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Recovery from Hospital-Acquired Anemia after Acute Myocardial Infarction and Effect on Outcomes

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Abstract

New onset, hospital-acquired anemia (HAA) during acute myocardial infarction (AMI) is independently associated with poor outcomes. Patterns of recovery from HAA after AMI and their association with mortality and health status are unknown. In the prospective 24-center TRIUMPH registry, we identified 530 AMI patients with HAA (defined as normal hemoglobin at admission with development of anemia by discharge) who had repeat, protocol-driven hemoglobin measurement 1 month after discharge. The 1-month measures were used to define persistent (persistent anemia) and transient (anemia resolved) HAA. Patients' health status was assessed at 1, 6 and 12-months after AMI using the Short-Form 12 Physical Component Summary (SF-12 PCS), and the health status of patients with persistent and transient HAA was compared using multivariable repeated measures regression. Mortality was compared using the log-rank test and proportional hazards regression. Overall, 165 patients (31%) developed persistent HAA. Adjusted mean SF-12 PCS scores in follow-up were significantly lower in those with persistent HAA as compared with transient HAA (−2.0 points [95% CI −3.6, −0.3], $p=0.02$). Over a median follow-up of 36 months, crude mortality (13% vs. 5%, $p=0.002$) and multivariable-adjusted mortality (HR 2.08, 95% CI 1.02–4.21, $p=0.04$) was greater in patients with persistent HAA. In conclusion, HAA persists 1 month post-discharge in nearly 1 of 3 patients and is associated with worse health status and higher mortality. Further investigation is needed to understand whether HAA prevention, recognition, and treatment, particularly among those persistent HAA, improves outcomes.

Keywords

myocardial infarction; anemia; hemoglobin; health status; quality of life; mortality

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Hospital acquired anemia (HAA) develops in nearly 1 in 2 acute myocardial infarction (AMI) patients who have normal hemoglobin at admission, and is associated with higher mortality and worse health status.¹ It is unclear to what extent HAA during AMI hospitalization resolves quickly in follow-up or persists after the acute episode of care. Moreover, it is unknown whether clinically important outcomes, such as mortality and health status, differ between patients with transient and persistent HAA. Understanding the patterns of HAA recovery after hospital discharge and their association with clinical outcomes would further support the importance of preventing and recognizing HAA. Furthermore, it may help identify patients at risk for poor recovery after AMI who may benefit from more intensive follow-up. To address these gaps in knowledge, we studied patients enrolled in the Translational Research Investigating Underlying disparities in acute Myocardial infarction Patients' Health Status study (TRIUMPH); a prospective 24-center observational registry of AMI treatment and outcomes. We identified patients with HAA at the time of discharge and compared the 1-year health status and 3-year mortality of those with persistent and transient HAA at 1 month after an incident AMI.

METHODS

The design and methods of the TRIUMPH study have been reported.² Briefly, a total of 4340 patients were enrolled in TRIUMPH between April 11, 2005, and December 31, 2008. Patients were 18 years of age, with elevated cardiac biomarkers (troponin or creatine kinase-MB fraction assessed within 24 hours of admission), and supporting evidence of AMI (electrocardiographic ST-segment changes or prolonged ischemic signs/symptoms). Participants were required to present to the enrolling institution or to have been transferred to that hospital within 24 hours of presentation so that the primary clinical decision-making occurred at the enrolling center. Patients with elevated cardiac biomarkers from elective coronary revascularization were excluded. Trained data collectors performed detailed baseline chart abstractions to document patients' medical history, the processes of inpatient care, laboratory results and treatments. Each patient underwent a standardized interview by research staff to document sociodemographic and clinical data. Patients were contacted for follow-up interviews at 1, 6 and 12-months after AMI to reassess health status and interval events. Patients who consented to follow-up laboratory assessment then underwent protocol-driven hemoglobin assessments 1 month after discharge. All patients signed an informed consent approved by the participating institution, and Institutional Review Board approval was obtained at each participating center.

Our goal was to describe the prevalence of persistent HAA and to assess the association between HAA persistence and subsequent outcomes. Accordingly we first excluded 24 of the 4,340 patients in TRIUMPH who died in-hospital. We then restricted the cohort to include only the 3,251 patients who were not anemic at admission, and further restricted our analysis to patients who developed HAA before discharge from the hospital (n=1,599). Of these patients, 1,069 did not have 1 month follow-up Hgb collected because they were deceased (2%), too ill (5%), refused interview (5%), lost-to-follow (31%), or had phone interviews instead of in home visits (57%), yielding an analytic cohort of 530 AMI patients discharged with HAA who had follow-up hemoglobin assessment 1 month after discharge.

