REVIEW
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Prevention and management of wound complications

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A wound can be defined as the disturbance of the skin's normal structure and function and the soft tissue beneath it.[1] Wound complications are major contributors to both early and late postoperative morbidity.[2] It can cause significant physical and emotional distress to both patients and the treating physician. Most surgeons will agree that wound complications can be challenging to treat and everything possible should be done to prevent it from happening.

Wounds are classified as clean-, clean-contaminated-, contaminated- or dirty wounds. Clean wounds can be described as an incision into tissue, other than hollow viscera, in which neither infection nor inflammation is present followed by primary closure afterwards. Clean-contaminated wounds result from intentional entry into the respiratory, gastro-intestinal, genital or urinary tracts without remarkable contamination. Contaminated wounds can be characterized by unintentional entry into a hollow viscus, a major disruption in sterility, or encountering areas of nonpurulent inflammation. Dirty wounds include old wounds with residual devitalised tissue, perforated viscera or current clinical infection.[3]

The physiology of wound healing

Tissue injury triggers a cellular response which lead to keratinocytes, fibroblasts, endothelial cells, macrophages, and platelets to be activated in order for wound healing to ensue.[4] Wound healing occurs in an orderly, integrated and overlapping fashion, and consists of distinct phases: haemostasis, inflammation, epithelialization, fibroplasia, and maturation.[4-6]

Haemostasis

Haemostasis starts with constricting blood vessels and fibrin clot formation as soon as tissue injury occurs. Pro-inflammatory cytokines and growth factors (eg, transforming growth factor-B, platelet derived growth factor, fibroblast growth factor, and epidermal growth factor) are released by adjacent tissue and the fibrin clots. These factors are crucial for commencement and progression of wound healing. The fibrin matrix that forms helps to stabilize the wound and provide an initial framework for the healing process to follow. Bleeding from bigger vessels may require additional measures to attain haemostasis.[4,5]

Inflammation

This phase is characterized by increased vascular permeability and cellular recruitment. In the absence of infection and other factors that can lead to a chronic wound, this phase is completed within 72 hours. Mononuclear leucocyte infiltration and differentiation to macrophages occur secondary to several events, including vimectin secretion. Histamine and other substances facilitating vasodilation and cellular migration are released by mast cells leading to increased permeability of small blood vessels. The build up of plasma and cellular components result in clinically evident oedema or swelling. Polymorphonuclear leukocytes migrate and concentrate secondary to chemotaxis. These leucocytes are responsible for removing bacteria, foreign material and dead tissue. Failure to remove this could lead to abnormal production of metalloproteases, ultimately causing chronic wounds.[4,5,7,8]

Epithelialisation

This phase, also called migration, normally follows and overlaps with the inflammatory phase, and is characterized by basal cell proliferation and epithelial migration over the temporary matrix within the wound.[5,9] Proliferation usually lasts until individual cells are surrounded by cells of the same or similar type. Epithelial cells descend deep in the dermis and migration stops when the layer is revitalized. The superficial re-epithelialisation layer is usually completed within 48 hours of surgery and forms a barrier to bacteria and foreign material.[4]

Fibroplasia

This process entails proliferation of fibroblasts, ground substance build-up, and production of collagen. Fibroblasts originate from local mesenchymal cells and produce collagen, glycosaminoglycans and proteoglycans, which form the major components of the ground substance or extracellular matrix. Fibroblasts also produce myofibroblasts, contractile proteins with similar characteristics as smooth muscle cells, and assist in pulling the wound edges together. Myofibroblasts are later removed by apoptosis as scar formation evolves.[4,5]

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Maturation
The main characteristics of this phase include collagen cross-linking, collagen remodelling, wound contraction and repigmentation. The amount of collagen present in the wound correlates directly with the tensile strength and is further enhanced by covalent cross-linking of the collagen. Around 80% of original tissue strength is present after six weeks following surgery and collagen fibres have the appearance of normal skin after roughly 180 days.[4,10]

