MINISTRY OF HEATH OF UKRAINE
POLTAVA STATE MEDICAL UNIVERSITY

Department of general surgery

METHODICAL RECOMMENDATIONS

FOR INDEPENDENT WORK OF THE STUDENT

IN TIME FOR PREPARATION TO THE PRACTICAL CLASSES

(auditorium work)

Study discipline	**General surgery**
Module №2	**Surgical infection. Necrosis. Basics of clinical oncology. Curing of surgical patients.**
Substantial module 1. Surgical infection. Necrosis	**Surgical infection. Necrosis.**
Lesson theme №27	Deadness. Necrosis. Gangrene. Ulcers. Fistula. Causes of occurrence. Clinical manifestations, diagnosis, treatment.
Course	***III***
Faculty	***International***

Poltava

Substantial module 1. Surgical infection. Necrosis	**Surgical infection. Necrosis.**
Lesson theme №26	Deadness. Necrosis. Gangrene. Ulcers. Fistula. Causes of occurrence. Clinical manifestations, diagnosis, treatment.

1. Results of training:

General competence - Relevance of the topic One of the typical pathological processes is necrosis (H). H defines a (often in combination with other processes) the development of many diseases : myocardial infarction , burns , frostbite , gangrene of extremities (including anaerobic) , lung , intestine , scrotum , gangrenous appendicitis, gangrenous cholecystitis, acute pancreatitis (pancreatic) strangulated hernia , intestinal obstruction , trophic ulcers, osteomyelitis. This is evidence of the importance of H in the development of surgical pathology. Therefore, the study of this topic is especially important for students of the third year of medical school .

Subject competence - Know the causes of necrosis, gangrene, ulcers, fistula, diagnostic methods, basic principles of treatment

2 . Learning Objectives :

1. Know the causes necrosis , gangrene , ulcers , fistulas
2 . Learn the classification of necrosis , gangrene , ulcers , fistulas
3 . Know the clinical signs of necrosis , gangrene , ulcers , fistulas
4 . Know the diagnostic features of certain types of necrosis , gangrene , ulcers , fistulas
5 . Learn the advanced phase - necrotic ulcerative process
6. To know the methods of necrosis , gangrene , ulcers , fistulas
7. Know the principles of diagnosis and treatment policy for foreign bodies
8. To capture the method of dressing for ulcers , fistulas
9. Capture the method of care for bed sores
10 . To master the technique of fistulography
11. To master the technique of painting samples

3 . Basic knowledge , skills necessary for studying the topic (inter-disciplinary integration)

The names of the preceding disciplines	the acquired skills
1. anatomy	Anatomy of the hand, the structure of the tendon sheaths, blood supply and innervation of the fingers and wrist
2. microbiology	The principles of the research content of the microflora and its sensitivity to antibiotics. Sterility control. Tech fence material.
3. pathophysiology	Signs of inflammatory process
4. pharmacology	Antibiotics principles and their application. Know the drugs, ways of administration, mechanism of action

The student must have an idea :

- On the etiology, pathogenesis , classification , clinic, and the structure of festering wounds ;
- The modern classification of gangrene , ulcers , fistulas , foreign bodies of the body;
- The mechanism of healing of gangrene , ulcers , fistulas ;
- The general reaction of the organism to necrosis , gangrene , ulcers, fistula, foreign body in the tissues and body cavities ;
- On special methods of clinical examination of patients.

The student should know :
- Basics of aseptic and antiseptic ;
- Definition gangrena , ulcers, fistula, foreign body of an organism;
- Etiology and pathogenesis of gangrene , ulcers , fistulas , foreign bodies of the body;
- The histological structure of the skin , adipose tissue, mucous membranes;
- Possible general and local complications ;
- The main stages of surgery ;
- Features of gangrene , ulcers , fistulas , foreign bodies of the body;
- Methods of gangrene , ulcers , fistulas , foreign bodies of the body;
- Different types of dressings that can be used ;
- The principles of care for patients with wounds ;
- Basic mechanisms and timing of regeneration gangrene , ulcers , fistulas , foreign bodies of the body.

The student should be able to:
- Apply the principles of care for surgical patients (hygiene of patients with the disease site) ;
- Inspect the patient and the site of disease ;
- Be able to provide clinical interpretation of the identified symptoms;
- Establish a clinical diagnosis ;
- Appoint conservative treatment for wounds of various locations ;
- Justify the indications for surgical intervention ;
- Conduct post-operative monitoring of patients and to provide care ;
- Apply preventive agents of infection in the wound

Mastering the skills of students:
- Master the technique of palpation to determine the boundaries of the inflamed tissues and organs ;
- Identify the symptoms of inflammation ;
- Learn to identify fluctuation or softening in the inflammation ;
- Learning how to wash the wound with antiseptics ;
- Identify the clinical and morphological characteristics of the primary and wound healing by secondary intention ;

- To improve the technique of applying different types of bandages , depending on the location of the wound ;
- Master the technique of collection of material on the sensitivity of microorganisms to antibiotics.

4.Tasks for self-study in preparation for the lesson.

4.1. The list of basic terms, parameters, characteristics, which the student must learn in preparation for the class:

Termin	definition
necrosis	the death of cells, tissues or organs in a living organism.
bionecrosis	irreversible degenerative changes prior to N.
Patobioz	bionecrosis, extended in time.
gangrene	necrosis part of the body (organ) that contacts with the external environment.
heart attack	ischemic necrosis.
detritus	state of tissue necrosis and subjected to decay.
sequestration	area of necrotic tissue is available in living tissues.
paranecrosis	set of morphological manifestations of reverse cell changes that characterize the local distribution of excitation density increase of colloids of the cytoplasm and the nucleus, changes electolit structure, enhance the sorption properties of the cytoplasm. Paranecrosis - parabiotic morphological expression.

4.2 . Theoretical questions for the class :

1. The modern view of the etiology , pathogenesis, clinical and morphological characteristics of gangrene , ulcers , fistulas , foreign bodies of the body.
2 . The clinic, diagnosis gangrene , ulcers , fistulas , foreign bodies of the body.
3 . Diagnosis and monitoring of the wound process in gangrene , ulcers , fistulas , foreign bodies of the body.
4 . Modern principles and methods of treatment.
5 . Types of wound healing after treatment of gangrene , ulcers , fistulas , foreign bodies of the body.
6. Technique of surgical treatment of gangrene , ulcers , fistulas , foreign bodies of the body.
7. General characteristic of the drugs that are used for the topical treatment and prevention of infectious complications.
8. Treatment of gangrene , ulcers , fistulas , depending on the phase of wound healing .
9. Preventive measures for the further progression of the disease .

