

Pulmonary complications after coronary revascularization

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Pulmonary complications are common after coronary artery bypass grafting. Identifying those individuals with increased risk of respiratory complications allows for appropriate preoperative intervention. The most commonly seen pulmonary complications include pleural effusion, hemothorax, atelectasis, pulmonary edema, diaphragmatic dysfunction, and pneumonia. Clinical features and appropriate management of these common problems are discussed. *Curr Opin Cardiol* 2000, 15:309–315

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Current Opinion in Cardiology 2000, 15:309–315

Abbreviations

CPAP continuous positive airway pressure
PEEP positive end-expiratory pressure

ISSN 0268–4705 © 2000 Lippincott Williams & Wilkins, Inc.

Since being introduced in the 1950s, the number of cardiac procedures performed each year has increased exponentially. Improvements in surgical technique, shortening of intraoperative pump time, and advances in the perioperative support of patients with limited cardiopulmonary reserve have made it possible for these individuals to survive such stressful interventions. With the evolution of invasive cardiology, patients with lesser degrees of disease are often treated in the cardiac catheterization laboratory, and surgeons now find themselves dealing with higher risk patients [1].

Many of these patients have chronic respiratory diseases that contribute to postoperative complications. However, pulmonary complications are common even in patients without underlying pulmonary disease. One study has shown that nearly 20% of otherwise healthy patients who underwent elective coronary artery bypass grafting suffered postoperative pulmonary complications [2]. Because these complications have been shown to increase hospital costs and prolong length of stay [3,4], their prevention is of considerable importance.

Risk reduction

Seven core variables (priority of operation, age, prior heart surgery, sex, left ventricular ejection fraction, percent stenosis of the left main coronary artery, and number of major coronary arteries with significant stenoses) are the most consistent predictors of mortality after coronary artery surgery. The greatest risk is correlated with the urgency of operation, advanced age, and one or more prior coronary bypass surgeries. Additional variables related to mortality include coronary angioplasty during index admission; recent myocardial infarction; history of angina, ventricular arrhythmias, congestive heart failure, or mitral regurgitation; and comorbidities such as diabetes, cerebrovascular disease, peripheral vascular disease, chronic obstructive pulmonary disease, and renal dysfunction [5].

Identifying patients with increased risk of postoperative pulmonary complications allows for preoperative interventions to minimize the undesirable effects of such a significant intervention. Increased pulmonary complication rates have been associated with a history of cigarette smoking or chronic obstructive pulmonary disease, hypercapnia, American Society of Anesthesiologists category two or higher, age greater than 59, weight

greater than 250 pounds, low serum albumin, or preoperative hospital stay greater than 4 days [6–8]. However, pulmonary function testing is recommended only for those patients with a history of routine cigarette smoking or those complaining of dyspnea [9].

Smoking cessation should be a primary goal in attempts to reduce postoperative pulmonary complication rates. Smoking more than ten cigarettes per day doubles the risk for postoperative pneumonia [10] because ongoing exposure to combustion products induces mucous gland hypertrophy with excess secretion production, mucous plugging, and resultant atelectasis. The timing of smoking cessation is crucial, with a minimum of 8 weeks of tobacco avoidance required to reduce the frequency of complications. Smokers who quit less than 8 weeks before surgery have higher complication rates than those who continue to smoke [11]. Any active infection should be eradicated before surgery.

It is also necessary to maximally alleviate airflow obstruction preoperatively. Chronic obstructive pulmonary disease is associated with an increased risk of postoperative atelectasis, bronchitis, pneumonia, ventilatory failure, and prolonged intensive care unit stay. Preoperative optimization of medical therapy such as inhaled bronchodilators, inhaled or systemic steroids, antibiotics, smoking cessation, and chest physiotherapy can reduce this risk [12,13]. Individuals refractory to preoperative intensification of therapy have particularly high rates of postoperative respiratory failure and prolonged mechanical ventilation [14].