Factors affecting wound healing
Numerous factors impair wound healing and can be divided into local and systemic. Local factors have a direct effect on the wound, whereas systemic factors refer to the patient's general health that influences their capability to heal.[5]

Local factors
Oxygenation
Sufficient oxygen levels are essential for optimal healing to take place. Oxygen protects wounds against infection, stimulates angiogenesis and promotes all the subsequent phases of normal healing. Although initial hypoxia following injury stimulates wound healing, sufficient oxygenation is imperative to sustain it.[5,11,12]

Infection
Invasive infection is present when microorganisms multiply in a wound, leading to underlying tissue injury.[13,14] Secondary to inflammatory substances, infections can hinder both the inflammatory and epithelialisation phases of wound healing.[15,16] Foreign material can also influence wound healing locally.

Systemic factors
Aging
Age-related physiological changes of the skin include decreased blood and nerve supply, thinning of the different layers and failure to sustain and produce more collagen.[17,18]

Stress
Stress can influence human health significantly. By inducing negative states like anxiety and depression, stressors may limit health outcomes by influencing physiological and behavioural patterns.[5] Impaired wound healing was demonstrated among students under examination stress, as well as people caring for Alzheimer patients.[19]

Diabetes
Pathophysiological impairment of vascular, neurological and immunological function, predispose diabetic patients to poor wound healing. Secondary to poor perfusion and diminished angiogenesis, diabetic patients' wounds may be subjected to prolonged periods of hypoxia.[5] Wound healing may also be affected by the effect of the disease on growth factor production, macrophage and fibroblast function, neovascularization, metalloprotease levels, local immune response and epidermal nerves.[5,20]

Obesity
Obesity is currently showing an upward trend among adults and increases the chance of several diseases and health risks, including poor wound healing.[5] Local sepsis, wound dehiscence and hematoma and seroma formation are some of the complications obese patients are more likely to suffer from.[5,21] These complications may be due to underperfusion and ischemia of the subcutaneous fatty tissue and impaired delivery of antibiotics to the wound site.

Smoking
Smoking is an important risk factor for impaired wound healing.[5] Apart from delayed wound healing smokers have an increase in post-operative complications, which include infection, wound disruption and anastomotic leakage. The tensile strength of wounds is also diminished.[21,22] There are clear benefits in quitting smoking, which include enhanced wound healing and a decrease in wound infection.[24,25]

Medication
There are many different medications that might influence wound healing through their effect on clot formation, platelet aggregation, inflammatory processes and cell growth. Some of these medications most commonly used, include glucocorticoid steroids and non-steroidal anti-inflammatory drugs.[5]

- Glucocorticoid steroids
Systemic administration of glucocorticoid steroids, impair wound healing by inhibiting fibroblast growth, collagen production and cellular responses and may increase the chance of wound infection.[5]

- Non-steroidal anti-inflammatory drugs (NSAIDs)
Although there is limited information that NSAIDs used for short periods halters wound healing, the consequence of chronic NSAID usage is undetermined. Among animal studies long-term usage is associated with fibroblast reduction, lower tensile strength, decreased wound tightening and slower epithelialization.[5]

Cancer treatment
The majority of chemotherapeutic agents inhibit cellular pathways, which are essential to wound healing.[5] These drugs can impair wound healing by affecting fibroblast proliferation, neovascularization, cellular migration, wound matrix formation, collagen synthesis and wound contraction.[26,27] Angiogenesis inhibitors target vascular endothelial growth factor (VEGF), which is significant in regulating cancer growth, leading to a reduction in blood supply to the tumour. Because this is also important in wound healing, these agents may increase wound complications, including wound dehiscence.[28,29] Radiation treatment also may contribute significantly to impair wound healing, especially by reducing the vascularity of the tissue in previously irradiated areas.[4]

Other
Despite the mentioned risk factors for impaired wound healing, there are many other causes that may directly or indirectly impact on healing and contribute to wound complications.

These factors include malnutrition, sickle cell disease, spinal cord disease and immobility, peripheral arterial disease and chronic alcohol abuse, to name a few. The effect of HIV on wound complications will be discussed later.

