Traumatic illness. Traumatic shock. Polytrauma. Syndrome of long crushing, etiology, pathogenesis.

Speaker PhD Iurii Piven

Scientific and methodological substantiation of the topic

The term "polytrauma" is widespread and has taken root among medical workers of various specialties, especially for those who are engaged in the provision of urgent trauma care. The short word "polytrauma" is a symbol of anxiety, which characterizes both the complexity of the injury, and the severe general condition of the patient and the presence of shock, blood loss, etc.

At the same time, this is a call to provide emergency care to such patients at all stages: first aid, admission department, resuscitation service, operating room. This is especially important in the development and development of disaster medicine.

The frequency of polytrauma is 5-8% of all injuries of the musculoskeletal system and internal organs.

Learning objectives of the lecture

• To familiarize students with the clinical and radiological picture of polytrauma.

• Know the clinical and radiological picture of the disease. To master the methods of examination, to determine the indications, to know the methods of surgical treatment.

• Be able to make a differential diagnosis of the disease.

• To master methods of diagnosing traumatic illness.

• Assess the general condition of patients and choose treatment tactics depending on the patient's condition and complications. Determine the indications and methods of surgical treatment.

• Know the principles of medical, social and labor rehabilitation of patients with traumatic illness.

Goals of future personality development

• Convince internists in the practical meaning of this topic.

• During the presentation of lecture material, emphasize the contribution of domestic scientists in the treatment of polytrauma, traumatic shock.

• Educate the sense of professional responsibility, the general ethical qualities of future doctors.

• Promote healthy lifestyles, explain to students the dangers of smoking, stay in dusty rooms, etc.

• To form an idea about the need for preventive measures in relation to the development of the pathology of the musculoskeletal system.

The plan and organizational structure of the lecture

Means of activating students. Materials of methodical support Distribution of time

1. Preparatory stage

Determining the relevance of the topic, the learning objectives of the lecture, motivation See points 1 and 2.

2. Main Stage

The presentation of the lecture material according to the plan:

Thematic lecture

Means of clarity:

1. Multimedia presentation
2. Radiographs.
3. Thematic patients.
4. Issues, problem situations, tasks

1. Classification of polytrauma.

2. Anatomico-morphological features of traumatic disease.
3. The maintenance of medical care for victims with polytrauma at the prehospital stage and the basic principles of rendering assistance at the hospital stage.
4. Traumatic illness. Classification, clinical and roentgenologic diagnostics.
5. The main principles of general and local treatment.

Actuality

There are statements that there are three killers of the age: cardiovascular disease, tumors and car. If in the fight against the first two reasons medicine is progressing every year, then this cannot be said with regard to traffic injuries.

With the increase in the number of different vehicles, their speed, as well as the poor state of roads and the lack of high self-discipline of road users, the number of road injuries in the world is increasing.


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In addition, as the trauma progress increases, their consequences often lead to disability, which is not only a medical problem but also a social one.

Currently, the general and local processes that occur in any trauma are considered within the concept of traumatic illness.

The introduction of the concept of traumatic illness allows us to consider its clinical manifestations in each victim as a system of processes that develop sequentially. It also allows linking the relationship between general and local changes that occur in the post-traumatic period.

Traumatic disease is a set of general and local changes, pathological and adaptive reactions that occur in the body from the moment of injury until its release.

Traumatic disease, like any other disease, must be characterized, on its own, with the following criteria in mind:

reasons;

morphological substrate;

basic pathogenetic mechanisms;

speakers;

severity;

clinical forms and manifestations.

1. *The cause of this pathological condition is the effect of damage factors.*

2. *The morphological substrate of traumatic disease is the damage to organs and tissues that occur in excess of damaging action and vary in localization and nature.*

3. The pathogenesis of traumatic disease is based on a combination of various pathological processes, "damage responses" and "defense responses".

Adaptive reactions are aimed at ensuring the vital activity of the organism in extreme conditions with the subsequent restoration of impaired functions and structures.

4-5. Important criteria for assessing a traumatic illness are its severity and dynamics, which are strongly interrelated.

The dynamics of traumatic disease largely depends on its severity and is characterized by several periods, which are different by a set of pathological processes specific to each of them and characteristic clinical manifestations - syndrome-symptom complexes. (The pathological process is the local manifestation of the disease, which determine its specificity in a particular period).

In severity distinguish traumatic disease I, II, III degree.

Such gradation of traumatic illness helps to determine the nature of the course of the post-traumatic period, to set the terms and scope of possible surgical interventions, to solve the main tasks of treatment of a specific victim.

So, the victims with the most severe injuries will be dominated by problems associated with general treatment,

and in the case of the slightest injuries (grade I), on the contrary, with the local one.

6. The clinical manifestations and forms of traumatic disease are diverse and characterized by specific and nonspecific features. Specific features depend on the nature of the underlying damage.

From a clinical point of view, it is advisable to distinguish four periods of traumatic disease:

Periods of traumatic illness

- Acute
- Early manifestations of the effects of trauma
- Late manifestations of the effects of trauma
- Rehabilitation

Acute period

Its duration depends on the localization and severity of traumatic injuries, age, general human condition, the degree of homeostasis. In mild injuries, this period can be short or last for several hours, in severe - it can last for several days.

Acute period

Conditionally in this period there are three phases:

1) the phase of instability of vital functions (duration of several hours, the maoment of its end coincides with the completion of resuscitation);

2) the phase of relative stabilization of vital functions (the main phase of the first period);

3) phase of stable stabilization of vital functions (completion of the first period).

The total duration of the first period is up to 2 days.

The clinical picture is dominated by the general manifestations of major pathological processes, the nature of these for this period - traumatic shock, acute blood loss, traumatic toxoid goat, as well as processes associated with primary organ damage.

The second period

Period of detailed clinical picture of traumatic disease. It is determined by post-resuscitation, post-shock, postoperative changes. Its duration is 4-6 days. The clinical picture is quite bright, depends on the nature of the dominant lesion and is most often represented by such syndromes as acute heart failure, adult respiratory distress syndrome, DIC, endotoxicosis.

The second period

It is these syndromes and their associated complications during this period that directly threaten the life of the victim.

In the second period of traumatic disease, with multiple organ failure, it is especially important to keep in mind that those multiple disorders that occurred in the patient - are manifestations of a single pathological process.

Late manifestation of the effects of trauma

The third period - or the period of clinical recovery: is characterized by the completion of plastic remodeling, ie the formation of the scar and its adaptation. The duration of this stage is from several weeks to several months and even years. This stage is characterized by impaired function of different organs and systems, as well as complications.

The most common complications are:

1) purulent-inflammatory (out of the area of damage - pneumonia, pleurisy, tracheobronchitis, inflammatory diseases of the urinary system, sepsis, etc., in the area of damage - suppuration);

2) toxic (mental disorders, acute renal and hepatic impairment);

3) post-hypocirculatory and trophic disorders (pressure ulcers, thromboembolism, thrombosis, brain edema, pulmonary edema).

Rehabilitation period is the fourth period

It is characterized by physical and social, complete or incomplete recovery. The duration of this period can be many months, and sometimes years

As a result of trauma and suffering, patients are often physically impaired, depressed, and not yet adapted to physical and mental work. During this period, they still need medical and, especially, social rehabilitation.

Traumatic shock

Shock - the "gateway to death" from the injuries and injuries of millions of able-bodied people, is a typical evolutionary process that has a phase development and is observed in an acute period traumatic disease.

Pathogenesis of traumatic shock

According to modern views, the leading role in the pathogenesis of traumatic shock belongs to blood loss.

It belongs to the category of hypovolemic shock or shock with circulating blood volume deficiency.

Acute blood loss forms a disproportion between BCC and vascular volume.

acute blood loss

The severity of blood loss is determined by two main factors: the BCC deficit and the rate of bleeding. The main pathogenetic factor in blood loss is a decrease in BCC. Thus, the rapid loss of 30% of BCC causes (under other different conditions) more pronounced violations of the life of the body than moderate leakage of the same volume of blood.

Pathogenesis of traumatic shock

Trauma and acute blood loss stimulate the nervous and (to a greater extent) endocrine system. Stimulation of the sympathetic - adrenal system leads to the release of catecholamines (adrenaline, norepinephrine, dopamine) and to generalized arteriospasm. Vasoconstriction is not uniform.

Pathogenesis of traumatic shock

It covers the area of the circulatory system of the internal organs (lungs, liver, pancreas, intestines, kidneys), as well as the skin and muscular system.

Due to this, the shock in the stage of compensation to the heart and brain blood flows more than in normal conditions. Such a change in the circulation situation is called centralization of circulation.

Pathogenesis of traumatic shock

Centralization of blood circulation, when considered in the short term, is an appropriate adaptive response. Unless there is rapid normalization of BCC, vasoconstriction, which continues and is associated with a decrease in capillary blood flow - a "microcirculation crisis" - causes a decrease in the supply of oxygen and energy substrates to the tissues and the elimination of end products of intracellular metabolism.

