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Comparison of pain in two methods of varicose vein surgery

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Abstract

Lower limb DVT is a common clinical entity and a clear history of DVT is found in about 20% of patients with CVU. In addition, an unknown and probably unquantifiable number of people develop asymptomatic DVT without ever knowing it. The presence of thrombus within a vein, especially where it is occlusive, leads to an inflammatory, possibly Ischaemic phlebitis. This is thought to be due to obstruction of the vena venorum and direct humoral and/or cellular damage to the endothelium. Patients presenting to hospital medical college and research institute with varicose vein to undergo surgery for varicose vein. Each patient with a typical history of symptomatic varicosities will be subjected to physical examination and preliminary investigations. Pain at 24 hours measured by visual analog scale (0: No distress, 10: Agonizing) is significantly more associated with conventional with $P < 0.001$.

Keywords: DVT, Varicose Vein Surgery, Pain

Introduction

Ageing, disability and a range of musculo-skeletal deformity can impair the ability of the calf muscle pump to expel venous blood. Loss of transverse and longitudinal arch of the foot can also impair the venous foot pump. Muscle bulk and tone are also important in the maintenance of perforator competence. Failure of perforator competence leads to calf pump inefficiency (akin to mitral regurgitation) ^[1].

Valvular reflux in the deep and/or superficial veins is present in more than 90% of patients with CVI. In approximately a third to a half of cases this is confined to the superficial system. In the remainder, both systems are affected. Furthermore, distal (crural, popliteal) reflux appears to have a greater adverse haemodynamic and clinical effect than proximal (thigh) reflux.

In this condition, loss of elastin and weakening of collagen in the vein wall, particularly around the valve commissures, leads to dilatation, separation of the valve leaflets and reflux. SMCs derived from varicose veins are more dedifferentiated and demonstrate increased proliferative and synthetic capacity than SMCs derived from normal veins.

In some circumstances, a (relatively) normal venous segment may be rendered incompetent because of excessive distending pressures elsewhere in the patient's venous system ^[2].

Lower limb DVT is a common clinical entity and a clear history of DVT is found in about 20% of patients with CVU. In addition, an unknown, and probably unquantifiable number of people develop asymptomatic DVT without ever knowing it.

The presence of thrombus within a vein, especially where it is occlusive, leads to an inflammatory, possibly Ischaemic phlebitis. This is thought to be due to obstruction of the vena venorum and direct humoral and/or cellular damage to the endothelium.

In the short term, partial or complete occlusion of the deep venous system with thrombus leads to flow through collaterals that are Valveless, thus permitting reflux of narrow diameter, so increasing the resistance to venous outflow.

Later, the thrombus undergoes 'organization' that is accompanied by an inflammatory infiltrate, neovascularization, fibrinolysis and partial or complete recanalization of the affected segment. However, even though the segment may no longer be physically obstructed it: ^[3]

- Will have no functional valves, so permitting reflux
- May pose a functional obstruction to venous outflow because of its reduced diameter and compliance.

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Physical and functional deep venous obstruction leads to the formation of collaterals and the establishment of venous return through pathological channels. Thus blood may return from the leg by being forced out from the calf deep compartment via perforators to the skin of the gaiter area and through distended so-called secondary varicose veins. Failure of haemodynamic compensation may quickly lead to the development of the post-thrombotic syndrome (PTS).

This comprises the skin changes of CVI leading to ulceration, swelling and a bursting discomfort in the leg following exercise relieved only by resting and elevating it (venous claudication).

It is generally accepted that CVI and CVU are due to sustained ambulatory venous hypertension. However, there is still uncertainty as to how a raised AVP actually leads to the pathological changes observed in the affected skin.

At one time it was believed that varicose veins contained slow flowing, 'stagnant' blood that was low in oxygen and that ulceration of the overlying skin was due to hypoxia. However, modern studies have shown that this is not the case.

P02 measurements in varicose veins in the supine position is higher than that observed in non-varicose superficial veins. Although varicose veins pO2 does fall on standing, it is still similar to that seen in normal individuals [4].

The increased oxygen tension observed in the superficial veins of legs affected by venous disease led to the suggestion that CVI, and specifically ulceration, might be associated with the development of Arteriovenous fistula which were 'stealing' blood from the skin. This hypothesis has not been supported by recent studies using microspheres and macro aggregates.

