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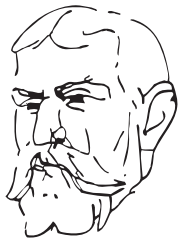

Memory of
dr Władysław
Biegański

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CONTENTS

EDITORIAL ARTICLES
REVIEW ARTICLES

Viacheslav M. Zhdan, Iryna A. Holovanova, Maksym V. Khorosh, Inna V. Bielikova, Natalia A. Lyakhova
ANALYSIS OF THE LEGISLATIVE ACTIVITY OF THE MINISTRY OF HEALTH OF UKRAINE IN THE CONDITIONS OF THE RUSSIAN-UKRAINIAN WAR IN 2022 1425

ORIGINAL ARTICLES

Ruslana M. Orlovska, Vasyl I. Popovych, Ivanna V. Koshel, Olexandr O. Bondarenko
FEATURES OF THE CONNECTIVE TISSUE COMPONENT OF THE PALATINE TONSILS IN PATIENTS WITH RECURRENT TONSILLITIS 1434

Oleksandr Yu. Ioffe, Stepan L. Kindzer, Mykola S. Kryvopustov, Yuri A. Dibrova, Yuri P. Tsiura, Mykhailo O. Havrylchenko, Oleg V. Lobanov
SURGICAL ISCHEMIC ASPECTS OF COVID-19: MANAGEMENT OF PATIENTS WITH COVID TOES AND FINGERS 1439

Grygoriy P. Griban, Olena O. Kosheleva, Olena O. Mitova, Alla M. Harlinska, Olena V. Solodovnyk, Bogdan S. Semeniv, Kostiantyn V. Prontenko
PHYSICAL DEVELOPMENT OF STUDENTS AS AN INDICATOR OF THE PHYSICAL EDUCATION SYSTEM FUNCTIONING IN THE EDUCATIONAL INSTITUTION 1446

Yulia G. Kolenko, Tetiana O. Timokhina, Olesya V. Lynovytska, Konstantin O. Mialkivskyi, Nina S. Khrol
EPIDEMIOLOGICAL SITUATION OF PRE-CANCER DISEASES OF THE ORAL MUCOUS IN UKRAINE 1453

Elżbieta Ewa Szczygieł-Pilut, Anna Zajączkowska-Dutkiewicz, Daniel Pilut, Jarosław Dutkiewicz
HYPERAMMONAEMIA AND COGNITIVE IMPAIRMENT IN EPILEPSY PATIENTS TREATED WITH VALPROIC ACID – PRELIMINARY STUDY 1459

Galina A. Yeroshenko, Alona S. Grygorenko, Kostyantyn V. Shevchenko, Olga D. Lysachenko, Nataliia T. Maksymenko, Angela V. Vatsenko, Olena V. Klepets
THE FEATURES OF THE NORMAL ULTRASTRUCTURE OF THE RAT DUODENUM AND UNDER THE COMBINED EFFECT OF THE FOOD ADDITIVES COMPLEX 1466

Inna V. Baranets, Nataliya G. Pakhomova, Ivan M. Okhrimenko, Olha H. Hubar, Valentyna S. Padun, Larysa V. Drozd, Iryna A. Holovanova
COMPREHENSIVE APPROACH IN CORRECTIONAL WORK WITH OLDER PRESCHOOL CHILDREN WITH SPEECH DISORDERS 1471

Liliia V. Burya, Nataliia V. Moisieieva, Anna A. Kapustianska, Andrii V. Vakhnenko, Iryna M. Zviagolska, Mariia O. Rumiantseva
NEUROMETABOLIC STRATEGY OF PHARMACOTHERAPY FOR PATIENTS WITH SERONEGATIVE ARTHRITIS 1477

Mariia V. Kryvopustova
PREDICTION OF ASTHMA CONTROL STATUS IN SCHOOL-AGE CHILDREN SENSITIZED TO CAT ALLERGENS 1481

Maksym M. Potyazhenko, Tetiana V. Nastroga, Nina L. Sokolyuk, Oksana Ye. Kitura, Nelya M. Motorna, Angelina S. Korpan
EFFICIENT COMPREHENSIVE TREATMENT OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE EXACERBATION AND POSTCOVIDAL SYNDROME IN ELDERLY PATIENTS 1486

Volodymyr H. Hryn, Vitaliy S. Drabovskiy, Dmytro A. Sytnik, Roman M. Ryabuschko, Mykola M. Riabuschko, Serhii M. Bilash, Bohdan I. Gonzhak
PECULIARITIES OF MORPHOETIOPATHOGENESIS OF ACUTE APPENDICITIS AND CONSEQUENCES AFTER APPENDECTOMY 1492

Ivan M. Okhrimenko, Victoria A. Shtykh, Hanna L. Boiko, Yurii V. Novytskyi, Olha M. Pasko, Liudmyla M. Prudka, Tetyana V. Matiienko
CADETS' PHYSICAL HEALTH AND PSYCHO-EMOTIONAL STATE DURING COMBAT SPORT TRAINING 1500

Igor M. Skrypnyk, Ganna S. Maslova, Tetiana V. Lymanets, Roman I. Skrypnyk
THE OVERWEIGHT AND OBESITY ROLE IN THE OCCURRENCE OF CHEMOTHERAPY-INDUCED HEPATOTOXIC REACTIONS IN PATIENTS WITH ACUTE MYELOID LEUKEMIA 1506

