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Clinical manifestations and diagnosis of edema in adults

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INTRODUCTION — Edema is defined as a palpable swelling produced by expansion of the interstitial fluid volume; when massive and generalized, the excess fluid accumulation is called anasarca. A variety of clinical conditions are associated with the development of edema, including heart failure, cirrhosis, and the nephrotic syndrome, as well as local conditions such as venous and lymphatic disease (table 1). (See "Pathophysiology and etiology of edema in adults".)

The clinical features and diagnosis of generalized edematous states in adults are reviewed here. The general principles of the treatment of edema in adults, including the use of diuretics to remove the excess fluid, the treatment of refractory edema, and the approach to edema in children, are discussed separately. (See "General principles of the treatment of edema in adults" and "Treatment of refractory edema in adults" and "Evaluation and management of edema in children".)

OVERVIEW OF PATHOPHYSIOLOGY — The pathophysiology and etiology of edema formation are discussed in detail elsewhere, but the pathophysiology is briefly reviewed here. (See "Pathophysiology and etiology of edema in adults".)

An increase in interstitial fluid volume that could lead to edema does not occur in normal subjects because of the tight balance of hemodynamic forces along the capillary wall and the function of the lymphatic vessels. For generalized edema to occur, two factors must be present:

- An alteration in capillary hemodynamics that favors the movement of fluid from the vascular space into the interstitium. Such movement requires a change in one or more components of Starling's law: increased capillary hydrostatic pressure, decreased capillary oncotic pressure (ie, hypoalbuminemia), and/or increased capillary permeability. (See "Pathophysiology and etiology of edema in adults", section on 'Capillary hemodynamics'.)
- The retention of dietary or intravenously administered sodium and water by the kidneys. Edema (other than localized edema as with an allergic reaction) does not become clinically apparent until the interstitial volume has increased by 2.5 to 3 L, an amount that is almost equal to the plasma volume. Thus, patients would develop marked hemoconcentration and shock if the plasma volume were not maintained by renal retention of sodium and water.

The retention of sodium and water can either be a primary event, as in renal failure, or a secondary event resulting from a primary reduction in either cardiac output (as in heart failure) or systemic vascular resistance (as in cirrhosis). In the latter settings, fluid retention tends to return the effective circulating volume toward normal. As will be described below, diuretic therapy in such patients may have a deleterious effect on systemic hemodynamics even though it reduces the edema. Thus, careful monitoring is required. (See "General principles of the treatment of edema in adults", section on 'What are the consequences of the removal of edema fluid?'.)

CLINICAL MANIFESTATIONS AND DIAGNOSIS — The many diseases that can produce heart failure, cirrhosis, the nephrotic syndrome, or renal failure and the methods used in their diagnosis are discussed elsewhere. (See appropriate topic reviews.)

The history is clearly important, and several general questions should be addressed:

- Is there a history of any disorder (eg, coronary disease, hypertension, or alcohol abuse) or drug that can cause cardiac, hepatic, or renal disease?
- Where is the edema located? Patients with a primary complaint of shortness of breath may have left heart failure and pulmonary edema; those with ascites may have cirrhosis; and those with only peripheral edema may have right-sided heart failure, pericardial disease, renal disease, or local venous or lymphatic disease. Among patients with heart failure, the localization of edema may provide diagnostic information. Patients with left ventricular dysfunction (eg, due to ischemia, hypertension, or valvular disease) typically present with pulmonary congestion; those with cardiomyopathy may have equivalent involvement of both the right and left ventricles, often leading to the simultaneous onset of pulmonary and peripheral edema; disease mainly affecting the right ventricle or venous return to the heart causes mainly peripheral edema.
- Is the edema intermittent or persistent? Intermittent edema is a common premenstrual symptom. Some women are given chronic diuretic therapy for this disorder; such treatment is not necessary and can produce a form of diuretic dependence that accounts for some cases of idiopathic edema. (See "Idiopathic edema".)

The physical examination can help establish the diagnosis. Of particular importance are: (1) the pattern of distribution of edema, which reflects those capillaries with altered hemodynamic forces; (2) the central venous pressure; and (3) the presence or absence of pulmonary edema (table 2).

Pulmonary edema — Patients with pulmonary edema complain primarily of shortness of breath and orthopnea. Chest pain is typically a prominent symptom when pulmonary edema is due to an acute myocardial infarction. (See "Evaluation of acute decompensated heart failure".)

