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(CASE REPORT)

Primary Cutaneous Gamma-Delta T-Cell Lymphoma: Case Report of a Rare Lymphoma and a Brief Review of the Literature

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Abstract

Primary cutaneous gamma-delta T-cell lymphoma (PCGDTCL) is a rare and highly aggressive subtype of cutaneous T-cell lymphoma characterized by rapid progression, resistance to therapy, and poor prognosis. We report the case of a 66-year-old man with a four-month history of rapidly progressive, painful ulcerative cutaneous lesions involving the lower extremities and trunk. The patient also reported systemic "B" symptoms, including fever, weight loss, and drenching night sweats.

Physical examination revealed multiple violaceous plaques and necrotic nodules without clinically evident lymphadenopathy or hepatosplenomegaly. Laboratory evaluation demonstrated pancytopenia, elevated lactate dehydrogenase levels, and hypoalbuminemia. Positron emission tomography–computed tomography (PET/CT) showed intense fluorodeoxyglucose uptake limited to the skin and subcutaneous tissues.

Histopathologic examination revealed dense atypical lymphocytic infiltration of the dermis and subcutaneous tissue with areas of necrosis and angioinvasion, sparing the epidermis. Immunophenotyping demonstrated a cytotoxic T-cell phenotype (CD3+, CD2+, CD56+, CD4−, CD8−) with a high proliferative index. Molecular analysis confirmed clonal rearrangement of the T-cell receptor gamma gene, establishing the diagnosis of PCGDTCL.

Despite treatment with CHOEP (Cyclophosphamide, Doxorubicin, Hydroxydaunorubicin, Vincristine Oncovin, Etoposide, and Prednisone) chemotherapy followed by second-line therapy, the disease progressed rapidly. It was complicated by hemophagocytic lymphohistiocytosis, leading to death 11 months after diagnosis. This report highlights the aggressive clinical course, clinicopathologic features, and underlying pathophysiology of PCGDTCL, and compares this case with findings from the current literature.

Keywords: Primary cutaneous gamma-delta T-cell lymphoma; Chemotherapy; CHOP; Molecular studies, Chronic immunosuppression; Prolonged antigenic stimulation; Immunohistochemistry; Hemophagocytic lymphohistiocytosis (HLH).

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1. Introduction

Primary cutaneous gamma-delta T-cell lymphoma (PCGDTCL) is a rare and highly aggressive malignancy derived from gamma-delta T lymphocytes, accounting for less than 1% of primary cutaneous lymphomas. [1] Gamma-delta T cells originate from thymic precursors and function as components of the innate immune system, recognizing antigens in a major histocompatibility complex–independent manner. [2] Chronic immunosuppression and sustained antigenic stimulation are increasingly recognized as potential drivers of lymphomagenesis in PCGDTCL. [3]

The earliest cases were described in 1991. One report by Berti et al. documented disseminated pagetoid reticulosis with epidermotropic gamma-delta T-cell infiltration, while other reports described T-cell receptor (TCR) delta–positive subcutaneous plaques without epidermotropism. [1,2] [4,12] Although PCGDTCL can occur in younger individuals, it most commonly affects adults between 50 and 60 years of age, with no significant sex predilection. [2,5,6]

Clinically, PCGDTCL typically presents as plaques, nodules, or masses involving extremities, often with ulceration or necrosis; however, lesions may occur at any cutaneous site. In some cases, the disease manifests as erythematous lesions resembling nonspecific panniculitis. [5,6] Although the disease frequently involves mucosal and other extranodal sites, lymph node, splenic, and bone marrow involvement are relatively uncommon. [1,6] Bone marrow infiltration has been reported in 0%–20% of cases and may be associated with a more aggressive clinical course, although its prognostic significance remains uncertain. [6]

