Background

LEG oedema, or swelling of the legs, is common and often poses a difficult diagnostic dilemma because of the diverse nature of its possible causes. Patients seek medical advice for a variety of reasons, including pain and inability to walk.

The distribution of oedema is an important guide to the underlying cause. The most important initial question is whether the oedema is localised or generalised. LEG oedema may be either unilateral or bilateral. Bilateral swelling is more commonly due to systemic conditions whereas unilateral swelling more often represents local pathology.

Table 1 lists the most common causes of leg oedema that present to GPs today. Local causes are usually related to venous or lymphatic disease. Systemic causes include heart, liver and kidney dysfunction.

Management is based on the underlying cause, so an accurate and timely diagnosis is paramount. Investigations should be directed to the postulated aetiology but should also be geared to excluding other possibilities. This article discusses the management of common local causes of leg oedema.

Systemic
- Cardiac dysfunction:
  - left ventricular systolic impairment: ischaemic heart disease, valvular disease
  - left ventricular diastolic dysfunction: hypertension
  - right ventricular dysfunction (pericardial effusion, chronic cardiac failure, end-stage chronic airflow limitation)
- Fluid overload
- Renal disease:
  - acute renal failure (eg, acute glomerulonephritis)
  - chronic renal failure
- Hypoproteinaemic states:
  - chronic liver disease
  - nephrotic syndrome
  - malnutrition
  - protein-losing enteropathy
  - malabsorption
- Endocrine:
  - hypothyroidism
  - Cushing's syndrome
- Drugs:
  - calcium-channel antagonists
  - corticosteroids
  - NSAIDs
  - oestrogens
  - Anaemia
- Others:
  - hereditary angioneurotic oedema
  - pregnancy
  - idiopathic
- Local
  - Venous:
    - acute DVT
    - post-thrombotic sequelae (eg, venous incompetence, venous hypertension)
    - varicose veins
    - obstruction to venous return (eg, pregnancy, pelvic tumours, inferior vena cava obstruction)
  - Lymphatic:
    - primary lymphoedema
    - secondary lymphoedema
  - Stasis:
    - paraesthesia
    - poor mobility
    - obesity
    - Inflammation:
      - cellulitis
      - allergic reactions
    - Trauma
    - Congenital:
      - arteriovenous malformations

Table 1: Causes of leg oedema

Local causes of LEG OEDEMA

The authors

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Deep venous thrombosis

DEEP venous thrombosis (DVT) is a prominent cause of lower limb swelling. It tends to be unilateral and the extent, site and number of previous episodes will determine the degree of swelling. DVT can cause two clinical scenarios: acute DVT and post-thrombotic syndrome (PTS).

Risk factors

There are many independent risk factors for DVT and pulmonary embolism (PE); however, it is not until there is a combination of these that a DVT will occur. Risk factors include:

- Increasing age.
- Male gender.
- Obesity or trauma.
- Hospital or nursing home confinement.
- Malignancy.
- Neurological disease with extremity pain.
- Central venous catheter or transvenous pacemaker.
- Prior superficial vein thrombosis.
- Varicose veins.
- Pregnancy, oral contraceptives and HRT can all increase the risk for women. The relative risk increases progressively from 2.4 in those with one risk factor to more than 20 among those with three or more risk factors.

In the acute setting, signs and symptoms vary with the cause of symptoms and signs alone is inaccurate; in up to half of patients with classic clinical findings, a DVT is not confirmed by diagnostic testing. Furthermore, up to half of all patients with acute DVT may lack any specific sign or symptom.

Investigations

Unfortunately, the diagnosis of DVT based on clinical signs and symptoms alone is inaccurate; in up to half of patients with classic clinical findings, a DVT is not confirmed by diagnostic testing. Furthermore, up to half of all patients with acute DVT may lack any specific sign or symptom.

Contrast venography has historically been the gold standard for diagnosing acute DVT. However, venography is inconvenient, expensive and may cause significant discomfort.

Deep vein thrombosis

PTS is the residual effect in the limb from the venous hyper tension caused by residual obstruction and resultant incompetence from the destruction of venous valves. It typically occurs at least 10 years after the initial episode of DVT.

The symptoms of pain, oedema, hyperpigmentation and/or ulceration are typical of PTS, which occurs in 29-79% of patients with a history of DVT. There is no clear relationship between the extent of thrombosis or symptoms on initial presentation and ultimate clinical outcome.