Physical examination usually reveals a tachypneic, diaphoretic patient with wet rales and possibly a diastolic gallop (S3) and heart murmurs. The diagnosis of pulmonary edema should be confirmed by radiologic studies because other disorders, such as a pulmonary embolus, may produce similar symptoms but will require different therapy (image 1A-B). (See "Auscultation of cardiac murmurs" and "Auscultation of heart sounds".)

Although cardiac disease is the most common cause of pulmonary edema, it can also be produced by volume overload due to primary renal sodium retention (such as acute glomerulonephritis) or by increased capillary permeability in the acute respiratory distress syndrome.

If the correct diagnosis cannot be established from the history, physical examination, and laboratory data, measurement of the pulmonary capillary wedge pressure can be helpful. The wedge pressure exceeds 18 to 20 mmHg when pulmonary edema is due to heart disease or primary renal sodium retention [1], but it is relatively normal (unless there is concurrent heart disease or fluid overload) in the acute respiratory distress syndrome [2]. (See "Evaluation of acute decompensated heart failure" and "Noncardiogenic pulmonary edema", section on 'Permeability pulmonary edema due to ARDS'.)

In contrast to cardiac and renal disease, uncomplicated cirrhosis is **not** associated with pulmonary edema. The intrahepatic sinusoidal obstruction in this disorder leads to selective increases in venous and capillary pressures **proximal to** the hepatic vein [3]. The systemic vascular resistance decreases in cirrhotic patients, and this generates relatively reduced blood volume in the cardiopulmonary circulation [4]. (See "Hyponatremia in patients with cirrhosis".)

Pulmonary edema also does not occur due to isolated hypoalbuminemia [5]. Thus, in the absence of a concurrent rise in left atrial and pulmonary capillary pressures, pulmonary edema is not usually seen with hypoalbuminemia, even at a plasma albumin concentration low enough to induce peripheral edema [6]. The reasons for this are presented elsewhere. (See "Pathophysiology and etiology of edema in adults", section on 'Compensatory factors'.)

Peripheral edema — In contrast to the potentially life-threatening nature of pulmonary edema, peripheral edema is cosmetically undesirable but produces less serious symptoms. These include swollen legs, difficulty in walking

and, with chronic tense edema, stasis dermatitis. Patients with the nephrotic syndrome may also have prominent periorbital edema due to the low tissue pressure in this area. (See "Stasis dermatitis".)

Pitting edema — Peripheral edema is usually detected by the presence of pitting after pressure is applied to the edematous area for at least five seconds [7]. Pitting reflects movement of the excess interstitial water in response to pressure.

Since peripheral edema locates preferentially in the dependent areas, it is primarily found in the lower extremities in ambulatory patients and over the sacrum in patients at bed rest. Nonpitting edema suggests lymphatic obstruction or hypothyroidism. (See 'Nonpitting edema' below.)

Although clinicians commonly grade pitting edema from 1+ to 4+ (mild to severe), there is no agreed upon definition of these grades. However, this type of grading scheme may help the clinician record relative changes in edema (eg, less edema after diuretic therapy). Since the degree of edema is also influenced by posture, documenting weight loss is another component of monitoring the efficacy of diuretic therapy.

Ascites — Ascites is associated with abdominal distention and both shifting dullness and a fluid wave on percussion of the abdomen. Patients with tense ascites may complain of shortness of breath due to pressure on the diaphragm. (See "Evaluation of adults with ascites".)

Distribution of edema and central venous pressure — The distribution of edema and estimation of the central (jugular) venous pressure can aid in the differential diagnosis of heart failure, cirrhosis, primary renal sodium retention, and the nephrotic syndrome (table 2). This is particularly important in some patients with chronic right-sided heart failure in whom the cardiac disease can lead to both cirrhosis (due to chronic passive congestion of the liver) and hemodynamically mediated proteinuria, which on rare occasions approaches the nephrotic range [8].

In view of the importance of accurate venous pressure measurements in patients with edema, it is essential to use proper technique to estimate the central venous pressure. (See "Examination of the jugular venous pulse", section on 'Jugular venous (right atrial) pressure'.)

