodists and Podiatrists March 2010

Liza Dunkley Senior Lecturer

Peter Roberts

# By the end of this session you will be able to:



- understand the causes of Peripheral Oedema
- differentially diagnose between local oedema and systemic causes
- recognise the clinical signs and symptoms of DVT, thrombophelebitis, and other causes of peripheral oedema
- Appreciate the podiatric implications and management strategies for patients with peripheral oedema

#### Peripheral Oedema



- Balance between extracellular and intracellular fluid is disturbed
- Changes of extracellular fluid volume
- 50% of body weight
  - 75 kg man 42 litres 2/3 intracellular 1/3 extracellular
- Interstitial fluid & blood plasma
- Na CI extracellular

#### Systemic Causes



- Heart failure
- Kidney impairment / failure
- Liver cirrhosis /failure
- Hypoalbuminaemia
- Idiopathic oedema women
- Localised oedema

#### Causes



#### Lifestyle

- Immobility
- Hot weather
- Exposure to high altitudes
- Burns
- Menstruation
- Pregnancy,
- Medication such as high blood pressure medicine and corticosteroids
- High Salt intake

Inspiring tomorrow's professionals

#### **Systemic**

- kidney disease
- heart failure
- chronic lung disease
- thyroid disease
- liver disease
- malnutrition.



- Cellulitis
- Ischaemia
- Muscle rupture compartment syndrome









#### What would you expect to occur if drainage was impaired?

## Drainage/Removal of waste products











•The lymphatic system is a network of closed but permeable vessels containing valves

•The function of the lymphatic system is to clear interstitial fluid from tissues

#### Lymph Vessels







#### Travels to the sub-clavian vein





Inspiring tomorro

#### Lymphatic Pathology



- Fluid accumulation can arise due to the absence, damage to, or obstruction of lymphatic vessels affecting the transporting capacity of the system
- When fluid formation is increased to a point that it overwhelms lymphatic absorption oedema occurs





#### Lymphoedema



- Can present as unilateral or bilateral
- Is local or systemic in origin
- Soft and pitting or firm and woody
- Oedema presents primarily on the dorsum of the foot and disappears overnight
- As the condition becomes chronic oedema extends proximally, in severe cases to the thigh

#### Lymphoedema





#### Classifying Lymphoedema



- Primary (congenital)
- Secondary (acquired)

# Primary Lymphoedema

Idiopathic/ Milroy's Disease



#### •Congenital Lymphoedema (autosomal dominant trait)

# •Characterised by chronic lymphatic obstruction



# Secondary Lymphoedema

Acquired



Intrinsic

- malignancy
- radiotherapy
- surgery
- infection
- congenital conditions

Extrinsic

- trauma
- plaster cast
- obesity

# **Presentation**



- Initial soft swelling of the foot and/leg
- Over time this becomes firmer and heavier
- Skin becomes thickened and keratinised, may see lichenification
- May weep oedematous fluid
- May see secondary superficial bacterial infections and cellulitis

#### **Clinical Diagnosis**



- Depends on history of symptoms and on characteristic skin changes termed "elephantiasis"
- Speed of onset
- Differential diagnoses include:
  - Venous oedema
  - "armchair legs"
  - Lipoedema

### **Grades of Lymphoedema**



- Grade 1 swelling will pit if the skin is pressed and is reduced if the limb is elevated
- *Grade 2* has much more excess fibrous tissue, so it does not pit, and is not much reduced by elevation (e.g. in bed overnight or for 2-3 days). The limb gradually 'hardens' because of the excess fibrous (scar) tissue due to the chronic inflammation
- Grade 3 Elephantiasis has gross changes to the skin

#### Kaposi-Stemmer Sign



 Skin becomes thicker, demonstrated by an inability to pinch a fold of skin at the base of the 2<sup>nd</sup> toe



#### Papillomatosis



- Skin creases become enhanced
- A warty texture develops



#### Treatment of Lymphoedema



- Swelling is treated by elevation and compression (hosiery or multi-layer bandaging)
- Manual lymphatic drainage- massage
- Meticulous skin care required
- If secondary, then need to treat underlying cause
- Severe cases surgical bypass

#### Diuretics



- Loop
- Thiazide
- Potassium sparing
- Carbonic anhydrase inhibitors

#### Refer on



- GP
- Lymphoedema clinic
- Lymphodema support groups

#### **Podiatric Implications**



- Psychosocial factors- body image
- Quality of life implications
- Reduced mobility
- Altered gait
- Footwear implications

#### **Podiatric Implications**



- Reduced tissue viability
- Increased risk of foot ulceration
- Poor wound healing
- Severe oedema can reduce arterial perfusion and sensation

#### Anatomy of a Vein (and arteries)







University of HUDDERSFIELD

#### Venous Return



- Venous return (blood flowing back to the heart from the veins) depends on pressure differences from venules (90 mmHg) to the right atrium (0 mmHg)
- Venous return is assisted by contraction of the heart, skeletal muscle pumps and respiration

#### Calf muscle pump




# **Respiratory Pump**



- During inhalation the diaphragm moves inferiorly, this results in compression of abdominal veins and blood flows upwards to the right atrium
- Pressures reverse during exhalation but valves within the veins prevent backflow





Pathologies develop when venous return is impaired thus leading to chronic venous hypertension. (The back pressure of blood in the veins is elevated)

Common pathologies include:

Varicose veins Superficial thrombophlebitis Deep vein thrombosis Venous Ulceration Chronic Venous Hypertension (From Valvular Incompetence)



- Perforator Incompetence
- Superficial venous incompetence
- Deep venous incompetence
- A combination of these three

