Acta Biomed 2025; Vol. 96, N. 4: 16610 DOI: 10.23750/abm.v96i4.16610

Case report

Cutaneous plasmablastic plasmacytoma: A case report with literature review

Khalid M. AlHusain[1], Haya A. AlHemli[2], Meshal A. Bukhari[3], Nadya A. Al-Faraidy[4], Lenah Y. Shaikh[4], Reem AlRabeh[5], Ayed AlGarni[5]

[1]Department of Dermatology, College of Medicine, Imam Abdulrahman Bin Faisal University, Dammam, Eastern Province, Saudi Arabia; [2]College of Medicine, Imam Abdulrahman bin Faisal University, Dammam, Eastern Province, Saudi Arabia; [3]Department of Medicine, Dermatology Section, E1 Health cluster, Eastern Province, Saudi Arabia; [4]Department of Medicine, Dermatology Section, King Fahad Specialist Hospital, Dammam, Eastern Province, Saudi Arabia; [5]Department of Pathology, King Fahad Specialist Hospital, Dammam, Eastern Province, Saudi Arabia

Abstract: Multiple myeloma (MM) is a hematologic malignancy often involving bone marrow and osteolytic lesions. Cutaneous involvement in MM is rare and typically signifies advanced disease. Here, we present a rare case of MM with cutaneous involvement and provide an updated review of the literature on this unusual dermatologic presentation as latest comprehensive review on cutaneous plasmacytoma dates back to 2003. A 50-year-old male with sacral plasmacytoma and elevated immunoglobulin G (IgG) was diagnosed with IgG Kappa MM, ISS stage II. After receiving radiation, chemotherapy, and stem cell transplantation, he achieved a very good partial response and began maintenance therapy with bortezomib-dexamethasone. Three years post-diagnosis, he developed biopsy-confirmed cutaneous plasmacytomas. A retrospective review of case series and reports from 2003 to 2024 using PubMed database identified 11 publications encompassing 27 patients. Our review found that IgG was the most common immunoglobulin subtype in cutaneous plasmacytoma (11/27), primarily affecting older males (15/27) (median age 73). Clinically, nodular lesions (17/27) were the most frequent presentation, predominantly located on the chest area (7/27). Histologically, plasmacytic cellular morphology (12/27) and plasmablastic morphology (11/27) were nearly equally prevalent. Prognosis varied from days to four years post-diagnosis. Despite numerous case reports, significant gaps remain in our understanding of cutaneous plasmacytoma, its risk factors, and clinical associations. Further research should prioritize prospective studies with larger, more diverse cohorts to enhance comprehension of the disease, improve diagnostic accuracy, refine treatment approaches, and ultimately improve patient outcomes. (www.actabiomedica.it)

Key words: cutaneous plasmacytoma, case report, multiple myeloma, plasmablastic morphology

Introduction

Multiple myeloma (MM), a hematologic malignancy that originates from the uncontrolled proliferation of monoclonal plasma cells (1). The classical triad of MM includes plasmacytosis in the bone marrow (affecting over 10% of cases), the presence of lytic bone lesions, and detectable monoclonal immunoglobulin (Ig) in serum and urine (1). While MM primarily affects the bone marrow, extramedullary involvement has been documented in various sites, including the respiratory tract, oropharynx, gastrointestinal tract, spleen, lymph nodes, and rarely the skin (2). Skin infiltration by malignant MM cells can lead to the development of plasmacytomas, typically resulting from direct extension from underlying bone lesions (2).

In rare cases, however, metastatic spread to the skin may occur through hematologic or lymphatic pathways without adjacent bone involvement, often signifying an advanced stage of MM with substantial tumor burden (3). In this report, we present a rare case of MM metastasizing to the skin in the absence of underlying bone lesions. This case report, along with an updated literature review, provides insights into patient demographics, clinical presentation, pathological and serological findings, and the prognosis associated with cutaneous metastasis in MM.

