Ineffective left atrial function following cardioversion has been suggested as a significant contributing factor in the pathogenesis of postcardioversion pulmonary edema. This has been presumed on clinical grounds, and to our knowledge, no echocardiographic studies or bedside hemodynamic measurements have been reported in a case of postcardioversion pulmonary edema. The demonstration of absent left atrial booster activity in our patient indicates left atrial mechanical failure. The return of right atrial contraction in the absence of left atrial contraction may result in increased pulmonary venous volume and pressure causing pulmonary edema.

It has been shown previously that there is a failure of a chronotropic response to hypotension in patients with postcardioversion pulmonary edema, leading to a suggestion that a widespread aberration in the cardiovascular reflex response to the changing rhythm may underlie this complication. This neurogenic mechanism has not been validated.

A recent report has suggested that the atrial natriuretic peptide level is elevated in elderly patients with either atrial flutter or atrial fibrillation regardless of the presence or absence of heart failure. The level decreases promptly after successful cardioversion, independent of the mode of cardioversion or presence of congestive heart failure. Atrial natriuretic peptide has beneficial counterregulatory effects in heart failure. It is tempting to postulate that the fall in atrial natriuretic peptide levels may result in fluid retention and vasoconstriction, thus contributing to the development of pulmonary edema in patients with left ventricular dysfunction or mitral valve disease.

The mechanism of postcardioversion pulmonary edema is probably multifactorial involving hemodynamic changes caused by ineffective left atrial function, left ventricular dysfunction, as well as neurohumoral regulatory mechanisms. Invasive hemodynamic studies and assessment of neurohumoral factors are required to elucidate this longstanding issue.

SUMMARY

Pulmonary edema is a rare complication of conversion of a tachyarrhythmia to sinus rhythm. Left atrial mechanical failure following cardioversion has been suggested as a contributory factor. This report has described a patient in whom this complication occurred 29 h following successful cardioversion. Echocardiography showed evidence of impaired left atrial mechanical function, an association which has been postulated previously but not documented echocardiographically.

REFERENCES

Nonfebrile Mitral Valve Endocarditis Due To Neisseria subflava*

Bram J. Amsel, MD, and Adriaan C. Mouljin, MD, FCCP

Native valve endocarditis normally presents with fever and only later in its course demonstrates dysfunction of the affected valve. We describe a case of endocarditis due to Neisseria subflava, a Gram-negative diplococcal saprophyte of the oral cavity, which was unsuspected clinically and found unexpectedly during a mitral valve operation performed for symptomatic prolapse with regurgitation.

(CHEST 1996; 109:280–82)

Key words: endocarditis; mitral regurgitation; Neisseria subflava; valve repair; valve surgery

Endocarditis due to Neisseria subflava, a Gram-negative diplococcal saprophyte of the oral cavity, rarely has been described in the literature. All patients presented with fever. We recently saw an afibrile man admitted for correction of long-standing mitral regurgitation due to prolapse, who at the time of the surgery was also found to have mitral valve endocarditis and leaflet perforation due to N. subflava.

CASE REPORT

A 56-year-old man was hospitalized for evaluation of his mitral regurgitation. He had been seen elsewhere 2 years previously where a diagnosis of moderately severe mitral regurgitation due to prolapse of the posterior leaflet was made. Left ventricular dysfunction and atrial fibrillation were documented at that time. There had been no prior episodes suggestive of endocarditis. The patient was treated conservatively, and he remained asymptomatic while receiving digitalis and anticoagulant treatment until several weeks before the present admission, when he noticed progressive shortness of breath, finally culminating in dyspnea at rest and orthopnea. He denied having had a fever. He had smoked a pack of cigarettes a day for many years and admitted to drinking 20 L of beer a week. There was no history of illicit drug use, and there was no recent medical history of antibiotic administration, prescription of anti-inflammatory drugs, or dental procedures.

At the time of examination, he was not ill, underfed or intoxicated, and he was not short of breath. His blood pressure was 130/90 mm Hg. The jugular veins were flat. There was a sustained

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Table 1—Published Cases of Definite and Probable Endocarditis Due to Neisseria subflava

