Electrocardiographic Diagnosis Of Critical Left Coronary Arterial System Stenosis

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Citation

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Abstract

Coronary angiography is undoubtedly the gold standard diagnostic tool for coronary artery stenosis. However, if we could diagnose or highly suspect non-invasively critical and proximal stenotic lesions in the left coronary arterial system, this could salvage more myocardium and minimize the morbidity and mortality rate. There is no signal noninvasive variable indicating the culprit lesion in the coronary artery tree. However, in the emergency room we correctly suspected the presence of tight proximal stenosis in the left anterior descending artery just by following serial resting electrocardiogram done during chest pain and after relief of the pain. This was suggestive of Wellen's syndrome. In another patient presenting with chest pain and normal resting ECG an exercise treadmill test (ETT) was done. This test gave us a good clue for the presence of significant left main coronary artery stenosis and highlighted the significance of ST-elevation during ETT. Coronary angiogram supported our suggestion in the two cases.

In view of the large area of the ventricle at risk, the recognition of such ECG patterns take on critical importance and provide important complementary information to that given by coronary angiogram and shorten the door-to-treat time.

CASE REPORT NO. 1 & REVIEW

A 55-year-old male Pakistani patient known to have hypertension and hyperlipidemia presented with typical anginal pain with no other symptoms. Electrocardiogram during chest pain showed sinus rhythm, normal axis, biphasic T-wave in V4-V5 minimal ST depression in V6. The pain was resolved then ECG was repeated and showed minimal ST elevation in V1 with inverted T wave in V2-V4, ST depression with inverted T in V5-V6 (fig. 1a,b).

Figure 1

Figure 1 A & B: ECG on arrival to the emergency room with typical anginal pain and ECG 30 minutes after pain relief



Half an hour later, chest pain recurred for a few minutes and then resolved. During the pain-free period, the ECG showed marked T-wave changes (fig.2a&b).

Figure 2

Figure 2 A & B: ECG on arrival to the CCU with typical anginal pain and ECG 30 minutes after pain relief



Echocardiography revealed normal left ventricular dimension, EF 58%, hypokinetic anterior and an apico-septal wall with diastolic dysfunction. 3 sets of cardiac enzymes were within normal range. Early coronary angiography revealed 95% proximal LAD stenosis with extensive clots (fig. 2c).

Figure 3

Figure 2c: coronary angiogram showed the tight stenotic lesion in the proximal left anterior descending coronary artery



The present case demonstrates the typical features of an interesting syndrome ($_1$). Between 1981 and 1989, Wellens and others described certain criteria by which critical stenosis high in the left anterior descending coronary artery could be diagnosed from specific ST-T changes on or shortly

after admission to the hospital (2, 3). Recognition of such a pattern and subsequent cardiac catheterization identifies the need for urgent intervention (CABG VS PTCA) for proximal LAD coronary artery stenosis. Such intervention would prevent the development of extensive anterior wall MI. In view of the large area of the ventricle at risk, the recognition of this ECG pattern takes on critical importance. The criteria to diagnose Wellens syndrome are summarized in table 1. The ST segment in leads V2 and V3 turns down into a negative T wave at an ST-T angle of 60 to 90 degree. During chest pain, these T-wave changes are replaced by positive T wave with either ST-segment elevation or depression. It is during this time that the coronary vessel is critically narrowed or occluded.

The T-wave inversion of Wellens Syndrome after pain relief represents reperfusion. Although leads V2 and V3 are the diagnostic leads for Wellens Syndrome, the T-wave inversion is not necessary limited to these leads. The location of the occlusion between the 1st and 2nd septal branches of LAD is typical but not essential; it can be more proximal with more widespread T-wave inversion

Figure 4

Table 1: Criteria of Wellen syndrome

Criteria for diagnosis	Comments
Chest pain with normal ECG	Represents critically narrowed or occludedvessel
or +ve T wave with either ST elevation or depression	
Progressive symmetrical deep	-Represents reperfusion
T-wave inversion in V2-V3 during	-not necessarily limited to V2-3
pain-free periods	
Little or no enzymes elevation	If not treated early→ extensive anterior MI
No loss of precordial R waves	
Tight proximal LAD stenosis	Typically between 1 st & 2 ^{sd} septal branches

CASE-REPORT NO. 2 & DISCUSSION

A 48-year-old Indian male patient with positive family history of CAD, non-diabetic, not hypertensive, and nonsmoker presented to the hospital. The patient had atypical chest pain for few days and subsequently was referred for exercise treadmill test (ETT). On the day of ETT, the patient was stable and his BP was130/80, pulse 70/min. His resting ECG was normal (Fig.3a). At the end of stage 1 he felt tired and developed mild chest pain with ST elevation in V2 – V4 (Fig3b). The test was stopped and the patient was given one tablet Nitroglycerin sublingual. In the recovery stage, there was no chest pain and ST elevation resolved within one minute. Then, the ECG showed ST depression in leads II, III, AVF, V5 and V6 (Fig.3c) that lasted for 5 minutes after that the ECG became completely normal (fig.3d) and the patient was admitted to the coronary care unit (CCU).

