Review Article

Monoclonal Gammopathies in Africa

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Abstract

People of African descent have a reported higher incidence of multiple myeloma (MM) and increased prevalence of its precursor conditions, monoclonal gammopathy of undetermined significance (MGUS) and smoldering MM (SMM). Despite this, research focusing on people of African descent remains sparse. Even in the absence of robust studies across African populations, major disparities are consistently reported. West Africans and South African Black men have a higher prevalence of MGUS than individuals of European descent. MM has been shown to occur in African individuals at a younger age of diagnosis compared to European individuals, with a relatively higher proportion of females (M/F ~1 vs. 1.4 in Europeans), delayed diagnosis (symptoms to diagnosis 10-12 months), and a higher prevalence of bone disease at presentation. This review summarizes the existing literature on monoclonal gammopathies for African people and highlights critical gaps in our understanding of the disease within the diverse African population. Importantly, differences in disease biology, with respect to cytogenetic and immunologic differences, which contribute to disparate disease outcomes are discussed. Concerted efforts to bridge knowledge gaps through collaborative research initiatives, both within and beyond the African continent, are urgently needed.

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Introduction

Monoclonal gammopathies represent a spectrum of disorders characterized by the presence of clonal plasma cells (PCs), which produce monoclonal immunoglobulins or immunoglobulin light chains.[1,2] These conditions show striking differences in prevalence, biology, and progression among racial groups within the United States (U.S.), particularly among people of African descent.[3,4] People of African descent in the U.S are frequently enriched for Western African or West-Central African genetic ancestry[5] and experience disease onset at an earlier age compared to individuals of European descent.[6] For instance, a U.S. study reported a mean age at diagnosis of multiple myeloma (MM) to be 65.8 years in African Americans, compared to 69.8 years in individuals of European ancestry.[6] In another U.S. population-based study, prevalence of Monoclonal Gammopathy of Undetermined Significance (MGUS) was 6 times higher in Black individuals than White individuals.[7] Similarly, cytogenetic patterns also differ by ancestry.[3,8-10] The prevalence of t(11;14) was approximately 1.4 times higher in individuals with an abnormal PC fluorescence in situ hybridization (FISH) and with ≥ 80% African ancestry compared to those of European ancestry (< 0.1% African ancestry and <30% Asian ancestry). The same study found that African ancestry was associated with a higher likelihood of having t(14;16) or t(14;20), compared to those of European ancestry.[3] However, most studies rely on self-reported race, a poor proxy for Africa's unparalleled genetic diversity, which includes significant heterogeneity across the African continent.[11-14] Additional studies are therefore necessary to confirm these early observations.

The reasons for these variations in disease incidence and presentation, especially among different populations of African descent remain unclear. This review synthesizes current data on monoclonal gammopathies, particularly MGUS and multiple myeloma (MM), across diverse African populations, including North Africans (classified in U.S. census data as Middle Eastern and North African (MENA), and sub-Saharan African communities).[15] The key challenges, opportunities and critical knowledge gaps are explored

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Table 1 Studies on Prevalence of MGUS in Africa

Country	Prevalence	Age Range	Participants	Citation
Ghana	5.84%	50-75	917 Ghanaian men	Landgren *et al.*[17]
Ghana	0	17-95	149 healthy Ghanaian	Oyeyinka *et al.*[20]
Nigeria	0.24%	20-80	410 healthy Nigerians	Onwa et al.[19]
South Africa	8.03%	>30	386 Black South African men	Cicero et al.[18]
South Africa	13.20%	35-80	Random selection of 515 participants from 13,339 Eswatini adult population	Cicero et al.[21]

in this review, offering a roadmap for future research to address these disparities and improve outcomes in underrepresented populations.

Search Methods

We reviewed primary research on monoclonal gammopathies from studies involving individuals of African descent living in Africa, including those conducted by research groups within Africa and by external research groups outside of Africa. In some instances, we also included studies involving African American and European American patients for comparative context. We utilized PubMed, Medline, and Embase databases. These studies reflected the diversity of African populations showcasing the varied cohorts included in this review.

