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Skin and subcutaneous tissue infections

Weronika Biaduń, Jakub Buczkowski, Aleksandra Romanowska, Aleksandra Padkowska, Kinga Rogowska, Michał Borawski, Aleksandra Ciuła, Klaudia Arciszewska, Mateusz Dobosz, Jakub Maciej Pieniążek

Weronika Biaduń

Casimir Pulaski University of Radom Faculty of Medical Sciences and Health Sciences Chrobrego 27, 26-600 Radom, Poland

w.biadun1@gmail.com

https://orcid.org/0009-0009-2650-5991

Jakub Buczkowski

Casimir Pulaski University of Radom Faculty of Medical Sciences and Health Sciences Chrobrego 27, 26-600 Radom, Poland

jakubbuczkowsky@gmail.com

https://orcid.org/0009-0005-0309-5222

Aleksandra Romanowska

Casimir Pulaski University of Radom Faculty of Medical Sciences and Health Sciences Chrobrego 27, 26-600 Radom, Poland

olarrromanowska@gmail.com

https://orcid.org/0009-0001-9829-2659

Aleksandra Padkowska

Casimir Pulaski University of Radom Faculty of Medical Sciences and Health Sciences Chrobrego 27, 26-600 Radom, Poland

ola.padkowska@gmail.com

https://orcid.org/0009-0000-3939-7349

Michał Borawski

Brothers Hospitallers of Saint John of God Hospital in Cracow Trynitarska 11 Street, 31-061 Cracow, Poland

michal.borawski1@gmail.com

https://orcid.org/0009-0008-4864-7336

Aleksandra Ciuła

Brothers Hospitallers of Saint John of God Hospital in Cracow Trynitarska 11 Street, 31-061 Cracow, Poland

https://orcid.org/0009-0000-0425-863X

ciula.aleksandra@gmail.com

Klaudia Arciszewska

Casimir Pulaski University of Radom Faculty of Medical Sciences and Health Sciences Chrobrego 27, 26-600 Radom, Poland

k-arciszewska@wp.pl

https://orcid.org/0009-0009-5099-6313

Mateusz Dobosz

Casimir Pulaski University of Radom Faculty of Medical Sciences and Health Sciences Chrobrego 27, 26-600 Radom, Poland

mdobosz.mat@gmail.com

https://orcid.org/0009-0006-2407-7522

Kinga Rogowska

Casimir Pulaski University of Radom Faculty of Medical Sciences and Health Sciences Chrobrego 27, 26-600 Radom, Poland

kinga.rogowska0504@gmail.com

https://orcid.org/0009-0003-7987-6477

Jakub Maciej Pieniążek

Casimir Pulaski University of Radom Faculty of Medical Sciences and Health Sciences

Chrobrego 27, 26-600 Radom, Poland

kubapieniazek99@gmail.com

https://orcid.org/0009-0005-9063-6575

Abstract

Necrotizing infections of the skin and subcutaneous tissues are life-threatening conditions characterized by rapid progression and high mortality. The most common types include Fournier's gangrene, gas gangrene, necrotizing fasciitis, and necrotizing myositis. These infections are often polymicrobial and involve both Gram-positive and Gram-negative bacteria capable of producing potent toxins. Diagnosis is based on clinical presentation, laboratory tests (e.g., LRINEC score), imaging techniques (ultrasound, CT, MRI), and microbiological evaluation. Effective treatment requires prompt surgical intervention, broad- spectrum empiric antibiotic therapy, and supportive care. Early recognition and aggressive management are essential to improve patient outcomes and reduce the risk of death.

Key words: necrotizing infections, Fournier's gangrene, gas gangrene, necrotizing fasciitis, necrotizing myositis

Introduction

These infections are of diverse etiology and may be caused by bacteria, viruses, fungi, or parasites. They encompass a wide range of conditions, from mild infections that usually do not require hospitalization, to more severe cases requiring surgical management, and even life-threatening necrotizing infections.The risk of skin and subcutaneous tissue infections increases in individuals with diabetes, obesity, cachexia, hepatorenal syndrome, advanced age, immunodeficiency, chronic lung and heart diseases, trauma, and peripheral lymphatic or arterial-venous insufficiency.

