

# **Acute and Chronic Mediastinal Infections**

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- Mediastinal infections = mediastinitis.
- **Acute mediastinitis:**
- usually secondary to infections ( operations requiring sternotomy or arising from perforation of the aerodigestive tract).
- Acute mediastinitis due to the spread of oropharyngeal infections, =descending mediastinitis, represents a less common but extremely lethal form of this disease.
- **Chronic infections** : uncommon; a minor amount of change in the presentation, diagnosis, and management of this disease has evolved.
- (Most fungal disease)( few are secondary to mycobacterial organisms).
- Chronic fungal or tubercular infections maybe self-limiting but may progress into the clinical entity of chronic fibrosing mediastinitis.

# Postoperative Sternal Infection and Mediastinitis

- The incidence of mediastinitis after cardiac surgical between **1%and 4%**.
- **causes and risk factors:**
- diabetes, chronic obstructive pulmonary disease , congestive heart failure, use of internal mammary artery grafts (unilateral or bilateral), smoking, reoperation, lower ejection fraction, prolonged ventilation, obesity, high body mass index (BMI), immunosuppressive therapy, older age, use of bone wax, preoperative renal failure, duration of operation, prolonged cardiopulmonary bypass and aortic cross-clamp times, off-center sternotomy, improper stabilization of the sternum, poor hemostasis, use of pacing

- wires, need for repeated blood transfusions in the early postoperative period, use of electrocautery, presence of infection elsewhere, extended intensive care unit stay and overall hospitalization, readmission to the hospital.
- ***Theories for mechanism of infection :***
  - 1. local osteomyelitis at the sternotomy
  - 2. Sternal instability contributes to the superficial wound dehiscence and that this serves as a portal of ingress for infections.
  - 3. Inadequate drainage in the retrosternal space serves as a culture medium for the source of mediastinal infection.
  - 4. Concomitant infections, such as a nosocomial pneumonia

- **Bacterial pathogens** :usually **Staphylococcus aureus** and **Staphylococcus epidermidis**, (50% to 80% ).
- skin flora at the time of operation.
- **Perioperative contamination** :
- leg incision used to harvest a saphenous vein graft.
- **Postoperative contamination**:
- Gram-negative (Pseudomonas, Serratia, and Klebsiella ).
- increased nosocomial infections and prolonged antibiotic use in the postoperative care of more complex and challenging cardiac patients.
- **Mixed infections account for up to 40% of cases.**
- **Fungal mediastinitis** :infrequent cause of post sternotomy mediastinitis .
- should be considered in the setting of failed therapy or prolonged antibiotic use.
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# Diagnosis

mortality mediastinitis after CABG: **10% to 50%.**

first-year survival rate after coronary artery bypass graft was 78% with mediastinitis and 95% without, with a threefold increase in mortality rate at 4 years' follow-up.

# Clinical Manifestations

- Clinically, postoperative mediastinitis should be suspected in the infected sternotomy wound.
- occur **early** or **late** in the clinical course.
- **Classic signs :**
- erythema, purulent discharge, and sternal instability.
- A history of pain with breathing or difficulty lying in the lateral decubitus position is indicative of the two halves of the sternum moving against each other.
- It has been suggested that sternal instability as assessed by bi manual alternating sternal compression is the most helpful diagnostic maneuver.
- Fever, sepsis, or leukocytosis, especially without an obvious source may, be a presentation.

# ***Radiographic studies***

- are not routinely used,
- particularly in the acute early postoperative period.
- ***computed tomography (CT)*** scanning( in the late presentation ) or in the evaluation of unresolving sepsis due to an untreated source of mediastinitis.( An undrained fluid collection or air–fluid level). >30 days after surgery
- ***nuclear imaging studies***



# Treatment

- has evolved tremendously in recent years.
- Despite the large number of series evaluating the risk factors for post sternotomy mediastinitis, the majority of these reports do not include an analysis of its management.