MGUS in Africa: Unraveling Patterns and Prevalence Across a Diverse Continent

The literature on MGUS, the earliest stage of the monoclonal gammopathy spectrum,[16] is limited within Africa (Table 1). Landgren et al.[17] examined 917 Ghanaian men aged 50 to 74 years and reported an age-adjusted prevalence of MGUS of 5.84%, which was notably higher than 3.2% reported for White men over 50 years from Olmsted County, Minnesota, USA. Similarly, Cicero et al.[18] reported an age-adjusted prevalence of 6.79% among 386 Black South African men aged 50 and older, surpassing the rates observed in Western populations. However, in contrast to the studies above, a Nigerian study of 414 individuals between ages 20 to 80 years reported a strikingly low prevalence of 0.24% and similarly another Ghanaian study including 149 individuals aged 17 to 95 years found no cases of MGUS.[19,20] These differences may stem from variations in study demographics, such as age and sex distributions, and/or differences in detection methods.

The prevalence of MGUS in African populations may be associated with an increased occurrence of the light chain subtype, emphasizing the importance of detection methods and the necessity to distinguish between different MGUS subtypes. Cicero et al.[18] highlighted that 39% of MGUS cases among South African men represented light chain only, a subtype that could be missed in studies employing less sensitive techniques. Supporting this observation, a recent study by the same group found a higher prevalence of light chain MGUS (11.1%) in Eswatini (formally known as Swaziland), while the prevalence of conventional (heavy chain) MGUS was 2.14% similar to that of Olmsted County, Minnesota.[21] In another study comparing serum free light chain ratios across different populations, excluding IgG, IgA, IgM MGUS, Black South African population in the study had the highest free light chain levels, followed by African Americans, who also had higher FLC levels than White Americans. The increase was particularly striking in kappa light chains levels, resulting in higher FLC ratios in the Black South African populations relative to the other groups.[22] This study suggests that population-specific reference ranges must be considered when defining light chain MGUS in Africans, as using the reference range determined primarily from White populations may overestimate the prevalence of MGUS in Africans.[22]

Unique risk factors may partially drive the prevalence of MGUS in African populations. Among Black South African men, human immunodeficiency virus (HIV) infection (OR: 2.39) and cigarette smoking (OR: 5.6) were associated with increased MGUS risk.[18] Additionally, both the South African and Ghanaian studies (Landgren et al.) reported a predominance of IgG isotypes and in South Africa, 78% of MGUS cases presented with at least 1 International Myeloma Working Group (IMWG) high risk feature for progression to MM. Although these findings are compelling, many questions about MGUS remain unanswered. The role of light chain MGUS, the impact of HIV, and the natural progression from MGUS to MM in African populations remains poorly understood. These studies underscore the need for larger, methodologically robust investigations to untangle the biological, environmental, and demographic factors that shape MGUS across the African continent.

Multiple Myeloma in Africa (MM)

Epidemiological and Clinical Landscape

The incidence and characteristics of MM vary significantly across Africa. According to the Global Cancer Observatory (GLOBOCAN) database, the age-standardized incidence rate (ASI) of MM in Africa spans from no reported cases in some regions, such as Sierra Leone, to 3.5 per 100,000 in Zimbabwe.[23] Likewise, the Global Burden of Disease study estimated ASI rates of MM to be 1.4, 0.8, and 2.2 per 100,000 for East, West, and Southern African populations, respectively.[24] Analysis of 25 cancer registries across 20 sub-Saharan African countries reported ASI values as low as 0.1 per 100,000 population in West Africa (e.g., Côte d'Ivoire, Abidjan), 0.4 per 100,000 in East African populations (e.g., Ethiopia, Addis Ababa), and 0.6 per 100,000 in South African populations (e.g., South African females in 2007).[25] In contrast, studies in the U.S. report significantly higher age-standardized incidence rates of 11 and 4.9 per 100,000 for Black and White populations, respectively.[6] These differences may be attributed to lower detection rates of MM within the African continent. Additionally, delays in MM diagnosis have also been reported in some countries. For instance, in

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Ghana and Senegal, the time from symptom onset to MM diagnosis averages 10 to 12 months, compared to 3 months in the U.S.[26-28]

