Edematous syndrome

Classification of edema

Etiology

1. Edema as a part of nephritic syndrome, which can be caused by:
• glomerulonephritis,
• renal amyloidosis,
• diabetic glomerulosclerosis,
• nephropathy pregnant
• rheumatoid arthritis,
• serum sickness,
• systemic lupus erythematosus,
• lymphocytic leukemia,
• Hodgkin's disease.
2. Swelling due to circulatory failure, which developed as a result of:
• Heart Diseases,
• cardiosclerosis,
• decompensated pulmonary heart.
3. Orthostatic edema.
4. Swelling during pregnancy.
5. Swelling in diseases of major joints:
• deforming osteoarthritis,
• infectious arthritis,
• reactive arthritis.
6. Swelling in venous disease:
• acute deep vein thrombosis,
• chronic venous insufficiency (CVI).
7. Lymph edema (lymphedema).
8. Mixed swelling.

Thus, the main factors leading to disruption of local water balance can be:
1. Increase in hydrostatic pressure in the capillaries.
2. Decrease oncotic pressure of plasma.
3. Increased interstitial fluid oncotic pressure.
4. Reduction of tissue mechanical pressure.
5. Increased capillary permeability.
6. Violation of lymph drainage.

We distinguish local edema (localized) associated with fluid retention in a limited area of body tissue or organ, and total (generalized) – display of positive water balance in the body as a whole. Generalized edema includes edema in heart failure, liver cirrhosis, nephrotic and nephritic syndromes, dropsy pregnant, cachectic and idiopathic, and the chronic loss of body potassium, laxatives abuse.

Localized edema includes: cerebral edema, pulmonary edema, swelling of the extremities. Local edema in the absence of common violations of water-elecrolyte exchange and due to the presence of local hemo- and limfodynamic disorders, capillary permeability and metabolism.

The differential diagnosis of lower extremity edema in venous insufficiency with edema caused by heart failure, venous pressure should be measured in the ulnar vein. Swelling of the veins in the defeat mild or moderate density, edematous skin is warm. When lymphostasis often occur irreversible subcutaneous seal. For recognition of hydrothorax and ascites may require both basic and additional studies (X-ray, laboratory analysis of intracavitary fluid, etc.).

Pathogenesis of renal edema

Along with hypertension and urinary syndrome swelling occur in many renal diseases, especially

such as acute and chronic glomerulonephritis, nephropathy, pregnancy, renal amyloidosis, diabetic glomerulosclerosis. Edema – an almost constant and the most striking clinical manifestation of nephrotic syndrome. Severity of edema syndrome may be different. In some cases the swelling are negligible as face and legs puffiness, in others – modest and very apparent when viewed from the patient, in the third - pronounced, massive, often in the form of anasarca with ascites, hydrothorax, hydropericardium that the most typical in nephrotic syndrome . Sometimes the body may be delayed by 15-20 kg of fluid, but no more than 10-12% of body weight. Now, however, such massive edema is rare for the early use of diuretics and salt restriction.

In contrast, cardiac edema, which are typically located on sloping ground (feet and legs, in the lumbar region), renal edema are ubiquitous, on the face, trunk, extremities. In some cases they are formed quickly (within hours or days), in others – slowly, gradually, increasingly for many days. They can be solid to the touch, or, conversely, soft, when pressing a finger hole is a long time, such as nephrotic edema. Some patients with apparent edema may be absent, despite the apparent fluid retention in the body, determined by reduce the amount of urine (oliguria).

An adult's body may be delayed up to 2-3 and even up to 6-7 liters of fluid without the appearance of visible to the eye and established palpable edema. This is so-called latent edema. To confirm them and determine the degree of fluid retention in the body it is necessary to weigh the patient each day and determine the daily water balance, ie measure the amount of liquid consumed during the day (including soups, compotes, etc.) and urine (daily urine output).

When fluid retention before the appearance of visible edema is marked not only an increase in body weight (up to 0,5-1 kg per day), but also reducing the number of urine compared to the amount patient drink fluids. When the descent of hidden edema spontaneously or under the influence of diuretics rapidly decreases body weight through fluid loss, manifested significant increases urine output in excess of amounts of urine over the amount patient drink fluids during the day.

Edematous tissues readiness, or tendency to edema formation can be determined by the blister test, or McClure-Aldrich test. Intradermally on the anterior surface of the forearm with a fine needle syringe with small divisions introduced 0.2 ml of isotonic sodium chloride solution. In normal tissues hydrophilicity, lack of delay in the body fluid and edema resorption of formed bubble (blister) is slow, for 60 minutes, not less. In patients with a tendency to fluid retention and edema formation, resorption of blister is faster - for 40-30-10 minutes (depending on the degree of hydrophilicity of tissue), with a pronounced edematous tissues ready blister generally not formed as isotonic sodium chloride solution immediately absorbed.

