Bacterial infection of blood

The blood is a hostile environment to most micro – organisms due to its high content of antibodies & complement, & large numbers of circulating phagocytes, therefore, bacteria entering the blood are usually destroyed rapidly.
The presence of bacteria in blood is classified into bacteraemia, septicaemia & pyaemia.

Bacteraemia: Indicates the presence of low virulence bacteria in the blood in small numbers in normal subjects, or in individuals with minor, subclinical lesions but they do not multiply. For example, streptococcus viridians may be cultured from the blood after vigorous tooth brushing, particularly when there is dental sepsis. The importance of bacteraemia is that, whenever, bacteria enter the blood, they may settle in various parts of the body & cause lesions.

Septicaemia: Means the presence & multiplication of bacteria in the blood, especially the rapid multiplication of highly pathogenic bacteria, the term thus implies a serious infection with profound toxaemia, in which the bacteria have overwhelmed the host defenses. If the septicaemia is not rapidly fatal, foci of suppuration may develop in various parts of the body as a result of haematogenous spread.

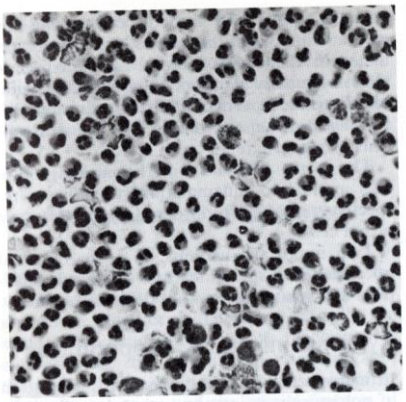
Pyaemia: literally means pus in the blood. A localised pyogenic infection is mainly the cause, when toxic injury to the endothelium of the veins involved in the lesion results in thrombosis, & then bacteria multiply in the thrombus & becomes heavily infiltrated by polymorphs & brocken by their digestive enzymes, then fragments of these septic thrombi may be carried off in the blood causing pyaemia.

Pyogenic bacterial infection (suppuration)
It is an important variant of acute bacterial inflammation. It shows the usual course of acute inflammation with special features.

Pyogenic Bacteria: It is applied to the bacteria which causes suppuration. The common pyogenic bacteria are *Staphylococcus aureus* and *Streptococcus pyogens*.
Abscess: It is a cavity formed which contains polymorphs rich (purulent) exudates or pus in some bacterial infections(emigration of polymorphs is intense).
Suppuration: is the process of abscess formation.

Composition of pus

1- Inflammatory exudate containing very large numbers of neutrophil polymorphs which gives it an opaque appearance (recent pus –living neutrophils, old pus- mostly they are dead, degenerated and digested). Release of DNA from these cells accounts for the sticky nature of pus.
2- Some red cells especially in newly formed pus.
3- Fragments of tissue debris.
4- Fibrin.
5- Old pus contains an increased number of macrophages as well as cholesterol crystals and globules of fat.



Smear of pus. Most of the cells are neutrophil polymorphs: some are undergoing autolysis.

The process of abscess formation (suppuration) (the pathogenesis)

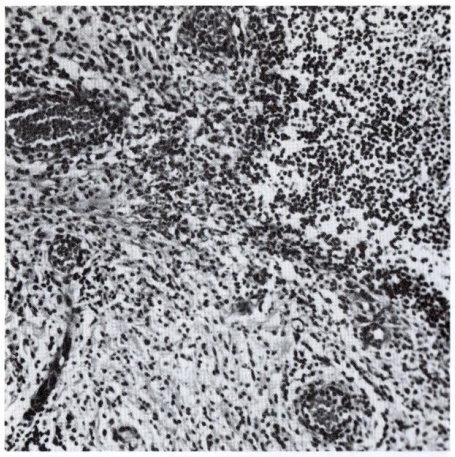
As the pyogenic bacterial infection progresses, local bacterial spread results in enlargement of the lesion, and unless the bacteria are destroyed rapidly, the tissue in the center undergoes necrosis due to:-
1- High concentrations of powerful toxins produced by pyogenic bacteria.
2- The pressure of inflammatory edema slows the blood flow.
3- Sometimes thrombosis due to endothelial injury.

Subsequently the central dead cells and tissue frame work will be digested by lysosomal enzymes released from phagocytic polymorphs infiltrated from the surrounding inflamed tissue; gradually a space or abscess cavity containing fluid is formed. Gradually an abscess may become enclosed in a layer of granulation tissue (the pyogenic membrane), and then its outer layer matures to a fibrous tissue.

