ACUTE MYOCARDIAL INFARCTION AND THE WOLFF-PARKINSON-WHITE SYNDROME

W.H. Ng

Department of Medicine
School of Medical Sciences
Universiti Sains Malaysia
Minden, Penang
MALAYSIA

W.H. Ng, MBBS, MRCP, AM
Assoc Professor

SYNOPSIS

The Wolff-Parkinson-White Syndrome may either simulate myocardial infarction or mask the electrocardiographic abnormalities of acute myocardial infarction. A case of the simultaneous occurrence of Wolff-Parkinson-White Syndrome and acute myocardial infarction in a 57 year old man is reported. Problems in the management of recurrent tachycardia and persistent cardiac failure are described.

INTRODUCTION

The association of the Wolff-Parkinson-White Sydrome with normal individuals and in those with congenital or acquired cardiac disorders is well established (1). Predisposition of such people with this electrocardiographic abnormality to supraventrcular tachyarrhythmias was first described in 1930 (2). When the Wolff-Parkinson-White Syndrome occurs in an individual with heart disease, interpretation of the electrocardiogram may be difficult. This is because the Syndrome may mask the underlying electrocardiographic abnormalities or may simulate other cardiac disorders. Acute myocardial infarction in a patient with the Wolff-Parkinson-White Syndrome was first reported in 1945 (3), and up to 1950 other reports that appeared on this association were in patients with inferior (posterior) infarction. Goldberg and Lewis (4) were the first to report the Wolff-Parkinson-White Syndrome in a patient with anterior infarction.

This is a case report of acute myocardial infarction and the Wolff-Parkinson-White Syndrome in a 57 year old man. Electrocardiographic changes are illustrated, and problems in the management of persistent cardiac failure and recurrent supraventricular tachycardia are described. As there has been no previous reports from this region of the uncommon simultaneous occurrence of this Syndrome with acute myocardial infarction, it was deemed pertinent to record this case.

CASE REPORT

A.W., a 57 year old Pakistani businessman, was admitted with severe chest pain associated with profuse sweating and dyspnoea. Prior to this, he was well with no past medical history of diabetes mellitus, hypertension, palpitations or angina pectoris. Physical examination on admission revealed an apprehensive, well built gentleman in pain and severe respiratory distress. The blood pressure was 110/70 mm. Hg. and the pulse rate was 106 per minute which was regular. Examination of the heart showed no cardiomegaly or any significant murmurs. A gallop rhythm was audible. Diffuse crepitations were detected in both lungs. The haemoglobin was 16 grammes %, total white count of 12,000 with a normal differential, and the erythrocyte sedimentation rate was 5mm. in the first hour. The peak serum glutamate oxaloacetate transaminase was 142 IU/liter, the lactic dehydrogenase was 226 IU/liter. Serum electrolytes and blood urea were normal. The fasting blood sugar was 86 mg. %. Electrocardiogram on admission showed a fresh antero-septal infarction (Fig. 1), and chest X-ray confirmed the clinical findings of acute pulmonary oedema.

Drug treatment instituted on admission with Morphine, Frusemide and Aminophylline intravenously produced good clinical response. Four hours after admission, he developed a supraventricular tachycardia with a heart rate of 155 per minute. Digoxin was prescribed and the supraventricular tachycardia aborted with Verapamil (Isoptin) intravenously. A repeat electrocardiogram on the next morning revealed typical electrocardiographic features of the Wolff-Parkinson-White Syndrome — Short PR interval (0.08 seconds), prolonged QRS complex (0.14 seconds) and initial slurring of the QRS complex (delta wave) — best seen in Leads V1-V4, II, III and AVF (Fig. 2). Loss of R wave in V1-3 and ST segment elevation in V1-3, I and AVL seen during the acute phase of infarction (Fig. 1) were no longer apparent.

During the next four days, he developed frequent episodes of supraventricular tachycardia with heart rates of 150 — 180 per minute requiring intermittent intravenous Verapamil. Control of cardiac failure was difficult because of the frequent episodes of tachycardia. Digoxin was then discontinued on the assumption that it could be contributory in the perpetuation of the tachyarrhythmias. However, frequency of recurrence of this arrhythmia remained unchanged despite the withdrawal of digoxin. On the eighth hospital day, he again developed supraventricular tachycardia with a heart rate of 190 per minute. He rapidly developed acute pulmonary oedema and lapsed into an unconscious state. Despite repeated doses of Verapamil, the tachycardia persisted.