Heart failure and pericardial disease — Patients with right-sided heart failure have peripheral edema and, in severe cases, anasarca with ascites and edema of the abdominal wall. Shortness of breath is commonly present and may be due to underlying coexistent left ventricular failure or pulmonary disease.

The peripheral edema in patients with heart failure is due to an increase in venous pressure behind the right side of the heart. Thus, the pressures in the right atrium and subclavian vein are elevated, changes that can be detected by estimation of the jugular venous pressure or by direct measurement with a central venous pressure catheter.

Cirrhosis — Cirrhotic patients can develop ascites and then edema in the lower extremities because of an increase in venous pressure below the diseased liver. As a result, the venous pressure above the hepatic vein, including the jugular veins and right atrium, is usually reduced or normal [3,4], not elevated as in heart failure. One exception to this general rule can occur in patients with tense ascites in whom upward pressure on the diaphragm can increase the intrathoracic pressure. Although elevated initially in this setting, the central venous pressure rapidly falls to normal following the removal of a small amount of ascitic fluid, which substantially reduces the intraperitoneal pressure [3]. (See "Evaluation of adults with ascites".)

The presence of other signs of portal hypertension, such as distended abdominal wall veins and splenomegaly, also is suggestive of primary hepatic disease. However, these findings are not necessarily specific since chronic right-sided heart failure can produce hepatic injury. The potential difficulties in distinguishing between primary hepatic and cardiac disease can be illustrated by the following example.

- Case history – A 56-year-old man has a three-year history of ascites, which requires removal of 3 to 4 liters of ascitic fluid by paracentesis every three weeks. The patient has been told he has cirrhosis, although a liver biopsy has not been performed. He has no history of hepatic disease and is only a social drinker. The physical examination reveals no acute distress, a soft abdomen with marked ascites, and moderate pedal

edema. The heartbeat is irregularly irregular. The heart sounds are distant, and no murmurs are heard. Several spider angiomata are present. The estimated jugular venous pressure is greater than 15 cmH2O.

The electrocardiogram shows atrial fibrillation and low voltage. There is 3+ proteinuria by dipstick; the plasma albumin concentration is 2.9 g/dL; and liver function tests are mildly abnormal.

- Comment – Despite the features suggestive of cirrhosis, the elevated jugular venous pressure pointed toward right-sided heart failure. As a result, the central venous pressure was measured directly and found to be 21 cmH2O (normal 1 to 8 cmH2O). Further evaluation established the diagnosis of constrictive pericarditis. After pericardiectomy, the patient had a complete recovery, including reversal of the liver function abnormalities and the proteinuria. (See "Constrictive pericarditis".)

Renal sodium retention — The physical findings associated with primary renal sodium retention are similar to those seen with biventricular failure: both pulmonary and peripheral edema may be present, and the jugular venous pressure should be elevated since these patients are truly volume expanded. An abnormal urinalysis (particularly if there are signs of active renal disease such as red cell casts) and elevations in the BUN and plasma creatinine concentration will help to distinguish underlying renal disease from heart failure. However, this differentiation can be difficult since cardiac disease can reduce glomerular filtration rate (GFR) (due to diminished renal perfusion) and cause proteinuria [8]. In this setting, the correct diagnosis may be best established by the presence of normal cardiac function when the excess fluid is removed.

Nephrotic syndrome — Patients with the nephrotic syndrome typically present with periorbital and peripheral edema and occasionally ascites. As described above, two factors contribute to the fluid retention in this condition: primary sodium retention due to the underlying renal disease; and, often less important, a diminished transcapillary oncotic pressure gradient, mostly with very severe hypoalbuminemia [9]. The central venous pressure is usually normal to high-normal in the nephrotic syndrome. (See "Pathophysiology and treatment of edema in patients with the nephrotic syndrome".)

The diagnosis of the nephrotic syndrome can be confirmed by documenting the presence of both heavy proteinuria (typically greater than 3.5 g/day, principally albuminuria) and hypoalbuminemia. Lipiduria and hyperlipidemia are also commonly seen; the former reflecting the abnormal glomerular filtration of lipoprotein molecules (mainly HDL) and the latter reflecting both increased hepatic lipoprotein synthesis (induced by the fall in plasma oncotic pressure) and decreased clearance of triglycerides. (See "Lipid abnormalities in nephrotic syndrome".)

