Research Article

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How Do Differences in Treatment Impact Racial and Ethnic Disparities in Acute Myeloid Leukemia?

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Abstract

Background: We previously demonstrated disparate acute myelogenous leukemia (AML) survival for black and Hispanic patients; these differences persisted despite younger ages and higher prevalence of favorable cytogenetics in these groups. This study determined: (i) whether there are differences in treatment delivered to minorities, and (ii) how these differences affect outcomes in AML. We hypothesize that differences in treatment explain some proportion of survival disparities.

Methods: We used California Cancer Registry data linked to hospital discharge abstracts for patients with AML (1998–2008). Logistic regression models estimated odds of treatment (chemotherapy and/or hematopoietic stem cell transplant) by race/ethnicity. Cox proportional hazard models estimated mortality by race after adjustment for treatment.

Results: We analyzed 11,084 records. Black race was associated with lower odds of chemotherapy [OR, 0.74; 95% confidence interval (CI), 0.61–0.91]. Black and Hispanic patients had decreased odds of transplant [(OR, 0.64; 95% CI, 0.46–0.87); (OR, 0.74; 95% CI, 0.62–0.89), respectively]. Black patients had increased hazard of mortality (HR, 1.14; 95% CI, 1.04–1.25) compared with whites. Adjustment for receipt of any treatment resulted in decreased mortality (HR, 1.09; 95% CI, 1.00–1.20) for black patients.

Conclusions: AML treatment differences for black patients explain some proportion of the disparity. Future AML disparities studies should investigate socioeconomic and other characteristics.

Impact: Study findings may better elucidate drivers of disparities in AML. *Cancer Epidemiol Biomarkers Prev; 24(2); 344–9. ©2015 AACR.*

Introduction

Previous institution-based studies describe racial/ethnic disparities in survival from acute myelogenous leukemia (AML) for black and Hispanic groups when compared with whites (1–6). Earlier work from our group, using the Surveillance Epidemiology and End Results (SEER) database, also show disparities in AML survival for black and Hispanic patients (1, 2). In one of our previous studies, we assessed the impact of favorable prognostic factors, specifically, younger ages at presentation and cytogenetic subtypes, on AML survival (2). Although whites and Asian Pacific Islanders (API) with these more favorable characteristics experience improved survival, disparities persist for other racial/ethnic groups (2, 3).

Appropriate treatment is known to be one of the most important predictors of survival in AML (7–16). Differences in receipt of appropriate or evidence-based treatment by race and ethnicity correlate with worse outcomes from several solid malignancies (12, 17, 18). For hematologic malignancies, however, there are few studies that assess whether racial/ethnic disparities in outcomes are due to differences in receipt of treatment (19–22). Many of these previous studies focus on outcomes after bone marrow transplant, using data from the International Bone Marrow Transplant Registry with or without linkage to SEER data. These studies show lower rates of transplant for black patients (19, 22) and higher transplant failure rates for Hispanic patients (21). These studies also show a strong correlation between low socioeconomic status (SES) and inferior outcomes after bone marrow transplantation in blacks (17). None of these studies, however, analyze differences in the receipt of chemotherapy, nor do they assess the impact of treatment differences on disparate survival outcomes. Finally, these studies include patients with various diseases amenable to transplantation, including multiple myeloma (19), lymphoma (19, 22), and leukemia (20)—each associated with unique risks for mortality. To date, there are no population-based studies that evaluate the impact of differences in any treatment on survival disparities in AML.