Asthmatics should be asked about symptoms suggesting suboptimal control such as nocturnal awakenings, declining maximal peak expiratory flows, or escalating reserve inhaler use. Because preoperative steroid use (inhaled or oral) does not increase the incidence of postoperative pulmonary complications in asthmatics [15], the most recent National Institutes of Health expert panel recommends that corticosteroid therapy be considered pre- and postoperatively [16]. The use of prednisone, 1 mg/kg (up to 60 mg) daily for 3–7 days preoperatively, or hydrocortisone 100 mg every 8 hours perioperatively, and a rapid taper over 1 week after surgery, is not associated with an increase in wound complication, adrenal insufficiency, or death [17].

Pulmonary complications

Pleural effusion and hemothorax

The incidence of pleural effusions varies, depending on surgical technique and the diagnostic modality, but is as high as 90% [18]. Pleural effusions are most commonly small, unilateral, and left-sided [19]. Small effusions are generally insignificant and resolve sponta-

neously. Larger volumes can cause respiratory compromise by decreasing chest compliance, reducing the effective volume of ventilation, and contributing to atelectasis with alteration of the ventilation-perfusion ratio [20]. Bloody pleural effusions usually occur earlier, are associated with a higher lactic acid dehydrogenase level and are frequently eosinophilic. Non-bloody effusions are usually recurrent, more difficult to manage and may require anti-inflammatory agents, tube thoracostomy or pleurodesis [20]. Postoperative hemothorax is more common in patients undergoing internal mammary artery grafting. This occurs as a complication of dissection of the internal mammary artery during revascularization.

Most postoperative effusions are properly managed with observation alone. Any patient with a large effusion causing respiratory compromise should undergo prompt drainage. Effusions that are moderate-sized, fail to resolve spontaneously, or are accompanied by fever should each be sampled by thoracentesis. If the fluid obtained suggests hemothorax or infection, tube drainage is required. If not adequately drained, a pleural peel may form, with eventual trapping of the underlying lung [21] requiring thoracotomy and decortication. If thoracotomy is performed it should be done within 10 days of development of the effusion because the plane between the peel and the lung parenchyma becomes difficult to dissect after this point.

Atelectasis

Postoperative atelectasis is common, typically being seen in the lung bases within 48 hours after surgery, and is generally multifactorial in nature. Impaired mucociliary clearance and bronchospasm lead to small airway obstruction by secretions with segmental postobstruction atelectasis. Hypoventilation secondary to use of narcotics and prolonged breathing at low tidal volumes caused by pain and splinting also contribute to development of atelectasis. In addition, thoracotomy alters chest wall mechanics and anesthesia can impair lung mechanics, muscular function, and mucociliary clearance. Compression of lung parenchyma by space-occupying conditions such as pneumothorax, hemothorax, or pulmonary effusion further predisposes to development of atelectasis. It has also been postulated that cold cardioplegia solution may affect the production and function of surfactant [22,23].

The risk of atelectasis increases with increasing number of grafts, longer bypass and operative times, entrance into the pleural cavity, lower core body temperature, and failure to use a pericardial insulating pad [23]. Atelectasis is detrimental to patient recovery because it reduces lung compliance and vital capacity with a subsequent impairment in gas exchange. This

often results in a widened alveolar-arterial oxygen gradient caused by right-to-left shunting and an increase in the patient's work of breathing. In patients with underlying pulmonary compromise the net effect is often significant hypoxemia and an increase in pulmonary infection rates.

Atelectasis is best managed by prevention. Pain should be adequately controlled to facilitate deep inspiration. Given the sedation and central respiratory depression caused by intravenous boluses of opiates, there has been concern that such a mode of analgesia may blunt respiratory drive, causing hypoventilation and atelectasis. As such, epidural analgesia is now commonly used to minimize the amount of opiate required to provide effective pain control [24,25]. Although epidural therapy has failed to provide a statistical improvement in spirometric values [26], it is clearly superior in providing pain relief and minimizing side effects [27].

