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Perspective

A guide to the Different Types of Congenital Coronary Artery Abnormalities

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DESCRIPTION

Congenital Coronary Artery Abnormalities (CAA) is a group of defects that appear at birth and can have a wide range of clinical presentations and severity levels. CAA may be discovered by chance during cardiac imaging or autopsy in patients who are asymptomatic. Ischemia-related signs and symptoms, which can lead to an elevated risk of Sudden Cardiac Death (SCD), might also appear as the initial symptom. We cover the normal morphology of the Coronary Arteries (CA) and the pathology of CAA at risk of SCD in this chapter, as well as our experience with SCD victims in the young population (under 40 years) and sports.

Coronary Arteries anatomy at a glance (CA)

The right (RCA) and left main (LCA), the latter branching into the Left Circumflex Artery (LCA) and the Left Anterior Descending Artery (LADA), arise normally from the aortic right anterior and left anterior sinuses of Valsalva, respectively, close to the sino-tubular junction, with no relation to the pulmonary trunk. The independent origins of the conal artery and the RCA from the right coronary sinus, as well as the LCX and the LAD from the left coronary sinus, are deviations from normal anatomy. When compared to the typical site at the sino-tubular junction, a coronary ostium can emerge from a higher position, up to 2.5 mm at most. Coronary dominance (right, left, or co-dominant circulation) is another deviation from the norm.

Anomaly of the Coronary Arteries (CAA)

CAA is an uncommon illness that affects about 1% of the general population, according to coronary imaging techniques and autopsy findings. CAA can cause myocardial ischemia, which can lead to Sudden Cardiac Death (SCD), even in young people and sportsmen. CAA has been classified in a number of ways. Angelina classified coronary anomalies into three categories based on anatomical features: anomalies of origin and course, anomalies of intrinsic CA architecture, and anomalies of coronary termination. While anomalies of origin and course will be explored in depth due to their possible link to SCD, anomalies of intrinsic CA anatomy and termination will be discussed briefly. Ostial stenosis/atresia and hypoplasia are examples of the latter.

Origin from the pulmonary artery of CA

Bland-White-Garland syndrome is a rare but potentially fatal CAA characterized by a reverse flow into the pulmonary artery and an abnormal origin of the LCA from the pulmonary artery. The RCA has a less common abnormal origin from the pulmonary artery, known as ARCAPA. Most individuals develop myocardial ischemia and heart failure in infancy and die within the first year of life if they are not treated and detected. The flow in the LCA actually decreases when the pulmonary vascular resistances diminish physiologically. Some patients, on the other hand, may stay asymptomatic and live into adulthood. We distinguish two forms of ALCAPA based on collateral vessel development: "adult type" with well-developed collaterals and "infant type" with inadequate collaterals and early onset of ALCAPA.

Myocardial connector of CA

When dealing with the inadvertent detection of myocardial bridge by angiography ("milking effect") and/or CT, the key problem is once again functional assessment for decision making. In certain patients, stress single-photon emission CT can reveal reversible myocardial perfusion abnormalities, with a connection between ischemia and systolic luminal narrowing. Physiological measures of the heart during pharmaceutical infusion are also beneficial.

The "half-moon" sign, an echolucent region between the bridging coronary segment and epicardial tissue that persists throughout the cardiac cycle, can be seen by IVUS imaging. However, there is no unanimity on whether additional diagnostic investigations of myocardial bridge are needed before medication, whether in symptomatic individuals or those with an "incidental" finding by angiography or CT.

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