<table>
<thead>
<tr>
<th>Reference, year</th>
<th>Age, yr/SEX</th>
<th>Predisposing Factor</th>
<th>Valve Affected</th>
<th>Surgery</th>
<th>Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kammerer &amp; Wegner, 2 1914</td>
<td>2/M</td>
<td>...</td>
<td>Aortic, mitral</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Connaughton &amp; Rountree, 3 1939</td>
<td>22/F</td>
<td>...</td>
<td>Mitral (stenosis)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Breslin et al, 4 1967</td>
<td>44/M</td>
<td>...</td>
<td>Aortic</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Clark &amp; Patton, 5 1968</td>
<td>47/M</td>
<td>...</td>
<td>Aortic valve prosthesis</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Moon et al, 6 1975</td>
<td>36/M</td>
<td>...</td>
<td>Mitral (stenosis)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Harris, 7 1981</td>
<td>35/M</td>
<td>Dental abscesses</td>
<td>...</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Pollack et al, 8 1984</td>
<td>43/M</td>
<td>IV drugs</td>
<td>Mitral, aortic</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Dickinson et al, 9 1985</td>
<td>49/F</td>
<td>IV drugs</td>
<td>Right sided</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Vlasveld, 10 1986</td>
<td>51/F</td>
<td>Asplenia, diabetes, dental abscesses</td>
<td>Mitral valve prosthesis</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Guelpa et al, 11 1986</td>
<td>50/F</td>
<td>...</td>
<td>Tricuspid</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Baquero et al, 12 1989</td>
<td>2/M</td>
<td>HIV, hepatitis B</td>
<td>...</td>
<td>No</td>
<td>?</td>
</tr>
<tr>
<td>Szabo et al, 13 1990</td>
<td>37/F</td>
<td>IV drugs</td>
<td>Tricuspid</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Ansotegui &amp; Moulijn, present report</td>
<td>56/M</td>
<td>Alcohol, dental abscesses</td>
<td>Mitral</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

and broadened apical cardiac impulse which was displaced to the left. The first heart sound was loud and was followed by a grade 3/4 murmur consistent with mitral regurgitation, and a dull third heart sound. Breath sounds were normal. The liver and spleen were not enlarged. There was no pedal edema, and there were no abnormal cutaneous findings. His teeth were in poor condition with significant periodontal disease, gingivitis, and dental plaque, and several elements required extraction. Laboratory examination showed a sedimentation rate of 1 mm/h, a C-reactive protein value of <0.5 mg/dL, a hemoglobin level of 15.6 g/dL, and a WBC count of 9.1 x 10^9/L with a normal differential cell count. Aspartate aminotransferase was 52 U/L (normal to 17 U/L); alanine aminotransferase, 88 U/L (22 U/L); alkaline phosphatase value, 121 U/L (166 U/L); γ-GT, 34 U/L (20 U/L); and lactate dehydrogenase, 285 U/L (220 U/L); protein level, 7.2 g/L; serum albumin value, 5.1 g/L. Plasma creatinine level was 1.2 mg/dL, and urinalysis was within normal limits. Blood cultures were not performed.

Transcostal and transesophageal echocardiography showed an enlarged, poorly contracting left ventricle and prolapate of the posterior mitral leaflet with grade 4/4 mitral regurgitation at Doppler examination. No vegetation or leaflet perforation could be visualized, and the direction of the regurgitant jet toward the anterior left atrial wall was compatible with the prolapate. The left atrium was enlarged, 49 mm. Coronary angiography demonstrated normal coronary arteries.

At the time of surgery, in addition to lengthened posterior chordae tendineae which explained the prolapse, a round perforation 1 cm in size was found in the posterior leaflet near the anterior commissure. The edge of the defect was rough and thickened, and endocarditis was suspected. The valve was repaired by means of a quadrangular resection of the posterior leaflet, shortening of two posterior chordae tendineae, and transposition of a secondary anterior chord to the posterior leaflet, and the annulus was configured by means of a 34-mm Carpentier-Edwards annuloplasty ring.

The patient had received 2 g of cefamandole nafate at the induction of anesthesia just prior to surgery. For the first few days after the operation, gentamicin and vancomycin were prescribed. When valve cultures were reported to have grown N subflava, therapy was switched to amikacin sulfate and 12 g of ampicillin a day for another 5 weeks, and the patient recovered completely.

During the hospital stay, the patient's dental disease was treated. Four months postoperatively, the patient felt very well. He denied smoking and consuming alcoholic beverages, and he rode a bicycle between 5 and 25 miles daily. A grade 1/6 mitral regurgitant murmur was heard. Transthoracic echocardiographic examination showed improved left ventricular systolic function and a reduced left atrial dimension and could not confirm the presence of mitral regurgitation.