Published case series consistently demonstrate a poor prognosis, reflecting the aggressive clinical behavior and relative resistance to conventional chemotherapy. [2,6] Mangala et al reported 5-year survival rates are generally below 20%, with a median overall survival ranging from 15 to 31 months. [2,6] Data from the National Cancer Database (NCDB) indicate 1-year survival of approximately 61% and 3-year survival of 47%. [6,7] Owing to the rarity of the disease, prospective clinical trials are lacking, and current management strategies are largely based on case reports and small series. Anthracycline-based regimens, such as CHOP, remain the most used first-line therapy. Although approximately half of patients achieve an initial response, relapses are frequent, and most patients ultimately experience disease progression and disease-related mortality. [2]

2. Case Presentation

2.1. Clinical Presentation

A 66-year-old male presented with a 4-month history of progressively enlarging, painful ulcerated skin lesions on his lower extremities and trunk. Initially, he noticed small reddish nodules on his right leg, which he attributed to insect bites and treated with over-the-counter topical antibiotics. The lesions gradually increased in size and number, becoming necrotic and ulcerated despite multiple courses of oral antibiotics prescribed by his primary care physician. He sought medical attention at the dermatology clinic when the lesions became severely painful, began draining purulent material, and he developed associated fever, drenching night sweats, and unintentional 15-pound weight loss over 6 weeks.

2.2. Physical Examination and Initial Assessment

Physical examination revealed a cachectic-appearing man with multiple violaceous plaques and nodular masses scattered across both lower extremities, with the largest measuring 8 cm on the right calf. Several lesions demonstrated central ulceration, necrotic bases, and purulent drainage. Smaller subcutaneous nodules were palpable on the trunk and left thigh. No significant lymphadenopathy was detected on careful examination of cervical, axillary, or inguinal regions. Hepatosplenomegaly was absent on abdominal palpation.

Initial laboratory studies showed pancytopenia with hemoglobin 9.2 g/dL, white blood cell count 3,200/μL, and platelet count 95,000/μL. Lactate dehydrogenase was markedly elevated at 892 U/L. Comprehensive metabolic panel demonstrated mild hypoalbuminemia (2.8 g/dL) but was otherwise unremarkable with normal liver and kidney function.

The clinical impression favored an aggressive cutaneous lymphoproliferative disorder, possibly an advanced cutaneous T-cell lymphoma variant, though atypical infections and other inflammatory dermatoses required exclusion.

2.3. Radiographic and Specialized Studies

Contrast-enhanced CT imaging of the chest, abdomen, and pelvis revealed no mediastinal or hilar lymphadenopathy, no hepatosplenomegaly, and no evidence of visceral organ involvement. Multiple enhancing subcutaneous soft-tissue masses correlated with clinically evident skin lesions on the lower extremities. PET-CT scanning demonstrated intense FDG uptake (SUV max 18.4) in the cutaneous and subcutaneous lesions with no extracutaneous sites of metabolic activity, confirming disease limited to the skin and subcutis.

Complete blood count with peripheral smear showed no circulating atypical lymphocytes. Flow cytometry of peripheral blood revealed no abnormal T-cell or NK-cell populations. Bone marrow aspiration and biopsy were performed, showing normocellular marrow with trilineage hematopoiesis and no evidence of lymphomatous infiltration; the cytopenias were attributed to systemic inflammatory effects. Serologic testing for HIV, hepatitis B, and hepatitis C was negative. Bacterial, fungal, and mycobacterial cultures from wound drainage were negative after extended incubation.

At this point, the differential diagnosis included PCGDTCL (given the aggressive presentation with ulcerated tumors and systemic symptoms), subcutaneous panniculitis-like T-cell lymphoma, extranodal NK/T-cell lymphoma (nasal type with cutaneous involvement), transformed mycosis fungoides, or cutaneous aggressive epidermotropic CD8+ cytotoxic T-cell lymphoma. A tissue core biopsy was obtained from the largest calf mass for initial evaluation, which showed an atypical T-cell population infiltrating the dermis and the subcutaneous tissue.