Oksana K. Kornosenko, Iryna V. Taranenko, Yevheniia Yu. Shostak, Yuliia V.Zaitseva, Oleksandr V. Petryshyn
PROJECTING OF COMPLEX HEALTH TRAINING FOR MATURE MEN WITH METABOLIC SYNDROME 1512

Andrii A. Rebryna, Anatolii A. Rebryna, Natalia A. Lyakhova, Iryna I. Shaposhnikova, Svitlana M. Korsun, Liudmyla V. Shuba, Victoria V. Shuba
ANALYSIS OF STUDENTS' HEALTH INDICATORS IN THE COURSE OF PHYSICAL EDUCATION CLASSES WITH A SPORTS FOCUS 1519

Haliia M. Mustafina, Ivan I. Starchenko, Boris M. Fylenko, Volodymyr M. Koka, Valentyna V. Cherniak, Nataliia V. Roiko, Serhiy A. Proskurnya
MORPHOLOGICAL FEATURES OF THE LIVER PARENCHYMA IN THE EXPERIMENTAL SUPPLEMENTATION OF RATION WITH THE FOOD ADDITIVES 1525

Oksana V. Kononova, Tetiana O. Timokhina, Anatoliy V. Borysenko
CHANGES IN IMMUNOLOGICAL INDICATORS UNDER THE INFLUENCE OF TREATMENT OF GENERALIZED PERIODONTITIS IN PATIENTS WITH MANIFESTATIONS OF PSYCHOEMOTIONAL STRESS 1529

Valery G. Arefiev, Viktor G. Riabchenko, Volodymyr M. Piddiachyi, Iryna V. Zenina, Mariia A. Redkina, Serhii M. Novik, Vasyl D. Hohots
APPROPRIATE LEVELS OF PHYSICAL CAPACITIES DEVELOPMENT IN ADOLESCENTS WITH DIFFERENT STATE OF HEALTH 1534

Vitalii I. Liakhovskyi, Arkhyp V. Sydorenko, Roman M. Riabushko
COMPLICATIONS AND EVALUATION OF QUALITY OF LIFE IN PATIENTS AFTER LESS-INVASIVE TREATMENT OF VARICOSE VEINS OF THE LOWER EXTREMITIES 1540

Volodymyr Hryn, Yurij Kostylenko, Viktoriia Pinchuk, Valeriy Zhamardiy, Viktoriia Donchenko, Olha Honchar, Oksana Hordiienko
EXPERIMENTAL ANALYSIS OF WAYS OF VIRAL INFECTIONS INTO THE HUMAN BODY 1544

Ihor V. Ksonz, Denys V. Khmilevskyi, Ievhen M. Grytsenko
PROGRAMMED LAPAROSCOPY IN THE OF DIFFUSE APPENDICULAR PERITONITIS TREATMENT OF CHILDREN 1550

Roman I. Skrypnyk, Ganna S. Maslova, Igor N. Skrypnyk
THE EFFECT OF S-ADEMETIONINE ON PLASMA CITRULLINE LEVEL DURING CHEMOTHERAPY-INDUCED OXIDATIVE STRESS IN PATIENTS WITH CHRONIC LYMPHOPROLIFERATIVE DISORDERS 1553

Serhii M. Bilash, Svitlana V. Donchenko, Olena M. Pronina, Mykhailo M. Koptev, Yaryna O. Oliinichenko, Valentyna V. Onipko, Volodymyr I. Ischenko
MORPHOMETRIC FEATURES OF THE ELEMENTS OF THE HEMOMICROCIRCULATORY BED IN THE CORTEX OF THE ADRENAL GLANDS INFLUENCED BY THE FOOD ADDITIVES COMPLEX 1558

Sergei O. Guryev, Olexiy S. Solovyov, Dmitriy M. Lysun, Natalia I. Iskra, Vitaly A. Kushnir, Andriy I. Tsvyakh, Nataliia O. Marchenkova
RISK-ORIENTED ANALYSIS OF LIMB LOSS IN VICTIMS OF MODERN HOSTILITIES 1564

Liudmyla I. Voloshyna, Oleksandr A. Toropov, Ihor V. Boyko, Pavlo I. Yatsenko, Dmitriy V. Steblovskyi, Olha P. Bukhanchenko, David S. Avetikov
COMPARATIVE CHARACTERISTICS OF CLINICAL INDICATORS OF THE CONDITION OF SCAR TISSUE OF THE FACIAL SKIN AT DIFFERENT STAGES OF THE POSTOPERATIVE PERIOD DEPENDING ON THE CHRONOTYPE OF THE PERSON 1569

REVIEW ARTICLES

Valentin N. Dvornik, Nataliia N. Deviatkina, Roman N. Opatsky, Vyacheslav V. Garkusha, Rostislav Yu. Molchanov
PECULIARITIES IN PERFORMING MEDICAL EXAMINATION TO ASSESS THE EXTENT OF ALCOHOL INTOXICATION OF DRIVERS ACCORDING TO THE LEGISLATION OF UKRAINE AND SOME OTHER COUNTRIES 1573

Anna V. Fastivets, Anatoliy V. Emetc, Yevheniia O. Skrinnik, Oleksandr V. Petryshyn
INTRODUCTION OF INNOVATIVE TECHNOLOGIES IN EDUCATIONAL PROCESS OF FUTURE SPECIALISTS IN PHYSICAL THERAPY AND ERGO THERAPY 1578