Pathogenesis of renal edema

Pathogenesis of renal edema, as edema of other origins, complex and not fully understood.

From the pathophysiological point of view edema is an accumulation of fluid (water) in cell-cell interstitial spaces, accompanied by swelling of the ground substance of connective tissue and to a lesser extent the cells themselves that tissue (Vovsi, 1960). Edematous fluid is always formed from blood plasma, which in pathological conditions influenced by various factors not able to hold water in the bloodstream, and the water in excess accumulates in the tissues.

The most significant factors influencing the formation of edema, include the level of hydrostatic pressure of blood plasma concentration of protein (especially albumin), and sodium, the state of permeability of vascular walls, particularly the capillaries and the osmotic pressure in the tissues, which depends on the content of sodium. Under physiological conditions, there is a balance between hydrostatic and colloid osmotic (oncotic) pressure of the blood. In the arterial capillary hydrostatic pressure exceeds oncotic, resulting in water from the vascular changes in the tissue. In the venous capillary hydrostatic pressure below the oncotic, so the liquid from the interstitial space goes inside the vessels. Change the level of hydrostatic and colloid osmotic pressure, disturbance of physiological balance between them is one of the pathogenetic factors of edema formation.

Increased hydrostatic pressure plays a leading role in the pathogenesis of so-called heart (congestive) edema arising from various chronic diseases of the heart. Elevated hydrostatic pressure enhances the water filtration through the walls of arteries capillaries and hampers its absorption from the tissue through the wall of venous capillaries.

Oncotic pressure of blood, which is formed mainly due to albumin and normaly is 25-30 mm hg., promotes water retention in the bloodstream. Decrease oncotic pressure in some diseases, particularly in the nephrotic syndrome due to reduced total amount of protein and albumin, leads to increased maintenance of water into the tissues and promotes the development of edema.

Increased capillary permeability plays an important role in the origin of many types of edema, primarily of edema resulting from vascular injury because of inflammatory or allergic nature, including in glomerulonephritis.

The pituitary and adrenal function state is important in the regulation of water and salt metabolism in the body. Undoubtedly part of pituitary-adrenal system in the pathogenesis of edema. In particular, we establish the leading role of the pituitary antidiuretic hormone (ADH) in the regulation of facultative water reabsorption in the distal renal tubules. There is no doubt, and the value of adrenal hormones - mineralocorticoids aldosterone in the regulation of sodium ions exchange. Increased secretion of adrenal hormones is blocking all avenues of sodium excretion from the body (through sweat, saliva, through the gastrointestinal tract, in urine). Aldosterone plays especially great role in sodium reabsorption in renal tubules. Following the reabsorption of sodium and increased reabsorption of water, can lead to the formation of edema, including renal one.

In the mechanism of the edema syndrome in renal disease both renal and extrarenal factors are involved. The most important factors in edema developing are: improvement of vascular and capillary permeability, decreased oncotic blood pressure due to hypoproteinemia and mainly hypoalbuminemia, increased blood concentrations of aldosterone and ADH, the delay in the body of sodium ions with the increase of its content in the tissues, increased hydrophilicity of tissues, as well as reduction in glomerular filtration and increased tubular reabsorption of water by the kidneys. Importance of each factor individually in the pathogenesis of this complex phenomenon is estimated differently. Thus, a pivotal role in occurrence of nephritic edema play overall vascular and capillary permeability, and the origin of nephrotic edema (with nephrotic syndrome) is mainly due to low oncotic blood pressure due to hypo-and dysproteinemia.

Therefore, at various kidney diseases pathogenesis of edema has some peculiarities. The view of the essential role of increased vascular and capillary permeability in the genesis of nephritic edema is shared by many researchers. Increased vascular permeability is associated with fast rise and ubiquity of edema in nephritis, the higher the protein content of edema fluid in these patients compared with the hungry or cardiac edema, hypoproteinemia and hypoalbuminemia development with a drop of blood oncotic pressure as a result of withdrawal from the bloodstream into tissue fine protein.

In the mechanisms of disturbances of vascular permeability in general and in renal disease in particular (especially during acute exacerbation and chronic glomerulonephritis) an important role play changes in the normal state of the enzyme system of hyaluronic acid – hyaluronidase. Hyaluronic acid belongs to a group of mucopolysaccharides and is found in all organs and tissues. This is one of the most important constituents of the intercellular (cementing or gluing) of the substance of the walls of blood vessels and especially the blood capillaries, as well as the basic substance of connective tissue.