The risk of wound infection grows with the virulence of bacteria present. Crush-type injuries, especially those affecting the lower limbs, carry the highest risk. Contamination with soil, feces, or saliva further increases this risk. A key factor contributing to infection in extensive wounds is the delay between injury occurrence and surgical intervention. (Burnham et al. 2018, Stasiak et al.2012, Ramakrishnan et al. 2017)

Necrotizing infections.

Among necrotizing infections of the skin and subcutaneous tissue, we can distinguish Fournier's gangrene, gas gangrene, necrotizing fasciitis, and necrotizing myositis.

They are caused by bacteria producing various toxins and are characterized by a high degree of virulence. These infections are extremely dangerous to humans, and without proper and timely treatment, they can lead to death. (Ramakrishnan et al. 2017)

Fournier’s Gangrene

This type of infection involves the perianal area, perineum, and genital organs. Fournier’s gangrene is usually of mixed bacterial etiology. The most commonly isolated microorganisms include Gram-negative bacteria such as Escherichia coli and Pseudomonas aeruginosa, as well as Gram-positive bacteria like Staphylococcus aureus and Streptococcus pyogenes.

It most frequently occurs in individuals with alcohol abuse, diabetes, immunodeficiency, malignancies, obesity, and in elderly patients. (Leslie et al. 2022)

Gas Gangrene

This is a necrosis of muscle or connective tissue, accompanied by gas formation and severe toxemia.

It is mainly caused by Clostridium perfringens, but other species such as Clostridium novyi, Clostridium septicum, Clostridium histolyticum, Clostridium sordellii, and Clostridium bifermentans may also be responsible.

Gas gangrene occurs primarily in patients with extensive wounds, especially crush injuries (e.g., from traffic accidents), particularly if contaminated with soil. Abdominal surgery is also a risk factor. (Leiblein et al. 2020, Wiercińska 2021a)

Necrotizing Fasciitis

Usually spares the muscles but involves the subcutaneous tissue and fascia.The main pathogen is group A Streptococcus pyogenes, but other organisms like Staphylococcus aureus, Vibrio vulnificus, and Aeromonas hydrophila may also be involved. Patients with diabetes, alcohol abuse, or liver cirrhosis are at higher risk. (Albadri and Salman 2019)

Necrotizing Myositis

This may be caused by mixed infections, although Staphylococcus aureus or Streptococcus pyogenes are often the causative agents. Risk factors include diabetes, chronic kidney failure, and immunodeficiency. (Plakoutsis and Mitsionis 2015)

Diagnosis of Necrotizing Skin and Subcutaneous Tissue Infections.

Clinical symptoms play an important role in diagnosing necrotizing infections, although they often appear when the infection is already advanced. These symptoms include disproportionately severe pain compared to clinical signs, the presence of hemorrhagic blisters, skin and subcutaneous hemorrhages, gas in tissues (crepitus), hypoesthesia and hyperesthesia, swelling, and rapid progression of lesions. Systemic signs of toxemia may also be present such as hypotension, fever or hypothermia, and tachycardia.

Laboratory tests included in the LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score may be useful: CRP, hemoglobin, white blood cell count, sodium, glucose, and creatinine levels. Imaging methods such as ultrasound can help differentiate necrotizing from non- necrotizing infections. CT and MRI are particularly useful when the diagnosis is uncertain or in evaluating necrotizing myositis.

The 'finger test' is another diagnostic tool. It is a quick procedure performed under local anesthesia, involving a 2 cm incision into the fascia. If cloudy, foul-smelling fluid is observed,

along with necrotic tissue, absence of bleeding, lack of muscle contraction, or resistance to dissection, it indicates necrotizing soft tissue infection.

Blood cultures are mandatory for all patients with suspected necrotizing soft tissue infections. Another microbiological test is Gram staining of a direct smear obtained from deep tissue samples, which allows definitive diagnosis. (Szewczyk 2019, Żukowska and Hryniewicz 2020, Albadri and Salman 2019, Burnham et al. 2018, Ramakrishnan et al. 2017)

Treatment of Necrotizing Skin and Subcutaneous Tissue Infections.

Treatment protocols for skin and subcutaneous tissue infections should include early diagnosis, prompt surgical intervention, empiric antimicrobial therapy, and supportive care.

