LEG ULCERS EFFECTIVE COMPRESSION PREVENTION OF PTS

Practice Nurses Programme GPCNE Rotorua 2010

Peter Chapman-Smith Skin and Vein Clinic

Whangarei – Otago - Hibiscus Coast

Leg Ulcers

1-2% public health cost annually in NZ80% are venous in origin20% arterial, diabetic, decubitus, malignant etc...



Incidence of Varicose Veins

40-70% of the population Men and women Sedentary occupations FH Hormonal- grand multips Urban > rural



Treatment of Leg Ulcers

Nursing – debridement, dressings, sepsis
 Compression – class 2 hose, layered bandaging
 Rx of underlying cause = varicose veins – CVI, chronic venous hypertension, interstitial oedema, local ischaemia and hypoxia







Bimalleolar Ulcers x10yrs. 18cm diameter



Pre

6mths post

12 mths

EVLA – hairdresser 43yrs



Compression Therapy

Graduated Compression

- Higher pressure at ankle
- Promote cephalad flow of blood

Reducing ambulatory venous pressure:
 Compress varicose veins
 Prevent pooling at ankle
 Reduce oedema

PVD can be a problem – ABIs

Tubigrip second best



Compression Classes

Compression power, height on leg
Level of compression at the ankle
European Standard

- 1 18-21 mmHg
- 2 25-32 mmHg
- 3 36-48 mmHg
- 4 48+ mmHg
- Travel 8-15mmHg





TED stockings

■ 18 mmHg

Use: prevention of DVT intra and post-operatively

Manufacturers Recommendation: "For use in the non-ambulant convalescing patient"





NOT designed for ambulant use

Dressings

Duoderm, Comfeel, Allevyn
Honey
Silver, seaweed (arginates)
Ichthyopaste

THEY ALL WORK

Frequency 1-2 x weeklyXXX saline and guaze





Long-term Complications

Varicose eczema
Thrombophlebitis
Pigmentation
Bleeding
Ulceration
DVT risk
Reduced QOL



Lipodermatosclerosis

= skin infarction

Lower venous pressure (top)

Higher hydrostatic pressure lower leg

Bluish feet, poor oxygenation and skin nutrition

VVs may not be obvious

Lateral Thigh Vein





Skin & Vein Clinic, Whangarei.Otago.Hibiscus Coast

25% incidence VVs









Normal scan, Blood flow to the heart

Skin & Vein Clinic, angarei.Otago.Hibiscus Coast Severe varicose veins. Blood retrograde flow

Rx of Varicose Veins

Surgical Non surgical – EVLA, RF, UGS

- Issues: efficacy, safety (VTE risk), scarring, recurrence, QOL, patient compliance and cosmesis
- Risk v gain
- Chronic relapsing disease
- Often not diagnosed

Varicose Veins RX Options

- None ignore them
- External Lasers
- □ Creams....."Vein Away"
- Graduated compression hose
- Surgery- ambulatory phlebectomy, stripping, flush ligation, stab avulsion, endoscopy, morcillation.
- Free injection (blind)
- UGS- US Guided Sclerotherapy
- VNUS
- EVLA
- ELLE catheter
- Combined UGS & EVLA



Surgery for VVs

Time off work 1-3 weeks
Scars
GA risks
VTE risk 5%
Recurrence post Surgery:
SFJ : 23% at 3 yrs
SPJ : 52% at 3 Yrs
Clinical recurrence 47.1% at 5 yrs, 62% at 11yrs
Physiologic recurrence 66% at 5 yrs

1.Recurrence after varicose vein surgery: A prospective long-term clinical study with duplex ultrasound scanning and air plethysmography. Andre' M. van Rij, MD, FRACS, Perry Jiang, MB, ChB, Clive Solomon, FRACS, Ross A. Christie, NZCS, and Gerry B. Hill. (J Vasc Surg 2003;38:935-43.)

2. Winterborn RJ, Foy C, Earnshaw JJ. Causes of varicose vein recurrence: late results of a randomised controlled trial of stripping the long saphenous vein. J Vasc Surg 2004;40: 634-9.

