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Advanced Management of Mediastinitis Post Cardiac Surgery

Essay

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General Surgery**

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Aim of the work

Review of risk factors and advanced diagnostic&management modalities of post cardiac surgery mediastinitis.

Introduction

Mediastinitis is a life-threatening condition with an extremely high mortality rate if recognized late or treated improperly. Although long recognized as a complication of certain infectious diseases, most cases of mediastinitis are associated with cardiac surgery. This complication affects approximately 1-2% of these patients. Although small in proportional terms, the actual number of patients affected by mediastinitis is substantial. This significantly increases mortality and cost. After 10 years of evolution, the optimal therapy for mediastinitis is more clearly understood (*Abboud et al., 2004*).

Risk factors for the development of mediastinitis postoperatively include the use of pedicled Bilateral Internal Mammary Artery (BITA) as conduits for Coronary Artery Bypass Grafting surgery (CABG), this risk is even higher among patients with diabetes, other risk factors include emergency surgery, external cardiac compression (conventional cardiopulmonary resuscitation), Obesity (>20% of ideal body weight), postoperative shock, especially when multiple blood transfusions are required, prolonged bypass and operating room time, reoperation and reexploration following initial surgery, sternal wound dehiscence, and surgical technical factors (e.g. excessive use of electrocautery, bone wax, paramedian sternotomy) (*Braxton et al., 2000*).

Most mediastinitis cases occur after cardiac surgery and indicate gram-positive cocci, with *Staphylococcus aureus* and *Staphylococcus epidermidis* accounting for 70-80% of cases. Mixed gram-positive and gram-negative infections account for approximately 40% of cases. Isolated gram-negative infections are rare causes (*De Paulis et al., 2005*).

The origin of infection following open heart operations is not known in most patients. Some believe that the process begins as an isolated area of sternal osteomyelitis that eventually leads to sternal separation. Others hold that sternal instability is the inciting event, and bacteria then migrate into deeper tissues. Inadequate mediastinal drainage in the operating room may also contribute to the development of a deeper chest infection. The patient's own skin flora and the bacteria in the local surgical environment are possible sources of infection because some bacterial contamination of surgical wounds is inevitable, host risk factors are likely critical in promoting an active infection (*El Oakley and Wright, 1996*).

The infection of surgical wounds of sternotomies should be considered as Superficial if only the skin and subcutaneous tissues are involved, Deep when the infection reaches the sternum but doesn't involve it, and Organ/ Space when sternal osteomyelitis or mediastinitis occurs. This classification enables a better comparison of related research (*Fowler et al., 2005*).

Approximately two thirds of patients present within 14 days following surgery. Although a delay of months is occasionally observed. The Typical postoperative Patient presents with sternal pain that has increased since surgery, drainage from the wound site, an audible click due to sternal nonunion, progressive redness over a variable period and leukocytosis (*Gardlund et al., 2002*).

The first step in treating mediastinitis is to assess the degree of sternal stability and classify the infection by type. Sternal preservation is a primary goal of treatment for a deep wound infection such as mediastinitis if surgery is performed early, before the sternum

deteriorates. Operative exploration includes reopening the previous sternotomy and debridement of necrotic and infected tissue. The sternum is separated from the ventricle, coronary grafts and the aorta carefully not to cause bleeding. Cultures are sent to direct antibiotic therapy (*Friberg et al., 2005*).

Wound closure is usually delayed until reasonable control of infection is achieved; however, some surgeons perform closure with muscle flaps at the initial debridement with good results. Delayed closure is usually accomplished with muscle flaps (pectoralis, rectus), omental flaps, and recently is aided by vacuum-assisted closure, great care should be taken to exclude active infection before rewiring the sternum in cases of sternal nonunion (*Haas et al., 2005*).

When the infection is under control, the surgeon (usually a plastic surgeon) will use muscle and skin flaps to close the incision by secondary intention. The bone may be approximated, depending on the degree of sternal resection required. The surgeon may use a technique known as the Robicsek procedure to give the weakened sternum additional support (*Milano et al., 2007*).