Admission hemoglobin was defined as the first in-hospital hemoglobin (g/dl) value available for each patient. If a patient was transferred to a TRIUMPH center from another hospital (n=221), the initial hemoglobin at the transferring facility was obtained and used as the admission value. Discharge hemoglobin was defined as the last hemoglobin value obtained within 48 hours of discharge from the hospital. HAA was defined as absence of anemia on admission, but development of anemia by discharge. Consistent with prior work,¹ we defined anemia using the age-, gender-, and race-specific criteria described by Beutler and

Waaen. Accordingly, a hemoglobin value less than 13.7 g/dl for white men aged 20 to 59, 13.2 g/dl for white men 60 years, 12.9 g/dl for black men aged 20–59, 12.7 g/dl for black men 60 years, 12.2 g/dl for white women and 11.5 g/dl for black women was used to identify anemia. This classification has been previously shown to be more accurate than the World Health Organization definition (WHO).³

Data abstracters, using the Thrombolysis In Myocardial Infarction (TIMI) classification, systematically recorded bleeding episodes.⁴ TIMI major bleeding was defined as intracranial hemorrhage or a hemoglobin decline ≥ 5 g/dl in the setting of overt bleeding. TIMI minor bleeding was assigned if the drop in hemoglobin was 3 to 5 g/dl in the setting of observed bleeding. Any bleeding episode with a decline in hemoglobin < 3 g/dl was classified as TIMI minimal bleeding. All TIMI categories accounted for blood transfusion, with adjustment of hemoglobin values by 1 g/dl per unit transfused.

Health status was assessed using the Short Form-12 Physical Component Summary score (SF-12 PCS). The SF-12 is a valid and reliable instrument, with higher scores representing better health status.⁵ A score of 50 is normalized to the mean health status of the US population and each 10 points represents 1 standard deviation from that mean. Using criteria proposed by Cohen, the minimum clinically important difference on this scale is 2 points ($0.2 \times \text{SD}$).⁶

Baseline characteristics, in-hospital treatments, in-hospital complications, and lab values of patients who had persistent HAA at 1 month were compared to patients with transient HAA. We also described the proportion of patients with persistent HAA across strata of HAA severity at discharge. For outcome analyses, we compared patients with persistent and transient HAA. For descriptive purposes, categorical data are presented as frequencies and differences between groups were compared using chi-square or Fisher's exact tests, as appropriate. Continuous variables were reported as the mean $\pm$ standard deviation, and differences were compared using independent Student's t-tests. The Wilcoxon rank-sum test was used to compare patients' length of stay due to its skewed distribution, and results are reported as the median and interquartile range.

The primary outcome of interest was health status, as assessed using the SF-12 PCS score. We used multivariable repeated measures linear regression with autoregressive covariance structure for SF-12 PCS that incorporated 1-, 6-, and 12-month health status assessments. This model adjusted for baseline SF-12 PCS score, age, gender, chronic kidney disease and chronic heart failure, which are potential confounders identified in the prior literature. Since patients discharged with more severe HAA may be more likely to develop persistent HAA, we also adjusted for patients' discharge hemoglobin. We then compared the mortality of patients with and without persistent HAA using Kaplan-Meier analyses and tested this relationship using the log-rank test. Follow-up time was censored at the time of the most recent mortality update to the TRIUMPH database, with a mean follow-up of 3 years. We used Cox proportional hazards regression to identify the relationship of HAA persistence with mortality after adjusting for the GRACE 6-month mortality risk score, age and discharge hemoglobin. The GRACE 6-month mortality risk score⁷ is strongly predictive of long-term mortality and incorporates important potential confounders. Variables in the GRACE score include age, heart rate, systolic blood pressure, creatinine, history of CHF, prior MI, in hospital PCI or CABG, ST-segment depression on the initial electrocardiogram and elevated cardiac biomarkers. We also adjusted for discharge hemoglobin values, since patients with severe HAA might be more likely to have persistent HAA at 1 month. Proportional hazards assumption was tested visually and using Schoenfeld residuals.