Case Report

A 50-year-old male with a history of cervical Hodgkin lymphoma in the right neck, treated 25 years prior with surgical excision and radiotherapy, presented to the emergency department with progressively worsening back pain unresponsive to analgesics. His condition had recently worsened, with symptoms including acute urinary retention, saddle anesthesia, dyschezia, and right leg weakness. Initial radiological assessments included magnetic resonance imaging (MRI) of the lumbosacral spine and computed tomography (CT) of the chest, abdomen, and pelvis. MRI revealed a presacral mass infiltrating the sacral neural foramina, while CT identified a mass in the right vertebral body at T10. A biopsy of the sacral mass confirmed a sacral plasmacytoma. Further investigation for MM yielded the following findings: serum IgG level of 50.1 g/L (normal range: 5.4-18.2 g/L), IgA level of 0.4 g/L (0.06-4.8 g/L), and IgM level of 0.3 g/L (0.2-2.4 g/L). Bone marrow biopsy showed 40% plasma cell infiltration. Serum and urine immunofixation confirmed IgG Kappa, establishing a diagnosis of IgG Kappa MM, ISS stage II.

Additional MM workup results included:

- SPEP: M band 48.4 G/L
- Immunofixation: IgG Kappa
- Serum Free Light Chain: Kappa 157, Lambda 1.91, Ratio 82.2
- Urine Free Light Chain: Kappa 26.5, Lambda 0.5, Ratio 53.0
- Beta-2 Microglobulin: 2.3 mg/L
- LDH: 117 U/L

The patient initially received 20 Gy of radiation therapy in 5 fractions from 24/12/2020 to 29/12/2020, with remarkable clinical improvement. VRD (lenalidomide, bortezomib, and dexamethasone) was initiated on 31/12/2020 but was discontinued on 5/4/2021 Treatment was discontinued due to grade 4 neutropenia and multiple interruptions. During VRD, cycle 1 lenalidomide was administered for only one week before discontinuation due to neutropenia; cycle 2 was interrupted for three weeks due to pneumonia; cycle 3 was rescheduled for one week due to COVID-19 vaccination. Despite stable M protein, FLC kappa showed progression to >13600. The patient subsequently received two cycles of KPd (Carfilzomib, Pomalidomide, Dexamethasone) starting 12/4/2021. Due to neutropenia after the first cycle, cycle 2 was modified to pomalidomide 4mg every other day alternating with GCSF. Partial remission was achieved after the second cycle. Autologous stem cell transplantation with melphalan conditioning was initiated on 24/6/2021. By July, the patient achieved very good partial response with normalization of FLC and M-band of 2.6. Due to grade 3-4 neutropenia with immunomodulatory drugs, maintenance therapy with biweekly bortezomib-dexamethasone was initiated on 14/9/2021. Relapsed in the form of multiple cutaneous plasmacyomas in June 2023 with no clear bone marrow involvement but started to show biochemical progression July 2023.

On 27/7/2023 the patient presented to the Dermatology Outpatient Clinic with two non-pruritic nodules, persisting for three months. Physical examination revealed a well-demarcated, deep red, firm, smooth-surfaced nodule measuring 5x2 cm on the right arm (Figure 1) and a round, violaceous, firm nodule with a verrucous surface measuring approximately 4x5 cm on the back (Figure 2). The nodule on the back exhibited bleeding and had increased in size. Notably, this second nodule was associated with an underlying osteolytic lesion, whereas the first nodule was not.

Biopsies of both nodules were performed. Histological analysis of the skin biopsy showed diffuse dermal infiltration by atypical plasma cells, abutting the dermal-epidermal junction. The infiltrate was composed of pleomorphic cells with abundant cytoplasm, large nuclei, and prominent nucleoli (plasmablasts),