Studies have found that the risk factors for PTS appear to be ipsilateral recurrence of DVT, poor quality of initial anticoagulation for the treatment of DVT, and increased BMI. Factors that reduce the risk of development of PTS are early thrombolysis, early effective anticoagulation and the long-term wearing of graduated calf-compression stockings.

Renal trauma.

The high sensitivity of D-dimer measurements makes it theoretically possible to exclude a diagnosis of DVT on the basis of a negative result, as long as the patient is in a low-risk group (ie, outpatients with no previous DVT, no PE symptoms).

Patient with recurrent DVT/PTS involving more than the tribial vessels should be evaluated for a thrombophilia.

Anticoagulant therapy for venous thromboembolism

The objectives of treatment in patients with venous thromboembolism (VTE) are to prevent death from PE, prevent recurrent VTE and prevent progression.

The accepted anticoagulant therapy for VTE is either continuous IV heparin or subcutaneous (SC) enoxaparin (Clexane) then changing to oral warfarin. Heparin or enoxaparin is continued until the INR has been within the therapeutic range (2.0-3.0) for two consecutive days.

Calf vein thrombosis

Isolated calf vein thrombosis is usually considered relatively low risk for causing PE. However, this risk is not nil, and proximal propagation (occurring in up to 30% of patients) significantly increases the risk for PE and PTS.

Debate still exists on whether to anticoagulate these patients; however, patients with a calf DVT have been shown to benefit from 6-12 weeks of anticoagulation. If anticoagulation is not given, an antiplatelet agent such as low-dose aspirin (100mg daily) should be started and graduated compression stockings used. A repeat venous duplex scan should be ordered within 5-7 days to determine progression.

Femoropopliteal venous thrombosis

Anticoagulation is standard therapy but does not dissolve the clot. However, circumstances of effective anticoagulation, natural thrombolysis may at least partially recanalise occluded veins. Even without recanalisation, morbidity may be minimal if thrombosis is limited to the femoral vein in the thigh. This treatment can be for 6-6 months, depending on the patient’s risk.

Iliofemoral thrombosis (proximal DVT)

Patients with proximal thrombosis are usually symptomatic. Characteristically these patients have thigh and calf oedema and pain on both rest and ambulation. PE from these large veins may be fatal.
**Full Text**

**How to treat — leg oedema**

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These patients need immedi-
ate anticoagulation with IV heparin, followed by warfarin for at least 12 months. The young physically active patients, with previous thrombosis should be consid-
ered for catheter-based throm-
bolysis followed by anticoagulation to lessen the long-term morbidity of DVT. It has been demonstrated with early clot clearance by either thrombectomy or catheter-
directed thrombolysis has better long-term outcomes than anticoagulation alone.

**Long-term treatment of VTE**

In addition to local manage-
ment, patients with recurrent VTE or continuing risk fac-
tors such as cancer, antithrombin deficiency or the antiphospholipid syn-
drome should be treated indefinately with warfarin (unless contraindicated).

Patients with activated protein C resistance (factor V Leiden mutation) should probably receive indefinite treatment if they have recur-
rent disease, are homozy-
gous for the gene or have multiple thrombotic conditions.

Primary valvular incompetence

- Pain or discomfort.
- Venous pain.
- Skin pigmentation.
- Ulceration.
- Itching.
- Pain.
- Tiredness.
- Skin discoloration.
- Venous ulceration (at the most severe form).

Symptoms and signs specific to abnormal venous function arise from chronic ambulatory venous hyper-
tension, and are known as chronic venous insufficiency (CVI). CVI is now thought of as a disease of chronic inflammation due to a sus-
tained injury secondary to venous hypertension.

The primary injury is extravasa-
tion of macromolecules and ery-
throcytes into the dermal intersti-
cial. Enzymes, growth factors, and inflammatory products and interstitial proteins are potent chemotactants and pro-

General indications

- Deep venous thrombosis or documented thromboembolism in a patient with a contraindication to anticoagulation.
- Complications of anticoagulation that have forced therapy to be discontin-
ned.
- Recurrent pulmonary embolism despite adequate anticoagulation.
- Prophylactic placement in high-risk patients.

**Relative indications**

- Demonstrated free-floating Iliofemoral thrombus.
- A propagating iliofemoral thrombus despite adequate anticoagulation.
- Septic pulmonary embolism.
- Chronic pulmonary embolism in a patient with pulmonary hypertension and cor pulmonale.
- A patient with >50% occlusion of the pulmonary vascular bed and who would not tolerate any additional thrombolysis.
- Severe atrial and risk of falling.

