a major complication of this procedure, is reported in 10 to 50 percent of the cases. Although the indirect method of inferior vena cava interruption using an umbrella filter has resulted in significant reduction of morbidity and mortality, it is not free of complications. Because of the risks associated with different methods of inferior vena cava interruption, this procedure is advisable only when the diagnosis is conclusively established and when the previously mentioned indications are present.

Our experience and that of others suggests that perfusion lung scan is not a very reliable test for diagnosing pulmonary embolus and that pulmonary emboli should be demonstrated arteriographically before interruption of the inferior vena cava is performed for the prevention of recurrent pulmonary emboli.

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Complete Heart Block due to Acute Nonspecific Carditis*

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The scarcity of reports of complete heart block complicating acute nonspecific carditis suggests that this is a rare occurrence. We observed and herein report three such cases from the same geographic area within a two-week period, which suggests the contrary. Consequently, in the "viral syndrome" or after pharyngitis, symptoms of dizziness or a pulse rate inappropriately slow for the acute illness require further investigation. At times, support of cardiac rhythm will be required.

A cute nonspecific carditis, usually benign and self-limited, is generally thought to be part of a viral infectious process, although frequently acute rheumatic fever also enters into the differential diagnosis. High-grade disturbances in conduction have been considered to be rare in both of these conditions, especially in the absence of the occasionally severe form with flagrant cardiac decompensation. The purpose of this report is to present a cluster of three cases of otherwise mild, acute "nonspecific" carditis complicated by complete heart block that were seen in a two-week period during the summer of 1975 in the area of southeastern Connecticut. Although the true incidence of this complication is still not known, it should not be considered a rarity.

CASE REPORTS

Case 1

A 20-year-old woman was hospitalized on July 15, 1975, complaining of syncope. Two weeks earlier, she experienced a syndrome characterized by fever, myalgia, sore throat, and earache. These symptoms lasted several days, but for the week prior to admission, the patient was asymptomatic. On the day of admission, she had multiple episodes of syncope. The electrocardiogram demonstrated complete heart block with a slow junctional escape rhythm. Physical examination revealed blood pressure of 110/70 mm Hg, temperature of 37.4°C (99.4°F), and pulse rate of 40 to 50 beats per minute. The lungs were clear. First and second heart sounds were normal. A grade 2/6 mitral regurgitant murmur and an S3 filling sound were heard. The liver and spleen were not palpable. There was no peripheral edema and no cutaneous rash. Laboratory examination revealed normal values for a complete blood cell count, serum glutamic oxaloacetic transaminase level and creatine phosphokinase level. The erythrocyte sedimentation rate was 74 mm/hr. Blood cultures were negative, a throat swab grew normal flora, and throat and rectal swabs for enterovirus culture were negative. Samples of serum from the acute and convalescent phases examined for viral antibodies were nondiagnostic and were negative for streptozyme and antinuclear antibody. The chest x-ray film was normal. The patient was treated by insertion of a temporary transvenous pacemaker. Her heart block resolved over several days, and therapy with the pacemaker was discontinued on the seventh day. On a follow-up examination one month after discharge from the hospital, the patient was asymptomatic. Her ECG was normal. The mitral regurgitant murmur was not detected.

Case 2

A 28-year-old man entered the hospital on July 16, 1975, following two weeks of fever, fatigue, arthralgia, and a transient rash of the lower abdomen and proximal lower extremities. He consulted his physician because of palpitations, at which time an ECG demonstrated frequent blocked P waves, which resulted in his admission. The patient's blood pressure was 110/70 mm Hg, his temperature was 37.8°C (100°F),
and his pulse rate was 60 beats per minute and irregular. There was no jugular venous distention, the lungs were clear, and the heart was not enlarged. First and second heart sounds were normal, and there was a soft late-systolic apical murmur. The liver and spleen were not palpable, and there was no peripheral edema. A chest x-ray film was within normal limits. Other laboratory data included a hematocrit reading of 33 percent and a white blood cell count (WBC) of 10,200/cu mm, with 67 percent neutrophils and 14 percent band forms. The erythrocyte sedimentation rate was 55 mm/hr. Titers of antinuclear antibody and anti-DNA were negative. The titer of antistreptolysin O was 1:32; the serum level of complement was normal. Blood cultures were negative, and a throat culture grew normal flora. Throat and rectal cultures were negative. Blood drawn on admission showed viral titers of Coxsackie virus, types B3 and A9, each of 1:28. There was no change in titer nine days later.

On the third day of hospitalization, complete atrioventricular block developed, with a junctional escape rate of 40 beats per minute. Therapy with prednisone (15 mg orally every six hours) was given, with resolution of the patient’s disturbances in conduction over a 48-hour period. Therapy with steroids was then tapered, and a 24-hour ambulatory ECG prior to discharge showed normal sinus rhythm, occasional blocked atrial premature contractions, and no other evidence of a disturbance in atrioventricular conduction. At a follow-up examination four months later, the ECG was normal.

**CASE 3**

A 24-year-old man was hospitalized on July 31, 1975, having been ill for 4 weeks. The illness began with fever (38.9°C [102°F]), myalgia, rash, headache, lethargy, and sore throat. The patient’s physician had diagnosed a viral illness which was prevalent in the community at that time and treated the patient with salicylates. His fever abated, but the patient had only partial relief of the other symptoms and, therefore, after a month, came for further evaluation.

Physical examination revealed an afebrile healthy-appearing white man with a pulse rate of 84 beats per minute and regular and a blood pressure of 140/80 mm Hg. The patient had a faint pink macular rash on the trunk and proximal extremities. Findings from the remainder of the examination, including the heart, were unremarkable. Laboratory data included a hematocrit reading of 39 percent and a WBC of 7,100/cu mm, with a normal differential count. Corrected erythrocyte sedimentation rate was 34 mm/hr. The chest x-ray film was normal.

