

# Identifying a Prognostic High-Sensitivity Troponin Threshold for Hemodynamic Decompensation and 30-Day Pulmonary Embolism-Related Mortality

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**Background:** High-sensitivity cardiac troponin (hs-cTn) is an established biomarker for risk stratification in acute pulmonary embolism (PE). However, conventional dichotomization at the assay-specific upper reference limit does not account for the magnitude of troponin elevation. We aimed to identify a clinically meaningful, assay-normalized hs-cTn threshold for predicting adverse outcomes in initially hemodynamically stable patients with acute PE.

**Methods:** We analyzed 1,474 patients with acute PE and available hs-cTn measurements from a multicenter cohort. Troponin concentrations were normalized to assay-specific cutoffs. Receiver operating characteristic (ROC) analysis was performed to identify the optimal prognostic threshold according to the Youden index. Subsequently, we evaluated the proportion of hemodynamic decompensation events and 30-day PE-related deaths captured at progressively higher hs-cTn thresholds.

**Results:** Hemodynamic decompensation occurred in 92 patients (6.2%), PE-related death in 39 (2.6%), and all-cause death in 110 (7.5%). The hs-cTn ratio showed good discrimination for hemodynamic decompensation (AUC 0.716) and PE-related mortality (AUC 0.727). ROC analysis identified an optimal threshold of 2.32 times the assay-specific cutoff for predicting decompensation. At an hs-cTn ratio  $\geq 2$ , sensitivity and specificity were 81.5% and 56.7% for hemodynamic decompensation, and 84.6% and 55.3% for 30-day PE-related mortality. For decompensation, PPV was 11.1% (95% CI 8.9–13.7) and NPV was 97.9% (96.6–98.8). For PE-related mortality, PPV was 4.9% (3.4–6.8) and NPV was 99.2% (98.4–99.7). No significant differences were observed at 2.32 times. Increasing the threshold to  $\geq 3\times$  reduced these proportions to 60.9% and 53.8%. Patients with hs-cTn  $\geq 2\times$  had a higher incidence of decompensation than those below this threshold (11.8% vs 2.5%). This association remained significant after multivariable adjustment (adjusted OR 2.62, 95% CI 1.46–4.72).

**Conclusions:** ROC analysis identified an hs-cTn threshold of approximately twice the assay-specific cutoff as a potentially useful marker of hemodynamic decompensation in initially stable PE. Higher thresholds substantially reduced the proportion of adverse events identified, supporting 2× as a pragmatic candidate threshold for further validation.