The median age of onset of MM across regions in Africa ranges from 58 to 64 years, compared to 66 years for African Americans and 70 years for populations of predominantly European ancestry in a U.S based population study.[26,29-34] Individuals from West Africa are reported to present with a median age between 58 and 59 years,[26,30] whereas patients from North Africa present with a slightly elevated median age of 61 to 63 years. Consistent with these findings, studies from Kenya, Nigeria, Libya, and Ghana indicate that 16% to 29% of MM cases occur in individuals under 50 years of age, compared to approximately 10% in European populations.[26,30-32,34,35] The earlier age of onset of MM in Africa compared to the U.S. may be partially due to differences in overall life expectancy. Further, studies from U.S. and Europe report a male predominance (male-to-female ratio of approximately 1.5:1),[31] whereas data from Africa show greater variability, with some studies reporting equal incidence between sexes and others suggesting a slight predominance in females.[26,27,30,33,34,36] Immunoglobulin G (IgG) is the most common isotype identified in African MM studies, followed by IgA.[26,27,34,36,37] A higher prevalence of kappa light chains compared to lambda light chain subtypes has been observed.[26,27,33,34,36,38] Light chain MM constitutes about 9% to 10% of cases in African studies,[27,39] compared with 20% of cases in predominantly White populations.[31] The immunoglobulin isotype in MM is associated with differences in disease biology, with non-IgG subtypes often linked to more aggressive clinical behavior and poorer prognosis. Based on the studies reviewed, the pattern of Ig subtype in Africans is similar to those reported in other non-African populations.[31,40]

Infections

Infections such as hepatitis C virus (HCV) and HIV may influence MM presentation and prognosis in African patients.[41-43] MM patients with HIV tend to be younger at presentation, with fewer bone lesions, less renal impairment, exhibit higher $CD4^+$ *T* cell counts than HIV positive non-Hodgkin lymphoma patients (controls) and demonstrate a normal kappa to lambda ratio and lower monoclonal protein levels than MM patients without HIV.[41] Notably, only 2 of the 16 HIV-positive MM patients in the study were reported to be on highly active antiretroviral therapy (HAART). Another South African study of a predominantly Black cohort (81.5% of participants) reported a 21.5% prevalence of HIV infection among MM patients, with no differences in outcomes between HIV-positive and HIV-negative individuals.[43] These findings, along with other epidemiological and clinical observations, underscore the heterogeneity of MM across populations and highlight the influence of regional, genetic and environmental factors on disease presentation, progression and outcomes.

Immunophenotypic Findings

Few publications characterize the immunophenotype of MM patients in Africa. Among 83 Kenyan patients, reduced expression of CD56 and CD117 in MM clones was observed compared with to European and Asian cohorts.[44] An Egyptian study noted a shift in monocyte subsets, with lower classical monocytes ($CD16^-/CD14^+$) and elevated nonclassical ($CD16^{++}/CD14^+$) and intermediate ($CD16^+/CD14^{++}$) monocytes compared to healthy control participants.[45,46] In newly diagnosed MM patients from Egypt, the immune derangement is marked by reduced $CD4^+$ helper T cells, NK cells, and NKT cells, with an increase in cytotoxic $CD8^+$ *T* cells in the blood compared to healthy controls.[47] Reduced NK cells at diagnosis was associated with poorer prognosis.[47] The observation of reduced $CD14^+$ classic monocytes and elevated $CD16^+$ nonclassical monocytes in the peripheral blood and reduced CD4+ helper T-cells along with increased cytotoxic $CD8^+$ *T* cells in the blood was consistent with previously reported data from studies conducted in White populations.[48-52] However, future studies are needed to comprehensively compare the immune tumor microenvironment between White, African American and African populations.

Cytogenetic Findings

Cytogenetic abnormalities in MM significantly impact prognosis and treatment decisions. However, our understanding of these abnormalities in African populations is incomplete due to the limited use of FISH, a crucial tool for identifying recurrent cytogenetic abnormalities in MM cells.[43,53-58] Most reports have small sample size and are from Northern Africa including studies from Egypt ($n = 60$), Tunisia ($n = 70$), Morocco ($n = 93$), and Sudan ($n = 15$). One study reported cytogenetic findings in South African patients with MM ($n = 75$)[43,53-56] and there is limited data on cytogenetic abnormalities in patients with MM from other regions of Sub-Saharan Africa. Similar to European populations, IGH translocations were commonly reported in 26% to 45.7% of Moroccan and Tunisian populations.[55,56] Translocation(11;14), was observed in 26% of Tunisian MM patients, and In Egypt t(4;14) (10%) was more prevalent than t(11;14) (7%).[53] The prevalence of t(4:14) in Moroccan and Tunisian patients with MM was 14% and 11.4% , respectively.[53,55,56] Deletion of 17p varied between different countries, with incidences of 12%, 6% , 12% , and 5% in Morocco, Tunisia, Egypt, and South Africa, respectively.[43,53,55,56] Evaluation of 13q deletion showed diverse rates of 19%, 7%, 55%, and 19% in Tunisia, Egypt, Sudan, and the South African studies, respectively.[43,53,55,56] Only the Egyptian study correlated cytogenetic data with outcomes, highlighting that hyperdiploidy correlated with a better prognosis, while 17p and t(4:14) abnormalities indicated a worse prognosis, aligning with the established risk patterns of cytogenetic abnormalities.[53] Further studies incorporating larger genome sequencing studies of patients with MM in Africa are needed to validate these findings. A summary of FISH findings in African studies are presented in Table 2, with a comparison of FISH data from U.S. studies.[3,9,40]

