A *actinomycescomitans* among others. Because of the rarity of encounter in the medical community, *Hemophilus aphrophilus* will probably continue to be confused with other organisms.

*Hemophilus aphrophilus* has been implicated in a variety of clinical human infections of various organs including skin, sinuses, respiratory tract, but the majority have been related to endocarditis and brain abscesses. Thirty-three cases of *Hemophilus aphrophilus* endocarditis are known and the majority involve a valve previously damaged by rheumatic disease or congenital deformity. At least three cases have involved a normal valve. In this case the valve was normal.

Two cases of *Hemophilus aphrophilus* infection have been associated with positive saliva cultures from dogs with whom close contact was maintained, although humans may be the reservoir rather than animals. The dog and family contacts of this patient were culture negative.

Peripheral embolization is a common feature with this organism and several cases have had persistent emboli in the face of massive antibiotic therapy. A similar course was noted in this case. *Hemophilus aphrophilus* is usually sensitive to penicillin and streptomycin and these are considered the therapy of choice, although kanamycin, gentamicin, ampicillin, chloramphenicol are considered useful.

Although tube dilution and disc sensitivities were requested on the isolated organism, they were not done and attempts to revive the organism for these studies were unsuccessful. The rapid abatement of fever, improved clinical state, lack of positive blood cultures after the start of therapy and the inability to culture the organism from the surgical material does suggest sensitivity to the antibiotics used, but due to the lack of sensitivity studies, the adequacy of antibiotic treatment remains in doubt.

In the case presented, progressive aortic insufficiency occurred in spite of continued medical therapy and acute cardiac decompensation necessitated emergency valve replacement. This appears to have been lifesaving, as evidenced by the gross destructive changes found on the aortic valve and rapid clinical deterioration. Cases of valve perforation and aortic sinus aneurysm have been previously described with *Hemophilus aphrophilus* endocarditis, but this is the first reported with this organism requiring valve replacement. Prosthetic valve replacement for endocarditis is now an accepted approach to complications of this illness and the triad of bacterial endocarditis, aortic insufficiency and congestive failure requires valve replacement to insure an acceptable survival.

ACKNOWLEDGMENT: I am grateful for the assistance and care rendered by the medical and surgical staff of the Albuquerque Veterans Administration Hospital. I am also grateful for technical assistance given me in this paper by Mrs. Margaret Tudor.

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Rapid Development of Constrictive Pericarditis in a Patient with Systemic Lupus Erythematosus

Ralph H. Starkey, M.D. and Bevra H. Hahn, M.D.

A 28-year-old man presented with systemic lupus erythematosus manifested by nondeforming polyarthritis, lymphadenopathy, acute pericarditis with pericardial effusion, LE cells, and antibodies to native DNA. Constrictive pericarditis developed over the following seven weeks, although corticosteroids were being administered. Constrictive pericarditis in systemic lupus is quite rare, but this entity does occur, may develop rapidly, and may not be prevented by corticosteroid therapy.

Acute pericarditis is a well-recognized manifestation of systemic lupus erythematosus, occurring in 17-48 percent of patients. Furthermore, pericardial effusion is not uncommon and has been reported in 9-15 percent. In contrast, the development of clinically significant constrictive pericarditis is rare. Constrictive pericarditis as a manifestation of systemic lupus has been reported previously in only two patients and is notably absent from large series of lupus patients such as those of Harvey and co-workers and Dubois. This report describes a young man with systemic lupus in whom acute pericarditis progressed to constrictive pericardial disease over a period of seven weeks in spite of...
RAPID DEVELOPMENT OF CONSTRUCTIVE PERICARDITIS

A 28-year-old white man was admitted to St. Louis Veterans Hospital on May 26, 1971, complaining of chest pain. For three years he had suffered intermittent attacks of migratory pain, warmth, and swelling in joints of the fingers, wrists, elbows, knees and ankles. Completely asymptomatic intervals occurred between attacks. During the six weeks prior to admission, the joint syndrome had recurred, accompanied by malaise, anorexia, and shortness of breath. During the preceding week, vague left precordial chest pain had appeared. He had no fever, orthopnea, nocturnal dyspnea, or pedal edema. There was no history of antecedent upper respiratory infection or exposure to tuberculosis.

Physical examination revealed blood pressure 100/80 mm Hg, regular pulse rate of 100/min and temperature 99.6°F. Pea-sized cervical, axillary, epitrochlear, and inguinal lymph nodes were palpable. There were no skin rashes or subcutaneous nodules. The only cardiac abnormality was a loud, three-component pericardial friction rub. Jugular venous distension, pulmonary abnormalities, liver enlargement or tenderness, ascites, and peripheral edema were absent. There was synovial thickening of the metacarpophalangeal and proximal interphalangeal joints bilaterally, but no joint deformity was present.

Laboratory studies included a hematocrit of 39 percent, white blood cell count 10,700/cu mm with a normal differential, corrected erythrocyte sedimentation rate of 30 mm/hour (Wintrobe), normal serum, and creatinine clearances of 70-100 ml/min. Electrocardiogram showed nonspecific T-wave abnormalities; x-ray film of the chest demonstrated an enlarged cardiac silhouette without pulmonary vascular congestion (Fig 1A). Cardiac fluoroscopy revealed a 1.5 cm distance between the epicardial fat pad and the fat pad of the internal mammary artery, consistent with pericardial effusion. An intermediate purified protein derivative (PPD) skin test was negative. Three lupus erythematosus (LE) cell preparations were strongly positive, with abundant LE cells and extracellular nuclear material. The latex-fixation test for rheumatoid factor was positive at a titer of 1:160. Serum protein electrophoresis showed slightly decreased albumin and increased a-2 globulin. Antibodies to native DNA, measured by modified Farr technique\(^7\) were present. Immuno-fluorescent tests for antinuclear antibody were repeatedly negative.

