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The clinical aspects of edema in general practice

Kliniczne aspekty obrzęków w praktyce lekarza ogólnego

CAUSES AND SYMPTOMS ASSOCIATED WITH EDEMA.

HEART

- Decreased heart perfusion rate leads to stagnant blood within lungs
- Left sided heart failure can lead to retention of fluid within the lungs because of blood back flow
- Accumulation of blood persists within the right atrium which can lead to portal venous congestion, hepatosplenomegly, kidney congestion, ascites, and peripheral edema
- Symptoms include: dyspnea, fatigue, and orthopnea

LIVER

- Reduction in albumin production can lead to disturbance in oncotic and hydrostatic pressure
- Hypoalbuminemia occurs with chronic liver diseases: cirrhosis, alcoholism, and viral infections (Hepatitis).
- Symptoms would include: peripheral pitting edema, ascites, pleural effusion, and portal hypertension

LUNGS

Cardiogenic Edema:

- increased end-diastolic ventricular volume
- increase in the hydrostatic pressure of the pulmonary capillaries, leading to an outflow of plasma into the alveoli

- increased capillary hydrostatic pressure caused by myocardial infarction, stenosis, or mitral valve regurgitation
- cardiac myopathy can persist with increased levels of epinephrine and norepinephrine secreted stunning the heart and causes decreased cardiac output, thus increasing capillary hydrostatic pressure
- catecholamines increase left ventricular afterload by increasing systemic vascular resistance. This leads to a reduction in left ventricular ejection, with increased end-diastolic volume and increased pulmonary wedge pressure with resultant edema.

Noncardiogenic Edema:

- aspirations
- trauma
- high altitude
- drug use
- reexpansions
- infections
- obstruction of airways
- hypoxia stimulated by vasoconstriction
- increase in the intrapleural negative pressure results in increased pulmonary blood pressure and increased hydrostatic pressure in the pulmonary capillaries leading to edema

KIDNEY

- prerenal, intrarenal, and postrenal diseases lead to edema
- decreased clearance of proteins, electrolytes, waste
- excess urea, water, creatinine, and sodium in the serum leading to change in oncotic pressure
- proteinuria (decreased protein leads to change of homeostasis between hydrostatic and oncotic pressures)
- defect in Renin Angiotensin System
- hyperaldosteronism (Conn's syndrome increases reabsorption of sodium and water
- syndrome of Inappropriate Antidiuretic Hormone leads to hyponatremia, leading to cerebral edema

LYMPHATIC SYSTEM

- obstruction of lymph drainage
- defects in lymphatic pumps: *Lymphedema Praecox* or *Lymphedema Tarda*
- diapedesis
- resection of lymph nodes or radiation can cause inflammation and fibrosis of drainage leading to edema
- intestinal lymphangiectasia

CLINICAL MANIFESTATION

Clinically there are several symptoms that will appear in almost all cases of edema. On a general examination, edema is visually identified as a swollen area covered with shiny, stretched skin [2]. Most commonly, edema presents with peripheral fluid retention. This may be present in locations such as in the lower limbs, around the eyes, and in the abdominal cavity. In the lower limbs, there is an increased occurrence of pitting edema, in which a dimple is formed after a certain amount of pressure is applied and removed from an area of skin. This occurs due to the displacement of interstitial fluid [3]. Pitting edema correlates with dependent edema, in which gravity mediates the accumulation of fluid into the lower limbs [refer to Figure 1]. It is common to see edema of the lower limbs with relation to varicose veins, which are dilated veins that usually occur in the superficial veins of the lower limb [2]. This phenomenon is most commonly seen with increased venous pressure. It is important to recognize whether the limb edema is unilateral or bilateral and whether there is pain upon pressure; this may indicate such conditions as thrombosis or acute cellulitis. Another common clinical manifestation of edema is dyspnea, or shortness of breath. This phenomenon occurs due to the accumulation of fluid in the pleural cavity and lung tissue, most commonly brought on by congestive heart failure. Patients may complain of such respiratory manifestations as paroxysmal dyspnea, orthopenia, and a condition known as Cheyne-Stokes respirations, in which there is a slowing and waning of breathing that occurs every 40-60 seconds [4]. Due to these respiratory difficulties, fatigue and weakness may be present, as well as cerebral symptoms. Cyanosis, most prevalent in the lips and nail beds, may also occur. While clinically examining the effects of edema, is it also important to notice the extent, warmth, and erythema of the area, which will further determine the primary cause of the edema.

