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Chapter

4

Bacterial Infections

INTRODUCTION

The skin surface harbours saprophytic flora, such as *Staphylococcus (S.) epidermidis*, Micrococci, Corynebacteria and Yeast. The number of organisms varies in different parts of the body; it depends upon the age of the individual, the environment and climate. Besides harmless organisms some pathogenic bacteria also grow in certain areas of the body especially the body folds. When the barrier horny layer is broken these organisms proliferate and cause disease.

The skin's own defences such as unsaturated fatty acids and sebum keep the flora in check. In hot and humid climate, the body defences are lowered and minor trauma or abrasions often result in infection. Bacterial infections can be primary or secondary. Infections may be secondary to scabies, fungal infections, eczema, etc. The severity of the disease depends upon the following factors:

- The causative bacteria and its severity of toxicity
- The target tissue
- The immunological status of the patient.

Normal Bacterial Flora

The skin is sterile at birth, but only for a short period. *S. aureus* colonisation of the umbilicus begins during the first day of life in 25% of neonates. This figure increases steadily, perhaps due to the fall in the pH of the skin from 7.0 to 5.5.

The normal skin flora can be transient or resident. Organisms capable of multiplying as well as survival are classified as resident flora; these are found almost all over the skin. The transient flora, are deposited in the skin from the mucous membrane drop out or from the environment.

Opportunistic organisms are the nonpathogenic members of the resident or transient flora that produce infection in debilitated or immunocompromised patients.

Resident Flora

Propionibacterium acnes: This is an anaerobic diphtheroid present in the sebaceous follicles of the skin. Its presence in acne does not indicate a causative role, but the acne lesions are due to the physiological changes in the underlying disease.

Aerobic diphtheroids: These organisms are usually present in the intertriginous areas; *Corynebacterium* (C.) *tenuis* causes trichomycosis axillaris. *C. minutissimum* causes erythrasma.

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Staphylococcus epidermidis: This organism is uniformly present on the normal skin; it exerts a suppressive action on the colonisation of the skin by other organisms. It is an important pathogen in prosthetic valve endocarditis and indwelling venous catheters.

Micrococci: These are not increased in number in dermatological diseases.

Gram-negative bacilli: These are present in the intertriginous areas; these include *Escherichia* (*E.*) *coli*, *Proteus*, *Pseudomonas*, *Enterobacter alcaligenes*. They often give rise to bacteraemia in patients with indwelling venous catheters.

Transient Flora

Aerobic spore forming organisms, such as Streptococci of various groups and Neisseria, are present on the skin surface for short periods. *S. aureus* is present in about 20% of individuals, their presence is associated with nasal or perineal discharge. Rifampicin is said to eradicate *S. aureus* from chronic nasal carriers. At times Streptococci can also colonise deeper in the skin and may become a part of the resident flora. Atypical mycobacteria occur as transient bacilli in the intertriginous areas.

INFECTIONS CAUSED BY *STAPHYLOCOCCUS AUREUS* AND *STREPTOCOCCUS HAEMOLYTICUS*

Staphylococcus and Streptococcus are the two common pathogens that infect skin. Most of the skin infections are caused by coagulase positive Staphylococcus, such as *S. aureus* producing invasive and toxin-mediated infection. *S. epidermidis* which is coagulase negative causes most of hospital infection. About 20% of individuals carry *S. aureus* in the nasal cavity and perineum; they act as carriers of infection. It is often the cause of recurrent furunculosis and wound infections.

Methicillin-resistant *Staphylococcus aureus* (MRSA) is becoming increasingly common because of the excessive use of antibiotics. The patient should be isolated to prevent spread of infection. Vancomycin is effective against MRSA, but resistance to this is also reported.

About 90% of streptococcal infection is caused by *S. pyogenes,* which belongs to β-haemolytic Streptococcus of group A, *S. viridans* (α-haemolytic Streptococci) and *S. pneumoniae* are also important members of streptococci. Subacute bacterial endocarditis and glomerulonephritis are complications of streptococcal infection; the patients should have a close follow-up after treatment. Patients should be monitored for anti-streptolysin O (ASO) and antistreptodornase titres till they normalise.

