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Characteristics and Outcomes Among Heart Failure Patients With Anemia and Renal Insufficiency With and Without Blood Transfusions (Public Discharge Data from California 2000–2006)

David P. Kao, MD[a], **Elma Kreso, MD**[b], **Gregg C. Fonarow, MD**[b], and **Mori J. Krantz, MD**[a,c,*]
[a]Division of Cardiology, University of Colorado Denver, Aurora, Colorado
[b]Division of Cardiology, University of California, Los Angeles, Los Angeles, California
[c]Colorado Prevention Center, Aurora, Colorado.

Abstract

Renal insufficiency and anemia are increasingly recognized as predictors of adverse events in heart failure. The impact of blood transfusion on mortality in patients with heart failure has not been previously characterized. We examined temporal changes in admissions and in-hospital mortality using public discharge data from California (2000 to 2006) and then evaluated the impact of renal insufficiency, anemia, and transfusion on in-hospital mortality in univariate and multivariate analyses. In total 596,456 unique patient admissions for heart failure were recorded. Renal insufficiency and anemia were common co-morbidities (27.4% and 27.1%, respectively) and 6.2% of patients received a transfusion of red blood cells. Renal insufficiency and anemia were associated with increased mortality (unadjusted odds ratio [OR] 2.45, 95% confidence interval [CI] 2.39 to 2.52, and 1.27, 95% CI 1.24 to 1.30, respectively). After adjustment, renal insufficiency (OR 2.54, 95% CI 2.46 to 2.62) and anemia (OR 1.12 95% CI 1.07 to 1.17) remained significant; however, transfusion emerged as the strongest single predictor (OR 3.81, 95% CI 3.51 to 4.13) of mortality. In conclusion, these data suggest that anemia and renal insufficiency are independently associated with mortality in an unselected heart failure population. This is the first study to demonstrate that transfusion magnifies this effect and is associated with a particularly poor prognosis.

Heart failure is the leading cause of hospitalization and rehospitalization in elderly patients in the United States.[1] Elderly patients with heart failure frequently have additional co-morbid conditions that complicate their inpatient care and worsen overall prognosis. Specifically, anemia and chronic renal insufficiency are common co-morbidities in the aging population and are associated with increased mortality in the setting of heart failure.[2] The simultaneous presence of anemia and renal insufficiency in patients with heart failure is often referred to as the cardiorenal—anemia syndrome and has been shown to magnify long-term morbidity and mortality irrespective of patient age.[3] The impact of red blood cell transfusion in patients with acute decompensated heart failure, however, has not been previously described, yet may be an important mediator of adverse events. The prognostic importance of these cofactors varies depending on the size of the study population and data source used. Because current heart failure population studies are derived from voluntary quality improvement initiatives,[4] industry-funded registries,[5-8] or demographically limited cohorts such as Medicare,[9] the generalizability of findings to the diversity of patients with heart failure in the United States is uncertain. The present study, therefore, evaluated

*Corresponding author: Tel: 303-436-7818; fax: 303-436-7739. mkrantz@dhha.org (M.J. Krantz)..

discharge data from a large, unselected database in California. We sought to quantify the effects of renal insufficiency, anemia, and the receipt of blood transfusion on in-hospital outcomes.

Methods

The California Office of Statewide Health Planning and Development collect data on all hospitalizations in the state of California as mandated by state law. We selected this state because it is currently the largest single United States population base with standardized, deidentified, and publicly available patient discharge data (PDD). Diagnoses and procedure codes for all admissions are classified using the International Classification of Disease, Ninth Revision, Clinical Modification (ICD-9-CM). After obtaining an exemption from Colorado Multiple Institutional Review Board, all PDD from California from January 1, 2000 to December 31, 2006 were obtained and admissions with the principal diagnosis of heart failure were extracted for analysis. Admissions where heart failure was not listed as the principal diagnosis were excluded as were hospitalizations where age was >18 years. These data were linked with census data from California[10] to calculate population-adjusted hospitalization and mortality rates.

