Useful Clinical Criteria for the Diagnosis of Ventricular Tachycardia

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Misdiagnosis occurs upon initial presentation to medical attention in a considerable number of patients referred for evaluation of wide QRS tachycardia. In order to improve diagnostic accuracy (ventricular versus supraventricular tachycardia), the answers to two key bedside questions were prospectively evaluated: (1) Had the patient experienced a prior myocardial infarction? (2) Did symptoms of tachyarrhythmia start only after the infarction? A patient presenting with a wide QRS tachycardia was considered to have ventricular tachycardia if he or she answered in the affirmative to both of these questions. Of 31 consecutive patients referred with electrocardiographically documented sustained wide QRS tachycardia that was reproduced in the electrophysiology laboratory, the diagnoses made when the patients first presented to medical attention were ventricular tachycardias in 17 patients and supraventricular tachycardias in 14 patients. Following electrophysiologic evaluation, 29 were diagnosed as having ventricular tachycardia and two as supraventricular tachycardia. If the diagnoses were made solely on the basis of responses to the bedside questions mentioned earlier, 28 of the 29 patients having a final diagnosis of ventricular tachycardia would have been correctly identified. It is concluded that the use of these two questions can be very helpful in improving the clinical diagnosis of ventricular tachycardia.

Despite availability of many electrocardiographic and clinical criteria for the diagnosis of sustained ventricular tachycardia [1–8], a patient presenting with this arrhythmia in an urgent setting is still frequently misdiagnosed and sometimes treated in an inappropriate manner with serious, deleterious consequences [9–11]. It would be quite helpful if clinical data, which can be readily obtainable at the time a patient presents with wide QRS tachycardia, could be used to enhance diagnostic accuracy. We prospectively evaluated the value of answers to two bedside questions in assigning a probability that a wide QRS (120 msec or more in duration) tachycardia is ventricular in origin. These two questions pertained to whether the patient had experienced a remote myocardial infarction in the past and whether symptoms of tachyarrhythmia had occurred only after infarction. A positive answer to these two inquiries in the presence of a sustained wide QRS tachycardia would indicate a high likelihood that the proper diagnosis is ventricular tachycardia. The purpose of this report is to demonstrate the usefulness of this clinical information in helping to arrive at an accurate diagnosis.

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Indeed, a number of vasoactive hormones have been shown to directly stimulate the release of ANP from cardiac atria in vitro [38,39] and could be responsible for high levels of ANP secretion independent of atrial stretch. In addition, there is the possibility of diminished degradation or clearance of ANP in cirrhotic patients with ascites, which remains to be evaluated.

Following PVS, there was an immediate rise in ANP in five of the six patients who demonstrated the typical hemodynamic responses that reflect a large intravascular volume load [2–6]. The only previous observation of the acute effect of PVS on ANP levels, which showed a similar marked rise after PVS, both in experimental ascitic dogs and in a cirrhotic patient, was reported recently in a letter to the editor [40]. In these patients, right atrial pressure and cardiac output increased, but mean arterial pressure did not change, indicating a fall in systemic vascular resistance. The rise in ANP that accompanied these hemodynamic changes was associated with an increase in urine cyclic guanosine monophosphate excretion, which serves as a marker for its biologic activity. That this rise was related to PVS and not a non-specific effect of anesthesia and surgery [41–43] in indicated by the failure of ANP levels to rise initially following induction of anesthesia and the abdominal incision in three patients.

The immediate renal consequences of PVS were a significant rise in urine volume and fall in urine osmolality without a change in plasma osmolality (Figures 2 and 3). It has been documented previously that arginine vasopressin levels do not fall during this acute period [6]. This prompt fall in urine osmolality is consistent with a direct antagonism of the action of arginine vasopressin by ANP at the level of collecting tubule, as has been demonstrated in the isolated perfused tubule preparation [28]. Whereas the fall in urine osmolality indicates that there was a change in collecting duct water handling, this resulted in no net change in solute-free water clearance, since both urine volume and osmolar clearance rose pari passu.

In five of the six patients who experienced a prompt rise in ANP, there was also an immediate significant natriuresis. For the entire group, the change in sodium excretion correlated with the change in ANP from preoperative values. However, the natriuresis appeared delayed in that peak sodium excretion did not occur until four hours, whereas both ANP and urine volume peaked at two hours.

The change in sodium excretion from preoperative values did not correlate with the change in creatinine clearance. Furthermore, we have previously documented that immediately after PVS, although there is a significant rise in renal plasma flow (as determined by para-aminohippurate clearance), there is no change in mean filtration fraction (creatinine clearance/para-aminohippurate clearance) [5]. These results, taken together, suggest that the acute natriuresis after PVS is not entirely mediated by changes in glomerular filtration or by associated adjustments in proximal peritubular capillary physical forces and are consistent with an additional direct tubular action of ANP.

Patient 6 was of particular interest in that the shunt did not appear to function properly until four to six hours after insertion. Only at six hours did cardiac output and right atrial pressure rise, accompanied by a fall in hematocrit and systemic vascular resistance (Figure 4). ANP remained unchanged from baseline valves until this time, when it rose progressively followed by a natriuresis and diuresis at six to eight hours.