Therefore, we considered whether differences in receipt of appropriate treatment (chemotherapy and hematopoietic blood and stem cell transplantation) may explain some of the observed racial/ethnic survival disparities from AML. In our previous evaluations, we could not compare differences in treatment due to limitations of the SEER stand-alone database (23). Linking SEER with Medicare claims that data might address this limitation; however, because a high proportion of minorities are diagnosed before the age of 65 (1, 2), these patients would be excluded from SEER-Medicare database based on age and lack of Medicare

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coverage. In addition, because the SEER-Medicare data represent a population with homogeneous insurance status (Medicare), use of these data may miss some of the population-level differences in AML survival that we are able to detect using an all-payer dataset. Therefore, in this study, we use a population based, all-payer dataset linked with treatment variables to evaluate differences in receipt of treatment [chemotherapy or hematopoietic stem cell transplant (HSCT)] for AML and investigate the effect of treatment of AML on racial/ethnic survival disparities. We hypothesize that there are differences in the receipt of both chemotherapy and HSCT by race/ethnicity. Second, we hypothesize that these differences explain some of the survival disparities observed in black and Hispanic populations with AML. Because differences in treatment are mutable and actionable targets for change, the results of this study could inform both national and local efforts to eliminate disparities in cancer.

Materials and Methods

Sources of data

The award-winning California Cancer Registry (CCR) has information for every new invasive cancer, with the exception of nonmelanoma skin cancers, diagnosed in the state of California as mandated by state law (24). The registry is quite complete with fewer than 2% of cases ascertained through death certificates and fewer than 3% missing race data (24). There is very little loss to follow-up, because any treatment delivered, regardless of the location of care, must be reported to the registry by California state mandate. The dataset contains demographic information, including the following: age, gender, race, clinical information regarding primary cancer diagnosis, first course of treatment, and census block group data. In addition, the dataset includes information about survival time (in months) and cause of death.

Using a probabilistic algorithm based on date of birth, social security number, and gender, registry staff linked the CCR data to hospital discharge data records from the Office of Statewide Health Planning and Development (OSHPD; ref. 25). The OSHPD-Patient Discharge Data (OSHPD-PDD) contains detailed information for each discharge from all nonfederal, general acute care facility in the state of California and details about primary diagnosis for the index admission and up to 24 additional diagnoses, including International Classification of Disease, ninth clinical modification (ICD9-CM) codes for AML cytogenetic variants [(t(8;21), acute promyelocytic leukemia (APL), and 11q23)]. All secondary diagnoses are recorded with a concomitant indicator about whether or not the condition was present on admission that facilitates the calculation of Deyo-modified Charlson comorbidity score (26). The OSHPD-PDD also records the patient's disposition, including inpatient mortality. Registrars stripped matching variables, with the exception of gender after linkage to the CCR data. We obtained Institutional Review Board approval from the state of California and Stanford University before we obtained the linked data.

Inclusion/exclusion criteria

We included patients diagnosed with AML through the International Classification of Disease Third Edition (ICD- 03) codes (9840, 9861, 9866-9867, 9871-9874, 9895-9897, 9910, and 9920). We included records for all patients diagnosed between 1998 and 2008. We excluded patients with American Indian/Alaskan Native race/ethnicity due to small cell size, in accordance with our data use agreement.

Patient-level predictors of care and outcomes

We identified cytogenetic subtypes, t(8;21), 11q23, and APL, by ICD 03 code (9896, 9897, and 9866, respectively) as guided by the World Health Organization classification systems (27). We defined chemotherapy treatment as receipt of any chemotherapy, including oral therapies, in the CCR, and supported this with documentation in the OSHPD-PDD for inpatient treatments (ICD 9 codes V58.1-V58.12). The chemotherapy variable in CCR also contains report on whether therapy was recommended, but never received, or if the patient refused treatment. These patients were coded as no receipt of chemotherapy. We identified receipt of HSCT, which occurs on an inpatient basis by ICD9 coding (41.0-41.09) in the OSHPD-PDD.

Statistical analyses

Logistic regression models estimated odds of receiving treatment adjusted for risk factors, including patient demographics (age, gender, and race), comorbidities, year of diagnosis, and presence of cytogenetic AML subtype. Baseline Cox proportional hazard models predicted the hazard of mortality by race. Cox proportional hazard models were built sequentially to evaluate the impact of demographics and clinical factors. Because loss to follow-up may vary by the racial/ethnic group, we only included patients with known vital status at 60 months from the date of diagnosis. Models were adjusted for age, gender, comorbidities, year of diagnosis, and cytogenetic profiles t(8;21), 11q23, and APL. A subsequent model predicted the hazard of mortality after accounting for delivery of appropriate treatment (chemotherapy and/or HSCT).