4.3. Practical work assignments used in class:

1. transport the patients to the dressing .
2. Laying according to the patient's dressing table area of the manipulation.
3. Preparation of hands to perform the procedure.
4. To be able to put on a sterile gown .
5. To collect anamnesis of patients .

6. To evaluate the results of laboratory research methods and plan further investigation .
7. A plan of treatment a particular patient .
8. Drawing up of a landmark epicrisis .
9. Development of primary documentation (history).
10 . Technique of the necrectomy .
11. Prepare a kit for washing ulcers.
12. Prepare a kit for performing ligation .
13. Specimen collection for bacteriological control .
14. Different types of cleaning in contaminated dressing .
15. Imaging technique of foreign bodies.
16. Methods for closure of ulcers of the skin.
17. Determine the prevalence of necrosis.
18. Probing fistula.
19. Preparing the patient for fistulography .
20. Manufacturing tables and other illustrative material (photos , slides, drugs) .
22. Disposal of dressing.
23. An analysis of archival material.

5. Contents training

Structural and logic threads

Necrosis and gangrene : Definition - the circumstances of occurrence - the classification

Classification - especially hospitals - particularly treatment

During the necrotic process - characteristics of individual species - their symptoms - treatment

Symptoms of necrosis and gangrene - general and local manifestations

Methods - selection based on determining the type of necrosis and the spread of the process

Ulcer : definition - the circumstances of occurrence - the classification

Classification - structural features - features of clinic - especially treatment

For necrotizing process - symptoms - treatment

Symptoms of ulcers - general and local manifestations

Methods - selection based on determining the nature of the origin of the phases of the process and the structural features of the wound

Decubitus - a special kind of trophic ulcers

Fistula : definition - the circumstances of occurrence - the classification

Classification - structural features - features of clinic - especially treatment

Symptoms of fistulas - general and local manifestations

Specific diagnostic methods fistulography , coloring sample .

Methods - selection based on the determination of the structure and origin

Foreign bodies :

definition - the circumstances of occurrence - the classification

reaction of the body - especially the clinic - especially treatment
Symptoms of foreign body - the general and local manifestations
Methods - selection based on localization , origin, possible consequences.

BASIC CONCEPTS

Necrosis (death) is the death of cells, tissues or organs in a living body.

Necrobiosis is irreversible dystrophic changes that precede necrosis.

Patobiosis is a necrobiosis that is stretched over time.

Gangrene is the death of a part of the body (organ) that contacts the outside environment.

A heart attack is an ischemic necrosis of internal organs that do not contact the external environment.

Detritus is the condition of tissue that has experienced necrosis and decay.

Sequester is a site of necrotized tissue that is freely located among living tissues.

Paranecrosis is a collection of morphological manifestations of reverse changes of cells, which characterize local common excitation: increase of density of colloids of cytoplasm and nucleus, changes of electrolyte composition, strengthening of sorption properties of cytoplasm. Paranecrosis is the morphological expression of parabiosis.

Necrosis reasons.

1. Factors that directly involve N (direct N):

• Mechanical (coarse mechanical force that exceeds the resistance of fabrics, for example, when the fabrics are crushed or crushed);

• Physical (high (> 60 ° C) and low (< 150 ° C) temperature, which causes burning or freezing, electric current of high voltage, which causes necrosis of tissues at the places of entry and exit, penetrating radiation);

• Chemical (acids that coagulate protein followed by coagulation necrosis, alkalis that dissolve protein and wash fats followed by colicvestive necrosis, mustard, calcium chloride upon subcutaneous administration);

• Biological (microbial toxins, specific infection (TV, lepra, syphilis), proteolytic enzymes that digest mucous membrane in DPC ulcer and stomach, acute pancreatitis).

2. Factors that contribute to N development (indirect N)

• Critical reduction of blood circulation in arteries in thrombosis, embolism, long-term spasm, endarteritis, atherosclerosis, diabetic macroangiopathy, artery injury, external compression (harness, impaired hernia, mobile organ germination), circulation failure. Examples include myocardial infarction, lower limb gangrene on atherosclerosis, intestinal necrosis in pinched hernia.

• Critical disturbance of blood outflow along veins with varicose veins expansion, deep vein thrombosis, TCP, thrombophlebitis. An example is venous gangrene.

• Sharp decrease and deceleration of blood circulation in small finite arteries and microcirculatory chain in case of Reino disease, diabetic microangiopathy, infectious and inflammatory processes, external or internal compression (lying).

• Vascular wall changes in endarteritis, atherosclerosis (Mortarel sclerosis), diabetes mellitus

• Disorders of trophic innervation (neurogenic, trophoneurotic N) Example - ulcers in damage to peripheral nerves, foot ulcer, which perforates (malum perforans pedis), on the heel or in the site of the 1st or 5th metatarsal bones.

• Metabolic disorders (avitaminosis, diabetes mellitus, anaemia).

• Ulcer formation in the tumor site (their decay).

EMERGENCE OF NECROTIC CHANGES

Critical microcirculation parameters in which tissue necrosis develops (A.A. Polynsky, 1985):

• Volumetric pulse blood filling of tissues < 20, 6 ± 2.7 ml/xv per 100 cm3 of tissue;

• Volumetric circulation rate < 2, 4 ± 0.2 ml/xv per 100 cm3;

• Capillary pressure < 18, 5 ± 3.1 mm Hg.

Hypoxia reduces oxidative enzyme activity in tissues and capillaries; The composition of the isoenzyme spectrum of LDG changes towards the advantage of fractions that function under anaerobic conditions. As a result of reduced redox processes, enzyme activity, lysosome decay occurs and hydrolases are released, which lyse cells and tissues. They increase the content of histamine, serotonin, kinins, prostaglandins, which have membrane toxic effects.

There is a necrosis of fabrics. Proteases accumulate that promote the spread of necrotic changes. Sensitization to protein decay products can lead to autimmune processes that deepen microcirculation disorders, tissue hypoxia, and the progress of necrotic changes.