**How to treat — leg oedema**

**Venous insufficiency**

**Table 2: Indications for insertion of a vena caval filter**

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**Table: Differential diagnosis of chronic venous ulcers**

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*Source: Padberg 2005*

**How to treat — leg oedema**

**Histology**

- Suppuration, is almost completely accepted as effective treatment for venous ulceration.

**How to treat — leg oedema**

**Compression therapy**

Compression therapy remains the pri-
mary treatment for CVI. It promotes fluid resorption and resolution of oedema, with improved diffusion of nutrients to the skin and subcuta-
aneous tissues.

**Pharmacological therapy**

Despite the oedema, diuretics have no role in the treatment of CVI. Zinc has been found to be of bene-
fit in the healing of venous ulcers, but only if pre-treatment zinc levels are low, and not in all studies.

**Varicose veins**

**VARIOSCE. venae, or super-
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cial venous incompetence, can lead to CVI. Patients with lower-limb oedema and superficial venous incompetence can be treated by sur-

gical or endovascular laser therapies.**

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*Source: Padberg 2005*
green reticular veins are not normal physical findings. They are evidence of venous dysfunction. Telangiectasias are defined as a confluence of permanently dilated intradermal venules of <1mm in diameter. Reticular veins are defined as permanently dilated, bluish intradermal veins, usually 1mm to <3mm in diameter. Longstanding venous dysfunction causes marked skin changes, from woodiness and fibrosis to hypodermosteocclerosis (from haemosiderin deposition) and atrophy blanche (a consequence of scarring from multiple episodes of skin breakdown and poor healing occurring in severe disease).

The aching pain is related to pressure of the dilated vessels on a network of somatic nerve fibres in adjacent subcutaneous tissue and the oedema caused by a fluid shift from venous to subcutaneous compartment by venous hypertension.

### Treatment

Extended availability of duplex ultrasonography has revealed another reason for intervening in varicose veins, in addition to those listed in Table 4 — deep venous function is improved by superficial venous surgery.

Doppler evaluation and duplex scanning have shown that saphenofemoral junction reflux is present in 70% of symptomatic limbs selected for surgery. In such limbs the saphenous vein at the femoral junction must be treated.

About 15% of patients are found to have short saphenous vein incompetence, which requires flush ligation and removal of some of the vein.

Surgical removal of the long saphenous vein is now performed by the stripping or laser ablation of the vein from the groin to the knee. Supplement ablations or sclerotherapy are used to remove other clusters of varicose veins. Sclerotherapy without addressing the saphenofemoral or saphenopopliteal reflux will fail.

Recent developments in treatment of the long saphenous vein have focused on attempting to minimise postoperative discomfort while maintaining the benefits of saphenous vein ablation. Endovascular laser treatment can be performed using local anaesthesia only, without a groin incision. It causes rapid thermal electrocautery of the vein wall and its valves, which in turn causes total loss of vessel wall architecture, disintegration and carbonisation.

There is now level I evidence indicating that such treatment is beneficial. At 1-3 years, reported results from ablation of the saphenous vein are as good as those from conventional surgical treatment, and clinical observations suggest that patients are much more comfortable after saphenous ablation than after stripping.

### Surgical treatment of chronic occlusions of the iliac veins and the inferior vena cava

Apart from DVTs, venous occlusion may also develop because of trauma or irradiation, or as a result of external compression of deep veins by retroperitoneal fibrosis.

- **Abnormally inserted muscle** (popliteal vein entrapment).
- **Fibrous bands or ligaments** (solar arch syndrome, femoral vein compression by the inguinal ligament).
- **May-Thurner syndrome** (compression of the left common iliac vein by the overriding right common iliac artery) is a rare but frequently overlooked cause of left iliofemoral venous thrombosis.

The approach to any of the above causes of proximal venous occlusion is to consider endovascular techniques first, as the advances being made in this field are significantly decreasing the number of patients requiring open surgical reconstruction. Endovascular techniques involve relatively open techniques of vein or synthetic grafting bypass the affected vein.
Lymphoedema

THE lymphatic vasculature consists of initial lymphatics, the lymphatic pre-collectors, lymphatic ducts and lymph nodes. The superficial system collects lymph from the skin and subcutaneous tissue and the deeper system drains sub-fascial structures. When lymphatic vessels are absent, underdeveloped or obstructed, lymphatic flow from the extremities is impeded and lymph accumulates. The resultant clinical presentation, lymphoedema, can affect any part of the body but tends to target the arms and legs. About 300,000 Australians have lymphoedema at any given time.