Volodymyr Hryn, Kseniia Yudina, Viktoriia Donchenko, Valeriy Zhamardiy, Viktoriia Horoshko
EPONYMOUS TERMS IN THE MORPHOLOGY OF ANGIOARCHITECTONICS OF THE HUMAN HEAD 1583

Oksana I. Krasnova, Iryna A. Holovanova, Svitlana M. Tanianskaia, Tetiana V. Pluzhnikova, Mariia O. Rumyantseva, Oleh H. Krasnov
STRATEGY FOR THE DEVELOPMENT OF PEDIATRIC SERVICE AT THE LEVEL OF MEDICAL INSTITUTION POLTAVA 1592

CASE STUDIES

Denys Kapustianskyi, Ihor Ivanytskyi, Tetiana Ivanytska, Valeriy Zhamardiy, Viktoriia Donchenko
DIFFERENTIAL DIAGNOSIS OF SOFT TISSUE TUMORS 1596

ABSTRACT BOOK

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ORIGINAL ARTICLE

PECULIARITIES OF MORPHOETIOPATHOGENESIS OF ACUTE APPENDICITIS AND CONSEQUENCES AFTER APPENDECTOMY

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ABSTRACT

The aim: To study the features of morphoethiopathogenesis of acute appendicitis and the consequences after appendectomy.

Materials and methods: 10 preparations of human appendix were studied, from which 5 appendixes were normal in people who died in adulthood and old age and 5 processes were taken intraoperatively during appendectomy. Morphometric characteristics of the tissue structures of the preparations were compared with the systematic review data, the literature search by the following keywords: morphoethiopathy", "vermiform process", "lymphoepithelial formations", "digestive system", "lymphoid nodule", "Peyer's patches", "mucous membrane".

Results: Pathogenesis of acute appendicitis – it is a consistent, staged process that is completely subject to the laws of exudative inflammation in response to microbial aggression. Removal of the appendicular process should be approached carefully and based on possible immunological consequences. Removal of the appendix as an immunocompetent organ Unreasonable removal of pathohistological unchanged appendix has medical consequences (can lead to consequences such as colon cancer) and not only.

Conclusions: Our results suggest that the vermiform appendix retains its active function throughout human life. The study provides an overview with current knowledge about the etiology, pathogenesis and possible consequences of appendectomy as the main method of treatment of acute appendicitis. The search for ways to prevent appendicitis can be successful only by finding out the causes and factors that in some individuals cause the inability of the appendix to resist bacterial invasion. Appendix is necessary to fully support the immune responses of the digestive tract, but it belongs to the category of those organs, the loss of which during forced surgery does not cause significant damage to the body.

KEY WORDS: Appendicitis, acute surgical pathology, lymphoepithelial formations

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INTRODUCTION

Currently, special importance in revealing the nature of the vast majority of diseases of the digestive system is given to the immune system, the relevant aspects of which are widely presented and discussed in many scientific journals. [1]. Although many publications on some issues are contradictory and incompletely studied, in recent years there has been a general concept of the mechanism of interaction between different parts of the immune system of the gastrointestinal mucosa, represented by the association of numerous lymphoid structures with mucosa due to migration and recirculation of immunocompetent cells between them provides the possibility of simultaneous activation of the immune response in all parts of the digestive tract, regardless of the focus of antigenic stimulus [2].

A special place in this system is occupied by the appendix, which, unlike other lymphoepithelial structures, is a relatively separate organ-type formation containing densely concentrated in the mucous membrane group of lymphoid nodules, known as Peyer's patches. [3]. The close anatomical connection between the appendix and the cecum is due to the fact that the latter breaks down food residues (mostly fiber) by putrefactive bacteria, which may include pathogenic microorganisms. It is possible that in some circumstances they are the cause of appendicitis.

Given the well-known theoretical position that any system in the body has mechanisms of adaptation and compensatory reserves, it can be argued that the immune system of the mucous membranes of the digestive tract in this regard is not an exception. Given this, not only theoretical but also practical interest is the question of why and in what form is the local immune defense compensation for the loss of the appendix after appendectomy.

THE AIM

The aim was to study the features of morphoethiopathogenesis of acute appendicitis and the consequences after appendectomy.

MATERIALS AND METHODS

10 preparations of human appendix were studied, from which 5 appendixes were normal in people who died in adulthood and old age and 5 processes were taken intraoperatively during appendectomy. After fixation in 10%

neutral formalin solution, the preparations were embedded in epon-812. Microscopic examination of the appendix tissues was carried out by obtaining sections of different thicknesses in their transverse and longitudinal directions. After thorough polishing of the end surfaces, they were stained with a 1% solution of methylene blue in a 1% solution of borax and studied using an MBS-9 and a Konus light microscope equipped with a Sigeta DCM-900 9.0MP digital microphotographic attachment with the Biorex 3 program adapted for these studies (serial number 5604). Morphometric characteristics of the tissue structures of the corresponding preparations were obtained using a system for visual analysis of histological preparations.