Impetigo

Impetigo is a contagious superficial skin infection caused by *S. aureus* or β-haemolytic streptococci. In some cases both the organisms are responsible for the lesion. It is the most common bacterial skin infection in children, but can occur at any age. The disease is common in the summer

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Clinical Features

There are two clinical types of impetigo:

- Non-bullous impetigo (Impetigo contagiosa) (Fig. 1)
- Bullous impetigo (Fig. 2).

***Non-bullous impetigo*:** This is more common, it is responsible for about 70% cases of impetigo. The disease is characterised by the formation of thin walled vesicles, these occur predominantly on exposed parts of the body. The lesions are the most common on the face, especially around the mouth and nose. The vesicles rupture readily to form golden, yellow or brown crusts. There is gradual peripheral extension. Healing occurs without scarring.

***Bullous impetigo*:** The initial lesions are vesicles, which rapidly progress to form flaccid bullae. Bullae develop due to the action of an epidermolytic toxin on the granular layer, if the toxin becomes widely disseminated it results in staphylococcal scalded skin syndrome (SSSS). The bullae are at first clear, but later become purulent. The bullae rupture in a day or two. After rupture a brownish crust is formed. The lesions are more common on the trunk and extremities.



Fig. 1: Impetigo-non bullous



Fig. 2: Impetigo-bullous

Complications

Nephritogenic strains of streptococci that are sometimes responsible for impetigo can cause acute glomerulonephritis. The nephritis occurs 18–21 days after the infection, as compared to 10 days after the throat infection.

Scarlet fever, urticaria and erythema multiforme may follow streptococcal impetigo. Rheumatic fever is not a complication.

Complications following staphylococcal impetigo are cellulitis, lymphangitis, SSSS and bacteraemia.

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months; prevalence is highest in tropical regions. Overcrowded and unhygienic conditions are important predisposing factors; it may complicate a pre-existing skin disease, such as scabies, pediculosis, atopic dermatitis and miliaria. Impetigo is contagious; infection can easily spread to other parts of the body and to close contacts.

Differential Diagnosis

The disease should be differentiated from herpes simplex in which the base is more erythematous and oedema more pronounced. In Tinea circinata the annular lesions have active vesiculations at the periphery, scraping with potassium hydroxide examination will show the fungus.

Treatment

Topical antiseptic creams, soaps or lotions containing hexachlorophene, triclosan, and chlorhexidine and povidone-iodine are effective for cleaning the lesion. Topical antibiotics are effective in most of the cases. The effective agents include bacitracin, gentamicin, mupirocin and fusidic acid.

Impetigo caused by erythromycin-resistant *S. aureus* is common in children. Amoxicillin plus clavulanic acid 25 mg/kg of body weight per day is given tid for 7–10 days. In extensive impetigo, penicillinase-resistant penicillin is initiated. Flucloxacillin or Cloxacillin is the antibiotic of choice and is given in a dose of 250–500 mg 6 hourly. Azithromycin 500 mg on the first day, followed by 250 mg for the next 4 days, is effective alternative therapy.

If impetigo appears resistant to treatment or is recurrent, then nasal swabs are taken for culture. Nasal mupirocin is useful to eradicate nasal carriage.

Tilbury Fox (1836–1879)

Tilbury Fox was an able and eminent English dermatologist. He was the first to describe dyshidrosis and impetigo. He described urticaria pigmentosa, lymphangioma circumscriptum, and epidermolysis bullosa but did not name these conditions.

Folliculitis

Folliculitis is an inflammation of the skin that is confined to the follicular ostium or slightly below it. Healing takes place without scar formation.

Aetiology

Infective: The offending agents include bacteria such as *S. aureus*, some gram-negative organisms, fungi such as *Pityrosporum ovale* and *Trichophyton rubrum*.

Chemicals: Oil folliculitis follows occupational exposure to mineral oil. Tar folliculitis is due to occupational or therapeutic exposure to tar.

Drugs: Topical steroids under occlusion.

Physical: Waxing, threading, plucking, occlusive dressings, etc.

Clinical Features

The lesions present as multiple, small follicular papules or pinhead-sized pustules. There is no pain. Tiny pustules with a central hair and an erythematous halo manifest Bockhart's impetigo. As the lesions dry, small yellow-brown crusts form. Folliculitis heals without scarring. The common sites are the scalp, beard region and the limbs.

