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Post-operative Pleural Effusions

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Pleural Effusions After Coronary Artery Bypass Graft Surgery

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In the United States, the annual number of coronary artery bypass graft (CABG) surgeries performed exceeds 500,000¹. Since pleural effusions complicate many of these procedures, post-CABG effusions are one of the most common types of exudative pleural effusions seen.

In the immediate postoperative period, the incidence of pleural effusions approaches 90% with the use of ultrasound². The majority of these postoperative effusions are unilateral, left-sided and small. The natural history of the post-CABG effusions is that they gradually disappear. However, in one prospective study, the prevalence of pleural effusions 30 days postoperatively was 63%³. Some post-CABG pleural effusions are large; the prevalence of pleural effusions occupying more than 25% of the hemithorax was 9.7% in one prospective study of 349 patients³.

There appears to be two different types of large post-CABG pleural effusions⁴. The first type occurs within the first 28 days following surgery and is thought to be due to the trauma of surgery itself. This effusion frequently appears bloody. Additionally, it is often eosinophilic, which is probably due to the blood in the pleural fluid. The second type reaches its maximum size more than 30 days post-CABG, but is neither bloody nor eosinophilic and has a predominance of lymphocytes⁵. This type of effusion

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is thought to have an immunological basis. Both types of post-CABG pleural effusions meet the exudative criteria.

The primary symptom with post-CABG effusions is shortness of breath. Fever and chest pain are unusual and their presence suggests the post-cardiac injury syndrome or a pleural infection. The recommended approach to a patient with a post-CABG effusion is as follows: a) if the patient has no symptoms, observation is warranted; b) if the patient has chest pain or fever, a thoracentesis should be performed to rule out pleural infection; and c) if the patient is short of breath, a therapeutic thoracentesis should be performed. The differential diagnosis of a large pleural effusion in the post-CABG patient includes post-CABG pleural effusion, congestive heart failure, pleural infection, chylothorax and pulmonary embolus. Accordingly, when a thoracentesis is performed, the following pleural fluid studies should be considered: a) if the pleural fluid is cloudy or milky, a triglyceride analysis is indicated; and if the fluid is bloody, a hematocrit is indicated; b) it should be determined whether the fluid is a transudate or an exudate, and c) a differential cell count, Gram stain and culture should be obtained to rule out pleural infection. If the shortness of breath is out of proportion to the size of the effusion or if the shortness of breath came on suddenly, the possibility of a pulmonary embolus should be evaluated.

The primary treatment for post-CABG effusions that are symptomatic is repeated thoracentesis. Most effusions will be controlled with one or two therapeutic thoracenteses, while occasionally three or four will be required. If more than four are required, consideration should be given to thoracoscopy as some patients will develop a thin membrane over their visceral pleura that prevents the underlying lung from expanding and leads to a chronic pleural effusion⁶. This membrane can be removed with thoracoscopy. There is no evidence that patients with post-CABG benefit from diuretics or anti-inflammatory agents.

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Pleural Effusions Post-orthotopic Heart Transplantation

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It has been estimated that nearly 2,300 heart transplants are performed each year in the United States¹. The prevalence of pleural effusions after coronary artery by-pass graft (CABG) surgery and other cardiac surgeries has been well established. However, the data on the prevalence of pleural effusions in the post-orthotopic heart transplant (OHT) population is limited to the results obtained from two previous studies^{2,3}. In the first study by Lenner et al², the authors retrospectively evaluated the frequency of all pulmonary complications that occurred after 159 OHT surgeries. The prevalence of pleural effusions was 6.7% (10 patients). In the second study, Misra et al³ retrospectively evaluated the prevalence of pleural effusions in 72 patients who underwent OHT. The authors reported that 61 patients (85%) developed an effusion at some time during the first year following surgery.

Misra et al have hypothesized that post-OHT effusions are due to the actual surgical procedure of entering the chest cavity and disrupting the pleural space³. On the other hand, the occurrence of pleural effusions in the post-OHT population might be related to immunosuppressive regimens. The former is more likely given the similarity in occurrence of post-OHT and post-CABG effusions⁴. Another possible explanation could be cardiac failure, which sometimes occurs in the heart transplant population. The biochemical analysis for pleural effusions was available for only four patients in the study by Misra et al³. Two of those four effusions were exudative, suggesting that at least not all of the effusions were due to congestive heart failure. In their series, none of the patients had evidence of post-cardiac injury syndrome, such as pericarditis or pneumonitis.

The only available data on the characteristics of post-OHT pleural effusions comes from the study by Misra et al³. In this study, 12 out of 72 patients (17%) had unilateral effusions (10 on the left side, 2 on the right) and 49 patients (68%) developed an effusion on each side at least once during the first 12 months after transplantation. The majority of the post-operative effusions were small. However, eight patients (13%) had 10 effusions occupying 25 to 50% of the

hemithorax, seven of which were left-sided. Four patients with fluid studies were available for review³. The fluid samples were obtained at a variety of times during the post-operative period, from 12 to 128 days post-op. Two of the effusions were exudates with LDH levels of 385 IU/L and 1,623 IU/L (upper normal limit for serum LDH, 220 IU/L). The protein level was measured in only one of these exudates and it was 3.8 g/dL.