To assess the potential impact of missing 1-month follow-up data on our results, we developed a non-parsimonious propensity score for having 1-month follow-up and conducted sensitivity analyses in which we weighted both the health status and mortality models with the reciprocal of this score. All analyses were conducted with SAS version 9.2 (SAS Institute, Cary, NC) and R version 2.11.1).⁸

RESULTS

Among 530 AMI patients discharged with HAA, 165 patients (31%) were persistently anemic at 1 month (Table 1). A greater proportion of patients with more severe grades of HAA at discharge had persistent HAA (Figure 1). Among patients with mild HAA at discharge, 82 (23%) had persistent HAA at 1 month, while patients discharged with moderate and severe HAA more frequently developed persistent HAA (65 patients (44%) and 18 patients (67%), respectively).

We observed significant differences in physical functioning in follow-up between patients with persistent and transient HAA (Figure 2). At the time of their AMI, patients who were ultimately identified as having persistent HAA had lower mean SF-12 PCS scores than those whose with transient HAA (41.8 ± 12.4 vs. 44.1 ± 12.1 , $p=0.04$). The difference between these groups increased at the time of 1-month follow-up, when patients with persistent HAA had a mean SF-12 PCS score of 39.4 ± 11.5 , as compared with 43.6 ± 10.6 for those with transient HAA ($p<0.001$). This gap between the groups remained at 6-month follow-up (persistent vs. transient HAA = 43.3 ± 11.2 vs. 47.1 ± 11.2 , $p=0.002$) and narrowed again by 12-month follow-up when no significant difference was found between these groups (persistent vs. transient HAA = 44.0 ± 11.9 vs. 45.7 ± 11.3 , $p=0.16$). Comparing patients with persistent to those with transient HAA, after adjusting for potential confounders and baseline health status, persistent HAA was associated with lower SF-12 PCS scores (-2.0 point, 95% CI, $-3.6, -0.3$ points, $p=0.02$) across the entire follow-up period. When these models were weighted with the reciprocal of a propensity score for having complete 1-month follow-up, the differences in health status between patients with persistent and resolved HAA were similar.

Vital status was known for the vast majority of patients across a median follow-up of 36 months (518/530, 98%). Patients with persistent HAA had a greater crude all-cause mortality over the follow-up period (13%) than patients with transient HAA (5%, $p=0.002$; Figure 3). After multivariable adjustment, patients with persistent HAA had more than twice the risk of death as patients with transient HAA (HR 2.08, 95% CI 1.02–4.21, $p=0.04$). When the mortality model was weighted with the reciprocal of a propensity score for having complete 1-month follow-up, we found no clinically meaningful changes in the hazard ratio for mortality.

DISCUSSION

Almost 1 in 3 AMI patients who developed HAA during hospitalization in this multi-center cohort had persistent HAA 1 month later. These patients had worse health status, particularly at 1 and 6 months after AMI, and greater long-term mortality than patients who had transient HAA. These relationships persisted after multivariable adjustment and in sensitivity analyses to assess for bias introduced by missing follow-up data. Since HAA is common, and its persistence is associated with worse clinical outcomes, further research is needed to understand whether HAA prevention, recognition, or management would improve outcomes.

Our findings extend prior reports establishing the incidence and clinical importance of HAA in patients with AMI. Previous studies have shown that patients who develop HAA have greater mortality in follow-up after AMI than patients who do not develop HAA.^{1,9,10} However, hemoglobin trajectories after discharge from the hospital also appear to influence outcomes. In the OPTIMAAL study cohort, Anker and colleagues found that hemoglobin changes in follow-up were prognostically important.¹¹ Specifically, hemoglobin declines during follow-up were associated with higher mortality, while increased hemoglobin in follow-up was associated with better survival. However, this study did not focus specifically on HAA since it also included patients who were anemic at enrollment into the study (a well-known risk factor for adverse outcomes), and examined changes between baseline and 1-year hemoglobin values, precluding the opportunity to identify those for whom early recognition and intervention might improve outcomes. Similarly, Hasin and colleagues studied the relationship between hemoglobin changes over a median follow-up of 4.5 months, relative to discharge anemia status, in a cohort of AMI patients admitted to the coronary intensive care unit.¹² Patients whose anemia resolved by follow-up assessment had similar outcomes to those who were never anemic, while those who were persistently anemic or developed new anemia in follow-up had greater risk of mortality and heart failure. They reported the subgroup of patients who were not anemic at admission, and results were consistent with their primary analysis. Consistent with these findings, we identified a high prevalence of persistent HAA in follow-up and a broader range of outcomes, including health status, even after adjusting for baseline severity of HAA. The finding that persistence of HAA is prognostically important, independent of HAA severity at discharge is an important, novel finding of this study, which suggests that further studies are needed to understand whether interventions to manage HAA at the time of hospitalization or early in the follow-up period improve outcomes. We also found important relationships between HAA persistence and health status, with significantly greater impairment in physical functioning among patients with persistent HAA.