This bibliographic analysis is based on published articles, books, textbooks, monographs, and abstracts of dissertations. For the purposes of this systematic review, the literature search (concerning the study of morphoethiopathogenesis of acute appendicitis and consequences after appendectomy) was carried out on the Internet, domestic literature sources, scientific and electronic libraries of Poltava State Medical University by the following keywords: morphoethiopathy", "vermiform process", "lymphoepithelial formations", "digestive system", "lymphoid nodule", "Peyer's patches", "mucous membrane". The search period covered the period from 2011 to 2021, but the review includes some data from earlier publications, as these literature sources are of great scientific value.

The following inclusion and exclusion criteria were used: inclusion criteria: original articles published in journals and conference proceedings, books, textbooks, monographs, dissertation abstracts, peer-reviewed, language of publication: Ukrainian, Russian, English; Exclusion criteria: reviews, case studies, editorials, letters, etc. that are not peer-reviewed, language of publication: others.

RESULTS

With age, starting from mature (40 years) and ending with extreme old age (90 years), the appendix retains its typical structural principle in an active state. From a constructive point of view, its wall mainly consists of two (except for the serous coating) coaxial membranes - muscular and mucosal, separated from each other by a well-defined layer of loose fibrous connective tissue with an admixture of fatty tissue (Fig. 1).

A special place in the immune system is occupied by the appendix, which, unlike other lymphoepithelial structures, is a relatively isolated organ-type formation containing densely concentrated in the mucosa group lymphoid nodules known as Peyer's patches (Fig. 2).

The close anatomical connection of the appendix with the cecum is explained by the fact that in the latter there is a breakdown of food residues (mostly fiber) by putrefactive bacteria, which may include pathogenic microorganisms. Apparently, in some circumstances they are the cause of appendicitis.

Acute appendicitis is a local infectious non-specific inflammatory disease of the appendix, which develops due to changes in the biological relationship between the human body and microorganisms under the influence of various factors that require immediate surgical treatment.

A significant number of etiological theories of acute appendicitis are known. In any case, the occurrence of non-specific acute inflammation in the appendix is impossible without the participation of pathogenic microflora, which is normally present in every person. Inflammation requires certain conditions: damage to the barrier function of the mucous membrane of the appendix, increased virulence of the microflora, violation of the body's defenses.

According to the analysis of literature sources, the microflora can get into the mucous membrane in two ways: enterogenic and hematogenous. Enterogenic penetration implies the presence of damage to the mucous membrane or increase the virulence of the microflora while maintaining the normal function of the mucous membrane. Damage to the barrier function of the mucosa can be caused by trauma (foreign body, worm infestation, fecal stone, etc.) and disruption of its blood supply (arterial thrombosis, reflex spasm, etc.). If there is no damage to the mucous membrane and its barrier function is preserved, the microorganisms can penetrate the mucous membrane only with increasing virulence and activity. This can be observed in sensitization of the body, the processes of putrefaction and fermentation in the colon, constipation, and decreased acidity of gastric juice. In the case of hematogenous penetration of the microflora, inflammation is possible only in violation of the body's defenses, i.e. in any condition in which the human body develops immunodeficiency [4].

Specific inflammation of the appendix is caused by disease or factors that are not present in a normally functioning body. In this case, the diagnosis is based on the underlying disease, and inflammation of the appendix is stated as its complication (N.: ascariasis, acute phlegmonous appendicitis, worm infestations).

All theories have a right to exist, but today the most accepted theory of Aschoff's primary effect. According to her, the action of certain factors leads to functional disorders in the intestine (bauginospasm). As a result of bauginospasm there is a spasm of the vessels of the appendix, ischemia of the wall of the appendix develops (primary effect). As a result, the intestinal autoflora easily penetrates into the wall and causes acute inflammation of the appendix.

Pathogenesis of acute appendicitis – it is a consistent, staged process that is completely subject to the laws of exudative inflammation in response to microbial aggression. This process begins acutely with serous inflammation and is localized in the serous and mucous membranes.

Morphological changes observed in inflammation of the appendix are very diverse and depend mainly on the stage of the inflammatory process. There are two clinical and anatomical forms of appendicitis: acute and chronic. Each of them has a certain morphological characteristic.

We studied intraoperatively 2 catarrhal, 2 phlegmonous and 1 gangrenous forms of appendicitis.

The initial form of inflammation of the 2 appendices is defined as acute catarrhal appendicitis. Pathologists

