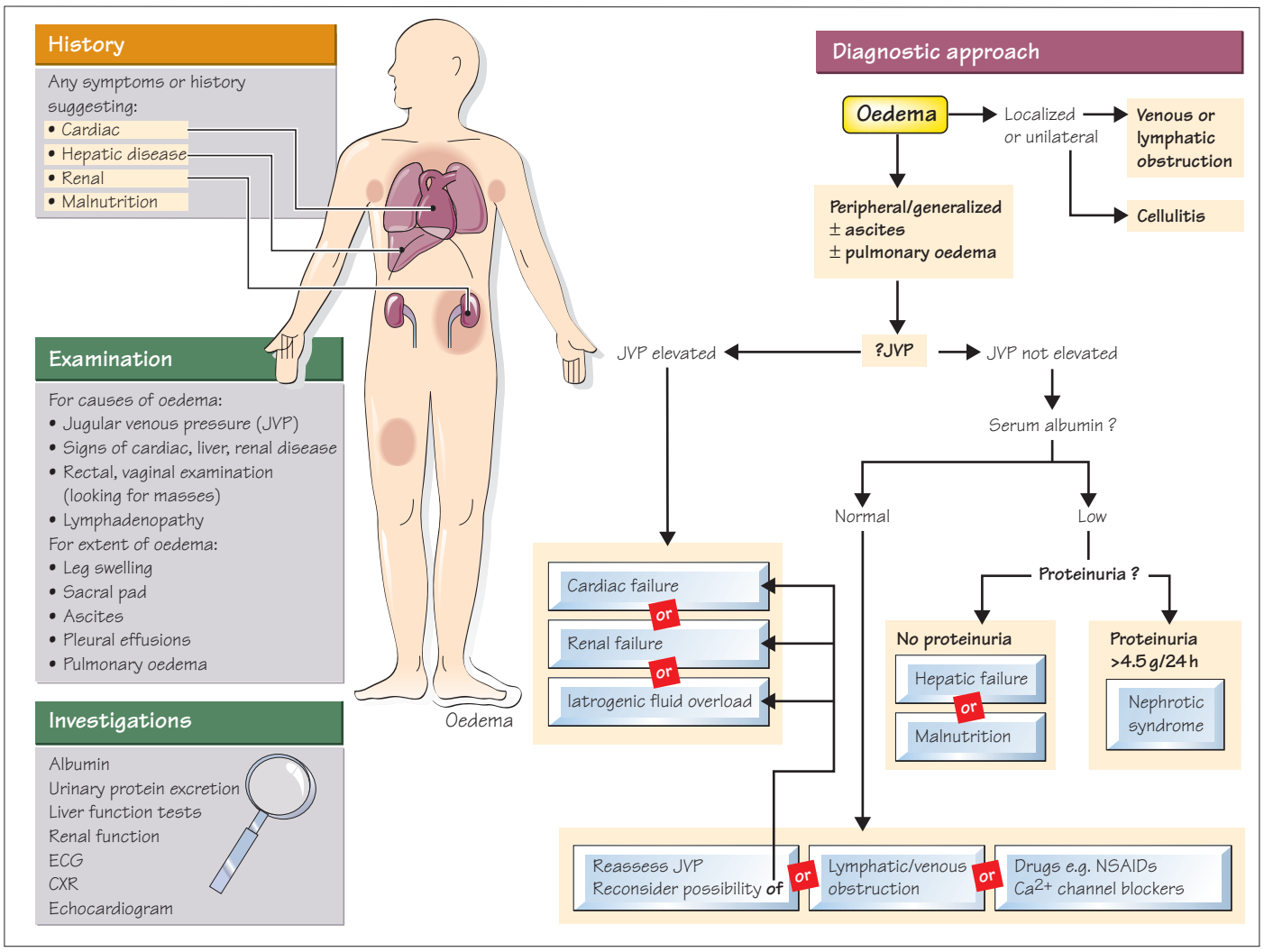
5 Oedema



Oedema ('an abnormal build-up of fluid in the tissues') can be a presenting feature of many serious medical conditions, notably congestive heart failure, liver failure, malnutrition and the nephrotic syndrome. Peripheral oedema can also result from venous or lymphatic obstruction or from excessive administration of salt and water. Agents such as non-steroidal anti-inflammatory drugs (NSAIDs) and calcium channel blockers can also produce peripheral oedema.

Presentation

Patients present complaining of swelling of the legs. In severe cases oedema extends to cause abdominal swelling (from ascites), sacral oedema, pleural effusions, pulmonary oedema and even facial swelling. Oedema is often, although not always, posturally dependent, and in bed-bound individuals it may be confined to the sacrum.

Diagnosis

Accurate history taking is vital. Symptoms and signs of cardiac, liver and renal disease should be sought. Two questions are the key to the diagnosis: Is the oedema unilateral or bilateral? Is the venous pressure raised or not? It is also important to determine whether oedema is present in other sites. Oedema diffusely affecting the whole body suggests a low serum albumin, or 'leaky' capillaries, rather than heart failure.

Bilateral leg oedema

In bilateral leg oedema, the diagnosis rests in determining whether the venous pressure is elevated and whether there are signs of liver disease, severe immobility or malnourishment.

