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Enhancing primary prevention: the incremental predictive value of high-sensitivity cardiac troponin T beyond ASCVD risk assessment

S. Andraz[1], J. Massa Pereira[1], H. Costa[1], M. Espirito Santo[1], L. Hamann[1], R. Fernandes[1], J. Bispo[1], D. Carvalho[1], D. Bento[1], P. Azevedo[1], J. Mimoso[1]

[1]Algarve University Hospital Center, Faro, Portugal
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Background: Atherosclerotic cardiovascular disease (ASCVD) is a global health challenge, prompting widespread use of conventional risk assessment tools (SCORE2 in Europe and the ASCVD Risk calculator in the United States) to guide preventive strategies. Emerging evidence indicates that high-sensitivity cardiac troponin (hsTn) may refine risk stratification.

Objective: We investigate whether measuring hsTnT in primary prevention correlates with mortality and enhances prediction compared to conventional risk score stratification.

Methods: Utilizing National Health and Nutrition Examination Survey (NHANES) data (1999 to 2004) linked to the National Death Index, our cohort included individuals without a history of CVD at baseline and suitable for assessment of the ASCVD ACC/AHA risk score. Individuals were grouped based on computed 10-year risk (low: <5%; borderline: 5-7.5%; intermediate: 7.5-20%; high: ≥ 20%). hsTroponin T levels were measured (Roche assay, 99 th percentile 22ng/L for men and 17ng/L for women). Cox regression estimated association between ASCVD risk categories, with or without hsTnT, and all-cause and cardiovascular mortality.

Results: Among 5257 adults, 47% male, mean age 54 ± 9.1 years, the mean 10-year ASCVD risk was 8.17 ± 8.2%, grouped as: low: 53.3%; borderline: 11.5%; intermediate: 23.9%; and high: 11.3%. hsTnT above the 99 th percentile was measured in 1.1%, 2.7%, 4.9% and 13.4%, respectively ($p < 0.001$). Over 16.2 ± 3.4 years, all-cause and cardiovascular death was 22.3% and 5.6%, respectively. Compared to low-risk, the hazard ratio (HR) for all-cause death increased to 2.85 (2.2-3.7), 5.6 (4.2-7.5), and 15.3 (11.7-19.9) for borderline, intermediate, and high-risk, respectively. A significant interaction with hsTnT above 99 th percentile was observed (low: HR 3.5 (1.3-9.2); borderline: HR 4.75 (2.1-10.8); intermediate: HR 2.6 (1.8-3.7), high: HR 2.2 (1.7-2.9), $p<0.01$ for all interactions, see Fig. 1). For cardiovascular death, a significant interaction occurred in intermediate (HR 3.3 (2-5.4), $p<0.001$) and high-risk (HR 3.6 (2.2-6), $p<0.001$) groups.

Conclusion: Our findings suggest that hsTnT provides incremental predictive value, at least doubling the hazard of all-cause death across all risk categories, and tripling the hazard of CV death in intermediate and high-risk groups, compared to ASCVD risk stratification alone. The integration of troponin measurements into risk assessment strategies could refine primary prevention approaches.

