# **Odessa National Medical University**

Department of Surgery #4 with the course of oncology

LECTURE " Shock. Shock therapy "

The lecture discussed at the methodical conference of department

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Actuality of the topic: The intransigence of mechanical, especially road traffic injuries, gunshot and burn injuries caused by spontaneous disasters, which are characterized by a multiplicity of damaged organs, causes a high frequency of severe forms of traumatic shock. Along with trauma in the development of traumatic shock, additional causes such as bleeding (external and internal), over-cooling or overheating, unsatisfactory state of the organism of the patient before an injury (dehydration and hypoproteinemia, hypovitamins, insufficiency of the function of endocrine Glands, in the frequency of the cortical substance of the adrenal glands, thyroid, pancreatic - diabetes mellitus, oncological and other diseases, children or the elderly, fear and depressed emotional formation, obesity, istochenie). Reduces the risk of developing shock, a good physical condition, drug-induced inhibition of the nervous system (narcotic state, including a small degree of intoxication) or chemical deactivation (denervation), the reception of a zone that is injured (for example, by local anesthesia), etc.

Shock is a serious disruption of the body's vital activity due to acute bleeding disorder (with a sharp decrease in blood supply, hypoperfusion of the tissue) under the action of exogenous or endogenous lesions. For a long time, the term "shock" was used to describe the severe form of the course of a mechanical trauma; over time, this term was used to refer to the general reaction of an organism to damage, giving the main importance to the pathogenesis of a given composition of the nervous system. At present, "shock" is a collective concept, while in spite of the general mechanisms of formation of geodynamic disturbances, the clinical picture of therapeutic measures, different forms of shock differ substantially. The material that is being lectured is important in the professional training of a specialist.

2. Objectives of the lecture:

- training

Know:

- Types of shock due to occurrence;

- Types of shock by type of circulatory disorders

• To master the etiology of the onset of shock.

• Know the pathogenesis of shock.

• Know the phases of shock.

• Know the methods of diagnosing the severity of shock.

• To master complex shock therapy depending on the severity of the condition of the victim and the severity of the injury.

• Know the complications of shock.

- Educational:

The material of the lecture is aimed at the formation in students of logical and clinical professional thinking, the responsibility of the doctor for the condition of the sick person. Issues of medical ethics and deontology are covered.

SHOCK is an acute critical condition of the organism with a progressive insufficiency of the life support system caused by acute circulatory insufficiency, acute respiratory failure, microcirculation disturbance and tissue hypoxia expressed in violation of all physiological systems. "Shock" in English - shock, shock, shock. The term was introduced by a scientist and physician of the army of Louis XV Erran (XVIII). Shock is a polyethological disease. Depending on the cause, types of shock are distinguished:

I. Traumatic shock:

1. As a result of a mechanical injury:

· Wounds

· Fractures of bones

· Tissue compression (crash syndrome)

2. Burn injury:

· Thermal

· Chemical

3. Cold shock.

4. Electric shock.

5. Radiation.

II. Hemorrhagic, or hypovolemic, shock.

1. Acute blood loss - bleeding.

2. Acute disturbance of the water balance - dehydration of the body (sludge syndrome).

III. Septic (bacterial-toxic) shock.

Common purulent processes caused by gram "+" or gram "-" microflora. Bacteremia, toxemia.

IV. Anaphylactic shock.

V. Cardiogenic shock.

1. Myocardial infarction.

2. Acute heart failure.

VI. Blood transfusion shock.

1. Inconsistency of blood groups according to the ABO system, Rh factor.

Depending on the speed of development of shock phenomena:

Primary shock - at the time of injury or immediately after it.

Secondary shock - a few hours after the injury.

Factors predisposing to the development of shock:

Preceded or developed at the time of exposure to shock factors, reduce the overall resistance of the organism, contribute to the development of shock and determine its severity.

1. Chronic debilitating diseases - beriberi, tuberculosis, anemia.

2. Subcooling.

3. Overheating.

4. Fasting.

5. Blood loss.

6. Nervous shocks.

7. Ionizing radiation.

8. Insufficient transport immobilization and insufficient anesthesia during immobilization and transportation.

9. Surgery for extensive injuries, especially with gunshot wounds.

Despite various causes and some features of pathogenesis, the main factor in the development of shock is vasodilation and, as a result, an increase in the capacity of the vascular bed, hypovolemia is a decrease in the volume of circulating blood (BCC) due to various factors: blood loss, redistribution of fluid between the vascular bed and tissues or inadequate blood volume increasing capacity of the vascular bed as a result of vasodilation. The resulting discrepancy between BCC and the capacity of the vascular bed leads to a decrease in the minute volume of the heart's blood and microcirculation disorders.

