

Wide Complex Tachycardia Due to Coexistent Supraventricular and Ventricular Tachycardia

Anne B. Curtis, MD, and Ken Grauer, MD

Gainesville, Florida

The differential diagnosis of wide complex tachycardias includes supraventricular tachycardia with aberrancy and ventricular tachycardia. Distinction between these two entities is important, since management of these conditions differs substantially. Differentiation can often be made by careful analysis of the 12-lead electrocardiogram (ECG), particularly if QRS morphology is characteristic or atrioventricular dissociation or fusion beats can be identified. If the diagnosis is still unclear from the 12-lead ECG, however, especially if management proves to be problematic, referral to a center that performs invasive electrophysiological studies may be appropriate.

Electrophysiological studies are performed by strategically positioning electrode catheters in the heart to record intracardiac electrical activity. Definitive diagnosis of the patient's arrhythmia is usually possible. The present report concerns a patient referred for management of a wide complex tachycardia of uncertain cause. Emergency electrophysiological testing enabled the diagnosis of coexistent atrial flutter and ventricular tachycardia to be made, and provided insight to treatment of the arrhythmia.

CASE REPORT

The patient was a 70-year-old man with a past history of three myocardial infarctions, heavy ethanol abuse, diabetes mellitus, and congestive heart failure. In the past, a ventricular demand pacemaker had been implanted for unclear reasons. He was admitted on this occasion with an acute myocardial infarction, having a positive creatine kinase (CK) MB fraction and a peak CK reading of 16.67 μ kat/L (1000 U/L). At the time of admission the patient was

in a hemodynamically stable wide complex tachycardia at a rate of 125 beats per minute. The rhythm was unresponsive to intravenous lidocaine, procainamide, and bretylium. Direct current cardioversion produced ventricular fibrillation, which was countershocked twice to yield a paced rhythm that quickly reverted to a wide complex tachycardia. An esophageal lead was placed, but no atrial activity could be recorded. An emergency echocardiogram suggested the presence of atrioventricular synchrony and was felt to be consistent with sinus tachycardia. Because managing the patient's tachycardia was difficult and the cause of the rhythm was unclear, the patient was referred to Shands Hospital at the University of Florida.

On transfer, the patient was still in a regular wide complex tachycardia at a rate of 125 beats per minute and had remained hemodynamically stable with a blood pressure of 120/80 mm Hg. He was resting comfortably without chest pain and in no acute distress. With the exception of bibasilar crackles, findings on physical examination were unremarkable. There were no cannon *a* waves to suggest atrioventricular dissociation.

Pertinent laboratory tests included a sodium of 132 mmol/L (132 mEq/L), potassium of 5.5 mmol/L (5.5 mEq/L), magnesium of 3.1 mmol/L (3.1 mEq/L), a total calcium of 2.17 mmol/L (8.7 mg/dL), blood urea nitrogen of 41.0 mmol/L (115 mg/dL), and creatinine of 239 μ mol/L (2.7 mg/dL). Arterial blood gases obtained on room air showed a pH of 7.4, a P_{CO_2} of 3.6 kPa (27 mm Hg), and a P_{O_2} of 14.1 kPa (106 mm Hg).

Hematocrit was 0.29 with a white cell count of 18.2×10^9 L. A chest radiograph showed a greatly enlarged cardiac silhouette with increased interstitial markings. The echocardiogram suggested a marked reduction in contractility, but no pericardial effusion.

The patient's 12-lead ECG at admission to Shands Hospital is shown in Figure 1. There is a regular wide complex tachycardia with a cycle length of 460 msec, corresponding to a heart rate of 125 beats per minute. Atrial activity is absent. The QRS complex is markedly prolonged, and manifests a bizarre morphology. Although the patient was alert and had remained hemodynamically stable, and al-

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From the Department of Medicine, University of Florida College of Medicine, Gainesville Veterans Administration Medical Center, and the Department of Community Health and Family Medicine, University of Florida College of Medicine, Gainesville. Requests for reprints should be addressed to Anne B. Curtis, MD, University of Florida, Box J-277, Gainesville, FL 32610.