Browne and Burnand suggested that the fibrin cuffs they had observed on histological sections surrounding the dermal capillaries of skin affected by CVI were acting as a barrier to oxygen diffusion and leading to local tissue Ischaemia.

These cuffs develop because of disturbed Starling's forces that lead to the deposition of peri-capillary protein and because of the reduced fibrinolytic activity observed in both the endothelium and vein wall of CVI patients [5].

It is important to note that 'fibrin' cuff is a much more complex structure than was originally imagined. For example, it also contains abnormal depositions of laminin, Fibronectin, Tenascin and type IV collagen. The cuff, basement membrane and surrounding extracellular matrix (ECM) may have a pathophysiological role in terms of its interaction with leucocytes, cytokines, polypeptide growth factors and proteases.

Methodology

Patients presenting to hospital medical college and research institute with varicose vein to undergo surgery for varicose vein. Each patient with a typical history of symptomatic varicosis will be subjected to physical examination and preliminary investigations.

Inclusion criteria

- Symptomatic varicosis of GSV
- Insufficiency of SFJ as determined by duplex ultrasound scanning.
- Age > 18 years
- Signed informed consent
- C₂, E_p, A_s, P_r (Clinical, Etiological, Anatomical, Pathological)
- Ipsilateral Recurrent varicose veins after stripping
- SSV insufficiency

- Previous GSV thrombophlebitis
- Malignancy, renal insufficiency, Uncontrolled Diabetes mellitus, immunosuppressive medication
- Deep vein thrombosis.

Patient who satisfy inclusion and exclusion criteria are subjected to clinical examination, Duplex ultrasound and Basic pre-operative investigations for surgery.

Clinical examination

History and examinations were completed as mentioned in the proforma. The patient was examined in standing position with good illumination, exposing both the lower limbs completely.

Results

Table 1: Comparison of pain At 24 hour in two groups of patients studied

Pain at 24 hours	Conventional		Invagination	
	No	%	No	%
No distress	0	0.0	0	0.0
Annoying	1	3.3	9	30.0
Uncomfortable	13	43.3	19	63.3
Dreadful	16	53.3	2	6.7
Horrible	0	0.0	0	0.0
Agonizing	0	0.0	0	0.0
Total	30	100.0	30	100.0

Pain at 24 hours measured by visual analog scale (0: No distress, 10: Agonizing) is significantly more associated with conventional with P<0.001**

Table 2: Comparison of pain at 48 hour in two groups of patients studied

Pain at 48 hours	Conventional		Invagination	
	No	%	No	%
No distress	1	3.3	10	33.3
Annoying	19	63.3	19	63.3
Uncomfortable	10	33.3	1	3.3
Dreadful	0	0.0	0	0.0
Horrible	0	0.0	0	0.0
Agonizing	0	0.0	0	0.0
Total	30	100.0	30	100.0

Pain at 48 hours measured by visual analog scale (0: No distress, 10: Agonizing) is significantly less in Invagination group with P<0.001**

Table 3: Comparison of Parasthesia in two groups of patients studied

Parasthesia	Conventional		Invagination	
	No	%	No	%
Absent	30	100.0	30	100.0
Present	-	-	-	-
Total	30	100.0	30	100.0

Discussion

Non-operative treatment has been the primary treatment for chronic venous insufficiency and venous ulceration for decades. The goal of this treatment for ulceration is to promote healing of the existing ulcer and prevent recurrence while allowing the patient to maintain a normal ambulatory status.

- In a modern health service, it is impractical to admit patients to hospital for long periods of bed rest.
- Confining elderly patients to bed in hospital is associated with a wide range of iatrogenic complications: colonization of the ulcer with resistant bacteria, thromboembolism,

bowel disturbances and so on.