On the first day of hospitalization, the ECG was abnormal only for a P-R interval of 0.24 second. On the second day of hospitalization, transient 2:1 atrioventricular block was seen, followed by complete heart block and a junctional escape rate as low as 30 beats per minute. The patient became dizzy and hypotensive, and a transvenous pacing wire was inserted. A His bundle electrogram recorded with that wire demonstrated the block to be at the level of the atrioventricular junction (Fig 1). Therapy with prednisone (60 mg/day in four doses) was given, and within 36 hours, predominately sinus rhythm with first-degree block had returned. The pacemaker wire was removed after six days, and therapy with steroids was tapered. There was one recurrent episode of transient 2:1 atrioventricular block without symptoms on the 12th day of hospitalization, for which the dosage of steroids was increased, but by the 16th day of hospitalization, normal sinus rhythm with a P-R interval of 0.20 second was present and remained unchanged until discharge from the hospital.

Additional laboratory studies included a negative titer for antistreptolysin O on Aug 1, greater than 400 but less than 600 Todd units on Aug 4, and a negative titer on Sept 1. Reactions for cold agglutinins and antinuclear factor were negative and for C reactive protein were weakly positive.

The titer of anti-DNA-ase was 1:340 the first week and negative the third. The level of antihyaluronidase was 1,024 units/ml. Numerous throat cultures grew only normal flora, and cultures of blood and urine were negative. Attempts at isolating Coxsackie virus from stool were unsuccessful, and viral titers of samples of serum obtained during the acute and convalescent phase of the illness revealed no remarkable patterns. As a result of the late serologic studies, it was elected to treat the patient with a two-week course of penicillin for the possibility of uncultured streptococcal infection. On follow-up examinations at three and six months, the findings from physical examination and ECG have remained normal.

**DISCUSSION**

The specific etiology of these three cases was not proven. As is frequently the case in this type of illness, postviral infection is thought to be likely, but no data from cultures or antibody titers were conclusive. Also common to such cases, rheumatic fever was considered a possibility, particularly in case 3, where some of the data were suggestive, but throat culture was negative and Jones’ criteria were not met.

The designation, “carditis,” as we have applied it to
these three cases, is purposefully general and is used to encompass episodes of objective cardiac involvement during or presumably due to an infectious type of systemic illness. Carditis at times can be evidenced exclusively by minor electrocardiographic changes in the S-T segment and the T wave. In some patients, pain and an audible rub suggests the more specific designation of pericarditis. In others, prominent heart failure with gallop rhythm, rales, etc, usually receive the designation of myocarditis. In the reports cited in the following paragraphs, previous investigators have generally combined the various manifestations of cardiac involvement in their reports.

Lev\(^1\) reported that severe heart block can be seen in any type of acute myocarditis: "It is possible to have marked involvement of the ordinary myocardium with minimal involvement of the conduction system and vice-versa." The frequency with which isolated inflammatory processes of the conduction system occur is not stated.

The clinical literature suggests that disturbances in conduction associated with viral illnesses presenting as mild pericarditis or myocarditis and with acute rheumatic carditis\(^2\) rarely progress to complete heart block. Sainani and associates\(^3\) reported their findings in 22 patients with heart disease secondary to infection with Coxsackie virus. Pericardial friction rubs were detected in eight patients, and six patients initially had acute heart failure. Chest x-ray films disclosed diffuse cardiac enlargement in 12 of these 22 cases. One patient had right bundle-branch block. None developed complete heart block. Helin and co-workers\(^4\) reported 18 cases of acute myopericarditis which occurred during a Coxsackie B5 epidemic in Finland in the autumn of 1965. Initially, the patients characteristically had fever, precordial pleuritic chest pain, and pericardial friction rub. Cardiac size on the chest x-ray film was reported as normal in ten of the 18 patients, the remaining eight patients demonstrating enlarged cardiac silhouettes. Electrocardiographic findings were limited to changes in the S-T segment and the T wave. No case was complicated by atrioventricular block.

Smith\(^5\) reported the findings in 42 patients with heart disease due to Coxsackie virus occurring in Australia from 1962 to 1969. Twenty of the patients had myocarditis without evidence of pericarditis. Heart failure was present in 11 patients, ten of whom had clinically isolated myocarditis. The chest x-ray film showed cardiomegaly in 22 patients, and pulmonary edema or congestion was present in 12. Four patients were admitted with patterns of bundle-branch block. One of these is described as having "severe congestive heart failure" and a period of complete heart block. That patient, a 23-year-old woman, eventually returned to normal sinus rhythm.

Lim et al\(^6\) have reported the largest series of cases of complete heart block complicating nonspecific carditis. Over a four-year period, these investigators saw ten such patients at the University of Singapore. Eight of these patients had no evidence of cardiomegaly. Two patients developed permanent complete heart block, and one developed "bifascicular disease of the atrioventricular conduction system."

The treatment of complete heart block, when seen in this setting, has been empirical in nature. Atropine has been given to facilitate conduction, and isoproterenol has been used to increase the rate of a lower focus. The place of corticosteroid therapy in this condition has been uncertain. These agents presumably reduce edema and infiltration of conduction tissues and have been used with some apparent success; however, debate continues over the risk of steroid-potentiated viral replication.\(^7\)

The indications for transvenous pacing in this condition are empirical as well. The need for such intervention is based upon overall hemodynamic status. Transient complete block with an escape focus rate over 40 beats per minute is usually well tolerated, and a trial of one of the pharmacologic interventions appears to be prudent. On the other hand, if perfusion of the target organ is diminished, if early heart failure is evident, or if the escape rate is a slow one, then transvenous pacing has been the treatment of choice.

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