Pattern of Clinical Presentations, Diagnosis, Treatment Outcomes, and Challenges

Bone pain is a prominent presenting symptom in African patients with MM, seen in 78-93% with more than 50% presenting with fractures at the time of diagnosis.[26,29,30,33,34,59] In contrast, African Americans and European Americans have a lower incidence of fractures at diagnosis, with rates of 16.7% and 23.5% respectively,[60] compared to African patients, this suggests that bone disease

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Table 2 Cytogenetics in MM in African Studies Versus Previous Report in African American and Europeans

Available Data From African Studies							Comparative Data From Published Articles				
Study Location	Morocco (Hamdaoui *et al.*[56])	Tunisia (Gmidene A et al.[55])	Egypt (Abel-Qader et al.[53])	Sudan (Dowd et al.[54])	South Africa (Chili LH et al.[43])	African American (A.J Greenberg et al.[9])	Non-Hispanic Black (Kaur et al.[40])	African descent (Baughn et al.[3])	White (A.J Greenberg *et al.*[9])	Non-Hispanic White (Kaur *et al.*[40])	European descent (Baughn et al.[3])
Year of publication	2020	2012	2022	2015	2013	2015	2021	2018	2015	2021	2018
Sample size (n)	93	70	60	15	75 For FISH, 36 Karyotyping	292	303	120	471	80	235
Conventional cytogenetics n (%)											
Normal	35 (78)		15 (25)		32 (88.9)						
Abnormal	10 (22)	13 (18.5)	45 (75)		49 (11.1)						
Complex	4 (9)	7 (10)									
Hyperdiploid	4 (9)		21 (35)				40 (13.2)			11 (13.7)	
Nonhyperdiploid	6 (13)		39 (65)				22 (7.3)			9 (11.2)	
Normal or none of the studied abnormalities were observed						185 (63.4)	241 (79.5)		163 (34.6)	60 (75)	
IGH translocations n (%)	(40)[a]	32 (45.7)						78 (65)			125 (53.2)
t(11:14)		18 (25.7)	4 (6.7)			19 (6.5)	70 (23.1)	46 (38.3)	83 (17.6)	17 (22)	63 (26.8)
t(4:14)	13 (14)	8 (11.4)	6 (10)		1 (1.4)	16 (5.5)	3 (1)	8 (6.7)	47 (10)	0	20 (8.5)
t(6:14)			1 (1.7)					1 (0.8)			4 (1.7)
t(14:16)		2 (2.45)					3 (1)	11 (9.2)		2 (2.6)	9 (3.8)
1q21 duplication/amplification n (%)	12 (13)							(25)			(29.2)
17p deletion n (%)	11 (12)	4 (5.7)	7 (11.7)		4 (5.3)	23 (7.9)	23 (7.6)	8 (6.7)	61 (13)	6 (7.8)	32 (13.7)
1p deletion n (%)	4 (4)										
11q deletion n (%)					14 (21.5)						
13q deletion n (%)		13 (18.6)	4 (6.7)	6 (54.5)	14 (18.9)	85 (29.1)		41 (34.2)	223 (47.3)		91 (38.7)
14q deletion/Monosomy 14 n (%)	4 (4)		2 (3.3)								

Details of fluorescence in situ hybridization (FISH) probes used across studies are summarized below:

Morocco (Hamdaoui et al.): 1. IGH/FGFR3 (t[4;14]) dual color/dual fusion (DC/DF) probe (Vysis) 2. TP53/CEP17 probe for 17p13.1 (Vysis) 3. 1q21 CKS1B/1p32 CDKN2C probe (Vysis).