Sinus rhythm was achieved only after intravenous Practolol (Eraldin). In the following two weeks, he remained fairly stable with occassional periods of supraventricular tachycardia each time responding to intavenous Practolol. Digoxin was reintroduced to control cardiac failure which persisted with fluctuating severity. Due to the recurrence of tachycardia which contributed to the persistence of cardiac failure, a beta-blocker, Metroprolol (Betaloc) was added cautiously. This produced a cessation of further episodes of supraventricular tachycardia and his clinical status improved gradually with subsidence of cardiac failure. Subsequent follow-up after discharge revealed that there had been no recurrence of tachycardia and cardiac failure. Other than occasional angina on effort readily relieved with Trinitrin, he was well. A repeat electrocardiogram done six months after hospital discharge showed normal atrioventricular conduction, old anterior infarction pattern with a left anterior hemiblock (Fig. 3).

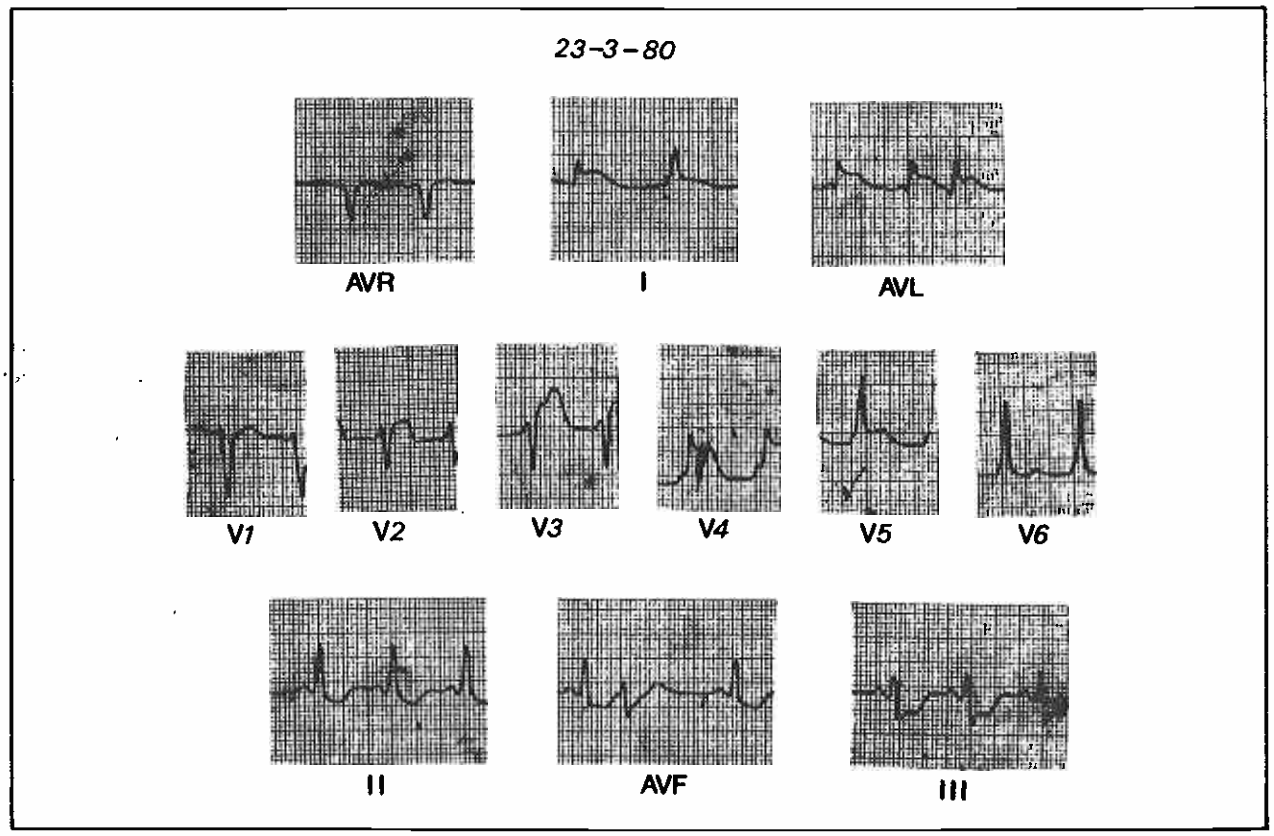

Fig. 1 Electrocardiogram on admission. Pattern of acute anterior infarction seen in Leads I, AVL and V1-V4

