

**MINISTRY OF HEALTHCARE OF UKRAINE
HSEEU "Ukrainian Medical Stomatological Academy"**

"Approved"
at the meeting of internal
medicine №1 department
Head of Department
Prof. Skrypnyk I.M.

Protocol № 4 from 13.10.2016

**GUIDELINES
FOR STUDENTS
INDEPENDENT WORK
IN THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Current practice of internal medicine
<i>Content module</i>	Management of the patients with main symptoms and syndromes in cardiology clinic
<i>Study subject</i>	Management of patients with edematous syndrome
<i>Course</i>	VI
<i>Faculty</i>	of foreign students training

1. The aims of the training course:

To Know:

1. Definition and classification.
2. Etiological factors.
3. Classification.
4. Pathogenesis of lesions in organs and systems, their clinical manifestations.
5. Clinic and laboratory parameters.
6. Differential treatment at different stages.
7. Weather and performance.

To be able to:

- Conduct surveys and examination of patients with edematous syndromes
- Know the basic invasive and noninvasive diagnostic techniques used in internal medicine, indications and contraindications for their conduct, possible complications
- identify major and atypical variants of the course and complications of edematous syndromes
- draft examination of patients with edematous syndromes
- Based on analysis of laboratory and instrumental examination to conduct differential diagnosis, justify and formulate diagnoses for diseases of cardiovascular, urinary and other systems
- prescribe treatment, determine prognosis, to conduct primary and secondary prevention
- diagnose and assist in edematous syndromes

The contents of topic:

Text

Merck Manual Professional Version <http://www.merckmanuals.com/professional>

By Lyall A. J. Higginson, MD

Edema is swelling of soft tissues due to increased interstitial fluid. The fluid is predominantly water, but protein and cell-rich fluid can accumulate if there is infection or lymphatic obstruction.

Edema may be generalized or local (eg, limited to a single extremity or part of an extremity). It sometimes appears abruptly; patients complain that an extremity suddenly swells. More often, edema develops insidiously, beginning with weight gain, puffy eyes at awakening in the morning, and tight shoes at the end of the day. Slowly developing edema may become massive before patients seek medical care.

Edema itself causes few symptoms other than occasionally a feeling of tightness or fullness; other symptoms are usually related to the underlying disorder. Patients with edema due to heart failure (a common cause) often have dyspnea during exertion, orthopnea, and paroxysmal nocturnal dyspnea. Patients with edema due to deep venous thrombosis (DVT) often have pain.

Edema due to extracellular fluid volume expansion is often dependent. Thus, in ambulatory patients, edema is in the feet and lower legs; patients requiring bed rest develop edema in the buttocks, genitals, and posterior thighs. Women who lie on only one side may develop edema in the dependent breast. Lymphatic obstruction causes edema distal to the site of obstruction.

Pathophysiology

Edema results from increased movement of fluid from the intravascular to the interstitial space or decreased movement of water from the interstitium into the capillaries or lymphatic vessels. The mechanism involves one or more of the following:

- Increased capillary hydrostatic pressure
- Decreased plasma oncotic pressure
- Increased capillary permeability
- Obstruction of the lymphatic system

As fluid shifts into the interstitial space, intravascular volume is depleted. Intravascular volume depletion activates the renin-angiotensin-aldosterone- vaspressin (ADH) system, resulting in renal Na retention. By increasing osmolality, renal Na retention triggers water retention by the kidneys and helps maintain plasma volume. Increased renal Na retention also may be a primary cause of fluid overload and hence edema. Excessive exogenous Na intake may also contribute.

Less often, edema results from decreased movement of fluid out of the interstitial space into the capillaries due to lack of adequate plasma oncotic pressure as in nephrotic syndrome, protein-losing enteropathy, or starvation.

Increased capillary permeability occurs in infections or as the result of toxin or inflammatory damage to the capillary walls.

The lymphatic system is responsible for removing protein and WBCs (along with some water) from the interstitium. Lymphatic obstruction allows these substances to accumulate in the interstitium.

Etiology

Generalized edema is most commonly caused by

- Heart failure
- Liver failure
- Kidney disorders (especially nephrotic syndrome)

Localized edema is most commonly caused by

- DVT or another venous disorder or venous obstruction (eg, by tumor)
- Infection
- Angioedema
- Lymphatic obstruction

Chronic venous insufficiency may involve one or both legs.

Common causes are listed by primary mechanism.

Common causes are listed by primary mechanism (see Table: [Some Causes of Edema](#)).