A diagnosis of pericarditis secondary to systemic lupus erythematosus was made, and the patient was treated with 60 mg of prednisone daily. Over a two-week period, the cardiac silhouette diminished in size, the friction rub disappeared, and the pericardial effusion disappeared as evaluated by cardiac blood pool scanning. Tapering of prednisone was begun and he was discharged.

One month later, the patient returned complaining of ankle swelling, increased abdominal girth, and nonproductive cough when supine. He had gained 12 pounds. He had been taking 15 mg prednisone daily for two weeks. Jugular vein distension was evident in a sitting position. Tender hepatomegaly and pitting ankle edema were present. Roentgenogram demonstrated a reduction of cardiac size (Fig 1B), but cardiac pulsations were good as judged by fluoroscopy. Lupus erythematosus preps had become negative, the sedimentation rate was normal, and the rheumatoid factor titer had fallen to <1:20.

During the next month, diuretic therapy proved to be ineffective in relieving edema. Cardiac catheterization showed evidence of pericardial constriction, and pericardectomy was performed. At operation, the cardiac pulsations were dampened, and the pericardium was 2-4 mm thick. Microscopic examination revealed hyalinization of collagen fibers and areas of chronic nonspecific inflammatory reaction. No granulomas were seen; cultures for tuberculosis and fungi gave negative results.

The postoperative period was uneventful; his only remaining symptom was slightly decreased exercise tolerance.

Discussion

In this patient, constrictive pericarditis represented a rare but life-threatening manifestation of systemic lupus. The diagnosis of lupus was based on the clinical findings of intermittent, nondeforming polyarthritis, generalized lymphadenopathy, and pericarditis, and the laboratory findings of strongly positive LE cell preparations, transiently positive rheumatoid factor, and the presence of
antibodies to native DNA.

A search of the literature revealed only two previously reported cases of constrictive pericarditis attributable to systemic lupus. Yurchak and co-workers described a patient who had systemic lupus manifested by malar rash, antinuclear antibodies, and LE cells at the time when constrictive pericarditis developed. However, he also had an atrial septal defect and rheumatic heart disease; an association between atrial septal defect and constrictive pericarditis has been reported. The second case was described very briefly, and the diagnosis of lupus was made retrospectively, in that LE cell preparations were found to be positive an unspecified period of time after pericardiectomy. The case described here represents a report of constrictive pericarditis in a patient with clinically active systemic lupus in the absence of other disorders.

The rapidity with which constriction developed in this case was noteworthy. The time interval from onset of chest pain to clinical evidence of pericardial constriction was only seven weeks. Although corticosteroids relieved the symptoms of acute pericarditis, the development of constriction was apparently not inhibited.

This case illustrates that constrictive pericarditis can be a manifestation of systemic lupus erythematosus, that it can develop quite rapidly, and that it may not be prevented by corticosteroid therapy.

REFERENCES

Idiopathic Right Atrial Enlargement with Pericardial Effusion*

Abdul J. Tajik, M.D., James C. Broadbent, M.D., and Thomas T. Schattenberg, M.D., F.C.C.P.

An asymptomatic patient with idiopathic right atrial enlargement is described in whom the interesting feature was associated pericardial effusion; this association has not been reported previously. Both of these conditions had been present for 16 years and both remained non-progressive.

Idiopathic enlargement of the right atrium is a rare and perhaps a controversial clinical entity. Since the original description of Pastor and Forte in 1961, 15 cases have been reported in the English literature. Originally, the condition was regarded as benign, but more recently, there have been reports of symptomatic patients who had massive enlargement of the right atrium associated with atrial dysrhythmias. There is one reported incidence of sudden death, and some patients have undergone exploratory thoracotomy unnecessarily. The condition of idiopathic enlargement of the right atrium probably occurs more frequently and is less benign than early communications implied. Accordingly, this report describes a patient with enlargement of the right atrium in whom the feature of special interest is chronic pericardial effusion. This association has not been described previously.

CASE REPORT

A 26-year-old man was referred to the Mayo Clinic because of an abnormal cardiac silhouette on a routine chest roentgenogram. The patient was asymptomatic and had no history of rheumatic fever, pericarditis, hypertension, or heart murmur. A routine roentgenogram of the chest taken when the patient was ten years old was suggestive of cardiac enlargement.

Table 1—Hemodynamic Summary of Patient with Enlarged Right Atrium and Associated Pericardial Effusion*

<table>
<thead>
<tr>
<th>Site</th>
<th>Pressure, mm Hg</th>
<th>Percent Saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femoral artery</td>
<td>129/66</td>
<td>96</td>
</tr>
<tr>
<td>Wedge</td>
<td>18/10 (m = 12)</td>
<td>98</td>
</tr>
<tr>
<td>Main pulmonary artery</td>
<td>24/11</td>
<td>80</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>28/3</td>
<td>78</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>—</td>
<td>84</td>
</tr>
<tr>
<td>Low right atrium</td>
<td>—</td>
<td>83</td>
</tr>
<tr>
<td>Mid right atrium</td>
<td>10/4 (m = 6)</td>
<td>83</td>
</tr>
<tr>
<td>High right atrium</td>
<td>—</td>
<td>80</td>
</tr>
<tr>
<td>Low superior vena cava</td>
<td>—</td>
<td>80</td>
</tr>
<tr>
<td>High superior vena cava</td>
<td>—</td>
<td>81</td>
</tr>
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*Cardiac index = 3.3 liters/min/sq m.

*From the Mayo Clinic and Mayo Foundation, Rochester, Minnesota. Reprint requests: Section of Publications, Mayo Clinic, Rochester, Minnesota 55901

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