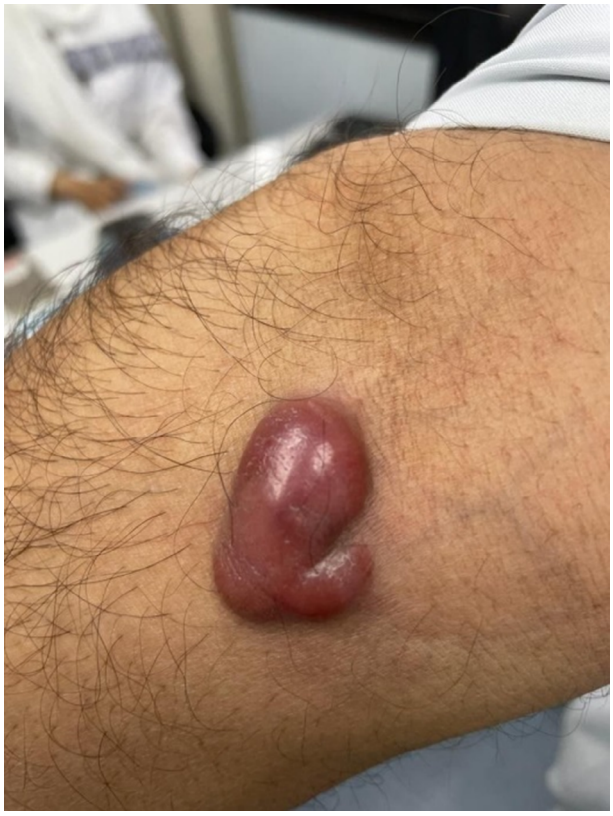

Figure 1. Purplish and erythematous nodule on the right arm.

with mature plasma cells exhibiting "clockface" chromatin rarely seen (red arrow) and scattered mitotic figures (black arrow) (Figure 3). The cells demonstrated a combination of nodular and diffuse growth patterns. Immunohistochemistry (with epithelial internal control) showed diffuse CD138 staining within the infiltrate, and κ light chain restriction was observed, consistent with a diagnosis of cutaneous plasmacytoma. Following the histopathological diagnosis, a PET-CT scan was performed. It revealed several intensely fluorodeoxyglucose (FDG)-avid cutaneous and subcutaneous lesions, including a large lesion in the lower back measuring 5.7 × 3.6 cm with a standardized uptake value (SUV) max of 16.80, consistent with disease relapse, and a lesion in the forearm measuring 2.5 × 1.9 cm with an SUV max of 12.10. Additionally, metastases were identified in the left palatine tonsil, left level 2 cervical lymph node, right axilla, left common iliac region, right inguinal area, and a lucent bony lesion in the left iliac bone.

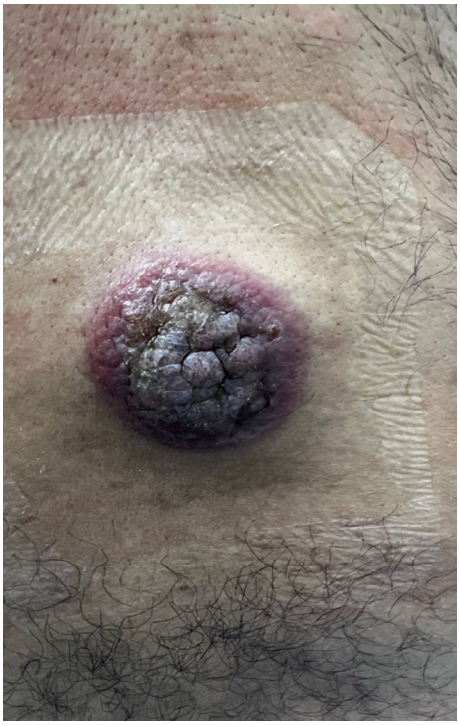

Figure 2. Violaceous nodule located at the back.

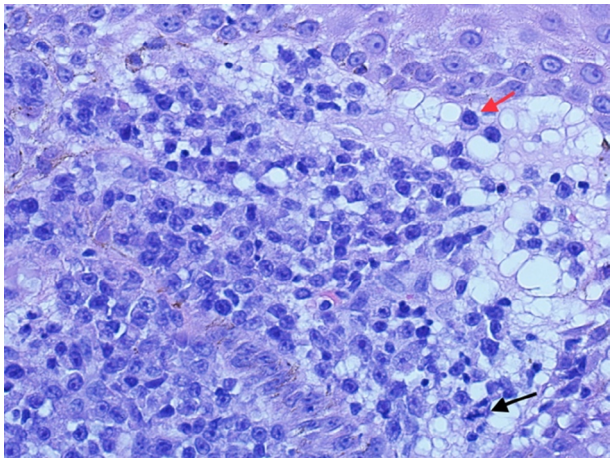

Figure 3. Skin biopsy showing plasma cell infiltration (H&E x 40).