Classification

The simplest classification requires differentiation between primary and secondary causes. Impedence to lymphatic flow may be due to an inborn defect (primary) or an acquired loss of lymphatic potency (secondary) (table 5).

Primary lymphoedema

Primary lymphoedema can be classified either according to the age of the patient when the oedema first appears, or by its lymphangiographic appearance.

Patterns of lymphatic dysfunction

There are four patterns of lymphatic dysfunction:
- Hypoplasia (or obstruction) of lymphatics to the inguinal nodes.
- Obstruction of pelvic vessels, with or without obliteration of the iliac system.
- Hyperplasia of lymphatic vessels.
- Underdeveloped, dilated lymphatic channels (megalymphatics).

The latter two patterns of disease are usually associated with a history of blockage or obstruction of the lymphatic system, be it related to primary lymphoedema or obstruction of the distal lymphatic system by relative lymphoedema of the proximal vessels. Swelling is usually bilateral and mild and women are affected more frequently. The prognosis is good; after the first year of symptoms there is usually minimal or no further extension in the same limb or to uninolved extremities. However, in about 40% of patients, affected limb growth continues to increase gradually.

In more than 50% of cases the defect primarily involves obstruction of the proximal lymphatics or nodes. There is possibly a slight female predominance. Swelling tends to be bilateral and severe, and the extent and degree is more likely to progress, with subsequent involvement of the distal lymphatics.

A minority of patients have bilateral lymphoedema. About 10% of the lymphatics or tortuous dilated inconstant lymphatics (megalymphatics), with a male predominance. There is primary lymphatic valvular incompetence and an increased number of lymphatic channels, with variations of the major collecting vessels. Megalymphatics are associated with a greater extent of involvement and a worse prognosis.

Age of first appearance

Congenital lymphoedema is apparent at birth or becomes recognised during the first two years of life. It represents about 15% of primary lymphoedema cases. There is typically aplasia or hypoplasia of lymphatic structures. In rare cases involvement of the multiple limbs, genitalia or facial structures may be seen.

Lymphoedema praecox is most commonly first detected at puberty but its appearance may be delayed until the third decade. It comprises about 50% of cases and is typified by a hypoplastic lymphangiographic pattern, usually beginning with foot and ankle swelling.

Lymphoedema tarda typically manifests after age 35. The lymphatic channels may be hypoplastic or hyperplastic.

A family history is evident in a minority of cases. The congenital autosomal dominant form is known as Milroy’s disease. The term Milroy disease is applied to the praecox familial form, also autosomal dominant. Autosomal recessive forms can sometimes occur.

Primary lymphoedema is also associated with Turner syndrome, Noonan’s syndrome, yellow-nail syndrome, intestinal lymphangiectasia, lymphangiomatosus and arteriovenous malformations.

Limb swelling may be the presenting and major manifestation of congenital lymphoedema, either in a pure form (e.g. diffuse lymphangiosis) or in combination with a congenital vascular syndrome, such as Klippel–Trénaunay syndrome (varicose veins, excessive long bone growth and vascular birthmark).

Secondary lymphoedema

Secondary lymphoedema results from disruption or obstruction of previously normal lymphatic pathways by other disease processes or as a consequence of surgery or radiotherapy. It is much more common than primary lymphoedema.

Infections

Recurrent episodes of bacterial lymphangitis cause obstruction of the lymphatics and fibrosis of the draining lymph node. It is almost always due to streptococci (predominantly beta-haemolytic strains) although Staphylococcus aureus and Gram-negative anaerobes have also been implicated. In addition, recurrent lymphangitis is a common complication of lymphoedema. Repeated bouts of cellulitis may also exacerbate the chronic oedema seen in CVI.

Tumour

Malignant cells may obstruct lymphatic vessels, inducing lymphoedema directly or preparing the patient to bacterial lymphangiitis. The most common causes of lower-extremity malignant lymphoedema are prostate cancer and breast cancer in men and lymphoma in women.