Pathogenesis of traumatic shock

The development of local metabolic disorders in tissues leads to the development of metabolic acidosis.

As the shock progresses, the local hypoxic metabolic disorder leads to dilatation of the precapillary vessels, while the postcapillary vessels remain constricted. Therefore, blood rushes to the capillaries, but the outflow from them is difficult. Decentralization is taking place.

Pathogenesis of traumatic shock

В системі капілярів уповільнюється кровотік, кров накопичується і підвищується внутрішньокапілярний тиск. В наслідок цього: 1) плазма переходить в інтерстіцій; 2) в повільно текучій крові настає агрегація клітин крові (еритроцитів і тромбоцитів); 3) підвищується в‘язкість крові; 4) уповільнення кровотоку та загальна тенденція підвищення згортаємості при шоку призводять до спонтанного згортання крові в капілярах, утворюються капілярні мікротромби.

Pathogenesis of traumatic shock

There is a process of disseminated intravascular coagulation.

At some stage of the shock, the factor of toxemia is included and plays a significant role.

Insufficient tissue perfusion causes severe metabolic, biochemical and enzymatic cellular disorders, which are secondary pathogenetic factors (toxemia) that form a vicious circle and cause progressive worsening of the shock course if the necessary treatment is not applied in a timely manner.

Shock organs

Some organs are particularly sensitive to circulatory shock. Such organs are called shock. These include the lungs, kidneys, liver.

Changes in the lungs. The blood flow decreases. The absorption of oxygen by the lungs is reduced. Swelling of the interstitial lung tissue is noted.

In the lungs, there are foci of stagnation, necrosis, and sometimes heart attack.

Shock organs

Kidney shock is characterized by a sharp restriction of blood circulation, impaired filtration and concentration function, a decrease in the amount of urine that is excreted. The development of a shock kidney may be accompanied by oliguria, and in more severe - anuria.

Liver. In case of shock, necrosis of liver cells, decrease in synthesis and detoxification function. Dysfunction of the liver is judged by the increase in the level of liver enzymes.

Violation of acid-base balance.

In traumatic shock, acidosis develops. It causes impaired myocardial contractile function, persistent vasodilation, decreased renal excretory function, and impaired higher nervous activity.

Shock phases

The excitation phase comes directly with the trauma and is characterized by generalized CNS excitation, metabolic intoxication, increased activity of some endocrine glands. Characteristic of this phase is motor and linguistic arousal while maintaining consciousness.

Excitation phase

Victims do not complain of pain, without appreciating the severity of the injury. The voice is muted, the phrases are fragmentary, the look is restless. Faces and visible mucous membranes, often pale, normal pulse, sometimes intense. Blood pressure is normal or slightly elevated. This phase is short-lived, observed at the scene and rarely in the clinic.

Torpedo or braking phase

It is characterized by marked inhibition of the CNS, impaired function of the cardiovascular system, the development of respiratory failure and hypoxia (oxygen starvation).

Torpedo or braking phase

In the victim falls blood pressure, the pulse becomes weak and more frequent, the breath is weak, the body temperature decreases, the skin becomes pale, the patient is covered with cold sticky sweat. A characteristic feature is the depression of the victim, timeless attitude to others while maintaining consciousness.

In cases of adverse traumatic shock, terminal conditions may develop at the end of the torpedo phase.

Distinguish: preahony, agony and clinical death.

Clinical assessment of the condition of the victims

In the clinical assessment of the condition of the victims who are in a state of shock, it is advisable to allocate the phases of compensation (reversible compensated shock) and decompensation (uncompensated reversible shock, decompensated irreversible shock) on the basis of blood flow parameters.

An important feature

polytrauma is the development of a syndrome of mutual aggravation.

The assessment of the severity of traumatic shock in its torpedo phase is one of the key issues in the diagnosis and treatment of shock in severe mechanical damage.

The benchmark for determining the severity is the value of systolic pressure:

shock I degree - 90,

shock of the second degree - 85-75,

shock of the third degree - 70,

shock IV degree - below 70 mm Hg

Shock index

Algower's Index	Blood loss, BCC
0.8 and less than	10%
0.9-1.2	20%
1.3-1.4	30%
1.5 or more	40%

The main criteria for assessing the severity of traumatic shock and the amount of blood loss.

The degree of shock	Systolic pressure	Algover Index	The amount of blood loss		Consciousness
			L	%	
I	**> 90**	**0,7-0,8**	**До 1**	**10**	**Збережена**
II	**70-90**	**0,9-1,2**	**1-1,5**	**20**	**Збережена**
III	**50-70**	**>1,2**	**1,5-2**	**30**	**Сопор**

The main criteria for assessing the severity of traumatic shock and the amount of blood loss.

The degree of shock	Systolic pressure	Algover Index	The amount of blood loss		Consciousness
			L	%	
IV degree (terminal conditions): pre-gonia (systolic pressure> 50 mmHg, surface breathing, no consciousness; **agony (pressure is not determined; breathing is fluid, convulsive, with the participation of additional muscles);** **clinical death (cardiac arrest and respiration, reflexion).**					

Fracture localization Approximate volume of blood loss, ml

Pelvic bones (shock) 2500 - 4000

Pelvic bones (without shock) 500 - 1500

Range 500 - 2000

Femur 1500 - 2000

Shin (closed) 300 - 500

Tibia (open) up to 1500

Bone, foot to 250

Shoulder bone 500 - 800

Forearm bones up to 300

Rib 100 - 150

Multiple fractures, shock 2500 - 4000

! The shock usually becomes irreversible if full assistance is delayed by 2-4 hours.

Indicators of systemic hemodynamics in traumatic shock are of great practical importance - determine the content of antitussive therapy at each specific moment. The stabilization of the parameters of systemic hemodynamics, or the presence of a clear tendency to improve them, is evidence of a way out of shock. Hemodynamic instability, which persists for a long time, is seen as a continuation of shock.

Features of a shock depending on localization of damages

In the case of trauma to the lower half of the body in the first hours, the leading pathogenic factor of shock in the case of rapid development of the process is blood loss, and then toxemia begins to play an increasing role.

Features of a shock depending on localization of damages

In chest injuries (except blood loss), the violation of gas exchange and pumping function of the heart, irritation of the large receptor field is of great importance.

Features of a shock depending on localization of damages

The greatest difficulties in the diagnosis of shock occur in traumatic brain injury. In terms of the severity of the condition of the victims, the number of complications and deaths, these are the most serious injuries. At the same time, massive severe blood loss in such patients occurs less frequently than in other cases, resulting in long-term blood pressure may be normal or even elevated. Sometimes such a situation can be with injuries to the chest cavity.

Increased blood pressure in head and upper body injuries and restless behavior of the patient is often evidence of increasing brain hypoxia and should warn rather than reassure the physician!

Features of shock in children

The peculiarity of the manifestations of shock in children is a rapid nonspecific reaction of the organism and a clinical picture of "imaginary" well-being. The most characteristic feature of traumatic shock at an early age is the ability of the infant to maintain normal blood pressure for a long time, even after severe trauma.

Features of shock in children

Traumatic shock in childhood is characterized by such a feature: prolonged and sustained centralization of blood circulation in the absence of adequate therapy is suddenly replaced by decompensation of hemodynamics, which in children is much more difficult than in adults.

Features of shock in children

The smaller the child, the more unfavorable prognostic sign of shock is hypotension.

Features of shock in elderly people

In the event of a shock of varying severity in the elderly, in addition to the damaging agent, the nature and the zone of injury, the functional changes of the internal organs and the central nervous system that have arisen before the injury play an important role.

Features of shock in elderly people

Elderly people often experience chronic cardiovascular insufficiency and a decrease in circulating blood volume; shock develops at a much lower baseline BCC than in younger people.

Features of shock in elderly people

Normal, and even more so, increased levels of blood pressure in the elderly, especially immediately after trauma, can be a cause for misleading overestimation of the condition. Such a patient may be in a precocious or latent shock state.

Features of shock in elderly people

Under normal or somewhat reduced pressure (up to 100-120 mm Hg), it will be almost right to suspect shock in elderly patients and to take appropriate preventive and curative measures.