We found clinically important differences in health status between patients with and without persistence of HAA, particularly in the early recovery period after AMI. Anemia is strongly associated with poorer physical functioning,^{13,14} which may be directly mediated by diminished oxygen carrying capacity in the setting of lower hemoglobin levels.¹⁵ Patients' physical functioning is an important outcome, and may also influence patients' participation with evidence-based treatments such as cardiac rehabilitation, or returning to work. Since patients may be particularly motivated to engage in lifestyle changes, such as increasing the frequency and intensity of exercise, soon after experiencing an AMI, this large, early impairment in physical functioning could worsen trajectories of lifestyle modification in recovery after AMI. We identified the largest differences in physical functioning between those with and without resolution of anemia at 1 and 6 months, suggesting that the association between persistent HAA and health status is strongest in the early recovery period, although it is possible that the greater death rates among those with persistent HAA may have attenuated the health status differences at 1 year. Alternatively, it may be that the anemia may have resolved in some of the persistent HAA patients, minimizing the health status deficits by 1 year. If so, however, it is possible that the early recognition and treatment of anemia might accelerate patients' recovery after AMI.

HAA is particularly important because it is potentially preventable by altering processes of care. Since HAA is multifactorial,¹ the most effective prevention strategies should address risk factors for HAA. Bleeding is one example of a modifiable risk factor for HAA and bleeding reduction strategies, particularly among PCI patients, have been well described.^{16–19} However, patients without overt bleeding also commonly develop HAA.¹ In these patients, novel risk factors such as blood loss from phlebotomy (which could be reduced using pediatric tubes, stored serum samples and by limiting unnecessary

phlebotomy)^{20–22} or unrecognized iron deficiency should be explored as components of a successful HAA prevention program. Moreover, further studies are needed to identify predictors of HAA persistence in follow-up. Fewer patients with persistent HAA were discharged on dual antiplatelet therapy, which may suggest that chronic gastrointestinal bleeding precipitated by dual antiplatelet therapy is not likely to be a frequent cause of persistent HAA. It remains unclear whether persistent HAA is a reflection of a greater burden of comorbidities, such as diabetes and renal failure, or other conditions that were not collected in the TRIUMPH registry, such as iron deficiency.

Our findings should be interpreted in the context of several potential limitations. We used discharge hemoglobin values to define HAA since nadir hemoglobin values were not available. This approach may underestimate the prevalence of HAA; although we accounted for blood transfusions, given the relatively short duration of hospitalization (median 3.0 days, (IQR) 2.0 – 4.0), it is unlikely that a substantial proportion of HAA cases were missed. A large proportion of patients did not have complete 1-month follow-up or did not consent to 1-month hemoglobin assessments. However, in sensitivity analyses that weighted our models with the reciprocal of a propensity score for having 1-month follow-up hemoglobin assessment we found similar results. This suggests that missing follow-up data did not introduce significant bias into our results. Finally, these observational data do not allow us to draw conclusions about causal relationships between HAA persistence and outcomes, and it remains unclear whether HAA is a marker for, or a mediator of, poor outcomes.

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Figure 1.
Persistence of Hospital-Acquired Anemia by Discharge Hospital-Acquired Anemia Severity.

Figure 2. Unadjusted SF-12 Physical Component Summary Scores of Patients with Persistent and Transient Hospital-Acquired Anemia During Recovery after Acute Myocardial Infarction
Mean unadjusted SF-12 PCS scores at the time of AMI and at 1, 6 and 12-month follow-up interviews. Bars through each point estimate reflect 95% confidence intervals around the mean value. M= months, PCS = Physical Component Summary.

