

Case Report

Mantle Cell Lymphoma Presenting as a Subcutaneous Mass of the Right Leg

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Abstract

Mantle cell lymphoma (MCL) is a relatively rare B-cell non-Hodgkin lymphoma, typically presenting with extensive lymphadenopathy, bone marrow involvement, and splenomegaly. Extranodal sites can also be involved. We discuss a 73-year-old man whose MCL presented with a 6-month history of a subdermal mass of the right upper thigh and no systemic symptoms.

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Introduction

Mantle cell lymphoma (MCL) is a relatively rare malignancy of the lymphatic system [1]. MCL arises from malignant transformation of mature CD5-positive B-lymphocytes in the outer edge (mantle zone) of lymph node follicles. Characteristically, MCLs carry a t(11; 14) (q13; 23) translocation resulting in overexpression of the cyclin D1 [1]. MCL accounts for approximately 6% of all non-Hodgkin lymphomas in the USA and about 7–9% of lymphomas in Europe [1, 2]. It is considered as a distinct aggressive lymphoma subtype, occurs predominantly in males (2:1), and usually affects individuals in the fifth and sixth decades of life with the median age of presentation being just under 70 years. MCL usually presents with advanced-stage (stage III or IV) disease. Consequently, patients often present with constitutional symptoms: weight loss, night sweats, persistent fevers, nausea, vomiting, and fatigue. For

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those with massive node involvement in the gastrointestinal tract, symptoms at presentation often include abdominal pain, bloating, and a feeling of fullness.

Extranodal involvement at presentation is very common and most often involves bone marrow, peripheral blood, the gastrointestinal tract, Waldeyer's ring, and the liver. Extranodal disease as the primary presentation occurs in approximately 25% of cases. Presentation outside of these main extranodal sites is rare and includes salivary glands, skin (see Table 1), thyroid, orbit, breast, epidural space, pleura, and spleen. Presentation which involves the leg is extremely uncommon and, to the best of our knowledge, has always been associated with skin manifestation [3]. Here, we report the first case of MCL that presented with a subdermal mass of the leg.

Case Report

A 73-year-old Caucasian male with a family history of cancer (his father and brother had prostate cancer and his sister had breast cancer) presented to his family physician with a 6-month history of a mass located medially on his right upper thigh. There was no history of trauma, the mass was nontender, and it had not grown in size. There were no systemic symptoms including fever chills, night sweating, or unintentional weight loss. He denied any nausea, vomiting, or generalized malaise. He had no prior history of smoking or tobacco product use, denied the use of alcohol, and had no history of being immunocompromised.

Physical examination revealed a subcutaneous firm, nonmobile, not warm, irregular mass of approximately 4 cm on the patient's right upper outer thigh with no associated inguinal lymphadenopathy. The patient's abdomen was soft, nontender, with no masses, hepatomegaly, or splenomegaly. The patient's afflicted leg showed no signs of muscle weakness, and the range of motion was normal.

A complete blood count (CBC) demonstrated a WBC of 7.9 K/ μ L (normal 4.8–10.8) with a normal differential, Hb 5.84 M/ μ L (normal 4.70–6.10), Ht 50.7% (normal 42.0–52.0), and platelet count 243 K/ μ L (normal 164–446). His electrolytes, liver and renal functions were normal. An ultrasound of the right leg was performed and showed a vascularized heterogeneous mass measuring 5.9 \times 3.8 \times 1.3 cm within the subcutaneous soft tissues, consistent with either a primary muscular or metastatic tumor.

A subsequent magnetic resonance imaging (MRI) with contrast of the right lower extremity (Fig. 1) demonstrated a 5.4 \times 3.4 \times 1.5 cm mass which was noted to be T2 hyperintense, T1 slightly hyperintense to muscle, and which avidly enhanced in a uniform fashion following contrast administration. The mass was found to be superficial to the rectus femoris muscle with no signs of muscular invasion. The remainder of the thigh was normal on imaging. Several small bilateral inguinal lymph nodes were noted, all within normal limits. At this point, the imaging findings were still primarily concerning for a soft tissue sarcoma or metastasis.