A variety of lung-expansion techniques, including deep breathing exercises, incentive spirometry, continuous positive airway pressure (CPAP), and intermittent positive pressure breathing have each been shown to be effective in the prevention and treatment of atelectasis [3,4,28]. The only expiratory maneuver with documented efficacy has been forced coughing. In compliant patients deep breathing exercises and incentive spirometry have been shown to reduce the frequency of radiologically and clinically apparent atelectasis. CPAP improves pulmonary mechanics more than any other modality, but because this has not translated into clear clinical benefit, CPAP is currently recommended only for patients who are unable to cooperate with deep breathing exercises or who have limited inspiratory capacity [29]. CPAP is most effective when applied for periods of 20 to 30 minutes, at least four times a day. Because intermittent positive pressure breathing is no more effective than the other methods, is much more expensive, and is often poorly tolerated, it is not recommended as first-line therapy [30]. Regardless of the modality chosen, therapy should be provided as frequently as possible for the first 4 days postoperatively.

In addition to lung expansion methods, an aggressive pulmonary toilet, including chest physiotherapy and nasotracheal suctioning, is often employed. If atelectasis of an entire lobe develops or if atelectasis persists more than 24 hours despite less invasive maneuvers, bronchoscopy is indicated for diagnostic and therapeutic purposes.

Pulmonary edema

Postoperative pulmonary edema is more common in patients with chronic pulmonary disease or in those with postoperative infection [31,32]. Because pulmonary vascular congestion may be caused by impaired left

ventricular function with subsequent elevation of pulmonary capillary pressure, it is often advisable to monitor central pressures with a pulmonary artery catheter for the first 24 hours postoperatively in patients with significant pulmonary disease. Hemodilution that occurs with cardiopulmonary bypass may lower oncotic pressure and contribute to formation of noncardiogenic pulmonary edema. Because of this, perioperative fluids should be minimized when possible.

Postoperative pulmonary edema should be aggressively treated with diuresis, positive inotropes, afterload reduction, angiotensin-converting enzyme inhibitors, and beta blockade as tolerated. Oncotic pressure should be corrected if abnormal. If necessary, patients should be electively intubated and initiated on mechanical ventilation. Ventilatory support with positive end-expiratory pressure (PEEP) should be continued until myocardial function is optimized and the patient can maintain adequate arterial oxygenation.

Diaphragmatic dysfunction

The effects of diaphragmatic dysfunction are potentially insignificant in otherwise healthy individuals, but patients with underlying pulmonary compromise are at increased risk of atelectasis and hypoxemia. Because early postoperative myocardial irritability is exacerbated by hypoxia, it is critical to consider phrenic nerve injury postoperatively in patients with unexplained hypoxemia.

Because myocardial oxygen demand decreases at lower temperatures, myocardial cooling confers cardiac protection intraoperatively. However, application of the cold cardioplegic solution results in phrenic nerve paralysis and associated diaphragmatic dysfunction in up to 26% of patients [33,34]. Use of a pericardial insulating pad to protect the phrenic nerve as it crosses the pericardium has been shown to significantly reduce the occurrence of nerve injury [35]. The time to recovery of diaphragmatic function varies from 30 days to 2 years, with 78% of patients regaining function at 1 year, and 97% recovering within 2 years [33].

Diaphragmatic dysfunction also occurs from other causes [23]. Direct injury to the phrenic nerve during mobilization of the internal mammary artery results in diaphragmatic dysfunction in almost 40% of patients [33]. Interruption of the blood supply to the phrenic nerve during mobilization of the internal mammary artery has also been described [36]. There are now numerous case reports of bilateral phrenic nerve injury. The occurrence of this complication is associated with significant morbidity and prolonged ventilator support [37,38]. Patients with chronic obstructive pulmonary disease who incur phrenic nerve injury after coronary artery bypass have a higher incidence of reintubation (23%), greater reduction

in forced expiratory volume in one second, and more frequent hospital readmissions [39].

Infection with herpes simplex virus (HSV) and cytomegalovirus infection (CMV) following cardiac surgery has been increasingly recognized as a serious problem associated with increased morbidity and mortality [47–49]. The clinical manifestations can range from asymptomatic viral shedding to persistent fevers, refractory bronchospasm, and difficulty weaning from mechanical ventilation [50,51].