Misra et al suggested that post-OHT effusions tend to resolve with time and no intervention is necessary³. Of the 61 patients with post-OHT pleural effusions, 46 (75%) had post-operative imaging available for review, which was obtained between days 30 and 365. Twenty-nine of these patients (63%) had a complete resolution of all effusions by the date of the last available image. The remainder of the 46 patients had small asymptomatic unilateral or bilateral pleural effusions, the exception being one patient who had bilateral effusions occupying more than 50% of the hemithorax.

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Pre- and Post-operative Pleural Effusions in Ovarian Malignancies

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Epithelial ovarian carcinoma patients with malignant pleural effusions are designated as having stage IV disease. Over the past few decades, optimal abdominal cytoreduction (debulking) at initial surgery has been established as one of the most important prognostic factors in patients with advanced epithelial ovarian carcinomas. Eitan et al, however, showed that the survival of successfully debulked patients with malignant pleural effusions was significantly shorter than that of patients with

optimally cytoreduced stage IIIC disease without effusions¹. The cause of this survival difference is currently unknown, but may be related to more aggressive tumor biology and/or undetected bulky intrathoracic disease in patients with malignant effusions.

Currently we, as previously described by others², utilize video-assisted thoracic surgery (VATS) in patients with moderate to large pleural effusions and presumed advanced ovarian cancer to assess the extent of intrathoracic disease and to identify candidates for optimal abdominal cytoreduction³. If VATS identifies unresectable intrathoracic disease, those patients are considered for neoadjuvant chemotherapy, without attempting abdominal cytoreduction, as the presence of substantial pleural tumor burden would negate the abdominal debulking procedure⁴. In patients with resectable intrathoracic disease, VATS cytoreduction may be performed, followed by the removal of as much abdominal tumor as possible. In patients with symptomatic malignant pleural effusions, talc pleurodesis at the time of the VATS procedure can also be performed to assist with pulmonary function. Currently, it is not clear if bulky intrathoracic disease is present in ovarian cancer patients with small pleural effusions and if VATS could be beneficial in these patients.

Extensive upper abdominal procedures such as diaphragm peritonectomy and diaphragm resection are being increasingly performed in an attempt to achieve optimal cytoreduction in patients with advanced ovarian cancers. Eisenhauer et al⁵ showed that more than half of the patients undergoing these specific procedures developed sympathetic pleural effusions postoperatively. Complete dissection of the hepatic ligaments and exposure of the diaphragmatic bare area is thought to be the main risk factor associated with the development of these pleural effusions postoperatively. It has been hypothesized that diaphragmatic defects developed during diaphragm peritonectomy/diaphragm resection in conjunction with ascites appear to cause fluid from the peritoneal cavity to be drawn up by negative intrathoracic pressure, forming pleural blebs that break and cause direct communication with the pleural space. We are increasingly utilizing placement of a chest tube prophylactically in patients undergoing extensive liver mobilization with exposure of the central tendon to help prevent the development of postoperative pleural effusions.

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Management of Postpneumonectomy Empyema

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The incidence of postpneumonectomy empyema (PPE) is reported to be between 2% and 16%¹. A bronchopleural fistula (BPF) is seen in 75-80% of the cases. The mortality rate of PPE with BPF is 30-40%, whereas in patients without BPF, it decreases to 5%. The treatment of PPE centers on the bronchial stump and the empty pleural space.

The key to the management of PPE, with or without a BPF, is the degree of pliability and induration of the mediastinal tissues and the extent of pleural space contamination. PPE can be classified as early (<2 weeks from pneumonectomy, EPE) or late (>3 weeks after pneumonectomy, LPE).

EPE is invariably associated with a BPF. The successful management of these patients revolves largely around the effective closure of the BPF. We favor a full thoracotomy and evacuation of the infected pleural fluid, debridement and lavage of the pleural space, as well as the definitive closure of the BPF. We have described a technique for irrigation of the EPE with excellent results².

The management of patients with EPE without a pliable mediastinum and those with a large degree of pleural contamination is similar to patients with a LPE. In these patients, basic surgical principles for wound management apply: 1) drainage, 2) prevention of repeated contamination, 3) debridement of nonviable tissue, and 4) obliteration of the dead space. In patients with LPE, these principles translate into: 1) open window thoracostomy with drainage of the pleural space, 2) closure of a BFP, 3) debridement of the pleural space, and 4) obliteration of the pleural space. The morbidity associated with the care of an

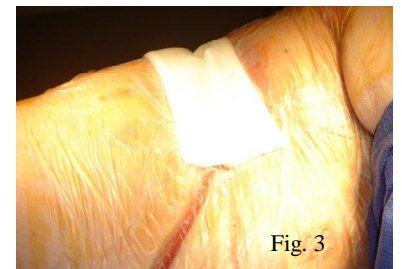
open thoracic window and prolonged hospitalization in patients with chronic PPE has led us to search for a more accelerated outpatient strategy.