- As soon as the patient leaves hospital, if no other measures are taken, the favorable haemodynamic environment is lost and early recurrence is inevitable.
- Short periods of hospital admission for the optimization of the wound environment with intensive nursing care may be clinically beneficial and cost-effective in certain circumstances, especially prior to surgery.
- Venoprotective drugs are a heterogeneous group of naturally occurring and synthetic compounds that are believed to improve the symptoms and haemodynamic abnormalities, specifically oedema, associated with venous disease [6].
- **Horse chestnut extract:** The active ingredient is believed to be aescin, a mixture of triterpenoid saponins.
- **Flavonoids:** This group comprises rutin and its derivatives, principally O-(Phydroxyethyl) - rutosides Diosmin is a synthetic flavonoid
- **F-Calcium dobesilate, Tribenoside** – Synthetic
- **Dihydroergotamine:** Ergot derivatives are rarely used because of their narrow therapeutic index and severe side-effects.

Closure of inter-endothelial capillary pores, so reducing capillary filtration. Improving venous tone (particularly ergot derivatives). Free radical scavenging and inhibition of Lipid peroxidation, Inhibition of Leucocyte activation and the release of inflammatory mediators.

Zinc, Stanazolol and Oxypentifylline are of minimal or no benefit in the treatment of the varicose ulcers. Several studies have been done and proved that these are not very significant.

Most venous ulcers are colonized with bacteria rather than infected. When there is no evidence of clinical infection, i.e. cellulitis, systemic antibiotic therapy is not indicated.

Topical antibiotics should not be prescribed as they are frequent sensitizers.

No large, randomized controlled clinical trials have shown improvement in CVU healing using topical applications, including zinc, selenium, antibiotics and various growth factors, even though growth factor therapy has been shown to be effective in both arterial and diabetic ulceration [7].

Poor calf muscle pump function is often overlooked as an important factor in the pathogenesis of CVU. Improvement in muscle function through (supervised), regular exercise will improve the AVP even if venous reflux and/or obstruction remain unchanged.

Compression therapy has been the mainstay of treatment for CVI for at least 2000 years. Today, there remains general agreement that compression retards the development and progression of CVI, and heals CVU.

Decrease wall tension, hence damage to the skin and collagen structure of the wall. Force blood from the superficial to the deep system and prevents the reflux of high pressure blood through perforating veins.

- Reduce reflux by opposing the vein wall and restoring valvular competency, and limiting the dispensability of the calf veins and sinuses. Reduce oedema and thus skin tension.

It helps in returning Starling's forces, Haemostasis and Leucocyte margination at the capillary towards normal by: Preventing excessive fluid and protein filtration, Decreasing pressure in the post-capillary venule, Augmenting lymphatic

clearance of excess filtrate augmenting the release of prostacyclin and plasminogen activator from the endothelium. Although both work when the patient is ambulant, Elastic compression exerts compression even in the supine position and tends to exert more sustained pressure. Non Elastic compression generate local areas of inappropriately high pressure over bony prominences, it also tends to give a less even pressure than elastic bandages. Randomized controlled trials have shown greater healing rates with elastic bandaging [8].

Because the shape of leg is obviously not a simple cylinder or cone, and varies enormously between individuals, large differences in pressure can be exerted by the same stocking or bandage.

Due to Laplace's law, the pressure will be highest over the shin, malleoli and Achilles tendon and lowest over the flatter areas and may be non-existent in hollows such as that found behind the medial malleolus [9].

The British Standard for compression hosiery describes four classes:

Class I: < 25 mmHg: Thrombo-embolic prophylaxis or early varicose veins

Class II: 25-35 mmHg: Advanced varicose veins, oedema, early CVI

Class III: 35-45 mmHg: Moderate to severe CVI, lymphedema

Class IV: > 45 mmHg: Severe CVI and lymphedema.

For most CVI patients class II compression represents a reasonable compromise between efficacy, compliance and patient comfort.

Compression should commence at the metatarsal heads and in most cases terminate at the tibial tuberosity. There is no evidence that extending compression above the knee confers benefit in patients with CVI [10].

The advantages of a multi-layer over a single layer bandaging system include:

- The shape of the leg ensures that graduated compression (40 mmHg ankle, 15-20 mmHg upper calf) will be obtained.
- Multi-layer bandaging little pressure is lost following application, even over a few days. The bandaging only needs to be changed once or twice weekly

Perhaps the single most important layer is the inner wool as it evens out the pressure, protects prominences from damage and absorbs moisture. Drawbacks of multi-layer, elastic bandaging include increased cost and the danger of exerting excessive pressures.

Conclusion

Invagination of long saphenous vein results in significantly less post operative pain as compared to conventional stripping of long saphenous vein.

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