Tunisia (Gmidene et al.): Locus-specific probes targeting:

1. IGH (14q32), D13S319 (13q14) 2. RB1 (13q14) 3. TP53 (17p13).

Egypt (Abdel-Qader et al.): 1. LSI D13S319 (13q14.3), LSI IGH break-apart probe (14q32) 2. LSI IGH/CCND1 dual fusion probe 3. LSI TP53/CEP17 probe 4. LSI D5S23/D5S721/CEP9/CEP15 5. LSI IGH/FGFR3 dual fusion probe.

Sudan- Dowd et. Al- Fluorophore labeled DLEU1 (13q14) LSI (local specific identifier) probe

South Africa- 1. 13q14.3 deletion (D13S319) 2. 17p13.1 /11q22.3 deletion TP53/ATM 3. 11q22 deletion ATM 4. t(4;14) IGH@/FGFR3/WHSC1

A J Greenberg et al.- Probe details not available

Kaur et al.- Probe details Not available

Baughn LB et al.- 1. RB1/LAMP1 (13q14) 2. TP53/D17Z1 (17p) 3. D3Z1/D7Z1/D9Z1/D15Z4 (enumeration probe for chromosomes 3, 7, 9 and 15) , 4. TP73/1q22 (1q22) 5. (MYC 8q24.1) rearrangement 6. IgH (custom probe) for 14q32 rearrangements and probes targeting individual IGH rearrangement. *Deletion or monosomy of chromosomes 13 and 17 and copy number gain of 1q were detected using enumeration strategy probes. Centromere probes were used to detect chromosomal aneusomy of chromosomes 3, 7, 9, and 15. Of note, normal cytogenetic cases were eliminated. Only those with a MM cytogenetic abnormality were included.*

Baughn LB et al.- 1. RB1/LAMP1 (13q14) 2. TP53/D17Z1 (17p) 3. D3Z1/D7Z1/D9Z1/D15Z4 (enumeration probe for chromosomes 3, 7, 9 and 15) , 4. TP73/1q22 (1q22) 5. (MYC 8q24.1) rearrangement 6. IgH (custom probe) for 14q32 rearrangements and probes targeting individual IGH rearrangement.

CEP = chromosome enumeration probe; DC/DF = dual color/dual fusion; LSI = locus-specific identifier.

[a] The number (n) was not mentioned.Countries represented include: Morocco, Tunisia, Sudan, Egypt, South Africa.

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may be more common and severe in Africans than in American populations. Another indicator of more severe disease is low hemoglobin(Hb) levels (<10g/dl), Hb levels <10g/dl were reported in 67% of Ghanaian MM patients (37.3% had Hb<7g/dL), 71% of Kenyan MM patients (26% had Hb<7g/dl) and in 77% Nigerian MM patients at diagnosis,[26,29,30] while hemoglobin of <10g/dl was reported in only 16.9% Europeans and 21.9% of African Americans with MM.[60] Whether bone disease and severe anemia signify a difference in disease biology or reflect late presentation requires further evaluation. Delayed diagnosis in African patients with MM may contribute to more advanced disease presentation, as most African patients present at stage III (70% to 77.9% according to either the International Staging System (ISS) or Durie-Salmon staging).[26,27,34,44,61] Interestingly, these findings are consistent with our recently published comparison of laboratory data between MM patients in Ghana and those in the U.S.[62]

Fewer patients from Africa received bortezomib-based treatments, while regimens such as Vincristine-Adriamycin-Dexamethasone (VAD) and melphalan-based treatments, with or without thalidomide, were more commonly utilized.[27,30,33,34] Additionally, 2 of these studies compared overall survival, between the few patients who had bortezomib-based treatment vs melphalan based or VAD treatment regimens and found no significant difference.[27,34] In 1 study, 28% of patients were untreated and had inferior median OS of 27.3 months, compared to 50.6 months in treated patients; 79.6% of those treated received a combination of melphalan, prednisolone, and thalidomide.[63] This study reported a higher median OS than most other African studies, which typically report OS in the range of 20–22 months.[26,27,33,34,64] The relatively better survival in the study may be attributable to the more recent cohort, with 59 of 75 patients seen in 2020, and the use of more uniform treatment regimens. Overall, survival outcomes for African patients with MM remain significantly lower than those reported in high-income countries such as the U.S., where median OS exceeds 64 months (Supplemental Table 1 and 2). Autologous stem cell transplantation (ASCT), a key component of standard MM treatment worldwide, remains largely inaccessible across most of Africa, its use and impact on patient outcomes are documented mostly in studies from North African countries with findings consistent with improved survival.[38,65]

