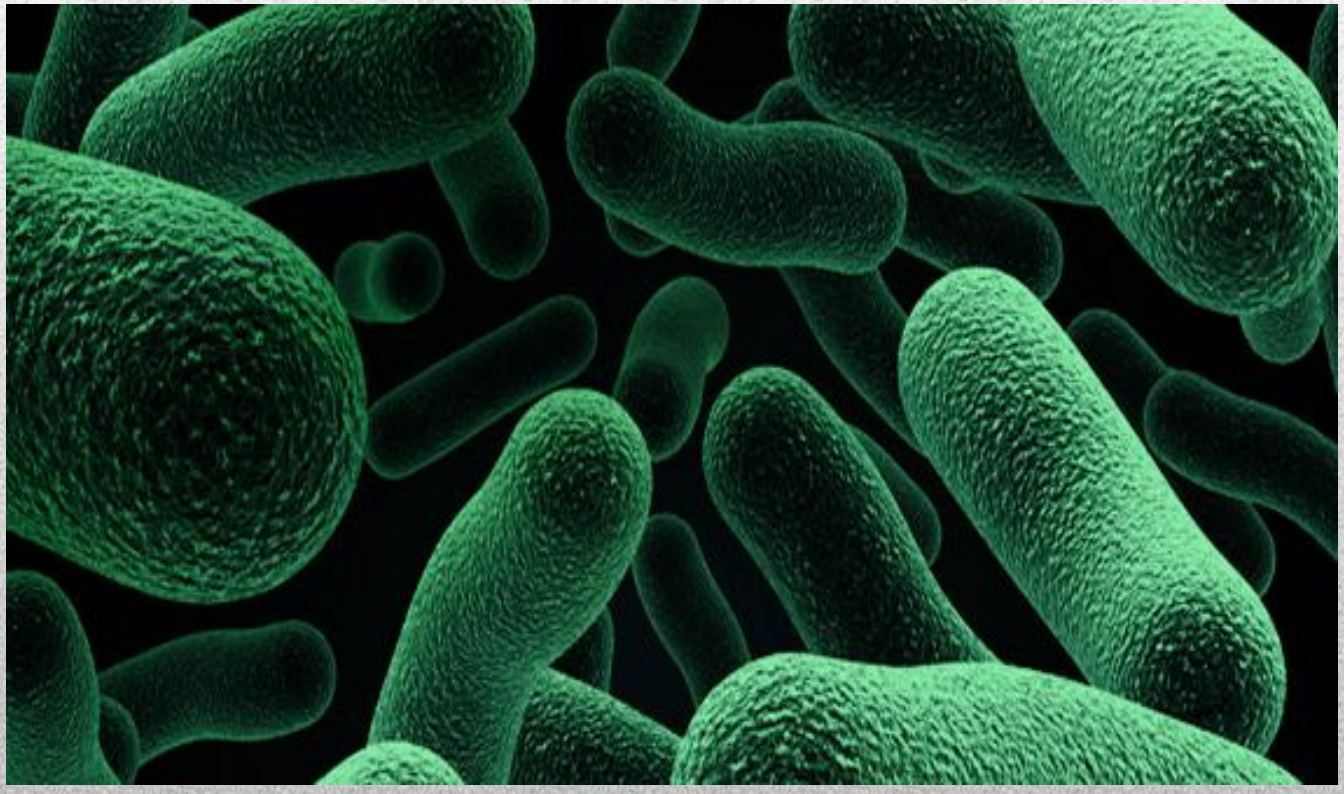
Infections in Diabetic Patients



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Infections in Diabetic Patients

Introduction:

- Diabetic patients are predisposed to infections.
- Infections may increase the morbidity and mortality in diabetic patients.

Why diabetic patients are at increased risk to have infections?

- Host related factors.
- Organisms related factors.

❖ *Host Related Factors:*

- **Vascular insufficiency** (low blood flow) result in:
  - Local tissue ischemia that enhances the growth of microaerophilic and anaerobic organisms.
  - Depressing the O2 dependent bactericidal functions of leukocytes.
  - Impairment of the local inflammatory response and absorption of antibiotics.