The drainage of abscess

In an abscess the removal of the fluid exudate by lymphatic is important in host defense, however, the exudate is relatively stagnant, some fluid can exude into the space from the surrounding inflamed tissues but lymphatic drainage is inadequate. Consequently the hydrostatic pressure in the cavity rises and the necrotic area expands more and more leading to abscess enlargement, so the pus

in the abscess tends to extend along tissues of least mechanical resistance, e.g. an abscess forming near the skin, a mucous membrane or a serosal cavity, tends to extend towards the surface and rupture, discharging its pus, accordingly surgical incision and drainage of an abscess is an important therapeutic measure, it promotes bacterial elimination and destruction which allows the abscess cavity to heal with minimal scarring.
The pressure in an abscess is well illustrated by the spurting out of pus upon incision as well as by the throbbing pain of an apical tooth abscess experienced by the patient.



Wall of an abscess. The abscess cavity is seen at the top left. The wall consists of vascular granulation tissue showing an inflammatory reaction.

EXAMPLES OF PYOGENIC BACTERIAL INFECTIONS

Staphylococcal infections .

Some diseases caused by staph. Aureus:

Furuncle or boil: a focal suppurative inflammation of the skin & subcutaneous tissue either solitary or multiple involving moist hairy skin e.g. face, axilla, groin, legs & submammary folds.

Carbuncle*:* this reveals deeper suppuration than the above, that spreads laterally beneath the subcutaneous fascia & erupts as multiple skin sinuses. It involves the skin of upper back & posterior neck.

Impetigo*:* this refers to infection of superficial epidermis.

Food poisoning: endotoxin stimulates emetic receptors in the abdominal viscera causing vomiting & diarrhea.

Others: pharyngitis, pneumonia, endocarditis, infections of burns & infections of surgical wounds as well as hospital infections.

Streptococcal infections

Streptococci are Gram-positive cocci, which grow in pairs or chains. They are either *β- hemolytic e.g.* ***St. pyogenes*** *(group A)* that is involved in pharyngitis, scarlet fever, erysipelas, impetigo, rheumatic fever & glomerulonephritis.

St. agalactiae *(*group B) involved in neonatal sepsis & UTI.

Enterococcus faecalis (group D) involved in the causation of some cases of endocarditis & UTI.

St. pneumoniae are involved in the causation of pneumonia & meningitis.

St. mutans are involved in dental caries.

St. viridans (α hemolytic) are involved in infective endocarditis.

Gastrointestinal infections

Normal defense against ingested pathogens include

1. Acid gastric juice
2. Mucus layer covering the gut
3. Lytic pancreatic enzymes & bile detergents

4- Secreted IgA antibodies made by B cells of mucosa associated lymphoid tissue (MALT)

5- Commensal bacteria residing in the lower GIT

Weakened host defenses

1. Low gastric acidity.
2. Antibiotic abuse.
3. Decreased peristalsis.
4. Mechanical obstruction.

Salmonellosis

Salmonellae are flagellated Gram-negative bacteria causing

1. Food born & water born gastroenteritis.
2. Food poisoning.
3. Typhoid & paratyphoid fevers.

Typhoid fever

This is caused by *Salmonella typhi.* Human is the only host. Bacteria are shed with feces, urine and vomits as well as oral secretions from sick persons & in feces of chronic carriers. Pathogenesis: Invasion of intestinal epithelium & macrophages is followed by bacteraemia and systemic dissemination cause proliferation of phagocytes with enlargement of reticuloendothelial and lymphoid tissues throughout the body, including peyer's patches in terminal ileum.

clinical features include

- Fever with bactraemia & chills in the first week.

- Wide spread reticuloendothelial involvement (spleen, lymph nodes, etc.,) with skin rash abdominal pain & prostration in the second week.
- Ulcerations of Peyer's patches with intestinal bleeding & shock in the third week.

Carrier state is associated with chronic cholecystitis & gallstones with colonization by bacteria.

Helicobacter pylori

Infection by H. pylori is the most important etiologic cause of chronic gastritis. Patients are at increased risk for the development of:
1. Peptic ulcer disease 2. Gastric carcinoma 3. Gastric non- Hodgkin's lymphoma.

H. pylori are curvilinear, gram-negative rods. They have adapted to survive within the superficial mucus layer on the surface and within the gastric pits, which is lethal to most bacteria. They do not invade the mucosa.

clinical features include

include epigastric pain that worsens with eating, postprandial belching and epigastric fullness, early satiety, fatty food intolerance, nausea, and occasional vomiting