Concealed Nonparoxysmal Junctional Tachycardia

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SUMMARY Electrophysiologic studies were performed in a patient who had an apparently uncomplicated complete trifascicular block. His bundle recordings revealed atrioventricular dissociation with: 1) an atrial rate of 58 beats/min, 2) an idioventricular escape rate of 45 beats/min, and 3) nonparoxysmal junctional tachycardia (His bundle rhythm) at a rate of 65−85 beats/min. The latter arrhythmia was electrocardiographically silent, influencing neither atrial nor ventricular events. The arrhythmia probably reflected digitalis intoxication (digoxin level of 3.3 ng/ml). A repeat electrophysiologic study 4 days after digoxin was discontinued revealed complete trifascicular block (distal to H) with intact conduction between the atrium and the His bundle (AH of 150 msec). Thus, electrophysiologic study demonstrated an electrocardiographically silent but clinically relevant arrhythmia, suggesting that His bundle recording should be part of diagnostic study during temporary pacemaker implantation in patients with atrioventricular block.

Recording of His Bundle electrograms may allow visualization of electrophysiologic findings that are invisible on the surface ECG. Examples include recording the HV intervals in patients with bifascicular block, demonstrating the site of atrioventricular (AV) block, and demonstrating pseudo AV block secondary to concealed His depolarizations.1-3 In these examples, the electrophysiologic demonstration of abnormalities by means of His bundle recordings may or may not be anticipated from surface electrocardiographic findings.1-4

In this report, we describe a major, clinically important arrhythmia that was unsuspected from the surface ECG. This arrhythmia was nonparoxysmal AV junctional tachycardia, concealed from the surface ECG because of simultaneous AV nodal and trifascicular block. The arrhythmia was secondary to digitalis intoxication.

Case Report

Clinical Summary

The patient was a 79-year-old male admitted to the University of Illinois Hospital on November 10, 1978 because of AV block and severe biventricular failure. The patient had a history of slow heart rate (undocumented) for 4 years. The patient had been on digoxin (0.25 mg/day), furosemide (40 mg/day) and intermittent potassium supplementation for approximately 3 years.

Electrophysiologic studies were performed and temporary transvenous pacing was instituted soon after admission. Digoxin and furosemide were discontinued immediately after the electrophysiologic study. Repeat electrophysiologic studies were performed on November 14, 1978 (see below), and a permanent transvenous pacemaker was implanted without difficulty on November 17, 1978 because of chronic established AV block.

The serum digoxin level was 3.3 ng/ml at admission, 2.3 ng/ml on November 11, and 1.1 ng/ml on November 12. Subsequent digoxin levels were in therapeutic range (on 0.125 mg digoxin daily). Serum potassium was 4.5 mEq/l on admission, with a pH of 7.43, a Pco2 of 55 and a Pco2 of 28.

Electrocardiographic Findings

The ECG at admission revealed AV block with complete AV dissociation (fig. 1). The atrial rate was approximately 65 beats/min and there was an idioventricular escape rhythm at rates varying from 28−45 beats/min. The escape rhythm was characterized by a left bundle branch block pattern, and there were occasional coupled premature ventricular depolarizations. The ECG was consistent with complete trifascicular block.

Electrophysiologic Studies

Electrophysiologic studies were performed on November 10, 1978, using standard catheter techniques, at the time of temporary pacemaker implantation. There was complete AV dissociation, reflecting AV block. The atrial rhythm (rate 58 beats/min) was characterized by P waves that were predominantly inverted in leads II and III, and biphasic in lead I (fig. 2). This rhythm was undisturbed by any other electrophysiologic events (see below). There was an

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idioventricular escape rhythm (rate 45 beats/min) characterized by wide QRS complexes with a left bundle branch block pattern (fig. 2). This escape rhythm was also unaffected by the timing of other electrophysiologic events (see below).

There was a third independent rhythm characterized by His bundle potentials occurring at a rate of 65–85 beats/min (predominantly 65–75 beats/min) (fig. 2). This rhythm did not affect the timing of atrial (P waves) or ventricular events (QRS complexes). The question arose whether the irregularity of this His bundle rhythm was influenced by atrial (AH conduction) or ventricular (VH conduction) events. The occurrence of shorter HH cycles did not relate identifiably to the timing of atrial complexes, nor was there any relationship between the short HH cycles and the timing of ventricular complexes. The possibility that the atria sporadically conducted to the His bundle could not be absolutely ruled out.

The possibility that the recorded His bundle potentials were artifactual was also explored. These potentials were recorded from an electrode position typical of that associated with recording of His bundle electrograms (electrodes in the AV position close to the tricuspid valve). Withdrawing the catheter to the atrium, or placing the electrodes within the ventricle totally extinguished the recorded potentials, suggesting strongly that these originated from the His bundle.

In summary, the patient had three independent rhythms: an atrial rhythm (visible), a ventricular escape rhythm (visible), and a His bundle tachycardia (invisible on the surface ECG). There were several sites of block: an antegrade site between the atrium and the His bundle (AV node) (block at this site could conceivably have been incomplete), and another antegrade site between the His bundle and the ventricles (trifascicular or distal His bundle); there were
corresponding retrograde sites of block between the ventricle and the His bundle (trifascicular), and between the His bundle and the atrium (AV node).