Idiopathic edema — Patients with idiopathic edema behave as if they are volume depleted. They have an exaggerated fall in the plasma volume when they stand, which is exaggerated by diuretics. As a result, they have peripheral edema, but the central venous pressure is normal or low-normal, and pulmonary edema does not occur.

The diagnosis of idiopathic edema is one of **exclusion**. It should be considered in menstruating women who have a normal plasma albumin concentration, normal jugular venous pressure, and no evidence of cardiac, hepatic, or renal disease. (See "Idiopathic edema".)

Drug-induced edema — There are a variety of causes of drug-induced edema, which are discussed elsewhere (table 1). The diagnosis is suspected when one of these drugs is being used and there is no other apparent cause for the edema. (See "Pathophysiology and etiology of edema in adults".)

Among the most predictable causes of drug-induced edema are:

- Direct vasodilators, such as minoxidil, diazoxide, and nifedipine, which reduce the blood pressure. This effect probably plays an important role both directly and by activating the renin-angiotensin-aldosterone and sympathetic nervous systems, both of which stimulate renal sodium retention.
- The thiazolidinediones such as pioglitazone or rosiglitazone stimulate sodium reabsorption by the sodium channels in the luminal membrane of cortical collecting tubule cells. This is the same site stimulated by aldosterone. Edema due to these medications is particularly likely to occur in patients who also have

underlying heart disease. (See "Thiazolidinediones in the treatment of diabetes mellitus", section on 'Fluid retention/heart failure'.)

- Nonsteroidal anti-inflammatory drugs can exacerbate edema in patients with underlying heart failure or cirrhosis. This effect is largely due to increased renal sodium reabsorption in response to the inhibition of renal vasodilatory prostaglandins. (See "NSAIDs: Electrolyte complications".)

Unilateral edema — The generalized edematous states described above are associated with symmetric bilateral edema, and unilateral edema is much less common with these disorders. Unilateral or single limb edema may develop as a result of venous insufficiency or thrombosis, and, when impaired, lymphatic drainage causes lymphedema.

Venous insufficiency or thrombosis — A common cause of peripheral edema is venous insufficiency, which can produce both unilateral and bilateral edema. One cause of venous insufficiency is a post-thrombotic syndrome after an episode of thrombophlebitis. (See "Clinical manifestations of lower extremity chronic venous disease" and "Post-thrombotic (postphlebitic) syndrome".)

In addition to the history of venous disease and the absence of other apparent causes of edema, the following clues point to an etiologic role for venous insufficiency due to thrombosis:

- The edema is generally limited to an extremity, most commonly one of the legs. In contrast, **unilateral edema is unusual** in patients with generalized edematous states. Deep vein thrombosis should always be considered in patients with the acute onset unilateral leg edema. (See "Approach to the diagnosis and therapy of lower extremity deep vein thrombosis", section on 'Differential diagnosis'.)
- The central venous pressure is normal, a finding that excludes heart failure or pericardial disease, and renal function is normal. There is a poor response to diuretics, with some patients developing signs of hypoperfusion.

Localized upper extremity edema may occur with upper extremity venous thrombosis. This may be catheter induced or spontaneous. These patients often present with a feeling of fullness in the fingers or difficulty with finger rings feeling "too tight" (see "Catheter-related upper extremity venous thrombosis" and "Primary (spontaneous) upper extremity deep vein thrombosis").

Lymphedema — The major causes of lymphedema in adults in developed countries are axillary lymph node dissection in patients with breast cancer and axillary or inguinal lymph node dissection in patients with melanoma. However, worldwide, the most common cause is filariasis. The edema in such patients is typically limited to an arm or leg. The clinical hallmark of lymphedema is the presence of cutaneous and subcutaneous thickening, as manifested by cutaneous fibrosis, peau d'orange, and a positive Stemmer sign, which refers to an inability to tent the skin at the base of the digits. (See "Clinical manifestations and diagnosis of lymphedema" and "Epidemiology, pathogenesis, and clinical manifestations of lymphatic filariasis", section on 'Lymphedema'.)

Periorbital and scrotal edema — Periorbital and scrotal edema are localized forms of edema that can be seen in systemic edematous states but should **not** be the sole manifestation of edema in these disorders. The differential diagnosis of localized periorbital/facial edema and acute idiopathic scrotal edema are discussed separately. (See "An overview of angioedema: Clinical features, diagnosis, and management", section on 'Disorders resembling cutaneous edema' and "Evaluation of the acute scrotum in adults", section on 'Acute idiopathic scrotal edema'.)