2.4. First Multidisciplinary Tumor Board Discussion

The case was discussed on the hematology/oncology tumor board, and the discussion centered on the unexpectedly aggressive clinical course, absence of epidermotropism on preliminary pathology review, and the unusual distribution of subcutaneous involvement with rapid ulceration, atypical features for conventional mycosis fungoides or Sézary syndrome. The dermatopathologist noted the importance of comprehensive immunophenotyping to characterize the T-cell subset and exclude NK-cell lineage. Given the differential diagnosis of rare aggressive cutaneous lymphomas, consensus recommended multiple deep incisional biopsies from different lesional sites (both ulcerated tumor and intact subcutaneous nodule) for extensive immunohistochemical (IHC) analysis, T-cell receptor gene rearrangement studies, and Epstein-Barr virus evaluation before initiating treatment.

2.5. Tissue Diagnosis

Histopathologic examination of skin biopsies revealed a dense, monotonous infiltrate of medium-to-large, atypical lymphocytes involving the dermis and subcutaneous tissue with extensive necrosis and vascular invasion. The infiltrate showed preferential involvement of the subcutis and deep dermis, with relative sparing of the epidermis and no significant epidermotropism. (**Figure 1 A**) The neoplastic cells demonstrated irregular, hyperchromatic nuclei with coarse chromatin and moderate amounts of pale cytoplasm. Numerous mitotic figures and apoptotic bodies were present, indicating high proliferative activity.

IHC studies revealed the following profile: positive markers include CD3 (cytoplasmic and surface), CD2, TIA-1 (strongly positive, indicating a cytotoxic phenotype), and CD56. Negative markers: CD4, CD8, CD5, CD7 (dim/partial), betaF1 (negative, indicating gamma-delta T-cell receptor), CD30, ALK-1. EBER-ISH: Negative (excluding EBV-associated extranodal NK/T-cell lymphoma, nasal type). Ki-67 proliferation index was high with 85% nuclear staining. T-cell receptor (TCR) gene rearrangement studies by PCR demonstrated clonal rearrangement of the TCR gamma chain genes, confirming monoclonality and gamma-delta T-cell origin. TCR beta chain genes showed germline configuration, corroborating the betaF1-negative immunophenotype. Flow cytometry on fresh tissue confirmed an aberrant T-cell population with the unusual gamma-delta TCR phenotype (TCR gamma delta+, TCR alpha beta−).

The constellation of clinical, histologic, immunophenotypic, and molecular findings established the final diagnosis of PCGDTCL, representing one of the most aggressive primary cutaneous lymphomas with characteristic subcutaneous/dermal involvement, absent epidermotropism, cytotoxic phenotype (TIA-1+), gamma-delta TCR expression (betaF1-negative, TCR gamma clonality), and EBV-negative status.

2.6. Second Multidisciplinary Tumor Board Discussion

The tumor board reconvened with the confirmed diagnosis of PCGDTCL, acknowledging this as one of the most aggressive and treatment-refractory cutaneous lymphomas with historically poor outcomes. The hematology-oncology team emphasized that strongly positive cytotoxic markers (TIA-1) and a betaF1-negative gamma delta phenotype predicted particularly aggressive biology and a high risk of systemic dissemination despite current cutaneous-only disease.

Given the lack of standardized treatment protocols and uniformly poor responses to conventional CHOP-based chemotherapy reported in the literature, the board recommended enrollment in a clinical trial if available. In the absence of appropriate trials, consensus supported frontline systemic combination chemotherapy with CHOEP, given the patient's relatively preserved performance status, followed by consolidation with autologous stem cell transplantation if a chemosensitive response is achieved. Radiation therapy was considered for local disease control of symptomatic ulcerated lesions.