Therapy against malignancy may also result in lymphoedema. Oedema of the leg is comparatively common after treatment of pelvic or genitourinary cancer that involves pelvic or inguinal lymph node dissection or without, with or without radiotherapy. Cancer itself rarely presents with lymphoedema except in advanced cases presenting late, such as prostate cancer, where venous obstruction may coexist. Relapsed tumour should always be considered in those with swelling after apparent curative cancer treatment.

Post-surgical lymph node dissection in the setting of cancer therapy, and vascular surgery, particularly femoral artery endarterectomy or lower limb bypass surgery, may cause secondary lymphatic obstruction.

Filariasis

A nematicid infection, filariasis accounts for the most cases of secondary lymphoedema worldwide. It should be considered in any patient with lymphoedema who has travelled or lived in an endemic area.

Chronic venous insufficiency

Lymphoedema may complicate CVI that has resulted in marked venous hypertension.

Other causes

Other causes associated with obstruction of lymphatic channels include:
- Trauma.
- Tuberculosis.
- Contact dermatitis.
- Rheumatoid arthritis.
- Pregnancy.
- Subcutaneous injections of drugs.
- Autoimmune destruction remains a controversial aetiology.

Clinical features

Typical clinical findings include chronic oedema with loss of normal contour sparing at the level of the ankle joint and significant foot oedema relative to the forefoot, yielding a ‘buffalo hump’ profile. The toes may be on a ‘sausage’, or ‘squared-off’, appearance, accompanied by tense oedema.

The unique changes of dermal and subdermal fibrosis (orange peel) and stemmer’s sign (an inability of the soft tissues to tent the skin of the interdigital webs) also serve to distinguish lymphoedema from other causes of chronic lower-limb oedema.

Initially there is pitting oedema, which may decrease or disappear with elevation. Increased fluid retention contributes to a sense of heaviness and early fatigue of the limb is common. The skin retains its normal texture but may appear flushed, and increased skin blood flow will result in an increased temperature of the limb. These features are quite typical of mild swelling.

Over a period of years, or with more moderate disease, the thickening of the skin and the limb may take on a woody (‘classic peau d’orange’) appearance. Pitting is no longer apparent. The oedema may extend more proximally and may not diminish at night with elevation.

Pain is generally absent, but in the presence of lymphangiitis the limb may become acutely painful, with a diffuse, sharply defined burning sensation.

In advanced stages, changes are characterised by marked thickening and coarseness. The skin is grossly enlarged, skin creases become enhanced, the limb takes on a mossy, cobblestone appearance with a warty texture (hyperkeratosis) and papillary thickening.

Lichenification of the toes develops and intermittent bacteriology of skin fissures, minor trauma or break down induced by interdigital fungal infection. The skin may become broken and the lymph fluid can leak out onto the surface (lympho- or lymphorrhoea).

Infections in the form of cellulitis, lymphangitis and lymphatic obstruction and ulceration may occur in advanced stages, usually resulting from concomitant venous hypertension. In addition to their inherent danger, infections further damage the lymphatic system. Very rarely, in certain exceptional cases, lymphoedema untreated over many years can lead to a form of cancer known as lymphangiosarcoma.

Lymphoedema manifests with sudden onset of swelling of a whole leg suggesting proximal obstruction, and a search is made for a secondary cause. Pelvic causes of lymphatic obstruction such as tumour must be excluded.

Diagnosis

Table 6 lists the differential diagnosis of lymphoedema. CVI and PTS resulting in chronic venous hypertension may be confused with lymphoedema. However, the fluid distribution in CVI and PTS is different, with fluid in the ankle area and least over the toes. Chronic venous hypertension is typically associated with:
- Aching discomfort.
- Dull pruritus.
- Hypertension.
- Dusky discoloration, with dependence, lipodermatosclerosis and subcutaneous fat necrosis (panniculitis).
- Ulceration (if advanced).

Myxoidema may occur in relation to thyroid dysfunction when abnormal depositions of mucinous material accumulate in the skin. In thyrotoxicosis, the process is focal, whereas in hypothyroidism, it affects the entire skin and is characterised by:
- Roughening of the skin.
- Delicate thinning hair.
- Yellow-orange discoloration of the skin.
- Reduced sweat production.
Lipoedema is the accumula-
tion of fluid and fat deposits under the skin, generally in the legs. It almost exclusively occurs in females and nor-
mally appears during puberty or pregnancy. The size of the extremities cannot be reduced by losing weight. The legs may be very tender and ache around the knees. It charac-
teristically spares the feet and does not change the appear-
ance of the skin, which distin-
guishes it from lymphoedema. People with lipoedema also bruise easily.

