SuperAgs are responsible for fever, susceptibility to endotoxins, hypotension, multi-organ failure, erythroderma (rash), AMI immunosuppression.


**SUB-PART GF 02 (C): LAB DIAGNOSIS**

10. Lab diagnosis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specimen</td>
<td>Swabs, aspirates, pus, blood</td>
</tr>
</tbody>
</table>
**Staining**

<table>
<thead>
<tr>
<th>Gram staining/ smears for pus</th>
</tr>
</thead>
</table>

**Culture**

*BA: β-hemolysis
*NA: golden yellow colonies (due to keratinoid formation)
*MA: LF colonies

(L-R): BA, NA, MA

**Biochem**

*Coagulase, catalase +ve

*Urease +ve
*Ferments mannitol (+)

**SUB-PART GF 02 (D): DRUG RESISTANCE**

11. Drug-resistant *Staph. aureus* is an emerging pathogen in hospitals & communities worldwide

12. Treatment failure is common

13. Drug resistance patterns:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Treatment</th>
</tr>
</thead>
</table>

Preview from Notesale.co.uk
Page 9 of 172
<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specimens</td>
<td>Swabs, blood (serum for ASO), pus</td>
</tr>
</tbody>
</table>
| Microscopy | *Gram (+) cocci in chains  
*Have diagnostic value for **skin infections** but NOT pharyngitis   |
| Culture    | **BA**: β-hemolysis (pin-point colonies) – gold standard  
*Selective media: crystal violet BA                                               |
|            | ![Image](BA.jpg) BA (left) & crystal violet BA (arrow)                                                                                       |
| Biochem    | *Bacitracin*-sensitive  
*Catalase –ve                                                                                                                                     |
|            | ![Image](biochem.jpg)                                                                                                                       |
| Serology   | *ASO test:  
a) > 200 IUs is diagnostic  
b) High titre in group A Strep **throat** infection & acute **rheumatic fever**                    |
|            | *ADNaseB (anti-DNAse B):  
a) > 300 – 350 units (is diagnostic)  
b) High titre in group A Strep **skin** infection & acute **glomerulonephritis**         |
|            | *Igs to group-specific carbs in bacterial cell wall  
*Rapid fluorescent Ig test  
*PCR                                                                                         |
PART GF 03: GAS GANGRENE @ ANEROBIC MYOSITIS @ CLOSTRIDIAL MYONECROSIS

1. Etiology: *Cl. perfringens* / *edematiens* / *septicum*

2. General properties (of *Cl. perfringens*)
   
   1. Is a normal flora (of GIT)
   2. Gram (+) bacilli (plump)
   3. Capsulated
   4. Sub-terminal spores (occur only in soil)
   5. Anerobic (sometimes aerophilic)
   6. Non-motile

3. Virulence factors:

<table>
<thead>
<tr>
<th>Factor</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collagenase</td>
<td>Disrupts collagen barrier (in muscle)</td>
</tr>
<tr>
<td>Hyaluronidase</td>
<td>Breaks in connective tissue matrix</td>
</tr>
<tr>
<td><em>Hemolysins</em> (12 toxins)</td>
<td></td>
</tr>
<tr>
<td><em>α-toxin</em> (lecithinase):</td>
<td></td>
</tr>
<tr>
<td>a) Lethal, dermonecrotic, hemolytic toxin</td>
<td></td>
</tr>
<tr>
<td>b) Mechanism:</td>
<td>Hydrolyzes lipid cell membrane ➔ ↑ capillary permeability ➔ Fluid extravasation, ↑ muscle tension</td>
</tr>
</tbody>
</table>

*(If) toxin enters blood ➔ massive hemolysis, renal failure, death*
PART GF 06: VIRAL INFECTIONS
SUB-PART GF 06 (A): HSV

1. General properties (of HSV):

   1. Linear dsDNA
   2. Icosahedral capsid
   3. Lipid envelope (derived from modified host cell NUCLEAR membrane)
   4. Envelope carries surface spikes
   5. Tegument (amorphous structure between envelope & capsid) present
   6. Replicates in host cell nucleus → forms Cowdry type A (Lipschutz) intranuclear inclusion bodies
   7. Has 2 serotypes (HSV-1 & -2)
   8. HSV-2 shows biological & antigenic difference from HSV-1