2.7. Treatment Response and Clinical Outcome

The patient received six cycles of CHOEP chemotherapy with an initial partial response; the cutaneous lesions decreased in size, and several ulcers began re-epithelializing. However, after the fourth cycle, new subcutaneous nodules emerged on the upper extremities despite continued treatment. Restaging PET-CT after cycle 6 showed persistent metabolically active disease. Autologous stem cell transplantation was deferred due to chemoresistant disease.

Second-line therapy with gemcitabine and romidepsin produced a minimal response. The patient developed progressive disease with widespread cutaneous dissemination, hemophagocytic lymphohistiocytosis with fever, pancytopenia, and hepatosplenomegaly at 9 months from diagnosis. Bone marrow biopsy showed infiltration by the same cell population noted in the cutaneous masses (**Figure 1 B, C**). Despite supportive care and consideration of experimental therapies, his clinical condition deteriorated rapidly.

He transitioned to hospice care and died 11 months after initial diagnosis from septic complications and multi-organ failure. This clinical course, unfortunately, reflected the characteristically aggressive nature and dismal prognosis of PCGDTCL despite multimodal treatment approaches.

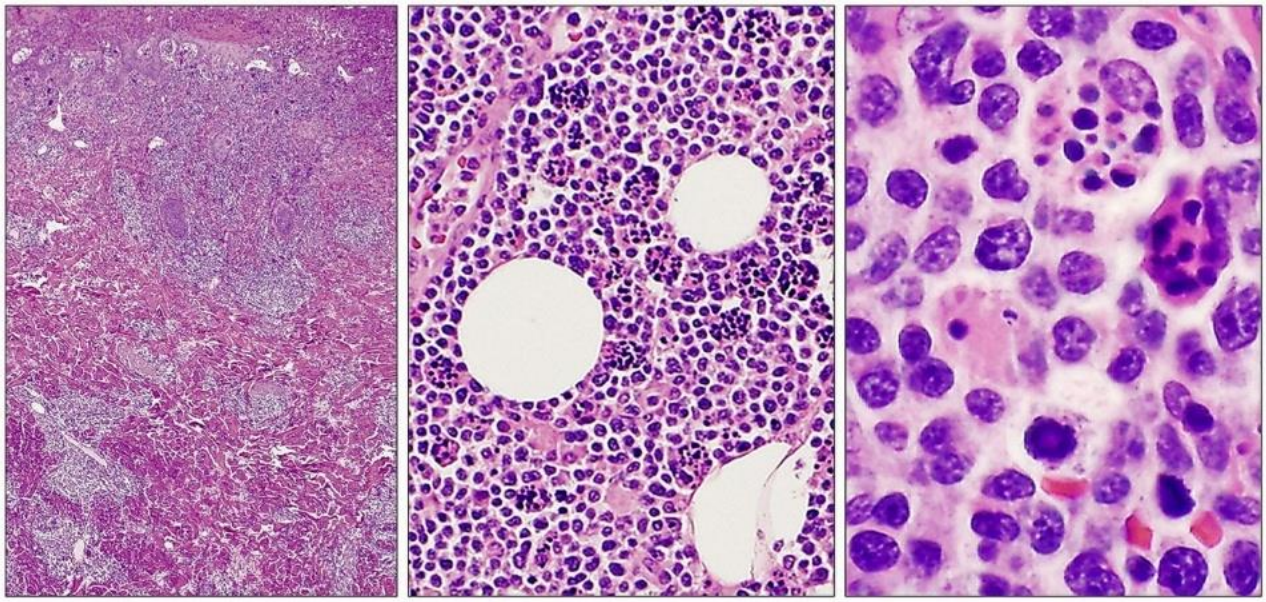

Figure 1 Microscopic examination of primary cutaneous gamma-delta T-cell lymphoma

- **1A:** Low-power view of initial cutaneous tissue biopsy showing infiltration of dermal and subcutaneous tissue by malignant lymphoid population and no significant epidermotropism (H&E X20).
- **1B:** Intermediate power view of bone marrow biopsy, later with disease progression showing diffuse infiltration of bone marrow spaces with malignant T-cells, prominent hemophagocytic lymphohistiocytosis, and vascular invasion (H&E X40).
- **1C:** High power view of bone marrow biopsy, later with disease progression showing hemophagocytic lymphohistiocytosis and the neoplastic T cells demonstrating irregular, hyperchromatic nuclei with coarse chromatin and moderate amounts of pale cytoplasm, apoptotic bodies, and abnormal mitotic figures (H&E X60).