**Discussion**

*N subflava*, formerly considered separately as *N subflava*, *N perflava*, and *N flava*, is a Gram-negative diplococcus saprophyte of the oral cavity that is chromogenic, grows at 22°C, ferments glucose, maltose and sucrose, produces H2S, and polymerizes sucrose. 1 It has been found to be associated with cases of sepsis, meningitis, and rarely, endocarditis. 2-13 Twelve cases of at least probable endocarditis, at least eight of which were reported after 1980, classified according to the von Reyn criteria, 14 due to *N subflava*, have been described in the literature (Table 1). Beyond possible preexistent valvular abnormalities, seven of the eight most recent cases, including our own, had evident predisposing conditions, five as a probable immune incompetence (HIV and chronic hepatitis, IV drug use, asplenia, or diabetes) and six with a possible source of infection (dental abscesses and IV drugs). There is no evidence that alcoholism without cirrhosis of the liver, as in our patient, may lead to susceptibility to endocarditis. The excessive alcohol intake, however, may have contributed to the left ventricular dysfunction and withdrawal of alcohol to postoperative improvement. All patients described, except our own, either had a fever or died or had the fever and died. Generally, patients with active endocarditis due to any bacterium have a fever unless they are taking antibiotics or anti-inflammatory drugs, are debilitated, in heart failure, or are suffering from renal failure. 15

In contrast to the gradually progressive symptoms of our patient, most instances of valve leaflet perforation lead to acute symptoms. When, as here, however, the left atrium is "prepared" for low-pressure volume overload by longstanding mitral regurgitation, worsening of the regurgitation might lead to a less dramatic progression of symptoms. This case illustrates the importance of careful inspection and, if suspicion of endocarditis arises, also that of culture of a heart valve at the time of corrective surgery even when a cause for valvular dysfunction other than endocarditis is known. More importantly subcutaneously worsening symptoms of valvular dysfunction should alert the physician to the possibility of endocarditis. The place of blood cultures in febrile valvular disease is unknown. Making a diagnosis of active endocarditis in valve-determined heart failure is most relevant because surgical correction should take place early in order to prevent further valvular or myocardial damage. Conversely, heart failure is the most frequent indication for
valvular surgery in active endocarditis, since it is present in 60 to 90% of cases.16 When possible, valvular repair is preferable to replacement because of the lower susceptibility of a repaired valve to infection and thromboembolic complications.

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Acute Myocarditis and Left Ventricular Aneurysm as Presentations of Systemic Lupus Erythematosus*

Andrea Frustaci, MD, FCCP; Nicola Gentiloni, MD; and Marina Caldarulo, MD

A case of systemic lupus erythematosus (SLE) associated with fever, heart failure, and left ventricular (LV) aneurysm is reported. A diagnosis of SLE was suspected owing to the presence of active lymphocytic myocarditis and fibrinous endocarditis at LV endomyocardial biopsy and was confirmed by identification of 4 of the 11 criteria proposed by the American Rheumatism Association for the definition of SLE. A 2-month period of steroid therapy was followed by a remarkable recovery of LV function and progression of endomyocarditis to a healed phase at control LV biopsy. The LV aneurysm disappeared, likely because thrombosis occurred as a result of the hypercoagulable state accompanying the presence of anticardiolipin antibodies. This is the first reported case of LV aneurysm induced by SLE and is a rare clinicohistologic documentation of the effectiveness of steroid treatment on lupus endomyocarditis.

(CHEST 1996; 109:282-84)

LV=left ventricular; SLE=systemic lupus erythematosus

Key words: cardiac aneurysm; endomyocarditis; systemic lupus erythematosus

While postmortem studies reveal myocardial inflammatory lesions in up to 70% of victims of systemic lupus erythematosus (SLE), clinical manifestations of lupus myocarditis are uncommon,1-3 and acute myocarditis as a presenting symptom of SLE is exceedingly rare.4 The pathologic spectrum of myocardial SLE includes severe manifestations such as myocardial infarction due to coronary arteritis5,6 and congestive heart failure.7 However, no previous report exists in the literature of lupus myocarditis causing cardiac aneurysm.

Herein is a case of SLE associated with acute myocarditis and localized left ventricular (LV) aneurysm. In this case, a 2-month period of steroid treatment was followed by a remarkable recovery of LV function and disappearance of the aneurysm, very likely as a consequence of a thrombotic event. Mechanical and histologic changes were detailed by sequential LV angiography and endomyocardial biopsy, respectively, which showed both active myocarditis and fibrinous endocarditis progressing, while the patient was receiving steroids, to a healed phase.

CASE REPORT

A 38-year-old man was admitted to the hospital because of progressively worsening fever, palpitations, weakness, and effort dyspnea, which had first appeared 2 weeks earlier. At the time of physical examination, ulcers in the oral mucosa were present, and basal pulmonary rales were audible; heart auscultation revealed the presence of a gallop rhythm, 120 beats per minute. BP was 110/70 mm Hg. The ECG showed diffuse ST-T abnormalities in the peripheral and precordial leads. A 2-dimensional echocardiogram showed normal cardiac volumes and wall thickness. LV ejection fraction was 45%. No dyskinetic segments and/or parietal thrombi were shown. No evidence of pericardial effusion was apparent. Doppler analysis showed mild mitral and tricuspid regurgitation. Myocardial enzymes, including creatine kinase-MB, were in the normal range. There was moderate anemia (3,250,000 RBC;