Abstract:
Wolff-Parkinson-White (WPW) syndrome is a complex arrhythmic syndrome with numerous pathways and varied ECG presentations. ST segment elevation in lead aVR with diffuse ST depression in most other leads is an ECG marker of Left main coronary artery (LMCA) occlusion but can also occur in lesions involving the left anterior descending artery and some left circumflex artery occlusions and in some tachyarrhythmias. We present here a case of concealed WPW syndrome presenting with ECG changes resembling left main disease.

Keyword: WPW syndrome, LMCA disease, ST elevation, lead aVR

INTRODUCTION:
Wolff-Parkinson-White (WPW) syndrome is a complex arrhythmic syndrome with numerous pathways and varied ECG presentations. ST segment elevation in lead aVR with diffuse ST depression in most other leads is an ECG marker of Left main coronary artery (LMCA) occlusion but can also occur in lesions involving the left anterior descending artery and some left circumflex artery occlusions and in some tachyarrhythmias. We present here a case of concealed WPW syndrome presenting with ECG changes resembling left main disease.

CASE REPORT: A 54 year old male presented to coronary care unit with palpitations, left sided chest pain and shortness of breath of 2 hours duration. There was no complaints of orthopnea or syncope. Patient was a chronic smoker and alcoholic. He was not a known case of diabetes mellitus, hypertension, ischaemic heart disease or valvular heart disease. On examination, patient was conscious, oriented with PR-210/minute, regular and BP-100/70 mmHg; CVS- S1S2 heard without any murmurs. Other system examination was also normal.
ECG at presentation (Fig. 1) showed a narrow QRS, regular tachycardia with ventricular rate of 260/min, retrograde P-waves with short RP interval of 80 milliseconds and secondary ST-T changes suggestive of atrioventricular reciprocating tachycardia (AVRT).

Patient was treated with Inj. Adenosine IV 6mg followed by 12mg and sinus rhythm was established. ECG repeated after sinus rhythm (Fig. 2) showed HR-70/minute, ST elevation in lead aVR and diffuse ST depression in lead I, II, aVL, aVF and V3-V6 with primary ST-T changes in leads with ST depression.

In view of chest pain at presentation and above ECG changes, a strong suspicion of LMCA disease was made and patient was started on Inj.Heparin, antiplatelets drugs and verapamil to prevent recurrent SVT.

ECG was repeated again after 6 hours (Fig. 3) which showed HR-80/minute, sinus rhythm with normal QRS axis, and no ST-T changes. There was no evidence of delta waves, short PR interval.

His routine biochemical investigations and chest xray were normal. Troponin-T assay was negative (qualitative assay). Echo showed no regional wall motion abnormalities, normal left ventricular function with ejection fraction of 60%, aortic valve sclerosis with aortic stenosis (mild), aortic regurgitation (moderate), normal mitral valve with trivial tricuspid regurgitation.

In view of suspected LMCA disease, patient was taken up for coronary angiography (CAG) on the third day. His CAG showed normal coronaries with no evidence of LMCA or other coronary artery disease (Fig. 4-6).
Patient was asymptomatic during rest of his stay in the hospital and was discharged with antiarrhythmic drug (verapamil), statins and enalapril. Electrophysiological studies to localise the accessory pathway could not be done as the facility was not available in our institution, but the patient was advised regarding the need for electrophysiological testing to be done from higher centers where facility is readily available.

DISCUSSION:
WPW syndrome is a complex arrhythmic disorder with numerous accessory pathways (AP) and varied ECG presentations. The classical triad of WPW pattern in a resting ECG is short PR interval <120 milliseconds, abnormally wide QRS complex > 110 milliseconds with secondary ST-T changes, and delta wave. It is more common in males and majority (two-thirds) have no associated heart disease (1). It is also noted with higher frequency in patients with dilated and obstructive cardiomyopathies, mitral valve prolapsed, hyperthyroidism (2).
Concealed WPW syndrome is the most common variant form of WPW syndrome and occurs in about 20-30% of patients with APs. Here, the AP can conduct only in the retrograde direction with antegrade block in the AP resulting in absence of the WPW pattern on the surface ECG (3). Our patient also had normal ECG with no delta waves or short PR interval suggesting the presence of Concealed AP conduction.

