Pulmonary actinomycosis usually occurs as a result of the aspiration of oropharyngeal material in the setting of poor dental hygiene or oral trauma. When pathogenic, it is invasive, often mimicking a malignancy in presentation and macroscopic appearance. Endobronchial disease due to this pathogen rarely is reported. A Ariel et al recently described five patients presenting with endobronchial actinomycosis presenting in a subacute fashion, similar to the presentation in our patient. Endobronchial infection is thought to be due to implantation of infected aspirated material, lymphohematogenous spread to the peribronchial region, or endobronchial implantation of infected secretions from draining cavitory lesions.

Although the diagnosis of actinomycosis was presumptive since the cultures of all specimens were negative, this was not an unexpected finding in light of antibiotic therapy administered prior to bronchoscopy. In addition, the finding of a spurt culture positive for H influenzae is common in the setting of Actinomycosis pulmonary infection. Coexisting organisms such as fusobacteria, streptococci and Eikenella may be cultured as well.

Even among immunocompromised hosts, such as patients on chronic steroid therapy or cancer chemotherapy, actinomycosis has not been shown to have an increased prevalence of infection. Actinomycosis is a rare pathogen in the HIV-infected population. This is most likely due to the partial susceptibility of the organism to antibiotics commonly used to treat persons with AIDS such as trimethoprim-sulfamethoxazole, isoniazid, rifampin and the cephalosporins. Why this particular patient developed actinomycosis infection with endobronchial disease is unclear. Although his oral hygiene appeared to be maintained, there may have been unsuspected aspiration of oropharyngeal secretions which subsequently led to endobronchial disease. The possibility that the endobronchial tissues may have been secondarily involved from a more distal infection, as seen in tuberculosis, also must be entertained. The subacute presentation, the initial lack of response to orally administered antibiotics and the development of a new infiltrate while receiving a broad-spectrum intravenously administered antibiotic regimen is consistent with this infection.

Actinomycosis also must be considered when obstructing lesions are noted at the time of bronchoscopy in a patient with AIDS and a suspected pulmonary infection.

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**Upper Extremity Deep Venous Thrombosis**

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The significance of upper extremity deep venous thrombosis (DVT) has been minimized in comparison to iliofemoral thrombosis, likely due to the erroneous belief that subsequent pulmonary thromboembolism is rare. The possibility of pulmonary thromboembolism originating in the upper extremity veins must now be seriously considered with catheters and medical instrumentation being performed more commonly in accessing the central venous system. It has been incorrectly assumed that the risk of pulmonary embolism was low due to the abundant collateral flow, and thus lack of stasis around an upper extremity even with venous occlusion. However, several studies, including a recent prospective trial, concluded that pulmonary embolism is not a rare complication in upper extremity DVT. Significantly, when comparing all sources of secondary upper extremity DVT, catheter-related upper extremity DVT is at greatest risk of subsequent pulmonary thromboembolism. We present an illustrative case documenting extensive pulmonary embolization that occurred following insertion of a central venous catheter and subsequent thrombosis of the right subclavian and innominate veins. With absolute contraindications to thrombolytic and anticoagulation therapy, prevention of further embolization was achieved by percutaneous insertion of a superior vena cava filter. (Chest 1993; 103:1887-90)

DVT = deep venous thrombosis; SVC = superior vena cava

Complications of subclavian vein thrombosis may be categorized into three main subgroups: pulmonary embolism, the postthrombotic syndrome, and venous gangrene. More common in the medical literature are discussions related to the postthrombotic syndrome and the extremely rare occurrence of venous gangrene. Since the significance of subclavian vein thrombosis has been minimized in comparison to iliofemoral thrombosis, this article will discuss the significance of deep venous thrombosis (DVT) of the upper extremity and its relevance to subsequent pulmonary embolism.

**CASE REPORT**

A 67-year-old white man was admitted to the hospital for further investigations of progressively worsening right lower limb claudication. The patient had bilateral claudication for approximately six years. The patient's history included a left thoracotomy for a bronchogenic adenocarcinoma four years previous to his current hospital admission. He continued to smoke against medical advice.

The patient had multiple attempted vascular reconstructions, each one unsuccessful ultimately due to thrombosis. The operations that failed were an in situ composite femoropopliteal bypass graft.