CONDITIONS that determine the speed and extent of N propagation and its appearance.

1. The general:

Acute and chronic infections, intoxication, exhaustion, avitaminosis (zinc), anaemia, heart failure, cold, hunger, metabolic disorder, blood composition disorder.

2. Local:

• Features of the structure of the vascular system (main or branched);

• Vascular wall condition (sclerosis, inflammation);

• Degree of development of vascular collaterals in the lesion site;

• Ratio of the rate of development of circulatory disorder and inclusion of compensatory mechanisms;

• Degree of tissue and organ differentiation, metabolic activity, intensity of enzymatic processes, functional load, hypoxia resistance;

• The degree of microbial contamination;

• Level of tissue hydration and protein content.

3. Nature, strength, duration of action of pathogenic factor

• Intensity of toxin formation by microflora;

• Physical conditions: action of low temperatures (leads to blood circulation disorder), excessive warming (increase of metabolism intensity in conditions of blood supply failure), penetrating radiation.

4. Mechanism of development of a necrosis:

• straight line;

• indirect

The specific ratio of these factors predeterminates the occurrence of dry (coagulation) or wet (colliquation) N

Microscopic signs of N.

Core changes: curl (cariopicnosis), lysis (cariolysis) or decay (cariopicnosis).

Changes in cytoplasm: protein denaturation and coagulation, death of ultrastructures, break-up into clamps (plasmorexis), hydrolytic melting (plasmolysis).

Changes of intercellular space:

• Intermediate swells and dissolves

• Collagen fibers swell, soak in plasma proteins (fibrin), turn into dense homogeneous masses, disintegrate or lyse.

• Elastic fibres swell, disintegrate, melt (elastolysis)

• Reticular fibers fragment, disintegrate into clamps

• Nerve fibers - similar.

Fibrinoid necrosis is the end of fibrinotic swelling, characterized by complete destruction of connective tissue. There is usually a pronounced reaction of macrophages, increased vascular wall permeability, fibrinoid formation (a substance that consists of protein, polysaccharides of fibers that disintegrate, basic substance, and blood plasma; Required component - fibrin).

Results of a necrosis:

1. Resorption.
2. Rejection.
3. Organization - replacement with connective tissue (scar, regenerate).
4. Encapsulation - fouling with connective tissue and capsule formation.
5. Ossification is the formation of bone tissue at the measurement site.
6. Petrification
7. Cyst formation is the restriction of the cavity that is formed by tissue detritus resorption by the connective capsule.
8. Purulent melting of the source of death.

Clinic of a necrosis

Depends from:

1. Localisation (surface, deep) - brightness of visual changes;
2. Prevalence - the area where H manifestations occur;
3. Functional value of the affected organ (areas) - corresponding functional disorders;
4. The type of death is local and general symptoms.

Local symptomatology:

Direct signs - changes in colour, density and elasticity of tissues, reduction of skin temperature, movement and sensitivity disorders

Indirect signs - reactive changes around located formations, for example, signs of peritonitis in intestinal gangrene on the basis of mesenteric vascular thrombosis or rotting plevra empiema in lung gangrene.

General symptoms - intoxication, multi-organ failure.

Diagnosis in case of necrosis:

1. Symptomatology assessment: local signs, signs of malfunction of body.
2. Identification of possible cause (main disease) based on physical examination and related additional examinations

General principles of necrosis treatment

1. Treatment of a causal disease
2. Necrectomy (amputation, exarticulation, resection, extirpation).
3. Improvement of the general state of the body: detoxification, antibiotic therapy, immunocorection, corection (support) of functions of life-supporting organs and systems.

Prevention:

1. Prevention, early diagnosis and treatment of diseases that cause H;
2. Resumption of circulatory disorders (elimination of vascular spasm, stimulation of collaterals opening - novocaine blockages, spasmolytics, vascular bypass and prosthetics, thromb- that embolectomy, b/a administration of fibrinolytics);
3. If blood circulation is disturbed to limit H or prevent its development - measures to improve trofics or reduce the intensity of metabolism in them: rest, novocaine blockages to eliminate reflex spasm of collaterals.

SEPARATE TYPES OF A NECROSIS

Dry gangrene (dry N, mummification). Gangrene in literal translation from Greek is a fire.

Etiology and pathogenesis. Slowly progressing circulatory disorders (in atherosclerosis, freezing, finger burn, thrombosis.), in depleted, dehydrated patients, in tissues rich on proteins and poor on liquid leads to the development of coagulation H, which is based on protein denaturation to form insoluble compounds, which may not succumb to hydrolytic cleavage for a long time. At the same time tissues are dehydrated and do not experience rot.

Gangrene begins with peripheral parts of limbs, spreads proximal to the level of sufficient collateral circulation. A demarcation shaft is developing here on the verge of dead and viable fabrics. It is a site of development of reactive inflammation that limits dead tissue. In this area blood vessels are expanded, hyperemia, swelling occur, a large number of leukocytes are accumulated, which release hydrolytic enzymes. The demarcation shaft prevents significant absorption of toxins, and endotoxicosis does not develop.

The blood pigment impregnated dead tissues and formed their characteristic colour (dark brown, grey-yellow, black).

Microflora in this case develops poorly. But in the initial period active development of rotten flora and transition to wet gangrene is possible.

CLINIC.

Complaints - in the beginning severe ischemic pain is possible below the place of blockage (occlusion) of the artery, parestesia.

Objectively - the skin is pale, then it is marble, cold. The pulse on the peripheral arteries is not determined. Hypo - (anesthesia is more rare). Disorders of function of an extremity. Movements can be preserved (by maintaining the work of muscles that have maintained viability). The fabrics are dry, mummified, dense, dark brown, grey yellow, black with a blue hue. General symptoms are generally absent or weakly expressed.

CURRENT

Necrotic changes usually do not progress. After a long time it is possible to reject necrotic tissues. During the initial period, the active development of rotten flora and the transition to wet gangrene is possible.

TREATMENT. Necrectomy or amputation after full and clear appearance of the demarcation shaft. Before that - drying of necrotic tissues (alcohol compresses, sorbents). To prevent the progress of necrotic changes (improvement of tissue trofics) and to prevent infection (aseptic bandages, antibiotics).