Features of shock in elderly people

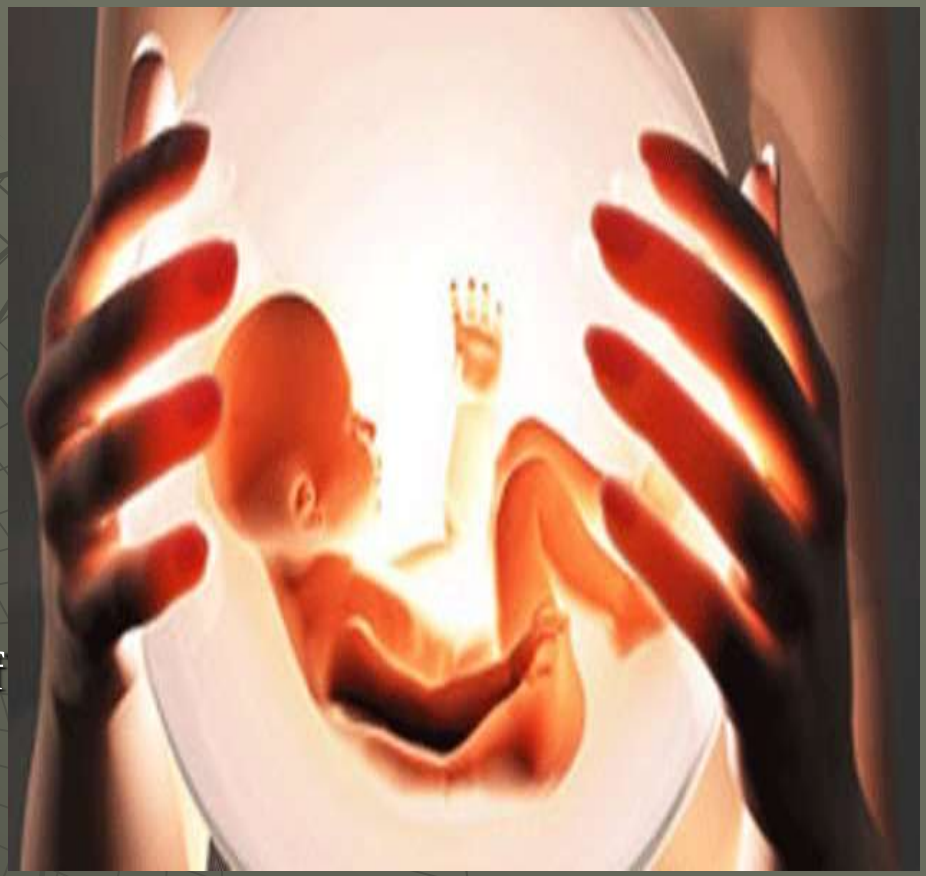
Traumatic shock in bone fractures in elderly patients more often than in young patients may be accompanied by fat embolism. With shock in patients with coronary atherosclerosis, decreased blood flow in the coronary vessels and increased blood viscosity observed in the elderly may accelerate coronary vascular thrombosis or cause myocardial ischemia with access to infarction.

Features of shock in elderly people

Brain thrombosis is also a common occurrence in age-related patients who are in a state of shock after bleeding, trauma, or surgery. In terms of the risk of traumatic shock, any significant injury should be considered potentially dangerous.

Features of assisting pregnant women

The timely provision of medical assistance to pregnant women who are traumatized is of great importance not only for the life of the expectant mother, but also for the outcome of pregnancy. A physician assisting a pregnant woman who is traumatized deals with two patients whose sensitivity to and response to trauma is different. Under the influence of stress, a woman's body tries to maintain her personal homeostasis at the expense of the homeostasis of the fetus. As a result, the development of shock and hypoxia in the mother decreases uterine-placental circulation, which adversely affects the fetus.



Features of assisting pregnant women

Urgent medical assistance to a woman in the first half of a traumatized pregnancy differs little from the methods of providing it to other victims with severe injuries.

In the later stages of pregnancy, the methods of treatment of the injured woman already have their own peculiarities. Physiological and anatomical changes associated with pregnancy affect not only the response of a woman's body to trauma, but also the interpretation of the results of diagnostic studies.

Features of assisting pregnant women

At the end of the second half of pregnancy, circulating blood volume increases by 15-30% compared to the level observed before pregnancy, and continues to slowly increase until delivery. In this regard, when carrying out countermeasures it is necessary to pour the amount of liquid, at least 25% exceeding the “calculated” volume. In a young woman, during pregnancy, the clinical symptoms of blood loss are often not determined until she is so large that it is too late to carry out any therapeutic measures.

Features of assisting pregnant women

A pregnant woman may lose up to 30-50% of the BCC before tachycardia, hypotension, pallor, and a weak pulse can be seen - signs that usually indicate the development of shock in its decompensated stage.

Features of assisting pregnant women

With the development of pregnancy hypotension, fluid transfusion improves the condition of both women and the fetus.

About 10% of women in the second half of pregnancy experience hypotension in their back position. It occurs as a result of contraction of the uterine vein. If a woman is left in this position for a few minutes, the minute volume of the heart decreases and hypotension may develop.

Features of assisting pregnant women

Although the obvious clinical manifestations of hypotension are not observed in all pregnant women, a greater or lesser degree of constriction of the empty vein occurs in each of them. This contraction can only contribute to the instability of a pregnant woman who is injured.

! Pregnant women who are injured in an accident should be transported in a prone position!

Even during surgery under the right thigh and the right half of the torso at the level of the wings of the iliac bone, a wedge-shaped roller should be inserted so that the uterus does not squeeze the inferior vena cava.

Treatment measures for shock

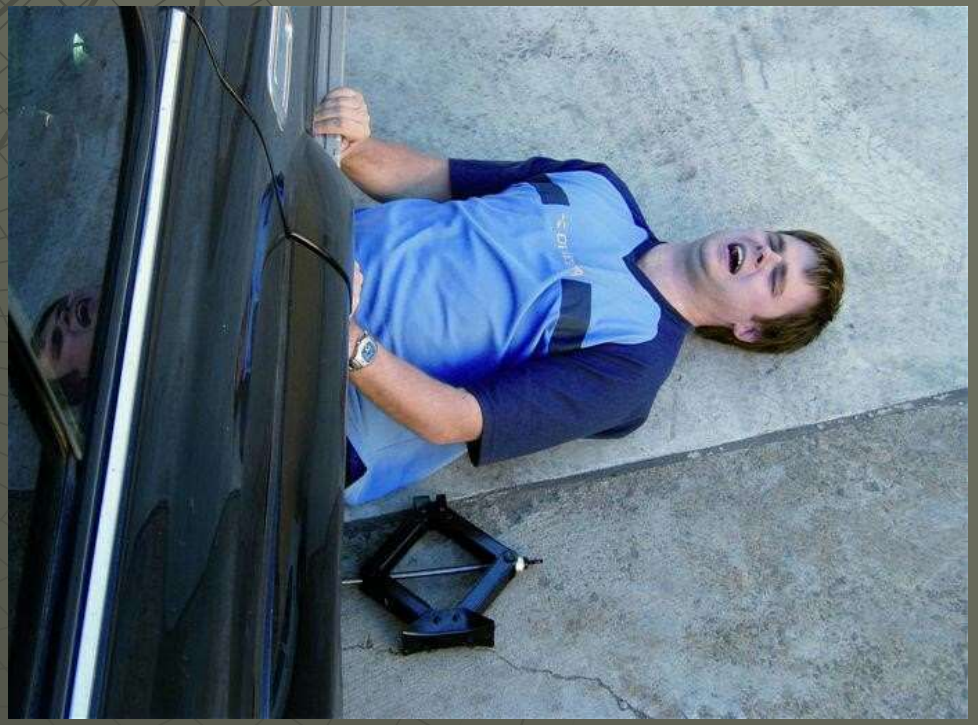
In case of shock trauma, active antitussive therapy should be initiated even in the absence of the expressed clinical manifestations of shock in the first time.

Starting from the first medical aid and further, all medicines to the victims with shock are introduced only directly into the vascular bed

Treatment measures for shock

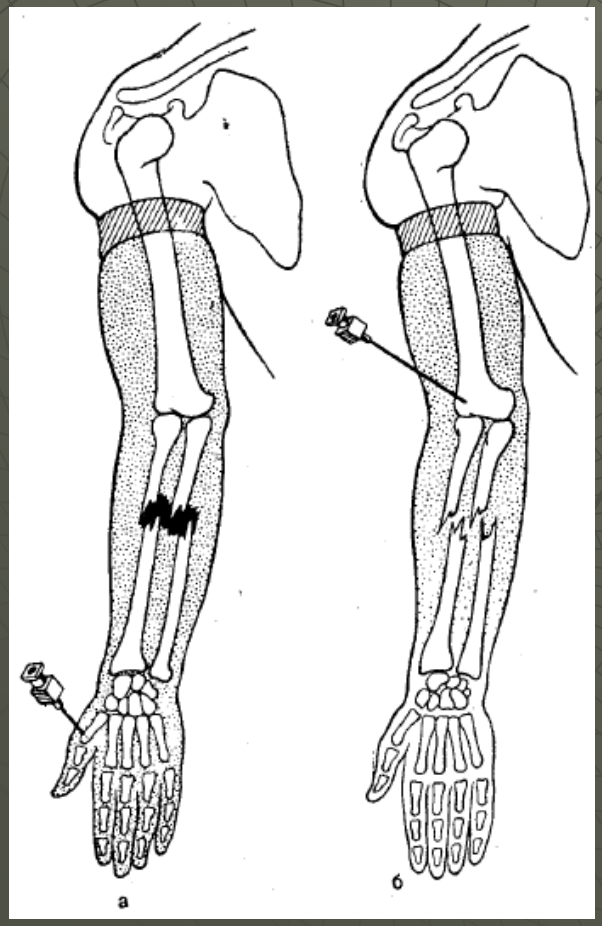
A set of countermeasures, which is mandatory when providing all types of medical care:

Elimination of the direct effect of the shock factor.



