EVOLUTION OF ERYTHROCYTE INDICES TOWARD AN IRON DEFICIENT PICTURE PREDICTS EARLY MORTALITY IN ACUTE DECOMPENSATED HEART FAILURE

Poster Contributions
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Background: Iron deficiency (ID) augments adrenaline synthesis as iron is a critical cofactor for monoamine oxidase, the rate limiting enzyme of catecholamine degradation. Because a high adrenergic drive further impairs the failing myocardium, we hypothesized that patients (pts) admitted for worsening heart failure (WHF) who manifest evidence of evolving ID might be at a heightened risk for mortality.

Methods: We analysed data from 337 pts with WHF (age 70±14y, 61% LVEF<45%). The combination of a rising red cell distribution (RDW) and a falling mean cell volume (MCV) identified evolving ID as it has a ≥90% sensitivity and specificity for this diagnosis.

Results: Over a median hospital stay of 10 days, 55%, 45%, 63%, and 17% of pts had a fall in hemoglobin (Hb), a rise in RDW, a fall in MCV, and evolving ID. Pts with evolving ID had greater reductions in Hb (-1.0 vs. -0.3 g/dl, P<0.01). From admission to 3 and 6 months post discharge, 37 (11%) and 55 (16%) pts died. Evolving ID predicted death at 3 (HR 2.10, 95% CI 1.00-4.37, P=0.04, Fig A) and 6 (HR 1.87, 95% CI 1.01-3.44, P=0.04, Fig B) months independently of worsening eGFR, CRP, Hb and sodium levels (adjusted 6 month HR 1.91, 95% CI 1.03-3.53, P=0.04). Addition of evolving ID to ΔeGFR, ΔCRP, ΔHb, and Δsodium improved the model χ²-value implying incremental utility.

Conclusion: Evolution of simple erythrocyte indices towards an iron deficient picture identifies hospitalized WHF pts at a 2-fold amplified risk for early mortality and could be utilised to refine risk or targeted.