In conclusion, the observed systemic vasodilatation together with failure of arterial blood pressure to fall plus diuresis and natriuresis, after PVS, are compatible with the combined effects of ANP and volume expansion, since in the absence of volume expansion, the blood pressure would be expected to drop. The demonstration that after PVS the acute immediate rise in right atrial pressure is associated with a rise in ANP suggests that in cirrhosis with refractory ascites, ANP is released appropriately in response to the acute volume load sustained immediately after PVS. However, despite the demonstration of an association between the rise in plasma ANP, urinary cyclic guanosine monophosphate, and a natriuresis and diuresis after PVS, conclusive evidence that ANP is responsible for these effects must await the development of a specific antagonist to ANP.

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fied. Statistically, there was an insignificant difference between final diagnoses and diagnoses arrived at through answers to the two clinical questions ($p = 0.5$).

In the 14 patients whose referring diagnoses were supraventricular tachycardia, all of whom had final diagnoses of ventricular tachycardia, the following treatments were used initially during acute presentation: carotid sinus massage, two patients; intravenous digoxin, two patients; propranolol, one patient; edrophonium chloride, one patient; and verapamil, seven patients. All seven patients receiving verapamil experienced acute hemodynamic decompensation. One was treated with the head-down position and intravenous fluids prior to spontaneous termination of the tachycardia. The remaining six required emergency direct current cardioversion to restore sinus rhythm.

COMMENTS

The results of this study indicate that answers to the two questions regarding a patient's past history would be very helpful in arriving at the correct diagnosis. In the referral patients described in this study, the sensitivity of this method for making the diagnosis of ventricular tachycardia was 97 percent. Since both patients who did not have ventricular tachycardia were correctly identified, the specificity was 100 percent. All patients presenting with a new wide QRS tachycardia who have had a previous myocardial infarction and denied any symptoms of tachycardia prior to infarct had ventricular tachycardia; that is, there were no false-positive results. There was only one false negative result, which occurred in a patient with ventricular tachycardia who had a myopathy without coronary disease. As the data herein indicate, misdiagnosis and consequent inappropriate treatment of these tachycardias continue to be quite common [9,10]. All patients in this report were hemodynamically stable during their tachycardia at the time they presented to medical attention. This factor may explain the high incidence of misdiagnosis [11]. Therefore, the availability of readily obtainable clinical criteria that could facilitate the diagnosis of ventricular tachycardia would obviously improve the therapeutic approach. The data presented here do suggest that patients meeting the just described criteria should be more prudently considered to have ventricular tachycardia and treated as such until definite proof to the contrary can be demonstrated. Treating such patients as if they had supraventricular tachycardia, especially with the use of verapamil, would frequently result in hemodynamic deterioration with potentially disastrous consequences.

Although the results of this study are quite striking, there are some limitations that are important to bear in mind. Patients included in this report were essentially a referral population sent for electrophysiologic evaluation and, therefore, may not be identical to patient populations seen by primary physicians in their offices or emergency rooms. Although that is not our impression from discussions with referring physicians, it is conceivable that patients presenting with a wide QRS tachycardia of supraventricular origin following myocardial infarctions were selectively not referred. Were they also to be referred, the number of false-positive diagnoses of ventricular tachycardia may be increased. Even so, when dealing with a potentially life-threatening problem such as ventricular tachycardia, one would settle for some increase in false-positive results if the tests were very sensitive. That is, one would rather err on the side of over-diagnosing ventricular tachycardia then risk mistreating it. This would allow for a more cautious approach to a patient's management until a firm diagnosis can be made.

The main reason there were so few false-negative diagnoses of ventricular tachycardia in this study was the relatively large prevalence of coronary disease as a cause of this arrhythmia in the general population. Other studies have revealed that a majority of patients (60 to 82 percent) presenting with malignant ventricular arrhythmias have underlying coronary disease [15–17]. If one only included patients with sustained monomorphic ventricular tachycardia that was inducible by programmed stimulation, the percentages would be even higher. Although not likely to occur in the immediate future, should the prevalence of coronary disease as a cause of ventricular tachycardia decrease significantly or should the prevalence of some other etiologic causes increase dramatically, the utility of these two questions as a test for ventricular tachycardia would probably diminish. Under these circumstances, one could conceivably improve the diagnostic value of the question regarding presence of myocardial infarction by including presence of dilated cardiomyopathies as well.

It is interesting to note the small number of patients in this study with final diagnoses of supraventricular tachycardia. Such a small number probably represents relative infrequency of supraventricular tachycardia presenting as a new, sustained wide QRS tachycardia. Patients who have experienced myocardial infarction are expected to have an increased incidence of atrial arrhythmias including atrial flutter and fibrillation. Yet, no patient in this series presented with these arrhythmias with new onset of aberrant conduction. This may be due to the fact that such patients are often receiving cardioactive drugs known to slow AV nodal conduction such as digitalis, beta blockers, or calcium channel blockers. With slowing of AV nodal conduction, sustained functional bundle branch block during supraventricular tachycardia is more difficult to achieve. Therefore, when supraventricular tachycardia occurs in this patient population, the QRS complex is likely to be narrow or at least similar to QRS morphology during sinus rhythm [8].

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