Amyloidosis and Other Monoclonal Gammopathies

There were few studies examining SMM, plasma cell leukemia, AL amyloidosis, solitary plasmacytoma, Waldenström macroglobulinemia or POEMS syndrome in Africa, underscoring the difficulty in diagnosing already rare and clinically elusive conditions, especially in areas with limited resources. A study evaluating renal amyloidosis in South Africa reported AL amyloidosis in all 7 White participants, 7 of 17 Black, and 12 of 22 mixed-race individuals, suggesting a higher prevalence of AL amyloidosis in White compared to Black, in another study on renal amyloidosis in Tunisian population, AL amyloidosis was reported I 9 out of 40 participants.[66,67] The role of radiotherapy was assessed in a study of 12 patients with solitary plasmacytoma, with findings indicating a median age of 53.8 years at presentation, a male predominance, and a good response to radiotherapy alone.[68] Finally, there have been few reports on the involvement of Africans in MM clinical trials.[69]

Discussion and Recommendations

The landscape of monoclonal gammopathies in Africa remains largely uncharted, particularly for MGUS and SMM. Most studies have focused on MM, often with small sample sizes, leaving the biology and progression of the precursor conditions poorly understood. The lack of routine health screening across much of Africa likely contributes to the underdiagnosis of MGUS.[58] Established screening programs, as demonstrated by Iceland's national initiative, provides a compelling example of how routine population level screening could significantly enhance early detection and decrease the deleterious disease defining events of MM.[58,70] Though there are thriving African economies, developing robust health infrastructure across Africa faces significant challenges, due to limited financial resources, shortage of skilled medical professionals, lack of facilities and inadequate access to diagnostics. Collaborative efforts between governments, nongovernmental organizations and international partners are essential to address these multifaceted challenges.[71,72] The International Myeloma Foundation, a nonprofit organization supporting patients with MM that has invested in various initiatives in underserved U.S. and Asian communities, offers a roadmap for improving diagnostics, care access, and research infrastructure in Africa. These, among other collaborations, have improved the quality of MM care in different countries.[73] In addition to the lower Hb at presentation, renal failure and advanced disease stage which may contribute significantly to shortened survival, transplant facilities and novel MM therapy are sparsely available in most parts of Africa.[24]

A higher prevalence of MGUS has been reported in Africans and among African Americans compared to European Americans.[74] Genetic variation may be a contributing factor, as the risk of developing MM is known to be higher in individuals with a family history of the disease, a risk that has been reported to be even greater in African Americans.[75] Similarly, a meta-analysis of genome-wide association studies (GWAS) among individuals of African ancestry identified genetic variations that may contribute to the increased risk of MM in this population.[76] This review also highlighted the possible association between HIV infection and MGUS, with studies reporting an even higher prevalence of MGUS among African patients with HIV.[77] Southern Africa has the highest prevalence of HIV infection globally,[78] however little is known about the impact of HIV on MGUS progression. Chronic antigenic stimulation may be a trigger for clonal expansion of plasma cells in the pathogenesis of MGUS.[79] Larger prospective studies are needed to further characterize the pattern and risk stratification in MGUS, as well as to investigate the role of HIV infection in the etiology and natural history of MGUS.

African patients with MM are more likely to have an earlier age of disease onset and are diagnosed at more advanced stages when compared to Europeans. Interestingly, the proportion of females with MM in Africa is equal or slightly higher, in contrast to the male predominance observed in other geographical regions[31]; whether this difference is explained by increased health-seeking behavior or higher life expectancy among women in Africa is unclear.[80]

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While some studies have explored differences in the disease biology between patients with MM in Africa and other geographic areas, there is still a significant knowledge gap. Even within Africa, variations exist and differences in the proportions of cytogenetic abnormalities may be due to the heterogeneity of populations across the continent, making direct comparisons with non-African populations challenging.[81] Most research projects about disease biology, including cytogenetic abnormalities and immunologic profiles describe patients from North Africa, leaving vast gaps in understanding MM across the diverse African continent. Recognizing the disparities in disease presentation and prognosis is a crucial step towards addressing the unique challenges faced by individuals of African ancestry.