We performed all analyses using SAS version 9.3 (Cary, March 2010). Results were considered significant when point estimates were not equal to 1, the associated confidence intervals excluded 1, and the associated P value was <0.05. All tests of significance were two-tailed. The proportionality assumption was tested by a thorough analysis of the Schoenfeld residuals (28).

Results

A total of 11,609 patients were included in the analysis. There were 64 patients who were excluded from the analysis for missing race ($n = 28$) or American Indian/Alaskan Native race/ethnicity ($n = 36$). Overall, the long-term loss to follow-up was low with small differences by race/ethnicity. We excluded a total of 4% of cases ($n = 462$; 3.9% NHW; 3% black; 8% Hispanic; and 6.8% API) because their vital status was unknown at 61 months. After exclusions, 11,084 patient records were retained for analysis. The cohort demographics are shown in Table 1. The majority of patients were white (67%), male (54%), and older than age 60 (61%) with low Charlson comorbidity score. APL was the most common subtype reported. The majority of patients received chemotherapy. There were low proportions of transplant among all patient populations. Compared with whites, black, Hispanic, and API patients presented at ages younger than 61 years, and had a higher proportion of the APL subtype. Overall, there were higher proportions of chemotherapy and transplant delivered to Hispanic and API compared with other racial/ethnic groups.

Table 2 shows the proportion of each racial/ethnic group receiving chemotherapy or bone marrow transplant in an

Table 1. Patient demographics (AML, California 1998–2008)

	Total *N* (%)	White *N* (%)	Black *N* (%)	Hispanic *N* (%)	API *N* (%)
Total	11,084 (100)	7,381 (67)	603 (5)	1,936 (17)	1,164 (11)
Gender					
Female	5,124 (46)	3,322 (45)	296 (49)	938 (49)	568 (49)
Male	5,960 (54)	4,059 (55)	307 (51)	998 (51)	596 (51)
Age, y					
< 21	604 (5)	201 (3)	46 (8)	292 (15)	65 (6)
21–40	1,116 (11)	473 (6)	65 (11)	403 (21)	175 (15)
41–60	2,585 (23)	1,610 (22)	187 (31)	478 (25)	310 (27)
61–80	4,741 (43)	3,483 (47)	217 (36)	587 (30)	454 (39)
>80	2,038 (18)	1,614 (22)	88 (15)	176 (9)	160 (14)
Median age, y		71	61	52	63
Comorbidity					
Char 0	6,615 (60)	4,342 (59)	307 (51)	1,262 (65)	704 (61)
Char 1	2,309 (21)	1,577 (21)	134 (22)	369 (19)	229 (20)
Char 2	1,123 (10)	774 (11)	86 (14)	139 (7)	124 (11)
Char $\geq$ 3	1,037 (9)	688 (9)	76 (13)	166 (9)	107 (9)
Translocation					
t(8;21)	124 (1)	71 (1)	4 (1)	28 (1.4)	21 (1.8)
APL (t(15;17))	893 (8)	451 (6)	59 (10)	288 (15)	95 (8.2)
t(11q23)	85 (0.8)	63 (1)	2 (0.3)	10 (0.5)	10 (0.9)

Abbreviations: Char, Charlson comorbidity index score; t(8;21), translocation 8;21; APL (t(15;17)), acute promyelocytic leukemia translocation (15;17); t(11q23), translocation 11q23.

unadjusted bivariate comparison. The table demonstrates that at least 60% of each racial/ethnic group received chemotherapy; higher proportion of Hispanics (75%) and API (72%) patients received chemotherapy. The proportion of patient records coded as chemotherapy recommended, but not received was less than 1%. Specifically, 79 (0.71%) patients for whom chemotherapy was recommended did not receive treatment. Approximately 10% of patients in each racial/ethnic group received HSCT with a similarly slightly higher proportion of Hispanics (12%) and API (13%) receiving this treatment.