The main pathophysiological process, caused by a violation of microcirculation, develops at the cellular level. Microcirculation disorders that unite the system of arterioles-capillaries-venules, lead to serious changes in the body, since it is here that the basic function of the circulation of blood occurs-the metabolism between the cell and blood. Capillaries are the direct place of this exchange, and capillary blood flow in turn depends on the level of arterial pressure, the tone of the arterioles and the viscosity of the blood. Slowing blood flow in the capillaries leads to aggregation of the formed elements, stagnation of blood in the capillaries, increased intracapillary pressure and the transition of plasma from the capillaries to the interstitial fluid. There is a thickening of the blood, which along with the aggregation of red blood cells and platelets leads to an increase in its viscosity and intracapillary coagulation with the formation of microthrombi, and consequently the capillary blood flow completely stops. Violation of microcirculation threatens to disrupt the function of cells and their death.

A peculiarity of the pathogenesis of septic shock is that the violation of blood circulation under the influence of bacterial toxins leads to the discovery of arteriovenous shunts and blood bypasses the capillary bed, rushing from the arterioles to the venules. Nutrition of cells is disrupted by reducing the capillary blood flow and the action of bacterial toxins directly on the cell, reducing the supply of cells with oxygen.

In case of anaphylactic shock, under the influence of histamine and other biologically active substances, capillaries and veins lose their tone, the peripheral vascular bed is dilated, its capacity increases, which leads to a redistribution of blood-congestion in the capillaries and veins, causing disruption of the heart. The existing BCC does not correspond to the capacity of the vascular bed, the minute volume of the heart decreases. Stagnation of blood in the microcirculatory bed causes a metabolic disorder between the cell and blood at the level of the capillary bed.

The disorder of microcirculation, regardless of the mechanism of its occurrence, leads to cell hypoxia and the disturbance of oxidation-reduction processes in it. In tissues, anaerobic processes begin to predominate over aerobic processes, metabolic acidosis develops. Accumulation of acidic metabolic products, primarily lactic acid, increases acidosis.

In the development of cardiogenic shock, the starting pathogenetic moment is a decrease in the productive function of the heart, followed by a violation of microcirculation.

The main initial pathogenetic factors determining the development of shock are:

1. decrease in the volume of circulating blood - hemorrhagic, hypovolemic, shock;

2. vasodilation, increased capacity of the vascular bed, redistribution of blood - anaphylactic, septic, shock;

3. violation of the productive function of the heart - cardiogenic shock.

Inadequate blood circulation at the level of capillaries during shock leads to changes in metabolism in all organs and systems, which is manifested by a violation of the function of the heart, lungs, liver, kidneys, nervous system. The degree of inadequate organ function depends on the severity of the shock, and this determines its outcome.

The developed circulatory disturbance, primarily the microcirculation disorder, leads to liver ischemia and impairment of its functions, which aggravates hypoxia in the severe stages of shock. Violated detoxification, protein-forming. glycogen-forming and other liver functions. Disorder of the main, regional blood flow, disturbance of microcirculation in the kidneys causes a violation of both filtration and concentration function of the kidneys with the development of oliguria, down to anuria. This leads to the accumulation of nitrogenous slags in the body - urea, creatinine and other toxic metabolic products.

Violation of microcirculation, hypoxia cause a violation of the function of the adrenal cortex and a decrease in the synthesis of corticosteroids (glucocorticoids, mineralocorticoids, androgen hormones), which aggravates the disorder of blood circulation and metabolism.

Disturbance of blood circulation in the lungs causes violation of external respiration, decreased alveolar exchange, shunting of blood, microthrombosis, resulting in the development of respiratory failure, aggravating hypoxia of tissues.

After (often during) stop bleeding, to address the need for, ways and volume of replenishment of blood loss you need to establish the amount of lost blood. Absolute figures can give incorrect information (100 ml blood loss in a one-year-old child is comparable to a loss of a liter of blood by an adult), so it is necessary to know what percentage of BCC (circulating blood volume), blood loss is in this patient. Tentatively, this can be done using the Algover-Burri index. The index is determined by dividing the pulse rate by the amount of systolic pressure.

THE IMPORTANCE OF THE ALGOVER-BURRY INDEX AT A DIFFERENT BODY LOOP

|  |  |
| --- | --- |
| index | Volume of hemorrage  ( % of Total volume) |
| 0,8 or less | 10 |
| 0,9-1,2 | 20 |
| 1,3-1,4 | 30 |

In cases of acute blood loss, the initial (due) bcc is calculated by multiplying the "ideal mass" by 85 ml / kg (if a man is examined) or 63 ml / kg (if a woman is examined).

"Ideal mass" - the proper weight of this person is calculated according to the Lorentz formula M = P - (100- (p-150) / 4), where P - human growth, M - ideal mass. This calculation allows to avoid mistakes in obese people, when recalculating for their weight, bcc will be overestimated, since subcutaneous fat contains a small amount of blood. To determine the amount of due BCC, you can use a different, less accurate method. First, the "ideal body mass" is determined, and then the BCC is calculated, based on the fact that it is 8-12 percent of the body weight (in men it is more than in women). To calculate the percentage of loss of BCC and thereby determine the severity of blood loss, it is necessary to establish the amount of blood loss. In a number of cases (bleeding in the body cavity), this can be done relatively simply. The cavities are punctured or opened, the blood is evacuated by an electric pump and measured.