***Conventional therapy—defined as opening and debriding***

- ***serially packing the wound, and eventually closing the wound primarily***
- ***debridement and primary closure*** has involved the use of retrosternal high-negative-pressure catheters, (Redon catheters)
- ***flap closure techniques*** in the treatment of poststernotomy mediastinitis is frequently accepted as (the standard therapy for these deep sternal wound infections). mortality rates of <10%
- ***mediastinal irrigation with either saline or antibiotic solution to the debridement and primary closure***

- . Dilute povidone-iodine or antibiotic irrigation was used until mediastinal fluid cultures dictated its cessation.
- simple debridement with rewiring of the sternum laterally for stabilization (Robicsek weave) and primary closure followed by postoperative closed mediastinal irrigation. Their closure technique, followed by a culture-driven antibiotic irrigation solution, result ed in a remarkable success rate of 98%. Equally impressive in both series was the fact that the mortality rates were 0%. Closed drainage irrigation using Redon catheters has been described as another variation of closed mediastinal drainage.<sup>79</sup> Comparisons of Redon catheters to closed mediastinal irrigation have shown that the use of the latter technique is associated with improved failure and mortality rates.<sup>19</sup> In contrast to these studies, others have shown that closed mediastinal irrigation following primary closure of sternal dehiscence in culture-proven mediastinitis is associated with an extremely high rate of failure. This has led some authors to caution against the use of this modality where an internal thoracic graft has been used and there is culture-proven infection.<sup>112</sup> Flap coverage is not limited to the pectoralis advancement flap. Omental flaps have been reported to be an adequate source of flap coverage and some have reported its benefits over muscle flaps. Milano and colleagues<sup>97</sup> demonstrated that omental flaps were associated with shorter operations and decreased lengths of hospitalization as well as lower rates of early complications. Furthermore, recurrent infections are more common with muscle flaps.<sup>97,145</sup> Brandt and Alvarez<sup>12</sup> have used both pectoralis flaps and omental flaps to cover the wound and occupy any potential dead spaces, with impressive results, including fewer major complications, shorter hospitalizations, decreased mortality rates, and increased overall survival.<sup>12</sup> Combination therapy with closed mediastinal irrigation using either primary closure alone or in combination with flap coverage has also been described with success. Rand and colleagues<sup>112</sup> showed that muscle flap closure with closed mediastinal irrigation. Hirata and colleagues<sup>65</sup> outlined the use of closed drainage irrigation following open debridement and omental flap closure in the setting of methicillin resistant *S. aureus* (MRSA) infections in four patients.

- Vacuum-assisted closure for the treatment of open wounds was first described by Argenta and Morykwas<sup>5</sup> in 1997. Since then, its use has been expanded to include poststernotomy wounds due to mediastinitis. The benefits of wound vacuum-assisted therapy have been postulated to be multifactorial, but they share the common theme of relying on the associated effects of negative pressure. An increase in local blood flow; decrease in tissue edema and bacterial load; and removal of stagnant fluid, necrotic debris and proteins impeding healing are all believed to promote wound healing. Furthermore, the mechanical effects exerted by the negative pressure is also thought to promote wound closure. <sup>52,86</sup> Obdeijn and colleagues<sup>106</sup> published one of the first reports of a vacuum-assisted closure of open wounds in three patients with poststernotomy mediastinitis. In this report all of the patients avoided the need for secondary surgical closure, as closure by accelerated secondary intention was achieved. Subsequent experiences, on a larger scale, have demonstrated that vacuum-assisted treatment of wounds can be an extremely useful adjunct in the management of poststernotomy mediastinitis. <sup>28,36,52,86,97,125,126,127</sup> Some of these studies have relied on vacuum-assisted closure exclusively. <sup>36,52,86</sup> However, not all investigations have employed vacuum-assisted therapy as the sole form of wound closure, since others have used this technique intentionally as a “bridge” to another form of definitive therapy. <sup>36,86</sup> However, even with this intention, vacuum-assisted therapy has, in some cases, precluded the need for flap coverage or sternectomy. <sup>28</sup> The depth of infection has been thought to determine which patients will require progression to a second operation for closure. <sup>36</sup> Direct comparisons to all forms of conventional therapy with omental or muscle flap closure have shown a significant advantage of vacuum-assisted therapy for wound closure in terms of decreased mortality, increased survival, lower frequencies of local failure, and shorter hospitalizations. <sup>52</sup> In contrast, studies of poststernotomy mediastinitis that have not included vacuum-assisted wound closure therapy have described a worse long-term survival. Sjogren and colleagues<sup>125,126,127</sup> have demonstrated that with the use of this treatment, long-term survival is no different than that of postoperative coronary artery bypass patients without mediastinitis. Others have shown that vacuum-assisted therapy can facilitate primary closure by accelerating granulation. <sup>37</sup> Catarino and colleagues<sup>21</sup> have shown that vacuum-assisted therapy compared with closed drainage irrigation alone is associated with no treatment failures and decreased lengths of hospitalization. Others have also demonstrated that there is a shorter length of hospitalization with vacuum-assisted therapy when compared with continuous irrigation. <sup>21,37</sup>