Nonpitting edema — Pitting reflects movement of the excess interstitial water in response to pressure. Testing for pitting involves applying firm pressure to the edematous tissue for at least five seconds [7]. (See 'Peripheral edema' above.)

Nonpitting edema is primarily due to one of two disorders:

- Moderate to severe lymphedema, as can occur after radical mastectomy or with lymphatic disease. However, pitting does occur in mild (stage I) and some cases of moderate (stage II) lymphedema [10]. (See "Clinical

manifestations and diagnosis of lymphedema".)

- Pretibial myxedema, which occurs in patients with thyroid disease and is associated with localized areas of swelling. (See "Pretibial myxedema (thyroid dermopathy) in autoimmune thyroid disease".)

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Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient information: Swelling (The Basics)")
- Beyond the Basics topics (see "Patient information: Edema (swelling) (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- Edema is a palpable swelling produced by expansion of the interstitial fluid volume. Generalized edema is produced by one or more of the following: increased capillary hydrostatic pressure, decreased capillary oncotic pressure, and/or increased capillary permeability. The retention of sodium and water by the kidneys must also occur for edema to develop. (See 'Overview of pathophysiology' above.)
- The retention of sodium and water by the kidneys can be the primary process, as in edema due to renal failure, or it may be a compensatory response to reduced effective arterial blood volume in edema due to other causes (such as heart failure and cirrhosis).
- The history should focus on disorders or drugs that can cause cardiac, hepatic, or renal disease or drugs directly associated with edema formation. (See 'Clinical manifestations and diagnosis' above.)
- The physical examination should be aimed at establishing the pattern of edema (pulmonary, peripheral, ascites, localized) and an assessment of the central venous pressure. The results can indicate the likely cause of edema (table 2). (See 'Distribution of edema and central venous pressure' above.)
- Acute onset unexplained unilateral leg edema should raise the possibility of deep vein thrombosis. Nonpitting edema should raise the possibility of lymphedema or pretibial myxedema. (See 'Venous insufficiency or thrombosis' above and 'Nonpitting edema' above.)

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Topic 6878 Version 14.0

GRAPHICS

Major causes of edema by primary mechanism

Increased capillary hydraulic pressure
Increased plasma volume due to renal sodium retention
Heart failure, including cor pulmonale
Primary renal sodium retention
• Renal disease, including the nephrotic syndrome
• Drugs*: Nonsteroidal anti-inflammatory drugs (NSAIDs), glucocorticoids, fludrocortisone, thiazolidinediones (glitazones), insulins, estrogens, progestins, androgens, testosterone, aromatase inhibitors, tamoxifen; and by multiple mechanisms: vasodilators (hydralazine, minoxidil, diazoxide) and calcium channel blockers (particularly dihydropyridines, ie, amlodipine, nifedipine); also see "Arteriolar vasodilation" below
• Refeeding edema
• Early hepatic cirrhosis
Pregnancy and premenstrual edema
Idiopathic edema, when diuretic induced
Sodium or fluid overload: Parenteral antibiotics or other drugs with large amounts of sodium, sodium bicarbonate, or excessive or overly rapid fluid replacement
Venous obstruction or insufficiency
Cirrhosis or hepatic venous obstruction
Acute pulmonary edema
Local venous obstruction
• Venous thrombosis
• Venous stenosis
Chronic venous insufficiency - post-thrombotic syndrome
Arteriolar vasodilation
Drugs*: Frequent - vasodilators (hydralazine, minoxidil, diazoxide), dihydropyridine calcium channel blockers; less frequent - alpha1 blockers, sympatholytics (ie, methyldopa), nondihydropyridine calcium channel blockers[1]
Idiopathic edema (?)
Hypoalbuminemia
Protein loss
Nephrotic syndrome
Protein-losing enteropathy
Reduced albumin synthesis
Liver disease
Malnutrition