3. Discussion

3.1. Background (History, Epidemiology, Risk factors, and WHO Classification)

Primary cutaneous gamma-delta T-cell lymphoma (PCGDTCL) was first described by Berti et al. in 1991 during the clinical evaluation of a patient with disseminated pagetoid reticulosis characterized by epidermotropic infiltration of gamma-delta T cells. [4] Although the disease affects both sexes, several series suggest a slight male predominance. PCGDTCL is an extremely rare malignancy, with an estimated incidence of approximately 0.4 cases per 10 million individuals per year. [4,8]

Clinically, PCGDTCL most commonly involves the extremities and trunk and presents as ulcerated or necrotic plaques, nodules, or masses. Histologically, the disease is characterized by dense atypical lymphoid infiltrates involving the epidermis, dermis, and, in many cases, the subcutaneous tissue. Subcutaneous involvement has been associated with a more aggressive clinical course and worse prognosis. The disease frequently disseminates to mucosal and other extranodal sites, whereas lymph node, splenic, and bone marrow involvement are absent in most patients at presentation. [5,9]

According to the World Health Organization (WHO) classification of lymphoid neoplasms, PCGDTCL represents a clonal proliferation of mature, activated gamma-delta T cells with a cytotoxic phenotype. The neoplastic cells typically express CD3 and CD2, show variable expressions of CD7, and are negative for betaF1 and CD5, while frequently expressing CD56 and cytotoxic proteins such as granzyme B, TIA-1, and perforin. Molecular studies demonstrate clonal rearrangement of the T-cell receptor gamma gene, supporting the diagnosis. [9]

Because of its rarity, well-defined risk factors for PCGDTCL remain limited. However, chronic immunosuppression, such as that seen in organ transplant recipients, and prolonged antigenic stimulation have been proposed as potential contributors to lymphomagenesis. [1]

3.2. Pathogenesis, Pathophysiology

In contrast to conventional alpha-beta T cells, gamma-delta T cells recognize cellular stress signals in a major histocompatibility complex in an independent manner and exert cytotoxic effects through perforin-and granzyme-mediated apoptotic pathways. In PCGDTCL, malignant transformation of this cytotoxic lineage results in dense, uncontrolled infiltration of the dermis and subcutaneous tissue by the cytotoxic T-cells. [9,10]

Molecular studies of cytotoxic T-cell lymphomas, including gamma-delta subtypes, have identified recurrent genetic alterations involving the JAK/STAT signaling pathway, particularly in STAT3, STAT5B, and JAK3, which are thought to play a central role in lymphomagenesis. [11, 12] Constitutive activation of this pathway promotes neoplastic cell survival and proliferation by enhancing anti-apoptotic signaling, including upregulation of BCL2 family proteins, and by increasing the production of proinflammatory cytokines. Additional mutations affecting epigenetic regulators, such as TET2 and KMT2D, further disrupt transcriptional control and contribute to clonal expansion and genomic stability of the malignant population. [12]

The characteristic clinicopathologic features of PCGDTCL, including angioinvasion, angiodestruction, ischemia, and extensive tissue necrosis, reflect the intrinsic cytotoxic activity of the malignant gamma-delta T-cell clone. Release of cytotoxic granules leads to endothelial injury, vascular occlusion, and subsequent tissue infarction, accounting for the rapidly progressive ulcerative plaques and tumors observed clinically. [9]

Moreover, sustained production of inflammatory cytokines, particularly interferon-gamma (IFN-gamma), can lead to macrophage activation and may precipitate secondary hemophagocytic lymphohistiocytosis (HLH), a severe hyperinflammatory syndrome frequently associated with aggressive cytotoxic lymphomas. [13] Overall, PCGDTCL is driven by genetically and epigenetically altered cytotoxic gamma-delta T cells, with persistent JAK/STAT pathway activation promoting malignant proliferation, vascular injury, tissue destruction, and immune dysregulation, mechanisms that collectively underline the disease's aggressive clinical behavior.