Discussion

MM is a plasma cell malignancy arising from the uncontrolled proliferation of monoclonal plasma cells that produce immunoglobulins (1). MM is relatively

rare, comprising 10% of hematologic malignancies and 1% of all cancers worldwide. In Saudi Arabia, MM represents approximately 5% of hematological cancers and 1 % of all cancers (4,5). Cutaneous involvement in MM is uncommon, generally occurring in advanced cases in approximately 1-4% of cases. A retrospective study by Woo et al. reported that only 1.14% (14 of 1,228) of MM patients exhibited cutaneous involvement (6). Cutaneous lesions in MM can arise either by direct extension from underlying bone lesions or through hematologic or lymphatic dissemination (6). Clinically, cutaneous manifestations of metastatic MM are variable (6), presenting as violaceous, reddish, or skin-colored papules, nodules, plaques, or subcutaneous masses and can appear on various body sites, including the head, chest, and back (7–17). Lesions can also develop at atypical locations (surgical scars) (17). Although the exact mechanism of cutaneous metastasis is unclear, some studies suggest that chemotherapy may select cell clones capable of disseminating to the skin (6,17,18). The differential diagnosis for cutaneous plasmacytoma is broad and includes inflammatory, infectious, and neoplastic conditions (19). Differential considerations include lupus erythematosus, cutaneous sarcoidosis, cutaneous tuberculosis, secondary syphilis, cutaneous lymphoma, pseudolymphoma, and cutaneous metastasis from other primary tumors. Definitive diagnosis requires a skin biopsy with immunohistochemistry and ancillary studies (16,19). Multiple imaging modalities used to diagnose plasmacytomas include computed tomography (CT), Magnetic resonance imaging (MRI), and positron emission tomography/computed tomography scans (PET/CT). The CT scan detects bone abnormalities that might be missed by simple radiographs. MRI provides better soft tissue characterization. Cellular masses like plasmacytomas appear isointense on T2 weighted images. Furthermore, MRI is helpful in disease staging and in confirmation of tumor mass reduction post-therapy. In the event of MRI unavailability, the role of PET/CT comes to light. Fluorine-18-fludeoxyglucose (18-F-FDG) PET/CT studies are superior in detecting plasmacytomas that do not get detected by other imaging modalities. FDG tumor uptake positively correlated to tumor size, providing significant diagnostic and prognostic value (20-21). Histologically, cutaneous metastasis of MM typically presents as a dermal proliferation of neoplastic cells, often displaying a diffuse or nodular pattern and, less frequently, an interstitial pattern (8,16). Multiple patterns can be within the same lesion (8). This infiltrate is primarily dermal, generally sparing the epidermis, creating a “Grenz zone” (19). The cellular morphology may include mature plasma cells with eccentric nuclei and “clockface” chromatin (plasmacytic) or immature cells with a high nuclear-to-cytoplasmic ratio and prominent nucleoli (plasmablastic) (16). That was the histopathology seen in our patient. Occasionally, small plasma cells with scant cytoplasm mimicking lymphocytes (lymphoplasmacytic) (16) and rarely sarcomatoid or spindle cell morphologies (8) are seen. Immunohistochemistry is crucial for identifying plasma cell neoplastic infiltrates. It utilizes markers such as CD38, CD138, MUM1, MUM 18, CD79alpha, and CD56 (8,16,19). MM cells are typically negative for B-cell markers, including CD20 (19). Immunostaining for κ and λ light chains can assess clonality (1). The prognosis in cutaneous metastasis of MM is generally poor. Jurczyszyn et al. reported the median overall survival (OS) from the diagnosis of cutaneous plasmacytoma as only 8.5 months (22). Furthermore, they found that both IgA heavy chain disease and plasmablastic cellular morphology were associated with worse OS (22). Woo. et al. reported that erythematous nodules, multiple skin lesions, and the absence of a Grenz zone were all associated with reduced OS (6). Our literature review found that IgG was the most common immunoglobulin subtype in metastatic cutaneous plasmacytoma. Notably, studies by Alberts and Lynch, and Kois et al., suggested that IgA MM increases the risk of cutaneous plasmacytomas (23,24). Woo et al. and Hobbs et al. proposed that the IgD subtype carries an elevated risk (6,25). Further studies are needed to clarify the relationship between the immunoglobulin subtype and the risk of cutaneous plasmacytoma. There is no established standard of care for cutaneous metastases in MM. Patients typically require a combination of treatments, including radiotherapy, bortezomib, and lenalidomide with dexamethasone (6). Studies comparing the efficacy of specific treatment regimens for cutaneous metastasis in MM are lacking. There are no established guidelines for early detection or prevention of cutaneous plasmacytomas. To enhance healthcare outcomes and provide better patient care, we suggest that