De-identified PDD were imported into a MySQL server (MySQL, Inc., Sun Microsystems, Cupertino, California), which was used for data collation. Patient age, gender, ethnicity, payer status, admission type, length of stay, and in-hospital mortality were quantified directly from the database. Presence of co-morbid conditions for each admission was determined using the ICD-9-CM codes in secondary diagnosis fields. Patients who underwent hemodialysis or received transfusions of packed red blood cells were identified by the presence of corresponding ICD-9-CM procedure codes in any of the procedure fields associated with each hospitalization. California population estimates from 2000 to 2006 were acquired from the United States Census Bureau and population-adjusted admission and mortality rates were calculated for each year and summarized across years by calculating an annual percent change (APC). The APC and 95% confidence intervals (CIs) were determined from the slope and standard error estimates from the regression line calculated on the log scale. Baseline clinical and demographic data were summarized using the number and percent per group. Overall mortality rates were presented for various clinical co-morbidities in addition to unadjusted odds ratios (ORs) and 95% CIs.

A multiple logistic regression model was fit to estimate the odds for predictors of mortality. Predictors for length of stay (LOS) were estimated using a Cox proportional hazards model censoring for deaths before discharge. Covariates used in the 2 models included demographics (age, gender, and ethnicity), admission type, the most common co-morbidities (hypertension, ischemic heart disease, diabetes, atrial fibrillation, renal insufficiency, anemia, chronic obstructive pulmonary disease, and dyslipidemia), and 2 prespecified procedures (transfusion and hemodialysis). All covariates remained in the model to assess their strength of association with the outcome in the presence of other factors. Interactions of anemia with transfusion, anemia with renal insufficiency, hemodialysis with renal insufficiency, and transfusion with renal insufficiency were included in the models and removed if their maximum likelihood estimate was not significant at the 0.05 alpha level. Because of the large number of observations in this study, overpowering resulted in most covariates showing statistical significance in the models. Therefore, to identify covariates that had a consistent reportable relation with the outcome, the models were run on smaller random samples of the data. Statistical analysis was performed using SAS 9.2 (SAS Institute, Cary, North Carolina).

Results

In total 596,456 of 27,522,042 (2.2%) hospital admissions in California met criteria for inclusion in the analysis from 2000 to 2006. Population-adjusted rates of hospitalization and in-hospital mortality are presented in Figure 1. Over the study period population-adjusted rate of heart failure admissions varied from 332 to 313 per 100,000 but did not exhibit a significant increase or decrease over the study period (APC −0.4%, 95% CI −1.07 to 0.28). However, population-adjusted mortality rates decreased moderately from 15 to 11 per 100,000, resulting in an APC equal to −5.1% (95% CI −3.02 to −7.04).

Baseline demographics and clinical characteristics are listed in Table 1. A majority (72.6%) of the population was >65 years old and correspondingly nearly 70% of the population was a recipient of Medicare. Hypertension and coronary heart disease were the 2 most common co-morbidities. In total 27.4% of patients had renal insufficiency and 27.1% had anemia. Overall, 6.2% of the population received a blood transfusion.

Effects of demographics and clinical characteristics on inhospital mortality and LOS are presented in Table 2. Overall, 4.23% of the population died, and median LOS was 4 days. Absolute mortality rates were higher than the overall population in subjects with renal insufficiency or who underwent hemodialysis or transfusion during their hospitalization. Median LOS was increased in subjects with renal insufficiency (5 days), anemia (5 days), and for those who received a transfusion (6 days). Patients who received a blood transfusion without concurrent anemia had the highest absolute mortality rate (17.46%) and longest median LOS (10 days).

Univariate predictors of in-hospital mortality and hospital LOS are listed in Table 2. Renal insufficiency was the strongest univariate predictor of mortality followed by receipt of transfusion. Presence of anemia was a modest predictor of in-hospital mortality. Additional co-morbidities such as chronic obstructive pulmonary disease and atrial fibrillation were significantly associated with adverse outcomes, whereas dyslipidemia and hypertension were associated with lower mortality rates in the univariate model. Notably, receipt of transfusion without a documented anemia diagnosis was strongly associated with mortality (OR 5.34, 95% CI 4.94 to 5.78) and hospital LOS (10 days, 6 to 17.5).

