Atrial Flutter with One-to-One Conduction

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A case of atrial flutter with one-to-one conduction is described, and the implication of digitalis and electrolyte shift is discussed. Cardioversion after diphenylhydantoin restored normal sinus rhythm, and appears the therapy of choice in this arrhythmia. The possible protective effect of diphenylhydantoin during cardioversion of digitalized patients is discussed.

INTRODUCTION

Atrial flutter with one-to-one A-V conduction is a rare occurrence.1 When atrial flutter is a manifestation of digitalis toxicity, it is usually associated with a high degree of atrioventricular block.2 A case of atrial flutter with 1:1 atrioventricular conduction is presented in which digitalis excess may be implicated.

CASE REPORT

The patient was a 44-year-old Negro man with a diagnosis of chronic glomerulonephritis established by renal biopsy in 1962. Over five years, he developed the nephrotic syndrome, hypertension and progressive uremia. He was rehospitalized with gastrointestinal symptoms and increasing uremia, with a creatinine of 14.2 mg/100 ml and a blood urea nitrogen (BUN) of 82 mg/100 ml. Three days after admission he suddenly became hypotensive with a blood pressure of 80/40, and a paradoxical pulse of 20 mm was elicited. A loud pericardial friction rub was also present. Fluoroscopy revealed an enlarged heart with decreased pulsations, and right heart catheterization demonstrated a right atrial pressure of 24 mm Hg and a right ventricular pressure of 70/24 with 20 mm variation with respiration. Atrial injection demonstrated a pericardial effusion, and 40 ml of serosanguinous fluid was removed on pericardiocentesis. During pericardiocentesis transient atrial flutter with 2:1 conduction occurred which spontaneously reverted to regular sinus rhythm.

The patient received 1.6 mg lanatoside C (Cedilanid) intravenously in divided doses over a 12-hour period. The following day peritoneal dialysis was begun, and 24 hours later the patient received a single 0.125 mg intramuscular injection of digoxin. Twelve hours later, while still being dialyzed, he developed a rapid supraventricular tachycardia at 240 beats per minute (Fig 1) and the blood pressure fell to 55/0. The heart rate initially did not slow with carotid massage and 500 mg diphenylhydantoin was given slowly, intravenously, with no effect. Carotid massage after DPH, however, slowed the ventricular rate transiently and flutter waves were seen (Fig 2). Electrolytes at this time showed a drop of the serum potassium predialysis from 6.8 to 4.3 mEq/L and a rise of the serum calcium from 3.8 to 5.5 mg percent. Despite strong suspicion that the rhythm was secondary to digitalis toxicity, because of the patient's poor clinical status, cardioversion with a 10 watt-second impulse restored normal sinus rhythm with immediate hemodynamic improvement (Fig 3). Approximately 90 minutes later he developed premature ventricular contractions which were abolished by 100 mg of intravenous diphenylhydantoin. Despite clinical and chemical improvement, the patient became septic four days later with a spiking temperature of 105.6, and with bilateral rales. Cultures returned after death showed Bacillus pyocyaneus and Klebsiella-Aerobacter resistant to penicillin, chloramphenicol and kanamycin, the drugs he had received prior to death. Autopsy revealed severe end-stage renal disease, hemorrhagic pericarditis, and acute bronchitis.

DISCUSSION

The occurrence of atrial flutter with one-to-one atrioventricular response is rare, approximately 60 cases having been reported in the world literature.4 It has been most frequently described in relationship to catecholamine release during exercise or emotional stress, the use of quinidine in atrial flutter with two-to-one block, and the use of atropine in atrial flutter or fibrillation.5,4 Finkelstein1 has listed the following electrocardiographic criteria for this arrhythmia: (1) an undulating baseline with waves at 225 to 315 beats per minute; (2) the presence of atrial flutter with a higher degree of atrioventricular block preceding or following the termination of this arrhythmia; and (3) response to digitalis with increasing block or atrial fibrillation. The present case meets the first two criteria, but because of the mode of therapy chosen, not the third. In addition, patient's response to carotid mas-

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Figure 1. Atrial flutter with 1:1 conduction at 240 beats per minute (lead 2).