3.3. Comparative Analysis of Our Case with Existing Literature. (Clinical-Radiology-Pathology, Lab, Diagnosis-Management-Outcome)

PCGDTCL is a rare and aggressive cutaneous lymphoma with a typically dismal prognosis. [6,14] The presented case, featuring a 66-year-old male with rapidly progressive, painful, ulcerated skin lesions, pancytopenia, and elevated LDH, aligns with the aggressive clinical course frequently reported in the literature. [14] Many studies highlight the common presentation of PCGDTCL with plaques, nodules, or tumors, often with ulceration, predominantly on the extremities and trunk, consistent with our patient's presentation. [19]

Diagnostic challenges are a recurring theme in PCGDTCL, often necessitating a multidisciplinary approach and comprehensive IHC typing to differentiate it from other cytotoxic T-cell lymphomas, such as subcutaneous panniculitis-like T-cell lymphoma (SPTCL). [3,20] Our case underscores this, with initial broad differential diagnoses and the need for extensive IHC and TCR gene rearrangement studies to confirm the gamma-delta phenotype (TCR gamma delta+, TCR alpha beta−, betaF1-negative) and cytotoxic markers (TIA-1+, CD56+). [14,21] This immunophenotypic profile is consistently associated with aggressive biology and poor outcomes in the literature. [6,14]

Regarding management, the lack of standardized protocols for PCGDTCL is a significant challenge. Conventional CHOP-based chemotherapy often yields unsatisfactory results, and the prognosis remains poor. [14,15] Our patient's initial partial response to CHOEP chemotherapy, followed by rapid progression and chemoresistance, mirrors the refractory nature of the disease described in various case series. [14,16] The development of HLH and subsequent multi-organ failure, leading to death within 11 months, is a tragic but characteristic outcome for this aggressive lymphoma, with median overall survival often reported in months rather than years. [17] This case reinforces the urgent need for novel therapeutic strategies and early identification of this aggressive subtype.

4. What have we learned from this case?

This case of Primary Cutaneous Gamma-Delta T-Cell Lymphoma offers several critical clinical insights. Diagnostically, the rapid progression of painful, ulcerated skin lesions, especially when accompanied by systemic symptoms like fever, night sweats, weight loss, and laboratory abnormalities such as pancytopenia and elevated LDH, should serve as significant red flags for clinicians, prompting early consideration of aggressive lymphoproliferative disorders. [14, 17] The case highlights the necessity of early and comprehensive tissue biopsies, including deep incisional biopsies, for extensive IHC and molecular studies (TCR gene rearrangement) to establish a definitive diagnosis, particularly when facing atypical presentations or aggressive clinical courses. [20, 21]

For management, this case reinforces the aggressive and often treatment-refractory nature of PCGDTCL. Despite initial response to systemic chemotherapy (CHOEP), the rapid emergence of chemoresistance and subsequent progression underscores the limitations of conventional approaches. [20,16] Clinicians should be aware that even with aggressive treatment, outcomes can be dismal, and early consideration of clinical trial enrollment or novel therapeutic combinations is crucial. [14,15] The development of HLH further complicates management and indicates a severe, often terminal, phase of the disease. [18] This case challenges the notion that aggressive chemotherapy alone can alter the natural history of this highly aggressive lymphoma and reinforces the need for more effective, targeted therapies and vigilant surveillance for disease progression and complications like HLH.

