AFRICAN AMERICAN PATIENTS WITH SMOLDERING MULTIPLE MYELOMA MAY HAVE A LOWER RISK OF PROGRESSION COMPARED TO WHITE PATIENTS


topic: 14. Myeloma and other monoclonal gammopathies - Clinical

Theresa Akhlaghi1, Kylee Maclachlan2, Neha Korde2, Sham Mailankody2, Alexander Lesokhin2, Hani Hassoun2, Sydney X. Lu2, Dhwani Patel2, Urvi Shah2, Carlyn Tan2, Andryi Derkach3, Oscar Lahoud4, Heather J. Landau4, Gunjan L. Shah4, Michael Scordo4, David J. Chung4, Sergio A. Giralt4, Saad Z. Usmani2, Ola Landgren5, Malin Hultcrantz2

1 Department of Internal Medicine, Icahn School of Medicine, Mount Sinai Morningside and West, New York, United States; 2 Myeloma Service, Department of Medicine, Memorial Sloan Kettering Cancer Center, New York, United States; 3 Department of Epidemiology and Biostatistics, Memorial Sloan Kettering Cancer Center, New York, United States; 4 Adult Bone Marrow Transplant Service, Department of Medicine, Memorial Sloan Kettering Cancer Center, New York, United States; 5 Myeloma Service, Sylvester Comprehensive Cancer Center, University of Miami, Miami, United States

Background: The incidence of multiple myeloma (MM) and its precursor stages is two to threefold higher in African Americans (AAs) compared to whites when adjusted for socioeconomic status, age, and sex. However, there is limited information on whether racial background affects the risk of progression from smoldering MM (SMM) to MM.

Aims: To assess the effect of race on the progression from SMM to MM.

Methods: Patients with SMM presenting to Memorial Sloan Kettering Cancer Center between the years 2000 and 2019 and who identified as either AA or white were included in this retrospective study. Baseline patient and disease characteristics were collected at the time of diagnosis including laboratory, imaging, and pathology reports. Differences in distributions of continuous and discrete characteristics were assessed by Kruskal-Wallis and chi-square tests. Time to progression (TTP) was assessed using the Kaplan-Meier method with log-rank test for comparisons. Univariate and multivariate Cox proportional hazard models were used to estimate effects of risk factors on TTP with hazard ratios (HR) and 95% confidence intervals (CI).

Results: A total of 576 patients were included (70 were AA, 12%). Median follow-up time was 3 years in AAs and 4 years in whites. Differences in baseline characteristics between AAs and whites included median age (60 years in AAs [IQR 51-67] vs 64 years in whites [IQR 56-72], p = 0.01), median hemoglobin level (12.3g/dL in AA [IQR 11.8-13] vs 12.8g/dL in white [IQR 11.8-13.9], p = 0.02), and immunoparesis including 1 or 2 uninvolved immunoglobulins (31% and 10% in AAs vs 56% and 27% in whites, p = 0.002). There was no difference in bone marrow plasma cell percentage (BMPC), M-spike, free light chain ratio, or Mayo-2018 SMM risk score. AA race was associated with a significantly decreased risk of progression in the univariate model (HR 0.57, CI 0.34-0.94). In the multivariate model adjusting for age, sex, and variables associated with an increased risk of progression in the univariate model (BMPC, M-spike, free light chain ratio, Mayo-2018 SMM risk score), AA race remained associated with a decreased risk of progression (HR 0.39, CI 0.16-0.95). Overall, AA patients with SMM had a significantly (p = 0.027) longer median TTP (9.7 vs 6.2 years), and a lower 2-year (12.6% vs 20.1%) and 5-year (34% vs 44.6%) progression rate than whites. Because AA patients were younger at diagnosis, we stratified patients by age group, < 65 vs ≥65 years. In patients < 65 years, there was no difference in progression rate. In patients aged ≥65 years, AA patients continued to have a longer TTP than whites (9.8 vs 5.2 years, p = 0.02).

Image:
Summary/Conclusion: In our retrospective single institution experience, AA patients with SMM had a lower risk of progression to MM compared to whites. Both groups had similar Mayo-2018 risk scores, however, AA patients had a lower degree of immunoparesis at baseline. Future studies are needed to better understand if these differences are explained by differences in disease biology including genomic mechanisms, immune microenvironment, and systemic immune response.