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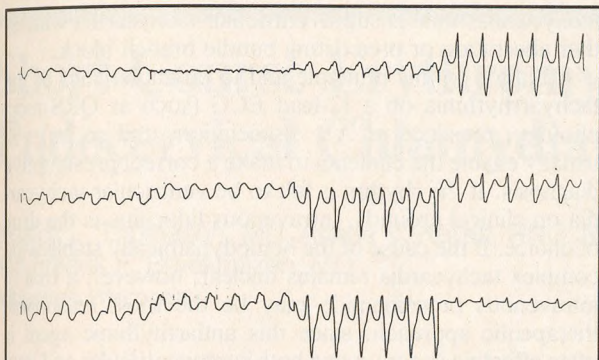


Figure 1. Admission ECG. There is a wide complex tachycardia at a rate of 125 beats per minute without identifiable atrial activity.

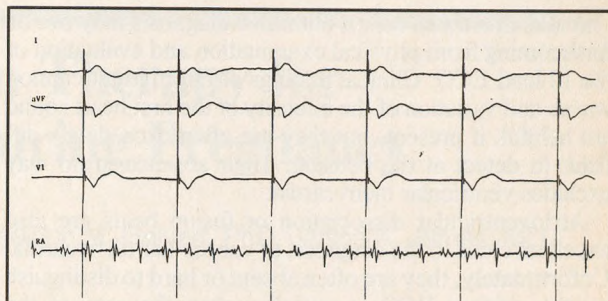


Figure 3. Additional recordings from electrophysiological studies. From top to bottom, ECG leads I, aVF, and V₁, and intracardiac recording from the right atrium. The patient's permanent pacemaker is seen pacing the ventricle at a rate of 72 beats per minute, while the atrial tachyarrhythmia continues unaffected at its own inherent rate.

though the echocardiogram findings suggested atrioventricular synchrony, the rhythm was felt to be ventricular tachycardia.

The patient was brought to the electrophysiological laboratory, where two quadripolar catheters were introduced transvenously into the right atrium and the right ventricle. The ventricular rhythm was regular with a cycle length of 480 msec (Figure 2). The atrial cycle length was approximately one half that of the ventricle but slightly irregular, varying from 220 to 280 msec. Although atrial flutter (with a slightly variable atrial cycle) with 2:1 atrioventricular conduction was considered, the fixed ventricular cycle length made this possibility unlikely. Failure to

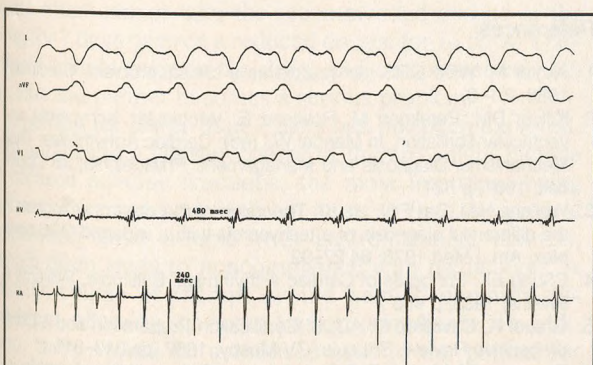


Figure 2. Recordings from electrophysiological study. From top to bottom are surface ECG leads I, aVF, and V₁, and intracardiac recordings from the right ventricle (RV) and right atrium (RA). At first glance, diagnosis of atrial flutter with aberrant conduction is suggested by the atrial rate, which appears to be twice that of the ventricular rate. Closer inspection shows the atrial rate to vary slightly while the ventricular rate remains quite constant.

demonstrate a His bundle deflection was further evidence in support of ventricular tachycardia.

In an attempt to provoke clear atrioventricular dissociation or higher grade atrioventricular block, a total of 5 mg of intravenous verapamil was given, and carotid sinus massage was performed. Neither intervention resulted in any change in the rhythm or the patient's clinical status. Ventricular burst pacing was then administered at cycle lengths from 400 to 280 msec. Following burst pacing at a cycle length of 280 msec, the ventricular tachycardia suddenly ceased and the rhythm reverted to demand ventricular pacing at a cycle length of 840 msec, corresponding to the patient's usual paced rate of 72 beats per minute (Figure 3). Atrial flutter continued with no change in the atrial cycle length, confirming that this rhythm had coexisted during ventricular tachycardia.

The patient went back into ventricular tachycardia less than 24 hours later. He was subsequently administered various combinations of quinidine, procainamide, lidocaine, and amiodarone with little success. Overdrive ventricular pacing was equally unsuccessful, and cardioversion was required on several occasions. Temporary control of his ventricular tachycardia was finally achieved with a combination of amiodarone and procainamide, but the patient's heart failure progressively worsened. Despite treatment with dobutamine, dopamine, and amrinone, he continued a downhill course, and died several weeks later.

