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The Spectrum of Pleural Effusions After Coronary Artery Bypass Grafting Surgery

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Coronary artery bypass grafting (CABG) is performed on more than 600,000 patients per year in the United States [1]. Patients commonly develop pleural effusions directly related to this operation, making this procedure one of the most common causes of a pleural effusion. The cause and management differ because of the varied pathogenesis and time course of these pleural effusions. These effusions can be best categorized by time intervals: (1) perioperative (within the first week), (2) early (within 1 month), (3) late (2–12 months), or (4) persistent (after 6 months). The pathophysiology of pleural effusion formation in the perioperative period after CABG can differ; however, these effusions usually resolve without intervention. The effusions that occur later than 1 week and within 1 month typically present with acute chest pain and fever and often require anti-inflammatory medication. The persistent effusions are the consequence of dysfunctional pleural healing, which results in a visceral pleural peel or fibrosis leading to a trapped lung, often requiring decortication.

Perioperative coronary artery bypass grafting pleural effusions

Pleural effusions have been reported to occur in the immediate postoperative period in 41% to 87% of patients [2–6]. These effusions are typically small and left-sided, but they can be bilateral. There are two distinct pleural effusions directly related to CABG in the perioperative period: effusions resulting from atelectasis from diaphragm dysfunction and hemorrhagic effusions resulting from internal mammary artery (IMA) harvesting. In addition, pleural effusions from congestive heart failure may occur after CABG. These pleural effusions are associated with different clinical characteristics, pathophysiology, pleural fluid (PF) analyses, management, and sequelae.

Pleural effusions from diaphragm dysfunction

Clinical characteristics

In the immediate postoperative period, patients often experience substantial chest pain and diaphragm dysfunction that results in a significant decrease in their forced vital capacity (FVC). With conventional chest tube placement, FVC falls by 66% during the first postoperative day and causes an average pain score of 5 of 10 during forced inspiration [7]. The decreased FVC is also likely related to decreased chest wall compliance, interstitial pulmonary edema, and poor bellows function from the median sternotomy. The drop in FVC, per se, may not produce symptoms; however, patients with decreased FVC often manifest a rapid and shallow breathing pattern with resultant atelectasis and small pleural effusions. The chest radiograph often reveals bibasilar-dependent atelectasis with blunting of the left costophrenic or bilateral costophrenic angles.

Pathophysiology

Atelectasis is extremely common after CABG and may be related to diaphragm paresis and/or paralysis. Fedullo and colleagues [8] observed left diaphragm dysfunction in 8 (16%) of 48 post-CABG patients as

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detected by comparing right and left diaphragm excursion by ultrasonography. Using the right diaphragm as a control for the bilateral effects of splinting and global reduction in FVC, the presence of left-sided phrenic nerve dysfunction was found in 16% of these patients. Direct cold cardioplegia, associated with an increased incidence of left lower lobe atelectasis [9] and effusion [10], induced phrenic nerve paresis [10]. The phrenic nerve paresis lasted from 6 to 28 days [9]. Lower lobe atelectasis from phrenic nerve paralysis has been replicated in animal models by direct application of cold to the ipsilateral phrenic nerve [11]. Intraoperative electrophysiologic phrenic nerve monitoring during cardiac surgery has confirmed left phrenic nerve paralysis [12]. All cases occurred when moderate hypothermic cardiopulmonary bypass was used as opposed to off-pump CABG [12]. The role of direct cold cardioplegia has been further clarified. Patients with aortic root cooling and cardioplegia with intracoronary cooling without direct cooling of the myocardium did not develop phrenic nerve paresis and/or paralysis in one study [13]. Two of 6 patients with topical ice cooling of the myocardium developed intraoperative left phrenic nerve paresis and/or paralysis, however, as documented by phrenic nerve monitoring [13]. In a study of 30 patients, Vargas and colleagues [6] found an 87% incidence of atelectasis by CT scan 2 days after CABG. These effusions were small and often bilateral. They also found a significant correlation between the degree of atelectasis and presence of pleural effusion between postoperative days 2 and 7 ($r = 0.53$, $P = .0025$) [6]. Left-sided atelectasis with effusion persisted more often than right-sided atelectasis with effusion at hospital day 7. Therefore, the literature supports the concept that the most immediate post-CABG effusions are caused by atelectasis related to splinting from pain, poor chest wall compliance, and diaphragm dysfunction.

Diagnosis, management, and sequelae

The pleural effusions that result from diaphragm dysfunction are often diagnosed by their radiographic appearance. The effusions are small, with associated ipsilateral atelectasis on the left; thoracentesis is rarely performed on these effusions. Management of these effusions is conservative. Avoidance of direct topical cold cardioplegia may lessen the risk of phrenic nerve paresis and/or paralysis and atelectasis. Early mobilization, incentive spirometry, and effective pain control also minimize atelectasis. An improvement in postoperative pain and FVC has been documented with the use of subxiphoid chest tubes [7]. These pleural effusions usually resolve spontaneously within 2 weeks without clinical sequelae.

Pleural effusions from internal mammary artery harvesting

Clinical characteristics

The surgical technique used can affect the development of pleural effusions after CABG in the immediate postoperative period. The use of an IMA graft [2,4,14,15] tends to result in an ipsilateral pleural effusion more often than a saphenous vein graft (87% versus 47% at postoperative day 6; $P < .05$; $n = 200$) [4]. These effusions usually present similar to the effusion from diaphragm dysfunction; the fluid appears in the immediate perioperative period, is usually small, is ipsilateral to the IMA harvesting, and may be associated with atelectasis. Large pleural effusions in the immediate postoperative period occur with an incidence of 0.5% to 8.5% [4,16,17]. Neither the operative time, number of grafts, nor type of cardioplegia (anterograde or retrograde) was found to be significantly different between patients with large effusions and those without effusions [18]. Most patients with large effusions had IMA harvesting (10.9% of 282 of patients with IMA ± saphenous venous graft (SVG) versus 4.5% of 67 patients with SVG alone, $P = .2$) [18].

Pathophysiology

The presumed cause of pleural effusions attributable to IMA harvesting is parietal pleural injury. The method of IMA harvesting affects the incidence of early post-CABG pleural effusions. When the IMA is harvested with the pleural and thoracic fascia intact, the reported incidence of postoperative pleural effusion is 5% to 11% [19–21]. When the IMA is harvested by incising the pleura and thoracic fascia, the incidence of postoperative pleural effusion increases from 20% [19] to 50% [21]. Furthermore, the chest tube drainage of patients with IMA harvesting with an open incision is significantly increased compared with the drainage of patients without an open incision [4,22] (1413 versus 1028 mL; $P < .01$) [4].

Diagnosis, management, and sequelae

These larger pleural effusions are typically hemorrhagic and inflammatory with elevated protein and lactate dehydrogenase (LDH) levels [18]. In a study of therapeutic thoracentesis for symptomatic patients after CABG, the early large effusions had a mean red blood cell (RBC) count of 706,000 cells/μL, mean white blood cell (WBC) count of 7000 cells/μL with an eosinophil predominance, and mean LDH level of