Common wound complications

Haematoma and seroma

Wounds complicated by a collection of blood are known as haematomas, whereas seromas are a collection of serum. Haematomas occur more frequently than seromas and are usually from when haemostasis is not achieved at the time of surgery or secondary to clotting defects (e.g. anticoagulation). Both haematomas and seromas increase the risk of infection by allowing bacteria access to the subcutaneous tissue following disruption of the wound surface.[2]

Patients usually present a few days post-operatively with painful or painless swelling around the wound site, and/or drainage of fluid. Seromas may present with drainage of straw-coloured fluid. If secondary infection is present there might be local and/or systemic signs of sepsis, like fever, erythema, cellulitis, pus draining from wound and leukocytosis. Diagnosis is usually clinical but ultrasound and CT-scan might be of use.[2]

Treatment usually depends on the size of the haematoma and seroma and ranges from expectant management to surgical drainage. In the absence of infection the wound may be closed primarily or left to heal by secondary intention. Seromas may be managed with sterile aspiration.[1,2]

Preventing these complications usually require proper surgical technique, meticulous haemostasis and obliterating large potential dead spaces.[2] Prophylactic drain placement may be indicated to prevent fluid collection and infection, however randomised trials and meta-analysis found that wound complications were not reduced by closed drainage of subcutaneous tissue.[30-32]

Fascial dehiscence

Fascial separation is usually a product of suture or tissue failure secondary to abdominal pressure. It can happen early or late following surgery and include partial or complete fascial dehiscence. Early fascial dehiscence, with or without evisceration, is regarded as an emergency requiring surgical intervention, while late dehiscence manifest as incisional hernias.[2]

A retrospectivestudy performed in Greece identified a number of risk factors associated with dehiscence.[33] Factors associated with dehiscence of the abdominal wound included advanced age (>65 years), emergency procedures, malignancy, haemodynamic unstable patients, local and intra-abdominal sepsis, low albumin, ascites, obesity and steroid usage. Gender, anaemia, diabetes mellitus and lung disease were not shown to be significantly associated with fascial dehiscence.[34]

Fascial dehiscence is usually diagnosed clinically and occurs on average around eight days post-operatively (4-14 days). Patients might report a popping feeling followed by excessive drainage of serosanguinous fluid. Surgical exploration is indicated if clinically suspected. Seeing as complete fascial dehiscence carries a mortality rate of up to ten percent, urgent intervention is mandatory. Treatment entails careful wound exploration, fascial edge debridement, mass closure with delayed absorbable sutures, with or without skin closure.[2]

A large review which included four meta-analysis concluded that the optimal technique to close a midline incision necessitate closure of all layers except the skin as a single structure, in a simple continuous fashion, with a delayed absorbable monofilament suture material (#1 or #2) and the suture length four times that of the wound length.[35]

Surgical site infection (SSI)

A wide spectrum of common endogenous bacteria produce wound infections, including most gram positive cocci and aerobic and anaerobic rods. Small numbers of bacteria are present in all surgical wounds, however, bacterial growth is facilitated by decreased tissue oxygen and excessive amounts of necrotic tissue. It requires between 100 000 to 1 000 000 bacteria per gram of tissue to produce an infection in surgical wounds. The incidence of superficial skin infections is directly related to the length of surgery. Every extra hour of operative time result in a doubling of the incidence of superficial skin infections.[2]

Infection complicates around four percent of clean wounds. The first symptoms of wound infections typically appear between five and ten days post-operative. Fever within the first 48 hours usually results from atelectasis. Rare types of wound infections caused by Clostridium species and acute β-haemolytic streptococci species are so virulent that they can produce toxicity within the first two days. Wound infections secondary to Clostridium species are boggy, oedematous and has a discharge smelling like sweat. β-haemolytic streptococci infections appear swollen, red and the discharge is odourless.[2]