Malignancy can cause the appearance or worsening of lymphoedema. There is a ten-
dency for rapid development and relentless progression, and pain may be a feature.

Diagnosis

In the patient with an oede-
matus extremity in which lymphoedema is suspected, diagnostic studies are used to establish the diagnosis, assess lymphatic function and doc-
ument the degree and severity of lymphoedema.

When the physical exami-
nation does not conclusively support the diagnosis of lym-
phoedema (typically in mild cases), additional objective evi-
dence is required to confirm lymphatic dysfunction. Initial investiga-
tors relied on lym-
phangiography but this pro-
cedure has now been largely replaced.

Isotopic lymphoscintigraphy and ultrasonic imaging is now the investigation of choice for identifying oedema of lymphatic origin. Radio-
labelled colloid or protein is injected into the first web space of each foot and moni-
tored using a gamma camera. Measurement of tracer uptake within the lymph nodes after a defined interval will distin-
guish lymphoedema from oedema of non-lymphatic origin.

In primary lymphoedema, the channels are obliterated or absent; in 10% they are lac-
tic and incompetent. In secondary lymphoedema the channels are dilated and the level of obstruction can be determined. In any form of lymphoedema, the appear-
ance of tracer outside the main lymph routes, particu-
larly in the skin (dermal back-
flow), indicates lymph reflux and suggests proximal pro-
gression is delayed. Poor trans-
port of isotope from the injec-
tion site suggests hypoplasia of the peripheral lymphatic system.

Direct contrast X-ray lympho-
graphy (lymphangiography)

After the lymph vessels have been identified with a viral marker, the subcutaneous lymph channels are administrated directly into a lymphatic vessel. This form of investigation is used rarely.

CT and MRI

Both CT and MRI detect a characteristic ‘honeycomb’ pattern in the subcutaneous compartment that is not seen with other causes of oedema. In lymphoedema, there is the char-
acteristic absence of muscle involvement in lym-
phoedema. MRI is more informative than CT because it can detect water. CT cannot localise the level of obstruction or differentiate surgically correctable lymphoedema from that which is not.

Table 7: Treatment of lymphoedema

<table>
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<td>Proper skin care — topical emollients</td>
<td></td>
</tr>
<tr>
<td>Manual lymphatic therapy</td>
<td></td>
</tr>
<tr>
<td>Multi-layer, short-stretch bandaging</td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td></td>
</tr>
<tr>
<td>Well-fitted compression garments</td>
<td></td>
</tr>
</tbody>
</table>

Surgical

- Lymphangioplasty with enteromesenteric flap grafts to bridge obstructed lymphatic segments.
- Lymphovenous shunt (anastomosis of lymphatic to vein).
- Lymphorrhaphy with enteromesenteric flap onmental transfer (pedicled portion of omentum trans-
posed to the affected limb).

References, further reading and online resources

- www.australiandoctor.com.au

Drugs

Surgical therapy is reserved for

- Excessive tissue enlargement.
- Inability to improve function
- Severe pain
- Recurrent cellulitis
- Recurrent lymphangitis
- Recurrent lymphoedema
- Recurrence after prior treatment
- Failure of non-surgical treatment

Prophylaxis

- Proper skin care
- Manual lymphatic drainage
- Pressure garments
- Compression pump therapy
- Exercise
- Corrective surgical therapy

Prevention

- Early detection and management
- Early intervention
- Regular exercise
- Early compression

Drugs

- Diuretics
- Corticosteroids
- Non-steroidal anti-inflammatory drugs
- Anticoagulants

Treatment

- Early detection and management
- Early intervention
- Regular exercise
- Early compression

Drugs

- Diuretics
- Corticosteroids
- Non-steroidal anti-inflammatory drugs
- Anticoagulants

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How to treat leg oedema

1. Linda, 45, presents with pain and swelling of the left calf for the past few days. She has been in a below-knee cast for a left ankle surgery with a two-week standing order. She states that she has been very swollen before, with ankle swelling. The foot swelling compared to the knee level, with loss of calf contour and heart rate, cardiovas- cular examination should be performed. Clinical assess- ment of general causes focuses on blood pressure and heart rate, cardiovascular examination looking for signs of peripheral vascular insufficiency and valvular disease. Examination to exclude chronic lower limb disease as well as thyroid examination should be performed.

