**DISCUSSION**

Bronchiolo-alveolar cell carcinoma represents between 1 and 9 percent of all lung cancers. It is less related to smoking than are other types of lung carcinoma, and is considered a distinct clinical entity. In spite of the presumed exfoliative nature of malignant cells, yield from cytologic diagnosis has been disappointingly low. As in this case, operative procedures have often been required for diagnosis. Surgical resection offers the best chance for long-term survival.

Bronchoalveolar lavage uses larger volumes of isotonic saline solution in more distal airways than a conventional bronchial washing. Since this type of carcinoma arises in bronchiolo or alveoli, it might be expected that a lavage would be more successful in recovering diagnostic cells, as we found in this case. In addition, bronchoalveolar lavage may be more useful than conventional bronchial washings in other types of peripheral lung cancers.

Physicians performing bronchoalveolar lavage should obtain cytologic evaluation of the specimen for neoplastic cells that may not be present in other types of bronchoscopic specimens.

**REFERENCES**


**Arteriosclerotic Heart Disease Following Correction of Tetralogy of Fallot**

**Gust H. Bardy, M.D.; and Robert H. Peter, M.D.**

An increasing number of people who have undergone surgical repair of tetralogy of Fallot are living long lives. Several late sequelae of corrected tetralogy of Fallot have been found, including residual ventricular septal defect, restenosis of the pulmonary outflow tract, ventricular tachycardia, and right ventricular failure from pulmonary insufficiency. A long-term survivor of corrected tetralogy of Fallot is reported with acquired coronary artery disease unrelated to the congenital anomaly or its correction as an additional cause of late morbidity in this select but growing population of patients.

Morbidity and mortality among patients with tetralogy of Fallot have diminished since systemic to pulmonic vasculature shunting was introduced by Blalock and Taussig in 1944. Further improvement in longevity followed the introduction of a corrective procedure by Scott et al in 1964. Because of these advances in surgical therapy, a large population of patients with tetralogy of Fallot has reached adulthood and some have reached middle age. Several investigators have reviewed the long-term sequelae of surgical correction of this anomaly and have recorded some late complications. These include the development of ventricular tachycardia at the surgical scar site, recurrence of right ventricular outflow tract obstruction, recurrence of the ventricular septal defect at the interface of the patch graft with the septum, and right ventricular dilatation secondary to chronic pulmonary insufficiency. All of these problems can be presumed to be directly or indirectly related to the congenital anomaly or its surgical therapy. To this list of problems in long-term survivors of surgically corrected tetralogy of Fallot, we add the first case of acquired coronary artery disease.

**CASE HISTORY**

A 44-year-old white man had been born cyanotic. Tetralogy of Fallot was diagnosed, and the patient was followed until 1948 when he underwent a Blalock-Taussig shunt for palliation. He responded well and developed normally with only mild restriction of activities until 1964 when he developed progressive cyanosis and fatigue due to increasingly severe pulmonic stenosis. Complete surgical correction of his congenital anomaly was then undertaken and included repair of a ventricular septal defect, excision of a stenotic pulmonic valve, partial patch reconstruction of the pulmonary artery and right ventricular outflow tract, and closure of the Blalock-Taussig shunt. He had an excellent result following surgery with complete resolution of symptoms. Subsequently, he was asymptomatic and worked as a farmer from 1964 to 1978. In 1978, without warning, he developed an inferior myocardial infarction. He recovered without incident and was again asymptomatic until September 1981 when he developed unstable angina culminating in an anterior myocardial infarction.

*From the Cardiovascular Laboratory, Department of Medicine, Duke University Medical Center, Durham, NC.
Reprint requests: Dr. Bardy, Box 31221 Duke University Medical Center, Durham 27710*
infarction. Risk factors for coronary artery disease were a 45 pack year history of smoking and hypertension. Following his second myocardial infarction, he continued to have intermittent episodes of rest angina associated with a stage 2 positive exercise tolerance test. He underwent cardiac catheterization and was found to have significant three-vessel coronary heart disease with total occlusion of a dominant right coronary artery, subtotal occlusion of the left anterior descending coronary artery, and subtotal occlusion of the left circumflex coronary artery. Distal coronary vessels were diffusely diseased. In addition, he had a persistent left-to-right shunt from a small ventricular septal defect, moderate right ventricular enlargement, mild right ventricular outflow tract obstruction, and marked pulmonary insufficiency. Because of the relatively high risk of reoperation on this patient with less than optimal coronary anatomy and previous thoracotomy, coronary artery bypass grafting and reconstructive surgery were not done. Medical therapy was improved and the patient has been asymptomatic, but at a reduced level of activity, after a five-month observation period.