PATHOPHYSIOLOGY OF EDEMA

The movement of fluid between vessels and the interstitial tissues are determined by a variety of physiological factors that are termed "starling forces." These include the hydrostatic and oncotic pressures of both the fluid in the vessels and in the interstitial tissue. Hydrostatic pressure is the physical pressure exerted by the fluid in the given compartment, while oncotic pressure is the pulling pressure provided by the negatively charged proteins in the tissue or fluid. In normal body physiology, the balance is slightly shifted toward fluid entering the interstitial tissue and being subsequently cleared by the lymphatic system. Edema fluid due to defects in the Starling equilibrium is typically protein-poor due to the lack of diffusion of the relatively large protein molecules across the capillary surface. This fluid is termed "transudate" and is defined as having a specific gravity of less than 1.012.

Changes in the "starling forces" of blood vessels that would favor edema are usually increased hydrostatic pressure and decreased oncotic pressure. Increased hydrostatic pressure is typically a result of increased local blood volume, such as that seen in impaired venous return due to, for example, distal venous obstruction or arterial dilation. Decreased oncotic pressure results from a relative lack of proteins in the plasma, leading to a deficiency of the pulling force present. This may be due to deficient intake, increased excretion, or decreased production of proteins [5].

Another important cause of edema is lymphatic obstruction, leading to an accumulation of fluid in the proximal interstitial fluid. The obstruction may be due to a variety of factors such as neoplasm and post surgical consequences. This specialized form of edema is termed "lymphedema."

A common cause of edema is inflammation, which leads to mechanical and physical defects of capillary walls with resulting increased vascular permeability and increased blood volume due to vasodilation. As a result, the capillary epithelial cells may be shrunken with increased intercellular spaces for the movement of molecules across the capillary interface. Due to these spaces, fluid with a relatively high concentration of proteins is able to leak through the walls of the capillaries and into the interstitial fluid from the plasma. This protein rich fluid is termed "exudate" and has a specific gravity of 1.020 or higher.

A number of renal syndromes [along with renal hypoperfusion] also cause an increase in the RAA system, which ultimately leads to salt and water retention in the plasma. As a result, the increased blood volume leads to an increased hydrostatic pressure, leading to edema. Due to renal hypoperfusion due to decreased blood volume caused in many edematous states, this mechanism is known to complicate preexisting edema [1].