2. After further history and examination you suspect Linda has a DVT. Which TWO statements about investigating DVT are correct?
   a) Duplex ultrasonography is highly accurate in detecting DVT of the iliac veins as well as the lower-limb veins.
   b) D-dimer assays are highly specific as well as highly sensitive for DVT.
   c) The high sensitivity of D-dimer measurements makes it theoretically possible to exclude a DVT on the basis of a negative result.
   d) Patients with recurrent DVT or DVT's involving more than the tibial vessels should be evaluated for a thrombophilia.

3. Duplex ultrasonography confirms that Linda has an isolated calf DVT. Which THREE statements about managing DVT are correct?
   a) In the acute DVT setting, lower-limb oedema can be treated with graduated compression stockings and appropriate treatment of the DVT with anticoagulants.
   b) Accepted anticoagulant therapy for venous thromboembolism is either continuous low-molecular-weight heparin or unfractionated heparin, then changing to oral warfarin.
   c) Patients with proximal thrombus need IV or IVC filter placement followed by warfarin for up to six months.
   d) Indications for venous catheter filters include recent pulmonary embolism despite adequate anticoagulation.

4. Which TWO statements regarding the post-thrombotic syndrome (PTS) are correct?
   a) PTS typically occurs within the first two years of the initial episode of DVT.
   b) Risk factors for PTS appear to include increased BMI.
   c) PTS typically presents with pain, oedema, hyperpigmentation and/or ulceration.
   d) Wearing compression stockings long term has not been found to reduce the risk of development of PTS.

5. Which THREE statements about chronic venous insufficiency (CVI) are correct?
   a) CVI is now thought of as a disease of chronic inflammation.
   b) When examining the patient with CVI the long and short saphenous veins should be palpated and tested for a cough impulse.
   c) Investigation of CVI is best performed with venography.
   d) Raised edges, an irregular appearance, bleeding, and prolonged failure of a venous ulcer to heal raise the suspicion of a malignant ulcer.

6. Which TWO statements regarding management of CVI are correct?
   a) Compression therapy remains the primary treatment for CVI.
   b) Diuretics are very helpful in reducing the oedema of CVI.
   c) Arterial disease should be considered before proceeding with compression therapy for CVI.
   d) Topical antibiotics are useful in the treatment of venous ulceration to prevent development of infection.

7. Dolores, a 55-year-old teacher, consults you regarding long-standing bilateral varicose veins. She is troubled by aching and varicose veins. She is bothered by aching and varicose veins.

   a) Typical clinical findings in lymphoedema include concentric calf oedema with a loss of normal contour, significant hind-foot oedema; however, the fluid is non-pitting.
   b) Lymphoedema is best assessed in lymphoedema.

   c) Bacterial infections need to be treated aggressively in patients with lymphoedema.
   d) In lymphoedema tarda, which typically manifests after age 25-40, the lymphatic channels may be patent.

8. Which TWO statements regarding the treatment of lymphoedema are correct?
   a) Primary lymphoedema is much more common than secondary lymphoedema.
   b) Congenital lymphoedema is usually seen in infancy or early childhood.
   c) Lymphoedema praecox is most commonly due to primary lymphoedema.

   d) In lymphoedema tarda, which typically manifests after age 25-40, the lymphatic channels may be patent.

9. Which THREE statements about assessing lymphoedema are correct?
   a) Typical clinical findings in lymphoedema include concentric calf oedema with a loss of normal contour, significant hind-foot oedema related to the foot and a ‘sagging’ appearance of the foot.
   b) Lymphoedema can be confined to the foot with lymphoedema due to CVI or PTS; however, the fluid distribution in CVI and PTS is different, being greatest over the ankles and least over the toes.
   c) In lymphoedema manifesting with sudden onset of swelling of one whole leg, pelvic causes of lymphatic obstruction must be excluded.

   d) Direct-contrast X-ray lymphangiography is the investigation of choice for identifying lymphoedema of lymphatic origin.

10. Which TWO statements regarding the treatment of lymphoedema are correct?
    a) Combination therapy with sequential- gradient pump therapy and complete decongestive therapy results in the greatest overall reduction in limb volume.
    b) Long-stretch bandages are preferred over short-stretch bandages in compression therapy for lymphoedema.
    c) Bacterial infections need to be treated aggressively in patients with lymphoedema.
    d) Benzoylperoxide, such as coumarine or flavonoids, have been proven to be of benefit in lymphoedema.