If the patient had external bleeding, it is extremely difficult to make anamnestic assessment of the amount of blood loss. In such cases, it is possible to judge quite fully the degree of hemorrhage by the numbers of erythrocytes, hemoglobin, haematocrit

|  |  |  |  |
| --- | --- | --- | --- |
| Tests | Hemorrage level | | |
|  | light | moderate | sewere |
| Volume deficite | До 20% | 20-30% | 30% andmore |
| RBC | 4,4х10.12/l | 3,5х10.12/l | 2,5х10.12/land more |
| Hb | More 100 g/l | 85-100 g/l | less 85 g/l |
| Hct | More 30% | 25-30% | less 25% |

These indicators allow us to retrospectively estimate the blood loss that occurred, because the changes in them are related to the hydromic phase of compensation for acute anemia and they come from 2-3 hours from the moment of hemorrhage. The complete picture (severity) of hemorrhageis manifested in these indicators by the end of one and a half to two days.

MEASUREMENT OF CENTRAL VENOUS PRESSURE (CVP).

           The level of central venous pressure (CVP) assesses the perfusion ability of the heart and the volume of circulating blood, and monitoring of the infusion therapy is carried out.

Equipment

       Waldman's phlebotonometer consists of a tripod with a linear scale, moving with a screw handle. In the center of the scale, a glass manometric tube is fastened to the lower end of which a rubber tube is connected, connected to a three-way valve. To the second exit of this tap is attached a rubber tube, going to a glass tank with a capacity of 100 ml, reinforced in a special nest on a tripod. On the third exit, a rubber tube is put on to connect with the patient's vein. An isotonic solution of sodium chloride or distilled water is poured into the reservoir, which, by switching the three-way tap, fills the whole system of tubes. The level of the solution in the manometric tube is set on the zero scale line.

       The reservoir, rubber tubes, three-way valve, dropper, manometer tube should be sterile.

       CVP - pressure in the upper or inferior vena cava within the chest. To measure it, a catheter is inserted into the upper vena cava (through the internal jugular, subclavian or brachial vein) or the lower vena cava (through the femoral or popliteal vein) and connects it to the Waldman phlebotometer with a water manometer connected via a three-way valve and an infusion system. The device is placed next to the patient. The zero pressure of the phlebotometer scale is set at the level of the right atrium with the help of the level and the screw of the tripod. To do this, one end of the level is strengthened by the lower leg holding it, and the other is brought to the projection of the right atrium of the patient - the point of intersection of the III intercostal or IV rib with the mid-axillary line, or 5 cm below the angle formed by the connection between the handle and the sternum.

       After this, the apparatus is attached to the catheter inserted into the vein. By tapping the tank with liquid, the pressure in the vein displaces the blood into the system, which in turn displaces the solution. The latter rises through the glass tube to a value equal to the venous pressure.

       The normal value of the CVP is between 7-10 cm of water. Art. Slightly noticeable fluctuation in the rhythm of breathing indicates its normal functioning. A high level of CVP with large swings in the oscillations indicates a too deep insertion of the catheter when it reaches the cavity of the right ventricle - it must be tightened. Low CVP (0-5 cm H2O) indicates hypovolemia and effective heart function, it is necessary to replenish the blood volume. The critical value of CVP is a level of 1.5-2 cm of water. Art. Increase CVP beyond 10 cm of water. Art. is regarded as a sign of a likely heart failure.

Possible complications

       Obturation of the needle, catheter, rubber tube with a blood clot. This complication requires the replacement of thrombosed parts of the device.

Hemorrhagic shock develops with acute blood loss over 10 percent of BCC. In clinical practice, it is observed in a "pure form" with suicide attempts (vein dissection), ectopic pregnancy, interrupted tube rupture, spontaneous rupture of the spleen, ulcer bleeding, etc. In most cases, the pathogenesis of shock depends not only on the volume and rate of hemorrhage, but also on the mechanism of its occurrence (the volume and nature of the injury).

Pathogenesis. Acute blood loss, decreased bcc, venous return and cardiac output lead to the activation of the sympathetic-adrenal system, which leads to spasm of blood vessels, arterioles and precapillary sphincters in various organs, including the brain and heart. There is a redistribution of blood in the vascular bed, autogemodulation (fluid transfer to the vascular bed) against the background of a decrease in hydrostatic pressure. The cardiac output continues to decrease, there arises a persistent spasm of arterioles, the rheological properties of the blood change (the aggregation of erythrocytes "slaj" - a phenomenon).

Subsequently, peripheral vascular spasm causes the development of microcirculation disorders and leads to irreversible shock, which is divided into the following phases:

- phase of vasoconstriction with decreased blood flow in capillaries

- the phase of vasodilation with the expansion of the vascular space and a decrease in blood flow in the capillaries;

- the phase of disseminated intravascular coagulation (ICE);

- phase of irreversible shock.

In response to ICE, the fibrinolytic system is activated, lysed clots and blood flow is disturbed.

Clinic. It is determined by the mechanisms leading to deficiency of BCC, changes in blood CBC and electrolyte balance, peripheral circulation disorders and DIC syndrome.

Symptomatic complex of clinical signs includes: weakness, dizziness, thirst, nausea, dry mouth, darkening in the eyes, pale skin, cold sticky sweat, sharpening of facial features, tachycardia and weak pulse filling decrease in blood pressure, dyspnea, cyanosis.

