In Syncope or Sudden Death from Coronary Artery Anomalies, Hypotension and Bradycardia are More Frequent than Ventricular Fibrillation

The case report by Garcia-Arribas and colleagues is a welcome addition to the literature on anomalous origin of the left coronary artery from the opposite sinus of Valsalva with an intramural aortic course (L-ACAOS-IM), a rare and often misunderstood condition. Severe coronary artery anomalies with an intramural course and proximal stenosis frequently lead to syncope during strenuous exertion, which can be the first clear sign of severe disease; less often, these anomalies cause sudden cardiac arrest or death. Some authors claim that syncope during exertion is frequent yet benign in children; however, when chest pain precedes and follows physical collapse, an ischemic event has probably occurred. Recurrent episodes strongly suggest a coronary artery anomaly of this kind. Because L-ACAOS-IM is the anomaly associated with the highest risk of sudden cardiac death, it is crucial that primary care physicians and cardiologists be aware of similar pathognomonic presentations.

Questions are continually raised about the mechanisms of syncope, sudden cardiac arrest, and sudden cardiac death. Some authors maintain that ventricular tachycardia or ventricular fibrillation (VF) is the mechanism and that existing ischemic scars are the cause. Despite these assumptions, recent field experience suggests that episodic global left ventricular ischemia is the cause and that low cardiac output with bradycardia (up to asystole) is the mechanism leading to physical collapse. Chiefly supporting this theory is the clinical pattern of severe exertion with chest pain before and after physical collapse. Sudden-onset VF is less likely to be accompanied by chest pain and electrocardiographic evidence of ischemia; it typically begins abruptly, and it can be interrupted only by resuscitation that includes defibrillation. On the contrary, as in the case presented by Garcia-Arribas and colleagues, troponin elevation and electrocardiographic changes are consistent with L-ACAOS-IM–related transient ischemia and are frequently accompanied by hypotension, bradycardia, and transient left ventricular systolic impairment. Residual myocardial stunning is often involved after critical left main coronary artery ischemic episodes.

In addition, this and similar cases indicate that, absent spontaneous recovery, prompt external massage resuscitation on the playing field often resolves the crisis without electric-shock cardioversion—evidence that VF was not the initial culprit. Reperfusion arrhythmias, including VF, are not unusual in cases of prolonged or delayed resuscitation, especially when adrenergic stimulation is applied during resuscitation to treat low cardiac output that does not resolve.

Advanced imaging (typically cardiac magnetic resonance imaging or computed tomography) is often necessary to confirm a general diagnosis of congenital coronary anomaly. Various diagnostic situations and alternative imaging techniques were recently evaluated in cases similar to that presented by Garcia-Arribas and colleagues, who used clinical evidence—a strong factor in favor of intervention—supported by computed tomography and echocardiography, to determine their treatment plan. Although we agree that these tests can prove the presence of L-ACAOS-IM, they cannot prove the severity of intramural stenosis and its detailed anatomy; in most cases, this will require intravascular ultrasonography.
References