Vascular insufficiency → low blood flow → Tissue ischemia →Necrotic Tissue → a good environment for bacterial growth.

- **Sensory peripheral neuropathy**:
  Minor local trauma may result in skin ulcers, which leads to diabetic foot infections.

Loss of sensation of the peripheries makes them more susceptible to trauma → skin ulcers → diabetic foot.

- **Autonomic neuropathy**:
  Diabetic patients may develop urinary retention and stasis that, in turn, predisposes to develop UTIs.

Affect the internal organs such as bladder

- **Hyperglycemia and metabolic derangements:** in diabetes may facilitate infection (providing a good environment for bacterial growth & adherence).

- **Immune defects in diabetes such as:**
  - Depressed Neutrophil function.
  - Affected adherence to the endothelium.
  - Affected chemotaxis and phagocytosis.
  - Compromised intracellular bactericidal activity.
  - Opsonization.
  - Depressed cell mediated immunity.

- **Increased skin and mucosal colonization**:
  - Diabetics on insulin have asymptomatic nasal and skin colonization with ***S.aureus***, particularly MRSA.
  - Colonization predisposes to skin infection and transient bacteraemia which may result in distal sites infection such as damaged muscle.
  - In type 2 diabetes: mucosal colonization with ***C.albiacns*** *is* common.
    Vulvovaginitis caused by non-albicans *Candida* spp.

Presence of glucose will allow the bacteria to be attached to the mucosa.

Vulvovaginitis is caused by candida (either albiacns or non-albiacns but albiacns is the more common).
-But in comparison with non diabetics, non-albiacns are common in diabetics than non-diabetics.

Surgical site infections:
Associated with postoperative hyperglycemia, which is related to deleterious effect on chemotaxis, phagocytosis and adherence of granulocytes.

❖ *Organism Specific Factor:*

- ***Candida albicans*:** glucose inducible proteins promote adhesion of *C.albicans* to buccal or vaginal epithelium which in turn, impairs phagocytosis, giving the organism advantage over the host.

- ***Rhizopus* spp:** Ketoacidosis allow *Rhizopus* spp. (which cause **mucormycosis)** to thrive in high glucose acidic conditions.

Common infections in diabetic patients

- Upper & lower respiratory tract infections
- Periodontal infections
- Genitourinary infections
- Abdominal infections
- Skin and soft tissue infections & diabetic foot [Most prominent]

❖ *Upper Respiratory Tract Infections*

- Invasive (malignant) otitis externa (uncommon but potentially life threatening).
- Rhinocerebral mucormycosis.

Invasive otitis Extrna [Malignant Otitis Externa]

Pathogen: *Pseudomonas Aeruginosa.*

Pathogenesis: Slowly invades from the external canal into adjacent soft tissues → mastoid and temporal bone and eventually spreads across the base of the skull → brain.

Clinically: Pt present with severe pain, otorrhea, and hearing loss.
Intense cellulitis and edema of the ear canal.

Diagnosis: CT and MRI studies to define the extent of bone destruction.

Treatment: surgical debridement & IV antipseudomonas antibiotics.

Rhinocerebral Mucormycosis

- A life threatening fungal infection [it starts as sinusitis then it develops and invade the brain]

Pathogen: *Rhizopus*, *Absidia* and *Mucor* species.

Clinically: pt present with facial or ocular pain and nasal stuffiness, generalized malaise and fever, there may be intranasal black eschars or necrotic turbinates.

Diagnosis: biopsy of necrotic tissue.

Treatment: surgical debridement and prolonged IV therapy with Amphotericin B.