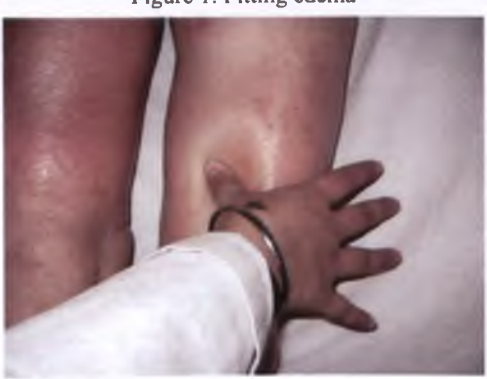
HEART FAILURE

Heart failure is the inability of the heart to pump enough blood to adequately perfuse bodily tissue, as well as the inability to prevent blood from collecting in the lungs. These pathophysiological consequences of heart failure are one of the primary causes of edema, which can gradually worsen as time progresses. There are two types of heart failure: left-sided heart failure and right-sided heart failure. Left-sided heart failure can be caused by a number of disorders, including ischemic heart disease, myocardial diseases, and aortic and mitral valve disorders [1]. In this situation, the left side of the heart is unable to properly pump the blood out towards the aorta and the rest of the body. Consequently, blood backflows into the blood vessels of the lungs, and in some cases may collect into the actual tissue of the lung. This causes pulmonary congestion and edema [6]. Morphologically, there is a sequence of events that occurs during pulmonary congestion. First is the presentation of perivascular and interstitial edema. Next, there is evidence of widening in the alveolar septa due to edema. Finally, edema fluid is accumulated in the alveolar spaces [1]. All of these manifestations are clinically present with dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and fatigue [6]. Left-sided heart failure is further divided into systolic failure, in which the heart's pumping ability is compromised, and diastolic failure, in which the heart is unable to properly expand to fill with blood; the latter may lead to a rapid onset of pulmonary edema [1]. In right-sided heart failure, the heart is unable to properly pump blood to the lungs for oxygenation [1]. The cause of right-sided heart failure is most commonly left-sided heart failure, as well as tricuspid valve failure and those causes that attribute to left-sided heart failure [1]. Due to the lack of blood that is pumped to the lungs, there is an accumulation of blood within the right ventricle and atrium. This eventually leads to such systemic manifestations as systemic and portal venous congestion, hepatosplenomegly, kidney congestion, ascites, and peripheral edema, especially of pedal and pretibial region. Morphologically, the right side of the heart presents with hypertrophy and dilation, and the liver is characteristically named "nutmeg liver" due to its centilobular discoloration and peripheral fatty changes [1] [6]. The most prominent clinical manifestations of right-sided heart failure include generalized edema [most prominent in the abdominal cavity and lower limbs], dependent edema due to gravity, and nocturia, which occurs due to the redistribution of fluid that occurs in the position of lying down [6].

LIVER FAILURE

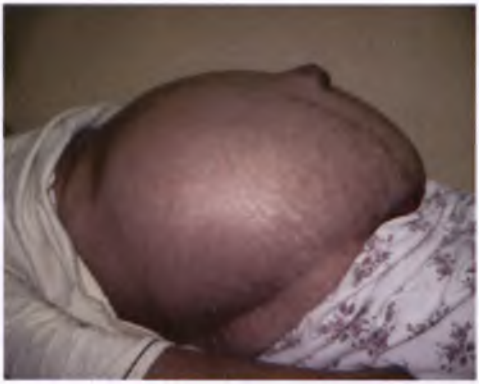
The human liver, representing just over 2% of the total human body weight, plays such vital roles as metabolizing important bodily components, filtering and storing blood, and forming bile [4]. In relation to the pathophysiology of edema, the most important aspect of liver function that is affected is the formation of albumin. Albumin, a serum protein that is produced in the liver, plays an important role in maintaining the plasma oncotic pressure, which is the pressure from plasma proteins that is exerted onto the capillary walls. This pressure, which normally values around 28 mm Hg, is the pressure that allows fluid to be pulled out of the interstitium and directed towards the plasma, hence holding the fluid within the blood vessels and maintaining a level of equilibrium. [4]. Therefore, any changes to this equilibrium will cause drastic changes in the fluid distribution within the body. The main cause of hypoalbuminemia is chronic liver diseases such as liver cirrhosis. Cirrhosis, a liver disease that is marked by severe scarring, is a condition that can be brought upon by such consequences as abundant alcohol intake, infection by hepatitis B and C, and nonalcoholic steatohepatitis. These conditions cause continuous injury to the liver. Since the liver is a self-repairing organ, each injurious episode is followed by a healing process. Eventually, the scar tissue that forms becomes so significant that it impairs the liver's ability to perform its usual function, with one such consequence being the inadequate formation of albumin [12]. The decrease of plasma albumin decreases the plasma oncotic pressure, which inhibits the ability of the blood capillaries to keep fluid within its vessel walls. Therefore, fluid escapes into the interstitial space, causing edema [13]. There are several clinical representations of edema caused by liver disease. Pitting edema of the lower extremities may be observed, in which fluid buildup in the lower legs causes an indentation in the skin when pressure is applied, This indentation remains for some time even after the pressure is removed [2].

Figure 1. Pitting edema



Ascites, or fluid accumulation in the abdominal cavity, can also occur with hypoalbuminemia, and this can be further complicated with portal hypertension.

Figure 2. Ascites



In some cases, pleural effusion may also occur, which is the accumulation of fluid within the pleural cavity [7].

