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9.13. Chagas Disease

Key Points

- The pathogenesis of ChD is poorly understood but often results in an inferolateral LV aneurysm.
- The arrhythmogenic substrate is located intramurally and on the epicardial surface, often necessitating an epicardial ablation procedure.

9.13.1. Chagas Disease

ChD is a chronic parasitosis affecting the heart and other organs and is caused by the protozoan *Trypanosoma cruzi*. ChD is transmitted to humans mainly through parasite-laden feces from a hematophagous insect vector found only in the Americas, where the disease is considered endemic. The World Health Organization estimates a prevalence of ChD ranging from 1%–6% in endemic areas, with 10 million people infected worldwide (mostly in Latin American countries), 100 million at risk of infection, and 300,000 new cases reported each year. The World Health Organization estimates that 50,000 CCM-related deaths occur annually, with 60%, 25%, and 15% related to sudden cardiac death, progressive heart failure, and stroke, respectively (S9.13.3.1,S9.13.3.2).

Cardiac involvement is the most frequent and serious manifestation of chronic ChD. Although the pathogenesis of cardiac damage is complex and not completely understood, at least 4 possible mechanisms have been suggested:

cardiac parasympathetic neuronal depopulation, immune-mediated myocardial injury, parasite persistence in cardiac tissue with secondary antigenic stimulation, and coronary microvascular abnormalities causing myocardial ischemia. Diffuse arteriolar dilatation found in CCM can result in a coronary steal phenomenon of blood flow from epicardial coronary arteries to dilated arterioles, producing low perfusion pressure in the distal microvasculature supplying susceptible areas distal to the coronary branches. The watershed areas between main coronary artery branches would be the most susceptible to this steal phenomenon, causing low perfusion pressure, secondary ischemia, microinfarctions, and reparative fibrosis. The most frequently observed areas of myocardial scarring in CCM are the posterolateral LV and the LV apex in the watershed zones between the right coronary and circumflex arteries, and the anterior descending and posterior descending coronary arteries, respectively. VT is typically related to an inferolateral LV aneurysm (S9.13.3.2).

9.13.2. Ventricular Tachycardia in Chagas Cardiomyopathy

Sustained VT is usually due to reentry, associated with an inferolateral LV scar in over 70% of patients. Occasionally, endocardial radiofrequency ablation can result in transmural injury, effectively treating all portions of the myocardium involved in the arrhythmia circuit. However, scars commonly exist intramurally and/or subepicardially in an area with an associated thick layer of subendocardial myocardium. Mapping and ablation from the epicardial surface is performed in up to 40% of patients (S9.13.3.3,S9.13.3.4). A myocardial isthmus of surviving tissue between the inferolateral LV scar and the mitral valve annulus can be involved in a macroreentrant submitral circuit, as has been described for postinfarction VTs (S9.13.3.5). BBR can occur but is unusual (S9.13.3.6).

In CCM that presents with VT storms, cardiac sympathetic denervation (CSD) can reduce VT episodes and is a potentially valuable treatment option (S9.13.3.7).

9.13.3. Epicardial Ablation of Sustained Ventricular Tachycardia in Chagas Heart Disease

The prevalence of epicardial VT origins in patients with CCM is high (approximately 37%). Electrograms obtained during epicardial mapping from the areas giving rise to VT are similar to those observed for other scar-related VTs, including delayed potentials, mid-diastolic potentials, and continuous electrical activity during VT. The critical isthmus of the reentrant circuit can be confirmed by entrainment maneuvers or interruption of VT with RF ablation. With the progressive nature of the disease, VT recurrences are common (S9.13.3.4,S9.13.3.8).

References

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