In vacuum-assisted closure the wound is debrided, and polyurethane foam is applied. An evacuation tube, vacuum pump, and transparent drape complete the dressing. Negative pressure is applied to the wound to drain fluid, inhibit bacterial colonization, stimulate tissue granulation, and reduce the frequency of dressing changes. Delayed primary closure may occur, or the wound may be prepared for secondary closure with flaps (*Omran et al., 2007*).

Anatomy and Development

The Sternum and its Joints:

The sternum is an elongated, flat bone that lies in the anterior midline. The manubrium upper border is marked on either side by a notch for articulation with the clavicle. Centrally, the widest portion of the manubrium is marked by bilateral indentations for articulation of the first costal cartilage, at the lower limits, demifacet for articulation of the upper half of the second costal cartilage. The lower margin of the manubrium articulates with the body of the sternum. The body or longest portion of the sternum articulates with the manubrium to form an angle, called sternal angle. The outer border of this angle is readily palpable. It is sufficiently flexible to allow movement of the body on the more stable manubrium during respiratory movements. Lateral margins of the body articulate with costal cartilages two to seven. The body ends at about the level of the tenth to eleventh thoracic vertebrae, where it forms a cartilaginous joint with the xiphoid process. The xiphoid is a cartilaginous process that is usually ossified by middle age (*Carlson, 1999*).

Ribs and Their Joints:

The size and shape of the thorax are largely determined by the ribs and costal cartilages (*Van De Graaff, 2002*). The upper seven pairs of ribs articulate direct with the sternum by way of costal cartilages and are therefore called true vertebrosteral ribs. In contrast, the lower five pairs are called false ribs because they do not articulate with the sternum at all. Of the false ribs, three pairs (the eighth, ninth and tenth) are called vertebrocostal because their associated cartilages articulate with the costal margin. The remaining pairs

(eleven and twelve) terminate in cartilaginous tips, ending muscles of the abdominal wall. Because their only articulation vertebrae, they are called vertebral ribs. The fused cartilages of ribs seven to ten course diagonally upward to the lower end of the sternum to form the infrasternal angle (*McMinn et al., 1995*).

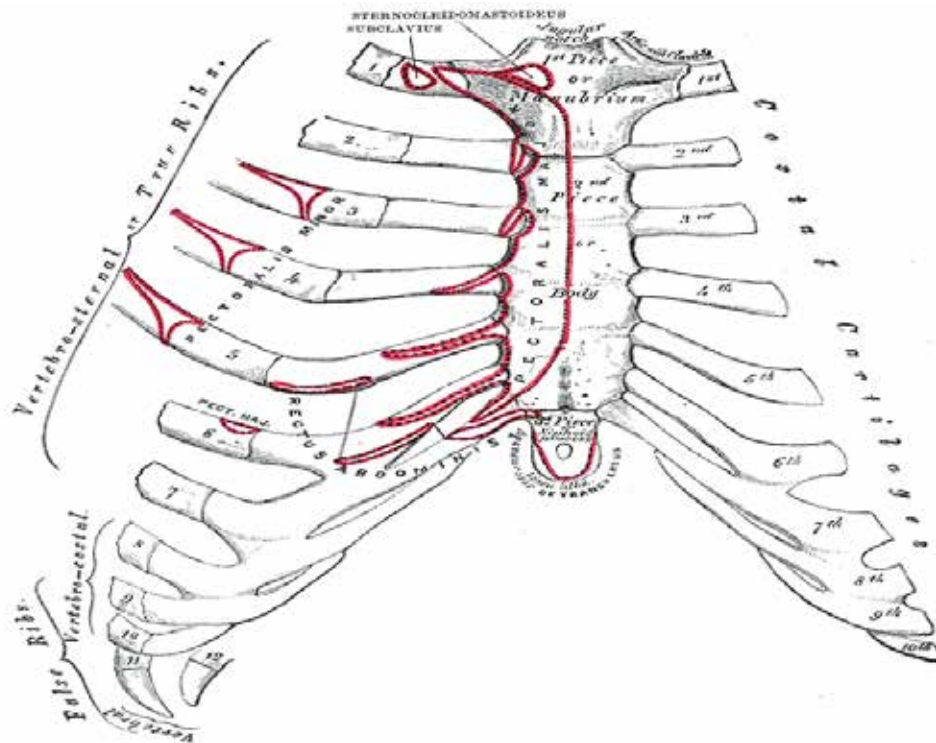


Fig. 1: Anterior surface of sternum and costal cartilages (*McMinn et al., 1995*)