ORs and 95% CIs from a multiple logistic regression mortality model are presented in Figure 2. Advanced age (≥65 years) was a significant predictor of mortality, whereas dyslipidemia and hypertension were associated with lower odds of mortality as observed in the univariate model. However, atrial fibrillation and renal insufficiency persisted as adverse prognostic factors. Undergoing hemodialysis (OR 2.2, 95% CI 2.1 to 2.3) and transfusion (OR 3.8, 95% CI 3.5 to 4.1) were also associated with an increased in-hospital mortality rate. Anemia alone, however, was associated with only a small increased risk for in-hospital mortality (OR 1.1, 95% CI 1.1 to 1.2), although interactions of anemia with transfusion (OR 1.7, 95% CI 1.6 to 1.8) and with renal insufficiency (OR 2.2, 95% CI 2.1 to 2.3) magnified the odds in the 2 cases. Receipt of a blood transfusion during the index hospitalization remained the strongest single predictor (OR 3.81, 95% CI 3.51 to 4.13) of mortality.

Discussion

Heart failure hospitalization remains a major cause of morbidity despite a modest temporal decrease in hospital mortality rates. This study represents the first report of unselected PDD to assess predictors of heart failure outcomes and provides confirmation that renal insufficiency and anemia are common co-morbidities and important prognostic indicators for LOS and mortality. A novel finding is the fact that blood transfusion is the single strongest predictor of in-hospital mortality even after adjustment for common co-

morbidities. Although transfusion is a well-established marker of adverse outcome in acute coronary syndrome, to our knowledge this is the first study to demonstrate the adverse impact of transfusion in patients hospitalized with heart failure. This stands in contrast to a much smaller study where blood transfusions were deemed to be safe and perhaps even beneficial in the setting of acute decompensated heart failure.[11] Previous studies evaluating the effects of iron repletion[12] and erythropoiesis-stimulating agents (ESAs) have produced mixed results, with ESAs being associated with an increased signal for acute myocardial infarction (risk ratio 1.3, 95% CI 0.9 to 1.9).[13] Because the present study used nonselected patient data to determine predictors of adverse outcome in heart failure, the findings have broad applicability to contemporary inpatient practice.

We sought to contrast our results from PDD with contemporary heart failure registry data to assess the reliability and usefulness of PDD. Our findings regarding the prevalence of renal insufficiency and anemia are remarkably similar to more selected heart failure registry studies, although the present study sample (>500,000) is somewhat larger compared to national databases such as the Acute Decompensated Heart Failure National Registry (ADHERE)[5,6] and the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE)[7,8] (>100,000 and >45,000 patients, respectively). For example, prevalence of renal insufficiency was >25% in our study population and the ADHERE population.[6] Use of PDD, however, has the potential advantage of eliminating selection bias because it does not require hospital participation in a specific registry. Instead it uses pre-existing data collected under the auspices of a state public health function. PDD are thus more analogous to Medicare-based registry studies yet does not have age-related enrollment thresholds. Despite these differences, demographics and co-morbidities such as coronary heart disease, hypertension, diabetes, and dyslipidemia were similar to those found in the ADHERE and OPTIMIZE-HF registries.[5-8]

Despite marked differences in data collection methods, our study confirms findings from ADHERE and OPTIMIZE-HF in 2 regards. First, our findings strengthen the reported adverse association between renal insufficiency and anemia on inpatient morbidity and mortality in the United States heart failure population. Second, publicly available PDD might be used to identify other co-morbidities gleaned from an array of procedure and diagnostic codes currently not included in the design of heart failure registries, which are tailored to data input by the enduser. These additional data elements could nonetheless have important prognostic value in the setting of acute decompensated heart failure admissions. In the current era of health care reform, tapping into publicly available data sources to gauge the impact of national quality improvement efforts may also prove useful.