In general wound infections present as an erythematous, indurated, warm and painful incision area. Purulent fluid may drain from the wound with local wound breakdown. Incisional abscesses may also be present. More severe infections may manifest with systemic signs, ranging from fever to septic shock. Although rare, necrotising fasciitis may occur in severe cases and is a life-threatening condition requiring emergency surgical intervention and support. These patients may present with excessive pain around the surgical site. Subcutaneous tissue is usually very friable with pale, devitalised fascia. The infection expands quickly in the subcutaneous spaces and often tracks far beyond the superficial margins of the involved skin. The disease carries a mortality of up to 50% and requires immediate surgical intervention including wide debridement of all the necrotic tissue followed by appropriate antibiotic administration.[2,36]

Treatment generally depends on the extent of the infection. Wounds complicated with cellulitis, without areas of fluctuation can generally be treated with oral or parenteral antibiotics. Antibiotic cover should usually be broad spectrum and include cover against grampositive cocci from the skin and bacteria expected at the operation site. Antimicrobial treatment should be tailored according to clinical response and microscopy, culture and sensitivity results.[2]

More severe infections and wound abscesses require surgical debridement with or without incision and drainage.

Debridement is done using a scalpel and scissors and requires removing all devitalised tissue that may impair healing and increase infection risks. Due to the toxic effect of chemical agents like povidine-iodine and hydrogen peroxide on fibroblasts, these agents are not recommended. Saline is preferred for irrigation because it is isotonic and does not halter wound healing.[2]

Following debridement, wounds should be covered by dressings that keep moisture and warmth, therefore holding tissue growth factors that assist with re-epithelialisation and allowing autolytic debridement.[2] In order for wounds to heal by secondary intention, the best dressing to facilitate this process will absorb exudates, resist water and micro-organisms and does not leave contaminants in the wound site after removal or damage granulation tissue.[37]

According to the Centre for Disease Control guidelines[38], the following preventative steps to limit surgical site infections are based on high quality evidence from well-designed trials: (1) Elective surgery should be avoided in patients with current infections. (2) Optimal concentration of perioperative antibiotics should be accomplished and prophylactic antibiotics continued for a couple of hours following surgery. (3) If hair removal is indicated, it is advised that it should be clipped and not shaved before commencing surgery in the operating room.

Based on good quality evidence and expert consensus, the following guidelines may also prevent SSI: (1) Optimal glucose control in diabetic patients peri-operatively. (2) Smoking or tobacco use cessation or abstinence for a month before surgery. (3) Bathing or showering with an antiseptic soap ahead of the operation. (4) Standards should be followed regarding sterilising instruments, maintaining an aseptic operating theatre, as well as appropriate air circulation. (5) Preventative measures from the surgical team include, disallowing members with active infections, always wearing sterile clothing and gloves, meticulous hand hygiene including short fingernails, scrubbing with antiseptic agents for two to five minutes up to the elbows and using sterile towels to dry afterwards. (6) Correct surgical technique is crucial, and leaving sterile dressings on the surgical site for 24 to 48 hours may also prevent this complication.

Special considerations

SSIs following lower segment caesarean section

Caesarean sections are one of the most frequently performed procedures globally and steadily rising. It remains one of the main reasons for SSI. SSIs following caesarean section, which includes all infections occurring within 30 days following surgery, may be superficial, deep, or in severe cases present as pelvic and/or abdominal abscesses. Endomyometritis can also be included as part of post-caesarean SSI.[39]

As mentioned before, there are many factors that increase the risk of wound infection as with non-pregnant patients. Other factors specifically to pregnancy include limited antenatal care, pregestational and gestational diabetes, hypertensive conditions, higher order pregnancies, emergency caesarean sections, labour duration, prelabour rupture of membranes and duration thereof, number of vaginal examinations and failure to administer prophylactic antibiotics, to name a few.[39]

Preventing SSI following caesarean delivery should be directed towards modifying specific risk factors. One such an important intervention is administrating prophylactic antibiotics before performing a caesarean section. Previously, antibiotic administration was delayed until after clamping of the umbilical cord because of concerns that it may conceal neonatal infections and cause bacterial resistance. However, this appears not to be the case and there is high quality evidence that by giving antibiotics earlier benefits the mother and does not harm the neonate.[40,41]