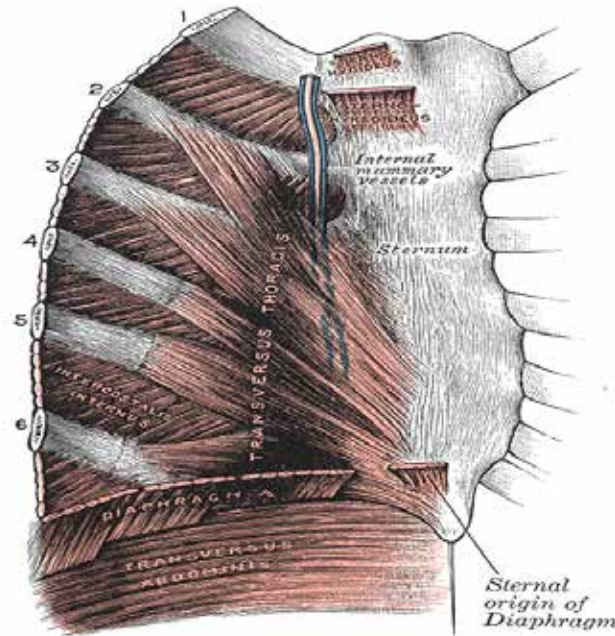


Fig. 2: Posterior surface of sternum and costal cartilages, showing Transversus thoracis (*McMinn et al., 1995*)

Complications of Sternal Wound Incisions

1. Complicated Healing:

- a. Hypertrophic scar and keloid:** Midline incisions have tendency to form raised, red hypertrophic scars (where the lumpy scar tissue exceeds the margin of the scar) are common in patients of African descent. There is no known way of avoiding these unsightly scars, apart from attempting to minimize the length of the sternotomy incision, or considering submammary incisions.
- b. Sternal wires sinus:** sometimes patients complain of prominent sternal wires or infected wire with sinus tract to the skin. This is commonest in very thin patients. If sternum is stable, sternal wires may be removed. This should be done under general anaesthetic with appropriate

monitoring; even a simple removal of sternal wires can turn into an unexpected resternotomy. If the patient is unhappy with 1 or 2 wires , these can be removed through stab incisions (*Chikwe et al., 2006*).

2. Failure of Healing:

- a. **Mediastinal dehiscence:** Median sternotomy wound breakdown in the absence of clinical or microbiological evidence of infection
- b. **Infection:** Mediastinal wound infection Clinical or microbiological evidence of infected presternal tissue and sternal osteomyelitis, with or without mediastinal sepsis and with or without unstable sternum (*Mills and Bryson, 2006*).

Ø Sternal Wound Infection and Dehiscence:

A number of patients suffer problems with delayed or impaired wound healing, wound dehiscence and infection. Recent work suggests that infection rates are about 1.9% (*Mills and Bryson, 2006*).

Sternal wound dehiscence and infection is a serious complication that carries significant consequences for the patient and for service provision. Some cases require further surgery, including repeated debridement and major surgical reconstruction. There is almost invariably considerable increase in the length of hospital stay, and the incidence of further complications is high (*Kappstein et al., 1992*).

Patients who develop sternal wound infection have an inpatient mortality of 14% (normally about 2%), a three fold increase in mortality over the first 4 years after surgery and a significantly higher short term and long-term

morbidity. Wound infection also carries a 2.8 times increase in the financial cost of the procedure (*Braxton et al., 2000*).

Median sternotomy wound complications range from sterile wound dehiscence to suppurative mediastinitis and it is difficult to make comparisons between cases and between treatments. This has been recognized and has been attributed, partly to the lack of a widely accepted and comprehensive definition of what constitutes wound infection (*El Oakley et al., 1996*).