



CUTANEOUS PURULENT DISEASES (PYODERMA): A REVIEW

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Abstract: Cutaneous purulent diseases (pyoderma) are inflammatory-purulent processes in the skin and its appendages resulting from bacterial infection. This group of diseases is one of the most common in dermatology, primarily caused by *Staphylococcus aureus* (including MRSA) and *Streptococcus pyogenes*. This review provides general information on the classification, etiology, clinical manifestations, diagnosis, treatment strategies, and problems of antibiotic resistance in pyoderma. Modern approaches recommend topical treatment as the first choice and systemic antibiotics only when necessary.

Keywords: Pyoderma, cutaneous purulent infections, impetigo, folliculitis, furuncle, carbuncle, MRSA, antibiotic resistance, topical treatment

Cutaneous purulent diseases (pyoderma) are bacterial infections accompanied by pus formation in the skin, classified into surface (superficial) and deep types [1]. These conditions are often secondary, developing against the background of atopic dermatitis, eczema, diabetes mellitus, immunosuppression, or poor hygiene [2]. Globally, skin and soft tissue infections account for a significant proportion of cases among hospitalized patients.

Pyoderma is classified according to the depth of infection. The causative agents are bacteria, and in some cases, secondary bacterial infection may occur in viral skin diseases. However, viral skin diseases (herpes, warts, molluscum contagiosum, chickenpox) typically do not result in purulent processes [3]. Table 1 presents the classification of pyoderma, main causative agents, and brief clinical features [4,5,6].

Table 1. Classification of Cutaneous Purulent Diseases

No	Classification Type (Level)	Main Types (Examples)	Main Causative Agent(s)	Brief Clinical Features
1	Surface Pyoderma (Surface pyoderma)	Impetigo contagiosa (common impetigo, sachratqi / honey-crusted sore) Bullous impetigo (intertriginous purulent dermatitis)	<i>Streptococcus pyogenes</i> (more common), <i>Staphylococcus aureus</i>	Erosions covered with honey-colored crusts, purulent discharge, rapid spread; often on face and perioral area



2	Superficial Follicular Pyoderma	Ostiofolliculitis (superficial folliculitis) Deep folliculitis Sycosis (chronic facial folliculitis)	<i>Staphylococcus aureus</i> (main)	Red nodules, pustules, purulent vesicles around hair follicles; possible itching
3	Deep Pyoderma	Furuncle (boil) Carbuncle Ecthyma Hidradenitis suppurativa Furunculosis (multiple furuncles)	<i>Staphylococcus aureus</i> (including MRSA)	Deep painful nodules, purulent masses, swelling, fever; carbuncle involves fusion of multiple furuncles
4	Streptococcal Pyoderma (Streptoderma)	Streptococcal impetigo (sachratqi) Perleche (angular impetigo) Paronychia (superficial) Simple ecthyma	<i>Streptococcus pyogenes</i> (main)	Thin vesicles, purulent discharge, rapid drying into black crusts; superficial erosions
5	Staphylococcal Pyoderma (Staphylo-derma)	Folliculitis, furuncle, carbuncle, hidradenitis, sycosis, neonatal vesiculopustulosis	<i>Staphylococcus aureus</i> (most common)	Involvement of hair follicles and apocrine glands; deep purulent inflammation, pain
6	Mixed Pyoderma (Staphylo-streptoderma)	Papulo-erosive staphylo-streptoderma Impetigo-like papular lesions Mixed forms	<i>S. aureus</i> + <i>S. pyogenes</i> or others	Combined effects of two pathogens; mixed superficial and deep features

As seen from the table, the main pathogens are *S. aureus* and *S. pyogenes*. Pathogenesis: Breach of skin barrier (trauma, scratching, maceration) + pathogen entry → inflammation → neutrophil infiltration → pus formation. Resistant strains (MRSA) are spreading in community and hospital settings [7]. Examples include:

Impetigo: Erosions covered with yellow-red crusts (often on face and hands).

Folliculitis: Red nodules and pustules around hair follicles.

Furuncle: Deep, painful purulent nodule.

Carbuncle: Fusion of multiple furuncles into a large purulent mass.

Ecthyma: Deep ulcer with hard crust. Common signs: redness, swelling, pain, purulent discharge, fever (in deep forms).

Diagnosis is established as follows: Clinical presentation, bacteriological examination (pus swab + antibiotic susceptibility testing – mandatory in suspected resistance), blood tests,

blood glucose (diabetes screening). Differential diagnosis: herpes, fungal infections, pyoderma gangrenosum, vasculitis [8].

Treatment varies depending on the depth and severity of infection.

• **Surface forms:** Topical antibiotics: mupirocin, retapamulin, fusidic acid (first choice). Antiseptics: chlorhexidine, povidone-iodine. Hygiene + removal of crusts.

• **Superficial and deep pyoderma:** Systemic antibiotics (7–14 days): Cephalexin, cefadroxil (first choice). Amoxicillin-clavulanate. Clindamycin or trimethoprim-sulfamethoxazole (for MRSA). If MRSA confirmed – vancomycin (severe cases).

• **Supportive measures:** Treatment of underlying causes (allergy, diabetes). Wet compresses, drainage (for furuncle/carbuncle). Surgery (for abscesses).

• **Prevention:** Personal hygiene. Wound cleaning. Rational antibiotic use (to reduce resistance) [9,10].

Cutaneous purulent diseases yield good results with timely diagnosis and proper treatment. Main focus should be on pathogen identification, consideration of resistance, and elimination of underlying factors. Collaboration between dermatologists and family physicians is essential

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