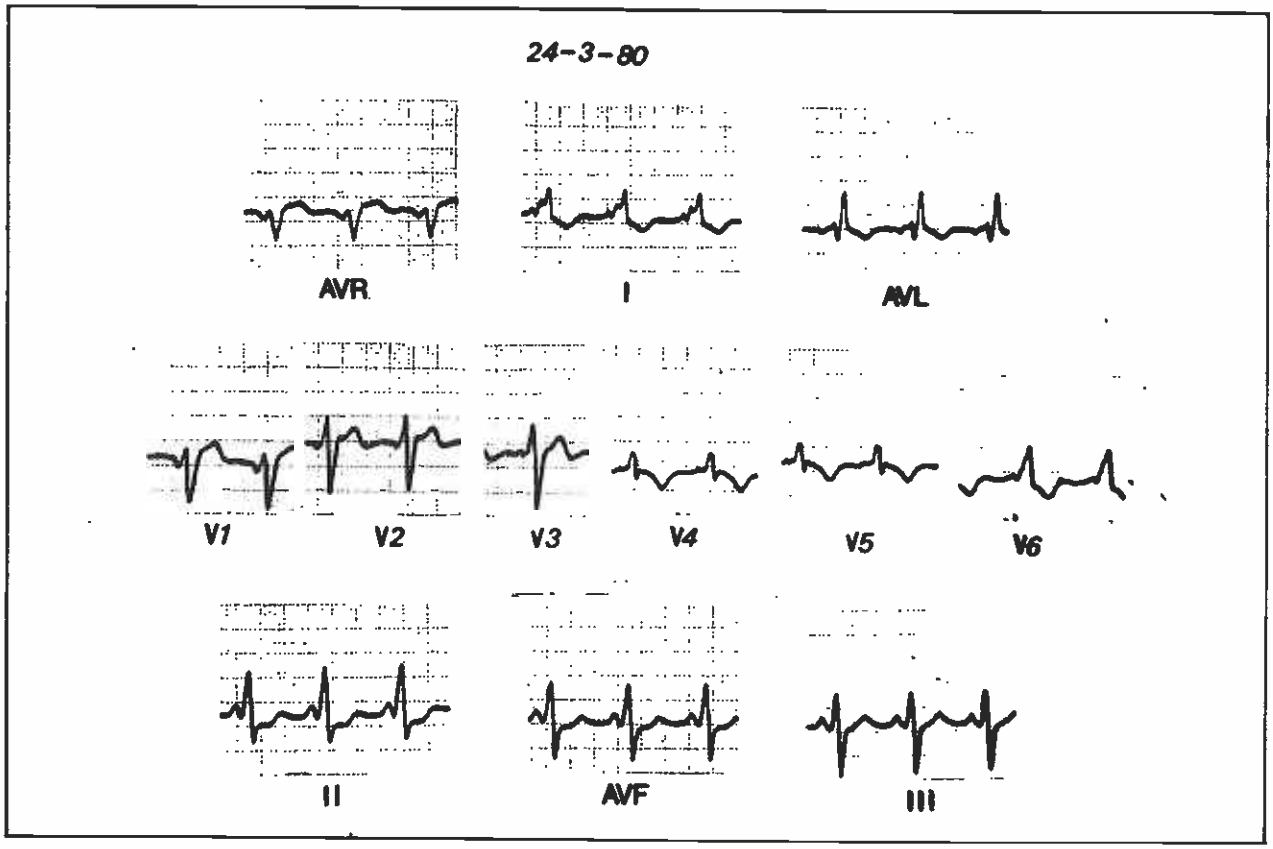

Fig. 2 Electrocardiogram one day after admission showing anomalous atrio-ventricular conduction especially in Leads II, III, AVF and V1-V3