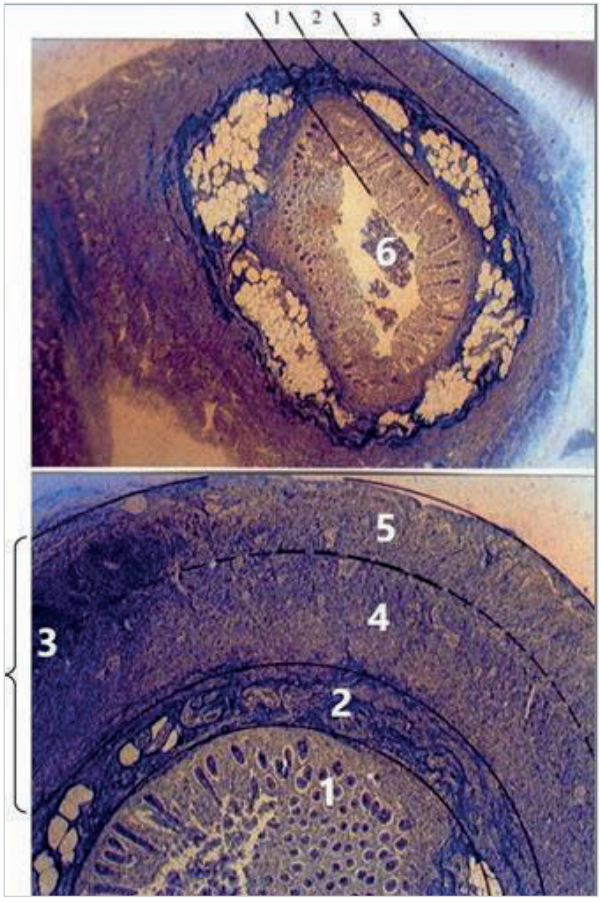

Fig. 1. Cross section of the base and middle part of the appendix. Epoxy sections; methylene blue color; 7x lens.
1 - mucous membrane; 2 - submucosal connective tissue base; 3 - muscular membrane; 4 and 5 - its inner and outer layers; 6 - internal lumen of the appendix

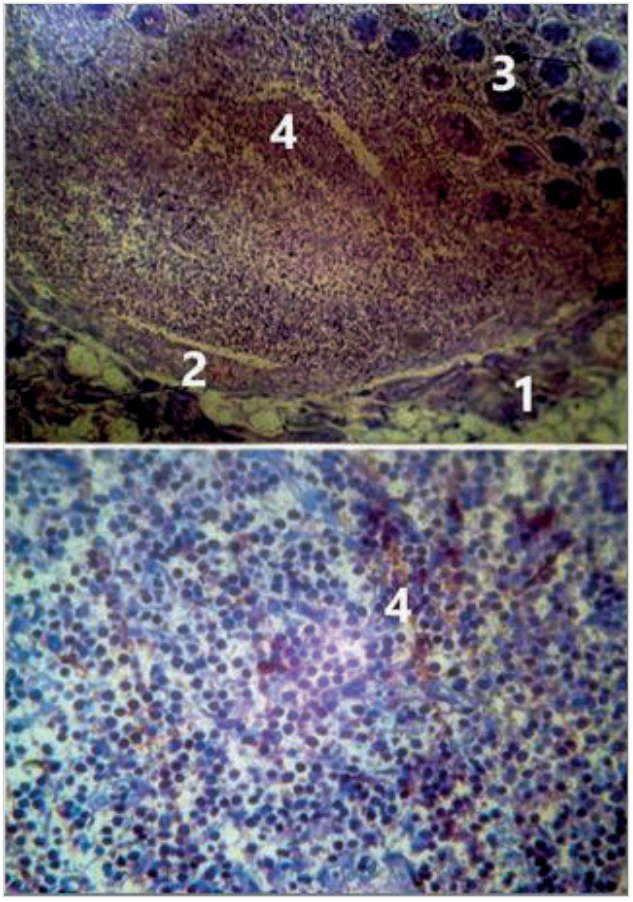

Fig. 2. Lymphoid nodule of the mucous membrane of the appendix. Epoxy sections; methylene blue staining; 10x and 40x lens.
1 - submucosa; 2 - base of the node; 3 - crypts; 4 - lymphocytes of the nodule

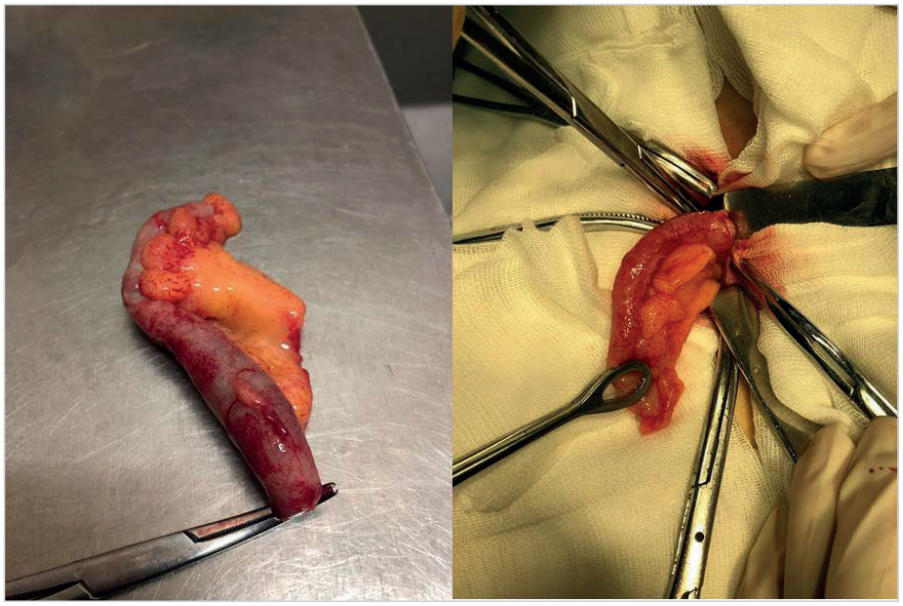

Fig. 3. Macroscopic picture of phlegmonously altered appendicular process

distinguish this form as "simple appendicitis" or "superficial appendicitis", which is more correct in terms of the essence of the inflammatory process. Macroscopically, the appendix looks a bit thickened, its serous membrane is dull, there are many small vessels filled with blood under it, which gives the impression of bright redness. In section, the mucous membrane of the appendix is swollen, gray-red, in the submucosal layer sometimes there are spots of hemorrhage. The lumen of the appendix often contains fluid of the genitalia. Microscopically, it is possible to de-

tect small defects of the mucous membrane covered with fibrin and leukocytes. Sometimes from a small defect the lesion spreads to tissues located deeper, having the shape of a wedge, the base of which is directed towards the serous membrane, this is a typical primary Aschoff's effect. There is a moderate leukocyte infiltration of the submucosal layer. The muscle membrane is not altered or slightly altered. The serous membrane contains a large number of dilated vessels, which can also be observed in the mesentery of the appendix. Occasionally there is a clear sterile reactive effusion in the abdominal cavity.