Pneumothorax

Pneumothorax is a frequent complication of open heart surgery and is more common if postoperative mechanical ventilation is prolonged or if high levels of PEEP are required. Patients who suffer other postoperative pulmonary complications are more prone to develop concurrent pneumothorax. Infection weakens the alveolar endothelium and predisposes to alveolar rupture. Atelectasis causes overdistention of normal alveoli, also predisposing to alveolar rupture. Pneumomediastinum and/or subcutaneous emphysema often precede pneumothorax and should heighten awareness of a possible case of pneumothorax.

Pneumothoraces may develop tension with resultant chest pain, restlessness, hypoxemia, hypotension, and electrocardiogram changes consistent with ischemia. These findings may be attributed to a myocardial infarction or cardiac tamponade with initiation of inappropriate therapy while the unrecognized pneumothorax continues to increase in size. Therapy of a postoperative pneumothorax includes discontinuation of PEEP and maneuvers to decrease the drive pressure in mechanically ventilated patients. A chest tube should be placed if the pneumothorax is large, develops tension, or if the patient cannot be effectively ventilated.

Pulmonary infection

Pneumonia has an incidence of approximately 20% in patients undergoing coronary artery bypass grafting [2]. The risk of pneumonia is doubled in patients who are active smokers [10] and in those with postoperative atelectasis. Introduction of organisms into the normally sterile lower respiratory tract is caused by microaspiration of oropharyngeal contents. This can occur during intubation, at any time while the endotracheal tube is in place, or postoperatively with excess sedation [40,41]. An increased length of preoperative hospital stay increases the likelihood of colonization by virulent hospital-acquired gram-negative bacilli, which are difficult to treat secondary to rising levels of resistance [6,42]. However, preoperative antibiotic decontamination of the gastrointestinal tract has consistently been ineffective.

Increasing gastric pH by gastric acid suppression or inhibition favors gastric colonization and more frequent inoculation of the respiratory tree. This has resulted in increased rates of pneumonia in these patients [43]. At present, recommendations to reduce postoperative pneumonia are limited to preoperative smoking cessation and prevention of postoperative atelectasis. Because the physiologic stress of open heart surgery is associated with viral reactivation and superinfection [44–46], any pneumonia or tracheo-bronchitis not responding to empiric antibacterial therapy should prompt an investigation for a viral etiology.

Infection with herpes simplex virus (HSV) and cytomegalovirus infection (CMV) after cardiac surgery has been increasingly recognized as a serious problem associated with increased morbidity and mortality [47–49]. The clinical manifestations can range from asymptomatic viral shedding to persistent fevers, refractory bronchospasm, and difficulty weaning from mechanical ventilation [50,51].

Pulmonary embolism

After cardiac surgery, patients are at increased risk of pulmonary embolism secondary to hypercoagulability, immobility during surgery, and surgical trauma to the lower extremities during saphenous vein graft harvesting. Despite this, pulmonary embolism is uncommon following cardiac surgery, with a published incidence of approximately 3% [52,53]. However, patients with pulmonary emboli postoperatively have a mortality rate nearing 20% [54] and invariably have a prolonged hospitalization. Patients with chronic lung disease and limited exercise tolerance are particularly predisposed to pulmonary embolism.

Sudden onset of dyspnea demands a rapid diagnostic evaluation because the differential diagnosis includes such life-threatening conditions as pneumothorax, pericardial tamponade, and myocardial infarction. If less invasive modes are not diagnostic, pulmonary artery angiography should be considered. Therapy is preventative, with early ambulation for all patients who are able to tolerate it. The use of sequential pneumatic compression stockings until the patient is fully ambulatory has been shown to further reduce the risk of pulmonary embolism [55].

Respiratory failure

Some degree of arterial hypoxemia is expected postoperatively due to reductions in total lung capacity, functional residual capacity, residual volume, and lung compliance, with a concurrent increase in pulmonary vascular resistance. Anesthesia, analgesia, and muscle relaxants depress respiration centrally and at the level of

the respiratory musculature. All these factors increase the work of breathing and oxygen demand. Intrapulmonary shunting develops as collapsed alveoli in regions of atelectasis are perfused. In patients with postoperative anemia or limited cardiopulmonary reserve, significant tissue hypoxia often results. As such, it is clear why patients with underlying pulmonary pathology and limited reserve are at higher risk of postoperative respiratory failure.