DISCUSSION

This case illustrates several interesting points. It is an excellent example of how certain patients may remain alert and hemodynamically stable despite persistent ventricular tachycardia, especially when the rate of the ventricular response is not exceedingly rapid.

It also illustrates how a definitive diagnosis may not be forthcoming from physical examination and evaluation of the 12-lead ECG. Clinical findings of intermittent cannon waves and variation of the intensity of the first heart sound are helpful, if present, but they are often exceedingly difficult to detect at the bedside. Their absence in no way excludes ventricular tachycardia.

Atrioventricular dissociation or fusion beats are also excellent clues to the diagnosis of ventricular tachycardia. Unfortunately, they are often absent or hard to distinguish on the surface ECG, especially when the rate of the ventricular tachycardia is rapid.^{1,2} In this patient, even an esophageal lead failed to reveal atrial activity.

QRS width and morphology may provide additional clues to the cause of the arrhythmia. The QRS duration of 200 msec, in conjunction with the lack of a typical right bundle branch block pattern in lead V₁, and marked right axis deviation (Figure 1) are all consistent with a diagnosis of ventricular tachycardia.³⁻⁵ In view of the patient's history of cardiomyopathy and the unavailability of a previous ECG for comparison, however, this diagnosis could not be made with certainty.

Another presumptive factor supporting a diagnosis of ventricular tachycardia was that the patient had a permanent pacemaker. Information was lacking on the indication for pacing, and the incessant nature of the tachycardia made diagnosis of the underlying rhythm impossible. But if the pacemaker had been implanted for advanced atrioventricular block, a supraventricular tachyarrhythmia with so rapid a ventricular response would be practically impossible.

Finally, the patient's age and history of underlying heart disease strongly favored ventricular tachycardia.⁶ Nevertheless, what should have been appropriate medical treatment (ie, use of lidocaine, procainamide, and bretylium) did not convert the arrhythmia. Thus, despite a strong clinical suspicion that the rhythm was ventricular tachycardia, doubt remained until definitive evaluation in the electrophysiologic laboratory demonstrated coexistent atrial flutter and ventricular tachycardia.

Admittedly, in most non-tertiary-care centers, invasive electrophysiologic studies are not often both indicated and available for emergency diagnosis of a wide complex tachycardia. Cases such as this one illustrate the tremendous potential utility of this procedure in such situations.

Finally, this case illustrates the most important clinical principle in managing patients who present in a wide complex tachycardia of unknown cause: Ventricular tachycardia must always be assumed until proven otherwise regardless of the patient's physical appearance, hemodynamic status, or the amount of time that the rhythm has been sustained. The rhythm should be treated accordingly. It is important to emphasize that ventricular tachycardia is much more common as a cause of wide complex

tachycardias than is supraventricular tachycardia with either aberration or preexisting bundle branch block.

Attention to this principle and to characteristics of the tachyarrhythmia on a 12-lead ECG (such as QRS morphology, presence of AV dissociation, and so on) will usually enable the clinician to make a correct presumptive diagnosis. If the rhythm is felt to be ventricular tachycardia on clinical grounds, intravenous lidocaine is the drug of choice. If the cause of the hemodynamically stable wide complex tachycardia remains unclear, however, a trial of intravenous procainamide may be the most reasonable therapeutic approach, since this antiarrhythmic agent is often effective in converting both supraventricular and ventricular tachyarrhythmias.⁷ In most instances this approach will be successful. If it is not, and the tachyarrhythmia persists, cardioversion should be considered. As long as the patient remains hemodynamically stable, cardioversion can be performed under relatively controlled conditions. Thus, the patient can be sedated, an anesthesiologist can be called to the bedside to assist in ventilation, if needed, and lower energies (ie, 50 to 100 J) may be used. On the other hand, should hemodynamic compromise occur at any time during the process, immediate cardioversion is in order.