Our impression after the initial electrophysiologic study was that the patient had an established chronic complete trifascicular block, with concealed digitalis intoxication manifested by nonparoxysmal junctional tachycardia with AV nodal block.

Repeat electrophysiologic studies were performed on November 14. Again, we found complete AV dissociation due to AV block. There was sinus tachycardia at a rate of 120 beats/min, with biphasic P waves in leads II and III. Conduction was intact between the atrium and the His bundle, with a prolonged AH interval of 150 msec (fig. 3A). There was complete block distal to H, with an idioventricular escape rhythm (left bundle/branch block pattern at a rate of 46 beats/min) (fig. 3A). AV nodal Wenckebach periodicity was noted at an atrial paced rate of 150 beats/min (block proximal to H) (fig. 3B).

Discussion

Nonparoxysmal junctional tachycardia was described by Pick and Dominguez in 1957. This arrhythmia was characterized by junctional beating with a ventricular rate generally between 70-130 beats/min, and lack of both abrupt onset and termination. Nonparoxysmal junctional tachycardia usually occurred as a manifestation of digitalis intoxication, acute myocardial infarction, or acute rheumatic fever. In many cases of nonparoxysmal junctional tachycardia, there was retrograde AV nodal block (a combination of block and interference), and retrograde capture of the atria was not noted. In many cases, there was also complete or incomplete AV dissociation (antegrade), reflecting a combination of interference from retrograde penetration of junctional impulses to the AV node and depressed antegrade AV nodal conduction (frequently seen with digitalis intoxication and myocardial infarction). When secondary to digitalis intoxication, withholding of medication allowed ventricular rates to decrease, with reestablishment of intact AV conduction, and sinus rhythm with intact conduction was restored.

In some respects, the present case is a typical example of nonparoxysmal junctional tachycardia. The junctional rate was moderate (65-85 beats/min), digoxin levels were within the toxic range, and conversion of the arrhythmia was noted several days after stopping digoxin. Since the arrhythmia was diagnosed only on the basis of electrophysiologic study, the presence or absence of gradual onset and conversion could not be documented. However, no characteristics of the arrhythmia suggested paroxysmal reentrant junctional tachycardia.

The present case differed from previously described cases of nonparoxysmal junctional tachycardia.

Figure 3. A) Complete atioventricular (AV) block distal to H with an atrial rate of 120 beats/min, an AH interval of 150 msec and an idioventricular escape rhythm of 47 beats/min. B) Atrial pacing with AV nodal Wenckebach periodicity proximal to H at an atrial paced rate of 150 beats/min. There is persistent complete block distal to H. The time lines indicate 1 second, with a paper speed of 100 mm/sec. HBE = His bundle electrogram.
because of the prior existence of established complete trifascicular block. This totally prevented diagnosis of nonparoxysmal junctional tachycardia by means of the surface ECG. The diagnosis depended upon the His bundle electrograms, which allowed delineation of a junctional rhythm that was invisible on the surface ECG. The low atrial rhythm noted on admission was not considered a clue to the diagnosis.

The variation of HH cycle lengths in the present case was not totally explained. In patients with nonparoxysmal junctional tachycardia, the periodicity of regular RR intervals is often disturbed by: 1) ventricular captures if AV block is incomplete; 2) antegrade exit block from the AV junctional pacemaker producing Wenckebach periodicity or sudden dropped beats; or 3) resetting of the AV junctional pacemaker by premature depolarizations (concealed conduction). Of these possible mechanisms, the disturbance in regularity of His bundle complexes in the present case could have been related to capture of the His bundle by the atria. However, when the timing of A and H was scrutinized, no consistent pattern of captures could be delineated.

The question arises whether the delineation of concealed digitalis intoxication (concealed nonparoxysmal junctional tachycardia) was an electrophysiologic oddity or a clinically relevant event. In a previous report, Dreifus and co-workers noted that the continuation of digitalis or rapidly administered supplemental doses of digitalis in patients with nonparoxysmal junctional tachycardia resulted in a high mortality. In the present case, if cardiac glycosides had been continued (which is entirely possible because of the clinical diagnosis of congestive failure), the resulting arrhythmia could have been a more lethal manifestation of digitalis intoxication, such as ventricular tachycardia or ventricular fibrillation. Delineation of nonparoxysmal junctional tachycardia at the time of initial His bundle recording prompted us to draw a serum digoxin level — we cannot establish that this level would not have been drawn without the His bundle recording — and we immediately discontinued cardiac glycosides.

The demonstration of concealed digitalis intoxication in the present case is relevant to the clinical use of His bundle recording in patients with AV block. His bundle recording should be part of the diagnostic study at the time of temporary pacemaker implantation in patients with AV block. The recording of His bundle electrograms allows a delineation of the site of block (which frequently will be correctly anticipated from surface electrocardiographic findings), and occasionally of unanticipated electrophysiologic findings. One previously reported example of a possible unanticipated finding could be the demonstration of pseudo AV block due to concealed, nonpropagated His bundle premature depolarizations. Such an unanticipated finding could be an indication for treatment with antiarrhythmic drugs.

The present case is another example of unanticipated findings. Unanticipated findings at electrophysiologic study may or may not be clinically relevant. In the present case, they appeared to be relevant.

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