Increased capillary permeability
Idiopathic edema (?)
Burns
Trauma
Inflammation or sepsis
Allergic reactions, including certain forms of angioedema
Adult respiratory distress syndrome
Diabetes mellitus
Interleukin-2 therapy
Malignant ascites
Lymphatic obstruction or increased interstitial oncotic pressure
Lymph node dissection
Nodal enlargement due to malignancy
Hypothyroidism
Malignant ascites
Other drugs* (uncertain mechanism)
Anticonvulsant: Gabapentin, pregabalin
Antineoplastic: Docetaxel, cisplatin
Antiparkinson: Pramipexole, ropinirole

* Patients with decreased cardiac output, preexisting renal insufficiency, and/or receiving higher doses are more likely to experience edema and edema-associated adverse events. This is not a complete list of drugs associated with edema. For additional information, refer to the Lexicomp individual drug monographs included with UpToDate.

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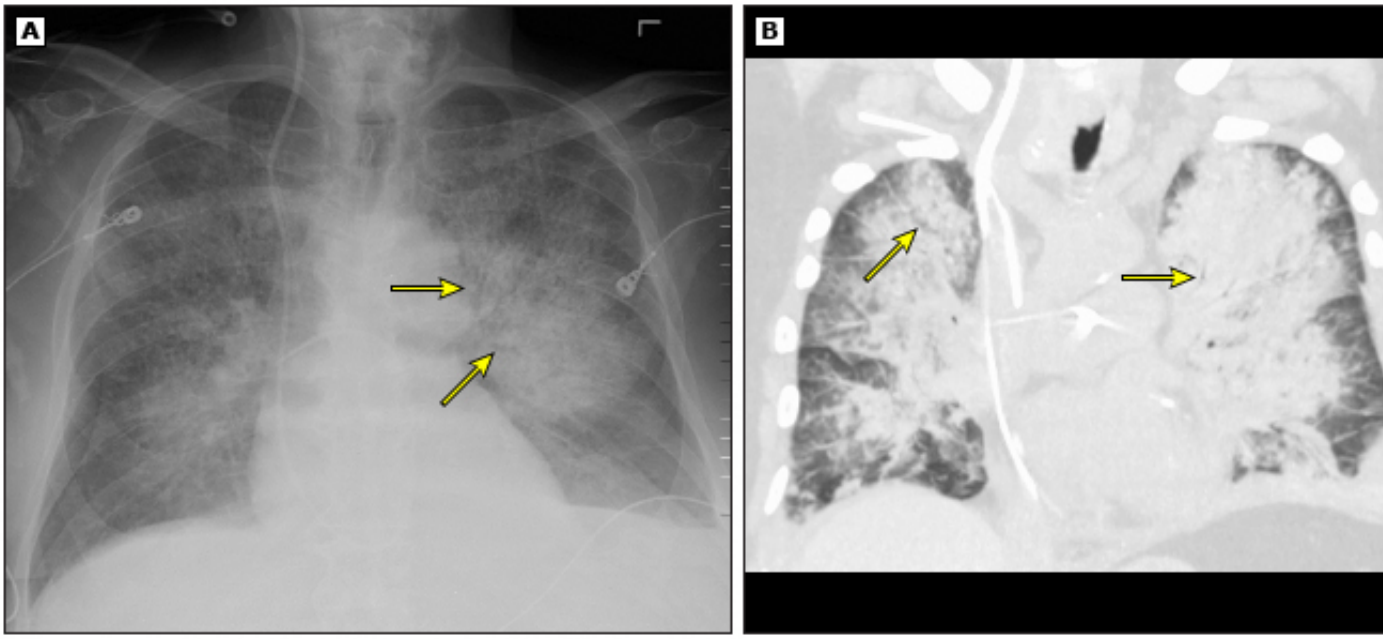
Graphic 53550 Version 8.0

Major physical findings in edematous states

Disorder	Pulmonary edema	Central venous pressure	Ascites and/or pedal edema
Left-sided heart failure	+	Variable	-
Right-sided heart failure	-	↑	+
Cirrhosis	-	↓-Normal	+
Renal disease	Variable	↑	+
Nephrotic syndrome	-	Variable	+
Idiopathic edema	-	↓-Normal	+
Venous insufficiency	-	Normal	+, edema may be asymmetric

