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COMPLICATIONS OF ISCHEMIC HEART DISEASE

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Ischemic heart disease often leads to a cascade of downstream problems once myocardial blood flow is compromised. One of the earliest and most frequent sequelae is the development of electrical instability within the heart. As ischemic or infarcted tissue alters the normal conduction pathways, patients become prone to arrhythmias-ranging from benign premature beats to life-threatening ventricular tachycardia or fibrillation. These ventricular arrhythmias are the most common cause of sudden cardiac death in IHD, while atrial fibrillation may arise from atrial stretch due to elevated filling pressures or scarring.

Progressive loss of viable myocardium also impairs the heart's pumping ability. Left ventricular dysfunction initially manifests as exertional breathlessness and fatigue; over time, chronic systolic and diastolic dysfunction culminate in overt heart failure. Patients describe increasing dyspnea on minimal exertion or when lying flat, and physical findings may include pulmonary crackles, jugular venous distension, and peripheral edema. Neurohormonal activation (renin-angiotensin-aldosterone system, sympathetic nervous system) perpetuates remodeling, leading to chamber dilation, wall thinning, and worsening function. Although less common today with early reperfusion, mechanical complications of myocardial infarction still occur and carry high mortality. Rupture of the interventricular septum produces a harsh holosystolic murmur with acute hemodynamic collapse; papillary muscle rupture leads to severe mitral regurgitation, acute pulmonary edema, and hypotension; free-wall rupture results in hemopericardium and tamponade, often causing sudden death. Ventricular aneurysm formation during the healing phase may lead to persistent heart failure, thrombus formation, or recurrent arrhythmias. Inflammatory responses following infarction can give rise to pericarditis, which typically presents days after the event with pleuritic chest pain relieved by leaning forward and a friction rub on auscultation. Dressler's syndrome, an autoimmune pericarditis occurring weeks later, adds fever, leukocytosis, and pericardial effusion to the picture.

Finally, chronic ischemia promotes progressive atherosclerosis and endothelial dysfunction in other vascular beds, increasing the risk of stroke, peripheral arterial disease, and renal impairment. Thus, ischemic heart disease rarely exists in isolation; its complications span electrical, mechanical, inflammatory, and systemic vascular domains, underscoring the need for comprehensive surveillance and multi-modal management.

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