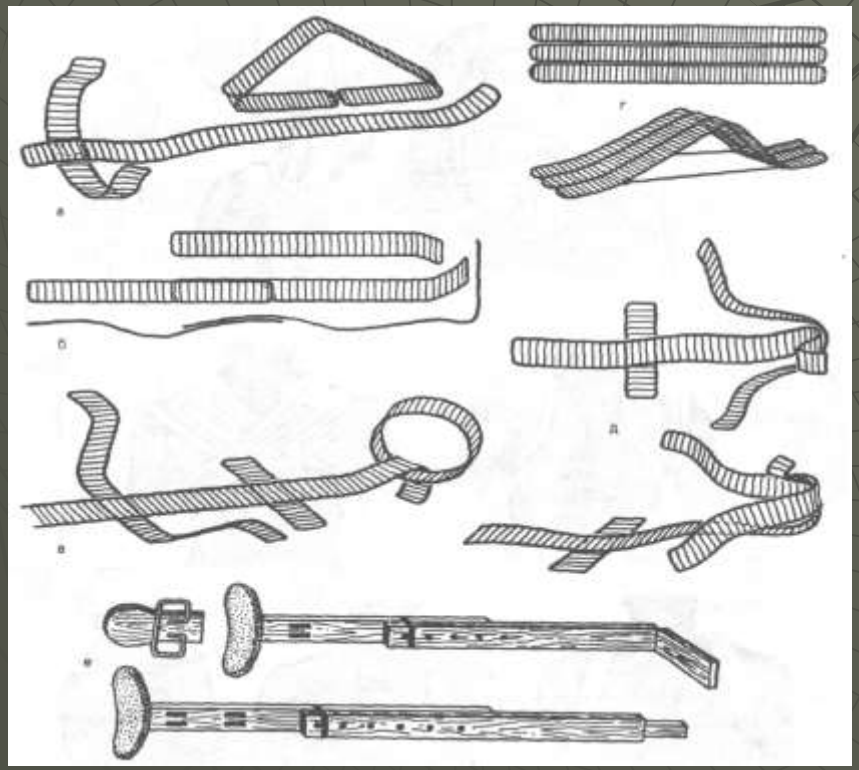
Treatment measures for shock

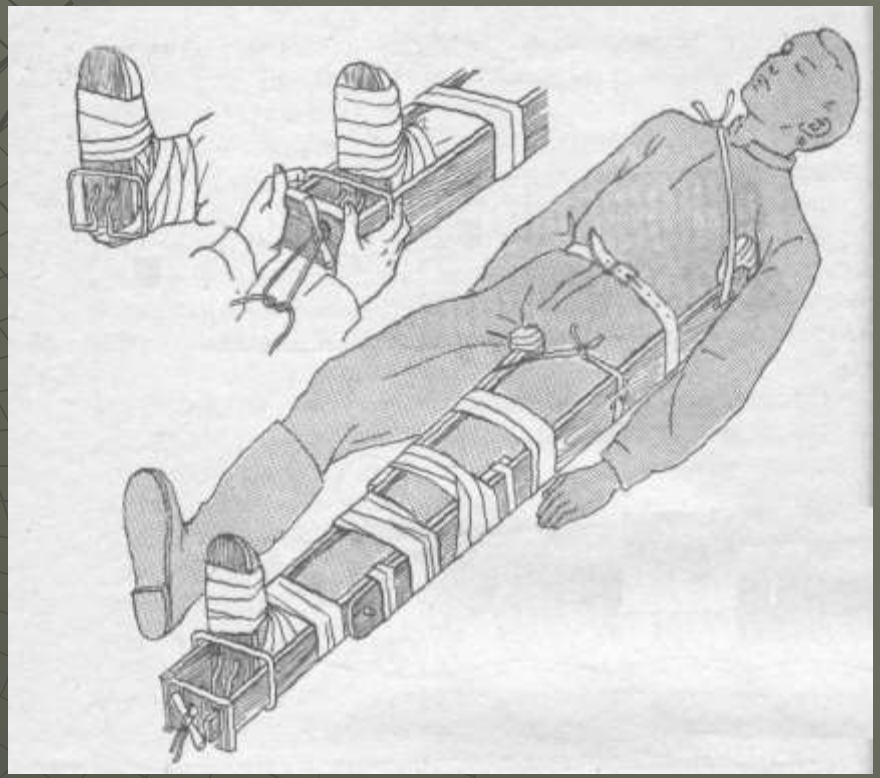
- anesthesia



Treatment measures for shock

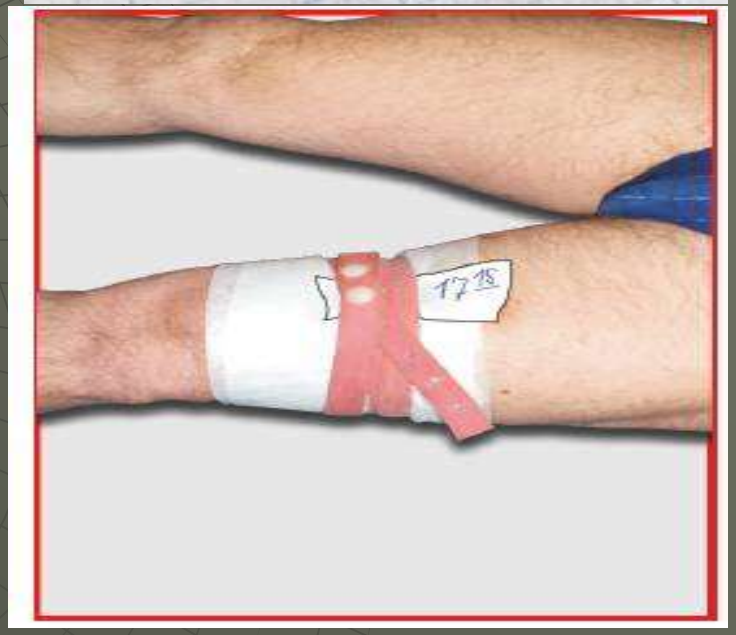
- immobilization.





Treatment measures for shock

- Stop bleeding..



Treatment measures for shock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Treatment measures for shock

- Respiratory and cardiac function support.
- Therapy is aimed at normalization of BCC and correction of metabolic disorders.
- !!! at the pre-hospital stage, if the time of transport to the hospital is not more than 1-1,5 g, the main thing is the presence of infusion therapy, not its content;

Treatment measures for shock

They must be performed as quickly and fully as possible, without which all counter-shock therapy cannot be effective.

Medical measures

The initial examination of the victim with severe trauma at the scene involves the following algorithm of actions:

A (Airway) - providing airway patency (removal of extraneous bodies, prevention of "sinking" of the tongue, aspiration of blood, vomiting masses, etc.) and control of the position of the cervical spine;

Medical measures

B (Breathing) - fight against the phenomena of hypoventilation, detection and elimination of dangerous complications of chest damage;

C (Circulation) - assessment of blood circulation adequacy, stopping of external bleeding, treatment of hypovolemia (infusion therapy);

D (Disability / neurology) - assessment of the degree of cognitive impairment, etc. neurological disorders (paresis, paralysis).

The incompatibility of therapy is often a major problem in the provision of medical care to victims of polytrauma. Thus, if the injury to the musculoskeletal system shows the introduction of narcotic analgesics to relieve pain, then when combined with these injuries with severe traumatic brain injury drug use is contraindicated. Thorax trauma does not allow the tire to be imposed on a shoulder fracture, and the large area of burns makes it impossible to adequately immobilize this segment with a gypsum bandage at a concomitant fracture.

Treatment of shock

The hospital phase of treatment of a polytrauma involves repeated (control) measures of primary examination (A, B, C, D), followed by the next stage of examination - E (Exposure) - dressing and head-to-toe examination.

For the correct solution of medical and tactical problems in the provision of medical care to victims with political trauma, it is necessary to identify the dominant damage, which determines the severity of the condition at the moment and is an immediate threat to life.

The main directions of correction of disorders in traumatic shock

Basic principles of anti-shock assistance:

1. observance of the principle of obligatory restoration of the anatomical integrity of organs and systems;

2. preservation of critical mass of blood;

The main directions of correction of disorders in traumatic shock

3. support of vital functions, first and foremost;
4. polyvalence of assistance - a complex effect on the circulatory system, respiration, water - electrolyte balance, kidneys

Healing moments in shock

1. *Infusion transfusion therapy (the basis of antitussive treatment)*

When developing a program of infusion therapy for blood loss and shock, the following issues need to be addressed:

determining the total volume of fluid injected;

determining the speed of bulk replacement;

determination of substitution rate of infusion - transfusion fluid composition

Approximate infusion transfusion therapy program

The degree of severity	Blood -loss-, ml	The volume of liquids that are injected			
		Blood	Colloids	Crystalloids	Total
I (reversible)	**500**	**-**	**300-500**	**700-1000**	**1000-1500**

1	2	3	4	5	6
II (shareware)	**1500**	**500**	**1000**	**1000**	**2500**
III (irreversible)	**2000**	**1000-1500**	**1500-2000**	**2000-2500**	**4500-6000**

Healing moments in shock

2. Vascular tone correction

It is possible to enter any medicines that have an active effect on vascular tone only after BCC recovery! It is unacceptable to consider the introduction of vasoconstrictor agents as an alternative to the infusion methods of BCC recovery.

