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Titel des Beitrags: Prognostic value of myocardial ischemia and necrosis in depressed left ventricular function: a multicenter stress cardiac magnetic resonance registry.

Abstract: The incremental prognostic value of inducible myocardial ischemia over necrosis derived by stress cardiac magnetic resonance in depressed left ventricular function is unknown. We determined the prognostic value of necrosis and ischemia in patients with depressed left ventricular function referred for dipyridamole stress perfusion magnetic resonance. In a multicenter registry using stress magnetic resonance, the presence (>= 2 segments) of late enhancement and perfusion defects and their association with major events (cardiac death and nonfatal infarction) was determined. In 391 patients, perfusion defect or late enhancement were present in 224 (57%) and 237 (61%). During follow-up (median, 96 weeks), 47 major events (12%) occurred: 25 cardiac deaths and 22 myocardial infarctions. Patients with major events displayed a larger extent of perfusion defects (6 segments vs 3 segments; P < 0.001) but not late enhancement (5 segments vs 3 segments; P = 0.1). Major event rate was significantly higher in the presence of perfusion defects (17% vs 5%; P = 0.0005) but not of late enhancement (14% vs 9%; P = 0.1). Patients were categorized into 4 groups: absence of perfusion defect and absence of late enhancement (n = 124), presence of late enhancement and absence of perfusion defect (n = ...
presence of perfusion defect and presence of late enhancement (n = 195), absence of late enhancement and presence of perfusion defect (n = 29). Event rate was 5%, 7%, 16%, and 24%, respectively (P for trend = .003). In a multivariate regression model, only perfusion defect (hazard ratio = 2.86; 95% confidence interval, 1.37-5.95; P = .002) but not late enhancement (hazard ratio = 1.70; 95% confidence interval, 0.90-3.22; P = .105) predicted events. In depressed left ventricular function, the presence of inducible ischemia is the strongest predictor of major events.