Figure 3.
Long-Term Mortality of Patients With and Without Persistent Hospital-Acquired Anemia.

Table 1

Baseline Characteristics of Patients by 1-month HAA Status

Variable	<u>Hospital-Acquired Anemia Status at 1 Month</u>		
	Transient (n = 365)	Persistent (n = 165)	P-Value
Age (mean $\pm$ SD)	60.2 $\pm$ 11.6	61.1 $\pm$ 11.9	0.41
Men	235 (64.4%)	118 (71.5%)	0.11
Caucasian	278 (76.6%)	110 (67.1%)	0.02
Hemoglobin: admit (g/dl, mean $\pm$ SD)	14.6 $\pm$ 1.3	14.1 $\pm$ 1.3	< 0.001
Hemoglobin: discharge (g/dl, mean $\pm$ SD)	11.8 $\pm$ 1.3	11.0 $\pm$ 1.4	< 0.001
Hemoglobin change from initial to final during hospitalization (g/dl, mean $\pm$ SD)	-2.7 $\pm$ 1.5	-3.2 $\pm$ 1.9	0.004
Hemoglobin: 1 month (g/dl, mean $\pm$ SD)	14.1 $\pm$ 1.1	12.0 $\pm$ 1.0	< 0.001
Platelets: discharge (per uL, mean $\pm$ SD)	257 $\pm$ 76	262 $\pm$ 101	0.54
Body mass index (kg/m ² , mean $\pm$ SD)	29.4 $\pm$ 6.3	29.3 $\pm$ 6.3	0.82
Ejection fraction (% , mean $\pm$ SD)	49.4 $\pm$ 11.3	48.6 $\pm$ 12.4	0.50
Killip Class			0.006
I	344 (94.8%)	140 (86.4%)	
II	16 (4.4%)	17 (10.5%)	
III	2 (0.6%)	3 (1.9%)	
IV	1 (0.3%)	2 (1.2%)	
GRACE mortality risk score (g/dl, mean $\pm$ standard deviation)	99.2 $\pm$ 25.9	106.0 $\pm$ 27.9	0.007
Chronic heart failure	15 (4.1%)	9 (5.5%)	0.49
Dyslipidemia	191 (52.3%)	89 (53.9%)	0.73
Hypertension	229 (62.7%)	120 (72.7%)	0.03
History of cancer	28 (7.7%)	18 (10.9%)	0.22
Peripheral arterial disease	19 (5.2%)	6 (3.6%)	0.43
Prior myocardial infarction	66 (18.1%)	37 (22.4%)	0.24
Prior coronary artery bypass surgery	30 (8.2%)	24 (14.5%)	0.03
Prior stroke	16 (4.4%)	13 (7.9%)	0.10
Diabetes mellitus	72 (19.7%)	57 (34.5%)	< 0.001
Chronic kidney disease	14 (3.8%)	12 (7.3%)	0.09
Final diagnosis			0.09

Variable	<u>Hospital-Acquired Anemia Status at 1 Month</u>		
	Transient (n = 365)	Persistent (n = 165)	P-Value
ST-segment elevation MI	197 (54.0%)	76 (46.1%)	
Non-ST-segment elevation MI	168 (46.0%)	89 (53.9%)	
In-hospital percutaneous coronary intervention	263 (81.7%)	81 (66.4%)	< 0.001
In-hospital renal failure	7 (1.9%)	6 (3.6%)	0.24
In-hospital bleeding	51 (14.0%)	27 (16.4%)	0.47
Thrombolysis in Myocardial Infarction bleeding severity			0.52
Major	10 (19.6%)	5 (18.5%)	
Minor	21 (41.2%)	8 (29.6%)	
Minimal	20 (39.2%)	14 (51.9%)	
Length of stay (Median [interquartile range])	4.0 (3.0, 5.0)	5.0 (3.0, 9.0)	< 0.001
In-Hospital Iron Supplementation	3 (0.8%)	5 (3.0%)	0.12
Aspirin at discharge	350 (95.9%)	154 (93.3%)	0.21
Thienopyridine at discharge	304 (83.3%)	106 (64.2%)	< 0.001
Warfarin at discharge	34 (9.3%)	17 (10.3%)	0.72
ACE inhibitor/ARB at discharge	270 (74.0%)	110 (66.7%)	0.08
Beta blocker at discharge	339 (92.9%)	154 (93.3%)	0.85