PULMONARY EDEMA

Pulmonary edema is categorized into two groups: cardiogenic and non-cardiogenic. Cardiogenic pulmonary edema is the most common form and is related to disorders of the heart, mainly left ventricular failure. Non-cardiogenic pulmonary edema may be due to other causes, such as respiratory disorders and renal failure. The pathophysiology of pulmonary edema consists of the extravasation of fluid from the pulmonary capillaries into the alveoli of the lung. In addition to blocking oxygen flow into the capillaries, this fluid creates an environment favorable to infection. Resultant symptoms include dyspnea, orthopnea, and persistent cough. Treatment is often aimed at the underlying syndrome.

Similarly to various systemic manifestations of edema present in patients with right-sided heart failure, patients with left ventricular heart failure present with pulmonary edema due to the impaired pulmonary venous return to the heart. In patients with left-sided heart failure, the left ventricle is unable to adequately pump enough blood into the systemic circulation. As a result, there is a "backward failure" caused by the increased end-diastolic ventricular volume. The resultant increase in left atrial and pulmonary venous pressures causes an increase in the hydrostatic pressure of the pulmonary capillaries, leading to an outflow of plasma into the alveoli in order to reestablish Starling equilibrium in the capillaries. The presence of hemosiderin-laden macrophages in the alveoli is a prominent histological marker of pulmonary edema secondary to heart failure [1].

Other forms of cardiogenic pulmonary edema largely center on other causes of increased capillary hydrostatic pressure [5]. These may include arrhythmias, heart attacks, and obstructive disorders such as aortic stenosis and mitral valve regurgitation. These obstructive disorders result in an increased end-diastolic ventricular volume such as that seen in heart failure due to the inability of the blood to leave the heart. Other causes are cardiomyopathies of the heart. Acute pulmonary edema may be seen in patients that have developed stress-related cardiomyopathy. This state is brought about by physical or emotional stress, which stimulates the body to secrete increasing amounts of epinephrine and norepinephrine. When added to a preexistent obstructive condition such as aortic stenosis, the toxic levels of these catecholamines

can "stun" the heart and decrease cardiac output, leading to the aforementioned pulmonary hypertension and increased capillary hydrostatic pressure. Catecholamines also increase left ventricular afterload by increasing systemic vascular resistance. This leads to a reduction in left ventricular ejection, with a resultant increased end-diastolic volume and increased pulmonary wedge pressure with resultant edema [8].

Among the non-cardiogenic causes of pulmonary edema, respiratory-related causes are the most exclusive to pulmonary edema alone. These causes are due to factors such as aspirations, re-expansions, infections, and obstruction of airways. These obstructions lead to an increase in the negative intrathoracic pressure. These obstructive conditions include syndromes such as ARDS [acute respiratory distress syndrome] and COPD [chronic obstructive pulmonary disorder] and other causes of obstruction, such as laryngeal spasms in anesthetic use. The increase in the intrapleural negative pressure results in increased pulmonary blood pressure and increased hydrostatic pressure in the pulmonary capillaries, such as that seen in the previous examples of heart failure. The hypoxemia that occurs due to the resultant edema and the preexistent airway obstruction leads to an increase of the excretion of catecholamines, further triggering edema by the mechanisms introduced in the previous paragraph. Another further trigger is the pulmonary vasoconstriction that occurs due to hypoxemia. Hypoxemia is also a cause of capillary wall damage, leading to leakage of other vascular elements into the alveoli and interstitium [9].

Other causes of pulmonary edema include renal failure [refer to appropriate section], ascent to high altitudes, trauma, and drug use.

Pulmonary edema is generally treated by treating the underlying cause [i.e. digoxin in patients with heart failure]. In case of emergency or specific treatment of pulmonary edema, nasal oxygen is administered in conjunction with morphine sulfate, furosemide, or sublingual nitroglycerin [10].

