
بسم الله الرحمن الرحيم

Edema

Present by:

dr.Mohammad Taghi Ashoubi

Assistant Professor of Vascular & Trauma Surgery

Department of Surgery, School of Medicine

Razi Hospital

Guilan University of Medical Sciences

Edema

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- Edema is swelling caused by excess fluid trapped in your body's tissues. Although edema can affect any part of your body, you may notice it more in your hands, arms, feet, ankles and legs.
- Edema can be the result of medication, pregnancy or an underlying disease — often congestive heart failure, kidney disease or cirrhosis of the liver.
- Taking medication to remove excess fluid and reducing the amount of salt in your food often relieves edema. When edema is a sign of an underlying disease, the disease itself requires separate treatment.

- A weight gain of several kilograms usually precedes overt manifestations of generalized edema. Anasarca refers to gross, generalized edema. Ascites and hydrothorax refer to accumulation of excess fluid in the peritoneal and pleural cavities, respectively, and are considered special forms of edema.

- Edema is recognized by the persistence of an indentation of the skin aer pressure known as “pitting” edema. In its more subtle form, edema may be detected by noting that aer the stethoscope is removed from the chest wall, the rim of the bell leaves an indentation on the skin of the chest for a few minutes. Edema may be present when the ring on a finger fits more snugly than in the past or when a patient complains of diiculty putting on shoes, particularly in the evening. Edema may also be recognized by puiness of the face, which is most readily apparent in the periorbital areas.

- GENERALIZED EDEMA
- The dierences among the major causes of generalized edema are shown. Cardiac, renal, hepatic, or nutritional disorders are responsible for a large majority of patients with generalized edema. Consequently, the dierential diagnosis of generalized edema should be directed toward identifying or excluding these several conditions.

Principal Causes of Generalized Edema: History, Physical Examination, and Laboratory Findings

Organ System	History	Physical Examination	Laboratory Findings
Cardiac	Dyspnea with exertion prominent—often associated with orthopnea—or paroxysmal nocturnal dyspnea	Elevated jugular venous pressure, ventricular (S_3) gallop; occasionally with displaced or dyskinetic apical pulse; peripheral cyanosis, cool extremities, small pulse pressure when severe	Elevated urea nitrogen-to-creatinine ratio common; serum sodium often diminished; elevated natriuretic peptides
Hepatic	Dyspnea uncommon, except if associated with significant degree of ascites; most often a history of ethanol abuse	Frequently associated with ascites; jugular venous pressure normal or low; blood pressure lower than in renal or cardiac disease; one or more additional signs of chronic liver disease (jaundice, palmar erythema, Dupuytren's contracture, spider angiomata, male gynecomastia; asterixis and other signs of encephalopathy) may be present	If severe, reductions in serum albumin, cholesterol, other hepatic proteins (transferrin, fibrinogen); liver enzymes elevated, depending on the cause and acuity of liver injury; tendency toward hypokalemia, respiratory alkalosis; macrocytosis from folate deficiency

Organ System	History	Physical Examination	Laboratory Findings
Renal (NS)	Childhood diabetes mellitus; plasma cell dyscrasias	Periorbital edema; hypertension	Proteinuria (≥3.5 g/d); hypoalbuminemia; hypercholesterolemia; microscopic hematuria
(CRF)	associated with uremic signs and symptoms, including decreased appetite, altered (metallic or fishy) taste, altered sleep pattern, difficulty concentrating, restless legs, or myoclonus; dyspnea can be present, but generally less prominent than in heart failure	hypertensive retinopathy; nitrogenous fetor; pericardial friction rub in advanced cases with uremia	creatinine and cystatin C; albuminuria; hyperkalemia, metabolic acidosis, hyperphosphatemia, hypocalcemia, anemia (usually normocytic)

Heart Failure

- In heart failure, the impaired systolic emptying of the ventricle(s) and/or the impairment of ventricular relaxation promotes an accumulation of blood in the venous circulation at the expense of the eective arterial volume. In addition, the activation of the sympathetic nervous system and the RAAS (see above) acts in concert to cause renal vasoconstriction and reduction of glomerular filtration and salt and water retention. Sodium and water retention continue, and the increment in blood volume accumulates in the venous circulation, raising venous and intracapillary pressure resulting in edema

- The presence of overt cardiac disease, as manifested by cardiac enlargement and/or ventricular hypertrophy, together with clinical evidence of cardiac failure, such as dyspnea, basilar rales, venous distention, and hepatomegaly, usually indicates that edema results from heart failure. Noninvasive tests such as electrocardiography, echocardiography, and measurements of BNP (or NTproBNP) are helpful in establishing the diagnosis of heart disease. The edema of heart failure typically occurs in the dependent portions of the body.

Edema of Renal Disease

- The edema that occurs during the acute phase of glomerulonephritis is characteristically associated with hematuria, proteinuria, and hypertension. In most instances, the edema results from primary retention of sodium and water by the kidneys owing to renal dysfunction. This state diers from most forms of heart failure in that it is characterized by a normal (or sometimes even increased) cardiac output. Patients with chronic renal failure may also develop edema due to primary renal retention of sodium and water.