2. Pathogenesis (of genital & neonatal herpes):

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Natural host</td>
<td>Humans</td>
</tr>
<tr>
<td>Transmission</td>
<td>*HSV-1: saliva</td>
</tr>
<tr>
<td></td>
<td>*HSV-2: sexual @ venereal</td>
</tr>
</tbody>
</table>

QUICK BYTZ

H
S: saliva
V: venereal
Serology

* IFA (detects virus Ag)
* ELISA (detects IgG to VZV @ past infection)
* PCR

5. Treatment: a-/ fam-cyclovir

6. Preventive measures (vaccines):

<table>
<thead>
<tr>
<th>Live Attenuated Vaccine</th>
<th>VZIG</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Composition: live virus (Oka-Merck stain)</td>
<td>* VZV lgs are prepared from patients recovering from the disease</td>
</tr>
<tr>
<td>* Efficacy: ≈ 95%</td>
<td>* Provides passive protection in immunocompromised children</td>
</tr>
<tr>
<td>* Duration of immunity: &gt; 7 yrs</td>
<td>* Limited availability</td>
</tr>
<tr>
<td>* Schedule: 1 dose (at age &lt; 13 years)</td>
<td>* NOT useful in treatment</td>
</tr>
<tr>
<td>* May be administered simultaneously with mumps, measles &amp; rubella (as MMRV vaccine)</td>
<td></td>
</tr>
</tbody>
</table>

*Vaccines for varicella (Varivax®, Proquad®) & herpes zoster (Zostavax®)
8. Prevention:

**MONOVALENT VACCINE**

1. Composition: *live attenuated* virus

2. Efficacy: $\approx 95\%$

3. Duration of immunity: *life-long*

4. Schedule: 2 doses

5. Should be administered as MMR or MMRV (measles, mumps, rubella, varicella)

**MMR VACCINE**

6. **12 – 15 months** is the recommended & min age

7. Booster dose at age **4 – 6**

8. Adverse effects: fever, rash, joint symptoms, thrombocytopenia, parotitis, deafness, encephalopathy

9. Contraindications & precautions:
   a) Severe **allergic** reaction to vaccine component of following prior dose
   b) **Pregnancy**
   c) **Immunosuppression/ immunodeficiency**
   d) Moderate/ severe **acute illness**

*Normal human γ-globulin is given to pregnant women & immunodeficient patients*
<table>
<thead>
<tr>
<th>Expanded Rubella syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Characterized by</strong> <strong>hepatosplenomegaly</strong>, <strong>thrombocytopenic purpura</strong>, <strong>myocarditis</strong>, <strong>bone lesions</strong></td>
</tr>
</tbody>
</table>

*Some congenital shedders are asymptomatic & w/o malformation (hence being diagnosed via virus isolation)*

*Congenitally-infected infants have significant IgM titre & persistent Ig titres long after maternal Ig has disappeared*
<table>
<thead>
<tr>
<th>Specimens</th>
<th>Exudate, tissues</th>
</tr>
</thead>
</table>
| **Microscopy** | *Budding yeast & pseudohyphae appear **Gram (+)** (visualized using calcofluor white staining)*  
*This is significant only when *C. albicans* is abundant*  
*Demo of *mycelial* forms indicates *colonization* & *tissue invasion* (greater significance)* |
| **Culture** | *SDA: **creamy white, smooth** & yeasty color*  
*Corn meal* agar: *C. albicans* form *chlamydospores* at 20°C (distinguishes it from other *Candida)*  
*Human serum: form *germ tubes* at 37°C (*Reynolds-Braude phenomenon)* |
| **Biochem** | *The following aspects differentiate *C. albicans* from other *Candida*:  
a) **Growth** characteristics  
b) **Sugar assimilation**  
c) **Fermentation test** |
| **Serology** | (Rarely helpful, as agglutinins appear in sera of patients & normal individuals) |
| **Skin test** | *Done using *Candida Ags*  
*Uniformly +ve in immunoCOMPETENT adults* (indicates that the individual can mount a cellular immune response)* |
| **Serology** | (Rarely helpful, as agglutinins appear in sera of patients & normal individuals) |
| **Skin test** | *Anergy* (absence of response to Candida Ags) indicates CMI deficiency |
### Susceptible individuals