RENAL DISORDERS

Renal disease is a prime marker for edema. There are three classifications of renal disease: prerenal, intrarenal, and postrenal disease. Prerenal disease is renal failure caused by decreased blood flow to the kidney. This can be stimulated by a thrombus in renal artery or hypotension. Blood flows from the cortex to the medulla. Since the medulla is exposed to less blood volume than the cortex, this may lead to susceptibility of the medulla to a thrombus or embolus. Ischemia is lack of oxygen readily available to the kidney, which can cause kidney failure. Intrarenal disease is evident by damage to the nephron. Acute intrarenal disease is caused by vasculitis, scleroderma, or malignant hypertension. Chronic intrarenal is known to be caused by nephrosclerosis. Postrenal disease pertains to the urinary tract distal to the kidney. This could be due to prostatic hypoplasia, stones, or increased inflammation. Renal disease is usually characteristic of decreased clearance. These diseases result in alterations in the filtration, secretion, or absorption leading to excess urea, water, creatinine, and sodium in the serum. If water is not cleared within the kidney as urine, it is retained as excessive fluids, leading to ascites and edema. In acute renal failure, there is a common occurrence of proteinuria, which leads to edema due to the decreased amount of protein in serum, leading to decreased oncotic pressure. Chronic renal failure is commonly caused by diabetes mellitus. Destruction of the nephron increases the workload of the functioning nephrons which increases glomerular filtration pressure. This would further destroy the healthy nephrons and contribute to mass fibrosis. In this instance, there is no longer an adequate secretion of waste metabolites. They are further kept within the plasma, leading to edema due to alterations in pressures of hydrostatic and oncotic volumes [11].

Edema may also be caused by a bodily mechanism called the renin-angiotensin system, which begins in the kidney. This system is regulated by the kidney and heart and provides the balance of blood pressure by mediating the amount of sodium and water retention. Hormones trigger this system into action. Renin is secreted by the juxtaglomerular cells of the kidneys in response to extracellular and intracellular levels of calcium. Release by the cells is dependent upon intrarenal baroreceptors of the afferent arterioles, the macula densa receiving adequate supply of sodium chloride, and sympathetic stimulation of the arterioles of the juxtaglomerular apparatus [12]. Renin cleaves angiotensinogen into angiotensin I. Angiotensin I is then cleaved to angiotensin II by angiotensin converting enzyme. Angiotensin II is responsible for systemic vasoconstriction of arteries, decreasing the amount of blood filtered by the kidney. This triggers aldosterone to be released from adrenal cortex. Aldosterone increases sodium and water reabsorption, while decreasing the amount of potassium reabsorption [12]. This whole system is designed to increase arterial blood volume during hypotension, but pathology in this system could cause considerable amounts of edema. Hyperaldosteronism is the oversecretion of aldosterone [Conn's syndrome]. This leads to increased reabsorption of sodium and water, which contributes to edema because of the increased hydrostatic pressure and decreased oncotic pressure. Retention of sodium can also be caused by poststreptococcal glomerulonephritis and acute renal failure [1]. The posterior lobe of the pituitary gland releases a hormone called antidiuretic hormone. This hormone functions to increase the amount of water being reabsorbed or secreted to keep a balanced blood pressure. Syndrome of inappropriate ADH is related to excessive ADH hormone secretion. Effects of hypersecretion of ADH include hyponatremia, a state of lowered sodium concentration in plasma leading to cerebral edema [13]. The edema is not characteristic for Conn's syndrome and Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH). Edema in a hyponatremic patient is inconsistent with SIADH and may represent another hyponatremic states such as CHF, cirrhosis, or nephrotic syndrome.

LYMPHEDEMA

Lymphedema is characterized by lymph nodes being swollen due to increased capillary filtration and an obstruction in the lymph drainage [14]. The lymph system in our body is designed to remove unwanted material from the plasma, filter it, reabsorb any usable products [protein, colloid, etc.], and then recirculate it throughout the blood. The homeostasis of hydrostatic pressure and oncotic pressure is important in the lymphatic system. Hydrostatic pressure is intravascular pressure within capillaries. Proteins are usually the molecules that produce intravascular colloid osmotic pressure [1]. Primary lymphedema is a consequence of defected lymph pumps. Our body's lymph system is not equipped with a main pump to allow for accurate circulation, as the heart does for the blood, so the lymphatic system must rely on muscular movement in order to contract these one-way valves to allow for lymph to flow fluidly. Pathology of these valves contributes to primary lymphedema. Primary lymphedema can be sorted into *Lymphedema Praecox* or *Lymphedema Tarda. Praecox* occurs at birth with defects in abnormal collection lymphatic vessels, while *Tarda* is manifested later in life. *Tarda* has been established for the later onset because of the atrophy of smooth muscles beginning proximally and moving distally [14]. The more common secondary lymphedema occurs with factors outside of the lymphatic system. Secondary lymphedema can consist of infections, cancer, and diseases of vital organs.