Graphic 72002 Version 1.0

Radiograph and CT of acute pulmonary edema

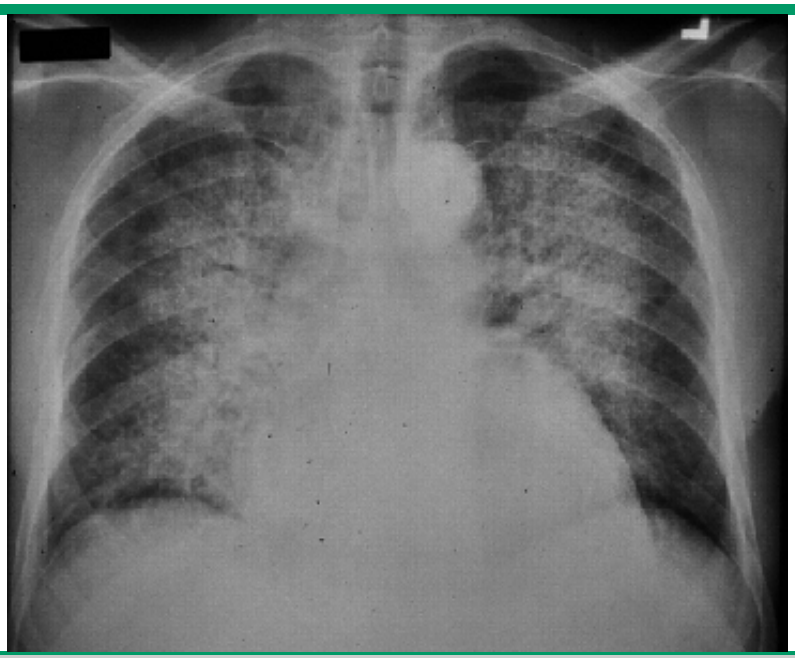


(A) An anteroposterior radiograph shows perihilar consolidations and air bronchograms (arrows) of acute edema.
(B) A coronal reconstruction of a CT scan of the same patient shows acute alveolar edema with diffuse pe infiltrates and air bronchograms (arrows).

CT: computed tomography

Graphic 98495 Version 2.0

Hydrostatic pulmonary edema

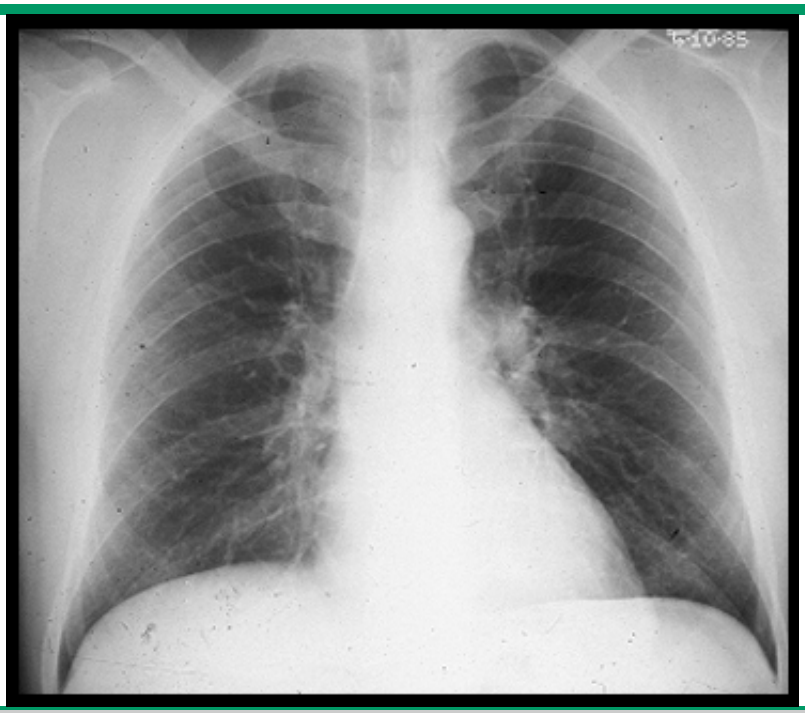


Pulmonary edema in a "butterfly distribution" due to left ventricular failure. Chest radiograph shows large perihilar opacities in patient with enlarged cardiac silhouette.

Courtesy of Paul Stark, MD.

Graphic 58394 Version 3.0

Normal chest radiograph



Posteroanterior view of a normal chest radiograph.

Courtesy of Carol M Black, MD.

Graphic 65576 Version 1.0

Disclosures

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