Moist (putrefactive) gangrene. **Moist collikvativ) necrosis.**

Etiology and pathogenesis. It develops in case of rapid circulation disorder (embolism, vessel trauma, etc.). Favorable conditions - expressed fat fiber, pasty, low protein content and considerable amount of water in tissues (i.e. conditions favorable for hydrolysis).

Thrombosis of the trunk veins of the lower limbs with an undeveloped collateral network and preservation of arterial blood supply also leads to the development of moist gangrene (venous gangrene).

Blood stagnation, swelling, hypoxia, paralysis of precapillary sphincters, deepening of hypoxia, subsequent melting of dead tissues with proteases.

Wet gangrene is observed in necrosis of internal organs (intestine, lungs, gall bladder, appendix), which exist under conditions of sufficient hydration. It 's always wet gangrene. The available conditions (nutrient medium, humidity, constant temperature, suppression of the immune system) contribute to the life of microflora, which is actively involved in the development of the process (bacteroids, proteus).

The occurrence of secondary necrosis leads to rapid progress of the disease and prevents the development of the demarcation shaft.

Dead fabrics do not have time to dry, experience wet decay. This leads to absorption of tissue decay products and microbial toxins, development of endotoxicosis, sepsis. There is a real threat to the patient 's life.

CLINIC. Complaints of pain in the affected area. Local - sharp increase of swelling of lower limbs, pale skin. Leather - marble, cold. A visible net of bluish subcutaneous veins, dark red spots, bubbles (filled with sucrovica) may appear. Pulse is not defined. Movements are absent. Upon decay, the tissues turn into a sane grey-green mass.

Abdominal gangrene is manifested by the peritonitis clinic. In gangrene, the lungs release a large amount of curly foam sputum in the form of "meat litter."

Common symptoms are endotoxicosis symptoms and signs of functional failure of vital organs (severe general condition, dry tongue, psychosis, sluggishness, stagnation, tachycardia, hypotension, oliguria, shortness of breath, hyperthermia, anemia, leukocytosis with neutrophylesis.

CURRENT. There is rapid progress of necrotic changes, endotoxicosis, sepsis. Especially difficult course in patients with diabetes mellitus.

TREATMENT. The main element is early amputation (removal of the altered organ or part thereof) in order to save life. Amputation should be carried out at a sufficient distance from the visual boundary of dead and viable tissues, which avoids the development of secondary H and the subsequent progress of gangrene. For example, in foot gangrene on the basis of diabetic angiopathy, in the presence of regional lymphadenitis, intoxication, sepsis development, amputation at the level of the middle third of the hip is shown. In case of intestinal gangrene from above mentioned boundary retreat proximal to 30-40 cm, distal - 15 - 20 cm.

It is mandatory to carry out detoxification, antibiotic therapy, correction of functional disorders of vital organs, immunocoresis.

ULCERS

Ulcer (U) is a defect of covering and deeper located tissues, which develops as a result of rejection of dead tissues. It is characterized by chronic flow, absence or weak regeneration and resistance to therapeutic measures.

For reasons - see "Necrosis" and "Violations of regional geodynamics."

Against the background of blood supply disorders of tissues (venous hypertension and microcirculation disorders, arterial anastomoses, hypoxia, acidosis), innervation, trophics. The role of the trigger factor can be played by minor injuries (microtraumas), long-term compression, microbial factor, auto-immune processes and allergies (body proteins acquire AG-properties, auto-AT is produced, immune complexes are formed, which block T- and B-lymphocyte systems and involve a local immunological reaction in the form of leukocyte infiltration and plasmocytes of paravasal tissues). In some patients, the cause cannot be determined. Contributing and supporting factors are anemia, diabetes mellitus, hypoproteinemia.

Kinds of ulcers:

1. Ulcers in chronic venous insufficiency (postthrombotic, varicose, congenital venous dysplasia)
2. Ischemic ulcers (atherosclerosis, endarteritis, diabetic angiopathy, hypertension-ischemic ulcers - Mortarel syndrome).
3. Ulcers caused by congenital and acquired arteriovenous compounds (post-traumatic arterial-venous fistulas, congenital arterial-venous fistulas in Parks Weber syndrome)
4. Post-traumatic ulcers (burns, sores, bedding, scalped wounds)
5. Neurotrophic ulcers (N.U. injuries and diseases)
6. Ulcers on the basis of common diseases (collagenosis, diseases of exchange, blood, syphilis, FA, diseases of internal organs)
7. Ulcers on the soil of the local infectious, mycotic and parasitic process (phlegmon, necrotic form of oral inflammation, fungiotic, parasitic).

Current phases:

1. Pre-ulcer state - dystrophic tissue changes.
2. Necrosis and inflammation of skin and adjacent tissues.
3. Formation of defect

4. Cleaning and regeneration
5. Epitelization and scarring

Morphology. The bottom is formed by granulations which can be coated with fibrin, detritus. The granulation shaft passes into a layer of hard connective tissue. There is non-specific inflammation around the ulcer.

Clinic. Dimensions from microscopic (corneal ulcer) to 10-20 cm in diameter (tibia ulcer in venous pathology of lower limbs). The edges of the ulcer can be flat, dug, soft, hard (caloric), crater-shaped. The bottom can be pale, hyperemized, sebaceous (at syphilis), coated with granulations, fibrin, detritus. The secretions can be serous, purulent, hemorrhagic. In case of progress of tissue decay there are so-called phagedenic ulcers, in case of death - gangrenous. Sometimes the ulcer can have a characteristic appearance: in case of syphilis - upper third of the tibia, sebaceous bottom, clear edges; At TV-flat surface; At Ca - crater-shaped, with hard edges.

Complication of ulcers

• Eczema, pyodermia, cellulitis, mycosis, rye inflammation, skin and subcutaneous fiber indentation
• Thrombophlebitis, secondary lymphedema
• Bleeding (arrosia of bottom vessels)
• Perforation of hollow organ ulcers (e.g. stomach), penetration into surrounding organs (e.g. DPC ulcers into pancreatic head)
• Organ function disorder due to ulcer healing due to the formation of a hypertrophic scar (pilorostenosis, ankylosis, and contraction in case of burn ulcers)
• malignization

Treatment

1. Eliminate the causes of ulcer:
- Safenectomy - with varicose expansion of subcutaneous veins;
- Treatment of specific infection
- Gastric resection together with ulcer

2. To create conditions conducive to regeneration:
- Bed mode with elevated leg position (elimination of blood stagnation and lymph);
- Careful washing of the skin around the ulcer;
- Provision of tissue fluid outflow from ulcer into bandage (osmotic active substances);
- Infection control (local - antiseptics).
- Ulcer purification from necrotic tissues and fibrinose laminations (protease, CD, laser);
- After cleaning the ulcer from necrosis - bandage with neutral ointments 1 times every 3-6 days;
- Presence of hypergranulations (silver nitrate - lapis);
- balanced diet;
- LFK
- immunomodulators

- Operational closing of cover defect

Methods of surgical closure of ulcers

Preparation. Purification of ulcers from necrosis, prevention of purulent process, release of ulcer from pathologically changed granulations and scars, which prevent blood supply of ulcer bottom and walls.

