Patofisiologi Infeksi

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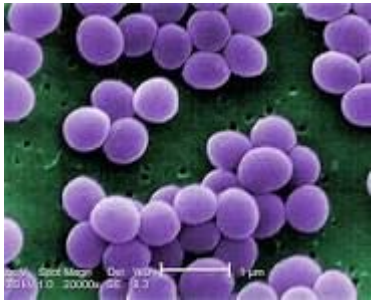
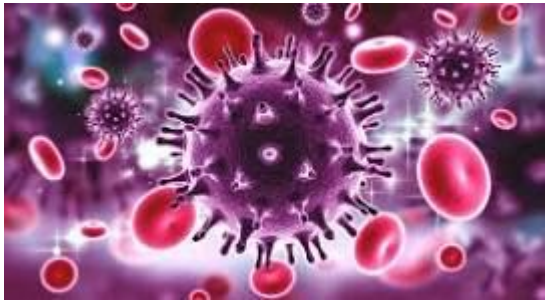
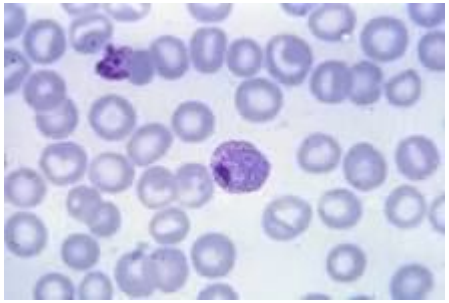
Infeksi: proses invasi dan multifikasi berbagai mikro-organisme (bakteri, jamur, virus dan parasit ke dalam tubuh







Bakteri Komensal	Peran	Lokasi
Escherichia coli (E. coli)	Membantu pencernaan	Usus besar
Staphylococcus epidermidis	Menyehatkan kulit & membantu melawan patogen	Kulit
Lactobacillus spp.	Flora normal usus dan vagina	Usus, vagina
Bakteri Patogen	**Peran**	**Lokasi**
Streptococcus pyogenes	Penyebab infeksi tenggorokan, impetigo, dll.	Tenggorokan, kulit
Salmonella spp.	Penyebab infeksi saluran pencernaan	Usus, makanan
Clostridium botulinum	Penyebab botulisme (keracunan makanan)	Makanan, luka

Faktor Resiko (Healthcare-associated infections (HAIs)

- Umur: neonatus dan lansia (lebih rentan)
- Status imun terganggu (imuno-kompromais); penderita penyakit kronis, penderita keganasan, obat-obat imunosupresan
- Interupsi barrier anatomis
- Implantasi benda asing
- Perubahan mikroflora normal

Cara Penularan (transmisi)

1. Kontak : langsung dan tidak langsung
2. Droplet (>5 um)
3. Airborne (< 5 um)
4. Vehikulum: makanan (salmonella), air (tifoid, kolera, hepatitis A, disentri), darah (Hepatitis B, Hepatitis C, HIV)
5. Vektor Nyamuk; demam berdarah, malaria, , lalat (makanan), tikus (leptospirosis)

Alur patogenesis infeksi

1. Paparan (exposure)
    - Patogen masuk tubuh lewat portal: saluran napas, cerna, kulit (luka), darah, atau kontak langsung.
    - Menentukan sejauh mana inang terpapar dan berisiko terinfeksi.
2. Adhesi (pelekatan)
    - Patogen menempel pada permukaan sel/jaringan inang dengan adhesin (protein, pili, fimbriae).
    - Tanpa adhesi yang kuat, patogen mudah disingkirkan oleh mekanisme pertahanan tubuh.
3. Invasi ke jaringan
    - Patogen menembus barier (epitel, mukosa, sawar darah-otak) lewat enzim proteolitik atau strategi intraseluler.
    - Masuk ke dalam sel atau jaringan dan memulai infeksi di lokasi tertentu

Alur patogenesis infeksi (lanjutan-1)

4. Kolonisasi dan perbanyakan
    - Patogen berkembang biak di jaringan inang, membentuk fokus infeksi lokal.
    - Jika mampu bertahan, populasi patogen bertambah dan menyebar ke jaringan sekitar atau sistemik.
5. Kerusakan jaringan dan toksin
    - Kerusakan terjadi lewat:
    - lisis sel langsung,
    - eksotoksin (toksin spesifik),
    - endotoksin/LPS (pada bakteri Gram negatif).
    - Kerusakan ini mencetuskan inflamasi dan gejala lokal.