Farmer, gardeners, florists

| Infection mode proper | Skin trauma by thorns (e.g. roses) $\rightarrow$ spores introduced into skin $\rightarrow$ subcutaneous mycoses |

9. **Clinical manifestations:**

1. Small \textit{papulae/ ulcer/ subcutaneous nodule} at lesion site

2. \textit{Ulceration} \& \textit{necrosis} of the nodules (localized @ \textit{fixed cutaneous sporotrichosis})

3. \textit{Lymphocutaneous sporotrichosis:}
   a) \textit{2° ulcers} on lymph nodes; lymphatics are \textit{hardened} \& \textit{cord-like}
   b) Occurs when infection spreads from skin/ subcutaneous tissue \rightarrow lymph nodes

4. \textit{Systemic dissemination} may occur to bones, joints, meninges (usually occurs in immunocompromised patients, e.g. HIV, underlying sarcoidosis/ Ca)

10. **Lab diagnosis:**

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specimens</td>
<td>Aspiration fluid, pus, biopsy material, skin scrapings, swabs</td>
</tr>
</tbody>
</table>
**Microscopy**

*Direct microscopy of KOH mounts of necrotic material*

*Exam of tissue sections stained by GMS*

*Asteroid body:*

a) Is the characteristic feature of sporotrichosis

b) Rounded/ovoid, basophilic, yeast-like body (with rays of an eosinophilic substance radiating from the yeast cell)

**Culture**

*SDA:*

a) Incubation at 25°C *(mycelia phase)* yields cigar-shaped cells

b) Incubation in 37°C *(yeast phase)* gives very thin, septate hyphae, carrying flower-like clusters of small conidia borne on delicate sterigmata (resembles daisies)

**Helpful in diagnosis of extracutaneous/systemic infections** (which lack distinct clinical features)

*Slide LAT:*

a) Peptide-L-rhamno-D-mannan (outer layer of fungal cell wall) is used as Ag

b) Titres of $\geq 1:4$ are presumptive evidence of sporotrichosis
"Trichophyton is the causative agent of athlete’s foot

16. Lab diagnosis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specimens</td>
<td>Skin scrapings, nails, hair</td>
</tr>
<tr>
<td>Microscopy</td>
<td></td>
</tr>
<tr>
<td><em>KOH</em> mount</td>
<td>(to demo hyphal elements)</td>
</tr>
<tr>
<td><em>LPCB</em> (to demo conidia):</td>
<td></td>
</tr>
<tr>
<td>Dermatophytes</td>
<td>Microconidia</td>
</tr>
<tr>
<td>Trichophyton</td>
<td>Rare, thin-walled, smooth, pencil-/ club-like</td>
</tr>
<tr>
<td>Epidermophyton</td>
<td>Numerous, smooth-walled, club-shaped (in groups)</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
</tr>
</tbody>
</table>
4. Clinical features:

<table>
<thead>
<tr>
<th>Stage</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intestinal invasion</td>
<td>*Occurs 2–3 weeks after infection due to ingestion of undercooked pork</td>
</tr>
<tr>
<td></td>
<td>*Inflammation of duodenal mucosa (due to penetration &amp; development of adult worms)</td>
</tr>
<tr>
<td></td>
<td>*Larvae shed, mucosal inflammation intensifies with neutrophils, eosinophils &amp; lymphocytes</td>
</tr>
<tr>
<td></td>
<td>*Malaise, vomiting, diarrhea, abdominal cramps</td>
</tr>
</tbody>
</table>
Catheterized patient | Depends on duration of catheterization
---|---
Immunocompromised | 
Renal tumors/ stones | 
BPH | 
Uncircumcised ♂ | 
DM | 

9. Bacterial factors:

1. Antiphagocytic activity
2. Capsular (K) Ag
3. Hemolysins (cause kidney membrane damage)
4. Urease (secreted by Proteus, causes renal stones & pyelonephritis)
5. Adhesion to uroepithelium

Uropathogenic E. coli (UPEC)