Figure 2. Rhythm strip showing effect of carotid massage following diphenylhydantoin. Note the undulating baseline with flutter waves marked.
sage favors this diagnosis. In atrial flutter with one-to-one conduction, carotid massage is frequently unsuccessful in increasing atrioventricular block.1,4 Carotid sinus stimulation was initially ineffective but did result in slowing the ventricular rate after diphenylhydantoin. The increased sensitivity to carotid sinus stimulation produced by diphenylhydantoin has been noted by others.14

Atrial flutter has been described due to digitalis toxicity, but usually a high degree of atrioventricular block is present.2 However, several reports of one-to-one conduction in digitalized patients have been described.1,3 Unfortunately, the etiological relationship between digitalis and atrial flutter cannot be proved in this case. The urgency of the clinical situation required prompt treatment, and therefore, simple withdrawal of the glycoside was not possible. However, several factors would support the possibility of digitalis toxicity. The full digitalizing dose of 1.6 mg of lanatoside C (Cedilanid) was followed first by an intravenous infusion of calcium and then by peritoneal dialysis. During the dialysis, the serum potassium fell acutely from 6.8 to 4.3 mEq/L, and the serum calcium rose concomitantly from 3.8 to 5.5 mg/100 ml of serum. Case reports of digitalis-induced arrhythmias following peritoneal dialysis have recently appeared.7,8 The temporal relationships of these electrolyte shifts led us to conclude that the arrhythmia was related at least in part to digitalis. The previous brief episode of atrial flutter with two-to-one block during cardiac catheterization may have indicated a predisposition to this arrhythmia made overt by the subsequent interventions described.

Patients with atrial flutter with one-to-one conduction are almost invariably symptomatic, with circulatory collapse, syncope or congestive heart failure occasionally culminating in death.1,5-6 The classic recommended therapy is large doses of digitalis,1,5 but we were reluctant to pursue this therapy for the above-stated reasons. Quinidine may be used after digitalis is unsuccessful, but occasionally this results in resumption of one-to-one response.3 Diphenylhydantoin was utilized initially in the management of this arrhythmia for several reasons. This antiarrhythmic agent is particularly effective in treating arrhythmias caused by digitalis excess.8,11 Although it has not been particularly useful in con-

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REFERENCES

Primary Lymphosarcoma of Lung*

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One case of primary pulmonary lymphosarcoma is presented. The treatment is surgical as thoracotomy is necessary to establish the diagnosis. The tumor remains localized for a long time; the lymph nodes are very rarely involved and an adequate surgical resection carries a better prognosis than in primary bronchial carcinoma and lymphosarcoma of lymph nodes. Routine postoperative radiotherapy is not indicated.

Primary pulmonary lymphosarcoma is in fact very rare. Although in 7 percent to 30 percent of cases of malignant lymphoma, there is radiologic evidence of secondary lung involvement as one of the manifestations of the disease, the appearance of this neoplasm as a primary pulmonary lesion is a rare entity. Sugarbaker and Craven found only one instance of primary lung tumor in a series of 196 cases of histologically proved lymphosarcoma.

Beck and Regnis while adding their one case reviewed 14 cases of primary pulmonary lymphoma published up to 1951 by Churchill, Spatt and Grayzel, Maier, and Anlyan and Lovingood. In 1956, nine cases were reported from Mayo Clinic. Rose reviewing the literature and including his two personal cases recorded only 23 cases of primary lymphosarcoma of lung published up to 1957. By 1962, the number increased to about 50. In 1965, Papaionnnou and Watson summarized primary lymphoma of lung, recorded a total of 93 cases; 77 of these were lymphosarcoma and 16 reticulum cell sarcoma. In 1966, Ehrenstein added two and Crismer, Rabb, Dornbach, and Kraft reported one case of primary pulmonary lymphosarcoma.

To the 80 recorded to date, an account of clinical and pathologic features of one additional case is presented.

CASE REPORT

F.W., 62-year-old man, a known case of controlled diabetes mellitus, following inadequate recovery from an influenza attack was found to have an abnormal chest x-ray. He lost about 28 lbs. in weight over the period of two years, had little sputum, no respiratory symptoms and smoked about 15 cigarettes a day for 30 years. He was admitted to the hospital on Dec. 15, 1967.

Clinical examination revealed nothing abnormal except the presence of diffuse and smooth enlargement of thyroid gland with no symptoms and signs of toxicity. He admitted its being enlarged for the last 30 years. There was no finger clubbing, no enlargement of cervical or other accessible lymph nodes of the body and no evidence of either a distant primary or secondary metastasis. Liver and spleen were not palpable.

The chest x-ray film (Fig 1) showed a well-defined solitary rounded and homogeneous opacity situated in the region of the right middle lobe with no evidence of hilar or mediastinal lymph node enlargement. Hemoglobin, sedimentation rate, blood glucose, red and white cell counts and electrocardiogram were normal. Exfoliative cytology of the sputum did not show any abnormal cells. Bronchoscopy was normal.

Figure 1. Chest x-ray film showing well-defined solitary opacity in the region of right middle lobe with no enlargement of hilar and mediastinal lymph nodes.

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