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Surgical Infection

Wound infection can be defined as the invasion of organisms through tissues following a breakdown of local and systemic host defences, leading to cellulitis, lymphangitis, abscess and bacteraemia.

The infection of most surgical wounds is referred to as **superficial surgical site infection (SSSI)**. The other categories include **deep SSI** (infection in the deeper musculofascial layers) and **organ space infection** (such as an abdominal abscess after an anastomotic leak).

Infection that follows surgery or admission to hospital is termed **health care-associated infection (HAI**). There are **four** main groups: *respiratory* infections (including ventilator-associated pneumonia), *urinary tract* infections (mostly related to urinary catheters), *bacteraemia* (mostly related to indwelling vascular catheters) and *SSIs*.

Background

Surgical infection, particularly surgical site infection (SSI), has always been a major complication of surgery and trauma and has been documented for *4000–5000* years. The understanding of the causes of infection came in the 19^{th} century. Microbes had been seen under the microscope, but *Koch* laid down the first definition of infective disease (Koch's postulates).

The principles of **antiseptic surgery** were soon enhanced with aseptic surgery at the turn of the century. The discovery of the antibiotic penicillin is attributed to *Alexander Fleming in 1928*, but it was not isolated for clinical use until *1941 by Florey* and *Chain.* Then, the policy of "**prophylactic antibiotics**" were widely applied & greatly **reduced** the rate of wound infection.

Advances in the control of infection in surgery

1_ Aseptic operating theatre techniques have enhanced the use of antiseptics

2_ Antibiotics have reduced postoperative infection rates after elective and emergency surgery

3_ Delayed primary, or secondary, closure remains useful in contaminated wounds

Risk factors for increased risk of wound infection

1_ Malnutrition (obesity, weight loss)

2_ Metabolic disease (diabetes, uraemia, jaundice)

3_ Immunosuppression (cancer, AIDS, steroids, chemotherapy and radiotherapy)

4_ Colonisation and translocation in the gastrointestinal tract

5_ Poor perfusion (systemic shock or local ischaemia)

6_ Foreign body material

7_ Poor surgical technique (dead space, haematoma)

There is a *delay* before host defences can become mobilized after a breach in an epithelial surface. The acute inflammatory, humoral and cellular defences take up to **4 hours** to be mobilised. This is called the '**decisive period**', and it is the time when the invading bacteria may become established in the tissues, so it's the appropriate **time to introduce the prophylactic antibiotics**.

Major And Minor Surgical Site Infections

A major SSI is defined as a wound that either discharges significant quantities of *pus* spontaneously *or* needs a secondary procedure to drain it. The patient may have *systemic* signs, such as tachycardia, pyrexia and a raised white count.

Major wound infections

_ Significant quantity of pus

_ Delayed return home

_ Patients are systemically ill

Minor wound infections may discharge pus or infected serous fluid but should *not* be associated with excessive discomfort, systemic signs or delay in return home.

The **ASEPSIS** wound score for severity of wound infection:

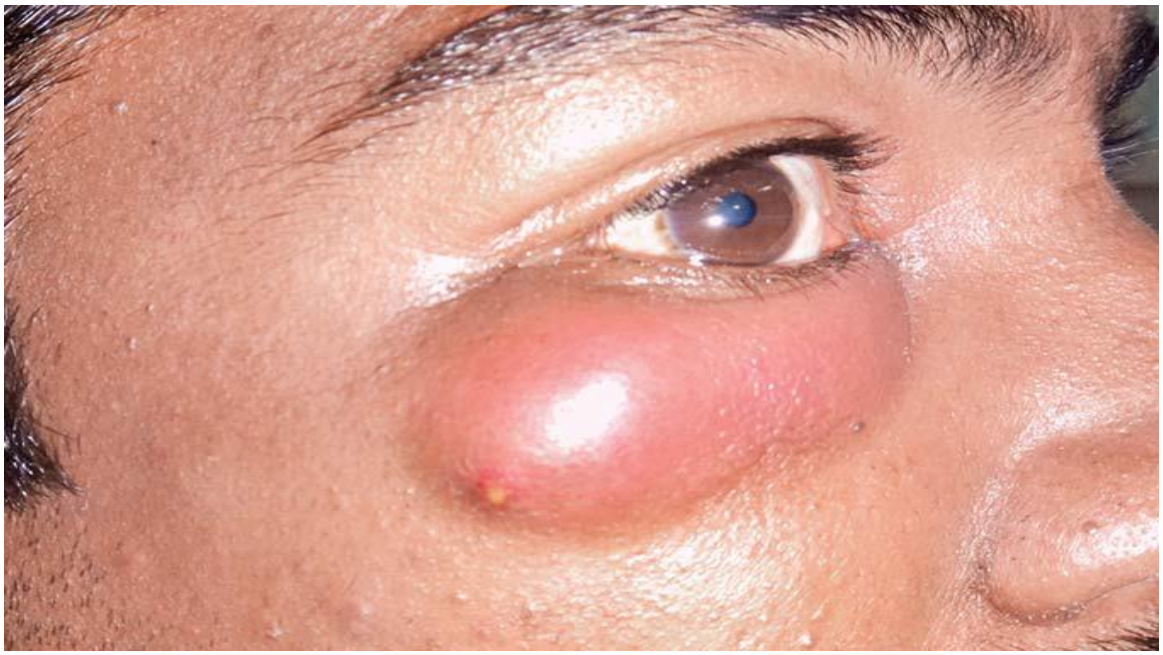
Additional treatment: Antibiotics for wound infection Drainage of pus under local anaesthesia or debridement of wound under general anaesthesia.

