presented. Moreover, patients with right-sided endocarditis may often have vegetations engrafted on more than one valve.¹ It is not known what degree of success one might expect from attempts at delineating lesions on more distal valves by right atrial angiographic techniques. Such attempts would be especially difficult in the presence of low output states or with tricuspid regurgitation.

Despite the problems outlined herein, we would suggest that forward angiographic studies be added to other diagnostic procedures in situations analogous to that described, and that such studies should be considered prior to resorting to more potentially hazardous maneuvers.

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REFERENCES


Cytomegaloviral Infection Presenting as a Solitary Pulmonary Nodule*

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Cytomegaloviral infection presenting in an immunologically compromised host as a solitary pulmonary nodule has not previously been reported. A patient with a renal transplant and with no pulmonary symptoms was noted to have a single nodule on a chest roentgenogram. At autopsy, this proved to be secondary to cytomegaloviral infection. Differential diagnostic considerations in the immunosuppressed patient are discussed.

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The high incidence of infection with cytomegalovirus in patients who have received renal transplants has been reported by several observers.¹-⁴ Frequently, infection with this virus occurs in conjunction with other opportunistic invaders, making radiographic analysis difficult.⁵,⁶ Initial reports stated that in those cases in which cytomegalovirus was the sole pathogen, the radiographic pattern consisted of small nodules scattered throughout the periphery of the lung;¹-³ however, more recently, Bragg and Janis⁴ have indicated that the pattern is commonly one of bilateral nodular infiltrates in the upper lobes of an asymptomatic patient who has received a transplant.

We have recently encountered cytomegaloviral infection presenting as a solitary nodule in the left lower lobe of a patient with a renal transplant. The unusual presentation in this immunologically compromised host suggested the possibility of several other serious infections, such as aspergillosis, nocardiosis, or phycomycosis.

CASE REPORT

This 22-year-old man with progressive renal failure secondary to systemic lupus erythematosus underwent renal transplantation on July 24, 1974. The surgical procedure was uneventful, and the kidney functioned well in the immediate postoperative period. Medications after the transplantation consisted of 150 mg of prednisone and 150 mg of azathioprine per day. The dosage of azathioprine was subsequently reduced by half because of developing neutropenia. An initial postoperative bacteriologic survey revealed only a throat culture positive for Candida albicans, which was treated with a mouthwash containing nystatin (Mycostatin).

On the eighth day after renal transplantation (Aug 1, 1974), fever developed, and renal function deteriorated. Acute rejection of the transplanted kidney was confirmed by renal biopsy and was treated with high doses of steroids (up to 1 gm of prednisone per day), 500 mg of actinomycin intravenously and radiation therapy. Renal function continued to deteriorate, and the kidney was removed on Aug 7, 1974. Therapy with prednisone was tapered after surgery to 40 mg/day.

A chest roentgenogram (Fig 1A) on Aug 5, 1974 showed a homogeneous round density measuring 3.5 cm in diameter in the left lower lobe. The remaining lung was normal, and there was no associated pleural effusion or hilar adenopathy. The previous chest x-ray film of July 27, 1974 had been normal, except for cardiomegaly secondary to the patient's underlying renal disease. Tomographic studies (Fig 1B) confirmed a homogeneous well-circumscribed lesion without evidence of cavitation or air bronchograms. No other pulmonary lesions could be identified. The lesion persisted essentially unchanged over the next three weeks, and at no time could signs or symptoms referable to it be noted. Bronchial brushing of the nodule was performed on two separate occasions, but no pathogens were seen microscopically or were cultured.

The remainder of the hospital course was one of persistent deterioration in mental status, although the findings from extensive evaluation of the central nervous system were remarkable. The patient died during a seizure on the 42nd day after renal transplantation.
Pathologic Findings

At autopsy a single well-circumscribed zone of consolidation measuring approximately 5 cm in its greatest diameter was found to involve a portion of the medial, lateral, and posterior basal segments of the left lower lobe. The lesion was firm, airless, and focally hemorrhagic. Microscopically, the injury proved to be an organizing pneumonia of the interstitial variety. Many epithelial cells were conspicuously enlarged and possessed large nuclei with an amphophilic intranuclear inclusion and surrounding halo typical of cells infected with cytomegalovirus (Fig 2). Intracytoplasmic inclusions were also demonstrated. The interstitial reaction consisted of an inflammatory infiltrate of mostly lymphocytes, histiocytes, and fibroblasts. There was focal hemorrhage and necrosis. Organization, with some early collagen production, was present throughout the lesion. Cells infected with cytomegalovirus were not found in the right lung nor in the uninvolved portions of the left lung. A focal hepatitis secondary to cytomegaloviral involvement of the liver was present. There was no histologic or microbiologic evidence for bacterial or fungal involvement of the lungs.

The findings from postmortem examination of the brain were unremarkable, except for mild proliferation of astrocytic nuclei consistent with a metabolic encephalopathy.

Discussion

The radiographic diagnosis of infectious processes occurring in an immunologically compromised host is a difficult one at best. Frequently compounding the problem is the simultaneous presence of more than one invader. A recent review by Bragg and Janis stressed that the radiographic characteristics of an opportunistic infection can often suggest the specific etiologic agent. In this case the single nodule in the lower lobe raised the diagnostic possibilities of aspergillosis, nocardiosis, and phycomycosis; however, these lesions have a tendency to enlarge and to cavitate, and in addition, the patient is most often symptomatic.

