WPW Syndrome and Myocardial Infarction

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R. W. was a 41-year-old television repairman who complained of a smothery, choking feeling in the throat accompanied by pain radiating down the distribution of the left ulnar nerve. These attacks appeared to be brought on by physical or emotional stress and were relieved by sublingual nitroglycerine.

The past history revealed that the patient had experienced attacks of fluttering of the heart since 1938 lasting approximately 20 minutes. These could often be stopped by deep inspiration. The cardiovascular history was otherwise negative.

A resting ECG (A) revealed anomalous atrioventricular conduction (Wolff-Parkinson-White Syndrome). A second tracing was taken immediately following the rapid intravenous injection of atropine sulfate gr. 1/15. The resultant change to a nodal rhythm is seen in Figs. B and D which revealed a completely different pattern. The QRS interval has shortened from 0.12 second to 0.06 second in duration, and the T wave has become upright in leads 1 and aV1 and inverted in leads 2, 3, aVf, V5 and V6. This was felt to represent posterior and anterolateral ischemia.

The patient was put on medication and instructed to stop all work and remain in bed. This he refused to do. He was seen again on December 16, 1961 when he stated that he was having no difficulties unless he became emotionally upset. Figure C consists of a tracing taken on that date following intravenous atropine sulfate medication. T has become more deeply inverted in leads 2 and 3, and ST is now elevated in aVf where the R wave has disappeared. Although both tracings were taken by the author, the appearance of the V leads has changed suggesting clockwise rotation. The T waves in V2 through V4 are now tall and peaked probably on the basis of ischemia. The changes in aVf are strongly suggestive of an acute posterior infarction. Again the patient refused to stop working, and he was found dead in his car several days later.

Tamagna et al. have reported that the electrocardiographic diagnosis of myocardial infarction can be made in anomalous atrioventricular conduction if the infarct is large, and particularly if the right ventricle is prematurely activated. Small infarctions could not be diagnosed in the presence of this conduction defect. It was recommended that when myocardial infarction is suspected in this syndrome, an effort be made to convert the conduction pattern to a normal one so that a definite electrocardiographic diagnosis can be made. This is particularly true in that T waves are unstable in anomalous atrioventricular conduction, and the accompanying attacks of paroxysmal tachycardia can often cause substernal pain. Thus, in this case, the intravenous use of atropine sulfate was helpful in revealing the electrocardiographic changes consistent with myocardial ischemia and probably acute infarction. It might be added that the patient experienced no untoward symptoms from the injections of the atropine sulfate other than a slight "catching" sensation substernally, when the rhythm changed to a nodal rhythm, and a mild transient euphoria. This has been the author's experience in other such cases.

Reference


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