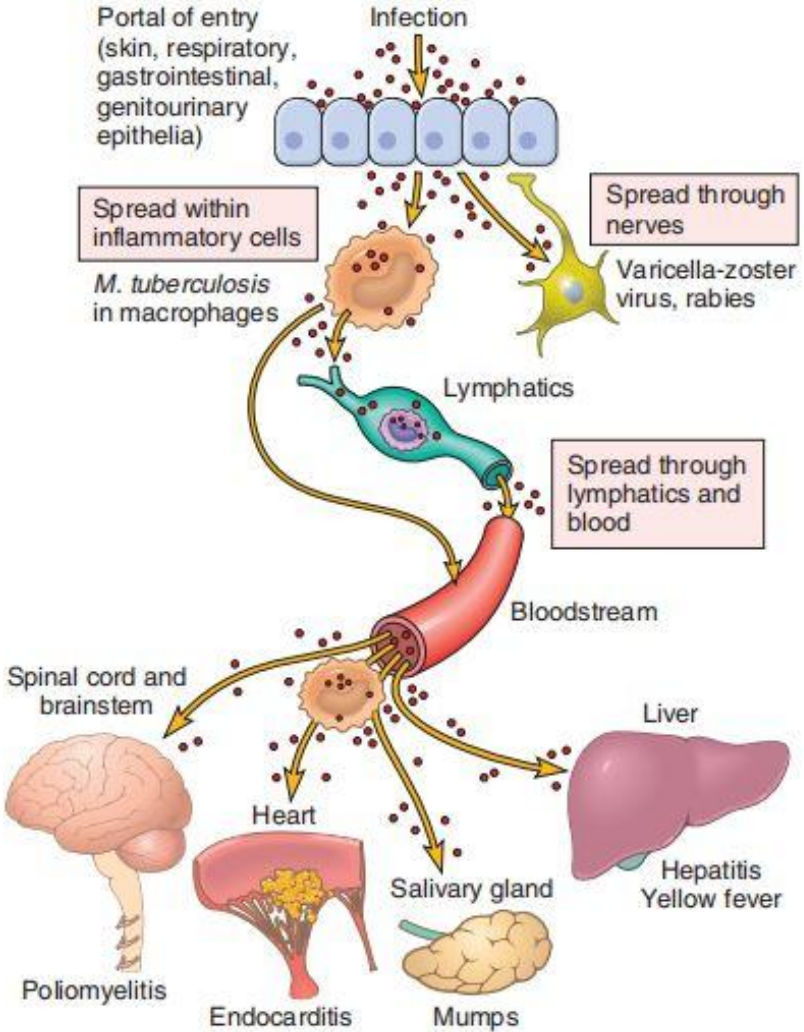
Alur patogenesis infeksi (lanjutan-2)

6. Inflamasi dan respons imun
    - Sel imun mengenali komponen patogen → pelepasan sitokin dan mediator inflamasi → vasodilatasi, edema, rekrutmen leukosit.
    - Neutrofil, makrofag, dan limfosit berusaha menghancurkan patogen; antibodi dan sel T membantu proses pembersihan.
7. Manifestasi klinis (penyakit)
    - Gabungan kerusakan jaringan + inflamasi + respons sistemik → gejala seperti demam, nyeri, bengkak, peningkatan leukosit, dan gejala organ tertentu (batuk, diare, sesak, nyeri abdomen).
    - Tingkat keparahan tergantung virulensi patogen dan status imun inang.

Alur patogenesis infeksi (lanjutan-3)

8. Resolusi atau komplikasi
    - Jika imun efektif: patogen berkurang, inflamasi reda, jaringan pulih (resolusi).
    - Jika tidak terkendali: infeksi berlanjut menjadi kronik, abses, sepsis, gagal organ, atau meninggal.