The inflammatory process is acute phlegmonous appendicitis (2 macropreparations). Macroscopically, the appendix looks significantly thickened; his serous and mesentery decreased, brightly hyperemic. The appendix is covered with a layer of fibrin, which in phlegmonous form is always more or less. Due to the fact that the process of transition to the peritoneum, you can see fibrinous layers on the dome of the cecum, parietal peritoneum, adjacent loops of the small intestine. Abdominal disease is often always exuded due to a large admixture of leukocytes. The effusion can be infected. The lumen of the appendix usually contains liquid, gray or green pus. The mucous base of the appendix is reduced, easily vulnerable and often you can see multiple and fresh ulcers, which gives pathologists the opportunity to distinguish the so-called phlegmonous-ulcerative form of acute appendicitis (Fig. 3.).

Microscopically in all layers of the appendix there is a massive leukocyte infiltration, covering the epithelial area and often exfoliated, sometimes there are multiple primary Aschoff's affects. In the mesentery of the appendix, in addition , there are leukocyte infiltrates.

A type of phlegmonous inflammation is an empyema of the appendix, in which a closed space filled with pus is formed in the lumen of the appendix due to obstruction by fecal stones or a connective process. The morphological feature of this form of appendicitis is that the process rarely passes to the peritoneum. The vermiform appendix in empyema is bulbous swollen and sharply tense, there is a clear fluctuation. Along with this, the serous membrane of the appendix looks like a catarrhal form of acute appendicitis: it is dull, hyperemic, but without layers of fibrin. Serous sterile effusion may be observed in the abdominal cavity. At the opening of the appendix, a large amount of smelly pus is poured out. Microscopically, there is a significant leukocyte infiltration in the mucous membrane and submucosal layer, which decreases to the periphery of the appendix. Typical primary effects are rare.

Gangrenous appendicitis develops (1 macropreparation), characterized by necrotic changes in the appendix. At the same time its total necrosis happens rather seldom, in the vast majority of cases the necrosis zone covers only a small part of a shoot. Contribute to the necrosis of the wall located in the lumen of the process fecal stones and foreign bodies. Macroscopically necrotic area is dirty green, loose and easily torn, the rest of the appendix looks the same as in the phlegmonous form of acute appendicitis. There are fibrinous layers on the organs and tissues surrounding the inflammatory appendix. The abdominal cavity often contains purulent effusion with fecal odor. Sowing this effusion gives the growth of typical colon flora. Microscopically in the area of destruction of the wall layers of the appendix can not be differentiated, they have the appearance of typical necrotic tissue, in other parts of the process there is a picture of phlegmonous inflammation.

If gangrenous appendicitis is not operated on in time, then perforation occurs and the process proceeds to the next stage - the so-called perforative appendicitis. The contents of the appendix are poured into the abdominal cavity. As a result, purulent peritonitis occurs. Macroscopically perforated appendix differs little from the gangrenous form of acute appendicitis. Areas of necrosis are also dirty green with signs of perforation, from which flows a smelly, often and chorous pus. The surrounding peritoneum is covered with massive fibrinous layers. The abdominal cavity contains abundant purulent effusions, sometimes fecal stones that have fallen out of the appendix.

DISCUSSION

According to modern literature, various etiological theories of the origin of this disease are known, which we will dwell in more detail [5].

L. Ashoff's infectious theory. According to this theory, the pathogen penetrates the mucous membrane of the appendix from the lumen, in the mucosa there is a "primary affect" - the area of violation of the epithelium of the mucous membrane, but the inflammatory process develops only when the virulence of microorganisms increases.

Cortico-visceral theory. According to this theory, pathological impulses from the gastrointestinal tract, entering the nerve plexuses and the CNS, after some time lead to dysfunction of the neuro-regulatory apparatus of the appendix. This is manifested by reflex spasm of blood vessels and muscles of the appendix, which leads to circulatory disorders (vascular stasis, edema). Trophic disorders end in necrosis of certain areas of the mucous membrane. The microflora joins the secondary. Kalitievsky PF, Saveliev VE and Savchuk VD believe that the dysfunction of intramural vegetative formations of the appendix causes contraction of the muscles of the appendix, which in turn leads to segmental disruption of blood supply to its wall.

Neurogenic theory. It is based on the fact that in the appendix under the influence of various factors there are pathological impulses that enter the CNS. Here the center of the "inert process of disturbance" is formed. When this focus is formed, any new irritation that comes from the body itself or from the outside, getting into the hearth, causes a clinic of acute appendicitis. This explains the discrepancy between the clinic and morphological changes in the process. Primary pathological impulses are associated with reversible functional and irreversible changes in the intramural nervous system of the appendix. Millller NA proposed to identify the reversible stage of acute appendicitis and call it "neurogenic appendicitis" [6].