Regarding health outcomes, our findings mirrored ADHERE, where renal insufficiency increased inpatient mortality and hospital LOS. Similarly, the OPTIMIZE-HF registry found an increase in mortality of 18% for every 0.3-mg/dl increase in serum creatinine >2.0 mg/dl up to 3.5 mg/dl.[8] From 2005 to 2007, the number of patients on hemodialysis had increased from 5% to 7%[14,15]—similar to the proportion who underwent hemodialysis in the present study. However, the mortality rate in our study was significantly higher (7.7% vs 5.1%), suggesting that unselected populations are at a higher baseline absolute risk and therefore may be more representative of real-world populations with greater underlying disease severity.

In multivariate analysis, transfusion of packed red blood cells was independently associated with mortality and LOS overall and compared to anemic patients who did not receive a transfusion. The impact of blood transfusion on outcomes in hospitalized patients with heart failure has not been studied extensively, although transfusion has been associated with increased mortality after percutaneous coronary intervention.[16] Transfusion may simply be a

marker of disease severity in heart failure and percutaneous coronary intervention populations, but this is unlikely the sole explanation because the highest observed mortality in our study (17.46%) occurred in patients transfused without concurrent anemia.

Several studies have demonstrated that anemia is independently associated with increased long-term mortality, although a definitive mechanism has not been identified.[17-24] None of these studies rigorously quantified the frequency of blood transfusion over the entire period studied. It is therefore possible that part of the observed increase in mortality was attributable to transfusion. The mechanism of the association between transfusion and mortality after percutaneous coronary intervention or in hospitalized patients with heart failure remains unclear but may include decreases in tissue oxygen delivery, disruption of nitric oxide–mediated vasodilation, platelet activation, and activation of the coagulation cascade.[2,3] The intravascular volume load resulting from transfusion may also exacerbate decompensation in hospitalized patients with heart failure. Nonetheless, applicability of these mechanisms to acute decompensated heart failure prognosis is unknown and requires further study. Currently, there are no prospective trials evaluating the safety of blood transfusion in patients with heart failure in the ambulatory or in-hospital setting. Most existing trials have centered on use of ESAs with or without iron supplementation and have provided inconsistent results.[25-27] Although not a primary end point of those trials, part of any beneficial effect of ESAs might be attributable to decreased rates of transfusion throughout the study.

We acknowledge several study limitations primarily that relate to administrative data. The interaction between transfusion and laboratory covariates including level of hemoglobin was not available and the clinical rationale for transfusion was not discernable. Other limitations include potential residual measured and unmeasured confounders. Although PDD have advantages over existing registries, they preclude identification of readmissions. Moreover, only in-hospital outcomes are available, so long-term prognostic implications of renal insufficiency, anemia, and blood transfusion cannot be ascertained. PDD include multiple diagnostic and procedure codes; however, an under-reporting bias for anemia is possible, and procedural codes such as transfusion might be more accurately recorded. Furthermore, coding of heart failure from hospital discharge data has been previously shown to have specificity, sensitivity, and positive predictive values of 96.8%, 58.5%, and 65.1%, respectively, somewhat less than for acute coronary syndrome admissions.[28] ICD-9 codes associated with a given hospital admission are often assigned by trained coding staff, which differs from previous voluntary registries that frequently use clinicians for data input. Nonetheless, we observed similar rates of co-morbidities and outcome patterns compared to other inpatient heart failure registries and the sheer number of admissions and hospitals captured in this database provides broad generalizability of the findings.

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References

1. American Heart Association. Heart Disease and Stroke Statistics—2010 Update. American Heart Association; Dallas, TX: 2010. Available at: http://americanhert.org/presenter.jhtml?identifier=3000090. Accessed: February 26, 2010
2. Kazory A, Ross EA. Anemia: the point of convergence or divergence for kidney disease and heart failure? J Am Coll Cardiol. 2009; 53:639–647. [PubMed: 19232895]