Highlighting the unique challenges faced by MM patients in Africa, we have an opportunity to address disparities, uncover new insights into disease biology, and ultimately improve outcomes for a historically underserved population. Addressing these gaps requires robust genomic studies, incorporation of ancestry-specific variables, and systematic evaluation of clinical and biological features unique to African populations. By prioritizing these efforts, the global community can better address the unique challenges of MM in Africa, paving the way for equitable care and improved outcomes.

Disclosure

The authors declare no relevant competing interests.

CRediT authorship contribution statement

Abiola Bolarinwa: Writing – review & editing, Writing – original draft, Conceptualization. **Lateef Odukoya:** Writing – review & editing. **Francis Buadi:** Writing – review & editing. **Vincent Rajkumar:** Writing – review & editing. **Shaji Kumar:** Writing – review & editing, Conceptualization. **Celine Vachon:** Writing – review & editing, Conceptualization. **Lily Paemka:** Writing – review & editing. **Linda B Baughn:** Writing – review & editing, Conceptualization. **Joselle M Cook:** Writing – review & editing.

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Supplementary materials

Supplementary Table 1: Presenting Laboratory data of African patients with MM.

Title	year	Period of participant's diagnosis	Country	Median Calcium level (mg/dl)	Percentage of participants with Calcium >11mg/dl	Median Creatinine in mg/dl	Percentage of participants with creatinine >2mg/dl	Median Hemoglobin levels in g/dl	% with Hb < 10g/dl	Albumin (g/dl)	Percentage of participants with serum Albumin <3.5g/dl
Presentation and survival of multiple myeloma patients in Ghana: a review of 169 cases.	2019	2002-2016	Ghana	10.4	35.80	1.3	32.7	7.9(3-16)	75.8	3.2	60.6
Diagnostic and evolutionary profile of multiple myeloma in Senegal: single-center study from 2005 to 2016	2017	2005-2016	Senegal				36.7				
Globulin fraction and albumin: globulin ratio as a predictor of mortality in a South African multiple myeloma cohort	2020	2003-2018	South Africa	11.43		3.8		7.9			
Clinical Profile and Treatment of Multiple Myeloma at a Tertiary Hospital in Kenya: A Five-Year Retrospective Review	2024	2014-2018	Kenya		55.40		38.2		71		
Characteristics and outcomes of patients with multiple myeloma at the Uganda Cancer Institute	2021	2008-2012	Uganda	9.6		1.15				3.18	
Multiple myeloma in Nigeria: a multi-center epidemiological and biomedical study	2015	2005-2014	Nigeria					8.3		3.4	
Laboratory Features of Newly Diagnosed Multiple Myeloma Patients	2019	2010-2017	Libya	9.4		2.9		10.2	50.5	3.4	
Retrospective Analysis of Presentation, Treatment, and Outcomes of Multiple Myeloma at a Large Public Referral Hospital in Eldoret, Kenya	2021	2009-2019	Kenya	10.5	54.80	0.84	13.7	10.3	46.5	3	64.1
Concomitant HIV infection in newly diagnosed multiple myeloma patients is hard to recognize and should be tested for routinely in areas of high endemicity	2017	2004-2011	South Africa			1.02		9.7			
Profile and outcome of multiple myeloma with and without HIV treated at a tertiary hospital in KwaZulu-Natal, South Africa	2023	2015-2020	South Africa		11.9		32.6		66.7		
Immunophenotypic expression profile of multiple myeloma cases at a tertiary hospital in Nairobi Kenya	2023	2009-2020	Kenya		17.5		40		50		52.2
Multiple myeloma: diagnostic, therapeutic and prognostic particularities of 123 cases collected at the Avicenne Military Hospital in Marrakech	2021	2012-2019	Morocco		25		28.5				50
Description of 48 cases of multiple myeloma seen at the hematology laboratory, Joseph Ravoahangy Andrianavalona University Hospital, Antananarivo, Madagascar	2017	2009-2015	Madagasca						79.2		
Musculoskeletal presentation of multiple myeloma at general Hospital douala, Cameroon Hospital Douala, Cameroon	2014	2007-2013	Cameroun					9.4			

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Supplementary Table 2 Clinical course and treatment details of MM patients in Africa