3. Hormone therapy.

The introduction of large doses (hydrocortisone -500-1000 mg) of glucocorticoids, especially in the first minutes of treatment, has a positive effect on the heart, reduces renal vascular spasm and capillary permeability; eliminates adhesive properties of blood cells; restores reduced osmolarity of intracellular and extracellular fluid spaces.

To correct BCC, crystalloid and colloidal solutions and blood components are widely used. At the same time, they pursue the goal not only to compensate for OCC, but also to combat generalized tissue dehydration and correction of water and electrolyte balance.

In decompensation conditions, it is necessary to control the acid-base state of the blood (pH and alkaline reserve), because, instead of apparent metabolic acidosis, shock is often metabolic alkalosis, especially 6-8 hours after injury. In this case, alkalosis occurs more often, the later the BCC deficit is replenished.

When conducting infusion-transfusion therapy for shock, it is necessary to consider:

- preservation of the principle of hypertransfusion (the volume of infusions should be higher than the BCC deficit by at least 1.5-2 times;
- maintaining the principle of achieving artificial hemodilution with hematocrit retention at the level of 30-35% (with further reduction of hematocrit, the transport of oxygen by blood is impaired (there is a loss of erythrocytes), and with hematocrit above 35%, the fluidity of blood (loss of plasma) is reduced, which also disrupts transport. to fabrics);

- systolic pressure during treatment should be equal to 90-100 mm Hg, which will provide relatively favorable conditions of circulation not only in vital organs, but also in peripheral departments;
- the criterion of sufficiency of infusion-transfusion therapy is stabilization of blood pressure, reduction of CVT (less than 6-16 cm of water), restoration of diuresis;
- the total volume of colloidal substances administered should not exceed 1500-2000 ml;

- saline substitutes can be administered at a higher rate of up to 2.5 l / h, especially in the initial period when transfusions cannot under any circumstances be initiated;
- it is necessary to take into account the metabolic needs of the body;
- the amount of blood that is injected should not exceed the amount of blood loss;

- for traumas accompanied by massive tissue fracture, it is advisable to combine infusion of saline blood substitutes with forced diuresis (administration of up to 6 l of crystalloid solutions in combination with lasix - 2-4 ml of 1% solution intravenously);
- with blood pressure less than 70 mm Hg, which cannot be increased by conventional infusion transfusion therapy, intraarterial administration of blood substitutes is shown;

- infusion-transfusion therapy should be performed taking into account the localization of major damage (especially in traumatic brain injury) and the predominant pathogenic factor (BCC deficiency, intoxication, etc.);

Healing moments in shock

Adequate volume of liquid - 3 l.

Input speed:

20 drops per 1 minute can be injected 24/7;

In extreme cases 300-400 per 1 minute

Surgical tactics at polytrauma

A short list of surgical interventions performed at polytrauma:

Surgery performed on a vital basis (external and internal bleeding that goes on, wounds of the heart with tamponade of the pericardium, increasing contraction of the brain).

Chest surgery (multiple rib fractures, “fractured” rib fractures, displaced sternum fractures, sternal dislocations of the clavicle with the risk of damage to the mediastinum);

Surgical tactics at polytrauma

- Operations on abdominal organs (operations on parenchymal organs; operations on hollow organs);
- Surgical treatment of the wound and operations on the musculoskeletal system (extracellular osteosynthesis with external fixation devices).

Fat embolism

The most severe complication of bone fracture is fat embolism syndrome (intravascular fat penetration), which occurs as a result of severe trauma, and 0.5-2% - after fractures of long tubular bones. The most common cause of fat embolism is a fracture of the femur and tibia. The syndrome is also observed in some conditions that are not associated with trauma: respiratory distress syndrome, sepsis after a massive blood transfusion, as well as in patients with systemic collagenoses.

Fat embolism

Classification.

All current classifications, and there are more than a dozen, reflect two aspects of the flow of fat embolism:

time of occurrence;

features of the clinical picture.

By time of occurrence distinguish the following forms:

lightning, which leads to the death of the patient within a few minutes;

acute, developing in the first hours after injury;

sub-acute, with a latent period of 12 to 72 hours;

subclinical, in which clinical symptoms are unclear or absent.

Fat embolism

According to the features of clinical manifestations acute form is divided into phases:

fatty embolism of blood vessels with symptoms of pulmonary heart failure;

fat embolism of a large circle of circulation, with lesions of internal organs and predominance of phenomena from the CNS.

Fat embolism

The clinical picture

The classic triad in the same is considered to be CNS dysfunction, respiratory disorders and petechial rashes on the skin.

! The first signs are manifested by a disturbance in the neuropsychic sphere (Neurological status, which is considered to be a true indicator of the development of fat embolism):

Fat embolism

- fear, anxiety, restlessness, emotional unbalance;
- cerebral form: weakness, headaches, drowsiness, dizziness, coma;
- pulmonary form: shortness of breath, chest pain, difficulty breathing, cough, sputum, shortness of breath, wheezing;
- integuments pale, pale cyanotic, cold, moist;
- blood pressure is increased, central venous pressure is increased.

Fat embolism

More recent symptoms:

decrease in hemoglobin;

high leukocytosis, ESR;

radiographically manifests "blizzard syndrome" (snowstorm) in the lung tissue;

changes in the retina, conjunctival petechia, characteristic pattern of the fundus - rounded clouded spots located near the vessels of the retina, multiple hemorrhages, vascularity (purcher symptom);

- petechial rash on the skin, which occurs for 2-3 days.

Fat embolism

Typical manifestations of fat embolism are petechial hemorrhages at the base of the neck, under vaginal folds, and mucous cheeks and conjunctiva. The distribution and intensity of the hives varies over time. In some cases, it can be determined using a magnifier.

Fat embolism

CNS lesions are manifested by various disorders of consciousness from resistance to coma, sometimes with transient focal neurological symptoms, meningeal symptoms. Typical is the presence of a so-called "light period", which can last from several hours to several days.

Fat embolism

Emerging acute respiratory failure is growing tachypnoe, dyspnoea, signs of shock lungs and is caused by progressive hypoxia and hypoxemia. Wet rales are heard over the lungs. Increase of pressure in a large circle of blood circulation with its subsequent fall, blood deposition in the pool of the portal vein, venous hypertension, bilateral pulmonary edema.

Fat embolism

- Cardiac disorders are expressed by bradycardia with the transition to progressive persistent unmotivated tachycardia, tachyarrhythmia, with certain signs of myocardial ischemia on the ECG.
- Another common feature is hyperthermia (fever up to 400C, which is caused by irritation of brain heat-regulating structures with fatty acids and endogenous pyrogens (interleukins, cachectin)).

Fat embolism

- In addition, jaundice, impaired renal function.
- Pathognomonic signs of fat embolism do not exist. Fat in the urine appears during the first days of trauma in 60% of patients, but in most traumatological patients it is determined in the urine and in the absence of fat embolism. In 50% of cases for 3-4 days increases the activity of lipase in the serum.

Fat embolism

Treatment begins at the pre-trial stage and should be aimed at maintaining vital functions (immobilization, anesthesia, bleeding arrest, infusion therapy). Performing these manipulations can significantly change the further course of traumatic illness.

Fat embolism

After hospitalization of the victim in hospital, therapy is directed to further support:

- vital functions,

- adequate gas exchange and oxygen level in the blood,

-normalization of plasma lipids.

First of all, the issue of the need for surgery is solved. It must be remembered that surgery is an additional stress factor that can push the body to manifest forms of fat embolism. When resolving the issue of active surgical tactics recommended by the method of choice - extracorporeal osteosynthesis with spike, rod and spike-rod devices in the earliest period since the injury.

Fat embolism

Along with these activities spend:

adequate analgesia;

oxygen therapy (oxygen inhalation) to ensure adequate oxygen delivery to the tissues;

administration of antihypoxants and antioxidants (Mexidol, Vit. C, cytochrome, berlithion 600-1200 mg / day);

Berlithion suppresses the processes of lipolysis, decreases the concentration of free fatty acids and triglycerides.

Fat embolism

- infusion-transfusion therapy in the mode of moderate hemodilution, elimination of hypovolemia:
- therapy of systemic disorders of the microcirculation involves the introduction of reopolyglyukin, kurantil, trental. To eliminate angiospasm of the peripheral vascular bed use glucose-novocaine mixture;

Fat embolism

Traditional for a long time was the introduction of 1-5 days from the moment of trauma drugs essential phospholipids (essential 10-25 mg / kg per day), which, according to existing ideas, contribute to the transition of large drops of fat into shallower.