In clinical electrocardiology, lead aVR was often a neglected lead until recent times. Yamaji et al (4) observed that aVR ST segment elevation greater than the ST segment elevation in V1 predicts acute left main coronary artery (LMCA) occlusion with a sensitivity of 81% and a specificity of 80%. Also, ST elevation > 1.5 mm in aVR, ST elevation in aVR plus aVL (>95% specific for LMCA disease) added to the specificity of diagnosing LMCA disease. These changes were associated with ST depression in most limb and precordial leads with maximal ST depression usually noted in V4-V6.

In LAD infarction, a high risk group with involvement of the basal part of the interventricular septum resulting from an occlusion site before the first septal perforator has previously been described (5). In patients with acute pulmonary embolism, acute right ventricular overload can present with ST elevation in aVR.

Identification of the presence and configuration of the P wave is of particular importance in the diagnosis of tachycardias. A dissociated negative P-wave in lead aVR is especially useful in the wide QRS tachycardia in diagnosing a ventricular origin of the arrhythmia. Also, during supraventricular tachycardia lead aVR is helpful in determining the site of origin of the tachycardia or the tachycardia pathway.

Ho et al (6) found that ST segment elevation in aVR during narrow QRS complex tachycardia suggests atrioventricular re-entry through an accessory pathway as the mechanism of the tachycardia. A total of 338 ECGs with narrow QRS tachycardia were analysed and they found that the prevalence of aVR ST-segment elevation was 71% for AVRT, 31% for AVNRT, and 16% for AT. However, after logistic regression analysis, they found that the sensitivity, specificity, and accuracy of aVR ST-segment elevation to differentiate AVRT from AVNRT and AT were 71%, 70%, and 70%, respectively. Among 117 episodes of AVRT with aVR ST-segment elevation, there were 76 (65%) left side, 23 (20%) right side, 14 (12%) posterior septal, and 4 (3%) antero- and mid-septal accessory pathways (p = 0.002). Finally they concluded that aVR ST-segment elevation during narrow QRS complex tachycardia favors the atrio-ventricular reentry through an accessory pathway as the mechanism of the tachycardia.

ST depression during SVT can occur both in AvnRT and in AVRT (manifest and/ or concealed pathway). This was shown by Riva et al, who showed that the presence of ST depression > 2 mm or T wave inversion, or both, during narrow QRS tachycardia suggests that AV reentry using an accessory pathway is the mechanism of the tachycardia. The phenomenon may be the consequence of a distinct pattern of retrograde atrial activation. Analysis of repolarization changes, especially ST depression, can guide preliminary localization of the accessory pathway even in the absence of ventricular preexcitation in the ECG during sinus rhythm (intermittent preexcitation, concealed accessory pathway) (7). There has been postulations as to why the ST-T changes are observed. Slavich et al suggested it was related to coronary artery spasm.
and went on to propose that echocardiographic examination during these ST-segment changes would be helpful (8). Zellweger et al showed that some degree of myocardial injury can occur during the episodes of tachycardia, which may also be associated with elevated troponin levels. The exact mechanism of injury however, remains unclear, but has been postulated to the increased demand during a relatively long period of tachycardia, combined with a reduction of oxygen supply to the myocardium due to shortened systole during tachycardia (9).

In our patient, the initial ECG presentation showed AVRT which later showed ST elevation in Avr with diffuse ST depression after sinus rhythm was restored making us strongly suspect LMCA occlusion but CAG showed normal coronaries. The absence of WPW pattern in the final ECGs taken were suggestive of a concealed AP pathology though confirmation needs further electrophysiological studies. So, the patient was treated symptomatically and discharged with antiarrhythmic drugs. This case report clearly shows that a high index of clinical suspicion must be maintained when interpreting ECGs in complex arrhythmias so that associated CAD is not missed. Also, it must be remembered that aVR – the forgotten lead – can be a useful tool in the diagnosis and prognosis of many clinical syndromes.

REFERENCES:


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