An increase in vascular permeability due to diapedesis is an important source of edema [1]. Injury to the body can be caused by foreign particles [bacteria/viruses] or endogenous agents [autoimmune antibodies]. Injury usually permits inflammation, which causes attraction of lymphocytes, macrophages, and inflammatory cells to the site of injury. These infiltrates will help the body rid itself of the harmful agent. One mechanism crucial to the participation of increasing infiltrate to site of injury is diapedesis or transcellular migration. The body allows for the basement membrane of the vascular epithelium to become more permeable for these cells to cross between barriers in a more efficient way to allow for speedy arrival to damaged area [15]. This allows for edema to occur because of increased capillary permeability to infiltrates.

Lymphedema is very common in parasite infestation with main contributions from *Wuchereria bancrofti* and *Brugia malayi*. The filarial parasites cause a weakness within the lymphatic structure by secreting harmful parasitic encoded proteins. These parasites also make a nest or living space within the lymphatic system and cause fibrosis or scarring of the lymphatic vessel or surrounding tissue. These obstructions lead to lymphedema and severe elephantitis [16]. Clinical manifestations of this type of lymphedema consist of massive edema of the legs and scrotum [elephantitiasis], lymphagitis, and cellulitis. Early in the infection, however, there are signs of coughing and wheezing; this is due to the parasite's larvae, also called microfilariae, infiltrating the lungs and are contributing to a hypersensitivity reaction [17].

Those patients that have undergone mastectomy, radiation therapy, or lymph node resection due to breast cancer, usually are at a higher likelihood of acquiring lymphedema [most commonly in the arm] [18]. In the treatment of breast cancer, lymphedema occurs due to obstruction, inflammation, or complete resection or destruction of the lymph nodes. Accumulation of lymph fluid, or chyle, allows for the edema to manifest. Unfortunately, lymphedema of the limbs is a common complication in the treatment of breast cancer.

Intestinal lymphangiectasia is a generalized dilation of the small intestine lymphatics. This results in hypoproteinemia and anasarca [generalized edema] [19]. Primary lymphangiectasia, Waldmann's Disease, is caused by congenital malformations in the lymphatic system. Waldmann's Disease leads to hypoalbuminemia, lymphopenia, and hypogammaglobulinemia [20]. These manifestations lead to bilateral lower limb edema and ascites.

Myxedema describes a specific form of cutaneous and dermal edema secondary to increased deposition of connective tissue components (like glycosaminoglycans, hyaluronic acid, and other mucopolysaccharides) in subcutaneous tissue as seen in various forms of hypothyroidism and Graves' disease [21]. Myxedema typically presents in specific areas (pretibial myxedema and exophthalmos) and is related to high levels of TSH receptor stimulation and/or inflammation mounted against the TSH receptor itself. Myxedema of the lower legs (called pretibial myxedema), can occur in 1-4% of patients with Graves' disease.

DIAGNOSIS

There are several diagnostic methods that are used to determine the presence of edema:

Physical exam: When physically examining a patient, there may be visual indications such as pitting edema, ascites, general swelling of certain tissues, dyspnea, and cyanosis.

Ultrasound: This test is very useful in the diagnosis of edema. It is especially useful in the instance of ascites, for the build-up of fluid within the abdominal cavity is clearly seen.