❖ *Lower respiratory tract infections [pneumonia and influenza]*

- Diabetic pts are 4 times more likely to die from pneumonia or influenza than non-diabetic patients.
- **Common organisms**: - Gram positive Bacteria: *S.aureus, S.pneumoniae.*
  - Gram negative bacteria: *Enterobacteria* & *Legionella.*
  - Other organisms: *Influenza virus* & *TB*
- Routine **pneumococcal** vaccination and **influenza** recommended. [for elderly patients only]

Most common cause of community acquired pneumonia = *S.pneumoniae*
Most common cause of hospital acquired pneumonia = *Gram negative bacteria*

❖ *Genitourinary infections:*

- **Asymptomatic bacteriuria** (> 10^5 /ml urine)**:**
  - It is common in diabetic pt.
  - Symptoms, signs and time of onset similar to non-diabetics.
  - Diabetes is an indication for screening for treating asymptomatic bacteriuria.

- **Cystitis**: (same as non-diabetics)
  - Incomplete bladder emptying and high incidence of unsuspected upper UTI.
  - Bacteria (Gram negative rods or group B streptococci) or *Candida albicans* may be involved.

Remember:
Urgency, frequency, fever, bloody urine & suprapupic pain → presentation of cystitis.
Vomiting, flank pain & fever →presentation of pyelonephritis

- **Pyelonephritis** (upper urinary tract infection):
  - Can be **Bilateral** or **Emphysematous** (like a sac full of gases)
  - **Emphysematous Pyelonephritis** exclusively an infection of diabetics (60%) and carries grave prognosis (30% fatal).
  - **Diagnosis:** flank mass & crepitus. CT shows gas in the renal tissues.
  - **Management**: supportive & IV antibiotics, nephrectomy may be needed.
- **Vulvovaginitis:** as mentioned earlier. (In obese ladies)

Most common organisms that cause UTIs in general:
1. *E.Coli*
2. *Group B streptococci*
3. *Candida albicans*

The signs and symptoms of infection are very light in diabetics due to the suppressed immunity

❖ *Abdominal infections*

- **Severe fulminating Cholecystitis**
  Common pathogens: enteric Gram negative bacteria and anaerobes.
  Clinical presentation: - Gall stone or peritonitis may be present.
  - Gas gangrene and perforation may occur.
  Management: Cholecystectomy and broad spectrum antibiotics

❖ *Skin and soft tissue infections*

- **Risk factors in diabetic patients:**
  - Sensory neuropathy → no pain perception.
  - Atherosclerotic vascular disease → low blood flow.
  - Hyperglycemia : >250 mg/ dl increased risk
  - History of celullitus, peripheral vascular diseases, Tinea, and dry skin.
- **Organisms:** *S.pyogenes* (GAS) and *S.aureus*.
  CA-MRSA (community acquired MRSA) is of concern (77%) of skin and soft tissue infections.

Remember:
-Celullitus → inflammation of dermal and subcutaneous layers of the skin.
-Osteomylitis → infection of the bone or bone marrow
Myocitis → muscle infection.
Strept & *staph* → skin & soft tissue infection

Necrotizing fasciitis:

- A deep-seated, life threatening infection of subcutaneous tissue with progressive destruction of fascia, fat, and muscle.

Pathogens: GAS (group A streptococci), with or without *S.aureus*, anaerobes may be involved.
Clinically: pain proportion to skin infected, anaesthesia of overlying skin. Violaceous discoloration (purple color) of skin that evolves into vesicles and bullae, crepitus, soft tissue gas seen in radiograph or CT.
Management: aggressive surgical debridement & IV antibiotics.

Diabetic foot infection: (important)

- The most common and most important soft tissue infection in diabetic patients because it is related to:
  - Peripheral neuropathy and compromised microvascular circulation which limits the access of phagocytic cells to the infected area and poor concentration of antibiotics in the affected area.
- Complicated by:
  - Chronic Osteomyelitis. (by anaerobes)
  - Gas gangrene (by blockage of an artery or clostridium perfringens)
  - Amputation
  - Death.
- The spectrum of foot infection ranges from superficial cellulitis to chronic Osteomyelitis.
- Combined infection involving bone and soft tissue may occur.

Pathophysiology:

- Microvascualr disease limits blood supply to the superficial and deep structures.
- Pressure from ill fitting shoes, trauma compromises local blood supply predisposing foot to infection.
- Diabetic neuropathy may lead to incidental trauma that goes unrecognized.
- Sinus tract may be present.