Some Causes of Edema

Cause	Suggestive Findings	Diagnostic Approach ^a
Increased hydrostatic pressure, fluid overload		
Right heart failure (primary or secondary to left-sided disease) directly increasing venous pressure	Symmetric, dependent, painless, pitting edema, often with dyspnea during exertion, orthopnea, and paroxysmal nocturnal dyspnea Commonly, lung crackles, S ₃ or S ₄ gallop or both, and jugular venous distention, hepatjugular reflux, and Kussmaul sign	Chest x-ray and ECG Usually echocardiography
Pregnancy and premenstrual state	Apparent by history	Clinical evaluation
Drugs (eg, minoxidil , NSAIDs, estrogens, fludrocortisone , dihydropyridine , diltiazem , other Ca channel blockers)	Symmetric, dependent, painless, usually mild pitting edema	Clinical evaluation
Iatrogenic (eg, excessive IV fluids)	Apparent by history and medical record	Clinical evaluation
Increased hydrostatic pressure, venous obstruction		
DVT	Acute, pitting edema in a single, usually lower extremity, usually with pain; sometimes Homans sign (pain in the calf when the foot is dorsiflexed) Redness, warmth, and tenderness; possibly less marked than in soft-tissue infection Sometimes a predisposing factor (eg, recent surgery, trauma, immobilization, hormone replacement, cancer)	Ultrasonography
Chronic venous insufficiency	Chronic edema in one or both lower extremities, with brownish discoloration, discomfort but not marked pain, and sometimes skin ulcers Often associated with varicose veins	Clinical evaluation
Extrinsic venous compression (by tumor, a gravid uterus, or marked abdominal obesity)	Nonpainful, slowly developing edema If tumor compresses the superior vena cava, usually facial plethora, distended neck veins, and absent venous pulse waves above the obstruction	Clinical evaluation Ultrasonography or CT if tumor is suspected
Prolonged absence of skeletal muscle pumping activity on extremity veins	Prolonged immobility (eg, being bedbound or on a long airline flight) Painless, symmetric, dependent edema	Clinical evaluation

Evaluation

History

History of present illness should include location and duration of edema and presence and degree of pain or discomfort. Female patients should be asked whether they are pregnant and whether edema seems related to menstrual periods. Having patients with chronic edema keep a log of weight gain or loss is valuable.

Review of systems should include symptoms of causative disorders, including dyspnea during exertion, orthopnea, and paroxysmal nocturnal dyspnea (heart failure); alcohol or hepatotoxin exposure, jaundice, and easy bruising (a liver disorder); malaise and anorexia (cancer or a liver or kidney disorder); and immobilization, extremity injury, or recent surgery (DVT).

Past medical history should include any disorders known to cause edema, including heart, liver, and kidney disorders and cancer (including any related surgery or radiation therapy). The history should also include predisposing conditions for these causes, including streptococcal infection, recent viral infection (eg, hepatitis), chronic alcohol abuse, and hypercoagulable disorders. Drug history should include specific questions about drugs known to cause edema. Patients are asked about the amount of Na used in cooking and at the table.

Physical examination

The area of edema is identified and examined for extent, warmth, erythema, and tenderness; symmetry or lack of it is noted. Presence and degree of pitting (visible and palpable depressions caused by pressure from the examiner's fingers on the edematous area, which displaces the interstitial fluid) are noted.

In the general examination, the skin is inspected for jaundice, bruising, and spider angiomas (suggesting a liver disorder).

Lungs are examined for dullness to percussion, reduced or exaggerated breath sounds, crackles, rhonchi, and pleural friction rub.

The internal jugular vein height, waveform, and reflux are noted.

The heart is palpated for thrills, thrust, parasternal lift, and asynchronous abnormal systolic bulge. Auscultation for loud pulmonic component of 2nd heart sound (P₂), 3rd (S₃) or 4th (S₄) heart sounds, murmurs, and pericardial rub or knock is done; all suggest cardiac origin.

The abdomen is inspected, palpated, and percussed for ascites, hepatomegaly, and splenomegaly to check for a liver disorder or heart failure. The kidneys are palpated, and the bladder is percussed. An abnormal abdominal mass, if present, should be palpated.

Red flags

Certain findings raise suspicion of a more serious etiology of edema:

- Sudden onset
- Significant pain
- Shortness of breath
- History of a heart disorder or an abnormal cardiac examination
- Hemoptysis, dyspnea, or pleural friction rub
- Hepatomegaly, jaundice, ascites, splenomegaly, or hematemesis
- Unilateral leg swelling with tenderness

Interpretation of findings

Potential acute life threats, which typically manifest with sudden onset of focal edema, must be identified. Such a presentation suggests acute DVT, soft-tissue infection, or angioedema. Acute DVT may lead to pulmonary embolism (PE), which can be fatal. Soft-tissue infections range from minor to life threatening, depending on the infecting organism and the patient's health. Acute angioedema sometimes progresses to involve the airway, with serious consequences.