The primary alteration in the nephrotic syndrome is a diminished colloid oncotic pressure due to losses of large quantities (≥3.5 g/d) of protein into the urine, and hypoalbuminemia (<3.0 g/dL). As a result of the reduced colloid osmotic pressure, the sodium and water that are retained cannot be confined within the vascular compartment, and total and eective arterial blood volumes decline. This process initiates the edema-forming sequence of events described above, including activation of the RAAS. The nephrotic syndrome may occur during the course of a variety of kidney diseases, including glomerulonephritis, diabetic glomerulosclerosis, and hypersensitivity reactions. The edema is diuse, symmetric, and most prominent in the dependent areas; periorbital edema is most prominent in the morning.

Hepatic Cirrhosis

- **This condition is characterized in part by hepatic venous outflow obstruction, which in turn expands the splanchnic blood volume, and hepatic lymph formation. Intrahepatic hypertension acts as a stimulus for renal sodium retention and causes a reduction of eective arterial blood volume. These alterations are frequently complicated by hypoalbuminemia secondary to reduced hepatic synthesis of albumin, as well as peripheral arterial vasodilation. These eects reduce the eective arterial blood volume, leading to activation of the sodium- and water-retaining mechanisms described above.The concentration of circulating aldosterone oen is elevated by the failure of the liver to metabolize this hormone. Initially, the excess interstitial fluid is localized preferentially proximal (upstream) to the congested portal venous system, causing ascites .In later stages, particularly when there is severe hypoalbuminemia, peripheral edema may develop. A sizable accumulation of ascitic fluid may increase intraabdominal pressure and impede venous return from the lower extremities and contribute to the accumulation of the edema.**

Drug-Induced Edema

- A large number of widely used drugs can cause edema .Mechanisms include renal vasoconstriction (NSAIDs and cyclosporine), arteriolar dilation (vasodilators), augmented renal sodium reabsorption (steroid hormones), and capillary damage.

Drugs Associated with Edema Formation

- Nonsteroidal anti-inflammatory drugs
- Antihypertensive agents
- Direct arterial/arteriolar vasodilators
- Hydralazine
- Clonidine
- Methyldopa
- Guanethidine
- Minoxidil
- Calcium channel antagonists

Drugs Associated with Edema Formation

- α-Adrenergic antagonists
- Thiazolidinediones
- Steroid hormones
- Glucocorticoids
- Anabolic steroids
- Estrogens
- Progestins
- Cyclosporine
- Growth hormone
- Immunotherapies
- Interleukin 2
- OKT3 monoclonal antibody

Edema of Nutritional Origin

- A diet grossly deficient in calories and particularly in protein over a prolonged period may produce hypoproteinemia and edema. The latter may be intensified by the development of beriberi heart disease, which also is of nutritional origin, in which multiple peripheral arteriovenous fistulae result in reduced eective systemic perfusion and eective arterial blood volume, thereby enhancing edema formation .Edema develops or becomes intensified when famished subjects are first provided with an adequate diet. The ingestion of more food may increase the quantity of sodium ingested, which is then retained along with water. So-called refeeding edema also may be linked to increased release of insulin, which directly increases tubular sodium reabsorption. In addition to hypoalbuminemia, hypokalemia and caloric deficits may be involved in the edema of starvation.

LOCALIZED EDEMA

- In thrombophlebitis, varicose veins, and in primary venous valve failure, the hydrostatic pressure in the capillary bed upstream (proximal) of the obstruction increases so that an abnormal quantity of fluid is transferred from the vascular to the interstitial space, which may give rise to localized edema. The latter may also occur in lymphatic obstruction caused by chronic lymphangitis, resection of regional lymph nodes, filariasis, and genetic (frequently called primary) lymphedema. The latter is particularly intractable because restriction of lymphatic flow results in both an increase in intracapillary pressure and increased protein concentration in the interstitial fluid, which act in concert to aggravate fluid retention.

Other Causes of Edema

- These causes include hypothyroidism (myxedema) due to deposition of hyaluronic acid, and hyperthyroidism (pretibial myxedema secondary to Graves' disease), in which edema is typically nonpitting and, in Graves' disease, exogenous hyperadrenocortism; pregnancy; and administration of estrogens and vasodilators, particularly dihydropyridines such as nifedipine.

DISTRIBUTION OF EDEMA

- The distribution of edema is an important guide to its cause. Edema associated with heart failure tends to be more extensive in the legs and to be accentuated in the evening, a feature also determined largely by posture. When patients with heart failure are confined to bed, edema may be most prominent in the presacral region.

