Hemoptysis Complicating AICD Patch Placement Controlled by Temporary Selective Bronchial Balloon Occlusion*

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This is the first reported case of an automatic implantable cardioverter-defibrillator patch placement complicated by hemoptysis. It is also notable because the patient's massive hemoptysis was managed by a balloon occlusion catheter inserted into the segmental bronchus through the endotracheal tube with fluoroscopic guidance.

(Chest 1991; 99:1301-03)

AICD = automatic implantable cardioverter-defibrillator

Pulmonary complications of automatic implantable cardioverter defibrillator (AICD) implantation have been described and include pneumothorax, pleural effusion, pneumonic infiltrate and atelectasis.1,4 Patch implantation has not been previously recognized as a source of massive hemoptysis. This report describes the findings and management of a patient who presented with life-threatening hemoptysis as a result of erosion of the patch into adjacent lung. A method for segmental bronchial occlusion via an indwelling endotracheal catheter was used in this patient and is described.

CASE REPORT

The patient was a 58-year-old white man with hemoptysis, who was transferred from another institution for diagnostic angiography. His pertinent past medical history began 11 months prior with resuscitation from sudden cardiac ventricular fibrillation. Severe triple vessel coronary artery disease was diagnosed by coronary angiography. A coronary artery bypass graft operation with five vessels was performed. Two epicardial AICD leads were placed, also without apparent complication (Fig 1). However, the subsequent electrophysiologic studies were unable to induce ventricular tachycardia. This implied that the myocardium had been adequately revascularized and there was no remaining electrically unstable tissue.

Therefore, the AICD was never implanted. After discharge, the patient's course was remarkable only for a small amount of drainage from the wound tract. Oral antibiotics were prescribed for a stitch abscess, but a small amount of drainage persisted. About five months later, he was hospitalized because of mild hemoptysis. His work-up included two bronchoscopies with negative findings, and neither carcinoma nor infection was identified as the cause. He was discharged with the diagnosis of idiopathic hemoptysis. The patient was readmitted eight days prior to transfer to LGH from another hospital, again with hemoptysis. Bronchoscopy showed bleeding into the middle lobe of the right lung. Specimens sent for cytology, washings and brushings, as well as a bleeding scan were all negative. Chest x-ray film showed a right middle lobe infiltrate (Fig 2). He was transferred for angiography, reportedly in stable condition. When he was placed supine for angiography, he began having massive hemoptysis and became hypotensive. Cardiopulmonary resuscitation was begun. Support measures included endotracheal intubation.

After normal angiographic studies of the intercostal and internal mammary arteries (Fig 2), an enlarged intercostal artery with an unusual branching pattern was identified. The patient's massive

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Figure 1. Patient S/P CAGB (coronary markers) and defibrillator patch placement (straight arrows) with epicardial sensors (curved arrow).
Hemoptysis persisted. The radiologist embolized this potential feeding artery with gel foam pledgets. Bronchial arteries were never identified angiographically. Hemoptysis persisted. In an effort to gain control of the hemoptysis without resorting to selectively intubating the contralateral lung, it was elected to attempt selective bronchus occlusion. A preshaped 5 Fr balloon occlusion catheter was placed through the T-piece of the endotracheal tube into the bronchus of the right middle lobe under fluoroscopic guidance, as confirmed by x-ray examination, and inflated (Fig 3). Hemoptysis abruptly ceased. He was monitored overnight in the ICU. He required blood transfusions. At thoracotomy the following day, the right AICD patch had eroded through the pericardium and into the middle lobe of the right lung. This lobe was resected. The right patch and accessible portions of the left patch were removed. A follow-up bronchoscopy showed no additional bleeding. His remaining course was remarkable only for an aspiration pneumonia, which responded to antibiotics.

**FIGURE 3.** Occlusion balloon in right middle lobe bronchus (arrows), lateral projection.

**DISCUSSION**

The AICD is being used with increasing frequency to treat recurrent ventricular fibrillation. Much research supports its efficacy. It is conceptually simple. Two patches are placed on the heart’s epicardial surface during open heart surgery. The patches are connected to an AICD. When it senses a life-threatening rhythm such as ventricular tachycardia or ventricular fibrillation, it delivers a cardioverting shock. Currently, research is focused on the mechanical problems of deciding which rhythms deliver a shock, when to start and stop defibrillating and improving the battery life.

After surgery, particularly where prosthetic material is used or a wound infection develops, it is well known that a small percentage of cases are complicated by the development of a fistula. For example, with abdominal aortic aneurysm resection and graft replacement, 1 percent of cases result in an aortoenteric fistula. These vascular fistulas can have life-threatening results. There are significant morbidity and mortality associated with AICD implantation. The reported inhospital complication rates, including death, range from 14 to 65 percent with infections occurring between 2 and 15 percent of the time. The AICD pulmonary complications include the following: pneumothorax, pherusal effusion, infiltrate, and atelectasis. Our reviews of the literature showed no previous reports of hemoptysis complicating AICD patch implantation. Causes of massive hemoptysis include the following: active pulmonary tuberculosis, bronchiectasis, chronic necrotizing pneumonia, lung abscess, carcinomas, fungal infections, bleeding diathesis, and iatrogenic (most commonly from Swan-Ganz catheters).