Dyspnea may occur with edema due to heart failure, DVT if PE has occurred, acute respiratory distress syndrome, or angioedema that involves the airways.

Generalized, slowly developing edema suggests a chronic heart, kidney, or liver disorder. Although these disorders can also be life threatening, complications tend to take much longer to develop.

These factors and other clinical features help suggest the cause.

Testing

For most patients with generalized edema, testing should include CBC, serum electrolytes, BUN, creatinine, liver function tests, serum protein, and urinalysis (particularly noting the presence of protein and microscopic hematuria). Other tests should be done based on the suspected cause, brain natriuretic peptide (BNP) for suspected heart failure or D-dimer for suspected PE.

Patients with isolated lower-extremity swelling should usually have venous obstruction excluded by ultrasonography.

Treatment

Specific causes are treated.

Patients with Na retention often benefit from restriction of dietary Na. Patients with heart failure should eliminate salt in cooking and at the table and avoid prepared foods with added salt. Patients with advanced cirrhosis or nephrotic syndrome often require more severe Na restriction (≤ 1 g/day). K salts are often substituted for Na salts to make Na restriction tolerable; however, care should be taken, especially in patients receiving K-sparing diuretics, ACE inhibitors, or angiotensin receptor blockers and in those with a kidney disorder because potentially fatal hyperkalemia can result.

People with conditions involving Na retention may also benefit from loop or thiazide diuretics. However, diuretics should not be given only to improve the appearance caused by edema. When diuretics are used, K wasting can be dangerous in some patients; K-sparing diuretics (eg, amiloride, triamterene, spironolactone, eplerenone) inhibit Na reabsorption in the distal nephron and collecting duct. When used alone, they modestly increase Na excretion. Both triamterene and amiloride have been combined with a thiazide to prevent K wasting. An ACE inhibitor–thiazide combination also reduces K wasting.

Geriatrics Essentials

In the elderly, use of drugs that treat causes of edema requires special caution, such as the following:

- Starting doses low and evaluating patients thoroughly when the dose is changed
- Monitoring for orthostatic hypotension if diuretics, ACE inhibitors, angiotensin receptor blockers, or β -blockers are used
- Evaluating for bradycardia or heart block if digoxin, rate-limiting Ca channel blockers, or β -blockers are used
- Frequently testing for hypokalemia or hyperkalemia
- Not stopping Ca channel blockers because of pedal edema, which is benign

Logging daily weight helps in monitoring clinical improvement or deterioration immensely.

Key Points

- Edema may result from a generalized or local process.
- Main causes of generalized edema are chronic heart, liver, and kidney disorders.
- Sudden onset should trigger prompt evaluation.
- Edema may occur anywhere in the body, including the brain.
- Not all edema is harmful; consequences depend mainly on the cause.

Last full review/revision August 2014 by Lyall A. J. Higginson, MD

Self preparation at class:

Listen information;

Work with patients (with cardiac pathology);

Ask about the problems that have not been found in information given.

Self preparation at home:

Compose the plan of your answer;

Answer the questions to the topic;

Do the test given above.

Questions

1. A patient who suffers from heart failure has enlarged liver, edemata of lower extremities, ascites. What is the leading mechanism in the development of this edema?
 - A Hydrodynamic
 - B Colloid osmotic
 - C Lymphogenous
 - D Membranogenic
 - E Mix
2. A patient with chronic cardiac insufficiency have edema of lower extremities. What is the leading link of their pathogenesis?
 - A. Hypothalamo-hypophysis system
 - B. Renin-angiotensin-aldosterone system
 - C. Sympato-adrenal system
 - D. Parasympathetic system
 - E. Kalikrein-kinin system
3. Strong decreased value of albumins and oncotic pressure of blood plasma takes place in a patient. What will be the result of it?
 - A. Increase of blood volume
 - B. Decrease of diuresis
 - C. Edemas
 - D. The ESR decrease
 - E. Increase of blood viscosity

Recommended literature:

A. Main:

1. "Harrison's principles of internal medicine", Editors: Anthony S. Fauci, Dennis L. Kasper, Stephen L Hauser, Dan L. Longo, Joseph Loscalzo, McGraw-Hill Education / Medical; 19 edition (April 8, 2015), 1-2 volumes, 3000 p.
2. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)
3. CURRENT Diagnosis and Treatment Emergency Medicine, Seventh Edition (LANGE CURRENT Series) by C. Keith Stone (May 23, 2011)
4. Goldman's Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer.—24th ed. Elsevier Sanders. Rev. ed. of: Cecil medicine. 23rd ed. – 2012. p.

Answers

1. A Hydrodynamic
2. B Renin-angiotensin-aldosterone system
3. C. Edemas

Methodical recommendations consisted by

Kulishov S.K.