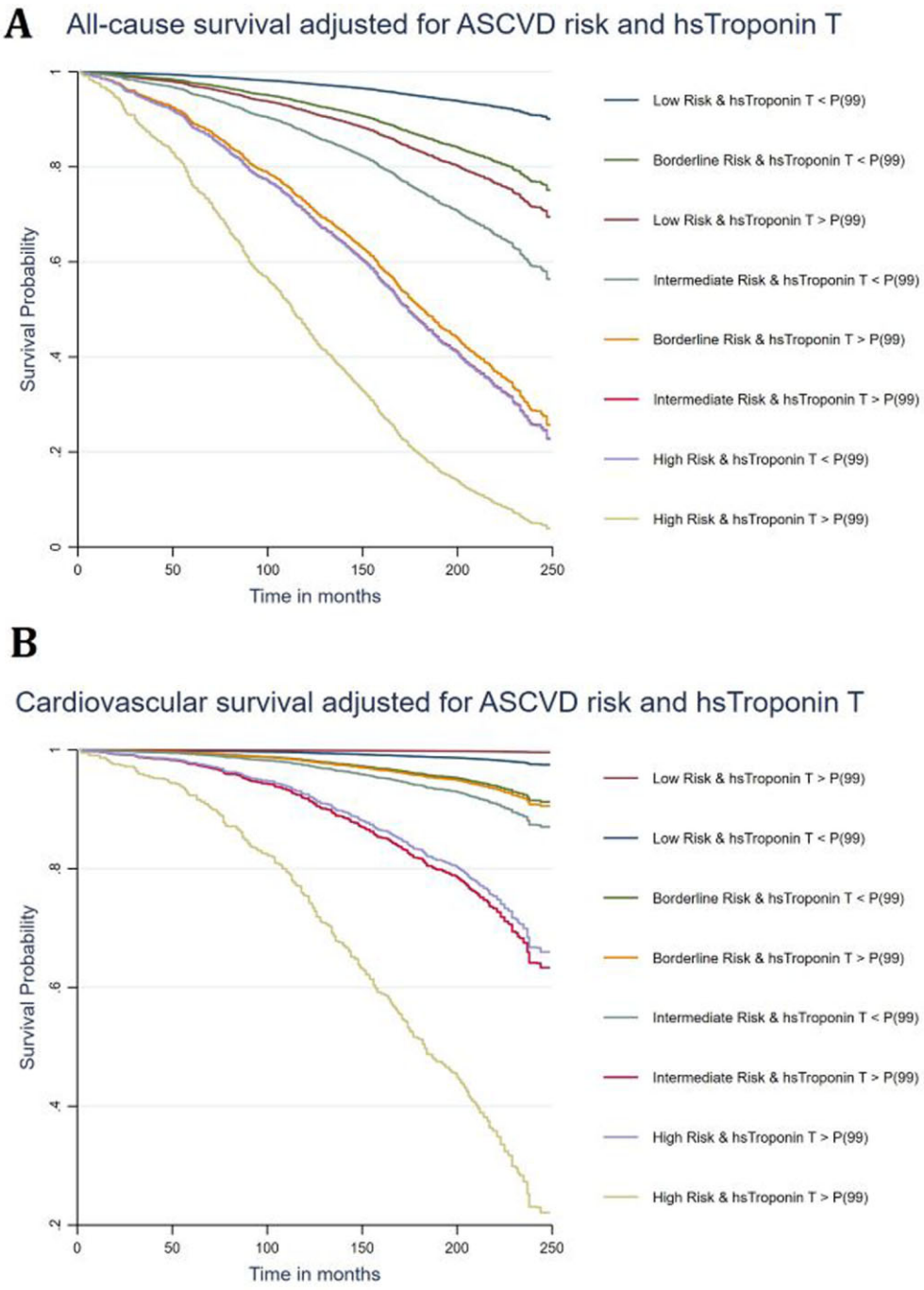

Figure 1 – All-cause (A) and cardiovascular (B) survival curves adjusted for ASCVD risk categories and hsTroponin T above the 99th percentile (P99). Note, for instance, that increased troponin in borderline and intermediate risk individuals confers an estimated higher risk of all-cause mortality compared to a high-risk individual with a normal troponin level. The hazard ratios (HR) and 95% CI, compared to Low-Risk and hsTnT < P(99), are as follow: **A**: Low Risk & hsTnT > P(99): HR 3.5 (1.3-9.2), p = 0.01; Borderline Risk & hsTnT < P(99): 2.7 (2.1-3.7), p < 0.001; Borderline Risk & hsTnT > P(99): 13 (6-28.2), p < 0.001; Intermediate Risk & hsTnT < P(99): 5.5 (4.1-7.4), p < 0.001; Intermediate Risk & hsTnT > P(99): 14.2 (9.4-21.2), p < 0.001; High Risk & hsTnT < P(99): 14.2 (10.8-18.7), p < 0.001; High Risk & hsTnT > P(99): 31.2 (22.7-43), p < 0.001. **B**: Low Risk & hsTnT > P(99): HR 0.16 (0.02-1.3), p = 0.08; Borderline Risk & hsTnT < P(99): 3.6 (2.3-5.8), p < 0.001; Borderline Risk & hsTnT > P(99): 3.9 (0.7-21.1), p = 0.1; Intermediate Risk & hsTnT < P(99): 5.5 (3.6-8.4), p < 0.001; Intermediate Risk & hsTnT > P(99): 18.1 (10.1-32.4), p < 0.001; High Risk & hsTnT < P(99): 16.5 (10.5-25.9), p < 0.001; High Risk & hsTnT > P(99): 59.8 (35.6-100.3), p < 0.001