The American Congress of Obstetricians and Gynecologists (ACOG) recommends the first-line antibiotic that should be used as a single dose within an hour of initiating surgery to be either a first generation cephalosporin or ampicillin. Patients allergic to penicillin should receive clindamycin and a aminoglycoside.[42] A recent study from India found the organisms most frequently associated with SSIs post-caesarean delivery were *Acinetobacter* species, *Staph. Aureus* and coagulase negative *Staphylococcus*.[33]

Operating room procedures that might contribute to reducing infections include clipping instead of shaving hair immediately before cutting, cleaning with chlorhexidine preperations, prophylactic antibiotics before skin incision, removal of the placenta with traction and not manually, closing subcutaneous tissue with a continuous suture if greater than 2 cm and cleaning the vagina with povidone-iodine just before the procedure.[39]

Vaginal cuff dehiscence/Evisceration

The overall incidence of dehiscence of the vaginal vault after hysterectomy is reported to range between 0.24% and 0.39% and varies depending on the route of hysterectomy. Total laparoscopic hysterectomy carries the highest risk for cuff dehiscence and vaginal hysterectomy the lowest risk. The average time it takes for this complication to occur varies between 45 to 100 days after surgery, but may occur as late as 20 months.[43,44]

Factors that increase the chance of dehiscence include sexual intercourse, vault haematoma or abscess, smoking, postmenopausal state and constant elevated intra-abdominal pressure. The incidence of vaginal cuff dehiscence may be reduced by administrating prophylactic antibiotics and by treating post-operative vaginal infections in symptomatic patients. Closing the vaginal vault with full thickness sutures, at least 10mm from the resection margin, and avoiding diathermy usage may reduce vault dehiscence. However, the type of suture material and technique used does not appear to make a difference.[45,46]

Vault dehiscence is diagnosed clinically and patients can present with vaginal bleeding, discharge and/or pain. More severe presentations include peritonitis, evisceration, bowel strangulation and septicaemia. Treatment usually involves broad-spectrum antibiotics with surgical repair. There is not sufficient evidence on the appropriate route to use for surgical repair. Vaginal repair is probably best for small, uncomplicated dehiscence whereas abdominal exploration and repair is mandatory for patients with evisceration.[45]

The obese patient

Obesity is one of the most significant risk factors for wound infections and disruption. In this group of patients, hyperglycaemia can further contribute to complications. Adipose tissue is poorly vascularised and causes suboptimal oxygenation leading to poor healing, collagen synthesis and reepithelialisation.[5]

Techniques to improve wound healing in obese patients can be summarised as follows:

- Chlorhexidine bath or shower the night prior to surgery
- Women with BMI >35 should receive double the dosage of prophylactic antibiotics
  Extending the dosing beyond surgery is not helpful
- Normothermia should be maintained during surgery
- Subcutaneous tissue more than two centimetres should be closed
- Cover the wound with a sterile dressing between 24 to 48 hours
- Maintain euglycaemia
- Excess oxygen administration during surgery and subcutaneous drains do not appear to improve outcome

Wound complications in HIV-infected patients

There appears to be conflicting evidence regarding post-operative morbidity and mortality among HIV-infected women.[47] Factors that may increase impaired wound healing, wound infections and post-operative sepsis include immunodepletion manifested by a low CD_4 cell count, malnutrition evident from a low albumin and/or a neutrophil count less than 500 cells.[48] Allaran et al. found more post-operative complications and less favourable outcomes six months afterwards among patients with a CD_4 cell count below 200 cells/microliter.[49] A pre-operative viral load more than 30 000 copies/milliliter and emergency surgery may also increase surgical complications.[47]

Conclusion

Wound complications can be complicated and challenging to manage and may cause significant distress for both the patients and the physician who cares for them. Everything possible should me done to prevent post-operative wound complications, but if they occur, early recognition and correct treatment is imperative.

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