During the hemorrhagic shock, there are 3 stages.

Stage 1 - compensated, reversible shock occurs when blood loss is 15-25% BCC (up to 1300 ml of blood). Arterial pressure in this case is reduced slightly, there is a moderate tachycardia.

Stage II - decompensated, reversible shock is accompanied by blood loss in 25-45% BCC (1300-1800 blood), there is a decrease in blood pressure (systolic below 100 mm Hg), tachycardia to 140 per minute.

Ill stage - irreversible shock occurs with acute blood loss more than 50% BCC (2000-2500 ml of blood), blood pressure below 60 mm. gt; Art. or is not determined, the pulse is more often than 150 beats per minute.

With compensated shock, the pallor of the skin, cold sweat, small and frequent heart rate, blood pressure within normal limits or reduced slightly, palate, and decreased urination. With decompensated reversible shock, the skin and mucous membranes are cyanotic, the patient is braked, the pulse is small, frequent, the arterial and central venous pressure decreases, oliguria develops, the Algover index is increased, and myocardial infringement is noted on the ECG. With an irreversible shock, consciousness is absent, blood pressure is not determined, the skin of a marbled species, anuria is noted - the termination of urination. Algover's index is high. To assess the severity of hemorrhagic shock, it is important to determine the BCC, the amount of blood loss.

Treatment of hemorrhagic shock involves the stopping of bleeding, the use of infusion therapy to restore BCC, the use of vasodilators.

Traumatic shock.

Erectile phase.

The erectile stage is characterized by:

1. Vast excitement of speech and motor.

2. Pale skin.

3. Frequent and deep breathing.

4. Tachycardia.

Pathogenesis of the erectile stage: in response to severe irritation (pain), the hypothalamic-pituitary-adrenal, sympathetic-adrenal, renin-angiotensin-aldosterone systems are activated.

In the blood, the concentration of vasoconstrictors rises: adrenaline, angiotensin II, glucocorticoids, vasopressin.

In the zones with α-adrenergic receptors, there is a vasospasm. Peripheral resistance of arterioles (resistant vessels) increases. In zones with β-adrenoreceptance, vasoconstriction does not occur: heart, lungs, brain. Those. there is a centralization of blood circulation during blood loss maintenance of blood pressure within the limits of the norm.

Torpid phase.

It is accompanied by general retardation, low blood pressure, a threadlike pulse.

Pathogenesis of the torpid phase: a major role in pathogenesis is played by BAS with a vasodilator capacity, which accumulate in the zones with α-adrenergic receptor where ischemia is observed, due to insufficient reception with blood O2.

Due to the lack of O2, the cells switch to anaerobic regimen. Lactic acid, carbon dioxide, H + ions accumulate. Metabolic acidosis develops, which leads to activation of LPO, enzymes of limited proteolysis released from lysosomes (BAS - callidinum, bradykinin).

Deficiency of ATP, due to a shift in pH to the acid side, because the denaturation of tissue respiration enzymes occurs, further increases acidosis, forming a vicious circle.

Acidosis leads to the degranulation of mast cells, stand out: serotonin, histamine. Vasodilators begin to predominate. As a result, the blood circulation is decentralized, the blood pressure drops, the resistive vessels expand.

1. Slows the blood flow in the capillaries.

2. The permeability of capillaries increases.

3. The yield of the liquid part of the blood and protein in the tissue.

4. Blood thickening.

5. Aggregation of uniform elements.

6. Thrombosis (disrupted vascular-platelet homeostasis, as endothelium is damaged, hypercatecholemia).

The severity of clinical manifestations of the torpid phase of shock is distinguished by 4 degrees:

I .. Consciousness is preserved, the patient is contact, slightly inhibited. ADSist up to 90 mm Hg, Algover - 0,8.

II ..It's inhibited. Skin covers pale, cold sticky sweat. ADSyst 90-70 mm.rt.st. Algover - 0,9-1,1.

III. Adynamic, inhibited, does not respond to pain. Skin covers pale, cold with a cyanotic shade. Urination stops. ADSyst 70-50 mm. Hg. Algover - 1,5 and above.

IV. ADSist below 50 ml. Hg. pulse frequent, weak filling, breathing frequent, superficial. Preagonal state.

Terminal stage.

Expanded arterioles do not react to cotecholamines, hypovolemia dramatically decreases blood pressure, which leads to brain hypoxia and agonal state development.

Principles of dealing with shock.

The most important is the early analgesia of the victim, transport immobilization.

Victims in a state of traumatic shock are treated with a complex application of a number of means. Treatment is designed to eliminate the disorders of vital body functions caused by shock. The most important elements of the complex method of treatment are as follows.