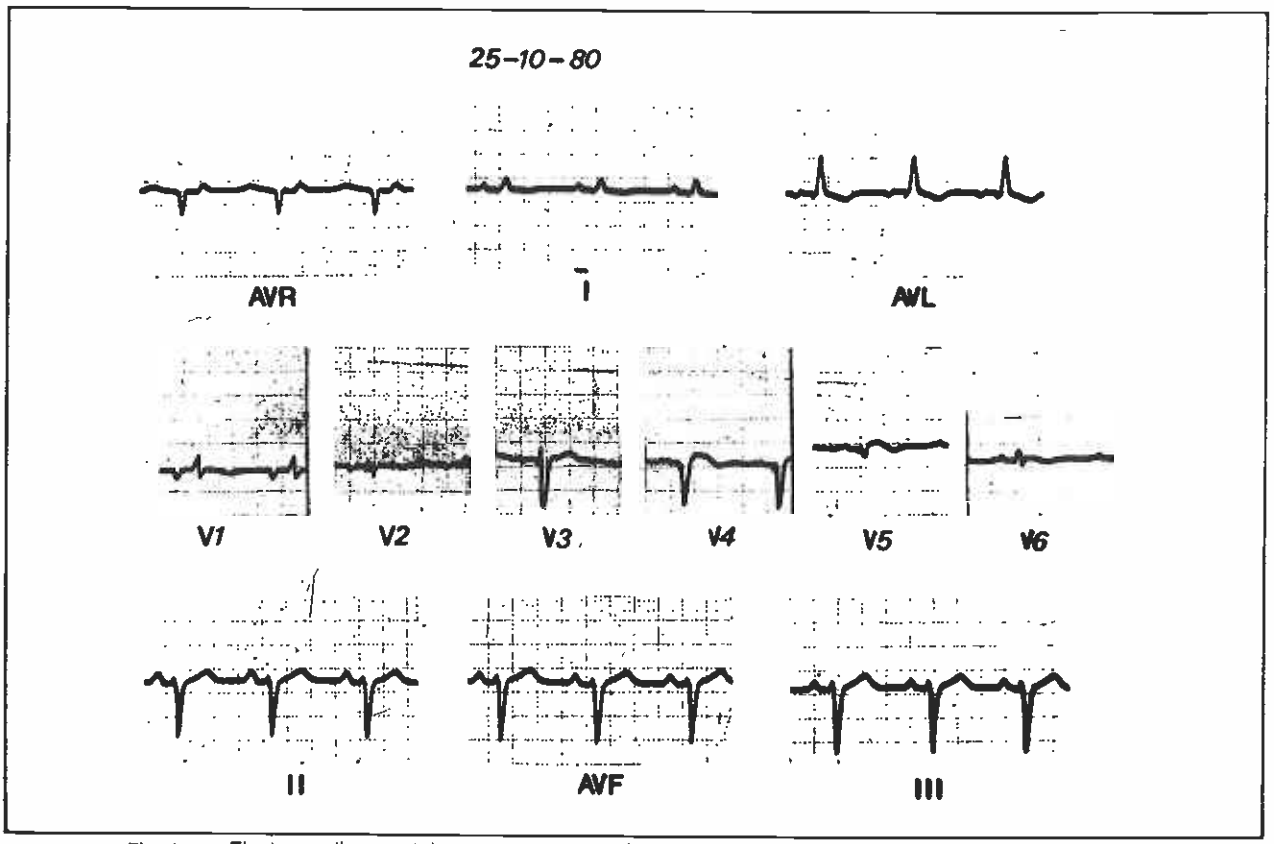

Fig. 3 Electrocardiogram taken seven months after admission showing normal atrio-ventricular conduction, old anterior infarction and left anterior hemiblock.

DISCUSSION

The commonest arrhythmia encountered in Wolff-Parkinson-White Syndrome is regular atrial tachycardia. Less commonly, atrial fibrillation or atrial flutter may occur (5). Atrio-ventricular conduction in Wolff-Parkinson-White Syndrome may be associated with a widened QRS complex which is normal in configuration, or with aberrant ventricular conduction resulting in bizzare, widened QRS complexes. The widened QRS complex may give rise to erroneous interpretation of the electrocardiogram as ventricular tachycardia (6) ventricular fibrillation (7) or simulate acute myocardial infarction (8.9). Simulation of myocardial infarction occurs because of the inscription of wide, deep and slurred Q or QS waves with secondary ST segment and T wave changes which usually characterise the 'electrical window' of myocardial necrosis. In most cases, the abnormal Q wave is seen in Leads II, III and AVF simulating inferior infarction, and less frequently in Leads 1, AVL and V1-3 simulating anterior infarction.