- ir colleagues and are beyond the scope of this chapter (see Chapter 146). Management strategies of esophageal perforation including that accompanying mediastinitis are based on four principles:  
Eliminate source of soilage by primary repair or diversion away from the esophageal perforation. Provide thorough and wide mediastinal drainage to control ongoing mediastinal suppuration occurring after primary repair or diversion. In addition, gastrostomy tube decompression should be performed to decrease gastric reflux and mediastinal soilage.  
P.2184 Appropriate antibiotics should be administered to augment host defenses, which must be effective against both gram-positive and gram-negative bacteria and against both aerobic and anaerobic bacteria. Maintain adequate nutrition. The ultimate goal is to restore alimentary tract continuity, as emphasized by Burnett and associates.18

# Descending Necrotizing Mediastinitis

- Estrera and associates<sup>46</sup> described acute purulent mediastinitis due to oropharyngeal infection as descending necrotizing mediastinitis. This infection remains an uncommon but still lethal form of mediastinitis. Etiology Of the reported cases, 60% to 70% are secondary to odontogenic infections.<sup>46,88,137</sup> Other common causes have included peritonsillar abscesses<sup>0,89</sup> (T et al.), retropharyngeal and parapharyngeal abscesses, <sup>27,46</sup> (Freeman et al.) and epiglottitis.<sup>23,78</sup> Other less common causes of descending necrotizing mediastinitis include trauma to the neck, including neck or mediastinal surgery; cervical lymphadenitis; and endotracheal intubation, as reported by Guardia and associates,<sup>63</sup> Uram and Hauser,<sup>133</sup> and Gould and colleagues. <sup>61</sup> Alsoub and Chacko<sup>4</sup> have listed the many causes of this lethal infection. Anatomically, there are three potential planes through which descending necrotizing mediastinitis can progress: (a) pretracheal, (b) perivascular, and (c) prevertebral spaces. The pretracheal space, also referred to as the superficial layer, is just anterior to the trachea. It is bound by the thyroid cartilage superiorly and pericardium and parietal pleura inferiorly at the carina. The perivascular space is bound by the carotid sheath and descends into the mediastinum along with the structures within the carotid sheath. This route of spread results in infections of the middle mediastinum. Finally, the prevertebral space, also referred to as the retropharyngeal space, is bound anteriorly by the posterior aspect of cervical fascia and posteriorly by the alar fascia; it extends inferiorly until these two fascia coalesce at the first thoracic vertebra. <sup>60,102,108</sup> Most cases of descending necrotizing mediastinitis are secondary to spread in this last plane and result in involvement of the posterior mediastinum (Fig. 173-1). All these spaces are joined by loose connective tissue, which facilitates direct spread within these planes.<sup>99</sup> Gravity and negative pressure during inspiration allow for the descent of the infected and purulent material into the mediastinum and pleura. <sup>99</sup> Odontogenic and peritonsillar abscesses may extend to involve the submandibular and parapharyngeal spaces, which, as McCurdy and colleagues<sup>93</sup> have noted, readily communicate with all major cervical fascial spaces. Chow<sup>24</sup> and Brook and Frazier<sup>15</sup> have recorded that the microbiological features of descending necrotizing mediastinitis are polymicrobial, with aerobes and anaerobes, reflecting the indigenous microflora of the oral cavity. The most common organisms isolated include Prevotella, Peptostreptococcus, Fusobacterium, Veillonella, Actinomyces, oral Streptococcus, Bacteroides, S. aureus, Haemophilus species, and Bacteroides melaninogenicus. Symbiosis between one or more species of gram-negative aerobic bacteria and an anaerobe can result in synergistic