The diagnosis of cytomegaloviral infection was not considered in this patient ante mortem because a solitary large pulmonary nodule secondary to infection with this virus has not been previously reported. Previously published reviews on the pathogenetic effects of this viral infection would lead one to expect a diffuse interstitial radiographic pattern, with or without a nodular component. In the case presented here, the radiographic density correlated well with the hemorrhage, necrosis, cellular infiltrate, and degree of interstitial organization seen microscopically. The cytopathic effects of cytomegalovirus found in the liver strongly suggest that the virus was pathogenic in this patient and that the pulmonary lesion was due solely to the effects of the virus. Further evidence exists in that no viral inclusions were found elsewhere in the lung, the lesion fits well within the spectrum of injury caused by cytomegalovirus, and there was no evidence for either bacterial or fungal pulmonary involvement. In retrospect, the history of renal transplantation, the paucity of symptoms relative to the chest, and knowledge of the pathogenetic effects of cytomegalovirus should have led to early consideration of this organism as the underlying etiologic agent, despite the unusual radiographic presentation.

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Paroxysmal Supraventricular Tachycardia with Unusual Induction*

Concealed Reentry or Automaticity?

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In a patient with documented paroxysmal supraventricular tachycardia, earlier atrial extrastimuli consistently induced His-ventricle (H-V) block and "atrioventricular junctional" beats, which were always followed by an echo or paroxysmal supraventricular tachycardia. In "atrioventricular junctional" beats, a V wave was preceded by an H deflection with the same interval as that of the sinus beat. As for the underlying mechanism of paroxysmal supraventricular tachycardia, two possibilities were considered: (1) concealed atrioventricular nodal reentry, and (2) "triggered automaticity."

Recent electrophysiologic studies have demonstrated that a common mechanism for the initiation of paroxysmal supraventricular tachycardia in man is atrioventricular nodal reentry. It has been suggested, on the other hand, that "triggered activity" may cause the types of cardiac arrhythmias that usually are attributed to reentry. It has been suggested, on the other hand, that "triggered activity" may cause the types of cardiac arrhythmias that usually are attributed to reentry.

This report describes electrophysiologic studies in a woman with a history of paroxysmal supraventricular tachycardia, as the underlying mechanism of which two possibilities were considered: (1) concealed atrioventricular nodal reentry, and (2) triggered automaticity.

METHODS

The patient was a 37-year-old woman with documented paroxysmal supraventricular tachycardia. She had atrial and ventricular septal defects (the former, ostium secundum defect and the latter, Roger's type). The defects were closed on Jan 8, 1974. The electrocardiogram of this patient at rest showed normal P-R intervals and an incomplete right bundle-branch block.

The patient was studied in a supine position and in a postabsorptive nonsedated state. She was not receiving medications at the time of the examination.

Electrophysiologic studies were performed, utilizing incremental and extrastimulus techniques with simultaneous electrocardiographic and His bundle recordings. Stimuli were delivered by a programmable digital impulse generator with a stimulation isolation unit (San-Ei Sokkai 3F-36).

The atrial, His bundle, and ventricular electrograms of the basic driving impulse were represented by A1, H1, and V1, respectively. The atrial, His bundle, and ventricular electrograms of the atrial test impulse were represented by A2, H2, and V2, respectively.

RESULTS

Electrophysiologic study during sinus rhythm showed P-A, atrio-His (A-H), and His-ventricle (H-V) intervals of 20, 90, and 36 msec, respectively. Rapid atrial pacing revealed progressive prolongation of the A-H interval (90 to 150 msec), resulting in Wenckebach's A-H block beyond a rate of 150 impulses per minute. The H-V interval remained constant.

Atrial extrastimuli were coupled at the driving cycle length of 780 msec. As A1-A2 intervals were decreased from 600 to 340 msec, A1-H2 intervals increased from 105 to 150 msec. At an A1-A2 interval of 330 msec, H-V block occurred with an A1-H1 interval of 160 msec. The breadth of the H1 deflection was slightly enlarged. At an A1-A2 interval of 320 msec, H-V block also occurred with an A2-H1 interval of 175 msec. In addition, a beat, which probably originated somewhere in the atrioventricular junction (i.e., an "atrioventricular junctional" beat), appeared in early diastole (Fig 1). In this beat, H deflection (H*) preceded the V wave with the interval of 36 msec, identical with that of sinus beats. An H2-H* interval was 450 msec (A1-H*, 625 msec). This beat was followed by two sinus beats; however, the P wave of the first sinus beat was considered to be atrial fusion because the high right atrial potential almost coincided in position with the low right atrial potential which was recorded on the His bundle electrogram. In consequence, retrograde conduction from the "atrioventricular junctional" beat was considered, the time interval between H* and the low right atrial potential (atrial fusion) being measured as 370 msec. At an A1-A2 interval of 300 msec, paroxysmal supraventricular tachycardia occurred, as seen in Figure 2. Similarly, H-V block occurred when the A2-H2 interval was 195 msec, and the "atrioventricular junctional" beat consistently appeared when the H2-H* interval was 480 msec (A2-H*, 635 msec). The P wave following this beat was of sinus origin because a high right atrial potential preceded a low right atrial potential by the same interval as...