Nowadays, in accordance with the principles of evidence-based medicine, the effectiveness of the introduction of methylprednisolone (salt-medrol) for the prevention of ZHE is convincingly confirmed. But the effect of the drug is effective only in the timely administration - 30 mg / kg twice (during hospitalization of the patient and after 4 hours).

Fat embolism

- administration of insulin and glucose to reduce the content of fatty acids in arterial blood (this reduces the toxic effect of free fatty acids on the lungs);
- medication therapy of brain hypoxia, fight against pathological impulsion includes antihypoxants (sodium oxybutyrate, opiates, barbiturates, etc.);
- nootropic and metabolic therapy is based on the same approaches as treatment of post-hypoxic encephalopathy (nootropil, cerebrolysin, actovegin, nimotope);
- correction of the system of coagulation and fibrinolysis (transfusion of fresh frozen plasma, low molecular weight heparins);

Fat embolism

- protection of tissues from free oxygen radicals and enzymes (kontrikal, trasilol 500000 IU, ascorbic acid up to 2 g per day, tocopherol up to 300 mg per day). A wide range of pharmacological activity has lipin in daily doses of 2-3 g, which has antihypoxic, antioxidant and membrane-protecting effect;
- restoration of the physiological state of demulsified fat in the blood is achieved by transfusion of lipostabil to 150 ml per day, essential to 40 ml per day;

Fat embolism

- in anemia, transfusion is performed not of whole blood, but only the necessary components - washed erythrocytes, albumin, but in a limited amount, so as not to cause an increase in blood pressure and pushing of the emboli from the pool of the pulmonary artery into a large circle of circulation and to prevent the development of brain form of fatty em;
- detoxification therapy;
- parenteral and enteral nutrition;
- prevention of purulent-septic complications;
- early operative stabilization of fractures.

Fat embolism

Prevention.

There is no specific prevention of fat embolism. But in a timely manner, qualified first aid in polytrauma (reliable transport immobilization, sufficient anesthesia, antitussive therapy aimed at filling the BCC) has an impact on the further course of traumatic disease and the occurrence of complications of bone fractures.

Fat embolism

herefore, in order to prevent fat embolism, it is recommended that:

- to consider all patients with polytrauma and monotraum of the lower extremities delivered by passing transport a risk group in relation to the development of the bile ducts;

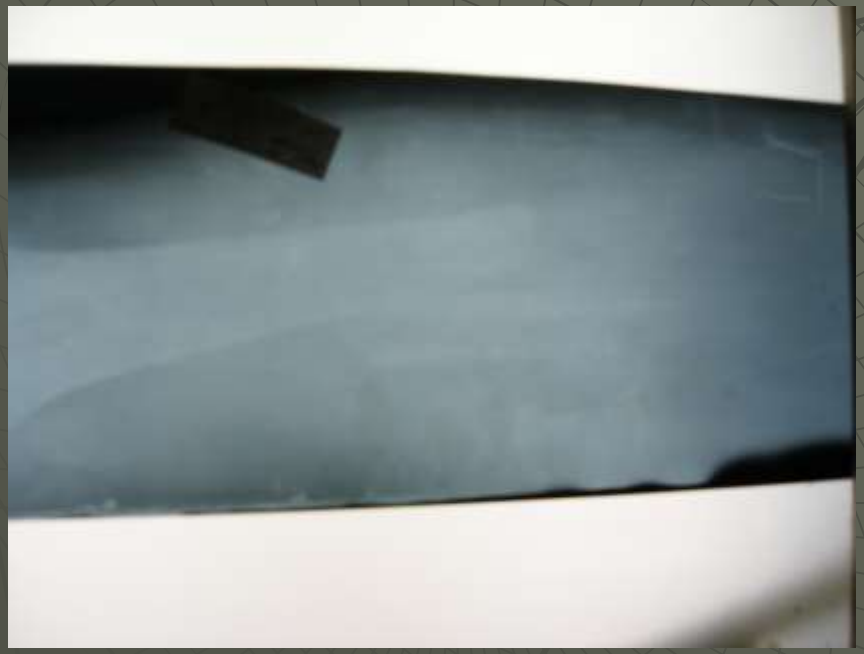
- all patients, irrespective of the method of delivery, who are in a state of traumatic shock, as well as all patients operated by the method of intramedullary osteosynthesis (especially with the use of cement), should also be classified as at risk of developing FE.

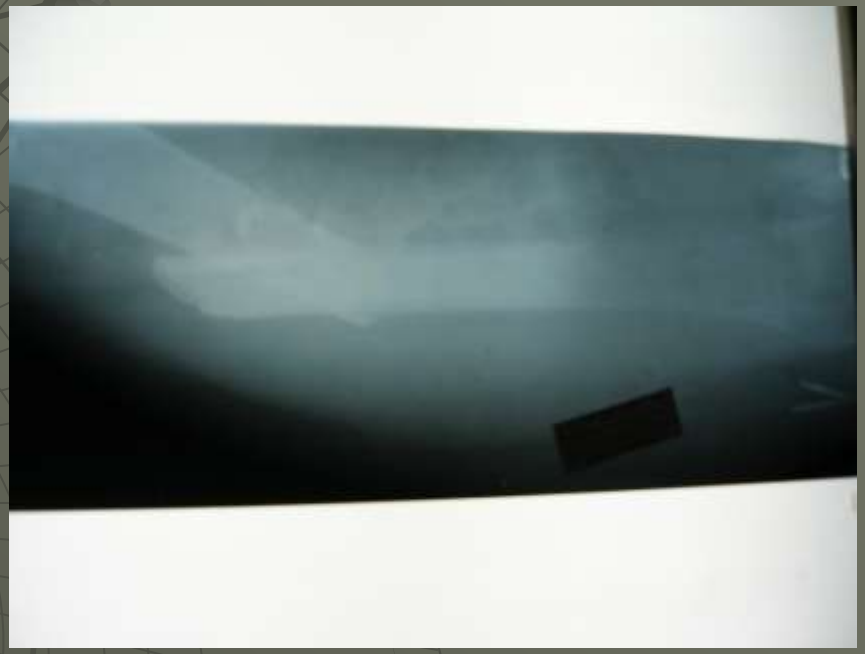
Case of practice (polytrauma)

Patient K., 19, was injured as a result of an accident.

Diagnosis: closed fractures of the right and left thighs, right lower leg, right shoulder, right forearm with displacement. Traumatic brain injury closed. Shock II-III. Fat embolism

Polytrauma





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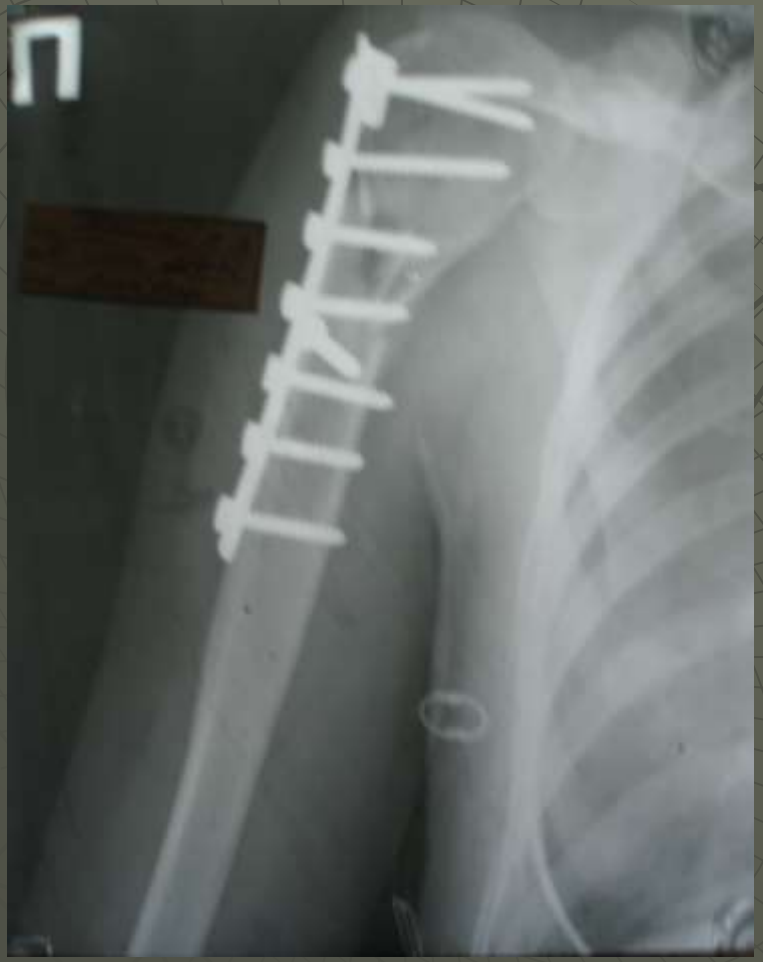