1. Moderate warming of the affected, while avoiding dangerous overheating. In the absence of a warm room, especially when evacuating, warming is achieved by wrapping in blankets and overlaying heaters. Damp clothes, underwear, shoes must be removed. Warming in anti-shock chambers is achieved due to a sufficiently high air temperature in the room (24-25 degrees). Contact heat in an anti-shock chamber should not be used. Warming is promoted by strong hot tea, small doses of alcohol, hot food. However, with injuries of the abdomen, as well as in the presence of vomiting (regardless of the nature of the lesion), the victims should not be given food or drink. In the case of shock associated with combined radiation damage, one should not apply more than 50 g of alcohol 40% at a time, taking intravenous fluids as a component of anti-shock liquids.

2. Attachment of the affected position in Trendelenburg (raise the end of the stretcher, remove the pillow from under the head).

3. The introduction of analgesics (promedol, omnopon, etc.) under the skin or better intravenously. The use of analgesics is contraindicated in cases of disturbances in external respiration or a decrease in blood pressure to a critical level and below, as well as in cases of craniocerebral trauma.

4. Production of Novocain blockades according to Vishnevsky. The blockade removes strong irritations, and acts as a weak stimulus, which facilitates the mobilization of compensatory mechanisms in shock. In case of breasts, one-or two-sided vagosympathetic blockade is used, with abdominal injuries - bilateral paranephalic blockade, with damage to the limb, a blockade.

5. Intravenous and intra-arterial blood transfusions, transfusions of blood plasma, albumin, infusion of anti-shock fluids. Depending on the degree of hemorrhage, the depth of shock and the availability of reserves of canned blood, 500-1000 ml or more of blood is poured. With a shock of grade 4, blood is initially injected into the artery (250-500 ml), and then transferred to intravenous drip. At a shock of 3 degrees blood or polyglucinum is poured in the beginning by a jet method, and after rising of a BP it is drip. If the level of blood pressure for a shock of grade 3 is very low, it is advisable to begin immediately intra-arterial blood injection. Along with hemotransfusions in shock, polyglyukin infusions are very important. The latter is administered at doses of 400-1500 ml, depending on the severity of the condition of the affected. With shock 3-4

6. degree, if there is no blood, a part of polyglucin is administered intraarterially in the same doses as blood. In the case of shock not accompanied by large blood loss, especially with burn shock, often limited to the introduction of polyglucin in combination with albumin or plasma. Along with this poured and reopoliglyukin. The introduction of crystalloid anti-shock solutions gives a good result only with shock of 1 degree.

7. The introduction of cardiovascular drugs (strophanthin, korglukon, in a 5% solution of glucose). In more severe cases, adrenomimetic drugs (ephedrine, norepinephrine, mezaton) and glucocorticoids (hydrocortisone and especially prednisolone) are shown. It should be emphasized that these drugs should be used in combination with blood transfusions or infusion of colloidal substitutes.

8. To combat oxygen deficiency prescribe inhalation of urine, injection of cititon or lobeline. In severe breathing disorders, tracheal intubation is applied or tracheostomy is applied and ventilator is used.

9. To combat metabolic disturbances, the administration of vitamins, especially ascorbic acid and vitamin B1, calcium chloride (10 ml of a 10% solution in a vein) is indicated.

10. Along with anti-shock therapy, tetanus antitetanus and toxoid, antibiotics are injected.

Each additional trauma aggravates the severity of the shock. Proceeding from this,it is necessary to abstain from surgical interventions before removal of victims from a shock condition. Life indications for surgery include:

• stop of continuing internal bleeding

• asphyxia

• anaerobic infection

• open pneumothorax

Surgical interventions in the presence of shock are performed simultaneously with ongoing anti-shock therapy.

In patients with severe forms of shock, the state of agony and clinical death may develop, which are considered as forms of terminal states.

Methods of treatment of burns and burn disease. Burn shock.

Methods for treating burned people are fundamentally different from the generally accepted ones even 10-15 years ago. Modern tactics of treatment for burns is developed largely due to success in studying the pathogenesis of burn disease and its complications. Wide opportunities were opened by the research of microbiologists and immunologists aimed at revealing mechanisms of interaction between the organism of the victim from burns and infection. The most important are the studies of metabolism with extensive burns. A special place is occupied by the study of the course of the wound process, depending on the severity of the burn injury, the use of various, including alternative therapies.

A significant improvement in the results of treatment of burned was made possible by the development and implementation of active surgical tactics in clinical practice. Its principal basis is an early necrectomy followed by autodermoplasty of burn wounds in order to quickly restore the integrity of the entire skin. In addition, the inclusion of intensive treatment of burned abacterial methods in the complex allows creating optimal conditions for healing burn wounds and maintaining homeostasis.

Classification

The choice of treatment tactics and methods depends on the severity of the injury, which, in the case of burns, is primarily determined by the depth and area of ​​thermal damage. In our country, a 4-degree classification of the depth of burns is used.

Hyperemia and small swelling of the skin in the burn area are clinical signs of the 1st degree burn. With burns of the second degree, in addition, bubbles are formed, filled with serous contents. With burns of the I degree, only the epidermis is affected, with the second degree burns - epidermis and papillary dermis. These burns heal independently, due to the preserved epithelial cells and are called superficial.