A contrast-enhanced computed tomography (CT) of the chest, abdomen, and pelvis revealed bilateral axillary lymphadenopathy. The liver, pancreas, adrenal glands, and kidneys were unremarkable. Multiple enlarged gastro-hepatic lymph nodes, the largest measuring 5 \times 3 cm in size, were visualized. Multiple mildly prominent retroperitoneal lymph nodes were also present, the largest measuring up to 10 mm in the short axis dimension. Based on these findings, the differential diagnosis included metastatic adenopathy and lymphoma.

Microscopic examination of a right thigh biopsy demonstrated sheets of intermediate-sized lymphocytes with somewhat dispersed chromatin, occasional inconspicuous nucleoli, irregular nuclear contours, and scant amphophilic cytoplasm (Fig. 2). Brisk mitotic activity was noted. The neoplastic cells were strongly and diffusely positive for CD20, CD5, and cyclin

Table 1. Clinical features of patients with MCL skin presentation

First author [ref.]	Patient	Age, years	Gender	Site/lesion	Skin as initial presentation	Other sites at diagnosis	Stage at initial diagnosis	Initial treatment	Outcome
A Geerts [7]	1	73	F	Scalp	+	+	4B	CLB + PRED	Relapse at 14 months and died 4 months later
	2	65	F	Back, chest, arm	+	+	4B	COP + CHOP	Relapsed at 11 months, died 11 months later
Bertero [6]	3	51	M	Breast	+	+	Late stage (3 or 4)	Multiple (COP-CHOP-XRT)	Multiple relapses, favorable response to treatments, alive and in CR after 17 years
	4	78	M	Breast and back	+	-	1	Observation	Died after 3 years, unrelated cause
	5	43	M	Back, face, sternum, arms	+	+	Stage 2 or 3	COP + CHOP	Obtained CR, unknown status
	6	22	M	Chest	+	-	1	Patient refused therapy	Unknown
Moody [9]	7	47	M	Earlobe	+	+	4B	CHOP	Alive with disease at 3 years
Marti [8]	8	61	F	Abdominal	+	+	4	CHOP	Initial CR, relapsed and died 15 months after diagnosis
Sen [11]	9	85	M	Leg/rash	+	+	4B	CHOP	Died 20 months after diagnosis
	10	76	M	Thigh/nodule	+	-	1	Hyper-CVAD	Relapsed, alive with disease at 30 months
	11	56	M	Chest/nodules	+	+	4A	Hyper-CVAD	Alive at 21 months
	12	57	M	Legs/rash	+	+	4B	Hyper-CVXD	Died 9 months after diagnosis
Dubus [10]	13	56	M	Breast, back	+	+	4A	CHOP, DEXA-BEAM + allograft	CR at 1 year, died of meningeal hemorrhage
	14	89	M	Breast, back, abdomen	+	+	4B	None	Died of cerebral and cardiovascular failure on day 5
Motegi [12]	15	62	M	Back, upper extremities	+	+	4	Hyper-CVAD + MTX/Ara-C + RTX	CR of skin lesions at 4 months
Estrozi [13]	16	72	M	Temporal (head)	+	-	1	Local XRT	Alive in CR at 6 months
Canpolat [14]	17	49	F	Face, shoulders, back, chest	+	+	4B	Intense chemotherapy	Died at 4 months
Lynch [17]	18	83	M	Thighs	+	Unclear	Likely 1 or 2	RTX, VMP	Died at 2 months, disseminated herpes zoster
Zattra [16]	19	77	M	Multiple body areas	+	-	1	VP16, PRED	Alive at 28 months with almost CR
Cao [18]	20	53	M	Legs	+	+	4A	Chemotherapy	Died at 5 weeks
Cesinaro [19]	21	75	F	Leg	+	-	1	Local XRT	Relapsed at 14 months, died at 40 months
Wehkamp [3]	22	68	M	Both legs	+	+	4A	RTX-CHOP	Dead at 62 months
	23	74	M	Leg	+	-	1	Surgery	Alive at 5 months
	24	82	M	Leg	+		4A	Surgery	Dead at 26 months
	25	50	M	Scalp	+	+	3	RTX-CHOP	Dead at 107 months
	26	84	M	Arm	+	+	4	RTX-bendamustine	Alive at 11 months
	27	70	M	Back	+	+	4	No therapy	Alive at 9 months
Hrgovic [20]	28	55	F	Face	+	+	4A	RTX-CHOP, IT MTX	Relapse at 16 months, intensive treatment including auto-PBSTC, alive at 4 years
Cherukuri [21]	29	61	M	Back	+	+	4A	Unknown	Unknown

