PATHOLOGICAL ANATOMICAL CHANGES, ETIOPATHOGENETIC MECHANISMS, AND CLINICAL-MORPHOLOGICAL FEATURES OF GANGRENE

***Jumaniyozova Feruza Sayfiddinovna*[1]**
***Jiyanmuradova Shakhlo Khamdamovna*[1]**
***Ergasheva Shahzoda Abror qizi*[2]**
[1]Teacher, Zarmed University
[2]First-year student, Higher Nursing Education Program, Zarmed University

Annotation. This article presents analytical information related to pathological anatomy concerning gangrene. Gangrene is a severe pathological condition characterized by necrosis (death) of living tissues followed by putrefaction processes. The article describes its etiology, pathogenesis, and morphological changes. Gangrene occurs in several forms: dry, wet, gas, and internal gangrene, each having its own specific clinical and morphological features. Dry gangrene develops slowly and usually occurs without infection, whereas wet and gas gangrene progress rapidly and are complicated by infectious processes. Gas gangrene is particularly dangerous as it develops under the influence of anaerobic bacteria and poses a serious threat to the patient's life. The article emphasizes that disturbances in blood circulation and metabolic changes in tissues play a crucial role in the pathogenesis of gangrene. In addition, detailed information is provided about clinical manifestations, complications, and modern treatment approaches. The management of gangrene requires urgent surgical intervention, antibiotic therapy, and detoxification measures. This analytical material contributes to understanding the main aspects of gangrene and demonstrates the practical importance of pathological anatomy.

Keywords: Gangrene, necrosis, dry gangrene, wet gangrene, gas gangrene, internal gangrene, pathological anatomy, infection, tissue death, sepsis, amputation, pathogenesis, morphology, bacteria, anaerobic flora.

Introduction. Gangrene is a pathological condition characterized by the death (necrosis) of living tissues and their subsequent decomposition due to exposure to the external environment or secondary infection under anaerobic conditions. In pathological anatomy, gangrene is considered a complex pathological process because it is associated not only with necrosis but also with microbial activity, immune responses, circulatory disturbances, and other contributing factors. Gangrene is a clinically significant condition frequently encountered in surgical practice, traumatology, diabetology, and many other medical fields.

Gangrene does not always develop as an independent disease; rather, it often arises as a complication of underlying conditions such as diabetes mellitus, atherosclerosis, arteritis, or traumatic injuries. The prognosis and treatment of gangrene largely depend on its type, the extent of tissue damage, the patient's general condition, and the timing of medical intervention.

Main Part. From a pathological anatomical perspective, gangrene is classified into the following forms:

1. Dry gangrene (Gangraena sicca). *Dry gangrene usually develops as a result of gradually impaired blood circulation. It most commonly affects extremities such as toes or fingers. The affected tissues lose moisture, become hard, and turn black or dark brown. They resemble mummified structures. It is mainly caused by atherosclerosis, diabetic angiopathy, or frostbite. Infection is usually absent or minimal.*

2. Wet gangrene (Gangraena humida). *Wet gangrene represents a typical form of infected necrosis. In this condition, tissues become soft, swollen, liquefied, and emit a foul odor. Color changes (blue-brown or greenish) are observed. It commonly occurs in patients with severe circulatory disorders and weakened immunity. It is caused by infectious agents such as staphylococci, streptococci, Escherichia coli, and various anaerobic microorganisms.*

3. Gas gangrene (Gangraena gaseosa). *Gas gangrene is the most severe and life-threatening form. It is caused by anaerobic bacteria such as Clostridium perfringens and Clostridium septicum. These bacteria produce toxins that lead to rapid tissue destruction and gas formation within tissues (emphysema). Patients develop severe intoxication, high fever, and shock. Immediate surgical intervention and antibiotic therapy are required. Gas gangrene often develops after deep wounds, surgical procedures, or childbirth complications.*

4. Internal (hidden) gangrene. *Internal gangrene may develop in internal organs such as the intestines, lungs, or gallbladder. Intestinal gangrene, often resulting from volvulus or thrombosis, is frequently diagnosed late and is complicated by peritonitis and sepsis.*

Pathogenesis and Morphological Changes. The primary mechanism in the pathogenesis of gangrene is circulatory disturbance leading to tissue ischemia. Reduced blood supply causes hypoxia, metabolic dysfunction, membrane damage, enzymatic imbalance, and ultimately cell death. Depending on the type of gangrene and the presence of infection, morphological changes include:

- In dry gangrene: tissue dehydration, hardening, and mummification
- In wet gangrene: swelling, liquefaction necrosis, and enzymatic autolysis
- In gas gangrene: gas bubble formation, necrotic foci, and bacterial colonies

Histologically, necrotic cells, hemorrhages, leukocyte infiltration, and microorganisms are observed.

Clinical Manifestations

- Increased body temperature
- Swelling and pain
- Changes in tissue color (gray, purple, black)
- Foul-smelling discharge
- In gas gangrene: crepitation (crackling sensation under the skin)
- Signs of systemic intoxication

Complications.

- Sepsis (blood poisoning)
- Toxic shock syndrome
- Multi-organ failure
- Life-threatening hemorrhage
- Death

Without timely treatment, gangrene progresses rapidly and causes severe systemic damage.

Principles of Treatment. The main goal in treating gangrene is the early removal of necrotic tissue, reduction of systemic intoxication, and control of infection. Treatment includes:

- Surgical removal of necrotic tissue (amputation, necrectomy)
- Broad-spectrum antibiotic therapy
- Detoxification therapy (infusion solutions, plasmapheresis)
- Oxygen therapy and, in some cases, hyperbaric oxygenation

Conclusion. Gangrene is a severe pathological condition characterized by irreversible tissue necrosis followed by infectious, inflammatory, and toxic processes. In pathological anatomy, gangrene is evaluated through the stages of tissue necrosis and its morphological progression. The disease exists in dry, wet, gas, and internal forms, each with distinct etiological, pathogenetic, and clinical features.

Dry gangrene develops due to gradual circulatory failure and usually occurs without infection, while wet and gas gangrene develop rapidly under the influence of infectious agents, especially anaerobic bacteria. Clinically, gangrene is extremely dangerous and may lead to sepsis, shock, and death.

Therefore, early diagnosis and urgent treatment are critically important. The main treatment strategies include surgical removal of necrotic tissues, antibiotic therapy, and detoxification of the organism. This article demonstrates the significant role of pathological anatomy in understanding the mechanisms, diagnosis, and effective treatment of gangrene.

REFERENCES:

1. Ergashev B. (2025). Gingivitning bakteriologik etiologiyasi va profilaktikasi. In International Scientific Conference "Innovative Trends in Science, Practise and Education", 1(1), 122–128.

2. Tursunaliyev Z., & Ergashev B. (2025). Bolalarda tish kariesini oldini olish usullari. Modern Science and Research, 4(4), 686–691.

3. Ergashev B. (2025). Karies va paradont kasalliklari profilaktikasi. Modern Science and Research, 4(4), 732–741.

4. Ergashev B. (2025). Psychological support for cancer patients. ИКРО Журнал, 15(01), 164–167.

5. Ergashev B., & Raxmonov Sh. (2025). Oral trichomoniasis: Epidemiology, pathogenesis, and clinical significance. Kazakh Journal of Ecosystem Restoration and Biodiversity, 1(1), 19–27.

6. Ergashev B., & Raxmonov Sh. (2025). Transmission dynamics of tuberculosis: An epidemiological and biological perspective. Kazakh Journal of Ecosystem Restoration and Biodiversity, 1(1), 28–35.