Hyaluronidase ("permeability factor") is an enzyme of microbial or tissue origin, have specific properties – to cause depolymerization and hydrolysis of hyaluronic acid. It contains in the serum of healthy human where its activity is 0-2,5 hyaluronidase units (city units). In some diseases its activity in serum is increased to 5-25 units., For example in patients with acute and exacerbation of chronic glomerulonephritis with nephrotic syndrome. Increased activity of this enzyme causes the depolymerization of hyaluronic acid, which is part of the intercellular substance walls of blood vessels and capillaries and is accompanied by an increase in the size of pores in the vascular and capillary walls, through which not only of water and ions (including sodium ions), but also fine fractions of protein (albumin) permeate in the tissue. In severe cases this can lead to hypoalbuminemia and a decrease in oncotic pressure of blood – one of the factors contributing to the development of edema.

Participation of hyaluronidase in the pathogenesis of renal edema confirmed by clinical observations, the signal increased diuresis and toe swelling (sometimes quite resistant to diuretic) in glomerulonephritis under the influence of heparin therapy. Apparently, heparin, as a potent inhibitor of hyaluronidase, inhibits the activity of this enzyme in serum and tissues, helps to reduce the vascular

permeability and hydrophilicity of the fabrics, the release of the tissue sodium ions and excretion of the latter from the body, and after them, and water.

In the genesis of renal edema important role play hypo-and dysproteinemia, often developing in kidney disease, and accompanied by a fall in oncotic (colloid osmotic) pressure of the blood below normal levels. Edema appears at lower levels of total protein in serum 50 g/l or less. Since the level of blood oncotic pressure is largely dependent on the concentration of albumin in it, then drop of it occurs mainly due to hypoalbuminaemia. Hypoproteinemia, hypoalbuminemia and reduced oncotic pressure of blood (up to 20 and even up to 15 mmHg) favor the formation of edema, when developing nephrotic syndrome. As a result, decreases the ability of colloids to keep blood in the bloodstream water, which in excessive amounts (more than it enters the blood vessels) goes into the tissue and accumulates in them, leads to fluid retention with the formation of latent or overt edema.

Sodium ions play a great role in the genesis of edema in general and in particular renal origin, because of their ability to hold water and increase the hydrophilicity of tissue. It was found that the concentration of sodium chloride in the edema fluid obtained from subcutaneous fat, increased substantially. In connection with this tissue osmotic pressure increases significantly, contributing to maintenance of water from the bloodstream into the tissue and retain it there. The mechanism of delayed sodium ion in the tissues is not entirely clear.

It can be assumed that the retention of positively charged sodium ions into the tissues occurs as a result of its connection with the molecules of hyaluronic acid carrying a negative electric charge, which may increase due to depolymerisation of hyaluronic acid under the influence of hyaluronidase. This explains not only retention of sodium in tissues, but also increased hydrophilicity of tissues and their ability to retain water in nephritic and nephrotic edemas.

Among other pathogenic factors of the edema syndrome in kidney diseases a considerable importance has increased secretion of cortical layer cells of the adrenal aldosterone and pituitary gland – the antidiuretic hormone. As a result, the concentration of these hormones in the blood increases significantly. Aldosterone is known to possess the ability to block all avenues of sodium excretion from the body. Delay in same sodium in the body and the accumulation of excessive amounts of it in the tissues are one of the reasons for increasing hydrophilicity of the latter, the accumulation of water in them and, consequently, formation of edema.

Strengthening the secretion of pituitary ADH and increase in the concentration of this hormone in the blood leads to increased water reabsorption in the distal renal tubules and collecting tubules and its accumulation in tissues. It was noted that during the growth of edema the activity of ADH is increased, whereas during the descent of edema and after its disappearance, it returns to normal ranges.

The above-mentioned pathogenetic factors edema attributed to extrarenal. However, in the pathogenesis of edema syndrome in renal disease an important role play kidneys by themselves, ie renal factor. It is associated with a decrease in filtration in the renal glomeruli and increased reabsorption of water and sodium in the tubules.

There is no doubt that the occurrence of nephritic edema involves all or most of these (and possibly other yet unknown) factors, but so far there is no consensus about which of them has the lead, and a minor role. With regard to the pathogenesis of nephrotic edema, there is unanimous opinion of most researchers that the main cause of them is a violation of the protein composition of blood with the development of hypoproteinemia, and hypoalbuminemia. As a result of blood plasma oncotic pressure decreasing and hydrostatic pressure dominates. Both of these factors, as well as increased vascular permeability contribute to the exit of water, sodium and protein from the bloodstream into the tissues, increase in their osmotic pressure and hydrophilic fabrics. Result of acute withdrawal of water from the bloodstream decreases blood volume, hypovolemia develops. This leads to volume receptors irritation and increased secretion of pituitary ADH and aldosterone adrenal glands, which is accompanied by increased reabsorption of water and sodium in renal tubules and lead to even greater rise of edema.