In fully adjusted logistic regression models predicting receipt of treatment (Table 3), black patients were less likely to receive chemotherapy [OR, 0.74; 95% confidence interval (CI) 0.61–0.91] and less likely to receive transplantation (OR, 0.62; 95% CI, 0.46–0.85) compared with whites. Hispanic patients with AML were slightly less likely to receive chemotherapy; however, the difference in odds of receipt of chemotherapy was not statistically significant ($P = 0.56$). This group was also less likely to receive transplant (OR, 0.74; 95% CI, 0.62–0.89) compared with whites. By contrast, API patients were more likely to receive chemotherapy (OR, 1.23; 95% CI, 1.05–1.44) but had no statistically significant differences in the odds of receipt of transplant (HR, 1.01; 95% CI, 0.83–1.24).

Cox proportional hazard models in Table 4 predict overall mortality for patients with 60 months follow-up. In the baseline model adjusted by age, gender, race, year of diagnosis, comorbidities, and cytogenetic translocation, there was an increased hazard of mortality for black patients compared with white patients (HR, 1.14; 95% CI, 1.04–1.25). There was no survival disparity associated with Hispanic ethnicity ($P = 0.99$). There was a slight reduction in the hazard of mortality associated with API race/ethnicity (HR, 0.90; 95% CI, 0.84–0.97). When the models were further adjusted for receipt of any treatment, the hazard of mortality associated with black race decreased from baseline (HR, 1.09; 95% CI, 1.00–1.20); nearly completely neutralizing the disparity. There was no substantive change in the hazard of mortality after adjustment for treatment for Hispanic ($P = 0.86$) or API patients (HR, 0.91; 95% CI, 0.85–0.98) as compared with the baseline.

A crude sensitivity analysis was conducted to assess the potential effect of differential lost to follow-up by race/ethnicity. We compared point estimates from models that exclude patients lost to follow-up after 36 months; and include all patients regardless of length of follow-up. The relative disparity in mortality between minority and white patients reported in Table 4 were observed in all three models and thus, the additional data are not shown.

Discussion

The purpose of this study was to determine whether there are differences in receipt of appropriate treatment for AML by race/ethnicity for minorities diagnosed and treated in California. Furthermore, we aimed to understand the impact of any treatment disparities on survival in these groups. In this study, we found that there are differences in receipt of treatment by race (lower proportions of black patients received chemotherapy; and lower proportions of black and Hispanic patients received HSCT) than other groups. There was no such disparity associated with API

Table 2. Distribution of treatment by race/ethnicity (AML, California 1998–2008)

	Chemotherapy *N* (%)	*P*	Transplant *N* (%)	*P*
Race				
White	4,512 (61)	(Ref)	660 (9)	(Ref)
Black	381 (63)	0.32	51 (8)	0.69
Hispanic	1,444 (75)	<0.001	235 (12)	<0.001
API	840 (72)	<0.001	153 (13)	<0.001

NOTE: Chemotherapy indicates any chemotherapy recorded as administered or recommended; a *P* value of <0.05 is considered significant.

Table 3. Logistic regression models predicting the odds of receiving treatment by race/ethnicity (AML, California 1998–2008)