NON-SURGICAL VARICOSE VEIN TREATMENTS

Endovenous Laser Ablation (EVLA)

Ultrasound Guided Sclerotherapy (UGS)

EVLA FU data

•97.5% closure at 1 yr (810nm diode)
•93.5% closure at 2 yrs, 3yrs
•Recurrences mostly in the first 3-9mths

•5yr data 1320nm Whangarei – 0.001% re Rx rate

Endovenous Laser Treatment of Saphenous Vein Reflux: Long-Term Results Robert J. Min, MD, Neil Khilnani, MD and Steven E. Zimmet, MD . 2003

SCLEROTHERAPY

Injecting a foreign substance into a vessel lumen causing endothelial damage , with or without thrombosis, inducing total FIBROSIS of the vessel.

Non Surgical RX UGS, EVLA

Popular Cheaper – procedure, time off Safer- low VTE risk (1:1500); no GAs, nerve damage, scars Immediately ambulant – RTW stat 1st choice Rx most Western countries

Combined EVLA and UGS best

Easily repeatable



Vein symptoms are not related to vein size













Pre UGS

30 days

2 yrs





Deep Vein Thrombosis and Pulmonary Embolism

Incidence of DVT

□ 1-2 per 1000 per year ■ 2/3 are DVT ■ 1/3 PE Risk doubles every decade after age 40 Major complications Post thrombotic syndrome (PTS) Death (PE) Bleeding (Warfarin)

Symptoms of DVT

Swelling
Pain, tenderness
Pitting oedema
Distension of superficial vessels
Positive Homan's sign
Shortness of breath
Cutaneous erythema



Risk Factors

Surgery	20%
Trauma	12%
PHx - DVT / PE	25%
Immobility (Hospital or Nursing Home)	8%
Lower Extremity paresis	3%
Cancer	4-6%
Hormone replacement therapy	2%
Oral Contraceptive pill	3%
Inherited Thrombophilia	
 Factor V Leiden (>50%) 	
 Protein C, S deficiency 	
Lupus	

- Pregnancy
- Heart Disease
- Obesity
- Sepsis
- Age
- □ Gender (Female > Male)
- Sedentary occupation



- 2 million cases DVT in USA pa
- PE 300,000 deaths pa in USA
- PE : leading cause of hospital preventable death
- 50% chance of PE with proximal DVT (?sx)
- 3-5% chance of PE with distal DVT
- DVT risk factors- PH DVT, FHx thrombophilia
- L term risk post thrombotic syndrome

Virchov's Triad

- Blood Vessel Wall (endothelium)
- Blood Flow smooth, turbulent, slow etc
- Blood factors as "Hypercoagulability"



Hypercoagulability

Recent hx: trauma, or surgery

- Thrombocytosis
- Activated platelets
- Elevated procoagulant proteins F VIII, fibrinogen (consider tissue factor)
- Reduced anticoagulant proteins AT3, plasminogen activator inhibitor.
- Blood flow venous stasis, local hypercoagulability

Generalised hypercoagulability ("thrombophilia") – congenital inherited acquired

Thrombophilia

Predisposition to venous thrombosis (rarely arterial)
Clinically: unprovoked DVT,PE ^{+/-} FHx
Laboratory: deficiency anticoagulant factors (usually hereditary)
or added procoagulant factors (usually transient and acquired)

Thrombophilia Blood Screen

- Antithrombin III High
- Protein C -
- Protein S Low
- Activated Prot C Resistance (FV Leiden) Low
- Factor V Leiden -

Low RR 8

Moderate to High

- Prothrombin Mutation (PTG20210A) Low RR 4-8
- Homocysteine high relevant ? Causal- acquired/congenital
- Lupus Anticoagulant (APLS) ? acquired , high risk
- Anticardiolipin Antibodies IgG and IgM
- Others platelets, fibrinogen, FVIII etc

30% with strong FH - no diagnosis

Suspected DVT and/or PE?