Paparan → Adhesi → Invasi → Kolonisasi → Kerusakan & Toksin → Inflamasi & Imun → Gejala → Resolusi


Portal of entry
(skin, respiratory,
gastrointestinal,
genitourinary
epithelia)
Infection
Spread within
inflammatory cells
M. tuberculosis
in macrophages
Spread through
nerves
Varicella-zoster
virus, rabies
Lymphatics
Spread through
lymphatics and
blood
Bloodstream
Spinal cord and
brainstem
Liver
Heart
Hepatitis
Yellow fever
Salivary gland
Poliomyelitis
Endocarditis
Mumps

Cara Pencegahan infeksi

- Cuci tangan dengan cara yang benar
- Penerapan kewaspadaan isolasi
- Inaktivasi agen penyebab infeksi: pengelolaan yang tepat a) bahan dan alat infektif, b) lingkungan yang terinfeksi
- Memutus mata rantai penularan (isolasi penderita)
- Imunisasi atau tindakan profilaksis paska pajanan

Table 8.1 Routes of Microbial Infection

Site	Major Local Defense(s)	Basis for Failure of Local Defense	Pathogens (Examples)
Skin	Epidermal barrier	Mechanical defects (punctures, burns, ulcers)	*Staphylococcus aureus, Candida albicans, Pseudomonas aeruginosa*
		Needle sticks	Human immunodeficiency virus, hepatitis viruses
		Arthropod and animal bites	Yellow fever, plague, Lyme disease, malaria, rabies
		Direct penetration	*Schistosoma* spp.
Gastrointestinal tract	Epithelial barrier	Attachment and local proliferation of microbes	*Vibrio cholerae, Giardia duodenalis*
		Attachment and local invasion of microbes	*Shigella* spp., *Salmonella* spp., *Campylobacter* spp.
		Uptake through M cells	Poliovirus, *Shigella* spp., *Salmonella* spp.
	Acidic secretions	Acid-resistant cysts and eggs	Many protozoa and helminths
	Peristalsis	Obstruction, ileus, postsurgical adhesions	Mixed aerobic and anaerobic bacteria (*Escherichia coli, Bacteroides* spp.)
	Bile and pancreatic enzymes	Resistant microbial external coats	Hepatitis A, rotavirus, norovirus
	Normal protective microbiota	Broad-spectrum antibiotic use	*Clostridioides difficile*
Respiratory tract	Mucociliary clearance	Attachment and local proliferation of microbes	Influenza viruses
		Ciliary paralysis by toxins	*Haemophilus influenzae, Mycoplasma pneumoniae, Bordetella pertussis*
	Resident alveolar macrophages	Resistance to killing by phagocytes	*Mycobacterium tuberculosis*
Urogenital tract	Urination	Obstruction, microbial attachment, and local proliferation	*Escherichia coli*
	Normal vaginal microbiota	Antibiotic use	*Candida albicans*
	Intact epidermal/epithelial barrier	Microbial attachment and local proliferation	*Neisseria gonorrhoeae*
		Direct infection/local invasion	Herpes viruses, syphilis
		Local trauma	Various sexually transmitted infections (e.g., human papillomavirus)

Table 8.3 Spectrum of Inflammatory Responses to Infection

Type of Response	Pathogenesis	Examples
Suppurative (Purulent) Infection	Increased vascular permeability Leukocyte infiltration (neutrophils) Chemoattractants from bacteria Formation of "pus"	Pneumonia (*Staphylococcus aureus*) Abscesses (*Staphylococcus* spp., anaerobic and other bacteria)
Mononuclear and granulomatous inflammation	Mononuclear cell infiltrates (monocytes, macrophages, plasma cells, lymphocytes) Cell-mediated immune response to pathogens ("persistent antigen") Formation of granulomata	Syphilis Tuberculosis
Cytopathic-cytoproliferative reactions	Viral transformation of cells Necrosis or proliferation (including multinucleation) Linked to neoplasia	Cervical cancer (human papillomavirus) Chicken pox, shingles Herpes
Tissue necrosis	Toxin- or lysis-mediated destruction Lack of inflammatory cells Rapidly progressive processes	Gangrene (*Clostridium perfringens*) Hepatitis (hepatitis B virus)
Chronic inflammation/ scarring	Repetitive injury leads to fibrosis Loss of normal parenchyma	Chronic hepatitis with cirrhosis (hepatitis B and C viruses)
No reaction	Severe immune compromise	*Mycobacterium avium* in untreated AIDS (T-cell deficiency) Mucormycosis in bone marrow transplant patients (neutropenia)