When acute myocardial infarction occurs in a patient with the Wolff-Parkinson-White Syndrome, a diagnosis of infarction electrocardiographically may be difficult, if not impossible (10.11.12). This is because pre-excitation conceals the initial QRS complex changes produced by necrosis thus masking or preventing the inscription of the pathological Q wave characteristic of transmural infarction. In addition, abnormalities of the ST segment and T wave, usually seen as part of the infarction pattern, is similarly seen in the Wolff-Parkinson-White Syndrome as secondary changes to the anomalous conduction. Diagnostic significance of these changes towards the diagnosis of myocardial infarction is therefore negligible. For a definite diagnosis of myocardial infarction to be made or excluded, it is vital to observe a normally conducted atrio-ventricular depolarization pattern in the electrocardiogram. This may arise spontaneously or by the abolition of pre-excitation by such measures as carotid sinus stimulation, parenteral administration of atropine, inhalation of amyl nitrite or with Quinidine (10).

This case highlights three important aspects of the simultaneous occurrence of the Wolff-Parkinson-White Syndrome and acute myocardial infarction. The masking effect of this Syndrome on the electrocardiogram is well demonstrated in Fig. 2. Classical anterior infarction pattern seen on admission (Fig. 1) changed significantly with the appearance of anomalous atrio-ventricular conduction. One is therefore unable to make an electrocardiographic diagnosis of infarction without both electrocardiograms for comparisons. When supraventricular tachycardia occurs in an otherwise normal heart, it may not be prognostically significant. However, its occurrence in myocardial infarction complicated by cardiac failure is very significant as the haemodynamic consequences of tachycardia on an already impaired pump will only aggravate cardiac failure and retard its control. Such was the situation with this case whereby the control of cardiac failure was both difficult and prolonged. Finally, in a clinical setting of acute infarction and cardiac failure, the choice of medications in the control of recurrent tachycardia is restricted and has potential risks. This is because drugs commonly used in the treatment of Wolff-Parkinson-White Syndrome have myocardial depressant properties with a tendency to precipitate or aggravate cardiac failure.

REFERENCES

1. Newman BJ, Donoso E, Friedberg CK: Arrhythmias in the Wolff-Parkinson-White Syndrome, Prog Cardiovasc Dis 1966; 9:147-65.
2. Wolff L, Parkinson J, White PD: Bundle branch block with short PR interval in healthy young people prone to paroxysmal tachycardia. Am Heart J 1930; 5:685-704.
3. Zoll PM, Sacks DR: Myocardial infarction superimposed on short PR, prolonged QRS complex; Case report. Am Heart J 1945;30:527-30.
4. Glodberg HH, Lewis SM: Acute myocardial infarction and the Wolff-Parkinson-White Syndrome. Am Heart J 1950;40:614-18.
6. Yahini JH, Zahari I, Neufeld HN: Paroxysmal atrial fibrillation in Wolff-Parkinson-White Syndrome simulating ventricular tachycardia. Am J Cardiol 1964;14:248-54.
7. Hermann Gr, Oates JR, Runge TM, Hejtmancik MR: Paroxysmal pseudoventricular tachycardia and pseudoventricular fibrillation in patients with accelerated atrioventricular conduction. Am Heart J 1957;53:541-61.
8. Eichert H: Wolff-Parkinson-White Syndrome simulating myocardial infarction. Ann Int Med 1944;21:907-12.
9. Kariv I: Wolff-Parkinson-White Syndrome simulating myocardial infarction. Am Heart J 1958;55:406-14.
10. Wolff L, Richman JL: The diagnosis of myocardial infarction in patients with anomalous atrio-ventricular excitation (Wolff-Parkinson-White Syndrome). Am Heart J 1953;45:545-70.
11. Grayzel J: Electrocardiographic criteria in the differential diagnosis of pre-excitation (Wolff-Parkinson-White Syndrome) and arteriosclerotic heart disease. N Engl J Med 1958;259:369-74.
12. Sodi-Pallares D, Cisneros F, Medrono GA, Bisteni A, Testelli MR, De Micheli A: Electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block, ventricular premature beats and Wolff-Parkinson-White Syndrome. Prog Cardiovasc Dis 1963;6:107-36.