Serous discharge **E**rythemaa **P**urulent exudate **S**eparation of deep tissues **I**solation of bacteria from wound **S**tay as inpatient prolonged over 14 days as result of wound infection.

Types Of Localised Infection

Abscess

It is a ***localised collection*** of pus in a cavity lined by granulation tissue, covered by pyogenic membrane. Pus *contains* dead WBC's, multiplying bacteria, toxins and necrotic material.



If it is *not* drained or resorbed completely, a *chronic* abscess may result. If it is partly sterilised with antibiotics, an *antibioma* may form. Antibiotics are only **indicated** if the abscess is still not localised (e.g. evidence of *cellulitis*) or the cavity is not left open (*aspiration*) to drain freely. Then healing by *secondary* intention is encouraged

Abscesses contain *hyperosmolar* material that draws in fluid. This increases the pressure and causes pain. If they spread, they usually track along planes of least resistance and point towards the skin, so they *discharge spontaneously*, or may need *drainage* through a surgical incision. Most abscesses relating to surgical wounds take *7–10 days* to form after surgery.

Modern imaging *techniques* e.g. ultrasonography, computed tomography (CT), magnetic resonance imaging (MRI) and isotope scans are all useful and may allow *guided aspiration* without the need for surgical intervention.

Pyogenic organisms, predominantly *Staphylococcus aureus*, & Streptococci are usually the causative agents of **acute** abscesses.

Persistent **chronic** abscesses may lead to sinus or fistula formation. Lymphocytes and plasma cells are seen. Common microorganisms are *Mycobacterium* and *Actinomyces*.

Cellulitis

It is ***spreading*** inflammation of subcutaneous and fascial planes. There is *poor localisation* in addition to the cardinal signs of inflammation & systemic signs. Spreading infection presenting in surgical practice is typically caused by organisms such as B-haemolytic streptococci, staphylococci and *C. perfringens*.

Tissue destruction, gangrene and ulceration may follow, which are caused by release of proteases.

Lymphangitis

It is an acute *nonsuppurative* infection and spreading inflammation of *lymphatics* of skin and subcutaneous tissues due to *beta haemolytic streptococci*, staphylococci, clostridial organisms. It is commonly associated with cellulitis & its part of a similar process and presents as *painful red streaks* in affected lymphatics. Lymphangitis is often accompanied by painful *lymph node* groups in the related drainage area.

Systemic Inflammatory Response Syndrome And Multiple Organ Dysfunction Syndrome

Systemic inflammatory response syndrome (SIRS) is a *systemic manifestation* of sepsis, although the syndrome may also be caused by multiple trauma, burns or pancreatitis without infection.

SIRS should *not* be confused with bacteraemia although the two may coexist.

Septic manifestations and multiple organ dysfunction syndrome (MODS) in SIRS are mediated by the release of proinflammatory **cytokines** such as interleukin-1 (IL-1) and tumour necrosis factor alpha (TNF_a). These cytokines stimulate neutrophil adhesion to endothelial surfaces adjacent to the source of infection and cause them to migrate through the blood vessel wall by chemotaxis. Coagulation, complement and fibrinolytic pathways are also stimulated as part of the normal inflammatory response. This response is usually beneficial to the host and is an important aspect of normal tissue repair and wound healing.

In the presence of severe sepsis or bacteraemia, this response may become harmful to the host if it occurs in excess, when it is known as *SIRS*. The activated neutrophils adhere to vascular *endothelium in key organs and damage* it, leading to increased vascular permeability, which in turn leads to cellular damage within the organs, which become dysfunctional and give rise to the clinical picture of **MODS**. In its most severe form, MODS may progress into multiple system organ failure (**MSOF**).