- Mathieu and associates<sup>90</sup> have described predisposing conditions that may favor this infectious process; such conditions include diabetes (13.3%), alcoholism (17.7%), neoplasm (4.4%), and radionecrosis (3.3%). In particular, they found that age >70 years and underlying diabetes were fatal risk factors.

# Diagnosis

- The criteria used for the diagnosis of descending necrotizing mediastinitis are clearly defined by Estrera and colleagues<sup>46</sup> and include (a) clinical evidence of severe oropharyngeal infection, (b) characteristic roentgenographic features of mediastinitis, (c) documentation of necrotizing mediastinal infection at the operation or postmortem or both, and (d) establishment of the relationship between descending necrotizing mediastinitis and the oropharyngeal process. Because this infection progresses rapidly, early diagnosis is essential. Computed tomography (CT) scanning is more reliable than chest radiography and can provide precise information on the extent of the infection, which will guide the optimal approach used for surgical drainage.



# Clinical Manifestations

- Descending necrotizing mediastinitis is seen most often in a patient who is under treatment for a deep cervical infection resulting from one of the aforementioned causes. Despite antibiotics and even drainage of the deep cervical space, the infection progresses to involve the mediastinum. Early diagnosis is often difficult because of the vagueness of early symptoms that would indicate mediastinal involvement. Unfortunately the usual delay in diagnosis contributes greatly to the high mortality associated with descending necrotizing mediastinitis.<sup>88</sup> Descending necrotizing mediastinitis may occur at any time after cervical infection, manifest by signs and symptoms of sepsis with stiffness, swelling, and neck pain. Cranial nerve deficits, trismus, and stridor have also been described.<sup>60</sup> Dysphagia may or may not be present. Mediastinal involvement may occur as soon as 12 hours to as late as 2 weeks, but it is most commonly seen within 48 hours after the onset of deep cervical infection. Diffuse brawny induration of the neck and upper anterior chest wall is seen. Pitting edema and crepitation may be present in the area. Substernal pain, increased dysphagia, cough, and dyspnea may also develop. Pleural and pericardial involvement may occur, since the necrotizing process involves the adjacent spaces. Pleural effusion, nonspecific electrocardiographic changes, and even infection of the retroperitoneal space of the abdomen may develop as the inflammatory process ensues. The capillary leak that occurs with sepsis can further exacerbate dehydration and lead to acute respiratory distress syndrome, cardiac tamponade, and empyema.<sup>78,108</sup>

# Radiographic Features

- CT is the diagnostic imaging modality of choice. Estrera and associates<sup>46</sup> reported four radiographic features of the neck and chest present in descending necrotizing mediastinitis: (a) widening of the retrocervical space with or without an air–fluid level, (b) anterior displacement of the tracheal air column, (c) loss of the normal lordosis in the cervical spine. Also, the superior mediastinal shadow can be widened, and findings of pleural or pericardial involvement may be evident (Fig. 173-2). CT scans of the chest are better than chest radiographs in delineating the infectious process. Carroll<sup>12</sup> and Breatnach<sup>20</sup> and their associates outlined several CT findings in descending necrotizing mediastinitis: (a) abscess formation, (b) soft tissue infiltration with loss of the normal fat planes, (c) absence of prominent lymphadenopathy, and (d) the presence of gas bubbles. Air and fluid can be seen in the visceral or anterior compartments, as can pleural or pericardial effusions. mediastinal emphysema, and (d)