- **Heart failure**: leg oedema occurs from right-sided heart failure and is always associated with a high jugular venous pressure (JVP) (Table 5.1). Hepatomegaly is often seen, as are signs of underlying cardiac pathology. If the oedema is mild in the legs, but severe in the abdomen, pericardial constriction should be considered.
- **Liver failure**: leg oedema is caused by a low serum albumin (usually < 20 g/dL). There may be signs of chronic liver disease, such as spider naevi, leuconychia, gynaecomastia, dilated abdominal veins indicating portal hypertension, and bruises (impaired liver synthetic function). The JVP is not elevated. In severe chronic liver disease (e.g. cirrhosis), liver enzyme tests may be only mildly disturbed, although the pro-

Table 5.1 Causes of oedema.

Bilateral oedema
Congestive cardiac failure
Hepatic failure
Renal failure
Nephrotic syndrome
Malnutrition
Immobility
Drugs (NSAIDs, calcium channel blockers)
Unilateral oedema
Lymphatic obstruction
Venous obstruction (usually DVT; rarely, external compression)
Venous valve incompetence from previous DVT
Cellulitis
Ruptured Baker's cyst
Localized immobility, e.g. hemiparesis

thrombin time (PT) is often prolonged (> 20 s). In acute liver failure, the patient is usually very unwell, cerebral disturbance is prominent and liver function tests are usually grossly abnormal.

• **Renal failure**: oedema is caused by either a low serum albumin (nephrotic syndrome, where the urine is frothy and contains 3–4+ of protein on dipstick testing—confirmatory tests include estimation of serum albumin (usually < 30 g/dL), urinary protein (usually > 4 g/24 h) and serum creatinine and urea) or an inability to excrete fluid (nephritic syndrome, associated with hypertension and low urine output).

• **General immobility**: the patient is usually elderly, and obviously immobile from general infirmity or cerebrovascular disease. The JVP is down, and there are no signs of liver or renal disease.

• **Malnutrition**: any chronic illness may be associated with a catabolic state and a degree of malnutrition that can be severe enough to depress serum albumin and cause leg oedema.

• **IVC compression**: rarely, bilateral leg oedema can be caused by compression on the inferior vena cava (IVC). This can be diagnosed by ultrasonographic studies of the abdomen, using colour flow Doppler to determine blood flow and computed tomography (CT), and occurs:
  • in extreme obesity
  • in severe (tense) ascites from whatever cause
  • with extensive venous thrombosis in the IVC, such as occurs in malignancy, or as a complication of the nephrotic syndrome.

Unilateral leg oedema

One-sided leg swelling is likely to have a local underlying cause, such as:

• A **deep venous thrombosis** (DVT) in the leg causes a slow onset (more than a few hours) of unilateral leg pain, swelling, with skin warmth, and possibly tenderness in the calves and along the veins, particularly the great saphenous vein. As symptoms/signs are unreliable for diagnosis, all patients with suspected DVTs should undergo definitive investigations (vein ultrasonography or venography) and be examined for complicating pulmonary embolisms (PEs) (see Chapter 7).

• **Ruptured Baker's cyst**: a Baker's cyst is a knee joint bursa that juts into the popliteal fossa and usually occurs in rheumatoid arthritis. It may rupture and cause sudden-onset leg pain and calf swelling. Ultrasonography is diagnostic.

• **Cellulitis**: consists of an intense spreading skin erythema, sometimes well demarcated, occasionally tracking up the line of the lymphatics. It is often very painful and is associated with a temperature, and raised erythrocyte sedimentation rate (ESR), C-reactive protein and white count. The organism is usually one of the staphylococci or streptococci species, and is occasionally grown from blood cultures, although rarely from skin swabs.

• **Lymphatic obstruction** results in a 'woody' form of unilateral oedema, sometimes described as 'non-pitting'. It is very rare in the West, and when found is usually the result of carcinomatous invasion and obliteration of the draining lymph nodes, e.g. in metastatic melanoma. In Africa lymph obstruction is very common, often bilateral, and caused by filarial infestation.

• **Pelvic tumours** can unilaterally compress veins, causing unilateral oedema.

• **Localized immobility** can cause unilateral leg oedema, e.g. long-standing hemiparesis.

Investigations

These vary depending on the features established by history and examination but determination of serum albumin, urinary protein loss, liver function tests, creatinine, ECG, chest X-ray and echocardiography are often appropriate.

Treatment

Therapy is directed at correcting the underlying cause. In bilateral oedema diuretics are often used to promote salt and water excretion, although their use should be balanced against the risk of hypovolaemia and worsening renal function, postural hypotension and falls. Several different classes of diuretic agent are used (see Table 5.2). The use of a loop diuretic in combination with a thiazide can produce a pronounced diuretic effect that is useful in resistant oedema. Spironolactone, a competitive aldosterone antagonist, produces a mild natriuresis and potassium retention, and is utilized in conditions with secondary hyperaldosteronism such as liver cirrhosis with ascites. Spironolactone and amiloride are 'potassium-sparing' diuretics, in contrast to the loop and thiazide diuretics which promote potassium depletion.

Table 5.2 Diuretics used in treating oedema.

Class	Example	Diuretic potency	Na^+/K^+ lowering potential
Thiazide	Bendroflumethiazide	+	++/+
Loop	Frusemide	+++	+/+++
K^+ sparing	Amiloride	±	±/0
	Spironolactone	±	±/0