Wounds with necrotizing soft tissue infections require debridement every 24–48 hours until necrosis resolves and clinical stability is achieved. Antibiotic therapy is crucial. Microbiological testing should guide appropriate therapy. Gram stain of direct smears can help in early selection of effective antibiotics. Broad-spectrum antibiotics are initiated empirically and later adjusted based on culture results. Empiric treatment for necrotizing fasciitis or Fournier's gangrene includes one of the following regimens:

- vancomycin
- a carbapenem (meropenem or imipenem/cilastatin)
- linezolid combined with piperacillin/tazobactam
- ceftriaxone with metronidazole

Empiric treatment for gas gangrene or necrotizing myositis should include:

- vancomycin combined with piperacillin/tazobactam
- a carbapenem (meropenem or imipenem/cilastatin)

When microbiological testing reveals the following pathogens, recommended antibiotics are:

- *Streptococcus pyogenes*: penicillin + clindamycin (or linezolid if clindamycin- resistant)
- *Staphylococcus aureus* (MSSA): cloxacillin or cefazolin
- *Staphylococcus aureus* (MRSA): vancomycin or linezolid
- *Vibrio vulnificus*: doxycycline + ceftazidime
- *Aeromonas hydrophila*: doxycycline + ciprofloxacin
- *Clostridium perfringens* and other *Clostridium* spp.: penicillin + clindamycin
- Mixed infections: piperacillin/tazobactam or a carbapenem (meropenem or imipenem/cilastatin).

Hyperbaric oxygen therapy is often used in cases of gas gangrene. However, it should not interfere with standard treatment approaches.Intravenous immunoglobulin therapy may be considered in toxic shock syndrome caused by Streptococcus pyogenes, although evidence supporting its effectiveness is limited. (Żukowska and Hryniewicz 2020, Wiercińska 2021a, Albadri and Salman 2019, Burnham et al. 2018, Plakoutsis and Mitsionis 2015)

Non-necrotizing infections

Non-necrotizing infections include: abscess, impetigo, furuncle, erysipelas, and cellulitis.These infections are usually mild, typically superficial, and generally not life-threatening. (Stasiak et al. 2012, Ramakrishnan et al. 2017)

Abscess

An abscess is a localized, encapsulated inflammatory focus filled with pus. It appears as a painful, firm lump surrounded by erythema.Staphylococcus aureus is the main causative agent, although Gram-negative flora and anaerobes may also be involved. (Żukowska and Hryniewicz 2020, Stasiak et al. 2012)

Impetigo

This is a localized infection of the connective tissue, usually affecting children. Lesions occur on the face or limbs and are typically honey-colored, though a vesicular form may also occur. Impetigo is most commonly caused by Streptococcus pyogenes, with Staphylococcus aureus as a less frequent cause. (Żukowska and Hryniewicz 2020, Ramakrishnan et al. 2017)

Furuncle and Carbuncle

These are purulent infections around hair follicles, presenting as bluish-red, painful, firm swellings due to inflammatory infiltration. Carbuncles are larger and involve both skin and subcutaneous tissue.The main causative agent is Staphylococcus aureus. (Żukowska and Hryniewicz 2020, Stasiak et al. 2012)

Erysipelas

A diffuse infection of the skin and subcutaneous tissue presenting as a red lesion with well- defined borders. The affected area is raised above the surrounding skin.The etiologic agent is Streptococcus pyogenes. (Stasiak et al. 2012)

Cellulitis (Connective Tissue Inflammation)

This is a purulent skin infection without clear borders, involving both the skin and underlying connective tissue. It typically results from puncture wounds but may also occur due to other skin integrity breaches.Erythema is present but lacks clear demarcation and elevation, unlike erysipelas.Cellulitis is caused by Streptococcus pyogenes, Staphylococcus aureus, and in children, Haemophilus influenzae. (Żukowska and Hryniewicz 2020, Burnham et al. 2018, Szewczyk 2019)

Treatment of Non-necrotizing Skin and Subcutaneous Tissue Infections.

Abscesses should be incised and drained.Antibiotic therapy is indicated only in cases of extensive inflammation, immunosuppression, incomplete surgical drainage,

recurrent abscesses, facial or hand involvement, or systemic signs of infection.Intravenous antibiotics include: cefuroxime (if Gram-negative flora is suspected), clindamycin (for penicillin allergy or deep abscesses with anaerobes), cefazolin, and cloxacillin. Oral therapy includes: cefadroxil, cephalexin, cefuroxime (for Gram-negative flora), clindamycin, or amoxicillin/clavulanic acid (for anaerobic infection), clindamycin (if allergic to penicillin). Antibiotics active against MRSA may be used in patients with risk factors, poor response to initial treatment, immunosuppression, or signs of shock. The recommended treatment duration is 5–7 days. .