- Treatment The management of descending necrotizing mediastinitis includes surgical drainage, antimicrobial therapy, and airway management. The surgical approach depends on the location of the abscess. Estrera and coworkers<sup>46</sup> stated that if the infection is in the space below the level of the tracheal bifurcation anteriorly or the fourth thoracic vertebra posteriorly, mediastinal drainage should be performed via a transthoracic approach. If only the superior mediastinum is involved and the infection is contained above the level of the carina or the fourth thoracic vertebra, standard transcervical mediastinal drainage may be adequate, as suggested by Wheatley and colleagues.<sup>137</sup>

P.218 Marty-Ane and colleagues<sup>88</sup> have proposed a more aggressive approach regardless of the level of infection, including the transthoracic approach through a standard thoracotomy in addition to cervical drainage. Transthoracic drainage has been demonstrated to result in better debridement and improved survival, as reported by Temes and coworkers.<sup>131</sup> Further evidence for the inclusion of routine transthoracic drainage is provided by Corsten and associates'<sup>27</sup> meta-analysis comparing neck and thoracic drainage (19% mortality) with transcervical drainage alone (41% mortality,  $p < 0.05$ ). Shimizu and colleagues<sup>124</sup> recently described the successful use of video-assisted mediastinoscopy with adequate drainage of the cervical neck, anterior mediastinum, and middle mediastinum to the level of the tracheal bifurcation. Numerous reports have described various different thoracic approaches. The standard posterior or lateral thoracotomy has unequivocally been the classic approach to mediastinal infections. In 1996, Ris and colleagues<sup>114</sup> reported on the use of the clamshell incision. Sternotomy has been described as both inadequate and dangerous in the treatment of descending necrotizing mediastinitis owing to inability to drain the posterolateral compartments of each thoracic cavity and the risk of introducing osteomyelitis and its attendant sequela of sternal dehiscence. With the increased popularity of minimally invasive surgery, several authors<sup>60,69,115</sup> have reported the use of thoracoscopic drainage in the management of descending necrotizing mediastinitis. The thoracoscopic approach has been reported to decrease morbidity versus a thoracotomy and to improve drainage of the mediastinum compared with cervical drainage. Percutaneous drainage has also been described, but its greatest utility may be in stabilizing of the critically ill patient rather than serving as the definitive form of therapy.<sup>59</sup> Irrespective of the treatment selected, mediastinal pleural irrigation following surgical debridement of the mediastinum with either saline or antibiotic solution akin to a modified Claggett procedure has achieved success in the setting of descending necrotizing mediastinitis by some,<sup>70</sup> but others have disputed its benefit.<sup>119</sup> Antimicrobial therapy should be given promptly and should cover both aerobes and anaerobes. At present, a single agent such as carbapenem, as noted by Sakamoto and coworkers,<sup>118</sup> will effectively cover both. Initial antibiotic choice should offer the broadest possible coverage, with combinations used as necessary. Later on, when culture results are available, the antibiotics can be tailored accordingly. The role of tracheostomy is controversial, as it may exacerbate the spread of infection via the pretracheal space.<sup>30</sup> Adequate drainage alone should suffice, as this is the major means by which patients can recover from their sepsis, and it obviates the need for prolonged intubation.

- Prognosis The mortality rate for patients with descending necrotizing mediastinitis before the antibiotic era was approximately 50%, yet the rate has only decreased to 40% despite the introduction of antibiotics, refined surgical techniques, and intensive care monitoring, as noted by Guardia,<sup>63</sup> Estrera,<sup>46</sup> and Levine<sup>83</sup> and their coworkers. The reasons for this are that the infection spreads rapidly, leads to fulminant sepsis, and that, as a rule, there is a significant delay in diagnosis and initiation of the appropriate therapy. Since 1990, there has been a decrease in the mortality rate to 15.4%, largely because of the more aggressive approach taken to treat these infections, as discussed by Brunelli and associates.<sup>16</sup> Death may result from fulminant sepsis, blood vessel erosion with exsanguination, aspiration, metastatic intracranial infection, empyema, and purulent pericarditis with tamponade.