1. Serotypes:
   a) O serotypes (e.g. O1, O2)
   b) K serotypes (e.g. K1, K2)
2. Pathogenicity islands in bacterial chromosomal genes (these code for virulence factors, e.g. P.fimbrae @ pyelonephritis-associated pili which adhere to bladder & urethral epithelium)
*Screening tests

Newer tests

*IV pyelography
*Voiding cystourethrography

12. Prevention (of nosocomial UTI):

Cautious insertion of urethral catheter, with consideration of the following:
   a) Motives/ indications
   b) Choice of catheter
   c) Insertion technique
   d) Sterility
   e) Duration
   f) Closed system of bag emptying

13. Treatment
   a) ↑ fluid intake (& urine output)
   b) Urine acidification (with cranberries/ citrus fruits)
**gp41** (transmembrane pedicle @ anchoring protein)

| Matrix & core proteins [coded by *gag*] | *p17/ p18*  
| | *p24*  
| | a) Major core Ag  
| | b) Can be detected in serum during **early stages** of disease (BEFORE Igs appear)  
| | c) In later stages, ↓ free anti-p24 Igs & re-↑ in p24 Ags indicate **disease exacerbation**  
| Pol Ags [coded by *pol*] | p31, p51, p66 (these form Pol reverse transcriptase, proteases, endonucleases, etc)  

### REGULATORY GENES

| **tat** | Regulates viral **transcription**  
| **rev** | mRNA transport  
| **nef** | -ve regulatory factor (inhibits MHC c1)  
| **vif** | Virion infectivity factor (inhibits certain host proteins)  
| **vpr** | Transcription activator  
| **vpu** | Required for budding  

3. **Pathogenesis:**

#### PHASE 0: SOURCES & TRANSMISSION

1. **Sources:** *infected humans*

2. **Transmission:**
   a) **Sexually**
   b) **Blood transfusion**
   c) **Transplacentally/ perinatal**
   d) **IV drug users**
   e) **Needlestick** exposure (e.g. tattooing, acupuncture)
PHASE 1: BINDING & ENTRY

1. Specific binding between gp120 & CD4 receptors
2. Fusion with cell membrane with help of gp41
3. Other receptors (e.g. CXCR-4 of lymphocytes/ CCR-5 on macrophages) also participate
4. HIV genome internalized & uncoated

*CD4 receptors are present in HTCs, (some) B cells, monocytes & macrophages
*CCR = co-chemokine receptors

PHASE 2: REPLICATION

1. Occurs via reverse transcription
2. Pro-viral (ds)DNA synthesis
3. Integration with host DNA (via integrase)
4. Viral proteins synthesized
5. Viral assembly (using host lipid membrane) & release
6. Host cell damaged in the process

PHASE 3: HOST CHANGES

1. 1° pathogenic mechanism: damage to CD4 cells
2. Viral infection...
   a) suppresses the function of infected cell w/o causing structural damage
   b) B cell & macrophage functions also affected
   c) ↓CD8 & NK cell activities
3. Marked damping effect on CMI
AIDS

*HIV test +ve

* ≥ 1 of the following conditions:

a) HIV encephalopathy
b) Pneumonia (life-threatening/ recurrent)
c) Weight loss
d) Invasive cervical Ca
e) Cryptococcal meningitis
f) Kaposi’s sarcoma
g) Esophageal candidiasis
h) TB

*Normal CD4 count: 500 – 1 600 cells/mm³

**Elite controllers: a small group of population who are resistant to HIV effects

6. Lab diagnosis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>CULTURE</td>
<td>Patient’s lymphocytes cultured with uninfected ones in presence of IL-2</td>
</tr>
</tbody>
</table>
| Ag DETECTION  | *Window period* (seronegative stage with high infectivity) occurs during the 1st 2 – 8 weeks or months after infection

*Infection can be detected during window period using this method
### Ig DETECTION

<table>
<thead>
<tr>
<th>ELISA</th>
<th></th>
<th></th>
</tr>
</thead>
</table>
| *Detects ↑ in Ig lvls:  
a) IgM appears 1st & lasts for 8 – 10 weeks  
b) IgG is present throughout the infection  |  |  |
| *Used as a screening test |  |  |