Abbreviations

- Primary cutaneous gamma-delta T-cell lymphoma (PCGDTCL);
- Immunohistochemistry (IHC).
- Hemophagocytic lymphohistiocytosis (HLH)

5. Conclusion

This case illustrates the aggressive clinical course of primary cutaneous gamma-delta T-cell lymphoma (PCGDTCL), which initially presented as subcutaneous nodules and rapidly progressed to necrotic, ulcerative lesions. Despite multimodal therapy, the disease demonstrated rapid progression with systemic complications, ultimately resulting in a fatal outcome.

This report highlights the diagnostic challenges associated with PCGDTCL, as early manifestations may mimic inflammatory, autoimmune, or infectious conditions, leading to delays in diagnosis. Prompt skin biopsy, comprehensive immunophenotypic analysis, and molecular studies are essential to establish an accurate diagnosis and enable timely

initiation of therapy. Clinicians should maintain a high index of suspicion for PCGDTCL when evaluating rapidly progressive or atypical cutaneous lesions to facilitate earlier recognition and management.

Given the limited effectiveness of current treatment strategies and the overall poor prognosis, there is a critical need for further research to define the molecular mechanisms of the disease in greater detail and to develop more effective targeted therapies and treatment approaches.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

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Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified before analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

References

[1] Alberti-Violetti S, Maronese CA, Venegoni L, Merlo V, Berti E. Primary cutaneous gamma-delta T cell lymphomas: a case series and overview of the literature. Dermatopathology. 2021 Nov 17;8(4):515-24.

[2] Mangala YO, Onukogu ID, Breen CM, Colvin GA. Primary cutaneous gamma-delta T-cell lymphoma: a case report and review of literature. Cureus. 2023 Feb 25;15(2):e35442.

[3] Goyal A, Goyal K, Bohjanen K, Pearson D. Epidemiology of primary cutaneous γδ T-cell lymphoma and subcutaneous panniculitis-like T-cell lymphoma in the USA from 2006 to 2015: a Surveillance, Epidemiology, and End Results-18 analysis. British Journal of Dermatology. 2019 Oct 1;181(4):848-50.

[4] Berti E, Cerri A, Cavicchini S, Delia D, Soligo D, Alessi E, Caputo R. Primary cutaneous gamma/delta T-cell lymphoma presenting as disseminated pagetoid reticulosis. Journal of investigative dermatology. 1991;96(5):718-23.

[5] Khallaayoune M, Grange F, Condamina M, Szablewski V, Guillot B, Dereure O. Primary cutaneous gamma-delta T-cell lymphoma: not an aggressive disease in all cases. Acta Dermato-Venereologica. 2020 Jan 23;100(1):5605.

[6] Arabi S, Vardell V, Hanley T, Florell S, Halwani A, Lim M. Primary Cutaneous Gamma–Delta T-Cell Lymphoma Presenting with Hemophagocytic Lymphohistiocytosis in a Young Polynesian Male. Case Reports in Hematology. 2025;2025(1):8582804..

[7] von Dücker L, Fleischer M, Stutz N, Thieme M, Witte M, Zillikens D, Sadik CD, Terheyden P. Primary cutaneous gamma-delta T-cell lymphoma with long-term indolent clinical course initially mimicking lupus erythematosus profundus. Frontiers in oncology. 2020 Feb 19;10:133.

[8] Gaillard F, Sharma R, Campos A, et al. (2022) WHO classification of haematolymphoid tumors. Radiopaedia.org. Accessed on 13 February 2026. https://doi.org/10.53347/rID-9250.

[9] Willemze R, Jaffe ES, Burg G, Cerroni L, Berti E, Swerdlow SH, Ralfkiaer E, Chimenti S, Diaz-Perez JL, Duncan LM, Grange F. WHO-EORTC classification for cutaneous lymphomas. Blood. 2005 May 15;105(10):3768-85.