Plastic skin closure of defect. Flaps (pieces) on the leg or free plastic are used.

Way Yatsenko-Reverdena. Skin pieces of 0.3-0.5 cm diameter are taken from external surface of hip, shoulder or abdomen and skull-like laying on defect surface.

Way Tirsha. Take up skin tapes that include the epidermis and tips of the nipple layer. Their length depends on the size of the defect, the width is not more than 3 cm.

Lawson-Krause's way. Significant epidermis transplants, which are fixed by sutures to the edges of the defect.

Way of Douglas. Punching of circles on stretched skin at a distance of 1.5 cm. The flap is prepared, leaving circles on maternal soil. The screen flap is fixed to the defect edges.

DECUBITUSES (decubitus)

Bedding is an ulcerative necrotic process that occurs in weakened patients in areas that experience constant pressure.

Etiology and pathogenesis. The main reason is external (with respect to the affected area) compression (exogenous beds) or neurotrophic tissue changes (endogenous beds). Compression causes circulation disorder (ischemia) and subsequent necrosis. Bedding is a form of gangrene.

In neurotrophic changes (disorder of trophic innervation), the trofics, metabolism of tissues changes, which also leads to the development of necrosis.

Factors that contribute to the development of poverty

1. Subcutaneous fiber atrophy contributes to increased pressure on protruding parts of the body.
2. The low mobility of the seriously ill contributes to constant pressure on the same areas of the body.
3. General hemodynamics disorders due to the underlying disease.
4. Metabolic frustration.

An example is pressing a gypsum bandage, a part of the body, a prosthesis, a foreign body on the vessel, a stone on the wall of the gallblthe, uncomfortable shoes.

Current phases:

1. Blood supply and trofics disorders.
2. skin necrosis.
3. formation of defect.

CLINIC. The skin is pale at the beginning, then cyanotic, swelling. Further - peeling of epidermis and formation of blisters. Next, skin necrosis. After its rejection, a defect is formed with the bottom covered with purulent-necrotic masses (detritus). Further, dystrophic granulations (pale, fine-grained, dull) are possible.

Typical localizations are the area of the sacral, the sharp processes of the vertebrae, the angles of the blades, the back of the head, the elbow processes, the anterior upper bone

of the ilium, the patella, the heel, the anterior surface of the tibia (the anterior edge of the large tibia).

TREATMENT - according to the rules of treatment of purulent-necrotic processes.

Prevention.

1. Prevention of prolonged compression: inflatable circles, sack bags, flax, frequent change of body position, sectional pneumatic mattresses.

2. Careful care of the patient and his skin (washing, straining with camphor or salicyl alcohol)

3. Clean

FISTULAS

Fistula is a pathological stroke that connects organs, body cavities, or deeper located tissues to the external surface of the body or to the cavity of the internal organ. Products of pathological process (gnoma, sequester), secret or content of corresponding organ (gastric juice, bile, urine, pancreatic juice) are separated through fistula.

Classification

By origin:

1. **Congenital** (based on and due to developmental defects, persisting some germ epithelial formations, obliterating normally) - middle and lateral neck fistulas, umbilical fistula, hoof fistula.

2**. Acquired**:

- Caused by pathological process (mastitis and other purulent processes, FA, foreign bodies, ligatures, destruction of tissues by tumor and its decay (urinary-uterine fistula in case of uterine chancellor)
- Fistula caused by trauma (intestinal, bronchial)
- Artificial fistula (cecostoma, sigmostoma in case of rectal injuries; Cystotome - in case of urethra injury).

With respect to the external environment:

• External - fistula between hollow organs, pathological cavities of soft tissues, bones with external environment. The external fistula of the gastrointestinal tract can be complete (the entire contents of the respective cavity are released outside) and incomplete (some of the contents are evacuated naturally). In case of purulent fistula, the complete one is considered to connect the purulent cavity, both with the external environment and with the cavity of the internal organ (for example, the complete pararectal fistula).

• Internal - connection between 2 bodies (inter-organ); Between the organ and the body cavity (e.g., jelly-pleural); Between the organ and the pathological focus (incomplete internal purulent fistula).

<u>In structure:</u>

• Tubular - with a clearly defined channel, which is usually expelled by granulations (the exception being epithelial penny moves);

• Spongy - practically without fistula canal with direct transition of epithelium of hollow organ mucosa to skin (hole of hollow organ into external environment). These fistulas are always epithelized.

Depending on the nature of the tissue lining the fistula:

• granulating

• epitelised

By the nature of excretions: milk, salivary, potassium, purulent, liquor, bile, intestinal, urinary, mucous.

Etiology and pathogenesis

Congenital fistula develops due to the persisting of some germ epithelial formations obliterating normally (neck fistula, umbilical cord, hoop). In some cases, they are formed primarily (with complete non-growth). In partial non-growth, the secret accumulates into within the formation. The cyst is formed. After a critical increase in pressure in the cyst cavity (secret accumulation, nudity), a break out occurs. At that fistula is formed.

Acquired fistula occurs in case of adverse course of pathological process:

- Inflammation, especially purulent;
- Presence of foreign bodies (ligatures)
- Tumor (decay or germination into adjacent organs and tissues)
- Injury and surgery (failure of sutures)
- Tissue trofics disorder, necrosis, undelivered sequester

At the same time, tissue destruction occurs simultaneously, and internal tissue pressure increases (due to limited accumulation of pus, detritus, secret). The strength and resistance of the tissues is reduced and the accumulated liquid breaks out or into the body (organ) cavity

Factors that prevent fistula healing:

1. Constant organ secretion across the fistula canal.
2. Irritating effect of reactive secrets on granulation (intestinal, gastric and pancreatic fistulas)
3. Not bought purulent-necrotic process, not remote sequester. Irritating effect of microbial toxins and decay products or suppression of granulation growth. Continuous expiration of exudate.