- History provoked or unprovoked
- Personal risk factors
- Clinical evidence
- Personal risk questionnaire
- Family history
- D-Dimer (useful if NEG)
- Wells Score



Imaging – duplex US, venography, pulmonary CTA



Clinical Score (Wells)

	Score
Active cancer	1
Paralysis or recent plaster immobilisation	1
Recent immobilisation for more than 3 days or major surgery within 12 weeks requiring general or regional anaesthesia.	1
Localised tenderness along the deep venous system	1
Entire leg swollen	1
Calf swelling (circumference >3cm more than normal side measured 10cm below tibial tuberosity)	1
Pitting oedema confined to the symptomatic leg	1
Collateral superficial veins (non-varicose)	1
Alternative diagnosis as likely or greater than that of DVT	-2



D-dimer

- Byproduct of Fibrinolysis
- Diagnoses thrombotic activity
- Non-specific in diagnosis of DVT
 - -ve D-dimer = DVT unlikely
 - +ve D-dimer = DVT or other coagulable state
- Other conditions cause raised D-dimer
 - Active cancer
 - Pregnancy
 - Infection
 - Post-surgery
 - Inflammatory processes
 - Trauma

Clinical Assessment

- Large differential diagnosis
 - Ruptured Baker's cyst
 - Cellulitis
 - Haematoma
 - Compartment syndrome
 - Superficial thrombophlebitis
 - Lymphoedema
 - CHF
 - Adenopathy
- Need standardised procedure...

Treatment of DVT,PE

- LMWH (Clexane)
- Start Warfarin same time
- Aim INR of 2-3 in 48hrs
- Optimum therapeutic effect 4-5 days, then stop LMWH
- Continue Warfarin for 3 6 months
- Monitor INR not >3 usual
- Consider risk of re thrombosis

Prevention of Post Thrombotic Syndrome, PTS



Healed skin ulcer and Chronic postthrombotic pigmentation and pos

Chronic (left) leg swelling, skin hardening, and postthrombotic pigmentation

Post Thrombotic Syndrome

Reported in 15-50% of patients with DVT
 Severe PTS 5% at 10 year follow up
 Higher thrombotic load (proximal DVT) higher risk
 PTS increases the risk of further VTE.

Symptoms of PTS

- Pain
- Oedema
- Hyperpigmentation (7-23%)
- Ulceration (4-6%)
- Lipodermatosclerosis (champagne glassieg)
- Heaviness
- Cramps
- Itchiness
- Numbness or tingling
- Dilatation of superficial veins
- Redness



PTS mechanism

Chronic mechanical problem- CVI, damaged valves. Reduced blood flow, swelling, and limb pain. Leg pain and swelling = post thrombotic syndrome.



PTS incidence

- Chronic complication of acute DVT
- Multicentre study
- □ 1 yr cumulative incidence 25%, severe PTS 7%
- Obesity increased risk (RR 1.5)
- Proximal DVT increased incidence (RR 1.3)
- Women more common (RR 1.5)
- □ Less common over 60yrs of age
- Reference: Risk factors for post-thrombotic syndrome in patients with a first deep venous thrombosis. Journal of Thrombosis and Haemostasis
 Volume 6, Issue 12, Date: December 2008, Pages: 2075-2081
 L. W. TICK, M. H. H. KRAMER, F. R. ROSENDAAL, W. R. FABER, C. J. M. DOGGEN

2 year RCT 2003

■ 180 patients

- Class 2 graduated compression hose
- 1-2 years post DVT
- **Significant** reduction of PTS risk of up to 50%

Recommendation: Prescribe compression therapy for DVT.



Level 1 evidence that wearing class 2 or 3 compression hose for 2 years will reduce the incidence of PTS by 50%, and reduces the severity of PTS.

Bernardi E and Prandoni P. The post-thrombotic syndrome. Current Opinions in Pulmonary Medicine 2000;volume 6:pages 335-42.

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Sanjeev Chunilal, Hematology, North Shore Hospital, Auckland

www.vascular.co.nz

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Compression hose should be worn lifelong.



www.skinandvein.co.nz

0800 1 4 VEINS Whangarei, Hibiscus Coast, Queenstown

VASCULAR ULTRASOUND NORTH

vasultra@clear.net.nz

67 Maunu Rd, Whangarei