ASM 2026
HFSA ANNUAL SCIENTIFIC MEETING
Where Heart Failure Teams Gather
October 9-12, 2026 | Phoenix, Arizona

Heterogeneity Of Volume Derangements In Advanced Heart Failure: Unmasking Red Blood Cell Volume As A Hidden Driver Of Plasma Volume Phenotypes

Marc A. Silver[1,2], Mohamed R. Mohamed[3], Radha Gopalan[4], Jonathan A. Feldschuh[5]. Banner University Medical Center-Phoenix, Phoenix, AZ; [1,2], University of Arizona, Phoenix, Phoenix, AZ; [3]The University of Arizona, Phoenix, AZ[1,2,4] Banner University Medical Center, Phoenix, AZ [1,2,4]; Daxor Corporation, Oak Ridge, TN [5]

THE UNIVERSITY OF ARIZONA COLLEGE OF MEDICINE PHOENIX

Banner University Medical Center Phoenix

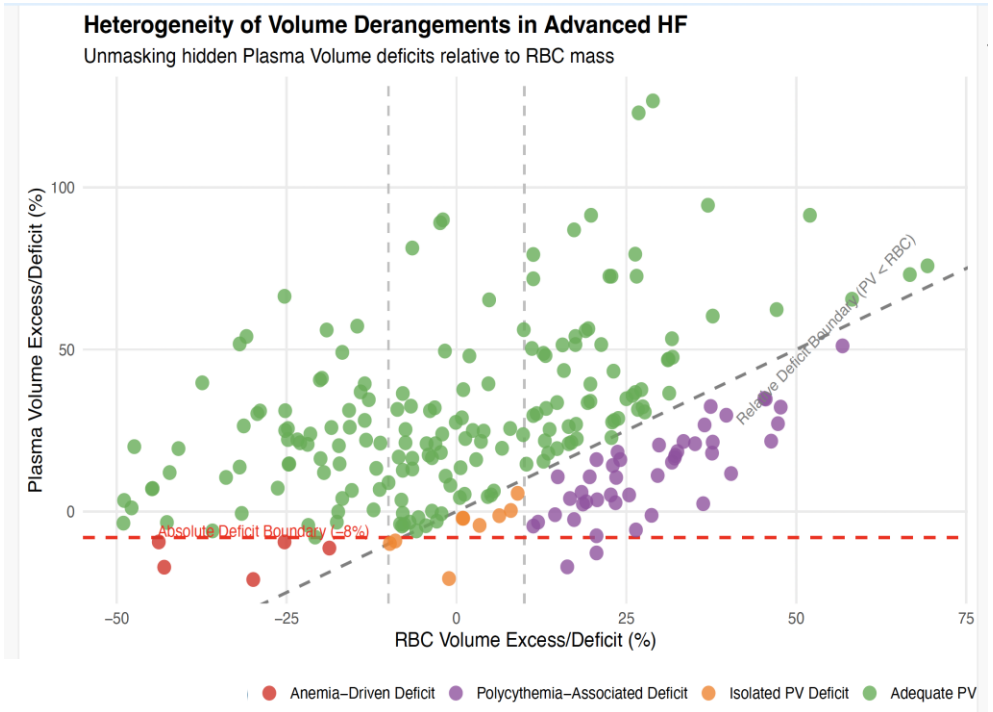
Background: Cardiac performance is dependent on intravascular euvolemia. Estimating blood volume often leads clinicians to overuse of diuretic therapies. Attention has focused on total blood volume (TBV) and red blood cell volume (RBCV), yet little attention is available on variations in plasma volume (PV). Adequate PV provides compensatory responses for **polycythemia** (excess RBCV) and **anemia** (deficient RBCV). If the PV Reserve **(PVR**) is exhausted, compensatory mechanisms are disabled.

Methods: We performed blood volume analysis in 238 patients with advanced heart failure.(Daxor, Oak Ridge, TN.) Patients were in-patients (n=89) and out-patients (n=149). All had a history of loop diuretic use. To account for both absolute and relative volume derangements driven by red cell mass, the selection criteria for "Deficient PVR" included:

Absolute PV Deficit: > -8% deficit from predicted normal.

Relative PV Deficit: Presence of PV excess that is quantitatively less than the concomitant RBCV excess (PV Excess < RBCV Excess).

ATR: Albumin transudation rate (ATR) evaluates excess capillary leak as a potential pathway to deficient PVR.



Results: The frequency of a deficient PV was 10 patients (4%). There were 8 women and 2 men. This cohort revealed distinct drivers of PVR deficiency:

Anemia-Driven Deficit: It was present in 7 patients who were anemic, with deficits ranging from -9% to -43.8% (Avg -25.6%). In these patients, the expected compensatory mobilization of plasma (PV reserve) to normalize TBV was depleted. Of the patients with impaired PVR, baseline HCTs ranged from 31 to 49% (avg. 40%) and the normalized Hct (nHCT) (the Hct if the PV was normal) declined to 22.8- 48% (avg 35%). Four of the low PVR patients were in-patients and 2 of these were on IV diuretics. 4 of the 10 were on oral diuretics and 4 were on no diuretics.(see figure, center, anemia and polycythemia associated deficits).

Polycythemia-Associated Deficit: Deficient reserve was also present in 2 patients who were polycythemic (Avg RBCV +18.5%). While polycythemia normally drives compensatory PV expansion to normalize blood viscosity, these patients demonstrated an absent or depleted reserve relative to their high red cell mass.

Isolated PV Deficit: Only 1 patient had a normal RBCV.

Additional signals: The ATR was measured for each patient and the low PVR group had values ranging from 0.17 to 0.62 %/min but averaged 0.36, suggesting potential increased capillary leak.

Conclusions: Large deficits in PV occur in patients with advanced heart failure. Current diuretic usage is not a requisite suggesting that long term PV depletion may be related to loss or decreased production initiated by RBC-driven derangements. The impaired RBCV derangements are then amplified by loss of PV dependent compensation. Specifically, the deficient PVR phenotype is most common in patients who are significantly anemic with increased ATR. Diuretic therapies maintain a role in HF care, but clinicians need to be mindful the role RBCV's play in circulatory homeostasis. Detecting loss of adequate PVR related to RCV-driven derangements are an essential pivot to enhance clinical decision-making, guide treatment and improve patient outcomes.