3. Ronco C, Haapio M, House AA, Anavekar N, Bellomo R. Cardiorenal syndrome. J Am Coll Cardiol. 2008; 52:1527–1539. [PubMed: 19007588]
4. Patel UD, Hernandez AF, Liang L, Peterson ED, LaBresh KA, Yancy CW, Albert NM, Ellrodt G, Fonarow GC. Quality of care and outcomes among patients with heart failure and chronic kidney disease: a Get With the Guidelines–Heart Failure Program study. Am Heart J. 2008; 156:674–681. [PubMed: 18946892]
5. Fonarow GC, Heywood JT, Heidenreich PA, Lopatin M, Yancy CW, ADHERE Scientific Advisory Committee and Investigators. Temporal trends in clinical characteristics, treatments, and outcomes for heart failure hospitalizations, 2002 to 2004: findings from Acute Decompensated Heart Failure National Registry (ADHERE). Am Heart J. 2007; 153:1021–1028. [PubMed: 17540205]
6. Yancy CW, Lopatin M, Stevenson LW, De Marco T, Fonarow GC, ADHERE Scientific Advisory Committee and Investigators. Clinical presentation, management, and in-hospital outcomes of patients admitted with acute decompensated heart failure with preserved systolic function: a report from the Acute Decompensated Heart Failure National Registry (ADHERE) Database. J Am Coll Cardiol. 2006; 47:76–84. [PubMed: 16386668]
7. Young JB, Abraham WT, Albert NM, Gattis Stough W, Gheorghiade M, Greenberg BH, O'Connor CM, She L, Sun JL, Yancy CW, Fonarow GC, OPTIMIZE-HF Investigators and Coordinators. Relation of low hemoglobin and anemia to morbidity and mortality in patients hospitalized with heart failure (Insight from the OPTIMIZE-HF Registry). Am J Cardiol. 2008; 101:223–230. [PubMed: 18178411]
8. Abraham WT, Fonarow GC, Albert NM, Stough WG, Gheorghiade M, Greenberg BH, O'Connor CM, Sun JL, Yancy CW, Young JB, OPTIMIZE-HF Investigators and Coordinators. Predictors of inhospital mortality in patients hospitalized for heart failure: insights from the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF). J Am Coll Cardiol. 2008; 29:347–356. [PubMed: 18652942]
9. Herzog CA, Muster HA, Li S, Collins AJ. Impact of congestive heart failure, chronic kidney disease, and anemia on survival in the Medicare population. J Card Fail. 2004; 10:467–471. [PubMed: 15599836]
10. California Census Data. Available at: http://quickfacts.census.gov/qfd/ states/06000.html. Accessed: February 13, 2010
11. Garty M, Cohen E, Zuchenko A, Behar S, Boyko V, Iakobishvili Z, Mittelman M, Battler A, Shotan A, Gottlieb S, Caspi A, Hasdai D, Heart Failure Survey in ISrael (HFSIS) Investigators. Blood transfusion for acute decompensated heart failure—friend or foe? Am Heart J. 2009; 158:653–658. [PubMed: 19781427]
12. Anker SD, Comin Colet J, Filippatos G, Willenheimer R, Dickstein K, Drexler H, Lüscher TF, Bart B, Banasiak W, Niegowska J, Kirwan BA, Mori C, von Eisenhart Rothe B, Pocock SJ, Poole-Wilson PA, Ponikowski P, FAIR-HF Trial Investigators. Ferric carboxymaltose in patients with heart failure and iron deficiency. N Engl J Med. 2009; 361:2436–2448. [PubMed: 19920054]
13. Unger EF, Thompson AM, Blank MJ, Temple R. Erythropoiesis-stimulating agents—time for a reevaluation. N Engl J Med. 2010; 362:189–192. [PubMed: 20054037]
14. Heywood JT, Fonarow GC, Costanzo MR, Mathur VS, Wigneswaran JR, Wynne J, ADHERE Scientific Advisory Committee and Investigators. High prevalence of renal dysfunction and its impact on outcome in 118,465 patients hospitalized with acute decompensated heart failure: a report from the ADHERE database. J Card Fail. 2007; 13:422–430. [PubMed: 17675055]
15. Adams KF Jr, Fonarow GC, Emerman CL, LeJemtel TH, Costanzo MR, Abraham WT, Berkowitz RL, Galvao M, Horton DP, ADHERE Scientific Advisory Committee and Investigators. Characteristics and outcomes of patients hospitalized for heart failure in the United States: rationale, design, and preliminary observations from the first 100,000 cases in the Acute Decompensated Heart Failure National Registry (ADHERE). Am Heart J. 2005; 149:209–216. [PubMed: 15846257]
16. Maluenda G, Lemesle G, Ben-Dor I, Collins SD, Syed AI, Li Y, Torguson R, Kaneshige K, Xue Z, Suddath WO, Satler LF, Kent KM, Lindsay J, Pichard AD, Waksman R. Value of blood transfusion in patients with a blood hematocrit of 24% to 30% after percutaneous coronary intervention. Am J Cardiol. 2009; 104:1069–1073. [PubMed: 19801026]