Discussion

None of the major series that followed patients with corrected tetralogy of Fallot14 have addressed the problem of coronary artery disease in these patients. However, many other complications have been observed in long-term survivors of corrected tetralogy of Fallot. Fuster et al15 reported 396 hospital survivors of surgically repaired tetralogy of Fallot. These patients were followed for 12 to 22 years. Eighty-seven percent had excellent late clinical results. Twenty-seven of these patients, however, died from late complications of their congenital anomaly or from its surgical correction. The most frequent problem was sudden death, presumably from ventricular arrhythmias that originated from surgical scars along the right ventricular outflow tract. Other causes of death included false aneurysms at the site of outflow tract reconstruction, progressive obstruction to right ventricular outflow, severe pulmonary incompetence, bacterial endocarditis, and residual ventricular septal defect. Katz et al16 reported long-term findings in 414 patients who underwent repair of tetralogy of Fallot between 1967 and 1977. There were only nine late deaths, six of which were directly related to the malformation or to its treatment. Eight patients required reoperation and ten patients had symptoms secondary to dysrhythmias. Eight had congestive heart failure that required treatment.

Despite the large number of patients followed for as long as 22 years after repair of tetralogy of Fallot, no study has found coronary artery disease in these patients. Although this is the first reported case of arteriosclerotic heart disease in a long-term survivor of corrected tetralogy of Fallot, we expect more of these patients to become apparent in ensuing years. Undoubtedly, as more and more of these individuals approach the fifth decade and beyond, we will see increasing numbers with arteriosclerotic heart disease. An etiologic relationship is not implied between tetralogy of Fallot and arteriosclerotic heart disease, but rather that survivors of tetralogy of Fallot will probably have the same likelihood of developing arteriosclerotic coronary disease as the general population. Because arteriosclerotic heart disease is common, growing numbers of survivors of corrected tetralogy of Fallot may develop it over the years. As observed in this patient, management decisions can be considerably more complex when the hemodynamic, anatomic, and surgical factors are taken into account.

References

1. Blalock A, Taussig HB. The surgical treatment of malformation of the heart in which there is pulmonary stenosis or pulmonary atresia. JAMA 1945; 125:189-202

Pulmonary Infiltrates with Eosinophilia due to Naproxen*

Daniel A. Nader, LCDB, MC, USNR, F.C.C.P.; and Richard F. Schillaci, CAPT, MC, USN, F.C.C.P.

Various drugs have been implicated in the development of the syndrome of pulmonary infiltrates with eosinophilia (PIE). In this study of a man who was being treated with naproxen, the findings are presented of the first such case of pulmonary hypersensitivity to naproxen as noted in our review of the literature.

An increasing number of drugs has been implicated in the etiology of transient eosinophilic pneumonia5 characterized by a benign clinical course, the development of pulmonary infiltrates, and blood eosinophilia. Naproxen is a widely used nonsteroidal anti-inflammatory drug which may be added to the list of pharmacologic agents which induce lung disease. A case of acute pulmonary hypersensitivity with eosinophilia, believed to have been caused by naproxen, is described.

Case Report

A 72-year-old man was referred to the pulmonary service for evaluation with a three-day history of fever (38.9°C), nonproductive cough, wheezing, and abnormal findings on chest roentgenogram. Additional history revealed that the patient had been taking ibuprofen during the previous six months for osteoarthritis. Two weeks prior to the onset of respiratory symptoms, ibuprofen had been discontinued, and naproxen was prescribed because of progressive osteoarthritisic pain.

Physical examination revealed the following: temperature,

*From the Pulmonary Division, Department of Internal Medicine; and the Clinical Investigation Center, Naval Regional Medical Center, San Diego.

The opinions or assertions expressed in this paper are those of the authors and are not to be construed as official or as necessarily reflecting the views of the Department of the Navy or the naval service at large.

Reprint requests: Dr. Schillaci, Clinical Investigation, Naval Hospital, San Diego 92134