Tropical folliculitis of the legs is common in Negroes and in other tropical races, its chronicity and atrophic sequelae characterise it. Clinically it resembles the Bockhart's type, but these are deep-seated nodules, as they heal they leave behind deep-seated atrophic depressions (Fig. 3).

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Fig. 3: Folliculitis

Treatment

- Remove the cause such as the physical and chemical agents
- One percent gentian violet or brilliant-green paint is a cheap and effective remedy
- Topical antibacterial agents: Fusidic acid, gentamicin cream, etc.
- Nasal and perineal carriage should be looked for and treated in chronic and resistant cases
- With availability of effective antibacterial agents, the need for systemic antibiotics is reduced
- Antimicrobial soaps are effective in treatment of folliculitis because they reduce the growth of Gram-negative, as well as Gram-positive bacteria. Soaps containing triclosan, chlorhexidine, hexachlorophene or povidone-iodine are beneficial. Systemic antibiotics should be used to treat tropical folliculitis of the legs.

Folliculitis in adults can be due to Ofugi's disease (Eosinophilic pustular folliculitis). The disease is seen mostly in Japan, the incidence is in the 3rd decade, most commonly on the scalp. It is often associated with AIDS. There is no effective treatment, topical and systemic and corticosteroids, topical tacrolimus, dapsone, colchicines and ultraviolet B (UVB) therapy have been used for treatment. Aetiology is unknown.

Furunculosis (Boils) and Carbuncles

Boil is an acute infection of the deeper part of the hair follicle, *S. aureus* is the most common pathogen. A carbuncle is a mass of boils that usually indicates an underlying debility.

Clinical Features

The initial lesion is a tender, hard red papule that rapidly develops into a nodule: this points and then discharges greenish-yellow pus. The sites of predilection are the face, neck, axillae and buttocks. The lesion should not be squeezed because this can lead to serious complications like osteomyelitis, cavernous sinus thrombosis or septicemia (Fig. 4).

A number of boils coalesce to form a carbuncle; it is often secondary to malnutrition, diabetes mellitus, prolonged steroid therapy, generalised debilitating diseases, etc. It presents as a red tender hard plaque, with a number of follicular orifices that discharge pus. Constitutional symptoms like fever may be present (Fig. 5).

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Fig. 4: Furuncles



Fig. 5: Carbuncle

Treatment

Local heat relieves discomfort and aids in localization of the infection. If the boil is large and fluctuant; it should be incised and drained. Carbuncles usually require surgical treatment. Predisposing factors that cause carbuncles should be treated, such as malnutrition, diabetes mellitus, and general debilitating diseases.

Systemic antibiotics are effective in treating boils; local antibiotics do not penetrate deep into the dermis. The effectiveness of erythromycin and tetracycline is limited owing to emergence of resistant strains of ***S. aureus***. Flucloxacillin is effective in most cases. Unresponsive cases can be controlled by ciprofloxacin or low-dose clindamycin.

If methicillin-resistant strain of ***S. aureus*** is suspected vancomycin 1.0–2.0 g IV once in 12 hours is indicated. The infusion should be given slowly. Linezolid oral or intravenous may also be used.

Recurrent furunculosis: Recurrent furunculosis is common if patients harbour ***S. aureus*** in the skin or anterior nares. Diabetes mellitus can also lead to recurring attacks of furunculosis. Both topical and systemic medication is required for recurrent furunculosis.

Topical agents: Fusidic acid 2% cream or ointment is used three times daily. Mupirocin is effective against MRSA. In recurrent furunculosis, mupirocin is applied to anterior nares two or three times daily for 1 week each month; this effectively eradicates the nasal organisms.

Systemic therapy: Rifampicin is effective against recurrent furunculosis. The recommended dose is 600 mg daily for 1 week each month for 3 months. Ciprofloxacin and minocycline have also been used for recurrent furunculosis.

Systemic isotretinoin may clear many types of folliculitis, presumably by changing the microenvironment of the pilosebaceous units. It is used in doses of 1mg/kg of body weight per day for 3–5 months. Isotretinoin should be reserved for patients who have failed other therapies.