patients with MM should undergo regular follow-ups and full-body skin examinations. Early dermatology referral should be made to facilitate further skin examination and skin biopsy for histopathological analysis. It is also imperative that patients with cutaneous plasmacytomas should be managed by a multidisciplinary team consisting of dermatologists, oncologists, radiologists, and pathologists. Such strategies might mitigate some risks associated with delayed diagnosis and help provide a more favorable prognosis. A comprehensive literature review was conducted via the PubMed database, covering studies published from 2003 to 2024. This review identified 11 publications involving 27 patients (7–14, 16, 17), as summarized in Table 1.

We extracted patient demographics, clinical presentation, pathological and serological findings, and prognosis. Most patients were male (15/27), with a median age of 73 at presentation. The chest was the most common site of cutaneous metastasis (7/27), and nodular lesions were the predominant clinical morphology (17/27). Serologically, IgG was the most frequently encountered immunoglobulin (11/27), and Kappa light chain restriction was more common (15/27). Histologically, Plasmacytic cellular morphology (12/27) was nearly as prevalent as plasmablastic morphology (11/27). Most patients succumbed to the disease within days to four years after skin metastasis. Novel therapeutic agents for MM includes antibody-drug conjugates, monoclonal antibodies, bispecific antibodies, immunomodulatory drugs and CAR T-cell therapy (26). In CAR T-cell therapy, patients' white blood cells identify and attack myeloma cells (after in-vitro modification) (27). Recently, FDA approved two

Table 1. Reported cases of secondary extramedullary cutaneous plasmacytoma since 2003

Case number	Age, sex	Site	Metastasis from underlying bony lesions	Tropism to trauma site	Clinical description	Serum immunoglobulin (Ig) subtype / Light chain restriction (IHC)	Histopathological pattern (Cytomorphology)	Prognosis	Reference
1	59, F	Arm[†], shin[§], chest[¶]	NA	Yes	Slightly compressible mass[†] Small area of tissue thickening[§] Subcutaneous nodules[¶]	IgG/κ	Plasmacytic	DOD 1 month after cutaneous metastasis	Rosenblum et al. (7)
2	75, F	Back	No	No	Violaceous nodules	igA/κ	Plasmacytic	DOD 7 months after cutaneous metastasis	Requena et al. (8)
3	73, M	Chest	No	No	Erythematous nodules	IgA/λ	Plasmacytic	DOD few days after cutaneous metastasis	Requena et al. (8)
4	70, M	Scalp, abdomen	No	No	Nodules	igG/κ	Plasmacytic	DOD 1 month after cutaneous metastasis	Requena et al. (8)
5	59, F	Abdomen, trunk	No	No	Nodules and livid plaques	IgA/λ	Plasmacytic	DOD 6 months after cutaneous metastasis	Requena et al. (8)

Table 1 (*continued*)

Case number	Age, sex	Site	Metastasis from underlying bony lesions	Tropism to trauma site	Clinical description	Serum immunoglobulin (Ig) subtype / Light chain restriction (IHC)	Histopathological pattern (Cytomorphology)	Prognosis	Reference
6	75, F	Abdomen	No	No	Nodules	IgA/λ	Plasmacytic	LTFU	Requena et al. (8)
7	70, F	Trunk	No	No	Papular violaceous lesions	IgA/λ	Sarcomatous	DOD 2 months after cutaneous metastasis	Requena et al. (8)
8	72, M	Abdomen	No	No	Papules and nodules	IgG/κ	Plasmacytic	DOD 3 months after cutaneous metastasis	Requena et al. (8)
9	49, M	Arms	No	No	Violaceous nodules	IgA/λ	Plasmacytic	DOD 3 months after cutaneous metastasis	Requena et al. (8)
10	77, F	Arm, forearm	No	Yes	Violaceous, fleshy, nodulo-tumoral lesions	IgG/λ	Plasmablastic	DOD 8 months after cutaneous metastasis	Pereira et al. (9)
11	72, M	Trunk, axilla, face	No	No	Erythematous violaceus tumors and nodules	IgG/κ	Plasmablastic	AWD	Santos et al. (10)
12	67, F	Heel	Yes	No	Erythematous nodules	IgG/κ	Plasmablastic	NA	Gómez-Armayones et al. (11)
13	66, M	Forearm	Yes	Yes	Erythematous to purplish nodule with black pigmentations and purpura	IgA/λ	NA	DOD 8 months after cutaneous metastasis	Yoo et al. (12)
14	82, M	Face, chest	No, Yes	No	Erythematous nodules	IgG/κ	Plasmablastic	AWD	Mohamed et al. (13)
15	73, F	Forearm	NA	NA	Nodular plaque-like lesions	NA	Plasmablastic	AWD	Chung et al. (14)
16	72, M	Thigh	Yes	NA	Fungating plaque	NA/κ	NA	DOD 4 years after cutaneous metastasis	Asavisanu et al. (15)
17	86, F	Chest, back	No	NA	Purple nodules	NA/κ	Plasmacytic	LTFU	Panse et al. (16)
18	83, M	Scalp	No	NA	Plaque	NA/κ	Plasmacytic	LTFU	Panse et al. (16)