With IIIA degree burns, the mesh layer of the dermis is included in the thermal damage zone, but many hair follicles, gland bags, sweat glands - skin derivatives, due to which the epithelization of burn wounds occur, remain intact. However, this does not always happen, often the mosaic of skin lesions and, consequently, of the majority of its derivatives, hinders the self-healing of wounds. Especially with extensive burns, when due to microcirculatory disorders and infection, burn wounds can "deepen", and as a result, autodermoplasty is required. Therefore, burns IIIA degree correctly called not superficial, but borderline. These burns can be diagnosed by a thin, sufficiently mobile scab or large blisters filled with serous contents, but already with intense yellow staining.

With deep burns, the skin affects its entire thickness (grade IIIB) or deeper tissues - subcutaneous fat, fascia, bones (grade IV). Burns of grade IIIB are often accompanied by the formation of a brownish shade, welded to the underlying tissues, less often - the formation of large blisters filled with hemorrhagic contents. With burns of the IV degree, a dense dark brown or black scab is always observed, closely adhering to the underlying tissues. Deep burns IIIB-IV degree always require surgical treatment.

The so-called nines rule is widely used to determine the area of ​​burns. According to this rule, the entire surface of the skin of an adult person is conditionally divided into eleven "nine": the head and neck - 9%, the upper limbs - 9% each, the lower limbs - 18% (2 times 9% each), the back surface torso - 18%, anterior surface of the trunk - 18% Remaining to 100% one percent of the body surface is in the perineal region. (In order to estimate the area of ​​burns in children, the rule of nines in the form presented is not applied).

At a primary examination of the victim, it may not be easy for a specialist to determine the depth of burns. Therefore, the clarification of the circumstances of the trauma becomes especially valuable. First of all, this concerns the etiologic factor of burns.

Practically determining the etiology of burns, the doctor indirectly evaluates the temperature of the affected agent and the exposure of his effect on the tissue of the victim. So, burns by flames, especially those received by a dressed person, usually lead to deep burns. Immersion in boiling water, hot water also leads to burns of III-IV degree. On the contrary, scalding with boiling water (usually household trauma) causes superficial burns. Contact or electric burns are the cause of limited but deep burns, and chemical burns are I, II, III degree burns.

It must also be remembered that fires in an enclosed area, directed explosions, can be accompanied by burns of the respiratory tract and poisoning by combustion products. Thermal inhalation trauma often results in life-threatening complications associated with the development of bronchospasm, pulmonary edema, and early pneumonia in the first days after the burn.

pneumonia already in the first days after the burn.

Burn disease

If deep burns occupy more than 10-15 percent. the surface of the body, or the total area of ​​burns is more than 30 percent. body surface, the patient develops a burn disease.

Burn disease is a complex complex of interrelated pathogenetic reactions and their clinical manifestations, which is based on a stress response in response to thermal damage as the first and determining the state of the victim, depending on the severity of the injury, the timeliness of the beginning and usefulness of the treatment.

In the structure of the burn disease, there are three main clinical syndromes: burn shock, intoxication, infection.

The total burn area and the Frank index

|  |  |  |  |
| --- | --- | --- | --- |
| **Clinical form** | **Space, %** | **Frank Ind** | |
| **without** | **with** |
| light | 10-19 | 30-59 | 30-49 |
| moderate | 20-50 | 60-120 | 50-100 |
| sewere | more50 | more 120 | more 100 |

Burn shock is caused by neuro-reflex and neuro-endocrine reactions, the appearance of a large number of various inflammation factors that lead to disruption of the central and peripheral circulation, including widespread microcirculatory disorders, increased permeability of vascular and cell membranes and hypovolemia due to the release of plasma from the vascular bed with the development of interstitial edema, as well as plasma loss through burn wounds.

The manifestation of these functional and morphological disorders is the corresponding clinical picture of shock, the severity of which depends on the depth and area of ​​burns, the time elapsed after trauma, and the adequacy of the treatment. In this case, the adequacy of intensive therapy of burn shock should be understood as the timeliness of its onset, pathogenetic validity, as well as the necessary volume and rational composition of infusion-transfusion media administered intravenously.

The calculation of intravenous infusions in the period of burn shock is based on the formula adopted worldwide to define a rational scheme for intensive therapy of burned:

V = m x S x 2, where V is the amount (volume) of intravenously administered fluid in the 1st day of burn shock in ml; S - total burn area in percent, but not more than 50%; m is the body weight of the patient in kg.

At the same time 2/3 of this volume must be poured already in the first 8 hours after the injury. Crystalloids should be 2/3 - 1/2 of this volume, and colloid preparations - 1/3 - 1/2, depending on the severity of the shock. In addition, it is necessary to administer about 2 liters of a 5% solution of glucose.

On the second day, the volume of intravenous infusions is reduced by 2 times, on the third day - to 1/3 of the initially established volume.

To implement this rule, of course, it is necessary to catheterize the central vein, which should be performed by all burned in severe or extremely severe burn shock. For elderly patients, as a rule, intravenous injection of half the volume calculated for the affected middle-aged group is sufficient.

