Abdominal wall defects: pathogenesis, prevention and repair

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Abstract

Abdominal wall defects often complicate the postoperative course of patients after abdominal surgery in the form of wound dehiscence in the short term and incisional hernia in the long term. Prevention and treatment remain a significant challenge to surgeons. This paper provides an update regarding the most recent insights regarding the pathogenesis of these common abdominal wall defects. In the 'How to' sections, the best methods of opening and closing the abdomen are presented, with a focus on prevention of short- and long-term complications. Also, the various options and indications for synthetic and autologous abdominal wall reconstructions are discussed. Finally, a treatment algorithm based on our extensive experience and the best available evidence in the literature is presented for the various forms of incisional hernias discussed in this paper.

Keywords Biologic graft; complex hernia; component separation technique; incisional hernia; synthetic mesh; wound dehiscence

Abdominal wall defects: wound dehiscence and incisional hernia

Abdominal wound dehiscence is defined as separation of the abdominal fascia soon after abdominal surgery, complete or partial, with or without dehiscence of overlying skin. If the abdominal organs protrude the term 'burst abdomen' is used. Wound dehiscence occurs in 0.4–1.2% of patients after elective laparotomy, while rates up to 12% are observed after emergency procedures. The consistency of its prevalence over the past century, despite improved surgical technique, is attributed to increasing co-morbidity within the surgical population. Wound dehiscence is commonest around postoperative day 9 and is associated with a high mortality, lying between 15 and 35%. The diagnosis of wound dehiscence not always easy to make, as it generally coincides with superficial tissue disruption and infection, but blood-tinged exudate may seep through the intact superficial tissue layer. Exaggerated abdominal pain and tenderness, and unexplained vomiting are also indicative of wound dehiscence. Radiographic imaging may be required to confirm the suspicion.

An incisional hernia occurs after wound healing which is insufficient at the level of the fascia. Incisional hernia occurs in 10–20% of patients after midline laparotomy. Repair of incisional hernia results in recurrences in 23.5% after first-time hernias and 34.8% after repair of recurring hernias after 5 years follow-up,[1] and continue to occur at a steady rate well up to 10 years after operation.

The majority of incisional hernias are asymptomatic. However, incarceration and strangulation are potential complications presenting with extreme pain, necessitating emergency surgery to prevent ischemia, necrosis, and ultimately perforation of bowel. A hernia may also grow larger and become physically debilitating.

Diagnosis of incisional hernia can usually be made at physical examination while the patient is standing, in which position the hernia often becomes evident directly or during Valsalva manoeuvre. However, clinical diagnosis may be a challenge in small asymptomatic hernias or in obese patients. Computed tomography imaging is considered the gold standard and is very accurate in geometrically defining the defect, and providing information on the musculofascial quality and on viscera outside the abdominal cavity (loss of domain).

Wound healing and mechanisms and risk factors of wound dehiscence and incisional hernia

Normal wound healing

The postoperative surgical wound proceeds through three stages of healing: the inflammatory stage, the regenerative stage, and the remodelling stage.

The *inflammatory stage* commences directly after the disruption of tissue (e.g. after incision) and generally lasts for about 4 days. Vasodilatation and angiogenesis take place, and proteinglycanes from mast cells cause formation of a gel matrix prepared for deposition of collagen later on during the regenerative stage. Macrophages are attracted and recruit the fibroblasts for the later stages, and further clear the environment of bacteria and debris. The presence of polymorphonuclear leukocytes causes phagocytosis of debris by proteinases, essentially weakening the tensile strength of the tissue in this stage. The area in which these proteolytic enzymes are active normally varies up to 5 mm on either side of the wound, but this can increase up to 1 cm in the presence of infection.

The *regenerative stage* (or proliferative stage) is characterized by movement of fibroblasts into the wound area, synthesis of collagen and contraction of the wound, and lasts for about 3 weeks. At the end of this stage, almost all new collagen has been formed in the wound. Tensile strength, although being increased relatively quickly due to the newly formed collagen matrix, is still not sufficient due to the lack of cross-linking between collagen molecules.

During the final and *remodelling (or maturation) stage* which may continue for years, the newly laid collagen matrix undergoes qualitative changes brought on by mechanical environmental forces, resulting in optimal alignment of fibres to withstand these

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pressures. Cross-linking, in which inter-molecular covalent bonds are formed within the collagen matrix, is responsible for the continued increase in tensile strength in this stage.