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Fig. 6: Sycosis barbae

Sycosis Barbae

This is a subacute or chronic pyogenic infection involving the whole depth of the follicle. Sycosis appears in males after puberty and commonly involves the region of the beard and upper lip near the nose. Many patients are seborrhoeic they often suffer from chronic blepharitis and have a greasy skin.

Clinical Features

The essential lesion is an edematous red follicular papule or pustule around the hair, usually the follicles are discrete, but sometimes they may coalesce to form a plaque with studded pustules. The pustules rupture after shaving, leaving an erythematous red spot that is later a site of fresh eruption of pustules. The disease is chronic with attacks of varying duration, occurring at irregular intervals (Fig. 6).

Treatment

Subacute forms are treated relatively easily with antibiotics both topical and systemic. If the nasal swabs show a chronic carrier state, then the topical antibiotic should be applied to the nasal vestibule. The chronic form does not respond well to antibiotics and a steroid antibiotic combination is preferred. The treatment should be continued for a long time.

ECTHYMA

It is a pyogenic infection of skin characterized by the formation of adherent crusts beneath which ulceration occurs. The causative organisms are similar to that of impetigo, i.e. *S. pyogenes* or *S. aureus*. Some authors consider ecthyma as a subtype or ulcerated form of impetigo. In ecthyma, the infection spreads in depth while in impetigo it spreads on the surface.

Clinical Features

The infection begins as a vesicle or a pustule with a red halo, this rapidly turns into a bulla that breaks down to form punched out ulcer covered with a dark-brown bloody crust. Removal of the crust shows an irregular crateriform ulcer. The ulcer heals in 6–8 weeks often with scar formation. The lesions are few or numerous. The sites of predilection are lower extremities, buttocks and thighs. It is especially common in children and the elderly. There is a potential for developing post-streptococcal glomerulonephritis (Fig. 7).

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Fig. 7: Ecthyma

Treatment

- Removal of crusts by starch poultice
- Topical antibacterials, e.g. mupirocin or fusidic acid ointment
- Systemic antibacterial: The antibiotic chosen should be effective against both Staphylococcus and Streptococcus. The antibiotics used are similar to that of impetigo, but the treatment should continue for 3 weeks.

Erysipelas and Cellulitis

Erysipelas is the infection of dermis and upper subcutaneous tissue due to *S. pyogenes* or occasionally *S. aureus*. Cellulitis is the infection of the loose subcutaneous tissue. The two conditions may often coexit. The association of erysipelas/cellulitis is more common than each disease alone.

Cellulitis: Cellulitis is characterized by red, hot, tender swelling around an ulcer or wound, the infection spreads into the surrounding tissue. The edge of the lesion is not well-defined. Cellulitis may be accompanied by fever and regional lymphadenopathy. It differs from erysipelas in being deeper and without a distinct edge (Fig. 9).

Cellulitis can occur on the face following sinus infection, otitis media, tooth extraction and trauma. In immunocompromised persons cellulitis can cause gangrene and

Clinical Features

Erysipelas: The onset of erysipelas is abrupt with fever, chills and pronounced malaise. The disease produces a sharply marginated red tender edematous plaque, which spreads peripherally, the borders are raised, the surface is shiny, bullae and vesicles may occur. It commonly affects the face, lower limbs and the feet may also be involved. There may be red streaks of lymphangitis with regional lymphadenopathy. Impairment of the lymphatic damage occurs in recurring erysipelas. The infection subsides in 1–3 weeks followed by slight pigmentation and desquamation. When located on the legs the swelling becomes covered with verrucous and papillomatous growths (Fig. 8).

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Fig. 8: Erysipelas- border raised



Fig. 9: Cellulitis

ecthyma gangrenosum. Other complications include fasciitis, myositis, subcutaneous abscess, septicemia and streptococcal nephritis.

Laboratory studies are usually not required for erysipelas/cellulitis; but they are required when the patient is immunocompromised. Successful identity of pathogens is difficult from the site of lesion, although injection of saline in the advancing border and then aspirating the fluid is tried, when the lesion is not open. Blood culture is a vital part of search for identifying the etiological agent. Magnetic resonance imaging (MRI) has gained favor in diagnosing soft-tissue infections.