Case number	Age, sex	Site	Metastasis from underlying bony lesions	Tropism to trauma site	Clinical description	Serum immunoglobulin (Ig) subtype / Light chain restriction (IHC)	Histopathological pattern (Cytomorphology)	Prognosis	Reference
19	73, M	Chest	No	NA	Nodule	IgG/λ	Plasmacytic	DOD 6 months after cutaneous metastasis	Panse et al. (16)
20	62, F	Back, buttock	No	NA	Violaceus nodules	igA/λ	Plasmacytic	DOD 10 months after cutaneous metastasis	Panse et al. (16)
21	78, M	Forearm	No	NA	Erythematous plaque	IgG/λ	Plasmablastic	DOD 7 months after cutaneous metastasis	Panse et al. (16)
22	80, M	Leg	No	NA	Plaque	NR κ	Plasmablastic	DOD in less than 1 month after cutaneous metastasis	Panse et al. (16)
23	73, M	Vertex scalp	No	NA	Plaque	IgA/κ	Plasmablastic	DOD 1 month after cutaneous metastasis	Panse et al. (16)
24	66, F	Mons pubis	No	NA	Erythematous nodule	K free light chain	Plasmablastic	DOD 3 months after cutaneous metastasis	Panse et al. (16)
25	89, M	Preauricular	No	NA	Plaque	IgG/λ	Plasmablastic and co-existing SCC	DOD 7 months after cutaneous metastasis	Panse et al. (16)
26	61, M	Chest	No	NA	Nodule	K free light chain	Lymphoplasmacytic and Co-existing amyloid deposition	AWD	Panse et al. (16)
27	70, F	Chest	No	Yes	Erythematous-to-violaceous plaques	IgG/κ	Plasmablastic	DOD after 4 months after cutaneous metastasis	Choong et al. (17)
28	50, M	Arm[†], back[§]	No[†], Yes[§]	No[†,§]	Deep red nodule[†] Violaceous nodule[§]	IgG/κ	Plasmablastic	AWD	Present case

Symbols used to link the lesion site with its clinical description: [†]Arm; Slightly compressible mass. [§]Shin; Small area of tissue thickening. [¶]Chest; Subcutaneous nodules. *Abbreviations:* M= Male; F= Female; DOD= Died of disease; LTFU= lost to follow-up; AWD= Alive with disease; NA= Not Available; SCC= squamous cell carcinoma.

new drugs to treat MM; 1) daratumumab (a hyaluronidase) (Darzalex Faspro) for patients who are eligible for an autologous stem cell transplant (28) and isatuximab (Sarclisa) for patients who are not eligible for a stem cell transplant (29). Both drugs include monoclonal antibodies and target a protein called CD38, often found at high levels in myeloma cells. Some of the newest drugs used to treat multiple myeloma do not fit the classification of any existing drugs, Xpovio (selinexor) being one of them. Xpovio is a selective inhibitor of nuclear export (SINE) (30). In conclusion, the clinical differential for cutaneous metastasis is broad.A comprehensive workup is required to achieve an accurate diagnosis and timely management. The prognosis remains poor for patients with cutaneous plasmacytoma. The current case report and literature review highlight the rarity of such cases, especially in our region. Furthermore, research in this area is limited to retrospective studies and small case series. Prospective studies, with large and heterogeneous populations may enhance our understanding, identify risk factors, and establish management guidelines for cutaneous plasmacytoma.

