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Understanding the Presence of Bundle Branch Block and Acute Myocardial Infarction:
Maybe not as Complicated as We Thought

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Understanding the Presence of Bundle Branch Block and Acute Myocardial Infarction: Maybe not as Complicated as We Thought

In the article “Relation of New Permanent Right or Left Bundle Branch Block on Short- and Long-term Mortality” the authors present a study of 5570 patients who were diagnosed with acute myocardial infarction (MI), of which 964 had documented bundle branch block (BBB) (either pre-existing, unknown, new-permanent, or new-transient). They reported that patients with BBB had higher 30-day and long-term mortality than patients without evidence of BBB, particular those with new-permanent right or left bundle branch blocks (1). New LBBB reportedly showed the greatest 1-year and long-term mortality amongst the groups. However, we wonder whether the presence of LBBB was actually the result of acute ischemia or whether its presence was entirely coincidental, reflecting the already diseased conduction system of these patients.

The fact that RBBB occurs far more frequently in the setting of acute myocardial infarction compared with LBBB has been established for several decades. A study published in 1970 by Norris et al describes a series of patients diagnosed with bundle branch block in the setting of acute MI. Autopsy findings revealed that the majority of patients discovered to have proximal LAD occlusion also had electrocardiographic evidence of RBBB prior to death. Those who had LBBB were more likely to have diffuse coronary atheroma and less likely to have primary LAD or left main occlusion on autopsy (2). Given what we know of the vast structure and coronary perfusion of the left bundle conduction system, it would conceivably require a catastrophic ischemic event across multiple coronary artery territories to knock-out this conduction pathway.

The authors in the present study admit that “new LBBB had an anterior location MI less frequently” which is consistent with the findings of previous studies. We would go further to propose that the development of “presumably new” LBBB, whether transient or permanent, in the setting of acute infarction in those patients had little, if anything to do with their ischemic event, and were solely a

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