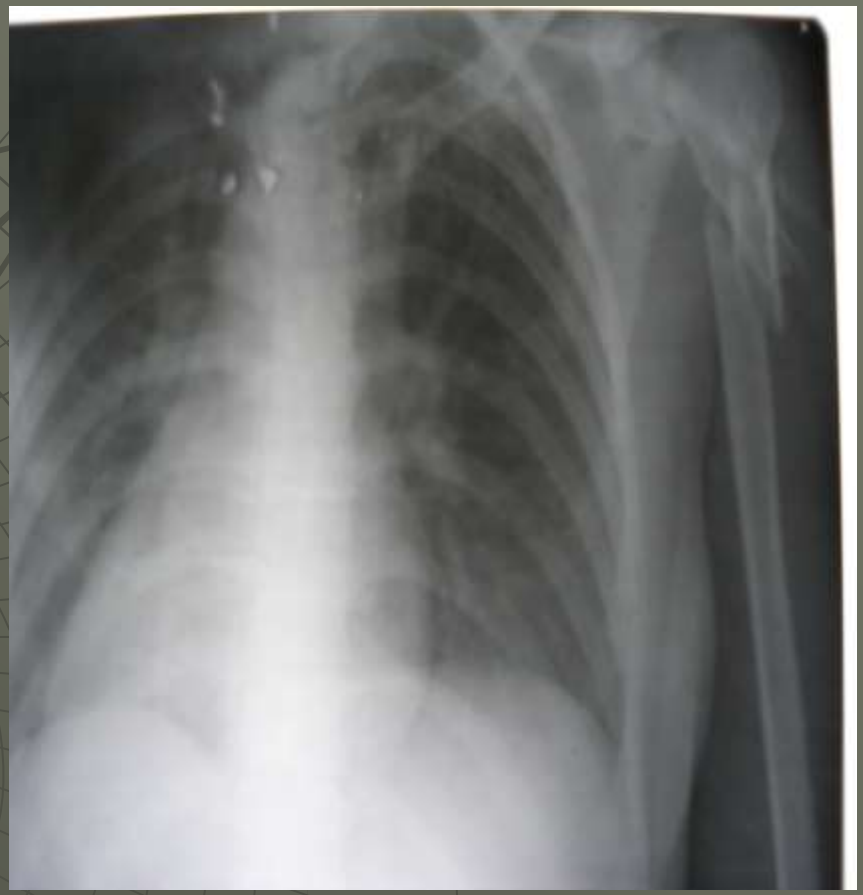

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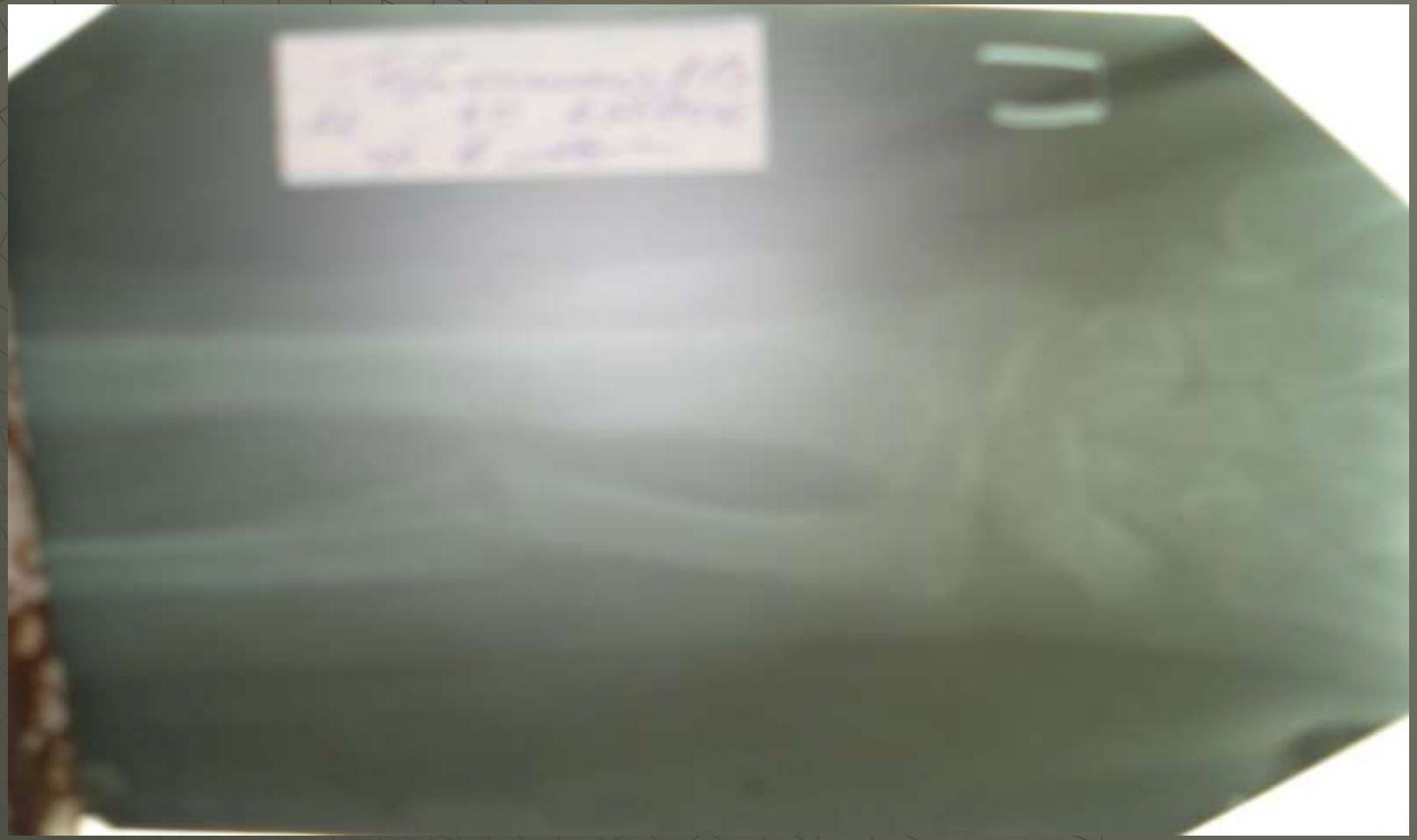

