Epidemiology:
- Endocarditis is infrequently reported following HSCT (prevalence is approximately 1.3%).

Clinical features:
- The clinical presentation of endocarditis following HSCT can be subtle (75% of cases are diagnosed at autopsy).
- Left-sided cardiac valves, especially mitral valve, are most commonly involved.

Risk factors:
- The main risk factors are:
  - (i) indwelling central venous catheters,
  - (ii) disruption of skin and mucosal barriers by high-dose chemotherapy and GVHD, and
  - (iii) the administration of immunosuppressive therapy.

Organisms:
- The usual organisms isolated are Gram-positive bacteria including S. aureus and S. viridans; however, there is a high prevalence of fungal endocarditis, including Aspergillus and Candida species.
- In one third of patients, no organisms are isolated, consistent with the diagnosis of nonbacterial thrombotic endocarditis.
- Most commonly associated with electrolyte abnormalities, hypoxemia, sepsis, MOSF, and use of vasopressor agents.

Pericardial tamponade:
- Pericardial effusion following HSCT is rare and is usually related to:
  - (i) cyclophosphamide toxicity,
  - (ii) viral syndrome,
  - (iii) chronic GVHD, or
  - (iv) renal failure.
- Rarely may it be due to bacterial infection (mainly S. aureus) or aspergillosis.

Risk Factors:
- Commonest cardiac complication requiring ICU admission is pulmonary oedema.

General:
- Risk factors are:
  - (i) pre-existing cardiac disease (even subclinical); EF <50%.
  - (ii) fluid overload associated with the infusion of chemotherapy.
  - (iii) acute renal failure.
  - (iv) venoocclusive disease.
  - (v) severe sepsis.
  - (vi) anemia.
  - (vii) High-dose chemotherapy used in the preparation for HSCT, such as cyclophosphamide, cytosine arabinoside, paclitaxel, etoposide, and cisplatin, may be associated with significant cardiac toxicity and congestive heart failure.