For abscesses in the abdomen, groin, or perineum (where anaerobes and Gram-negative flora are common), treatment should last 7 days. IV options include piperacillin/tazobactam; oral and IV use of amoxicillin/clavulanic acid is also recommended. For penicillin allergy, ciprofloxacin with metronidazole is advised. .

Depending on the size and location of lesions, either local or systemic treatment is applied. Topical treatment (5 days) includes fusidic acid or mupirocin. Systemic treatment (7 days) includes cloxacillin, cephalexin, and, for confirmed streptococcal etiology, phenoxymethylpenicillin.

The primary treatment is drainage. Antibiotics are indicated only if systemic signs of infection are present.Recommended antibiotics: cefazolin, cloxacillin, cefuroxime, clindamycin, cephalexin, cefadroxil. Duration: 5–7 days.MRSA-active antibiotics are used when initial therapy fails, with immunosuppression or shock symptoms.

Erysipelas, recurrent erysipelas. If treatment fails, or in immunocompromised or severe cases, culture testing should be performed. Recommended antibiotic: penicillin. If ineffective, use cefazolin or cloxacillin; if penicillinallergy exists, moxifloxacin, clindamycin, or clarithromycin are alternatives. Treatment duration: 10 days. Recurrent erysipelas (3–4 episodes) requires prophylaxis. Key is identifying and mitigating risk factors. Prophylactic antibiotics: clarithromycin, moxifloxacin, or clindamycin, continued until predisposing factors are controlled. Cellulitis (connective tissue inflammation). Cultures should be obtained in severe infections, immunodeficiency, or if initial therapy is ineffective. Mild cases: oral antibiotics such as cephalexin, cefadroxil, clindamycin. IV options for mild to moderate cases: clindamycin, cefazolin, cloxacillin, cefuroxime.MRSA-active therapy may be needed in high- risk patients or those with poor response or immunodeficiency. Duration: typically 5–7 days. If no improvement, suspect deeper

infection such as necrotizing fasciitis. . (Żukowska and Hryniewicz 2020, Stasiak et al. 2012, Szewczyk 2019, Ramakrishnan et al. 2017)

Infections in damaged skin

Burn Wounds

A burn is an injury to the skin, mucous membranes, and sometimes deeper tissues or organs caused by heat, chemicals, electric arcs, ionizing radiation, or friction. The severity depends on the energy source, exposure time, and affected surface area. Based on depth, burns are classified into four degrees (Table 1).

Table 1. Classification of burn wounds by depth

Degree of Burn	Skin Depth	. Injury Depth	Symptoms
I	Superficial	Epidermis	Erythema, severe pain
II A	Superficial partial-thickness	Epidermis and part of dermis	Blisters, severe pain
II B	Deep partial-thickness	Epidermis and dermis	Blisters, severe pain
III	Full thickness	Epidermis, dermis and subcutaneous tissue	Firm, waxy skin, no pain due to damage to skin nerves
IV	Full thickness, penetrating deeper tissues	Epidermis, dermis, subcutaneous tissue and deeper structures up to bone	Charred tissue, necrosis

The skin acts as the body's natural protective barrier. Its microbiota consists mainly of Actinobacteria, Firmicutes, Bacteroidetes, and Proteobacteria. During a burn, the skin is sterilized and becomes vulnerable. Within 48 hours, bacterial colonization occurs. The subcutaneous tissue becomes exposed, immune mechanisms are impaired, exudate appears, subcutaneous vessel thrombosis develops, and ischemia and hypoxia

ensue.Without proper treatment, infection may occur, often progressing to sepsis. (Korzekwa 2021, Jędrys 2017)

Microbiological Diagnosis. Initially, burn wounds are colonized by Gram-positive bacteria such as Staphylococcus spp. and Streptococcus spp., originating from the patient's endogenous flora, respiratory tract, or environment. Local antiseptic treatment with silver-based or chlorhexidine preparations is recommended at this stage.