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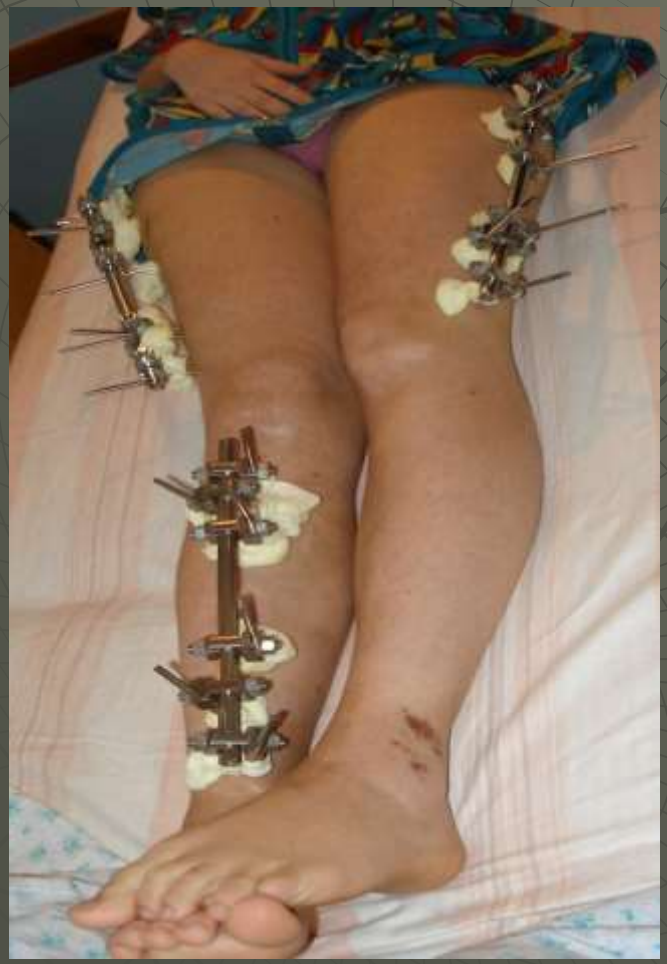
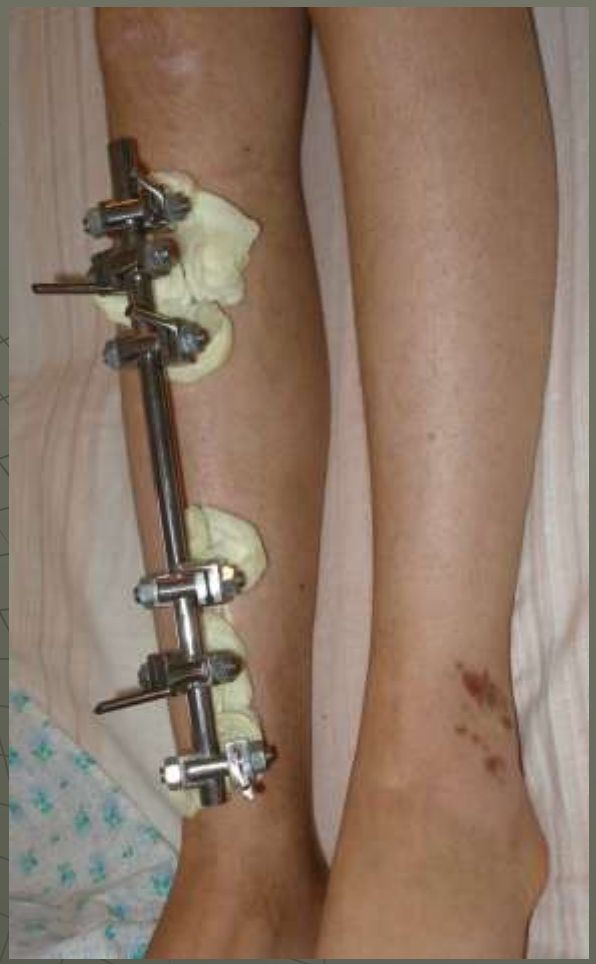
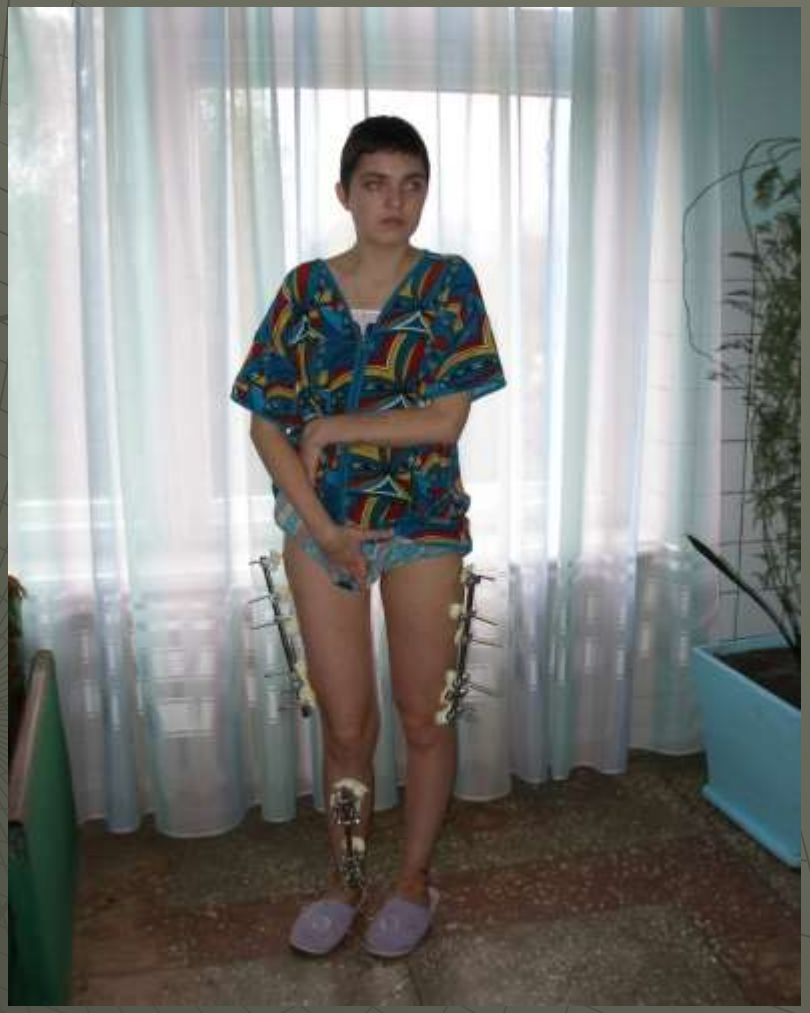
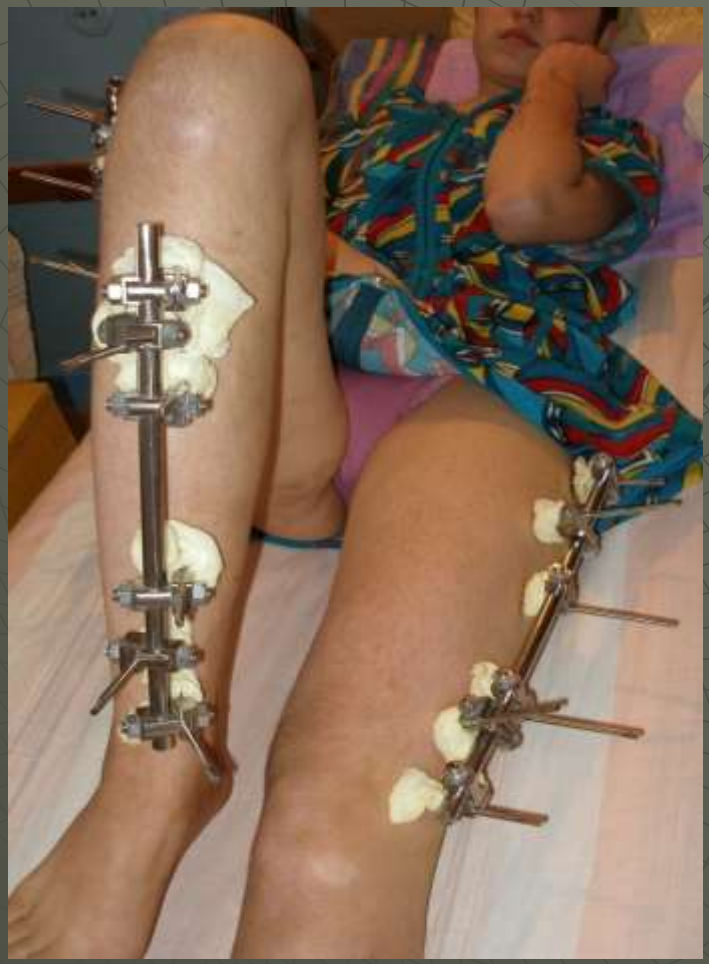


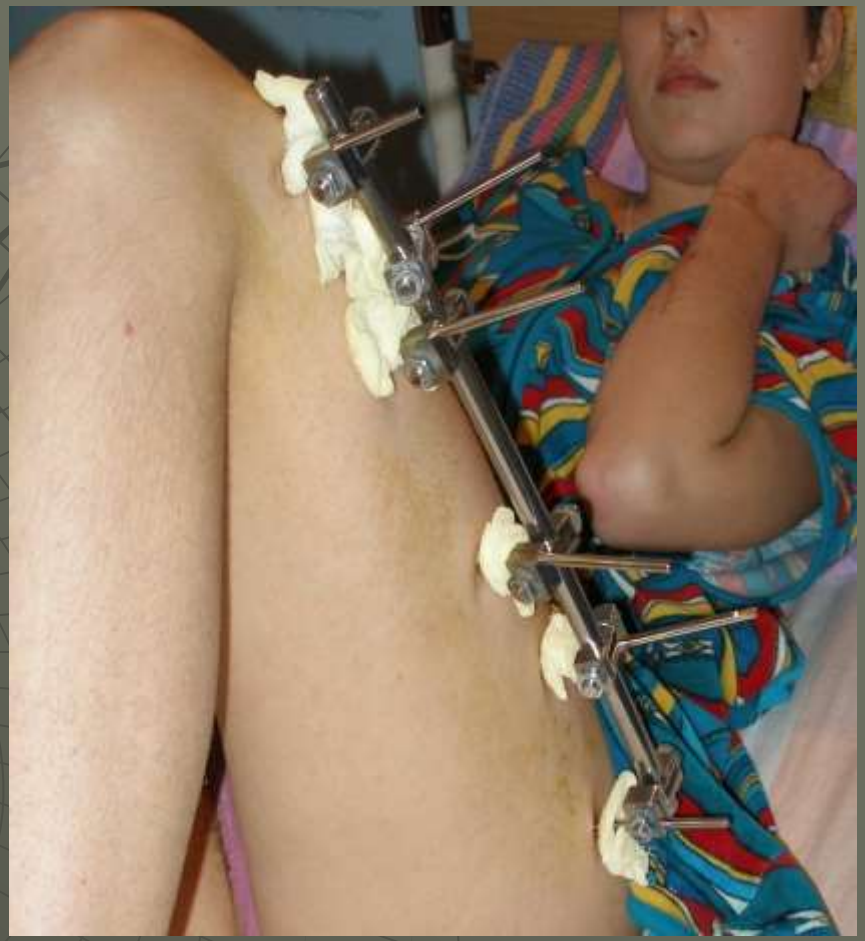


Result 2.5 months after injury



Result





THANKS FOR ATTENTION!