Organisms involved in diabetic foot infections:

- **Cellulitis**: beta-hemolytic streptococci (group A, B streptococci), *S.aureus*, Entertobacteriacae (E.coli, *Klebsiella, Proteus spp.*) In chronic ulcers.
- **Macerated ulcer or nail injury (sinus):** *P.aeruginosa*.
- **Deep soft tissue infections (necrotizing fasciitis, or myositis):** GAS & gas producing gram positive bacilli (Clostridium).
- **Chronic Osteomyelitis**: GAS and Group B.sterptococci, *S.aureus, Enterobacteriacae* (*E.coli* ,Proteus *mirabilis* , *K.pneumoniae.)* ,*Bacteroides fragilis (anaerobes)*
- **Factors that increases the development of Osteomyelitis:** grossly visible bone or ability to probe to bone, large ulcer size (>2x2 cm), deep ulcer (> 3mm), ulcer duration longer than 1-2 wks, ESR >70 mm/hr

Staph → skin & soft tissue infection.
Strept → skin & soft tissue infection.
Anaerobes → chronic osteomylitis.
Pseudomonas → ulcer & nail injury
Clostridium perfringens → gas gangrene

Clinical presentations of diabetic foot infections:

- **Cellulitis**:
  - Tender, erythematous non-raised skin lesion on the lower limb, may be accompanied with lymphangitis which suggests GAS.
  - Bullae suggest *S.aureus*, occasionally GAS.
- **Deep skin and soft tissue infections**:
  - Patient acutely ill, with painful induration of the limb especially the thigh. Foot may be involved.
  - Wound discharge suggests anaerobes.
- **Acute Osteomyelitis**: pain at the involved bone, fever, adenopathy.
- **Chronic Osteomyelitis**: fever ,foul discharge ,may be pain, no lymphangitis, deep penetrating ulcer ,and sinuses on the planter surface of the foot

Erythematous: redness of skin as a result of inflammation or infection.

Diagnosis of foot infections:

- Thorough examination to evaluate the patient's vascular and neurological status.
- Radiological examination including doppler ultrasonography, transcutaneous oxymetery, MR angiography.
- CT scan, MRI and gallium -67 scan for soft tissue and bone evaluation.
- Exploration of ulcer to determine its depth and presence of sinus tract.
- Deep specimens (tissues) for culture and susceptibility testing [tissue biopsy is the best specimen for diagnosing diabetic ulcer].

Management & treatment

- Control blood sugar and hydration
- Evaluation of neuropathy and vasculopathy
- Mild cases: debridement of necrotic tissues and use of antibiotics according to the causative bacteria
- **Moderate to severe cases:** places the foot at risk of amputation. Needs hospitalization, IV antibiotics and surgical intervention if needed.

Prevention:

- Prevention is the cornerstone of diabetic foot care.
- It is multidisciplinary including family physician, social worker, home care nurse and specialist.
- Patient education about the control and complication of diabetes.
- Blood sugar should be controlled promptly, a diet low in fat and cholesterol.
- Proper foot care, using protective footwear and pressure reduction.
- Self and family member examination of foot.

Summary

- Diabetic patients are predisposed to infections with increased morbidity and mortality rates because of 2 main factors

Host related factors	Organisms related factors
***Vascular insufficiency* (low blood flow)** (Local tissue ischemia that enhances the growth of microaerophilic and anaerobic organisms & depressing immune function)	***Candida albicans*** Glucose inducible proteins promote adhesion of *C.albicans* to buccal or vaginal epithelium which in turn, impairs phagocytosis, giving the organism advantage over the host.
Sensory peripheral neuropathy *(*Minor local trauma may result in skin ulcers, which leads to diabetic foot infections)	
Autonomic neuropathy (Diabetic patients may develop urinary retention and stasis that predisposes to UTIs)	
Hyperglycemia and metabolic derangements (providing a good environment for bacterial growth & adherence)	
Immune defects in diabetes - Depressed neutrophils function - Affected adherence to the endothelium - Affected chemotaxis and phagocytosis - Opsonization	***Rhizopus spp*** Ketoacidosis allow *Rhizopus* spp. (which cause **mucormycosis**) to thrive in high glucose acidic conditions.
Increased skin and mucosal colonization (Colonization predisposes to skin infection and transient bacteraemia which may result in distal sites infection such as damaged muscle) - Diabetics on insulin have asymptomatic nasal and skin colonization with S.aureus - mucosal colonization with *C.albiacns is* common	
Surgical site infections	