<table>
<thead>
<tr>
<th>Dot blot assay</th>
<th></th>
<th></th>
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</thead>
<tbody>
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</table>

<table>
<thead>
<tr>
<th>Comb test</th>
<th></th>
<th></th>
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<tbody>
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</tbody>
</table>

### CONFIRMATORY TESTS

<table>
<thead>
<tr>
<th>PCR</th>
<th></th>
<th></th>
</tr>
</thead>
</table>
| *Types:  
RNA PCR | Used in diagnosis & viremia monitoring |  |
| DNA PCR | Proviral DNA amplified & then characterized by NA hybridization |  |
|  |  |  |
| *Gives +ve results at ANY stage of infection  
*However, is costly & done only when other methods cannot give definitive result |  |  |

<p>| | | |</p>
<table>
<thead>
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<tbody>
<tr>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

| *Procedure:  
a) HIV proteins separated (by molecular weight & electrophoretic mobility) by gel electrophoresis  
b) Blotted onto strips of nitrocellulose paper  
c) Strips reacted with test sera & conjugated anti-human Ig |  |  |
6. Congenital syphilis:

1. Transmitted from infected mother (usually after 3rd month of pregnancy)

2. Effects
   a) Stillbirth
   b) Congenital abnormalities (Hutchison teeth, saddle-shaped nose)
   c) Snuffles (rhinitis)
   d) Hepatosplenomegaly
   e) (Rarely) skin & mucosal lesions

#This is the time when the fetus' immune system develops

![Hutchison teeth](image1.png)  
Saddle-shaped nose (image2.png)

7. Lab diagnosis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specimens</td>
<td>Exudates, blood/ serum</td>
</tr>
</tbody>
</table>
| Microscopy | *Dark-field microscopy
*UV microscopy (DFA for treponemal Ags) |
<table>
<thead>
<tr>
<th>Dissemination</th>
</tr>
</thead>
</table>
| *Spread from lymph nodes → lymphatics → tissues of rectum → (hemorrhagic) **proctitis**  
*Chronic granulomatous reactions in lymphatics & neighboring tissues → anal fistula/ genital elephantiasis |

![Proctitis (left) & genital elephantiasis](image)

<table>
<thead>
<tr>
<th>Other systemic (metastatic) complications:</th>
</tr>
</thead>
</table>
| *Fever  
*Hepatitis, pneumonitis, meningoencephalitis  
*Involvement of joints & eyes  
*3° stage: **scarring** & **lymphatic blockade**  
*Late sequelae (in women): **rectal strictures** & elephantiasis of vulva (esthiomene) |

6. Lab diagnosis [Refer Sub-part CB 02 (D), #8]

7. Treatment: [Refer Sub-part CB 02 (D), #9]
SECTION L: INFECTIONS IN COMPROMISED HOST  
CHAPTER LA: FACTORS AFFECTING IMMUNE SYSTEM

1. 1° & 2° immunodeficiencies:

<table>
<thead>
<tr>
<th>Etiology</th>
<th>1° Immunodeficiency</th>
<th>2° Immunodeficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>*Inherited</td>
<td>*Underlying disease state</td>
</tr>
<tr>
<td></td>
<td>*Exposure in utero to <strong>environmental</strong> factors</td>
<td>*Result of <strong>treatment</strong> for a disease</td>
</tr>
<tr>
<td></td>
<td>*Other unknown mechanisms</td>
<td></td>
</tr>
<tr>
<td>Factors which ↓ innate defences</td>
<td>*Complement deficiencies</td>
<td>*Burns</td>
</tr>
<tr>
<td></td>
<td>*Phagocyte cell deficiencies</td>
<td>*Trauma</td>
</tr>
<tr>
<td>Factors which ↓ adaptive immune response</td>
<td>*T-cell defects</td>
<td>*Major surgery</td>
</tr>
<tr>
<td></td>
<td>*B-cell deficiencies</td>
<td>*Catheterization</td>
</tr>
<tr>
<td></td>
<td>*SCID (severe combined immunodeficiency)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>*Foreign bodies (eg. shunts, prostheses)</td>
<td>*Obstruction</td>
</tr>
<tr>
<td></td>
<td>*Obstruction</td>
<td></td>
</tr>
</tbody>
</table>
Dissemination in immunocompromised:

- *Brain:*
  - a) Headache
  - b) Weakness
  - c) Confusion, seizures

- *Skin (occur when open wounds/ cuts come in contact with infected soil):*
  - a) Ulceration
  - b) Nodules (which sometimes drain & spread along lymph nodes)

*Kidney

This disease is non-communicable (no person-to-person transmission)

5. Lab diagnosis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specimens</td>
<td>Sputum, blood</td>
</tr>
<tr>
<td>Microscopy</td>
<td><em>Gram/ weak acid-fast</em> staining&lt;br&gt;<em>Branching rods/ filaments</em></td>
</tr>
</tbody>
</table>
H. capsulatum

*Hepatosplenomegaly & pulmonary disease* (are 1° signs in children & adults respectively)

*Types of manifestations:
   a) Acute pulmonary, progressive form
   b) Chronic pulmonary form
   c) Disseminated form

(Self-limiting in immunocompetent individuals)

P. marneffei

*(Similar to that of extrapulmonary TB)*

*Disseminated disease with…*
   a) fever, weight loss
   b) generalized lymphadenopathy
   c) hepatomegaly
   d) skin lesions

<table>
<thead>
<tr>
<th>Specimens</th>
<th>Rhizopus, Mucor</th>
<th>H. capsulatum</th>
<th>P. marneffei</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biopsy, nasal discharge</td>
<td></td>
<td><em>Lymph nodes aspirate</em></td>
<td>Marrow/ skin/ lymph node biopsy</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Marrow/ blood/ lesion secretion/ sputum/ biopsy</em></td>
<td></td>
</tr>
</tbody>
</table>

*Mesenteric lymphadenitis (left) & skin lesion*
<table>
<thead>
<tr>
<th>LAT</th>
<th>RIA, ELISA (Ag detection)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Others</td>
<td><strong>Skin test:</strong> delayed-type response</td>
</tr>
<tr>
<td></td>
<td><em>Fungi demo in urine/ serum (in disseminated cases)</em></td>
</tr>
<tr>
<td>Others</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td></td>
</tr>
<tr>
<td><em>Severe, fulminating gall bladder infection</em> (especially by gas-forming organisms)</td>
<td></td>
</tr>
<tr>
<td><em>Anerobic cellulitis</em></td>
<td></td>
</tr>
</tbody>
</table>

*↑ risk of *Staph, K. pneumoniae* infections
*↑ risk of TB reactivation
*↑ virulence of *cryptococcal* infections & *coccidioidomycoses* |


---

**PART LM 02: MUCORMYCOSIS @ ZYGOMYCOSIS**

2. **Causative agents:** *Rhizopus, Mucor, Absidia*

3. **Pathogenesis:**

<table>
<thead>
<tr>
<th><strong>Spores (in air/ dust) are inhaled</strong></th>
</tr>
</thead>
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<tr>
<td><strong>1° focus</strong> in URT/ nasal cavity (spores germinate &amp; mycelia invade orbits/ sinuses/ brain)</td>
</tr>
<tr>
<td><strong>Dissemination</strong></td>
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<tr>
<td><strong>Systemic infection</strong> (is usually a fatal complication of DM)</td>
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PART LN 02: CRYPTOSPORIDIUM PARVUM

1. General:
   a) Sporozoa of class Coccidea (related to malaria)
   b) Infects wide range of animals
   c) Found worldwide

2. Transmission:
   a) Oocysts in contaminated water
   b) Fecal-oral route

3. Oocysts:
   a) Size: 4 – 6 μm
   b) Thin-walled oocysts remain in human body & excyst endogenously → autoinfection
   c) Thick-walled oocysts are mainly removed in feces

4. Life cycle:
   a) Is completed in a single host
   b) Oocysts containing 4 sporozoites each are liberated in intestines
   c) Penetrate intestinal epithelial cells
   d) Undergoes both asexual (schizogony) & sexual (gametogony) multiplication

Quick Bytz

Thin-walled oocysts are unable to withstand the forces of external environment so they remain in human body (& vice versa)