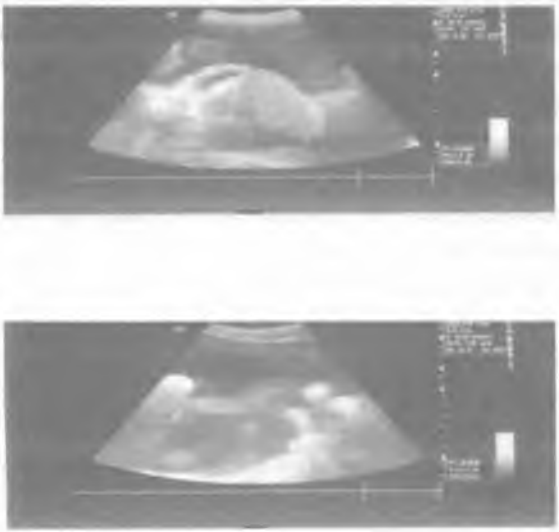


Figure 3 Ultrasonography of ascites

X-ray: This test is most useful in the diagnosis of pulmonary edema. In the initial stages of interstitial edema, thin horizontal lines called Kerley lines may be present due to cellular infiltration. As the edema eventually progresses into pulmonary edema, a distinct "butterfly" pattern is observed. Additionally, it is common to observe a noticeable enlargement of the heart [22].

Urinalysis/Blood tests: These tests are extremely important in the determination of the etiology of edema. The observed changes allow the determination of which of the organ systems is contributing to the resulting edema. For example, hypoalbuminemia diagnostically points to the liver as the source of the edema.

Table 1. Blood tests

Test	Reference Range	Comments
Albumin	3.2-5.0 g/dL In blood	Low levels are indicative of edema due to decreased oncotic pressure of hepatic or renal origin
B-type Natriuretic Peptide [BNP]	<100 pg/mL In blood	High levels indicative of edema due to heart failure
Blood Urea Nitrogen [BUN]	7-20 mg/dL In blood	Low levels indicative of nephritic syndrome
Free thyroxine [FT4]	0.7-1.5 ng/dL In blood	Low levels indicate possible myxedema due to hypothyroidism
C-reactive Protein [CRP]	<6 mg/L In blood	High levels indicate edema due to inflammation
Alanine Transaminase [ALT]	0-35 IU/dL In blood	Indicative of edema due to liver injury [i.e. cirrhosis]
Aspartate Transaminase [AST]	0-37 IU/dL In blood	
Urine Protein	<150 mg/day In urine	Indicative of edema due to renal disorder [i.e. nephrotic syndrome]

Electrocardiogram: This is a test that determines the electrical activity of the heart. This test measures aspects such as heart rate, cardiac problems such as arrhythmias and heart attacks, and problems with signal conduction [4]. This test is most useful in the presence of edema caused by heart failure.

Echocardiogram: This test is used to measure the function of the cardiac muscle. It focuses on the functionality of the heart valves, the size and thickness of the heart, and the overall pumping ability of the heart muscle. Like the electrocardiogram, this test is most useful in the presence of edema caused by heart failure [23].

Computed tomography/MRI: These tests are especially useful in the diagnosis of lymphedema. They allow the visualization of any obstruction that may occur in the lymph vessels [24].

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STRESZCZENIE

Obrzęk jest zdefiniowany jako klinicznie widoczne zwiększenie śródmiąższowej objętości płynu. Klinicznymi przyczynami obrzęków są niedrożność żylnego i limfatycznego odpływu z kończyn, niewydolność serca, zespół nerczycowy, marskość wątroby. Obrzęk miejscowy należy różnicować z obrzękiem uogólnionym. W artykule opisana została patofizjologia, objawy kliniczne i metody diagnostyczne obrzęków.

ABSTRACT

Edema is defined as a clinically apparent increase in interstitial fluid volume. Clinical causes of edema are: obstruction of venous and lymphatic drainage of a limb, heart failure, nephrotic syndrome, liver cirrhosis. Localized edema can usually be differentiated from generalized edema. In this article pathophysiology, clinical manifestation and diagnostics methods of edema were described.

Edema is the presence of increased fluid in the interstitial tissue. It is mostly a clinical indication of one of a variety of syndromes related to almost all of the organ systems of the body. Depending on the anatomical location of the edema, it may be termed "hydrothorax" [in the pleural space of the lungs], "hydropericardium" [in the pericardial space of the heart], or "hydroperitoneum" [in the peritoneal space of the abdomen]. Generalized edema throughout the body is termed "anasarca" [1]. Edema may be manifested by disorders of the heart, liver, lungs, kidney and lymphatic system.

Key words: edema, general practice.