Table 8.4 Selected Human Viruses and Viral Diseases

Organ System	Species	Disease
Respiratory	Adenovirus	Upper and lower respiratory tract infections, conjunctivitis, diarrhea
	Rhinovirus	Upper respiratory tract infection
	Influenza viruses A, B	Influenza
	Respiratory syncytial virus	Bronchiolitis, pneumonia
Digestive	Mumps virus	Mumps, pancreatitis, orchitis
	Rotavirus	Childhood gastroenteritis
	Norovirus	Gastroenteritis
	Hepatitis A virus	Acute viral hepatitis
	Hepatitis B virus	Acute or chronic hepatitis
	Hepatitis D virus	With hepatitis B virus, acute or chronic hepatitis
	Hepatitis C virus	Acute or chronic hepatitis
	Hepatitis E virus	Acute viral hepatitis
Systemic with skin eruptions	Measles virus	Measles (rubeola)
	Rubella virus	German measles (rubella)
	Varicella-zoster virus	Chickenpox, shingles
	Herpes simplex virus 1	Oral herpes ("cold sore")
	Herpes simplex virus 2	Genital herpes
Systemic with hematologic disorders	Cytomegalovirus	Cytomegalic inclusion disease
	Epstein-Barr virus	Infectious mononucleosis
	Human immunodeficiency viruses 1 and 2	Acquired immunodeficiency syndrome
Arboviral and hemorrhagic fevers	Dengue viruses 1 to 4	Dengue hemorrhagic fever
	Yellow fever virus	Yellow fever
Skin/genital warts	Human papillomavirus	Condyloma; cervical carcinoma
Central nervous system	Poliovirus	Poliomyelitis
	JC virus	Progressive multifocal leukoencephalopathy (opportunistic)

Table 8.6 Selected Human Protozoal Diseases

Location	Species	Disease
Luminal or epithelial	*Entamoeba histolytica*	Amebic dysentery; liver abscess
	Balantidium coli	Colitis
	Giardia duodenalis	Diarrheal disease, malabsorption
	Cystoisospora belli	Chronic enterocolitis or malabsorption or both
	Cryptosporidium spp.	
	Trichomonas vaginalis	Urethritis, vaginitis
Central nervous system	*Naegleria fowleri*	Meningoencephalitis
	Acanthamoeba spp.	Meningoencephalitis or ophthalmitis
Bloodstream	*Plasmodium* spp.	Malaria
	Babesia spp.	Babesiosis
	Trypanosoma spp.	African sleeping sickness
Intracellular	*Trypanosoma cruzi*	Chagas disease
	Leishmania donovani	Kala-azar
	Leishmania spp.	Cutaneous and mucocutaneous leishmaniasis
	Toxoplasma gondii	Toxoplasmosis

