Looking Behind the Block.

Basamad Z*.

*Assistant Professor, Department of Cardiology, University of Alberta, Edmonton, AL, Canada.

Correspondence: Dr. Z. Basamad, Assistant Professor, Department of Cardiology, University of Alberta, Edmonton, AL, Canada. Department of Cardiology, Edmonton General Hospital, Edmonton, AL, Canada, E-mail: z.basamad@gmail.com

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A 78-year-old man presents with a complaint of severe anterior chest pain which began suddenly one hour earlier. He gives a history of a heart attack several years ago, following which he had remained asymptomatic until today. His ECG is shown (Figure 1).

Figure 1, ECG on presentation
Diagnosis:
The ECG shows sinus rhythm with a slightly prolonged PR interval of 240 msec. The QRS complexes are abnormally wide at 150 msec with a left bundle branch block (LBBB) morphology. In most of the leads the ST segments and T waves are deviated in the opposite direction from the QRS complexes the expected finding in secondary repolarization changes due to the conduction abnormality. However, the ST segment are slightly elevated in lead III and markedly depressed in V2. These ST segment shifts are in the same direction as the main QRS deflection (concordant) and as such are not consistent with secondary changes due simply to abnormal depolarization. For practical purposes they have the same significance as they would in the absence of LBBB.

Figure 2, Fifteen-lead rhythm strip.

In a patient who presents with a clinical picture highly suspicious for acute MI, the presence of LBBB (whether new or old) may obscure the usual ST segment changes of transmural myocardial injury. Accordingly, administration of reperfusion therapy (thrombolysis or angioplasty) is usually considered appropriate. In this case, the presence of primary ST segment changes concordant with the QRS direction provides additional evidence that myocardial ischemia is present. In patients presenting with an acute coronary syndrome whose ECGs show ST segment depression in V1 to V3, the possibility of reciprocal change due to isolated posterior injury should be considered and a "15-lead" ECG obtained. The presence of ST segment elevation in the posterior leads should
prompt the administration of reperfusion therapy which otherwise might not be considered. A 15-lead ECG (Figure 2) was obtained in this case and demonstrated subtle ST segment elevation in V8 and V9, consistent with posterior wall injury. Subsequent coronary arteriography demonstrated proximal occlusion of a large, early, obtuse marginal branch of the circumflex artery.