17. Groenveld HF, Januzzi JL, Damman K, van Wijngaarden J, Hillege HL, van Veldhuisen DJ, van der Meer P. Anemia and mortality in heart failure patients a systematic review and meta-analysis. J Am Coll Cardiol. 2008; 52:818–827. [PubMed: 18755344]

18. Dunlay SM, Weston SA, Redfield MM, Killian JM, Roger VL. Anemia and heart failure: a community study. Am J Med. 2008; 121:726–732. [PubMed: 18691487]

19. Mitchell JE. Emerging role of anemia in heart failure. Am J Cardiol. 2007; 99(suppl):15D–20D.

20. Horwich TB, Fonarow GC, Hamilton MA, MacLellan WR, Borenstein J. Anemia is associated with worse symptoms, greater impairment in functional capacity and a significant increase in mortality in patients with advanced heart failure. J Am Coll Cardiol. 2002; 39:1780–1786. [PubMed: 12039491]

21. Luthi JC, Flanders WD, Burnier M, Burnand B, McClellan WM. Anemia and chronic kidney disease are associated with poor outcomes in heart failure patients. BMC Nephrol. 2006; 7:3. [PubMed: 16515712]

22. McClellan WM, Flanders WD, Langston RD, Jurkovitz C, Presley R. Anemia and renal insufficiency are independent risk factors for death among patients with congestive heart failure admitted to community hospitals: a population based study. J Am Soc Nephrol. 2002; 13:1928–1936. [PubMed: 12089390]

23. Ezekowitz JA, McAlister FA, Armstrong PW. Anemia is common in heart failure and is associated with poor outcomes: insights from a cohort of 12 065 patients with new-onset heart failure. Circulation. 2003; 107:223–225. [PubMed: 12538418]

24. Tang WH, Tong W, Jain A, Francis GS, Harris CM, Young JB. Evaluation and long-term prognosis of new-onset, transient, and persistent anemia in ambulatory patients with chronic heart failure. J Am Coll Cardiol. 2008; 51:569–576. [PubMed: 18237687]

25. Silverberg DS, Wexler D, Blum M, Sheps D, Schwartz D, Yachnin T, Baruch R, Tchebiner J, Zubkov A, Shaked M, Steinbruch S, Keren G, Iaina A. Aggressive therapy of congestive heart failure and associated chronic renal failure with medications and correction of anemia stops or slows the progression of both diseases. Perit Dial Int. 2001; 21(suppl):S236–S240. [PubMed: 11887828]

26. Silverberg DS, Wexler D, Blum M, Tchebiner JZ, Sheps D, Keren G, Schwartz D, Baruch R, Yachnin T, Shaked M, Schwartz I, Steinbruch S, Iaina A. The effect of correction of anaemia in diabetics and non-diabetics with severe resistant congestive heart failure and chronic renal failure by subcutaneous erythropoietin and intravenous iron. Nephrol Dial Transplant. 2003; 18:141–146. [PubMed: 12480972]

27. Besarab A, Bolton WK, Browne JK, Egrie JC, Nissenson AR, Okamoto DM, Schwab SJ, Goodkin DA. The effects of normal as compared with low hematocrit values in patients with cardiac disease who are receiving hemodialysis and epoetin. N Engl J Med. 1998; 339:584–590. [PubMed: 9718377]

28. Austin PC, Daly PA, Tu JV. A multicenter study of the coding accuracy of hospital discharge administrative data for patients admitted to cardiac care units in Ontario. Am Heart J. 2002; 144:290–296. [PubMed: 12177647]