In epithelial fistula, the resumption of cover integrity prevents the possibility of infection. In granulating fistulas, in the event of the destruction of granulations and the disruption of their normal growth, there are conditions for the penetration of toxins and microbes into the surrounding tissues. This leads to the development of reactive inflammation, a large number of scars around the canal and when the body weakens - purulent infection. (generalization of the last is possible).

Embryonic passage lined with epithelium releases mucus (endodermal formations) or skin lard (ectodermal formations).

Clinic. Complaints of discomfort, pain, weakness are predetermined by localization, the nature of the excretions and its number and the disruption of the function of the relevant organ. Objectively: topically - external hole of different diameter and shape, isolation (of different character and quantity), possible maceration and irritation of skin, dermatitis as a result of irritating action by juices of digestive organs, dense swelling of surrounding tissues (up to ivory - in case of urinary fistula).

Common symptoms are purulent intoxication (in case of insufficient outflow by purulent fistula), disorders of protein, water-salt metabolism in case of high fistula of the gastrointestinal tract, disorder of organ function when it is filled with non-natural secrets (food in bronchia in case of bronchoposkeletal fistula), secondary infection through fistula.

The course is long, without a tendency to self-heal epithelial fistulas (granulating fistulas can self-heal when the irritant is terminated).

Diagnostics:

1. Record of complaints, history, number and nature of allocations
2. Fistulography - to clarify the structure (iodylipol)
3. Chemical isolation study - HCl content
4. Samples with dyes (methylene blue, idigocarmin) to clarify fistula relation to certain structures)

Treatment. The main method of treatment is operative. Its basic principle is to cut within healthy fistula tract tissues and eliminate the causal factor. Examples:

1. Excision with pathological embryonic formation
2. Intestinal resection that carries fistulas
3. Resection of the part of the pancreas that supports fistula
4. Removal of sequester or bone resection
5. Removal of foreign body (ligature)
6. removal of necrotic tissues

Skin care around the outer opening is of particular importance. Lassar paste is often used to protect her from the aggressive effects of fluid that is released through the fistula. In case of intestinal fistula, calodant is used. In case of incomplete fistula of gastrointestinal tract obturators are used.

In terms of preparation for the operation - fistula rehabilitation. General treatment (correction of metabolic disorders).

ARTIFICIAL fistulas

Some authors (E.N. Wantzian, 1990) believe that the channel created by operational means to divert secret or excreta is wrong to call fistula.

Classification

1. In relation to the external environment:

- External or with t o m and (lat. stoma - hole) - holes that connect the cavity of the organ with the external environment. Examples are gastrostoma, cecostoma, sigmostoma, anus praeter naturalis, cholecystostoma, tracheostoma;

Internal or anastomoses are sufficiently broad inter-organ compounds in which the mucous membrane of one organ passes directly into the mucous membrane of another organ. An example is gastroenteroanastomosis, ileotransverzoanastomosis.

2. Depending on the purpose of formation:

- Temporary (tracheostoma in case of brain injuries, sigmostoma in case of operations for rectal injuries
- Permanent (if it is impossible to eliminate the cause that forces formation of such fistula - sigmostoma in case of inoperable tumor of rectosigmoid transition or impossibility to resume natural passage).

Purpose - resumption of organ function (passage of food along the gastrointestinal tract, breathing - tracheostoma)

- Withdrawal of hollow organ contents (cystotome in case of urethra injury, sigmostoma in case of rectal injury).

Closing of artificial fistula is carried out according to general rules of fistula treatment.

Temporary tubular fistulas may close on their own when the contents are no longer released therethrough**.**

SEPARATE TYPES OF FISTULAS

Congenital fistulas.

MEDIAL neck fistulas are formed due to an abnormality of thyroid development from the residues of tractus thyreoglossus. (Epithelial cord, which goes from foramen coecum pagan to the body of sublingual bone and further down between the lateral lobes of the thyroid gland). Thyroid isthmus is formed from it.

Clinic: the external hole is located exactly along the middle line, releases mucus.

Treatment - prompt (excision of fistula with sublingual bone section)

LATERAL (gill) neck fistulas - remains of gill furrows.

Embryology - gill arcs (4 on each side) and membranes are located between adjacent pairs, which form gill furrows, expelled from the outside and from the inside by epithelium. The preservation of their residues may give rise to the formation of gill (lateral) neck fistulas.

UMBILICAL FISTULAS

Reasons:

- Incomplete obliteration of the yellow duct (ductus omphalo-entericus), which leads to the formation of an intestinal fistula with an external opening in the umbilical area (Meckel diverticle is formed at incomplete growth).

- Incomplete urachus obliteration (URACHUS), which leads to the formation of bladder fistula with an external opening in the umbilical area (incomplete germination entails the formation of bladder diverticle).

The structure is a tubular epithelial fistula.

Clinic - external hole of fistula in the area of umbilical cord, urine or intestinal content is released.

Diagnostics - clinical data, history, fistulography.

Treatment is quick.

SMOKED FISTULAS (Epithelial Smoked Moves).

They are covered with epidermis, contain sebaceous glands secret, hair and secondary nudity products.

FOREIGN BODIES (FM)

IT is bodies of non-biological origin that are in tissues, organs or cavities of the body.

Circumstances of hit of IT:

1. Trauma is fragments of an object that damages.

2. Operation

- Permanent implants (artificial organs): prostheses of heart valves, vessels, ligatures
- Temporary implants (drains, vascular catheters)
- Careless leaving of napkins, tools

3. Conscious or unconscious actions of the patient (foreign bodies of the gastrointestinal tract, respiratory tract, urethra and bladder).

Classification

1. By the circumstances of the hit: traumatic, yatrogenic, autogenic.

2. On a contrast x-ray: contrast and low-contrast.

Pathological anatomy. Inflammation. Encapsulation. Migration. Thrombosis.

Clinic. Pain. Inflammation symptoms. Disorders of organ and system function (circulatory disorder, peritonitis).

Diagnostics. Taking into account complaints, circumstances of injury, history of disease and life. Radiological inspection. Endoscopy.