Title	year	period of participant's diagnosis	country	sample size	Median age of onset in years.	Males n(%)	Bone disease n(%)	Most prevalent symptoms at diagnosis	Number (Percentage) with ISS stage III Myeloma	Regimen used	Median Overall Survival in months
Presentation and survival of multiple myeloma patients in Ghana: a review of 169 cases.	2019	2002-2016	Ghana	169	58	87(51.5)	102(76.1)	Bone pain	39(50)	Vincristine-Adriamycin-Dexamethasone/ Melphalan-prednisolone	33
Diagnostic and evolutionary profile of multiple myeloma in Senegal: single-center study from 2005 to 2016	2017	2005-2016	Senegal	136	58.8*	69(50.5)	119(88.1)	Bone pain	19(59)	Melphalan/ prednisolone	20
Clinical Profile and Treatment of Multiple Myeloma at a Tertiary Hospital in Kenya: A Five-Year Retrospective Review	2024	2014-2018	Kenya	207	60	113(54.6)	126(91.9)	Bone pain		Melphalan based	
Globulin fraction and albumin: globulin ratio as a predictor of mortality in a South African multiple myeloma cohort	2020	2003-2018	South Africa	65 (59 Black,2 White African. 1 mixed race.)	58.57*	59(50.7)					
Epidemiological, clinical and prognostic aspects of multiple myeloma eligible for therapeutic intensification followed by autologous hematopoietic stem cell in the Algerian West: report of 147 cases	2017	2008-2015	Algeria	147	53	89(60.5)			29(30.8)	VD/VTD	30
Overview of Multiple Myeloma in Rwanda	2017	2011-2017	Rwanda	33	44.6						
Characteristics and outcomes of patients with multiple myeloma at the Uganda Cancer Institute		2008-2012	Uganda	217	59*	119(55)	111(69)			MP/MPT	30
Multiple myeloma in Nigeria: a multi-center epidemiological and biomedical study	2015	2005-2014	Nigeria	135	60	70(58.9)				MP/MPT	
Laboratory Features of Newly Diagnosed Multiple Myeloma Patients	2019	2010-2017	Libya	99	61.8*	42(42.4)					
Retrospective Analysis of Presentation, Treatment, and Outcomes of Multiple Myeloma at a Large Public Referral Hospital in Eldoret, Kenya	2021	2009-2019	Kenya	221	61	124(56.1)	60(58.8)	Bone pain	4(36.5)	TD/VTD	29
Concomitant HIV infection in newly diagnosed multiple myeloma patients is hard to recognize and should be tested for routinely in areas of high endemicity	2017	2004-2011	South Africa	146	59+	49(55)	76(89.4)	Bone pain	51(56.7)		
Profile and outcome of multiple myeloma with and without HIV treated at a tertiary hospital in KwaZulu-Natal, South Africa	2023	2015-2020	South Africa	136 (116 of African ethnicity)	58	61(45.2)		bone pain	49(49)	CTD	18
Immunophenotypic expression profile of multiple myeloma cases at a tertiary hospital in Nairobi Kenya	2023	2009-2020	Kenya	67	61	47(52.2)			21(56.8)		
Cytogenetic and FISH analysis of 93 multiple myeloma Moroccan patients	2020	2017-2018	Morocco	93	61.9	49(54)					
Therapeutic results of bortezomib multiple myeloma treatment: A monocentric study. HemaSphere. 2021:799-.	2021	2016-2019	Tunisia							Bortezomib	26
Multiple myeloma: diagnostic, therapeutic and prognostic particularities of 123 cases collected at the Avicenne Military Hospital in Marrakech	2021	2012-2019	Morocco	123	62.5*	98(79.7)		Bone pain			
Distribution and features of hematological malignancies in Eastern Morocco: a retrospective multicenter study over 5 years.	2016	2008-2012	Morocco	82	63.5	44(53.7)					
Hematological malignancies in East Africa-Which cancers to expect and how to provide services.	2020	2016-2019	Tanzania	43	58	24(56)					
Epidemiological and immunochemical parameters of monoclonal plasma cell dyscrasias of 2121 cases in Algeria.	2020	1998-2016	Algeria	1171	63.95*	569(48.6)					
A descriptive study of plasma cell dyscrasias in Egyptian population	2014	2000-2010	Egypt	217							
Description of 48 cases of multiple myeloma seen at the hematology laboratory, Joseph Ravoahangy Andrianavalona University Hospital, Antananarivo, Madagascar	2017	2009-2015	Madagascar	48	64.2	26(54.5)					
Musculoskeletal presentation of multiple myeloma at generalhospital douala, cameroon	2014	2007-2013	Cameroun	62	57*	24(38.7)		Bone pain			

*- mean age, + - The study reported median age of 59 for HIV negative participants and 50 years for HIV positive participants

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