[10] Alaggio R, Amador C, Anagnostopoulos I, Attygalle AD, Araujo IB, Berti E, Bhagat G, Borges AM, Boyer D, Calaminici M, Chadburn A. The 5th edition of the World Health Organization (WHO) classification of haematolymphoid tumours: lymphoid neoplasms. Leukemia. 2022 Jul;36(7):1720-48.

[11] Kücük C, Jiang B, Hu X, Zhang W, Chan JK, Xiao W, Lack N, Alkan C, Williams JC, Avery KN, Kavak P. Activating mutations of STAT5B and STAT3 in lymphomas derived from γδ-T or NK cells. Nature communications. 2015 Jan 14;6(1):6025.

[12] Daniels J, Doukas PG, Escala ME, Ringbloom KG, Shih DJ, Yang J, Tegtmeyer K, Park J, Thomas JJ, Selli ME, Altunbulakli C. Cellular origins and genetic landscape of cutaneous gamma delta T cell lymphomas. Nature communications. 2020 Apr 14;11(1):1806.

[13] La Rosée P, Horne A, Hines M, von Bahr Greenwood T, Machowicz R, Berliner N, Birndt S, Gil-Herrera J, Girschikofsky M, Jordan MB, Kumar A. Recommendations for the management of hemophagocytic lymphohistiocytosis in adults. Blood, The Journal of the American Society of Hematology. 2019 Jun 6;133(23):2465-77.

[14] Muhsen IN, El Fakih R, Hamadani M, Lazarus HM, Kharfan-Dabaja MA, Aljurf M. Clinical, diagnostic and prognostic characteristics of primary cutaneous gamma delta T-cell lymphomas. Clinical hematology international. 2022 Jun;4(1):1-0

[15] Moskowitz AJ, Lunning MA, Horwitz SM. How I treat the peripheral T-cell lymphomas. Blood, The Journal of the American Society of Hematology. 2014 Apr 24;123(17):2636-44.

[16] Ong SY, Zain JM. Aggressive T-cell lymphomas: 2024: updates on diagnosis, risk stratification, and management. American journal of hematology. 2024 Mar;99(3):439-56.

[17] Oon ML, Lim JQ, Bosch-Schips J, Climent F, Au-Yeung RK, Hutchison B, Sohani AR, Eren OC, Kumar J, Dogan A, Ong CK. Characterizing nodal gamma-delta T-cell lymphoma: clinicopathological and molecular insights. Modern Pathology. 2025 Mar 1;38(3):100685.

[18] Voruz S, de Leval L, Cairoli A. Successful salvage therapy for refractory primary cutaneous gamma-delta T-cell lymphoma with a combination of brentuximab vedotin and gemcitabine. Experimental hematology & oncology. 2021 May 13;10(1):32..

[19] Hu G, Song Z, Lv C, Sun Y, Zhang Y, Liu X, Han X, Li L, Qiu L, Qian Z, Zhou S. Clinical Features and Outcomes of Primary Cutaneous Peripheral T-Cell Lymphoma, Not Otherwise Specified, Treated with CHOP-Based Regimens. Cancers. 2025 May 15;17(10):1673.. PMC.

[20] Pit D, Hoinoiu T, Hoinoiu B, Cerbu S, Iordache M, Vaduva A, Szilagyi D, Bardan CR, Taskov P, Crainiceanu ZP, Samfireag M. Subcutaneous Panniculitis-like T-Cell Lymphoma: Diagnostic Challenge and Successful Multimodal Management with Integra® Dermal Matrix—Case Report and Review of the Literature. Diseases. 2025 Jun 30;13(7):201.

[21] Hirata C, Nakai K, Kurasawa Y, Maekawa N, Kuniyuki S, Yamagami K, Ohsawa M, Tsuruta D. Primary cutaneous gamma-delta T-cell lymphoma initially diagnosed as subcutaneous panniculitis-like T-cell lymphoma with dermatomyositis. Dermatopathology. 2022 Apr 29;9(2):143-7.