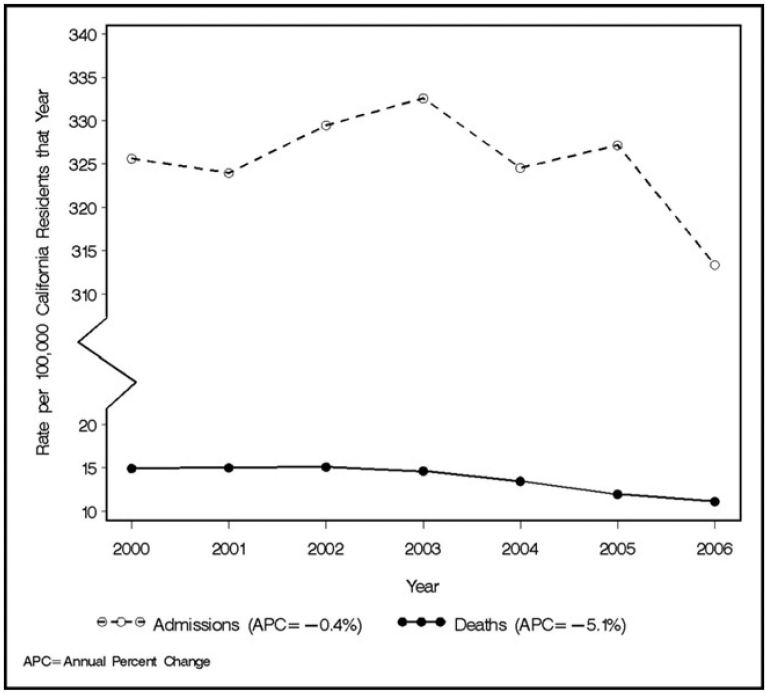

Figure 1.
Population-adjusted heart failure hospitalization and mortality rates during the study period.

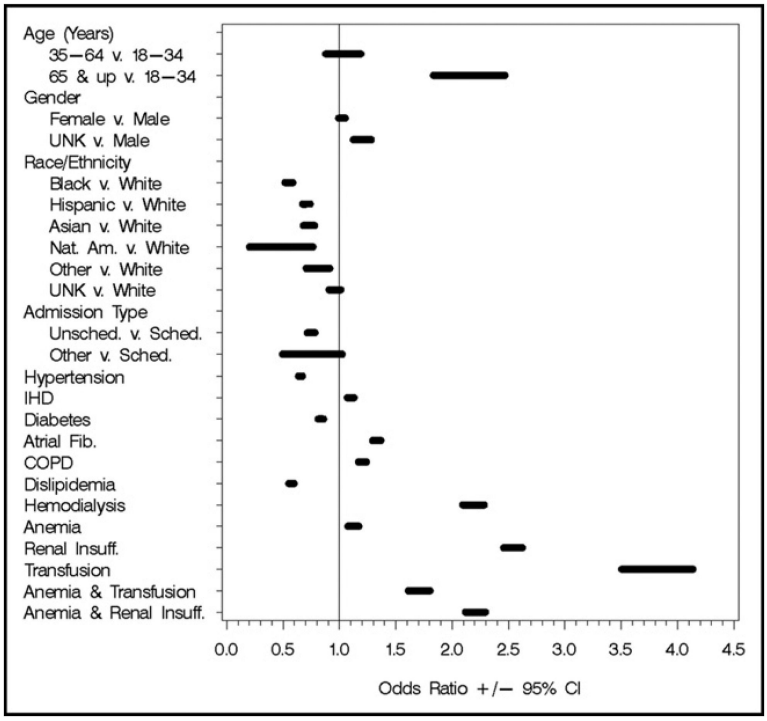

Figure 2.

Factors associated with mortality from multiple logistic regression. COPD = chronic obstructive pulmonary disease; Fib. = fibrillation; IHD = ischemic heart disease; Insuff. = insufficiency; Nat. Am. = Native-American; Sched. = scheduled; UNK = unknown; Unsched. = unscheduled.