Treatment. The main method is operational.

Observation of IT progress along the gastrointestinal tract.

Removal (endoscopic) from the gastrointestinal tract, respiratory and urinary tract.

Alone, single IT up to 10 cm long can leave the gastrointestinal tract. Appearance of clinical manifestations after "light interval," standing IT without movement - indication to surgery. Perforation of the hollow organ, occurrence of bleeding, infiltration or abscess of the abdominal cavity - indication for urgent operation.

6. Materials for self-control.

1. To master the technique of identifying areas of necrosis , gangrene , ulcers , fistulas.
2 . Make a differential diagnosis between different type of necrosis, gangrene , ulcers , fistulas.
3. Determinate method of anesthesia and the method of surgical intervention in various forms of necrosis , gangrene , ulcers , fistulas

6.1. Tasks for self-control .

question:

1. Causes of necrosis , gangrene , ulcers , fistulas .
2 . Classification of necrosis , gangrene , ulcers , fistulas .
3 . Clinical signs of necrosis , gangrene , ulcers , fistulas .
4. Diagnostic features of certain types of necrosis, gangrene, ulcers, fistulas.
5. Current views on the phase-necrotic ulcer process.
6. Methods of necrosis, gangrene, ulcers, fistulas.
7. Principles of diagnosis and treatment policy for foreign bodies.
8. Specific Methods of dressings for ulcers, fistulas.
9. Methodology care bed sores, prevention of pressure sores.
10. Methodology of fistulography.
11. The methodology of the sample with the use of dyes.

6.2. Situational problems.

Tests in the volume "KROK 1" and "2 KROK."

6.3 Tests for self-control (basic knowledge)

Tests and testing task source of knowledge .

1. The cause of the gangrene of the lower extremity :
A fracture of the femur
B. Dislocation of the hip
B. main artery thrombosis
G. Congenital hip dysplasia
D. Rheumatoid arthritis

2 . The cause of the gangrene of the lower extremity :
A. Dislocation of the hip
B. Fracture of the femoral
B. main artery embolism
G. Congenital hip dysplasia
D. Rheumatoid arthritis

3 . The cause of the gangrene of the lower extremity :
A fracture of the femur
B. Dislocation of the hip
B. Trauma main artery
G. Congenital hip dysplasia
D. Rheumatoid arthritis

4 . Dry gangrene is characterized by:
A. Development of putrid infection , the presence of a line of demarcation
B. the presence of the demarcation line , intoxication
B. Development of putrid infection , intoxication , mummification
, The development of putrid infection , the lack of toxicity
D. the presence of the demarcation line , the absence of intoxication, mummification

5 . Reasons for wet gangrene of the lower extremity :
A. The rapid development of circulatory disorders regionarnonogo
B. Obesity
B. Putrid infection
G. arteriosclerosis obliterans
D. The gradual development of regional circulation disorders

6. Clinic wet gangrene include:
A swelling of tissues, skin discoloration , no line of demarcation
B. Intoxication , tissue swelling , discoloration of the skin, the lack of a line of demarcation , putrid smell
B. A clear line of demarcation , putrid odor , intoxication, tissue edema
G. putrid smell , no demarcation line , the mummified diseased tissue
D. putrid smell, mummified affected tissues , the lack of toxicity , lack of sensation in the affected area

7. The causes of venous ulcers :

A thermal injuries
B. violation of innervation
B. decrease in blood volume
G. bowel dysfunction
D. fracture of the limbs

8. The causes of venous ulcers :
A thermal injuries
B. violation of arterial hemodynamics
B. decrease in blood volume
G. bowel dysfunction
D. fracture of the limbs

9. The causes of venous ulcers :
A thermal injuries
B. violation of venous hemodynamics
B. decrease in blood volume
G. bowel dysfunction
D. fracture of the limbs

10 . Basic principles of treatment of venous ulcers :
A. Vissichennya ulcer debridement , stimulation of regeneration,
B. Vissichennya ulcer debridement , stimulating regeneration , pathogenetic treatment
B. Vissichennya ulcers , stimulating regeneration , debridement , plastic ulcer
D. Stimulation of regeneration, pathogenetic treatment , debridement , plastic ulcer
D. The use of spa treatment

11. The method of treatment of colonic fistula gubopodibnih
A conservative
B. filling
B. diathermocoagulation fistula
G. intestinal resection loop carrier fistula
D. ligation on the course of the fistula

12. In which the surgical treatment of debridement is performed on the basis of direct evidence:
A. The primary
B. Secondary
B. Early
G. Late
D. delays

13. Based on what evidence necrectomy spend in the primary surgical treatment of wounds
A. direct

B. Late
B. Indirect
G. Backlink
D. Laboratory

14. Based on what evidence spend necrectomy in secondary surgical treatment:
A. Direct
B. Late
B. Indirect
G. Backlink
D. Laboratory

15. The most common form of purulent necrotic lesions of the foot in diabetes mellitus:
A. Boil
B. Carbuncle
B. Cellulitis
D. After injection abscess
D. Chemical burn

16. The most common form of purulent necrotic lesions of the foot in diabetes mellitus:
A. Boil
B. Carbuncle
B. Gangrene of fingers
D. After injection abscess
D. Chemical burn

17. The most common form of purulent necrotic lesions of the foot in diabetes mellitus:
A. Boil
B. Carbuncle
B. neuropathic ulcer
D. After injection abscess
D. Chemical burn

18. What causes the risk of foreign bodies :
A. Development lymphostasis
B. Encapsulation
B. Malignization
G. The development of inflammation and infection
D. Development of bleeding

19. The most informative method for studying fistula:
A. fistulography
B. dye sample
B. endoscopy
G. sensing fistula

D. plan radiography

20. Measures for the prevention of pressure sores include:
A. wiping "dangerous" areas of the patient 5 % alcoholic solution of iodine
B. wiping "dangerous" parts of the patient 10% solution of camphor alcohol
B. Application to the places of possible defeat alcohol compress
G. provide the patient a permanent position at the back
D. Application to the " dangerous " sites warmers

Case studies for the source of knowledge

The patient , 38 years after appendectomy of scar observed skin defect to 4 mm in diameter, which is connected to swing coated granulations . After it is released to 50 ml precipitates night . In conducting the course was fistulography diameter and 3 mm in length and 4 cm in which the contrast enters the cecum , well it fills . Violations of the motor- evacuation function of the intestinal tract is not marked. Install the diagnosis and suggest further treatment .