Risk factors and mechanisms of wound dehiscence and incisional hernia

It is a widely held concept that dehiscence is primarily a result of erroneous surgical technique. Indeed, during the first stage of wound healing the laparotomy wound has practically no strength, relying completely on the sutures to hold it together. Documentation of the mechanisms of wound dehiscence after major abdominal surgery indicate most cases are considered to be secondary to tearing of sutures through the fascia, followed by infection, broken suture, fascial necrosis, and loose knots. Independent patient-related risk factors and type of surgery associated with postoperative wound dehiscence based on a validated surgical cohort risk factor analysis are presented in Table 1.[2]

The mechanism of incisional hernia recurrence is most often infection or inadequate fixation or overlap of the prosthesis. Although incisional hernia mainly occurs in the remodelling stage or even after healing has taken place, its formation may be attributable to improper healing in the inflammatory and regenerative stages. Indeed, risk factors involved in wound dehiscence are also related to incisional hernia, and wound dehiscence is itself a strong predictor of incisional hernia. Specifically, patient-related factors that increase the risk of developing incisional hernia are mainly obesity, chronic pulmonary disease, wound infection and age. Interestingly, recurrent hernia is a risk factor for future incisional hernias, implying that these patients suffer a common underlying dysfunction in wound-healing that predisposes to hernia formation.[3]

Risk scores for postoperative wound dehiscence

Variable	Risk score[a]
Age category, years	
40–49	0.4
50–59	0.9
60–69	0.9
>70	1.1
Male gender	0.7
Chronic pulmonary disease	0.7
Ascites	1.5
Jaundice	0.5
Anemia	0.7
Emergency surgery	0.6
Type of surgery	
Gallbladder/bile duct	0.7
Oesophagus	1.5
Gastroduodenum	1.4
Small bowel	0.9
Large bowel	1.4
Vascular	1.3
Coughing	1.4
Wound infection	1.9
Theoretical score (min–max):	0–10.6

[a] Independent risk scores may be added up to create the cumulative risk. Absolute risk (mean probability) in a validation population: score 0–2: 0.1%, 2–4: 0.7%, 4–6: 5.5%, 6–8: 26.2%, >8: 66.5%. Adapted from Ramshort et al.[2]

Table 1

Malnutrition

Besides adequate intake of carbohydrates, protein, and fat, it has been shown that vitamins A, B complex and C, the micronutrients copper, zinc and iron, and certain essential amino acids are involved in one or more stages of the wound healing cascade, and deficiencies can cause poor and delayed healing. Creating an adequate preoperative nutritional state seems to be more important to successful wound-healing than the overall nutritional status of the patient, and proper preoperative assessment is essential.

Collagen metabolism and incisional hernia

There are many types of collagen, of which subtypes I and III are important in incisional hernia formation. Type I collagen is responsible for strong tensile properties, whereas type III collagen is thinner and more flexible, and is considered an immature variant in connective tissue, as it is expressed early on in the healing process later to be replaced by type I. A decreased collagen type I/type III ratio results in altered arrangement and smaller diameter of collagen fibrils and a decreased amount of cross-linking, overall reducing the mechanical stability of the connective tissue. A decreased type I/type III ratio has been observed in patients with incisional and recurrent incisional and inguinal hernia, suggesting an underlying causative role for abnormal collagen metabolism.[4]

Genetic disorders of connective tissue have also been linked to incisional or other types of hernia, including Ehlers–Danlos, Marfan's syndrome, autosomal dominant polycystic kidney disease, osteogenesis imperfecta, and congenital dislocation of the hip. Similarly, increased incisional hernia rates in patients treated for abdominal aortic aneurysm are possibly related to an underlying disorder in collagen metabolism pathogenic for both ailments. These observations support an important biological role (genetic or acquired) in the pathogenesis of incisional hernia.

How to open the abdomen

Consensus on the superior type of incision regarding early postoperative wound complications and development of incisional hernia remains elusive. A recently updated Cochrane review of randomized controlled trials comparing midline and transverse incisions revealed a trend of lower wound dehiscence and incisional hernia rates associated with transverse incisions.[5] Even though pulmonary function was significantly less affected after a transverse incision, pulmonary complications did not differ between midline and transverse incisions. Analgesic requirements were significantly lower with transverse incisions, although the studies involved displayed significant heterogeneity. Data on cosmesis could not be pooled due to differences in assessment criteria, although the two studies that evaluated this aspect both suggested a significant preference for transverse incision. No