Between days 2–4, Gram-negative bacteria such as Pseudomonas aeruginosa, Acinetobacter baumannii, Escherichia coli, Klebsiella pneumoniae, and Enterobacter cloacae become predominant, originating from the skin, respiratory tract, gastrointestinal tract, or hospital environment. As antibiotic therapy is initiated, fungal infections (e.g., Candida spp.) may also develop. In the final stage, colonization with resistant bacteria and fungi may occur. (Szewczyk 2019)

Treatment. Targeted antibiotic therapy, based on microbiological testing, is the treatment of choice for infected burn wounds. Topical antibiotic use should be avoided as it impairs skin function and blood flow. Surgical intervention and antiseptic wound care are the mainstays of treatment. In patients with infection and/or sepsis, empiric antibiotic therapy should cover both Gram-positive and Gram-negative bacteria. (Korzekwa 2021, Jędrys 2017)

Bite Wounds

Skin infections may also result from animal bites, most commonly from cats and less frequently from dogs. These injuries introduce bacteria such as Pasteurella multocida, Capnocytophagacanimorsus, and Eikenellacorrodens, leading to inflammation within 24 hours.Initially, the infection is localized to the skin and subcutaneous tissue, but may progress to abscesses, fasciitis, joint and bone infections, and systemic involvement such as nervous system or bloodstream infections.

Common symptoms include high fever, enlarged lymph nodes, pain, swelling, and erythema around the wound. (Żukowska and Hryniewicz 2020, Stasiak et al. 2012)

Risk factors for infection include:

- type of bite,
- wound size,
- location and time since the bite,
- patient factors such as respiratory, circulatory, or lymphatic disorders, and presence of prosthetic heart valves.

Microbiological Diagnosis. Diagnostic evaluation should consider anaerobes and fastidious pathogens. Samples may be obtained from: wound exudate, infected tissue, wound swabs, blood, cerebrospinal fluid, or stool. (Szewczyk 2019)

Antibiotic Therapy Prophylactic antibiotic therapy is indicated in high-risk patients: those with immunodeficiency, diabetes, chronic liver disease, post-splenectomy, and children under two years of age. Treatment duration is 3–5 days. The most commonly used antibiotic is amoxicillin with clavulanic acid. (Żukowska and Hryniewicz 2020, Ramakrishnan et al. 2017)

Treatment. Empirical therapy should be broad-spectrum, targeting both aerobic and anaerobic pathogens. Additional treatments include:

- primary wound closure (only in facial bite wounds),
- post-bite immunoprophylaxis – tetanus toxoid should be administered to patients who:
  - were never vaccinated against tetanus,
  - received their last booster over 10 years ago,
  - have contaminated wounds and their last booster was more than 5 years ago. (Żukowska and Hryniewicz 2020)

Diabetic foot infections.

Diabetic foot is a complication of type 1 or type 2 diabetes. It results from nerve damage, poor circulation, and excessive foot pressure. About 25% of diabetic patients develop

foot ulcers, 80% of which become infected. The ulcers typically result from skin disruption caused by poorly fitting shoes or walking barefoot. Neuropathy often masks pain, delaying wound detection and increasing infection risk.Infection is diagnosed when at least two signs are present: local swelling, erythema, pain or tenderness, warmth, and purulent discharge. However, clinical presentation may vary, especially in neuropathy or ischemia, complicating diagnosis. (Korzon-Burakowska and Łukaszewicz 2010, Witek and Katra 2017, Kowalska 2022)

Microbiological Diagnosis. Clinical signs suffice for diagnosing soft tissue infection. Bone infection requires additional imaging (MRI) or bone biopsy.

Microbiological testing should only be performed in symptomatic patients and helps identify dominant organisms. Acute wounds tend to harbor Gram-positive bacteria (e.g., S. aureus, beta-hemolytic streptococci A, B, C, G), while chronic wounds may also include Gram-negative and anaerobic organisms.

Samples should be taken after wound debridement, from:

- tissue biopsies, aspirations, or curettage,
- purulent discharge,
- deep wound swabs (only if proper samples cannot be obtained).