Ethical Approval: This study was approved by the Institutional Review Board (IRB) of King Fahad Specialist Hospital, Dammam, Saudi Arabia on the 30th of October 2024.

Conflict of Interest: All authors declare no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Authors Contribution: KMH, HAH, MAB, & LYH: writing–original draft preparation; NAF, RR, & AG: investigation; HAH, KMH, & MAB: conceptualization; KMH & LYH: methodology; NAF, LYH, KMH, MAB, & HAH: resources; data curation, formal analysis; AG, RR, & NAF: visualization & project administration, MAB, KMH, NAF & LYH: writing–review & editing.

Declaration on the Use of AI: None.

Consent for Publication: A written consent for publication was obtained from the patient.

Funding: None.

References

1. Rajkumar SV, Kumar S. Multiple myeloma: diagnosis and treatment. Mayo Clin Proc. 2016;91(1):101-19. doi: 10.1016/j.mayocp.2015.11.007
2. Gagelmann N, Eikema DJ, Iacobelli S, et al. Impact of extramedullary disease in patients with newly diagnosed multiple myeloma undergoing autologous stem cell transplantation: a study from the Chronic Malignancies Working Party of the EBMT. Haematologica. 2018;103(5):890-7. doi: 10.3324/haematol.2017.178434
3. Usmani SZ, Heuck C, Mitchell A, et al. Extramedullary disease portends poor prognosis in multiple myeloma and is over-represented in high-risk disease even in the era of novel agents. Haematologica. 2012;97(11):1761-7. doi: 10.3324/haematol.2012.065698
4. Abdrabou AK, Sharif FA, Fakih RE, et al. Outcomes of autologous stem cell transplantation for multiple myeloma in Saudi Arabia. Ann Saudi Med. 2021;41(4):198-205. doi: 10.5144/0256-4947.2021.198
5. Rajkumar SV. Multiple myeloma: 2022 update on diagnosis, risk stratification, and management. Am J Hematol. 2022;97(8):1086-107. doi: 10.1002/ajh.26590
6. Woo YR, Kim JS, Lim JH, et al. Prevalence and clinicopathologic characteristics of multiple myeloma with cutaneous involvement: a case series from Korea. J Am Acad Dermatol. 2018;78(3):471-8.e4. doi: 10.1016/j.jaad.2017.08.054
7. Rosenblum MD, Bredeson CN, Chang CC, Rizzo JD. Subcutaneous plasmacytomas with tropism to sites of previous trauma in a multiple myeloma patient treated with an autologous bone marrow transplant. Am J Hematol. 2003;72(4):274-7. doi: 10.1002/ajh.10296
8. Requena L, Kutzner H, Palmedo G, et al. Cutaneous involvement in multiple myeloma: a clinicopathologic, immunohistochemical, and cytogenetic study of 8 cases. Arch Dermatol. 2003;139(4):475-86. doi: 10.1001/archderm.139.4.475
9. Pereira MA, Baudrier T, Costa A, Magalhães J, Azevedo F. Cutaneous metastatic plasmacytomas with tropism for a previously injured limb. Dermatol Online J. 2008;14(9). doi: 10.5070/D337n527v0
10. Santos G, Sousa L, Fernandes T, João A. Case for diagnosis. An Bras Dermatol. 2014;89(1):173-4. doi: 10.1590/abd1806-4841.20142431
11. Gómez-Armayones S, Climent F, Servitje O. Cutaneous nodules in multiple myeloma. Actas Dermosifiliogr. 2015;106(7):581-2. doi: 10.1016/j.adengl.2015.06.009
12. Yoo J, Jo M, Kim MS, Jue MS, Park HJ, Choi KH. Cutaneous plasmacytoma: metastasis of multiple myeloma at the fracture site. Ann Dermatol. 2017;29(4):483-6. doi: 10.5021/ad.2017.29.4.483
13. Mohamed M, Alhillan A, Gupta V, et al. Relapsing cutaneous multiple myeloma responding to immunochemotherapy: a rare case report. J Med Cases. 2019;10(10):305-8. doi: 10.14740/jmc.v10i10.3382
14. Chung A, Liedtke M. Cutaneous plasmablastic plasmacytoma. Blood. 2019;134(23):2116. doi: 10.1182/blood.2019002821

