same socioeconomic background as were those patients in the Child Chest Clinic.

All the smoking patients from the Child Chest Clinic smoked less than one pack of cigarettes each day. Sixty of the 70 control patients also smoked less than one pack, and in two control subjects the amount of smoking was not recorded.

Age

The teenagers questioned were all between 13 and 19 years of age (Table 1). As might be expected, the percentage of smokers increased with age. In the 13 and 14 year age group, only 26 percent admitted smoking, while in those 17 to 19 years of age, 65 percent were smokers. Among the control group, too, the percentage of smokers increased with age.

Race and Sex

Since blacks constituted the preponderance (275) of those questioned, no conclusions as to racial incidence could be determined. Differences by sex were likewise inconclusive.

RESULTS AND COMMENT

In the Child Chest Clinic of the Medical College of Virginia Hospitals, 300 teenagers (13 through 19 years of age) were questioned as to their smoking habits, and 127 (42 percent) admitted the smoking habit. Among a control group chosen as a random sample from three local high schools and one junior high school in which students are, in general, of a similar socioeconomic background, 32 percent were smokers.

Although the dangers of smoking are well recognized, teenagers and college students have accepted the smoking habit; the results of the present study emphasize this fact. However, the large percentage of smokers in this particular study group (no one was a "heavy smoker," since daily consumption was less than one pack in all cases) was unexpected. It should be emphasized that all of the children in this study have been observed in the Medical College of Virginia Child Chest Clinic for more than ten years because of infection with tuberculosis.

There is no evidence that smoking is more likely to be harmful to teenagers who have healed primary tuberculosis than to those in the general population. However, the adolescents themselves were certainly not aware of this fact and smoking is discouraged by the Chest Clinic. It would appear that knowledge of the existence of any earlier pulmonary infection, in this case tuberculosis, and the need for an annual follow-up examination should be a deterrent to the acquisition of the smoking habit. This is apparently not the case.

A recent report by the Committee on Environmental Hazards of the American Academy of Pediatrics1 has listed aspects of the smoking problem which relate directly to children and teenagers.

The first of these is the short-term effect of smoking by teenagers and children, and in this regard it has been adequately shown that children who smoke have more respiratory symptoms, cough, phlegm, breathlessness, wheezing and colds than do nonsmokers.8,9 The second aspect is the effect of tobacco smoke on nonsmokers. Cigarette smoke, inhaled from either end of the cigarette, suppresses pulmonary defense mechanisms in vitro, and mucociliary activity and alveolar macrophage activity in vitro. Therefore, the pulmonary defense mechanisms of children are impaired from breathing smoke from cigarettes.9 Further, Cameron's studies10,11 indicate the children from homes where the parents smoked had a higher incidence of respiratory disease than did the children of nonsmokers.

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REFERENCES

10. Cameron P: The presence of pets and smoking as correlates of perceived disease. J Allergy 40:12, 1967

Aortopulmonary Septal Defect Due to Staphylococcal Endocarditis

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The first known case of aortopulmonary septal defect due to infective endocarditis is reported. A 21-year-old man with aortic insufficiency due to active staphylococcal endocarditis required aortic valve replacement. A week later a faint continuous murmur was first heard, which became very loud the next day. An aortopulmonary septal defect was confirmed by cardiac catheterization. Fifty-six hours after the continuous murmur was first heard, he developed florid pulmonary edema and died. Aortopulmonary septal defect was confirmed by staphylococcal endocarditis was confirmed at postmortem examination. This case illustrates the malignant nature of an aortopulmonary septal defect due to staphylococcal endocarditis.

Destruction of tissue sufficient to allow intracardiac shunting of blood is an unusual complication of infective endocarditis. Most frequently, this occurs when an aortic sinus aneurysm ruptures through the septal wall of the right atrium or the posterior wall of the right ventricular outflow tract. Rarely, peripheral arteriovenous shunting may occur in the setting of infective endocarditis.1

**CASE REPORT**

One week after the development of facial acne, a 21-year-old man developed shaking chills, fever, myalgia, pleuritic chest pain, nausea and vomiting. There was no history of intravenously administered drug use, heart murmurs, or heart disease.

Physical findings on admission included jaundice, fever, tachycardia, widened pulse pressure with bounding peripheral pulses, abnormal paradoxic pulse, multiple cutaneous, conjunctival and palatal petechiae, large tender cutaneous ulcerations, diminished left precordial activity, a fourth heart sound, a three-component pericardial friction rub, and a grade 2/6 blowing diastolic murmur along the left sternal border.

The electrocardiogram demonstrated ST-T abnormalities consistent with pericarditis, and the chest roentgenogram showed a significantly enlarged cardiac silhouette.

Cardiac catheterization demonstrated a large pericardial effusion and 4+ aortic insufficiency. After multiple blood cultures were obtained, the patient was given oxacillin, 2 gm every four hours, and gentamicin, 80 mg every 12 hours intravenously, on an empiric basis. Pericardiocentesis yielded 25 ml of purulent fluid containing massive numbers of Gram-positive cocci. After blood and pericardiocentesis fluid grew coagulase-positive *Staphylococcus aureus*, the gentamicin therapy was stopped. The organism was sensitive to oxacillin at a level of less than 0.3 µg/ml. During the course of antibiotic therapy, serum levels of oxacillin ranged between 85 and 200 µg/ml. All blood cultures obtained after initiation of antibiotic therapy demonstrated no bacterial growth.