Figure 5

Figure 3a: normal resting ECG



Figure 6

Figure 3b: 2 min exercise ECG showed ST-elevation in leads V2-V4



Figure 7

Figure 3c: I min recovery stage ECG showed ST depression in leads1, 2,3,aVF, V5-6



Figure 8

Figure 3d: 5 min during recovery ECG showed normal ECG



In the CCU, the patient was stable with no chest pain and was started on Aspirin 300 mg ,Atenolol 50 mg and Isosorbid dinitrate 20mg tid orally and LMW fractionated heparin 60 mg subcutaneously bid. Two sets of CPK were normal and the Troponin T-test was negative. Serum cholesterol was 4.8 and triglyceride was 2.0. Coronary angiogram revealed a left main 80% stenosis distally, a left anterior descending totally occluded, a left circumflex 80% stenosis, and RCA distal lesion 60% (fig 4). The echocardiogram revealed left ventricular ejection fraction of 58% with hypokinesia of a small apical segment and mid and basal infrolateral wall. The patient underwent urgent CABG, which has been done uneventfully.

Figure 9

Figure 4: coronary angiography showed 80% LMCA, proximal LCX and total LAD stenosis



Patients with 3-vessel disease with significant LMCA stenosis had a 60% 4-year survival compared to a 70% 4-year survival if no LMCA disease ($_4$). Good exercise performance will indicate a better prognosis in patients with LMCA and/or 3-vessel disease than those with similar lesions but have poor exercise performance ($_{5,6}$).

Most of the patients with LMCA stenosis have extensive disease elsewhere in their coronary artery tree as well (7). It is not surprising that patients with left main and 3- vessel CAD have similar exercise performance but the difference in prognosis reflects the difference in mortality associated with a chance event such as plaque rupture with thrombus formation when it occurs in the left main coronary artery rather than elsewhere

The clinical indicators of LMCA stenosis are well recognized but still have a low sensitivity and low predictive value to be of diagnostic value ($_8$).

During ETT patients with LMCA disease tend to show an earlier onset, longer duration and more widespread STdepression than those with 3-vessel CAD do. However there is no single variable that help either to distinguish LMCA from 3-vessel CAD or to predict the presence of LMCA stenosis .The predictive accuracy of ETT findings can be increased to 74% if more than one variable are recognized (₉) such as development of down-sloping ST depression, ST- depression >2mm, onset of ST changes in stage 1, persistence of ST changes beyond 3 minutes, appearance of ST changes in at least three ECG leads or exertional STelevation ($_{10,11}$). Exercise-induced hypotension occurred in 33% of patients had at least 75% stenosis of LMCA($_{12}$). The duration of exercise-induced ischemia is extended longer in the recovery period in patients with LMCA stenosis ($_{13}$). The presence of coexistent RCA disease exerted a major adverse effect on ETT performance in patients with LMCA disease and these patients had higher incidence of ventricular arrhythmia, exertional hypotension and exertional ST elevation more than those with normal RCA ($_{14}$)

ST ELEVATION DURING ETT

ST elevation during ETT on top of normal resting ECG represents severe transmural ischemia as shown in table 2, it is more arrhythmogenic and might localize the culprit vessel (55677889910911912913914915916917918919920).

Figure 10

Table 2: ST-elevation during exercise test

On normal ECG	On old Q-MI
Indicates severe transmural ischemia	Indicate: wall motion abnormalities,
	LV aneurysm, or residual viability
May indicate variant angina	Not known
Very arrhythmogenic	less
Localize the affected artery	Less accurate
Very rare event(0.1-0.5)	Not rare

ST-elevation in non-Q areas is very rare event, and when it occurs purely during exercise phase it indicates more severe lesions than when occurring in recovery period of ETT $(_{21})$.

On the other hand, ST elevation in the presence of Q-waves represents wall motion abnormalities, LV aneurysm, or residual viable myocardium within an infarct. Exercise-induced ST elevation when tested two weeks after uncomplicated myocardial infarction indicated higher morbidity and mortality (22,23,24).

Our patient had a normal resting ECG. He developed chest pain and ST elevation in leads V2-4 in stage 1 which may indicate tight proximal left system stenosis ($_{10}$, $_{25,26}$, $_{27}$), then he developed ST- depression in the infrolateral leads which may indicate a lesion in RCA and Circumflex artery. It is not a reciprocal change because it appeared after normalization of ST elevation and lasted for 5 minutes. All those proposed lesions were confirmed by angiography with 80% LMCA stenosis

THE GOLD STANDARD DIAGNOSIS OF LMCA DISEASE

Coronary angiogram is the gold standard for diagnosis of LMCA stenosis. However, this technique is not without its shortcoming $(_{28,29})$. This limitation has been confirmed by presence of significant LMCA disease by intravascular ultrasound (IVUS) despite normal or insignificant angiographic appearance $(_{30,31})$.

The presence of LMCA disease portends a higher than normal risk during coronary angiography. There is a 0.55%mortality for angiography in the presence of a > 50% LMCA stenosis while the overall mortality for angiography is 0.05%(₃₂). This will demand preangiographic suspicious of the presence of LMCAD.

CONCLUSION

The diagnosis of ischemic heart disease needs collaboration of the history, clinical examination, and laboratory tests including noninvasive and invasive investigations. Serial ECG recording in emergency room after pain relief is of great value for patients presenting with typical anginal pain and normal ECG. It could give a clue for the culprit lesion and hence the urgency for intervention. Patients with LMCA stenosis cannot be detected on the basis of their clinical presentation alone which may not be different from those with milder CAD. Thus additional investigations are needed. Exercise treadmill test may suggest the presence of such lesion and other serious coronary artery disease (CAD) by giving a high index of suspicious. ST- elevation during exercise test in the absence of prior infarction should be taken seriously as a clue for the presence of LMCA stenosis or its equivalent.

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