We have applied the Vacuum Assisted Closure Therapy System (VAC) (Kinetic Concepts Inc, San Antonio, TX) to patients with LPE (Fig 1). Our present protocol consists of the placement of the VAC granufoam dressing soaked in 1% acetic acid into the pleural space, application of the occlusive drape over the opening (Figs 2 and 3), and institution of VAC therapy using a portable suction device set at 75 mm Hg suction. Using this portable system,



the patient can be discharged home and return for weekly debridement under anesthesia. The VAC system is replaced on a weekly basis coincident with the operative debridement. We have been able to obtain a clean and more contracted pleural space after 5-6 weeks of therapy. Based on our protocol, once the pleural space is deemed to be clean enough, the patient undergoes muscle flap obliteration of the residual pleural space with closure of the wound over suction catheters.



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CASE REPORT

Empyema Necessitans as a Late Complication from Undertreated Streptococcus Milleri Pneumonia

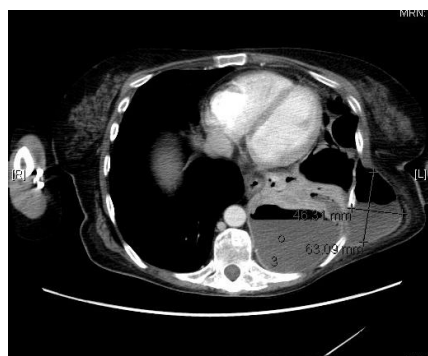
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A 62-year-old woman with a history of rheumatoid arthritis, asthma, and chronic gastroesophageal reflux presented to the emergency department with a history of several days of progressive left-sided chest discomfort ('like squeezing a sponge'), dyspnea, cough with gray sputum, chills, and night sweats. She had been treated as an outpatient 7 months earlier with 10 days of moxifloxacin for pneumonia of the right upper and middle lobes. She felt that she had never completely recovered.

Upon physical examination, she was not in respiratory distress, and blood pressure, heart rate and oxygen saturation on ambient air were normal. However, decreased breath sounds on the left base along with a palpable fluid mass over the posterolateral chest wall were noted. A chest radiograph showed multiple air-fluid levels in this area, which were thought initially to be loops of small bowel. A CT scan of the chest, abdomen and pelvis revealed a large multi-loculated left pleural effusion

in communication with a chest wall fluid collection measuring 6.3 x 4.6 cm (figure), and multifocal areas of tree-in-bud opacities throughout the right upper and middle lobes.



Routine lab tests showed prolonged PT and PTT (45.5 and 39.8 seconds respectively), INR 5.3, fibrinogen 590 mg/dL, leukocyte count $5.7 \times 10^9/L$, platelet count $573 \times 10^9/L$, and hemoglobin 7.3 g/dL. The patient underwent correction of the coagulopathy with vitamin K and subsequent drainage of the empyema. Pleural fluid cultures revealed

Streptococcus milleri, without evidence of fungal, anaerobic or acid-fast bacilli sources.

The pleural space, which is typically sterile, can become infected as a complication of pneumonia or by direct extension from infections of an adjacent organ (esophagus, mediastinum, vertebrae and sub-diaphragmatic organs). Empyema can be caused by a number of bacteria and fungi. Common aerobic pathogens include *Streptococcus*, *Staphylococcus* and *Pseudomonas aeruginosa*¹. *Bacteroides* and *Fusobacterium* species are the most common anaerobic pathogens^{1,2}. Many case reports have also mentioned *Mycobacterium tuberculosis*, *Actinomyces* and *Nocardia* species.

Empyema from any cause requires drainage of the pleural space with concurrent antibiotic therapy to ensure complete resolution and avoidance of late complications³. *Empyema necessitans* is a late complication resulting from inadequate treatment. The majority of *Streptococci* infections are caused by *S. pneumoniae* and *S. pyogenes*. Other *Streptococcal* species account for <10% of empyemas⁴. *S. milleri* is a member of the *viridans* streptococci family, which are colonizers of the gastrointestinal tract, oropharynx, auditory canals, and the genitourinary tract. Infections are usually due to aspiration and are most common in patients with alcohol consumption, esophageal disease or mental retardation, that is, those in whom aspiration is common⁴.

Treatment of *S. milleri* empyema involves complete pleural drainage^{4,5}. If there has been extra-thoracic extension, subcutaneous and rib resection may be required. Penicillin-based antibiotics are the treatment of choice. However, there are case reports of penicillin-resistant isolates⁴.

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If you have any comment on the Newsletter or interesting cases of pleural disease, contact:
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