It was fortunate in this case that verapamil was not given at the bedside as a diagnostic and therapeutic trial. Unless one is certain that the tachyarrhythmia is supraventricular, use of verapamil is to be condemned, since intravenous administration of this drug to a patient with a stable wide complex tachycardia that turns out to be ventricular tachycardia is extremely likely to result in hemodynamic collapse and precipitation of ventricular fibrillation.^{8,9}

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Short-Course Erythromycin Therapy for Endocervical Chlamydia During Pregnancy

Jessica L. Thomason, MD, Arlene V. Kellett, RNC, NP, Sheldon M. Gelbart, PhD, Janine A. James, MD, and Fredrik F. Broekhuizen, MD

Milwaukee, Wisconsin

The recent recommendation by the Centers for Disease Control (CDC) for treatment of *Chlamydia trachomatis* in pregnant patients is unfortunately not entirely practical.¹ The CDC recommends erythromycin base, 500 mg orally four times a day for 7 days, but notes that this regimen is not readily tolerated. Recommended alternatives are erythromycin base, 250 mg orally four times a day for 14 days, or erythromycin ethylsuccinate, 800 mg orally four times a day for 7 days, or erythromycin ethylsuccinate 400 mg orally four times a day for 14 days; if the patient cannot tolerate erythromycin, amoxicillin can be tried, even though its efficacy is questionable. Erythromycin estolate is contraindicated during pregnancy, as drug-related hepatotoxicity can result, and tetracycline in any form is contraindicated because of congenital limb abnormalities, cataracts, tooth discoloration, dysplasia, and inhibition of bone growth in the fetus.² Clearly the nonpalatable form of erythromycin is the only proven therapy, but patients often cannot take the recommended dosage of the drug for 7 days or even a reduced dosage for 14 days. They simply cannot tolerate the drug, and compliance of both patient and partner becomes a serious problem.

Therapy for fewer than 7 days has not been explored. Advantages to a shorter course of therapy might include decreased adverse reactions, the most important being nausea. Pregnant patients even more so than the general population have trouble with gastrointestinal distress, which often leads to noncompliance with medication. A shorter course of therapy would help to diminish this problem, allowing for better patient compliance. In addition, there would be a reduced cost to the patient for shorter term therapy.

Since optimal dosage and duration of therapy for eryth-

romycin ethylsuccinate have not been established, a pilot study was undertaken to evaluate the possible efficacy of a 4-day course of therapy compared with the CDC recommendation of 7 days of therapy.

METHODS

At their first clinic visit, 57 consecutive pregnant patients who tested positive for *Chlamydia trachomatis* by enzyme immunoassay (Chlamydiazyme, Abbott Laboratories), but who were negative for all other sexually transmitted diseases, were recruited, and informed consent was obtained. Patients were alternately assigned to one of two treatment regimens. Patients and their sexual partners were to receive oral erythromycin ethylsuccinate 800 mg four times a day for either 4 days of therapy or 7 days of therapy. When 15 patients had been recruited for the control group (7 days of therapy), all future patients were assigned to receive 4 days of therapy. All patients received liquid medication and were retested by enzyme immunoassay for chlamydia antigen 3 weeks after completion of therapy.

Forty-two patients received the 4-day course, and 15 patients were treated for 7 days. The groups were similar with respect to age, weight, gravidity, parity, and mean weeks of gestation. Both groups had mean ages of 15.7 years. Weights were 137.8 pounds for the 4-day group and 134.1 pounds for the 7-day group. Both groups were made up almost entirely of first-time pregnant women. The mean weeks of gestation was 19.2 weeks for the 4-day group and 19.4 weeks for the 7-day group.

RESULTS

Of the 42 patients treated for 4 days, 29 were negative for chlamydia by immunoassay after 3 weeks, for a success rate of 69%. In the group of 15 patients receiving the standard 7-day course of therapy, the cure rate was 80% with 12 patients being negative by immunoassay at 3

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From the Department of Obstetrics and Gynecology, University of Wisconsin Medical School, Milwaukee. Requests for reprints should be addressed to Jessica L. Thomason, MD, Division of Gynecology, Sinai Samaritan Medical Center, PO Box 342, Milwaukee, WI 53201-0342.

weeks. Using Fisher's exact chi-square method with Yates' correction for small sample size, there was no statistically significant difference between the success rates of the two groups ($P = .72$).

Analysis of the treatment failures in both groups revealed that five patients in the 4-day group failed to complete their medication because of gastrointestinal upset, and five patients confided that their partners refused medication. Two patients in the 7-day group failed to complete their medication because of gastrointestinal distress, and one patient admitted to a different sexual partner who received no treatment.

DISCUSSION

This study shows that a 4-day regimen might be adequate and suggests that a larger patient trial comparing the two

regimens should be undertaken. It is apparent that compliance remains a problem even with the shorter duration of therapy. Also of significance is the high number of treatment failures because patients were unable to convince their partners to take medication. When both patients and all partners took medication as prescribed, there was a higher treatment success rate as indicated by negative immunoassay. More emphasis must be placed on treatment of sexual partners to effect better cure rates in pregnant patients.

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