Septicaemia

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The presence of bacteria in the blood causes bacteraemia, septicaemia and pyaemia.

In *bacteraemia*, bacteria are present in the blood in relatively small numbers, but do not multiply significantly, while in septicaemia and pyaemia bacteria of high pathogenicity are present and also multiply in the blood. Bacteraemia subsides by itself when the resistance of the patient is more powerful than the causative organism. But sometimes the bacteria may settle in the various parts of the body and produce lesions e.g.: suppurative meningitis, suppurative arthritis and osteomyelitis.

Septicaemia is the presence and multiplication of bacteria, in the blood, of high pathogenicity causing symptoms e.g. pyogenic cocci, with profound toxaemia in which the bacteria have overwhelmed the host defence.

Sometimes it is very difficult to differentiate between bacteraemia and septicaemia, because in both cases the bacteria are present in the blood. In septicaemia small haemorrhages may occur due either to capillary endothelial damage from severe toxaemia or due to multiple minute metastatic foci of bacterial growth.

Pyaemia. In localised pyogenic infection toxic injury to the endothelium of the veins involved in the lesion may result in thrombosis. Bacteria multiply in the thrombus, then small fragments of septic thrombus break away and are carried by the blood. They become impacted in small vessels, resulting in necrosis, haemorrhage and suppuration with the formation of multiple pyaemic abscesses, either because of the bacteria or their toxin. Impaction occurs in various tissues depending on the site of original septic thrombosis. An example is portal pyaemia with abscesses only in the liver, because of septic venous thrombosis of the tributary of the portal vein in acute appendicitis. When a large vein is involved in the lesion the thrombus will cause septic infarction. Usually bacteria are released from the septic thrombus in pyaemia and frank septicaemia supervenes.

Septicaemia may be primary, usually caused by virulent organisms eg. Streptococcus pyogenes; or secondary, when it is initiated by other causes. The latter occurs especially when the alimentary tract is involved eg. coliform organisms, or during medical treatment when immune mechanisms are disturbed e.g. leukaemia and chemotherapy.

Predisposing factors of Septicaemia

1. Breach of the mechanical barriers i.e. skin and mucous membranes, either by a burn or by trauma.
2. Ischaemic necrosis of the bowel.
3. Serious underlying disease with immunosuppression, e.g. leukaemia.
4. Genetic disorders with defects of leucocyte motility.
5. Intra-abdominal infection after surgical procedures.
6. Diabetes.
7. Alcoholism and cirrhosis of the liver.
8. Nature of the bacteria.

Causative Agents of Septicaemia

1. Gram +ve and Gram -ve cocci:-
   - Staph. aureus
   - Strep. pyogenes
   - Strep. pneumoniae
   - Neisseria meningitidis
2. Gram -ve bacilli:-
   - E. coli
   - Enterobacter
   - Klebsiella
   - Pseudomonas
   - Proteus

Causes of Septicaemia

1. Penetrating wounds.
2. Burn wound sepsis - all cutaneous defences against bacterial invasion are virtually destroyed coupled with the destruction of the first line of defence to bacterial invasion or modification in the systemic response, such as hypovolaemia and and altered immune mechanisms.
3. Intravenous catheter sepsis - include superficial thrombophlebitis and septicaemia. The chances are greater in centrally placed catheters used for nutritional therapy.
4. Urinary catheters - when strict aseptic techniques for introduction of catheter and closed drainage system is not applied.
5. Post-operative respiratory infection is common in upper abdominal surgery, especially in the obese and smokers.
6. Abdominal sepsis - infection of the wound may cause septicaemia. Abdominal and pelvic abscesses are frequent complications of operation on the G.I.T. e.g. colorectal surgery, appendicectomy, biliary surgery, etc.
7. Endoscopies - cystoscopies, retrograde cholangiopancreatography and trans-hepatic cholangiography where septicaemia is a recognised hazard.
8. Decubitus ulcers.

Clinical Features

1. High fever, spiking and accompanied by rigors.
2. Profound circulatory collapse and oliguria.
3. Tachycardia.
4. Leucocytosis.
5. Petechial haemorrhages in the skin and conjunctiva.
6. Anaemia secondary to haemolysis.
7. Shock is common in Gram -ve infection.
8. Metastatic abscesses - involving the bone, brain and spleen.

Factors responsible for Sepsis in Surgical Wounds

1. Inadequate preparation of the patient.
2. Prolonged operative technique.
3. Inadequate haemostasis.
4. Use of non absorbable sutures in the wound.
5. Impaired blood supply to the part.
6. The use of open drains and soaking of the dressing.
7. Dead space in the wound.
8. Presence of chronic carriers of pathogenic organisms in the operating theatre.
9. A hollow viscus containing bacteria i.e. colon when opened, then there is a great risk of infection.

Laboratory Investigations

1. Complete blood picture.
2. Urine analysis.
3. Gram stain of any discharge from the wound and urine.
4. Chest X-Ray.
5. Fluoroscopy for movement of the diaphragm.
6. Blood culture.
7. Culture and sensitivity of sputum, urine, C.S.F. and stool in case of diarrhoea.
8. Pus for culture and sensitivity if there is sepsis in the wound.

Treatment

1. Rest and isolation.
2. Supportive treatment to build up the resistance of the patient.
3. Correction of fluid balance and electrolytes.
4. Correction of urinary output.
5. I.V. antibiotics (bactericidal) in high doses.
6. If abscess is present somewhere then drain.

Septicaemic shock is usually caused by Gram-ve organisms with the clinical findings of tachycardia, tachypnoea, hypotension leading to circulatory collapse, oliguria and fever with rigors. In post operative patients the sudden appearance of tachypnoea and hypotension is suggestive of Gram -ve septicaemia. This condition has high mortality rate.

Management of Septicaemic Shock

1. Correction of fluid and electrolyte balance.
2. Correction of renal function.
3. Some patients need assisted ventilation.
4. High doses of antibiotics (bactericidal) effective against the likely infecting organism should be started immediately. Type of antibiotic is guided initially by identification of source of infection and later by repeated blood culture and sensitivity.
5. Source of sepsis may be identified by careful clinical, radiological and biochemical examination and then antibiotic should be changed according to sensitivity results.
6. Localised intra-abdominal sepsis will need antibiotics and drainage.