The second syndrome of burn disease - intoxication - is caused by the appearance in the body of a large number of toxic products of tissue, enterogenic and partially bacterial origin. It manifests itself after the removal of the victim with extensive and deep burns from the shock of psychoemotional disorders, a persistent increase in body temperature during the day, loss of appetite and development of signs of toxic damage to internal organs (toxic myocarditis, hepatitis, etc.). This period is called the period of acute burn toxemia. An effective method of treatment in the period of toxemia is active disintoxication therapy using the method of forced diuresis, and, in more severe cases, the use of plasmapheresis or hemosorption. The development of sound methods and the rational use of modern medicinal preparations have now made it possible to significantly improve the results of treatment of burns that were severely burnt in early periods of burn disease. On the contrary, late onset, insufficient volume or inadequate composition of infusion-transfusion therapy in severe or extremely severe burn shock can lead to prolonged hypovolemic spasm of peripheral microvessels followed by paralytic expansion of capillaries, pronounced disturbance of water-electrolyte and protein balance.

The third syndrome, or period, is burn septicotoxemia. It is associated with the development and progression of the infection. Diseases of metabolism started in the period of burn shock in acute burn toxemia are manifested, and, as one of the consequences of this, the lack of an immunological response to infection.

Infection significantly burdens the course of the burn disease. It determines and maintains intoxication, suppresses reparative processes in wounds, affects various organs, and in some cases generalization occurs - it is difficult to healable burn sepsis.

The main cause of infection in burned is a burn wound. On it, in the future, an infection of enteric origin, as well as hospital infection, is superimposed. In addition, the prolonged existence of burn wounds causes burn burnout, the development of which creates extremely unfavorable conditions for regeneration as a whole.

Anaphylactic shock.

At the heart of anaphylactic shock is the interaction in the body of the antigen and antibodies. This is a state of sharply increased sensitivity of the organism, which develops with the repeated introduction of foreign proteins and serums, medicines, with the bite of the hymenoptera. One of the most formidable and complex complications of drug allergy, ending in about 10-20% of cases is lethal. The rate of occurrence of anaphylactic shock from a few seconds or minutes to 2 hours from the beginning of contact with the allergen. In the development of anaphylactic reaction in patients with a high degree of sensitization, neither the dose nor the method of administration of an allergen plays a decisive role. However, a large dose of the drug increases the severity of the duration of the shock.

In surgical practice, anaphylactic shock develops with the use of protein blood substitutes, immune preparations, antibiotics, certain chemical antiseptics (iodine preparations), as well as other antigens that cause a reaction in patients suffering from allergic diseases (bronchial asthma, drug dermatitis, etc.).

Pathogenesis. The common and most significant sign of shock is an acute onset decrease in blood flow with violation of peripheral and then central circulation under the influence of histamine and other mediators, abundantly secreted by cells. The skin becomes cold, moist and cyanotic-pale, anxiety, impaired consciousness, shortness of breath, urination is disturbed.

There are following forms of anaphylactic shock:

1) cardiovascular form, which develops acute circulatory failure, manifested tachycardia, often with a violation of the rhythm of heartbeats, ventricular and atrial fibrillation, a decrease in blood pressure;

2) the respiratory form, accompanied by acute respiratory failure: dyspnea, cyanosis, stridoriznym, bubbling breath, wet wheezing in the lungs. This is due to a violation of the capillary circulation, swelling of the lung tissue, larynx, epiglottis;

3) cerebral form, caused by hypoxia, a violation of microcirculation and brain edema. It manifests itself as a disorder of consciousness, development of a coma, the appearance of focal symptoms of a violation of the central innervation.

Clinical picture. The degree of severity of anaphylactic shock depends on the rapidity of the development of vascular collapse and impaired brain function.

According to the severity of the course, there are 4 degrees of anaphylactic shock:

1 degree (light): duration of development - from several minutes to 2 hours, characterized by skin itching, flushing of skin and rash, the appearance of headache, dizziness, a sense of tidal head, sneezing, pershenia, rhinorrhea, hypotension, tachycardia, a feeling of heat, increasing weakness, unpleasant sensations in various areas of the body;

2 degree of moderate severity: the average severity of anaphylactic shock is characterized by the most extensive clinical picture: toxicermis, Quinck'sedema, conjunctivitis, stomatitis, circulatory disorders - palpitation, pain in the heart, arrhythmia, lowering blood pressure, severe weakness, dizziness, anxiety, excitement, a sense of fear of death, trembling, pallor, cold sticky sweat, hearing loss, ringing and noise in the head, fainting. Against this background, it is possible to develop an obstructive syndrome according to the type of attack of bronchial asthma with the manifestation of cyanosis, the presence of gastrointestinal (nausea and vomiting, bloating, swelling of the tongue, abdominal pain, diarrhea with admixture of blood in the stool, sharp abdominal pain) and kidney urge to urinate, polyuria) syndromes.

3 degree (severe): manifested by loss of consciousness, acute respiratory and cardiovascular insufficiency (dyspnea, cyanosis, stridorous respiration, small frequent pulse, sharp decrease in arterial pressure, high index of Algovera);

4 degree (extremely severe): collapse rapidly (pallor, cyanosis, filiform pulse, sharp decrease in blood pressure), coma (with loss of consciousness, involuntary defecation and urination), pupils dilated, their reaction to light is absent. With a subsequent drop in blood pressure, the pulse and blood pressure are not detected, the heart stops, and breathing stops.