Cardiopulmonary bypass also leads to activation of complement, neutrophils, monocytes, macrophages, platelets, and endothelial cells [56]. A systemic inflammatory response can result from any of four forms of tissue injury: contact of blood components with artificial surfaces of the bypass circuit, ischemia-reperfusion injury, endotoxemia, or operative trauma. This systemic inflammatory response can cause various degrees of acute lung injury with the most severe form being acute respiratory distress syndrome. The majority of patients do not develop a clinically significant acute lung injury and less than 1% will develop acute respiratory distress syndrome. A recent prospective, randomized study suggested that the widened alveolar-arterial oxygen gradient associated with cardiac surgery is due to factors other than use of cardiopulmonary bypass [57].

The entity commonly called post-perfusion lung syndrome is likely a single point on the spectrum of acute lung injury. Its incidence is related to the duration of total ischemia-reperfusion time and is more common when pump time is greater than 2 hours. Clinically patients develop tachycardia, tachypnea, and progressive respiratory distress. Chest radiographs range from clear to asymmetric bilateral infiltrates. The alveolar-arterial oxygen gradient is widened and histologic specimens resemble acute respiratory distress syndrome. Proposed inciting events include pump-induced hemolysis with hemolyzed blood precipitating pulmonary vasculitis and reduction of surfactant or an immune response by the patient's leukocytes to the foreign proteins in transfused blood. Therapy is generally supportive, with early initiation of mechanical ventilation and application of PEEP. Corticosteroid therapy is reportedly beneficial.

Postpericardiotomy syndrome

This entity is characterized by the onset of fever, pleuropericarditis, and parenchymal infiltrates approximately 3 weeks after injury to the pericardium of myocardium. This injury is hypothesized to cause an immune reaction against antigenic proteins released into the circulation during surgery. Postpericardiotomy syndrome has been seen after myocardial infarction, cardiac surgery, chest trauma, pacemaker implantation, and angioplasty. For unclear reasons the postpericardiotomy syndrome is

seen more frequently after cardiac surgery than after myocardial infarction, with one study showing an incidence of 30% after surgical intervention [58].

The pathogenesis of this syndrome is unclear, but because many patients have antiheart antibodies an autoimmune etiology has been hypothesized [53]. It is possible that a concurrent viral infection may be necessary to trigger such an immune response because peak incidence correlates with the seasonal variation of viral infections [59,60].

Diagnosis is established by the clinical picture and by ruling out congestive heart failure, pulmonary embolism, and pneumonia. The most common presenting symptoms include fever, cough, pleuritic chest pain, and dyspnea. Leukocytosis and an elevated erythrocyte sedimentation rate are common findings. Evidence of pericarditis, including a friction rub and effusion, are nearly universal. The chest x-ray is almost always abnormal, with pleural effusions seen in 83% of patients [61]. Pulmonary parenchymal infiltrates are present in as many as 75% of patients, most commonly involving the left lower lobe [61].

Patients usually have good clinical responses to anti-inflammatory agents such as aspirin and indomethacin. Corticosteroids may be necessary in the more severe forms. Prompt diagnosis and therapeutic intervention are required to prevent premature graft closure [57] and to prevent iatrogenic complications from inappropriate therapy such as cardiac tamponade due to anticoagulation for presumed pulmonary embolism. Therapy with prednisone 30 mg daily for a week followed by a taper over five weeks and 600 mg aspirin four times daily reduced the graft occlusion rate from 86% to 16% [62].

Conclusions

Although pulmonary complications are common after coronary artery bypass grafting, several risk factors permit the identification of individuals with increased risk. Identifying this patient subset allows for interventions to minimize adverse outcomes. A clear understanding of the most common respiratory complications allows for earlier identification of complications, prompt intervention, and reduced adverse effect on patient outcomes.

Acknowledgment

The authors thank Margie Galkowski for secretarial assistance.

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