Open heart surgery was accomplished on the sixth hospital day because of progressive cardiac deterioration. Five hundred ml of thick purulent pericardial fluid were removed. A shaggy peel of fibrinous purulent material, 1 to 2 cm thick, surrounded the heart and great vessels. Multiple small epicardial ulcerations were noted. The right and left aortic valve cusps were destroyed, but the noncoronary valve cusp, valve ring, and aortic root were intact. No aortic wall ulceration was noted. A Smeloff-Cutter ball valve was placed in the aortic position without difficulty. After operation the valve sounds were normal and no murmur of aortic insufficiency was heard.

On the seventh day after operation a faint continuous murmur was heard for the first time at the second and third intercostal spaces along the left sternal border, which progressed in loudness the next day. Cardiac catheterization demonstrated an increase in oxygen saturation at the pulmonary artery, providing a pulmonary to systemic blood flow ratio of two to one. An aortic root injection confirmed an aortopulmonary septal defect. No aortic insufficiency was seen.

Thirty hours following cardiac catheterization, the patient suddenly developed florid pulmonary edema. Despite conventional therapy, two hours after the onset of the pulmonary edema, the patient died.

At autopsy, an aortopulmonary septal defect was located 2 cm below and to the left of the previous aortotomy. It measured 1.5 x 1.5 cm on the aortic side and 1 x 1.5 cm on the pulmonic side (Fig 1). The aortic valve was well seated, without evidence of perivalvular leak. Sections taken from the myocardium inferior to the left coronary artery and the wall of the aorta adjacent to the aortopulmonary septal defect showed multifocal abscesses, with destruction of tissue and dense accumulation of neutrophils. Stain for bacteria con-

![Figure 1A (upper). Aortopulmonary septal defect (arrow) as viewed from aortic side. Orifice measured 1.5 by 1.5 cm. Defect located between left coronary artery (LC) opening and right coronary artery (RC) opening in left coronary sinus. Aortic valve was well seated with no perivalvular defect. B (lower). Aortopulmonary septal defect (arrow) as viewed from pulmonic side. Orifice measured 1.5 by 1 cm. Defect located in posterior pulmonary sinus.](image-url)
firmed the presence of clusters of large Gram-positive cocci in these sections. Bacteria were not demonstrated in sections taken from other areas in the aorta.

DISCUSSION

Aortopulmonary septal defect is a well recognized congenital heart defect, occurring as frequently as in 10 of 1,000 patients with congenital heart disease. Acquired aortopulmonary septal defects are apparently infrequent. Causes of acquired aortopulmonary septal defect include syphilis and trauma. Pirani described a young man with staphylococcal endocarditis who, at autopsy, had a communication between the left and right side of the heart at the level of the semilunar valves. It is unclear from the description whether the defect was intracardiac or extracardiac.

The precedence for early valve replacement in patients with aortic insufficiency and heart failure due to infective endocarditis has been established. During the nine-month period preceding the admission of the present patient, two similar patients had successful early aortic valve replacement at the San Diego Naval Hospital. At the time of aortic prosthetic valve replacement in this patient, there was no gross evidence of aortic endarteritis at the site of the subsequent development of the aortopulmonary septal defect. No suture lines or surgical clamps were placed in that area. Possibly, an erosive process was underway on the pulmonary artery side.

Although surgical intervention in congenital aortopulmonary septal defect is well established, it was considered appropriate to observe our patient following the demonstration of the aortopulmonary septal defect. Closure of the aortopulmonary septal defect was to be delayed until a later date when the tissue was less friable. The rapid progression in the size of this defect was not appreciated. Fifty-six hours after the murmur was first heard faintly, the patient was dead. In retrospect, a more promising course of action would have been immediate surgery once the diagnosis was established. Although the problem, as demonstrated at autopsy, would be formidable in terms of surgical correction, autogenous tissue such as pericardium would be the obvious choice for patch material. An alternate solution might be to approach the defect through the pulmonary artery, excluding the involved pulmonary cusp and sinus, creating a bicuspid pulmonary valve. This, together with primary closure of the aortic defect, might provide the most secure closure without need for a patch graft.

The present case demonstrated many of the classic findings of infective endocarditis due to coagulase-positive Staphylococcus aureus: there was no antecedent heart disease; there was recent onset of facial acne; onset of endocarditis was fulminant; there were multiple metastatic abscesses; and pericarditis was associated with purulent effusion. Abscess formation, which may lead to rupture, is aided by the enzyme coagulase; this enzyme promotes clotting in small vessels with subsequent necrosis and produces fibrin, which protects the Staphylococcus from phagocytes. Despite appropriate antibiotic treatment and early aortic valve replacement in this case, the disease process continued with formation of an aortopulmonary septal defect, resulting in death.

REFERENCES


Staphylococcal Pericarditis in a Patient with Systemic Lupus Erythematosus

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A patient with systemic lupus erythematosus developed spiking fevers, which were controlled by parenteral steroids, and, later, pericardial friction rub. Staphylococcal pericarditis was diagnosed by pericardiocentesis; no pathogenic organisms were found in blood or sputum. The infection did not respond to methicillin, and irrigation with Dakin's solution through a cardiac window was required to cure the pericarditis.

Pericarditis is a common clinical and autopsy finding in patients with systemic lupus erythematosus. Despite its high frequency, the morbidity of pericarditis in systemic lupus is relatively low. A patient with systemic lupus erythematosus acquired acute, purulent staphylococcal pericarditis that was not diagnosed until pericardiocentesis was performed.

CASE REPORT

A 52-year-old woman was admitted to the University of California Hospitals in October 1971. She complained of a weight loss of 10 kg (22.05 lb), anorexia, fatigue, and severe arthralgias. For ten years she had had intermittent morning