- Edema resulting from hypoproteinemia, as occurs in the nephrotic syndrome, characteristically is generalized, but it is especially evident in the very so tissues of the eyelids and face and tends to be most pronounced in the morning owing to the recumbent posture assumed during the night. Less common causes of facial edema include trichinosis, allergic reactions, and myxedema. Edema limited to one leg or to one or both arms is usually the result of venous and/or lymphatic obstruction. Unilateral paralysis reduces lymphatic and venous drainage on the aected side and may also be responsible for unilateral edema. In patients with obstruction of the superior vena cava, edema is confined to the face, neck, and upper extremities in which the venous pressure is elevated compared with that in the lower extremities.

Edema

- An important first question is whether the edema is localized or generalized. If it is localized, the local phenomena that may be responsible should be identified. If the edema is generalized, one should determine if there is serious hypoalbuminemia, e.g., serum albumin <3.0 g/dl. If so, the history, physical examination, urinalysis, and other laboratory data will help evaluate the question of cirrhosis, severe malnutrition, or the nephrotic syndrome as the underlying disorder. If hypoalbuminemia is not present, it should be determined if there is evidence of heart failure severe enough to promote generalized edema. Finally, it should be ascertained as to whether or not the patient has an adequate urine output or if there is significant oliguria or anuria.

Symptoms

Edema in foot and ankle
Edema in foot and ankleOpen pop-up dialog box
Signs of edema include:

Swelling or puffiness of the tissue directly under your skin, especially in your legs or arms
Stretched or shiny skin
Skin that retains a dimple (pits), after being pressed for several seconds
Increased abdominal size

When to see a doctor

Make an appointment to see your doctor if you have swelling, stretched or shiny skin, or skin that retains a dimple after being pressed (pitting). See your doctor immediately if you experience:

- **Shortness of breath**
- **Difficulty breathing**
- **Chest pain**

These can be signs of pulmonary edema, which requires prompt treatment.

If you've been sitting for a prolonged period, such as on a long flight, and you develop leg pain and swelling that won't go away, call your doctor. Persistent leg pain and swelling can indicate a blood clot deep in your vein (deep vein thrombosis, or DVT).

- Edema occurs when tiny blood vessels in your body (capillaries) leak fluid. The fluid builds up in surrounding tissues, leading to swelling.
- Mild cases of edema may result from:
- Sitting or staying in one position for too long
- Eating too much salty food
- Having premenstrual signs and symptoms
- Being pregnant

Edema can also be a side effect of some medications, including:

- High blood pressure medications
- Nonsteroidal anti-inflammatory drugs
- Steroid drugs
- Estrogens
- Certain diabetes medications called thiazolidinediones

Several diseases and conditions may cause edema, including:

- **Congestive heart failure.**

If you have congestive heart failure, one or both of your heart's lower chambers lose their ability to pump blood effectively. As a result, blood can back up in your legs, ankles and feet, causing edema. Congestive heart failure can also cause swelling in your abdomen. Sometimes, this condition can cause fluid to accumulate in your lungs (pulmonary edema), which can lead to shortness of breath.

Cirrhosis.

Fluid may accumulate in your abdominal cavity (ascites) and in your legs as a result of liver damage (cirrhosis).

- **Cirrhosis.** Fluid may accumulate in your abdominal cavity (ascites) and in your legs as a result of liver damage (cirrhosis).
- **Kidney disease.** When you have kidney disease, extra fluid and sodium in your circulation may cause edema. The edema associated with kidney disease usually occurs in your legs and around your eyes.
- **Kidney damage.** Damage to the tiny, filtering blood vessels in your kidneys can result in nephrotic syndrome. In nephrotic syndrome, declining levels of protein (albumin) in your blood can lead to fluid accumulation and edema.

•**Weakness or damage to veins in your legs.** If you have chronic venous insufficiency, the one-way valves in your leg veins are weakened or damaged, which allows blood to pool in your leg veins and causes swelling. Sudden onset of swelling in one leg accompanied by pain in your calf muscle can be due to a blood clot (deep vein thrombosis, or DVT) in one of your leg veins. If this occurs, seek medical help immediately.

Inadequate lymphatic system. Your body's lymphatic system helps clear excess fluid from tissues. If this system is damaged — for example, by cancer surgery — the lymph nodes and lymph vessels draining an area may not work correctly, and edema can occur.

Severe, long-term protein deficiency. An extreme lack (deficiency), of protein in your diet over a long period of time can lead to fluid accumulation and edema

Risk factors

- High blood pressure medications
- Nonsteroidal anti-inflammatory drugs
- Steroid drugs
- Estrogens
- Certain diabetes medications called thiazolidinedione
- A chronic illness — such as congestive heart failure or liver or kidney disease — can increase your risk of edema. Also, surgery can sometimes obstruct a lymph node, leading to swelling in an arm or leg, usually on just one side.

Complications

- Increasingly painful swelling
- Difficulty walking
- Stiffness
- Stretched skin, which can become itchy and uncomfortable
- Increased risk of infection in the swollen area
- Scarring between layers of tissue
- Decreased blood circulation
- Decreased elasticity of arteries, veins, joints and muscles
- Increased risk of skin ulcers

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THANKS FOR YOUR ATTENTION