	Chemotherapy OR (95% CI)	*P*	Transplant OR (95% CI)	*P*
Race				
White	1.00 (Reference)		1.00 (Reference)	
Black	0.74 (0.61–0.91)	0.004	0.64 (0.46–0.87)	0.005
Hispanic	0.96 (0.84–1.10)	0.56	0.74 (0.62–0.89)	0.001
API	1.23 (1.05–1.44)	0.010	0.99 (0.81–1.22)	0.95
Gender				
Female	1.00 (Reference)		1.00 (Reference)	
Male	1.10 (0.99–1.20)	0.05	0.89 (0.78–1.02)	0.10
Age				
<21	1.00 (Reference)		1.00 (Reference)	
21–40	0.49 (0.32–0.76)	0.001	1.33 (1.04–1.70)	0.02
41–60	0.35 (0.24–0.53)	<0.001	0.88 (0.70–1.10)	0.26
61–80	0.08 (0.06–0.12)	<0.001	0.09 (0.07–0.12)	<0.001
>80	0.02 (0.12–0.03)	<0.001	0.01 (0.00–0.01)	<0.001
Comorbidity index				
Char 0	1.00 (Reference)		1.00 (Reference)	
Char 1	0.71 (0.63–0.79)	<0.001	0.74 (0.61–0.89)	0.002
Char 2	0.66 (0.57–0.77)	<0.001	0.52 (0.38–0.70)	<0.001
Char $\geq$ 3	0.52 (0.44–0.60)	<0.001	0.51 (0.36–0.72)	<0.001
Genetic translocations				
t(8;21)	2.10 (1.18–3.75)	0.01	2.12 (1.37–3.27)	<0.001
APL ((t(15;17))	1.09 (0.89–1.33)	0.41	0.06 (0.04–0.10)	<0.001
t(11q23)	2.11 (1.14–3.89)	0.02	0.86 (0.51–1.46)	0.58

NOTE: Models also adjusted for year of diagnosis (not shown). A *P* value of <0.05 is considered significant; genetic translocations compares presence or absence of the mutation under study.

race/ethnicity. We found a positive and independent association between treatment and survival; receipt of any treatment improved outcomes for all groups. For black patients, differences in receipt of treatment were associated with a significant reduction in the hazard of mortality, reducing, but not quite neutralizing the survival disparity noted in baseline models.

In agreement with studies of treatment disparities in other cancer types, we found differences in the receipt of treatment for various racial/ethnic groups (12, 14, 17, 18, 29). Our results also support the assertion in previously published studies that disparities may be eliminated by equalizing access to services (20, 30–32). In agreement with studies in other cancer types,

Table 4. Cox proportional hazard models predicting overall mortality (AML, California 1998–2008)

	Model 1, baseline	*P*	Model 2, baseline + treatment	*P*
Race				
White	1.00 (Reference)		1.00 (Reference)	
Black	1.14 (1.04–1.25)	0.004	1.09 (1.00–1.20)	0.05
Hispanic	1.00 (0.94–1.06)	0.99	0.99 (0.94–1.06)	0.86
API	0.90 (0.84–0.97)	0.004	0.91 (0.85–0.98)	0.008
Gender				
Female	1.00 (Reference)		1.00 (Reference)	
Male	1.05 (1.00–1.09)	0.03	1.05 (1.01–1.10)	0.02
Age				
<21	1.00 (Reference)		1.00 (Reference)	
21–40	1.42 (1.23–1.64)	<0.001	1.47 (1.27–1.70)	<0.001
41–60	2.20 (1.90–2.47)	<0.001	2.18 (1.91–2.48)	<0.001
61–80	4.12 (3.62–4.68)	<0.001	3.16 (2.77–3.59)	<0.001
>80	6.58 (5.76–7.52)	<0.001	3.83 (3.34–4.39)	<0.001
Comorbidity index				
Char 0	1.00 (Reference)		1.00 (Reference)	
Char 1	1.19 (1.13–1.25)	<0.001	1.14 (1.08–1.20)	<0.001
Char 2	1.24 (1.16–1.33)	<0.001	1.17 (1.10–1.25)	<0.001
Char $\geq$ 3	1.29 (1.21–1.39)	<0.001	1.19 (1.11–1.27)	<0.001
Genetic translocations				
t(8;21)	0.69 (0.55–0.87)	<0.001	0.72 (0.58–0.91)	<0.001
APL (t(15;17)	0.51 (0.46–0.56)	<0.001	0.48 (0.43–0.54)	<0.001
Treatment				
No chemotherapy	—	—	1.00 (Reference)	
Any chemotherapy	—	—	0.48 (0.46–0.50)	<0.001
No transplant	—	—	1.00 (Reference)	
Transplant	—	—	0.55 (0.50–0.60)	<0.001

NOTE: Model 1, baseline model includes age, gender, race, comorbidity index and year of diagnosis (not shown), and presence or absence of genetic translocations [excluding t(11q23) as this was not associated with survival advantage]. Model 2, baseline model + indicator for treatment compared with none. A *P* value of <0.05 is considered significant.

we demonstrate that survival disparities attenuate toward null for black patients when appropriate treatment is delivered (12, 18, 33). Our study also agrees with previous evaluations that demonstrate no survival disparities for Hispanics after adjustment for age (34–36), and with literature that show improved survival associated with API race/ethnicity in other types of cancers (29, 37).