Complications

- Deep cellulitis
- Septicemia and metastatic abscess
- Glomerulonephritis, endocarditis
- Lymphedema in recurrent cases.

Treatment

Elevate the limb and use cool compresses locally.

Systemic antibiotics are required for treatment. A single injection of procaine penicillin will arrest the process. Penicillin should be given for 10–20 days. If no improvement occurs in a day, penicillin-resistant Staphylococcus should be suspected and flucloxacillin should be initiated. Macrolide antibiotics are used as an alternate, such as erythromycin, azithromycin or clindamycin.

In recurrent cases penicillin V daily for 5–7 days, benzathine penicillin G 2.4 million units once a month or erythromycin 1 g daily for 5 days every month for 4–6 months can prevent attacks. Vigorous treatment of any local skin damage is required. Some patients may require longer-term or life-long prophylaxis.

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Lymphangitis

Lymphangitis is inflammation of the lymph vessels, often following an acute streptococcal infection of the skin, less often by staphylococci. Lymphangitis may be a sign that the skin infection is getting worse. It should raise concerns that bacteria may spread into the bloodstream, which can cause life-threatening problems.

Clinical Features

Lymphangitis is associated with constitutional symptoms, such as fever, malaise, headache, arthralgia, myalgia and loss of appetite. Painful, red streaks are visible along the course of the lymphatic vessels, which may be faint or obvious. The pain is throbbing in nature; the regional lymph nodes may be enlarged.

Complications

Abscess, cellulitis and septicaemia.

Diagnosis

A biopsy and culture of the affected area may reveal the cause of the inflammation. Blood cultures may be done to see if the infection has spread to the bloodstream.

Treatment

Lymphangitis may spread within hours. Treatment should begin promptly. Parenteral antibiotics may be required for a patient with signs of systemic illness; this should be started immediately without waiting for a culture report. Coverage should be provided for Group A Streptococci and *S. aureus.* These include penicillinase-resistant synthetic penicillin, cephalosporin or a second or third generation cephalosporin, such as cefuroxime, ceftriaxone. When erythema, warmth and edema are markedly reduced, antibiotics can be changed to the oral route.

Analgesics can help control pain, and anti-inflammatory medications can help reduce inflammation and swelling. Warm moist compressions may reduce inflammation and pain. Surgery may be needed to drain an abscess.

Necrotising Fasciitis

This is a dangerous, rapidly progressive and destructive inflammation of the dermis and underlying tissues including the deep fascia. It can be a polymicrobial infection following surgery, especially in a diabetic or immunocompromised patient; or it may be caused by *S. pyogenes* leading to streptococcal gangrene. The toxic bacterial products and necrotic debris trigger a massive destructive inflammatory reaction. Necrotising fasciitis is associated with profound toxaemia and multisystem failure (Fig. 10).

The condition often arises suddenly after a minor skin trauma. The legs are the most common site of

Investigation

Aspiration or Incision for culture and sensitivity, exclude clostridial infection with wound smear. Infections with clostridia and bacteriodes may lead to gas formation, which can then be diagnosed radiographically.

Magnetic resonance imaging scans are also helpful in diagnosing necrotising fasciitis or subcutaneous abscess. In patients with muscle damage or subcutaneous abscess, well-defined areas of high signal intensity are found.

Baseline laboratory tests should be performed, and then monitored for leucopenia, hypocalcaemia (fat necrosis), and elevated creatine phosphokinase (muscle necrosis).

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Fig. 10: Necrotizng fasciitis

infection. The clue to the presence of this infection is a rapidly spreading cellulitis in a very toxic patient, despite antibiotic therapy. Necrotising fasciitis is initially manifested as erythema and edema, the lesion progresses to brawny induration and cyanosis. Blister formation and gangrene rapidly follow. The central area of the lesion is anesthetic due to cutaneous nerve damage; the surrounding area is extremely tender and erythematous. In polymicrobial necrotising fasciitis subcutaneous gas may be present.

Necrotising fasciitis due to *Vibrio vulnificus* is found in tropical region, this is associated with liver disease and contact with shelfish.