The theory of stagnation of fecal masses. Proposed in 1892 by Talamone K. According to this theory, stagnation

of fecal masses leads to the formation of fecal stones in the lumen of the appendix, which damages the mucous membrane. The accession of pathogenic microflora is secondary [7].

The theory of constipation and "lazy gut". According to statistics, for those patients who later developed acute appendicitis, characterized by chronic, perennial constipation; defecation is less common in them than in those who did not suffer from acute appendicitis. Thus, at the turn of the 80's and 90's of the twentieth century in world literature was dominated by the idea that coprolites of the appendix occur due to delayed fecal content in the right parts of the colon during its prolonged passage. Limited epidemiological studies have found that cancer, diverticulosis, and glandular polyps of the colon are less common in populations that do not suffer from appendicitis. There is a well-known assumption (so far there is no strong evidence) that acute appendicitis may be an early precursor to the development of colon and rectal cancer, one of the main factors in the development of which is chronic constipation.

The theory of "closed cavities". If the outflow of contents from the lumen of the appendix is difficult or impossible, the secretion that has accumulated in it will cause overstretching and trauma to the mucous membrane. In addition, stagnant secretion serves as a nutrient medium for the growth and reproduction of pathogenic microorganisms, the attachment of which to the inflammatory process is secondary. The reasons for the formation of a "closed cavity" can be different: sclerosis with obliteration of the lumen of the appendix; obstruction of the lumen by a foreign body, helminth or fecal stone; inflammation with the development of edema of the mucous membrane; torsion of the appendix due to adhesions and scars caused by inflammation or injury of adjacent tissues, etc.

Hematogenous theory. Imagining that microbes enter the appendix mucosa hematogenously, we compared the pathological processes in the appendix and pharyngeal tonsils, based on the fact that often acute appendicitis occurs during or immediately after sore throat. This theory has been partially confirmed, because in sore throats, acute respiratory diseases, influenza, pharyngitis, pneumonia, often in the walls of removed worm-like processes were found the same microorganisms that were sown from the oropharynx of patients [8].

Alimentary theory. It has been noted that inflammation of the appendix is much more common in people who eat a lot of meat. According to the authors of the theory, protein foods enhance the processes of putrefaction in the colon, thereby increasing the virulence of the microflora of the cecum and appendix. In favor of this theory is the fact of a sharp reduction in the incidence of acute appendicitis during the starvation.

The theory of congenital bends. According to this theory , congenital anomalies can cause inflections, compression or fixation with impaired trophic process or obstruction of its lumen. Joining the microflora is secondary.

The theory of bauginospasm. Increased nervous excitability leads to the fact that any irritant etiology and strength causes spasm of the Bauginian valve, which causes pain and reflex stagnation of the contents in the lumen of the appendix, reflex vasospasm [9].

Allergic theory. Protein food, constantly sensitizing the human body, in some conditions causes an allergic reaction such as the Arthus phenomenon from the appendix [10].

The theory of immunodeficiency. Acute appendicitis was considered an autoinfection of a sensitized organism due to a "breakthrough of immunity". Many studies have found that acute appendicitis reduces the functional activity of T and B lymphocytes [11].

Viral theory. Predicts that the trigger for acute appendicitis is a virus. In the experiment, this assumption was not confirmed [12].

Theory of arterial thrombosis. According to this theory, thrombosis of the appendicular artery leads to destruction in the wall of the process up to perforation. Joining the microflora is secondary [13, 14].

Meteorological theory. The literature also notes the dependence of the incidence of acute appendicitis on meteorological conditions - increasing its frequency with fluctuations in air temperature, increasing relative humidity, fluctuations in solar activity and the state of the Earth's biosphere [15].

According to the literature described acute appendicitis caused by the presence of helminths in the lumen of the appendix. Helminths (pinworms, roundworms, spiral trichinae, hookworms, echinococci) were found in approximately 2% of worm-like appendages removed due to acute appendicitis. These parasites do not give a specific clinical picture, but the clinic of acute appendicitis may occur against the background of an erased clinical picture of this disease. As a rule, an accurate diagnosis is possible only after microscopic examination of the removed appendix. Defeat of a mucous membrane at worm invasion is twofold: direct mechanical damage by a parasite and chemical damage by those toxic substances which are allocated by parasites in the course of the vital activity. It should be emphasized that the roundworm can perforate the wall of the appendix [16].

According to the literature, amoeba was found in 7% of removed appendages, balantidial in 7%, trichomoniasis (Devian's intestinal form) in 12.5%, and in very rare cases - Plasmodium falciparum malaria. Surprisingly, no specific clinical picture of acute appendicitis caused by protozoa is observed, and the diagnosis is established only after microscopic examination of the removed appendix.

Many works are devoted to the defeat of the appendix by fungi. Actinomycosis and histoplasmosis are most often found, and in actinomycosis - only destructive forms of inflammation [17].

Changes in the appendix in typhus have been described. Changes in the appendix in typhoid fever and paratyphoid fever were divided into two groups: vulgar appendicitis (swelling of the lymphoid apparatus) and typhoid appendicitis with specific changes (ulcers) that can lead to perforation of the appendix wall. According to the literature, perforation of the appendix is 10% of all perforations of the intestine in typhoid fever [18].