# **Chronic Venous Hypertension**









#### Capillary fluid movement and venous hypertension





# Venous pathology flow chart





### Varicose Veins



- It is estimated that over six million people in the UK suffer from varicose veins - five million of which are women
- Onset usually reported between 20 40 years
- Varicose veins are not regarded as a particularly dangerous problem, but they can be very painful and unsightly
- However severe varicose veins can compromise tissue viability

# What are Varicose Veins?



- Dilated tortuous veins of the lower limb
- Arise due to incompetent venous valves
- Veins become enlarged and congested with blood
- Can range from minor dilatations to large grape like structures

#### Varicose Veins







# Causes of varicose veins



- Pregnancy: 70-80% of pregnant women develop varicose veins during the first trimester
- Pregnancy causes an increase in hormone levels and blood volume which can enlarge veins

# **Environmental factors**



- Occupational Standing or sitting in one position for too long, inactivity
- Diet- diet high in fat, refined sugar and low fibre is thought to be a contributory factor
- Injury Valves can be damaged by knocks
- More frequent with advancing age but can occur at any age.

# Symptoms of Varicose Veins



- Symptoms are exacerbated by long periods of standing
- Feelings of fatigue, heaviness, aching, burning, throbbing, itching, cramping and restlessness of the legs

#### Venous Insufficiency - Clinical Observations

- Ankle oedema
- Haemosiderosis
- Teleangectasia
- Varicose eczema
- Atrophie Blanche
- Venous ulceration





#### Teleangectasia (Dermal flare/ spider veins)







# Venous Ulceration: common causes



- Trauma- supermarket trolleys
- Varicose eczema- itching
- Reduced tissue viability



#### Varicose Eczema



- In the early stages the main changes are orange-brown discolouration of the skin. Later the skin surface becomes scaly, itchy and develops other features of eczema
- Weeping and infection are common which can result in venous ulceration











#### Eczema or Cellulitis?



- Eczema = Itchy
- Cellulitis = Pain



#### Infected Varicose Eczema





#### **Venous Ulceration**





#### Atrophie Blanche/ Lipodermatosclerosis









# Management of Varicose Veins



- Exercise
- Avoid standing for long periods
- Elevate legs when resting
- Use support/elastic stockings
- Avoid constrictive clothing/crossing legs
- Surgery (Ligation, Stripping, Sclerosing)

# **Compression Hosiery**



- Compression hosiery helps counteract stasis by applying a firm continuous graduated pressure to the muscles and veins in the leg
- More pressure is exerted at the ankle to assist venous return





## Sclerotherapy











- Ligation: Tying off of a varicosity with a suture or wire ligament
- Stripping: Avulsion of the varicosity (usually indicated in severe cases)

# Stripping a Varicose Vein





#### Venous Thromboses



- Presence of a thrombus in a vein
- Superficial thrombophlebitis
- Deep vein thrombosis

#### Arterial and Venous thrombi compared University of HUDDERSFIELD

4!

	ARTERIAL	VENOUS
Clinical setting	Person with atheroma	Immobile person
Pathogenesis	Turbulent flow Damaged Endothelium	Stasis Hypercoaguable Blood
Symptoms	Sudden onset	Slow onset
Complications/ Fate	Infarction Arterial embolism	Pulmonary embolism via the heart

# **Composition of Venous Thrombi**



- Erythrocytes
- Fibrin mesh
- Few platelets

red thrombi

 Pathogenesis is stasis, increased blood coagulability and vascular wall damage - 'Virchow's triad'

### Virchow's Triad



Altered Blood	Increased cells
	Increased platelets
	Increased protein
	Decreased fluid
Altered vessel wall	Endothelial loss
	Endothelial damage
Altered flow	Stasis
	Turbulence

# **Risk Factors**



- Vein wall alterations
- Stasis (immobility, long periods of sitting/standing)
- Coagulopathies (e.g. polycythemia)
- Inherited hypercoagulability
- Obesity
- Varicose veins
- Use of oestrogen-based oral contraceptives

# Superficial Thrombophlebitis



- Inflammation of superficial vein with associated thrombotic occlusion
- Pain, tenderness, erythema, warmth
- Vein may be raised, indurated and cord like
- Treat with:
  - NSAIDs
  - Rest
  - Elevation
  - Compression
  - Antibiotics if evidence of infection

### Thrombophlebitis




# Deep Vein Thrombosis (DVT)

- May be asymptomatic
- May be variable combinations of:
  - tenderness
  - pain
  - oedema
  - warmth
  - bluish skin discoloration
  - prominent superficial veins
  - mild fever
  - Homan's sign (but don't do it)



## **DVT Treatment**



- Prevention of pulmonary embolism and chronic venous insufficiency
- Require hospitalisation
- Anticoagulant therapy
- Thrombolytic therapy

# Leg post DVT





### PBL – Picture task



- Diagnosis
- Aetiology
- Risk factors leading to the pathology (i.e. factors you would reasonably expect to find in a patients history)
- How and why you would assess the vasculature and what results you might expect.
- What your podiatric management would be (remember this includes appropriate referral)
- What health promotion strategies might you employ

## Venous Reflux Testing



- With the Doppler it is possible to test for the presence/ absence of venous reflux (an abnormal backward flow)
- Testing should be performed with the patient standing, knee slightly flexed
- Locate the Popliteal vein with the Doppler





## Venous Reflux Testing



- Compress calf for 3 seconds to cause a surge of blood
- You will hear forward flow on the Doppler
- When the valves are competent they will close and prevent any backward flow
- No further noise should be detected





• Need good plumbing and waste removal systems



#### Thank you for listening

Any Questions???

I.dunkley@hud.ac.uk