Treatment

Necrotising fasciitis requires immediate treatment with generous surgical debridement. An appropriate antibiotic cover should be started immediately, without waiting for culture and sensitivity results; as the mortality rate is very high. Usually intravenous benzyl penicillin, a quinolone, and clindamycin or metronidazole for anaerobic Gram-negative organisms is started immediately. Even with prompt treatment mortality rate is high; in some cases amputation of the limb may be required.

Other adjunctive therapies include intravenous gamma-globulins; patients with poor granulocyte production may be helped by granulocyte stimulating factor. Recent literature recommends that nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided as they may interfere with leukocyte chemotaxis, decrease inflammatory response and therefore blur the cardinal signs of inflammation.

Eosinophilic fasciitis is a scleroderma-like disorder, seen in children and adults, although it is rare in children. It has a sudden onset, following strenuous exercise. The sites commonly affected are the arms and legs. The condition is characterized by pain, swelling, erythema and induration. The surface of the skin has a cobblestone appearance. The condition is associated with transient eosinophilia and elevation of sedimentation rate. The condition resolves spontaneously. Glucocorticosteroids, methotrexate and cimetidine are helpful.

Staphylococcal Scalded Skin Syndrome

Staphylococcal scalded skin syndrome (Ritter's disease) is characterized by epidermolysis and desquamation. The skin exfoliates below the granular layer. The disease is usually linked to infections with Group 2 *S. aureus*, phage type 71. Ritter von Ritterschein, who was a director of an orphanage in Prague; first described the disease. The focus of infection is not in the skin; the cutaneous lesions are due to the action of an epidermolytic toxin, elaborated by an infection at a remote site usually nasopharynx. The disease usually occurs in

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children under 5 years of age. Most patients are newborns or infants; the condition is primarily explained by a lack of immunity to the toxins and to renal immaturity that leads to a poor excretion of toxins. Epidermolytic toxin antibodies are present in 75% of normal people over the age of 10 years; this fact explains the rarity of SSSS in adults.

Histopathology

Intraepidermal cleavage occurs beneath and within the stratum granulosum. The remainder of the epidermis and the dermis contains no inflammatory cells.

Clinical Features

Initially a faint erythematous rash develops; cutaneous tenderness develops at this stage. Within 24–48 hours the rash progresses from a scarlatiniform eruption to spontaneous wrinkling of the skin, followed by the appearance of large flaccid bullae in the axillae, groins and around the body orifices, later spreading all over the body, but sparing the mucous membrane. As sheets of epidermis are shed, a moist erythematous base is revealed. The lesion dries quickly and healing is complete in 5–7 days.

The rare adult type of SSSS in adults is associated with underlying diseases related to immunosuppression, abnormal immunity and renal insufficiency.

Diagnosis

Examining the roof of the blister can make rapid diagnosis. The epidermis is cleaved below the stratum granulosum. Nikolski's sign is positive.

Treatment

Topical antiseptics or fusidic acid are used for local treatment. The body should be covered with nonadhesive covering. Intake-output chart should be maintained, fluid therapy and general supportive measures should be taken. Systemic antibiotic such as penicillinase-resistant penicillin or first generation cephalosporin is initiated as soon as possible. Erythromycin is also effective. The prognosis is good. Corticosteroids are contraindicated.

Toxic Shock Syndrome (TSS)

The condition is characterised by widespread erythema, and "circulatory shock". It is a multisystem disorder.

Aetiology

The syndrome is caused by the toxin of *S. aureus*. The organism is in most cases isolated from the vagina during menstruation, most women using tampons. The role of tampons remains uncertain; it probably provides a suitable condition for bacterial growth and injury to the vagina, which facilitates absorption of the toxin. TSS can occur after staphylococcal infection from any site, at any age and in either sex.

Clinical Features

The onset is sudden with fever and rash. The rash is erythematous and widespread usually clearing within 3 days. Edema of the hands and feet may occur. Conjunctiva is red. Desquamation occurs after 1–2 weeks, it may be localized on the fingers or it may be widespread.

Systemic manifestations: Diarrhea and vomiting occur due to gastrointestinal involvement. Liver, kidney, central nervous system (CNS) and cardiovascular system (CVS) may also be affected. Circulatory shock may be very severe.

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