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a revision of the previous graft, and finally a right axillofemoral and femoropopliteal graft. The patient eventually underwent a right above-knee amputation. The patient's postoperative course was complicated by the development of gastrointestinal bleeding, congestive heart failure, and pulmonary sepsis. During his intensive care unit (ICU) admission, continued and progressive respiratory failure prompted a ventilation-perfusion scan to rule out pulmonary thromboembolism. A venogram and a possible pulmonary angiogram were requested.

The ventilation-perfusion scan showed bilateral mismatch defects consistent with pulmonary emboli. A left leg venogram showed no evidence of DVT. Because the patient has a right above-knee amputation, a trans catheter venogram of the right femoral and iliac veins as well as inferior vena cava was obtained via a left femoral approach; all were free from clot. The femoral catheter was then advanced through the right atrium and a superior vena cavaogram was obtained (Fig 1). This revealed a large thrombus in the right innominate vein, the most likely source of the pulmonary emboli. The superior vena cava (SVC) measured 24 mm after correction for magnification. Pulmonary angiography was performed, confirming the presence of bilateral pulmonary emboli. Furthermore, there was a suggestion of extrinsic compression of the pulmonary vasculature suggestive of a recurrent bronchogenic carcinoma.

Because of a history of recent gastrointestinal bleeding contraindicating anticoagulant therapy, placement of an SVC filter appeared to be the best option for protecting the lungs from further emboli originating from the thrombus in the right innominate vein.

A filter introducer (Vena Tech) was inserted through the existing left femoral vein access and the filter was released in the proximal third of the SVC superior to the expected course of the azygos vein (Fig 2). A jugular vein introduction set was used to place the filter in the correct orientation (apex caudal), i.e., opposite to the filter's orientation when used in the inferior vena cava.

The patient's condition improved, and he was later transferred from the ICU to the surgical ward for convalescence. Unfortunately, his condition deteriorated and he eventually died of respiratory failure secondary to severe pulmonary sepsis weeks following

**Pathologic Findings**

A full autopsy demonstrated that the immediate cause of death was extensive bilateral pneumonitis. Further significant findings included the following: (1) recurrent adenocarcinoma of the left lung with left pleural metastases and mediastinal lymph node involvement; (2) a thrombotic state secondary to the pulmonary malignancy with recent massive pulmonary thromboemboli, mainly in the right lung, and thrombosis of the filter and occlusion of the SVC (Fig 3) with resulting marked edema of the right arm; and (3) no DVT found in the left leg or the right lower extremity stump.

**Discussion**

**Recognition**

The possibility of pulmonary thromboembolism originat-
ing from the upper extremity veins must now be seriously considered with catheters and pacemaker hardware commonly being utilized in accessing the central venous system. It is now well established that pulmonary embolism frequently presents in an asymptomatic fashion.4,4 It is therefore not surprising that 30 percent of those with angiographically proven pulmonary emboli have a normal lower extremity venogram.5 Possible explanations for the latter finding could include either (1) embolization of all thrombi to the pulmonary circulation or (2) emboli derived from a source other than the deep veins of the legs, ie, the deep veins of the upper extremity.5

Source

Our case provides direct evidence that DVT of the upper extremity may be the source of lethal pulmonary thromboembolism. Thrombosis of the subclavian veins may be either primary or secondary. The condition is rare, occurring in less than 2 percent of all cases of venous occlusion prior to 1967.6-7 Undoubtedly, the incidence has risen with more frequent use of these veins for treatment of a variety of medical problems. Primary thrombosis generally occurs following exertion, as exemplified by the high occurrence of cases in the dominant upper extremity. Approximately 25 percent of cases of primary thrombosis arise spontaneously without a recognized predisposing event.8,9,10 Secondary thrombosis, which occurs more often, can occur due to local sources of inflammation and/or compression, ie, sclerosing intravenous solutions, foreign bodies such as catheters, and anatomic variations at the thoracic outlet. Clots have been found to form at the site of venipuncture and extend along the venous catheter. Swinton et al11 suggested that pulmonary embolism was more frequent and severe in patients with secondary thrombosis of the upper extremities. Hypercoagulability states, a manifestation of serious preexisting systemic disease, such as neoplastic disorders, are well known to be associated with secondary thrombosis and a markedly reduced survival.12,13

Treatment

Unlike the rarer sequelae of upper extremity DVT, no treatment protocols have yet been advocated for the prevention of recurrent pulmonary thromboembolism. Early venous thrombectomy in the acute situation has been advocated in an attempt to prevent residual disability despite the initial effectiveness of anticoagulants.10,11,12,13 Anticoagulant therapy continues to be recommended solely or combined with surgery on the theoretical grounds of preserving venous collateral flow.11,12,13,14 thus possibly avoiding long-term disability. Fibrinolysis and anticoagulation therapies that seem most promising are likely to continue to serve as the primary noninvasive modalities prior to surgical correction of any existing abnormalities.15-17