15. Asavisanu K, Kasparis C, Hock YL. Cutaneous plasmacytoma: a case report. J Skin Stem Cell. 2019;6(2). doi: 10.5812/jssc.100545
16. Panse G, Subtil A, McNiff JM, et al. Cutaneous involvement in plasma cell myeloma. Am J Clin Pathol. 2021;155(1):106-16. doi: 10.1093/ajcp/aqaa122
17. Choong DJ, Ng JL, Delaney TA. Cutaneous involvement by multiple myeloma presenting as erythematous indurated plaques at the site of cardiac pacemaker insertion. JAAD Case Rep. 2021;12:54-7. doi: 10.1016/j.jdcr.2021.04.013
18. Torne R, Su WP, Winkelmann RK, Smolle J, Kerl H. Clinicopathologic study of cutaneous plasmacytoma. Int J Dermatol. 1990;29(8):562-6. doi: 10.1111/j.1365-4362.1990.tb03469.x
19. Zanelli M, Palicelli A, Sanguedolce F, et al. Cutaneous involvement in diseases with plasma cell differentiation: diagnostic approach. Curr Oncol. 2022;29(5):3026-43. doi: 10.3390/curroncol29050246
20. Caers J, Paiva B, Zamagni E, et al. Diagnosis, treatment, and response assessment in solitary plasmacytoma: updated recommendations from a European Expert Panel. J Hematol Oncol. 2018;11(1):10. doi: 10.1186/s13045-017-0549-1
21. Kilciksiz S, Karakoyun-Celik O, Agaoglu FY, Haydaroglu A. A review for solitary plasmacytoma of bone and extramedullary plasmacytoma. ScientificWorldJ. 2012;2012:895765. doi: 10.1100/2012/895765
22. Jurczyszyn A, Olszewska-Szopa M, Hungria V, et al. Cutaneous involvement in multiple myeloma: a multi-institutional retrospective study of 53 patients. Leuk Lymphoma. 2016;57(9):2071-6. doi: 10.3109/10428194.2015.1128542
23. Alberts DS, Lynch P. Cutaneous plasmacytomas in myeloma: relationship to tumor cell burden. Arch Dermatol. 1978;114(12):1784-7.doi:10.1001/archderm.1978.01640240026007
24. Kois JM, Sexton FM, Lookingbill DP. Cutaneous manifestations of multiple myeloma. Arch Dermatol. 1991;127(1):69-74. doi: 10.1001/archderm.1991.01680010079012
25. Hobbs JR, Corbett AA. Younger age of presentation and extraosseous tumour in IgD myelomatosis. Br Med J. 1969;1(5641):412-4.
26. Morè S, Corvatta L, Manieri VM, Morsia E, Poloni A, Offidani M. Novel immunotherapies and combinations: the future landscape of multiple myeloma treatment. Pharmaceuticals (Basel). 2023;16(11):1628. doi: 10.3390/ph16111628
27. Miller K, Hashmi H, Rajeeve S. Beyond BCMA: the next wave of CAR T cell therapy in multiple myeloma. Front Oncol. 2024;14:1398902. doi: 10.3389/fonc.2024.1398902
28. Arnall JR, Maples KT, Harvey RD, Moore DC. Daratumumab for the treatment of multiple myeloma: a review of clinical applicability and operational considerations. Ann Pharmacother. 2022;56(8):927-40.
29. Martino EA, Bruzzese A, Iaccino E, et al. Isatuximab in multiple myeloma. Expert Opin Biol Ther. 2023;23(4):315-8. doi: 10.1080/14712598.2023.219328
30. Syed YY. Selinexor-bortezomib-dexamethasone: a review in previously treated multiple myeloma. Target Oncol. 2023;18(2):303-10. doi: 10.1007/s11523-022-00945-3

Correspondence:
Received: 23 November 2024
Accepted: 29 January 2025
Haya A. AlHemli, MD
College of Medicine, Imam Abdulrahman bin Faisal University, Dammam, Eastern Province, Saudi Arabia. 31433, Dammam, Eastern Province, Saudi Arabia
E-mail: hayaahemli@gmail.com
ORCID: 0009-0001-6342-3225