Table 1

Characteristics of patients hospitalized with heart failure, 2000 to 2006 (n = 596,456)

Category	
Age (years)	
18–34	8,640 (1.5%)
35–64	155,066 (26.0%)
≥65	432,750 (72.6%)
Men	261,019 (43.8%)
Women	280,664 (47.1%)
Unknown/masked/missing	54,773 (9.2%)
Ethnicity/race	
White	334,625 (56.1%)
Hispanic	73,556 (12.3%)
Black	58,027 (9.7%)
Asian/Pacific islander	25,424 (4.3%)
Native American/Eskimo/Aleut	486 (0.1%)
Other	7,039 (1.2%)
Unknown	97,299 (16.3%)
Insurance	
Medicare	416,373 (69.8%)
Medicaid	76,333 (12.8%)
Private coverage	75,249 (12.6%)
Self-pay	12,513 (2.1%)
Other	
Admission	
Scheduled	41,426 (7.0%)
Unscheduled	554,265 (92.9%)
Other	765 (0.1%)
Co-morbidity	
Hypertension	362,919 (60.9%)
Coronary heart disease	296,177 (49.7%)
Diabetes mellitus	242,956 (40.7%)
Atrial fibrillation	182,361 (30.6%)
Renal insufficiency	163,402 (27.4%)
Anemia	161,468 (27.1%)
Chronic obstructive pulmonary disease	152,340 (25.5%)
Dyslipidemia	129,867 (21.8%)
Underwent hemodialysis	40,098 (6.7%)
Received transfusion	36,749 (6.2%)

Patients >18 years old were included. Masked age data preclude further breakdown of age categories.

Table 2

Association of patient characteristics with mortality and length of stay

Feature (ICD-9-CM code)	Mortality (%)	Unadjusted OR (95% CI)	LOS (Days), Median (Q1–Q3)
All	4.23%	—	4 (2–6)
Men	4.24%	—	4 (2–6)
Women	4.10%	0.96 (0.94–0.99)	4 (2–6)
White	4.86%	—	4 (2–6)
Hispanic	3.07%	0.62 (0.59–0.65)	4 (2–6)
Black	2.08%	0.42 (0.39–0.44)	3 (2–6)
Asian	3.76%	0.76 (0.0.72−0.82)	4 (2–7)
Native-American	1.85%	0.37 (0.19–0.72)	4 (2–6)
Other race	3.58%	0.73 (0.64–0.83)	4 (2–6)
Scheduled admission	5.60%	—	4 (2–8)
Unscheduled admission	4.12%	0.73 (0.69–0.76)	4 (2–6)
Hypertension	3.41%	0.61 (0.59–0.62)	4 (2–6)
Coronary heart disease	4.57%	1.18 (1.15–1.21)	4 (2–6)
Diabetes mellitus	3.75%	0.82 (0.80–0.84)	4 (2–6)
Atrial fibrillation	5.64%	1.60 (1.56–1.64)	4 (2–7)
Renal insufficiency	7.25%	2.45 (2.39–2.52)	5 (3–8)
Anemia	4.96%	1.27 (1.24–1.30)	5 (3–8)
Dyslipidemia	2.67%	0.56 (0.54–0.58)	3 (2–6)
Hemodialysis	7.43%	1.93 (1.85–2.01)	4 (2–8)
Transfusion	8.37%	2.22 (2.13–3.21)	6 (4–11)
Chronic obstructive pulmonary disease	5.35%	1.36 (1.31–1.40)	4 (2–7)
Neither anemia nor renal insufficiency	2.95%	—	3 (2–5)
Anemia and renal insufficiency	6.82%	2.40 (2.32–2.49)	5 (3–9)
Renal insufficiency, no anemia	7.56%	2.69 (2.61–2.78)	4 (2–7)
Anemia, no renal insufficiency	3.58%	1.22 (1.17–1.27)	4 (3–7)
Neither anemia nor transfusion	3.81%	—	3 (2–6)
Anemia and transfusion	7.09%	1.93 (1.84–2.02)	6 (4–10)
Transfusion, no anemia	17.46%	5.34 (4.94–5.78)	10 (6–17.5)
Anemia, no transfusion	4.43%	1.17 (1.14–1.21)	4 (3–7)

Q1 = quartile 1; Q3 = quartile 3.