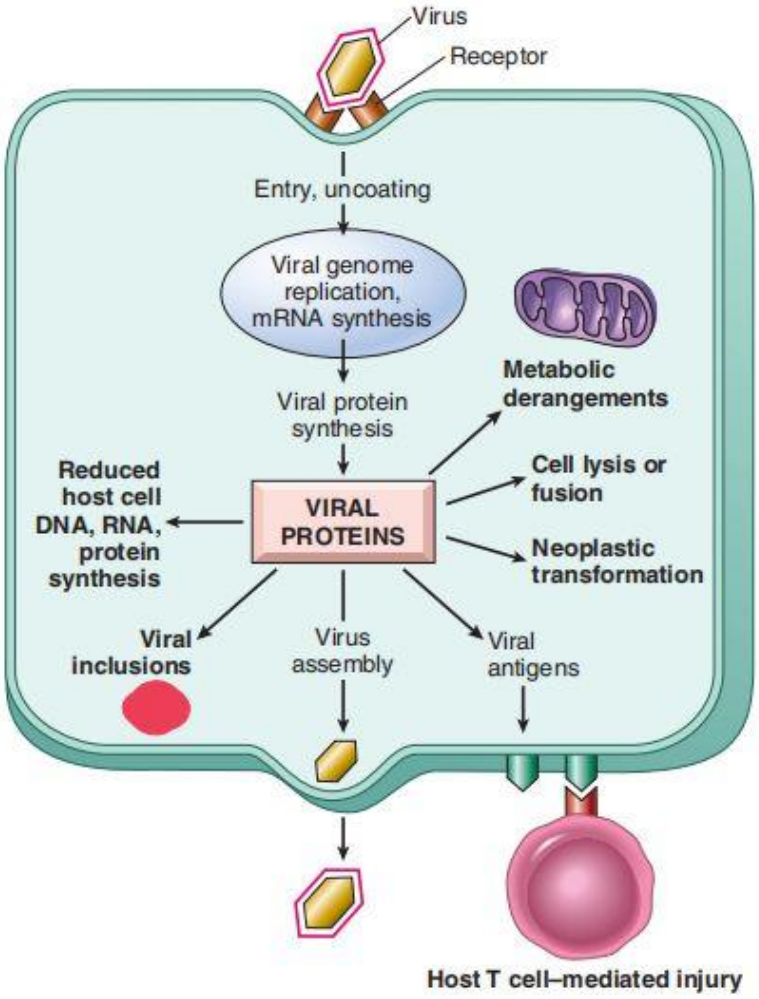


Figure 8.3 Mechanisms by which viruses cause injury to cells.

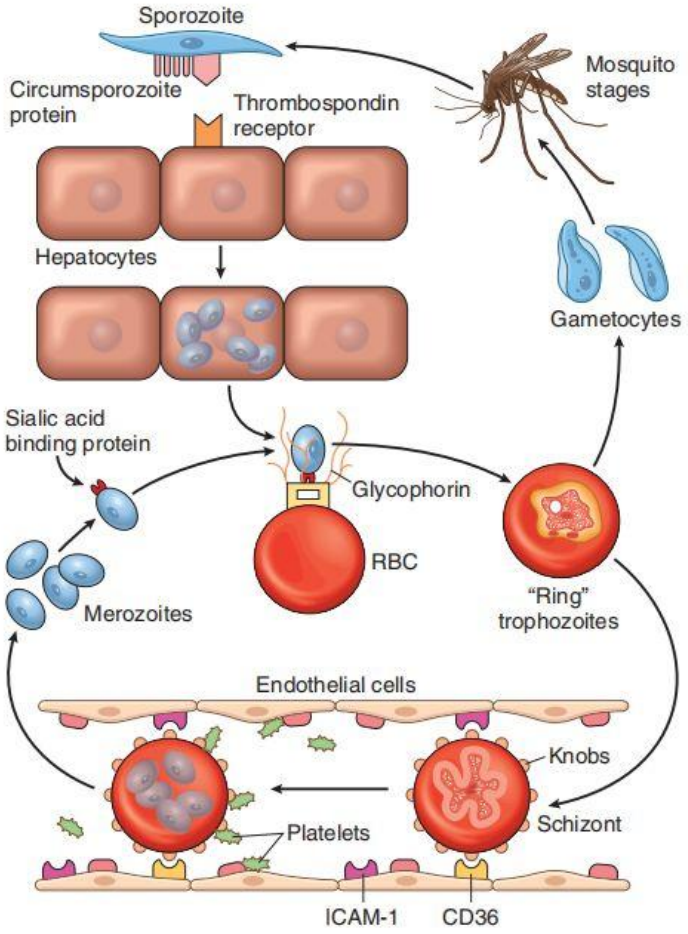


Figure 8.46 Life cycle of *Plasmodium falciparum*. Both exoerythrocytic and erythrocytic stages are depicted. *ICAM-1*, Intercellular adhesion molecule 1; *RBC*, red blood cell. (Drawn by Dr. Jeffrey Joseph, Beth Israel-Deaconess Hospital, Boston, Mass.)