Blinded by Virchow's identification that the lower limbs were the major anatomic source of pulmonary thromboembolism, technical advances and medical therapies for the prevention of further pulmonary thromboembolism have long centered around the inferior vena cava as the major avenue of subsequent thromboembolism.18 When contemplating the use of an inferior vena caval filter, the surgeon/invasive radiologist must be aware of not only the anomalous venous return, megacava, but also the possibility of pulmonary thromboembolism origination in the upper extremity veins. Insertion of an inferior vena caval filter in the latter circumstance would be disastrous!

It is hoped that upper extremity venography, and perhaps in the near future duplex scanning19 and light reflection rheography,20 will be contemplated whenever pulmonary thromboembolism occurs in the presence of upper extremity venous access. Superior vena caval placement of venous filters in humans has rarely been performed but may provide further protection of recurrent pulmonary embolism when other modes of therapy are contraindicated.20

Technical Information With Regard to SVC Filter Insertion

Prior to the insertion of the filter, the distance between the left groin and the SVC should be measured, using a guide wire clamping technique. In our case, this indicated that the introducer was of adequate length. It should be noted that in our patient, who was of average height, the total length of the introducer had to be inserted in order to deliver the filter to the desired location. We therefore suggest that for patients greater than 170 cm in height, longer introducers be used. A potential problem with these devices is that they may preclude the use of thrombolytic therapy because of fear of excessive retroperitoneal mediastinal bleeding or from the insertion site should further massive pulmonary thromboembolism occur. The recent development of temporary and vena caval filters may negate the latter fears.

Conclusions

A recent prospective trial concluded that pulmonary embolism is not a rare complication in upper extremity DVT, and catheter-related DVT seems to be at the greatest risk.4 Upper extremity DVT must now be taken seriously as a harbinger of possible pulmonary thromboembolism. As illustrated in our case, a combination of risk factors, ie, hypercoagulability secondary to recurrent pulmonary carcinoma, and the cannulation of the central veins made embolization from an upper extremity DVT possible. With the systemic and pulmonary venous systems being accessed increasingly for a multitude of invasive diagnostic and therapeutic procedures, upper extremity venography and possibly newer less invasive modalities should be contemplated whenever pulmonary thromboembolism occurs in the presence of upper extremity venous access. The insertion of an SVC filter may provide further protection against recurrent pulmonary embolism when other modes of therapy are contraindicated.

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Diagnosis of Pneumocystis carinii Infection in HIV-Seropositive Patients by Identification of P carinii in Pleural Fluid*


Pneumocystis carinii pneumonia (PCP) is the most common pulmonary complication of AIDS and is typically diagnosed by the identification of P carinii organisms in sputum, bronchoalveolar lavage fluid, or tissue obtained with transbronchial biopsy. We describe two HIV-seropositive patients with pleural effusions in whom the diagnosis of P carinii infection was made by examination of pleural fluid. Pleural effusions associated with PCP are very unusual but can provide a source of diagnostic material particularly in those HIV patients who have development of a spontaneous pneumothorax and require chest tube insertion.

(Chest 1993; 103:1890-91)

AFB = acid-fast bacteria; CMV = cytomegalovirus; HIV = human immunodeficiency virus; LDH = lactate dehydrogenase; MAC = Mycobacterium avium complex; PCP = Pneumocystis carinii pneumonia

Pneumocystis carinii pneumonia (PCP) occurs in 80 percent of all patients with acquired immunodeficiency syndrome (AIDS) at some time during the course of their illness. Although spontaneous pneumothorax is a well-described complication of PCP, actual involvement of the pleural space causing inflammation and a significant effusion is very uncommon. In fact, it has been stated that if a pleural effusion is present, it is probably indicative of a disorder other than PCP. In this article, we describe two patients in whom the diagnosis of P carinii infection was made by evaluation of pleural fluid. To our knowledge, identification of P carinii in pleural fluid has not been described previously.

CASE REPORTS

Case 1

A 23-year-old man with hemophilia A was examined because of 2 weeks of a nonproductive cough, dyspnea on exertion, fevers, and

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1890

Diagnosis of P carinii Infection by Identification of P carinii in Pleural Fluid (Schaumberg et al)