Appendicular form, which usually affects children, occurs in approximately 3% of cases of clinical dysentery [19].

Lesions of the appendix by tuberculosis are very rare and, according to various authors, account for 0.016% of all removed appendages. The specific clinical picture of appendicitis in the defeat of mycobacteria of tuberculosis is not described [20].

Serous inflammation under the influence of the body's defenses can have a favorable outcome. Inflammation can progress, progressing sequentially from serous inflammation to the stage of fibrinous and purulent inflammation up to gangrene. This sequence of stages is always preserved and does not depend on etiological factors. Only the duration of one or another stage of inflammation depends on them. The development of inflammation in the appendix is due to the specificity of the pathogen (pathogenicity and virulence), the state of immunological reactivity and allergic reorganization of the patient's body. The predominance of microbial aggression factors over protective factors leads to a relatively rapid development of the disease, in which the initial stage is short. At the initial relative balance of forces of aggression and protection the inflammatory process can be long and end with regression or progress of inflammation [21].

Another point of view is that all forms of appendicitis develop in the first 24 hours after the disease and are independent, not transitional stages of a single inflammatory process in the appendix.

There is another point of view - destructive forms of appendicitis can have a stage of development, and simple forms are independent forms of inflammation that do not turn into destructive. According to Kalitievsky PF simple and destructive forms develop at once depending on degree of ischemia to which initial disturbances of blood supply of a worm-shaped shoot lead. A simple form of acute appendicitis is considered the most morphologically complex. The author concluded that the boundary between the initial signs of inflammation and different variants of tissue leukocytosis is almost impossible to draw.

Some scientists have noted that catarrhal appendicitis has nothing to do with classical appendicitis and is neither the initial form nor the stage of appendicitis. In this regard, the theory of appendicopathies was put forward, according to which acute appendicitis and appendicopathy are distinguished as different nosological units. Appendicopathy, according to the authors, is a set of clinical symptoms of acute appendicitis without an anatomical picture of inflammation of the appendix (ie, vasomotor, functional changes in the appendix or in the ileocecal intestine). In addition, the authors introduced the term "pseudo-appendicitis", which referred to a complex of subjective sensations (suffering), similar in some clinical symptoms to acute appendicitis or appendicopathy, but which are the result of adjacent or distant diseases [22].

Many scientists have proposed various schemes for the development of inflammation in appendicitis. Some scientists distinguish three stages: the stage that corresponds to the "primary affect" - lasts up to 12 hours from the onset of the disease; stage of development of phlegmonous inflammation - lasts from 12 to 24 hours from the onset of the disease; stage of gangrenous and perforated appendicitis - develops after 24 hours of illness [23].

Another scheme has 4 stages: the stage of functional disorders (appendicular colic); stage of formation of "intraapendicular abscess", or "stage of illusions"; stage of spread of peritoneal inflammation; stage of diffuse peritonitis. The term "stage of illusions" is applied more to the clinical course of the disease, rather than to the morphological stages of the inflammatory process. The destruction of the wall of the appendix continues, but the patient feels a clear improvement - the pain subsides. This is due, on the one hand, to the death of the intramural nerve endings of the appendix, and on the other - before the peritoneal irritation has occurred. In domestic literature, this period is known as the "stage of imaginary well-being".

Morphologists distinguish the following forms of acute appendicitis: simple (catarrhal); superficial; destructive: phlegmonous, apostematous, phlegmonous-ulcerative, gangrenous [24].

According to statistics, appendectomy is one of the most common surgeries in surgical practice. Acute appendicitis is a surgical disease for which the largest number of urgent operations is performed. As a result, acute appendicitis sometimes becomes a source of diagnostic errors. The risk of complications of acute appendicitis, a common rule among surgeons - "you can not rule out acute appendicitis - operate!" leads to the fact that the decision to operate on the patient is made quickly and often it is unwise. Studies show that most patients are operated on by surgeons either immediately after hospitalization or after short-term monitoring of their condition and changes in laboratory parameters. Morphological changes in the appendix may increase to perforation, which causes peritonitis and sometimes death of the patient. Therefore, when the diagnosis is problematic, surgeons are traditionally more likely to have surgery than to wait until they are confident in the diagnosis. Among other factors, active tactics have reduced mortality from acute appendicitis. At the same time, this tactic leads to the removal of the unaltered appendix, according to various authors, in 15-40% of patients [25].

Thus, the problem of acute appendicitis has two opposite aspects: on the one hand - delayed surgery can cause serious complications and even death, on the other - the cause of unjustified, erroneous surgery. Such a "simple", at first glance, intervention as appendectomy for chronic, subacute or catarrhal appendicitis is associated with complications and even fatalities [26].

CONCLUSIONS

Our results suggest that the vermiform appendix retains its active function throughout human life. The study provides an overview with current knowledge about the etiology, pathogenesis and possible consequences of appendectomy as the main method of treatment of acute appendicitis.

Currently, the search for ways to prevent appendicitis can be successful only by finding out the causes and factors that

in some individuals cause the inability of the appendix to resist bacterial invasion.

Obviously, the appendix is necessary to fully support the immune responses of the digestive tract, but it belongs to the category of those organs, the loss of which during forced surgery does not cause significant damage to the body. Apparently, this is due to the large reserve capacity of other lymphoepithelial formations of the mucous membrane of the digestive tract, which can compensate for its loss during appendectomy.

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