The immediate goals of management are to prevent asphyxiation, control the bleeding, and treat the underlying cause. The first step should be to review the history and physical exam, appropriate laboratory studies and chest x-ray films. Other facts can aid in the decision-making process. These include knowing the etiology; baseline pulmonary function tests to see if there would be adequate pulmonary reserve; after resection, overall clinical status, with consideration of other medical and surgical problems; recurrent bleeding; ability to localize the source; and failure to achieve the above goals using the temporizing medical measures below. When volume of blood lost is less than 500 ml, usually medical therapy is initiated. This includes bronchial lavage with cold saline solution, bronchial artery embolization and selective mainstream bronchus intubation, using a Fogarty catheter and placing the patient in the lateral decubitus position so the side with bleeding is dependent. When this volume is greater than 500 ml of blood, there is a controversy between the medical and surgical literature how to best manage the patient. Several studies conclude that they can be managed without surgery. The surgical literature recommends a pneumonectomy with use of the same temporizing measures, then surgery if needed. If the site and cause of bleeding remain unknown, bronchoscopy should be performed. If active bleeding prevents its use, then an angiogram would be useful. There are few very randomized studies using operable patients. Thus, as always, an individualized decision must be made. With massive hemoptysis, the mortality for pulmonary resection varies from 10 to 17 percent, with medical treatment it is 50 percent.
With the increasing use of AICDs, an increasing number of complications will be seen. In these patients, the etiology of hemoptysis should be aggressively pursued. In this case, the standard angigraphic approach did not identify the bleeding artery. If a bleeding vessel cannot be identified or embolized, alternative measures must be considered. Life-threatening hemoptysis can be temporarily managed by unilateral, or, as in this case, segmental bronchial occlusion via endotracheal intubation using a balloon catheter to occlude the bronchus of the hemorrhaging lobe.

The technique of segmental bronchial occlusion limits the lung parenchyma lost to a lobe. This should be valuable in all patients, particularly those with a compromised pulmonary reserve. Furthermore, the remaining lung can be efficiently ventilated. As in this case, fluoroscopic guidance and familiarity with guidewire catheter technique would be essential. This technique should reduce the complications and mortality associated with massive hemoptysis when the hemorrhaging lobe can be isolated.

**REFERENCES**

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**Pulmonary Hypertension Associated with Long-standing Thrombocytosis**

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A case of thromboembolic pulmonary hypertension associated with long-standing thrombocytosis is presented. In this patient we found a significant local pulmonary platelet activation and thrombin generation as indicated by the existence of a transpulmonary gradient for thrombocytopenia Aβ, beta thromboglobulin and fibrinopeptide A. Prolonged hepatic and acetylsalicylic acid treatment resulted in improvement of clinical and hemodynamic conditions. These findings support the usefulness of anticoagulating and antiaggregating therapy in selected cases of pulmonary hypertension. (Chest 1991; 99:1303-05)

About half of all the cases of primary (unexplained) pulmonary hypertension are of the thromboembolic type.1-4 Pulmonary endothelial damage and local platelet activation have been hypothesized to play a significant role in the pathogenesis of this type of PPH.5-9 Moreover, abnormalities in platelet function and an increased thrombin generation have been demonstrated in patients suffering from primary pulmonary hypertension.10,11

We report a case of unexplained pulmonary hypertension in a young man with moderate thrombocytosis, secondary to splenectomy performed in infancy for minor thalassemia. This case underlines the possible role of chronic pulmonary endothelial damage and local platelet activation in the pathogenesis of the thromboembolic type of PPH and supports the usefulness of antithrombotic treatment in selected cases of pulmonary hypertension.

**CASE REPORT**

A 29-year-old man suffering from minor thalassemia underwent splenectomy at the age of nine years. Afterwards, repeated hematologic controls showed a mild thrombocytosis (platelet count between 400,000 and 700,000/mm mm) and leukocytosis (white blood cell count between 17,000 and 25,000/mm). However, after splenectomy the patient did not receive any treatment and was asymptomatic until the age of 25 years when he began to experience mild dyspnea on exertion. Cessation of smoking did not result in any improvement. In the following months, fatigue and dyspnea progressively worsened and the patient was compelled to limit his physical activity. Moreover, the patient noticed an increase in weight (about 5 to 6 kg) and the occurrence of dependent edema.

For these reasons, in May 1984 he came to our clinic for observation. On admission to the hospital, the patient had dyspnea with minimal effort. Physical examination showed cyanosis of the lips, distended jugular veins, hepatomegaly and dependent edema.

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