Superficial swabs or samples from skin/soft tissue have low diagnostic value. Additional tests include CBC, ESR, CRP, creatinine, and liver transaminases (especially prior to antibiotic therapy). (Korzon-Burakowska and Łukaszewicz 2010, Witek and Katra 2017, Kowalska 2022, Szewczyk 2019)

Table 2. Infection Classification in Patients with Diabetic Foot

Grade	Features	Type of Antibiotic
1	No signs of infection	None given
2	- Infection of skin and subcutaneous tissue - Presence of ≥ 2 characteristic symptoms (erythema 0.5–2 cm)	Clindamycin, amoxicillin with clavulanic acid
3	- Involvement of deeper tissues: bone, fascia - Presence of ≥ 2 characteristic symptoms (erythema >2 cm)	Amoxicillin with clavulanic acid, ceftriaxone, linezolid, ertapenem, cefuroxime with metronidazole, ciprofloxacin with clindamycin
4	General infection symptoms: temperature > 38°C, tachycardia > 90/min, respiratory rate > 20/min, partial oxygen pressure pO2 < 32 mm Hg, leukocytosis 12,000—40,000/mcl (10% immature forms)	Intravenous therapy: - Broad-spectrum cephalosporin, - Ciprofloxacin with clindamycin, - Vancomycin with ceftazidime and metronidazole

Infections in Chronic Skin Lesions - Pressure Ulcers and Venous Ulcers

A pressure ulcer is a wound resulting from prolonged pressure or friction on the skin. It most often occurs in immobilized patients in areas such as the sacrum, buttocks, or heels.

Venous ulcers result from chronic venous hypertension due to chronic venous insufficiency. They typically appear on the lower leg, above the ankle. These areas show swelling and venous stasis. The skin becomes thin, tight, dry, and itchy. Untreated ulcers can become infected. Diagnosis of infection is clinical: wounds that do not heal within two weeks

of proper cleansing, warmth, redness, severe pain, poor granulation, and foul odor suggest infection. Systemic signs may include fever, chills, malaise, and weakness.

Etiologic agents include Enterobacteriaceae, non-fermenting Gram-negatives (Pseudomonas aeruginosa, Acinetobacter baumannii), Staphylococcus aureus, Enterococcus spp., and anaerobes (Peptostreptococcus). (Wiercińska 2021b, Ludwikowska 2017, Chwała 2021)

Microbiological Diagnosis. Biopsy after proper surgical cleansing is recommended. Performing microbiological diagnostics in patients without clinical symptoms or using superficial swabs is inappropriate and lacks diagnostic value. (Chwała 2021, Szewczyk 2019)

Antibiotic Therapy. Antibiotics are indicated in non-healing chronic wounds with signs of sepsis, spreading cellulitis, muscle or fascia infection, or vasculitis or osteomyelitis.

Not recommended:

- prophylactic use in chronic wounds (proper care is usually sufficient),
- routine use in chronic wounds,
- topical antibiotics (except metronidazole to reduce pain or odor in suspected anaerobic infections).

Duration of antibiotic therapy varies by wound type and symptoms:

- Cellulitis: ~7 days
- Sepsis: 10–14 days
- Osteomyelitis: 6 weeks to 3 months (Żukowska and Hryniewicz 2020, Wiercińska 2021b, Ludwikowska 2017, Chwała 2021)

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Author'scontribution:

Conceptualization: Jakub Buczkowski, Weronika Biaduń, Jakub Maciej Pieniążek; Methodology: Aleksandra Ciuła, Klaudia Arciszewska; Software: Aleksandra Padkowska, Kinga Rogowska, Michał Borawski, Aleksandra Ciuła; Check: Jakub Buczkowski, Weronika Biaduń, Jakub Maciej Pieniążek, Aleksandra Padkowska;

Formalanalysis: Jakub Buczkowski, Weronika Biaduń, Jakub Maciej Pieniążek; Investigation: Michał Borawski, Aleksandra Ciuła, Jakub Maciej Pieniążek; Resources: Mateusz Dobosz, Jakub Buczkowski; Data curation: Weronika Biaduń; Writing - rough preparation: Michał Borawski, Aleksandra Ciuła; Writing - review and editing: Mateusz Dobosz, Jakub Buczkowski, Michał Borawski, Aleksandra Ciuła, Aleksandra Romanowska; Visualization: Klaudia Arciszewska, Aleksandra Romanowska; Supervision: Jakub Maciej Pieniążek, Aleksandra Padkowska; Project administration: Michał Borawski, Weronika Biaduń.

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