- ***common infections in diabetic patients: (important)***

	Infection	*Pathogens*
Upper respiratory tract infections	***Invasive (malignant) otitis externa***	*Pseudomonas Aeruginosa.*
	Rhinocerebral mucormycosis	*Rhizopus,Absidia* *Mucor species*
Lower respiratory tract infections	***pneumonia***	*S.pneumoniae (gm+)*
	influenza	*Influenza virus*
Genitourinary infections	***Cystitis***	*E.Coli*
	Bilateral Pyelonephritis	*Group B streptococci*
	Emphysematous Pyelonephritis	*Candida albicans*
	Vulvovaginitis	
Skin and soft tissue infections & diabetic foot	***Necrotizing fasciitis***	*S.pyogenes (GAS) + anaerobes*
	Diabetic foot infection	*beta-hemolytic streptococci A & B (cellulitis)* *P.aeruginosa (Macerated ulcer or nail injury)* *GAS & Clostridium (necrotizing fasciitis or myositis)* *GAS + anaerobes(Chronic Osteomyelitis)*
Abdominal infections *Periodontal infections*	***Severe fulminating Cholecystitis***	*Gram negative bacteria and anaerobes*

- *malignant otitis externa could invade to the mastoid & temporal bone → base of the skull → brain*
- *Diagnosis of malignant otitis externa is done by CT and MRI.*
- *Rhinocerebral Mucormycosis is diagnosed by biopsy of the necrotic tissue*
- *Routine pneumococcal vaccination and influenza recommended.*
- *Candida (either albicans or non-albiacns) causes Vulvovaginitis in diabetic patients*
- *Urgency, frequency, fever, bloody urine & suprapupic pain are presentation of cystitis.*
- *Vomiting, flank pain & fever are presentation of pyelonephritis*
- *Emphysematous Pyelonephritis exclusively an infection of diabetics (60%) showing flank mass and gas in renal by CT*
- *Necrotizing fasciitis present as pain proportion to skin infected, anaesthesia of overlying skin. Violaceous discoloration (purple color) of skin that evolves into vesicles and bullae, crepitus, soft tissue gas seen in radiograph or CT.*

- *Risk factors for Skin and soft tissue infections in diabetic patients:*
  - *Sensory neuropathy causing no pain perception.*
  - *Atherosclerotic vascular disease leading to low blood flow.*
  - *Hyperglycemia*
  - *History of celullitus, peripheral vascular diseases, Tinea, and dry skin.*

- *Complications of diabetic foot infection*
  - *Chronic Osteomyelitis.*
  - *Gas gangrene (by blockage of an artery)*
  - *Amputation*
  - *Death.*

- *Cellulitis, Deep skin and soft tissue infections, and acute and chronic Osteomyelitis are the clinical manifestations of diabetic foot infection*
- *Chronic osteomylitis caused by anaerobes & GAS*
- Clostridium perfringens *causes gas gangrene*

- *Diagnosis is done by*
  - *Evaluation of the patients' vascular and neurological status*
  - *Radiological examination*
  - *Deep specimens (tissue) for culture and susceptibility testing*
- *Management :*
  - *Control blood sugar and hydration*
  - *Mild cases: debridement of necrotic tissues and use of antibiotics according to the causative bacteria*
  - *Moderate to severe cases: places the foot at risk of amputation. Needs hospitalization, IV antibiotics and surgical intervention if needed.*