Respiratory, cardiac, intestinal, renal and liver failure ensue in combination with circulatory failure and shock. In this state, the body's resistance to infection is reduced and a vicious cycle develops where the more organs that fail, the more likely it becomes that **death** will follow despite all that a modern intensive care unit can do for organ support.

Definitions of infected states

_ SSI is an infected wound or deep organ space

_ SIRS is the body's systemic response to severe infection

_ MODS is the effect that SIRS produces systemically

_ MSOF is the end stage of uncontrolled MODS

SIRS

Two of:

*hyperthermia (>38°C) or hypothermia (<36°C)

*tachycardia (>90/min, no B-blockers) or tachypnoea (>20/min)

*white cell count >12 × 109/l or <4 × 109/l

Sepsis is SIRS with a documented infection

Severe sepsis or sepsis syndrome is sepsis with evidence of *one or more organ failures* (respiratory (acute respiratory distress syndrome), cardiovascular (septic shock follows compromise of cardiac function and fall in peripheral vascular resistance), renal (usually acute tubular necrosis), hepatic, blood coagulation systems or central nervous system)

Bacteraemia and sepsis

Bacteraemia is common after *anastomotic* breakdown (deep space SSI). It can also follow procedures undertaken through *infected* tissues (particularly instrumentation in infected bile or urine),or through bacterial *colonisation* of indwelling intravenous cannulae.

Sepsis accompanied by MODS may follow anastomotic breakdown. Aerobic Gram-negative bacilli are mainly responsible, but *S. aureus* and fungi may be involved, particularly after the use of broad-spectrum antibiotics.

Specific wound infections

Gas Gangrene

This is caused by *C. perfringens*. These Gram-positive, *anaerobic*, spore-bearing bacilli are widely found in nature, particularly in *soil and faeces*. This is relevant to *military and traumatic* surgery and colorectal operations. Patients who are immunocompromised, diabetic or have malignant disease are at greater risk, particularly if they have wounds containing necrotic or foreign material, resulting in anaerobic conditions.

Gas gangrene wound infections are associated with severe local wound *pain and crepitus* (gas in the tissues). The wound produces a thin, brown, *sweet-smelling* exudate. Oedema and spreading gangrene follow the release of collagenase, hyaluronidase, other proteases and alpha toxin. Early systemic complications with circulatory collapse and MSOF follow if prompt action is not taken.

Antibiotic prophylaxis should always be considered in patients at risk.

Synergistic spreading gangrene (synonym: subdermal gangrene, necrotising fasciitis)

A *mixed* pattern of organisms is responsible: coliforms, staphylococci, *Bacteroides* spp., anaerobic streptococci and peptostreptococci have all been implicated, acting in synergy. Abdominal wall infections are known as **Meleney's** synergistic hospital gangrene and scrotal infection as **Fournier's** gangrene. Patients are almost always *immunocompromised* with conditions such as diabetes mellitus. Severe wound pain, signs of spreading inflammation with crepitus and smell are all signs of the infection spreading.

Untreated, it will lead to widespread *gangrene and MSOF*. The subdermal spread of gangrene is always much more extensive than appears from initial examination. Broad-spectrum *antibiotic* therapy must be combined with aggressive circulatory support. Locally, there should be *wide excision* of necrotic tissue and laying open of affected areas.

Choice of antibiotics for prophylaxis

_ Empirical cover against expected pathogens with local hospital guidelines

_ Single-shot intravenous administration at induction of anaesthesia

_ Repeat only during long operations or if there is excessive blood loss

_ Continue as therapy if there is unexpected contamination or if a prosthetic is implanted in a patient with a septic source

_ Benzylpenicillin should be used if Clostridium gas gangrene infection is a possibility

_ Patients with heart valve disease or a prosthesis should be protected from bacteraemia caused by dental work, urethral instrumentation or visceral surgery

Avoiding surgical site infections

_ Staff should always wash their hands between patients

_ Length of patient stay should be kept to a minimum

_ Preoperative shaving should be done immediately before surgery

_ Antiseptic skin preparation should be standardised

_ Attention to theatre technique and discipline

_ Avoid hypothermia perioperatively and ensure supplemental oxygenation in recovery

Classification Of Surgical Wounds according to Potential For Infection

Type of surgery	***Infection rate (%)***	***Rate before prophylaxis***
Clean (no viscus opened)	1–2	The same
Clean-contaminated (viscus opened, minimal spillage)	<10	Gastric surgery up to 30% Biliary surgery up to 20%
Contaminated (open viscus with spillage or inflammatory disease)	15–20	Variable but up to 60%
Dirty (pus or perforation, or incision through an abscess)	<40	Up to 60% or more

Good Luck