Despite similarities to other studies, our study is novel because it is one of the first to evaluate differences in treatment and the effect on disparities in AML survival using population-based data (23). Previous evaluations have been limited to single institutions (where patients receive the same effective risk-directed treatment; refs. 30, 31) or clinical trial populations (where all participants meet strict selection criteria and receive protocol-based treatment; refs. 4, 9, 20, 32). Using a custom-built state-wide cancer registry dataset that includes hospital discharge abstracts and focuses on patients with one disease rather than many, we have improved on findings from prior studies, with increased granularity of patient and clinical level factors. Our results demonstrate that there are important differences in treatment associated with specific racial/ethnic groups. Furthermore, our findings suggest the potential to eliminate disparities in AML for select minority groups.

Our results highlight that equitable care delivery may reduce racial/ethnic disparities in cancer. However, because disparities are likely multifactorial, unmeasured factors such as SES and/or location of care may also explain these differences. Future studies should evaluate these factors.

Limitations

Some limitations should be considered for this study. First, our analysis only included the available cytogenetic profiles documented by ICD-9CM coding. These cytogenetic factors are not fully representative of all the possibilities that predict outcome in AML. At this time, all population-based datasets for cancer collect this limited panel of markers. The clinical relevance of the markers is underscored by their ability to indicate targeted treatment such as ATRA for APL subtype; or in their ability to predict who is more likely to respond to bone marrow transplantation (38–41). Nonetheless, in our study, we did not find that higher proportions of favorable genetic subtypes in black and Hispanic patient populations translated into higher odds of receiving clinically indicated care. Our findings, however, demonstrate that the application of these markers must practically include more equitable care otherwise their associated survival benefit cannot be fully leveraged to address disparities. Second, our analysis included many important patient level factors, but did not account for SES factors that have been shown to play a role in disparities in many solid tumors. Further linkage and evaluation of SES variables could improve the assessment of AML disparities in future studies. In addition, our study was limited to treatment as defined as "chemotherapy" and only included first course of therapy. We could not account for any potential differences in the completeness of treatment or use of consolidation therapy. No current population-based registries include this degree of clinical information. However, our work establishes a rational argument for delving deeper into disparities in the quality of treatments delivered. We tried to improve upon this well-known limitation of population-based registries, and used a novel dataset with both inpatient and outpatient treatments, including intensive cytoreductive induction therapy.

Conclusions

Despite limitations, our work has important implications. Using a robust dataset, we correlated racial/ethnic differences in receipt of treatment with worse outcomes. Our results advance our understanding the multifactorial etiologies for racial/ethnic disparities in AML. Because differences in treatment are actionable, the findings from this study suggest that more equitable delivery of care might improve outcomes for racial/ethnic minorities. Future studies should assess the impact of SES factors and/or location of care with differences in outcome.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

Authors' Contributions

Conception and design: M.I. Patel
Development of methodology: M.I. Patel, K.F. Rhoads
Acquisition of data (provided animals, acquired and managed patients, provided facilities, etc.): M.I. Patel, K.F. Rhoads
Analysis and interpretation of data (e.g., statistical analysis, biostatistics, computational analysis): M.I. Patel, Y. Ma
Writing, review, and/or revision of the manuscript: M.I. Patel, Y. Ma, B. Mitchell, K.F. Rhoads
Administrative, technical, or material support (i.e., reporting or organizing data, constructing databases): M.I. Patel, K.F. Rhoads
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