Possible variants of anaphylactic shock with a predominant lesion are: a) skin integument with increasing skin itching, hyperemia, the appearance of common urticaria, edemaQuincke; b) the nervous system (cerebral variant) with the development of severe headache, the appearance of nausea, hyperesthesia, paresthesia, seizures with involuntary urination and defecation, loss of consciousness with clinical manifestations by the type of epilepsy; c) respiratory organs (asthmatic variant) with dominant suffocation and development of asphyxia due to changes in patency of the upper respiratory tract due to laryngeal edema and disruption of middle and small bronchial passages; d) heart (cardiogenic) with the development of a picture of acute myocarditis or myocardial infarction and other organs.

Treatment of anaphylactic shock is based on:

1. blocking the receipt of antigen-drug in the bloodstream;

2. neutralization of biologically active substances abundantly secreted and entering the bloodstream as a result of the antigen-antibody reaction;

3. restoration of pituitary-adrenal insufficiency;

4. deducing the patient from collapse;

5. removal of bronchospasm;

6. Elimination of asphyxiation phenomena;

7. Reducing the permeability of the vascular wall;

8. Effects on psychomotor agitation;

9. prevention of late complications from the cardiovascular system, kidneys, gastrointestinal tract, central nervous system.

Anaphylactic shock requires urgent help, since minutes and even seconds of delay and confusion may lead to the death of the patient. First of all, it is necessary to lay the patient, turn his head to the side and push the lower jaw to prevent the tongue and tongue asphyxiation (if the patient has dentures, they should be removed), warm heaters should be applied to the feet. It is necessary to apply a tourniquet above the injection site. Place the injection of the allergen with 0.1% solution of adrenaline (1-0.5 ml) and apply ice to it to prevent absorption of the allergen, and in case of taking it inside, make a gastric lavage. Administer 1 ml of a 0.1% solution of epinephrine, 2 ml of cordiamine, 2 ml of a 10% caffeine solution, 60 mg of prednisolone or 125 mg of hydrocortisone, if necessary, administer 0.5 ml of epinephrine with 20 ml of a 40% glucose solution. as adrenaline restores vascular tone and is a powerful antagonist of histamine release. In case of anaphylactic shock from the use of penicillin, one million intramuscular injection of 1 000 000 units of penicillinase per 2 ml of sodium chloride solution should be administered, and in case of shock caused by bicillin - for 1 000 000 units of penicillinase per day.

To neutralize biologically active substances, 1-2 ml of a 1% solution of suprastin or 2-3 ml of a 2.5% solution of pipolpene, or 1-2 ml of a 1% solution of dimedrol intramuscularly, 10-20 ml of a 10% solution of calcium chloride or calcium gluconate intravenously (pipolfencan not be administered to patients who have allergic diseases due to the use of aminazine due to the common antigenic properties of these drugs). When bronhospazme intravenously prescribed 2.4% solution of euphyllin with 10 ml of 40% glucose solution, with swelling of the larynx, which does not disappear, despite ongoing therapy, produce urgent tracheotomy. In cases of cardiovascular insufficiency and pulmonary edema, 0.5 ml of 0.05% solution of strophanthin with 10 ml of 40% glucose solution and 10 ml of a 2.4% solution of euphyllinshould be injected intravenously; constantly through the nasal catheter give moistened oxygen, to carry out inhalation of vapors of ethyl alcohol. With pulmonary edema, 1 ml of a 1% solution of furosemide should be added to the dropper, and 0.2% to 0.5 ml of a 5% solution of pentamine intravenously, under the control of AD. To maintain blood pressure, 1% mezatone solution is used from 0.5 to 1 ml subcutaneously or 0.1% noradrenaline solution from 1 to 2 ml in injections with 5% glucose solution. To eliminate metabolic acidosis, 4% sodium bicarbonate solution (150-200 ml) is intravenously dripped intravenously. In the future, the patient is continued to drip intravenously an isotonic solution of sodium chloride together with antihistamine (dimedrol, suprastin, diprazine, pipolfen) and glucocorticoid agents - hydrocortisone (125-500 mg) or prednisolone (60-120 mg), dexamethasone (4-20 mg) . With convulsions and strong excitation, intravenous droperidol up to 1 ml is recommended. In case of cardiac arrest, 1 ml of a 0.1% solution of adrenaline should be injected intracartically with a long needle, to perform closed cardiac and artificial respiration (mouth to mouth, mouth to nose, or with the help of apparatuses), etc. Patients with anaphylactic shock, are hospitalized in the intensive care unit. In order to prevent late complications, they should be treated for 10-14 days with prednisolone in a dose of 40 to 60 mg or in equivalent doses with other hormonal drugs.

For the prevention of anaphylactic shock, it is important to identify allergic diseases in the anamnesis, which should be taken into account when prescribing agents that can cause an allergic reaction. In case of a dysfunctional allergic anamnesis, tests on the sensitivity of the organism to the drugs used, for example antibiotics, antiseptics, iodine preparations before angiography, etc., are shown.

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