The patient, 78 years old, underwent surgery for cancer of the cecum : eksterpatsiya rectum to form a single- rear unnatural . The entire intestinal contents is allocated to the anterior abdominal wall.

Give a full description of the intestinal fistula. Determine the tactics of the patient.

The patient , aged 68, in the area of the inner ankle right leg there is a defect of the skin, a rounded shape with a diameter of 4 cm , depth 4.3 mm , the defect is filled with granulation , peripheral epithelialization not. The appearance of the defect patient notes 6 months later after suffering microtrauma . Thickened skin around the defect , bluish-brown. Subcutaneous veins of legs and hips twisting , irregularly dilated.

Determine the nature of the defect and described the tactics of his treatment .

The patient, 68 years old , says chilly feet, of pain in the calf muscles when walking about 50 meters. Pulse on the popliteal arteries and the arteries of the foot is missing . In the area of the lateral malleolus defect rounded, large diameter 3 cm , depth 2 mm. The bottom of the defect is made dull granulation , covered with fibrin. Determine the cause of the origin and nature of the defect described . Specify tactics examination and further treatment of the patient .

A patient with a mental disorder has swallowed some coins (50 cents) . Complaints does not deliver . Breathing is difficult. No cough . Belly swollen, symmetrical , painless , soft, involved in the act of breathing. Peristaltic usual noises . Determine the tactics of the patient.

Patient V. , 77 years , 1 day after surgery was performed - amputation of the left leg over the wet gangrene. The patient's condition serious. Independently she can not move . On examination found flushing of the skin in the areas of both angles of the blades ,

sacrococcygeal region. What complication can occur in this patient ? What should I do in this situation?

Patient M. , 73 years old, is in a supine position in the sacrococcygeal region there is flushing of the skin , blisters available with bloody contents . What complication developed in this patient , we need to do in this case?

Tests III level of difficulty

A patient of 81, complained of a sharp pain in his stomach. The pain came suddenly after 2 hours after meals. Gastric lavage and cleansing enemas have brought no relief . The patient has coronary artery disease , 5 months after myocardial infarction . Patient's condition is severe , pale, cyanotic mucous membranes , acrocyanosis . Suffocation. Pulse - 100 punches in 1 minute , atrial fibrillation . Uniformly distended belly , soft , with deep palpation - painful . Percussion - high tympanitis , peristalsis is dramatically weakened. Peritoneal signs are not defined . Per rectum - the remnants of feces with mucus and dark blood . What is the likely diagnosis and what will be the treatment strategy ?

The patient S., 72 years brought to the emergency room in serious condition with complaints of abdominal distention, pain without clear localization within three days. The general condition is serious , skin and visible mucous membranes pale . Tongue dry . Pulse - 98 beats in 1 minute, blood pressure 100/60 mm Hg. Art. The abdomen is swollen , painful in all areas, defined fuzzy signs of peritoneal irritation . Auscultation - peristaltic noises do not listen. PER rectum - ampoule of the rectum is empty, on the glove - traces blood - mucous secretions . The most likely initial diagnosis ? The optimal method of treatment?

A patient 47 years old complained of a rise in temperature to 38 ° C , a pain in the neck , the presence of edema. When viewed from the back of the neck is determined by the infiltration of 7x5 cm , the skin underneath the purple- black, in the center - a lot of the fistula with necrotic content. Determine the diagnosis and treatment strategy .

The patient , 76 years old , was admitted to the surgical department with complaints of pain in his left foot and lower leg. For 12 years, sick atherosclerosis . About 2 weeks ago, suddenly there was a strong pain in my left foot. Medical care is not addressed . Increased rapidly swelling. The skin was pale in the beginning , then - marble , cold to the touch . There are bubbles exfoliation of the epidermis filled with ichor . Pulse on the popliteal artery or arteries of the foot is not defined . Motions are absent . Body temperature - 37.8 ° C , heart rate - 100 in 1 min, blood pressure - 90 /50 mm Hg . Determine the diagnosis and treatment strategy .

A patient 65 years old , 4 years ago, moved appendectomy . In the area of the scar there is a defect sheets (hole) diameter of 1.5-2 mm, through which allocated about 2 ml of yellowish turbid contents day. The skin around this defect is not changed. Palpation painless. Edema , infiltration of surrounding tissues do not.

Determine the necessary additional testing .

1. The modern view of the etiology , pathogenesis, clinical and morphological characteristics of gangrene , ulcers , fistulas , foreign bodies of the body.
2. The clinic, diagnosis gangrene , ulcers , fistulas , foreign bodies of the body.
3. Diagnosis and monitoring of the wound process in gangrene , ulcers , fistulas , foreign bodies of the body.
4. Modern principles and methods of treatment.
5. Types of wound healing after treatment of gangrene , ulcers , fistulas , foreign bodies of the body.
6. Technique of surgical treatment of gangrene , ulcers , fistulas , foreign bodies of the body.
7. General characteristic of the drugs that are used for the topical treatment and prevention of infectious complications.
8. Treatment of gangrene , ulcers , fistulas , depending on the phase of wound healing .
9. Preventive measures for the further progression of the disease .
10. Transport the patients to the dressing .
11. Laying according to the patient's dressing table area of the manipulation.
12. Preparation of hands to perform the procedure.
13. To be able to put on a sterile gown .
14. To collect anamnesis of patients .
15. To evaluate the results of laboratory research methods and plan further investigation
16. A plan of treatment a particular patient .
17. Drawing up of a landmark epikrisis .
18. Development of primary documentation (history).
19. Technique of the necrectomy .
20. Prepare a kit for washing ulcers.
21. Prepare a kit for performing ligation .
22. Specimen collection for bacteriological control .
23. Different types of cleaning in contaminated dressing .
24. Imaging technique of foreign bodies.
25. Methods for closure of ulcers of the skin.
26. Determine the prevalence of necrosis.
27. Probing fistula.
28. Preparing the patient for fistulography .
29. Manufacturing tables and other illustrative material (photos , slides, drugs) .
30. Disposal of dressing.
31. An analysis of archival material.

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rating	Points
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3 (satisfactory)	3
2 (poor)	0

Guidelines prepared
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