Table 1 (continued)

First author [ref.]	Patient Age, years	Gender	Site/lesion	Skin as initial presentation	Other sites at diagnosis	Stage at initial diagnosis	Initial treatment	Outcome	
Our patient	30	73	M	Thigh	+	+	4A	R-CHOP/R-DHAOX, R-BEAM + PBSCT	Alive at 12 months
B									
Ellison [5]	29	66	M	Temporal	-	+	4B		
Sen [11]	30	61	M	Flank, back, thigh/ plaques	-	+	4B		
Dubus [10]	31	72	M	Face, breast, arm	-	+	4		
Ishibashi [15]	32	68	M	Thigh, arm	-	+	4		
Wehkamp [3]	33	85	M	Thigh	-		1E		
	34	NK	M	Back	-	NK	NK		
	35	76	M	Skin NK	-	NK	NK		
	36	55	M	Leg	-	+	4		
	37	77	M	Both legs	-	+	4		
	38	69	M	Leg	-	+	4B		
C									
Wehkamp [3]	39	NK	M	Shoulder	NK	NK	NK		
	40	45	M	NK	NK	+	4		

F, female; M, male; CLB, chlorambucil; PRED, prednisolone; CTX, cyclophosphamide; DOX, doxorubicin; VCR, vincristine; BCNU, carmustine; VP16, etoposide; Ara-C, cytarabine; RTX, rituximab; VMP, bortezomib; MTX, methotrexate; IT, intrathecal; XRT, radiotherapy; CR, complete remission; PBSCT, peripheral blood stem cell transplant; COP, CTX + VCR + PRED; CHOP, CTX + DOX + VCR + PRED; hyper-CVXD, CTX + liposomal daunorubicin + VCR + dexamethasone; hyper-CVAD, CTX + VCR + DOX + DEX; DEXA-BEAM, DEX + BCNU + VP16 + Ara-C + melphalan; R-CHOP, RTX + DEX + Ara-C + oxiplatine; R-DHAOX, RTX + DEX + Ara-C + cisplatin; R-BEAM, RTX + BCNU + VP16 + Ara-C + melphalan; NK, not known.

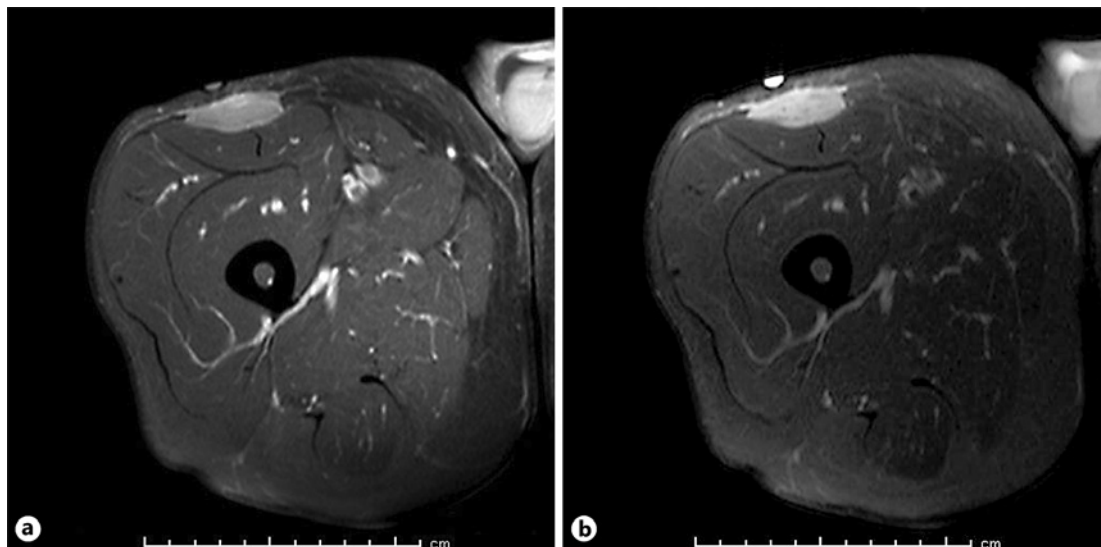


Fig. 1. MRI of the lower extremity demonstrates a subcutaneous mass which is well defined and T2 hyperintense on T2-weighted fat saturated images (a) and which avidly enhances following contrast administration (b).

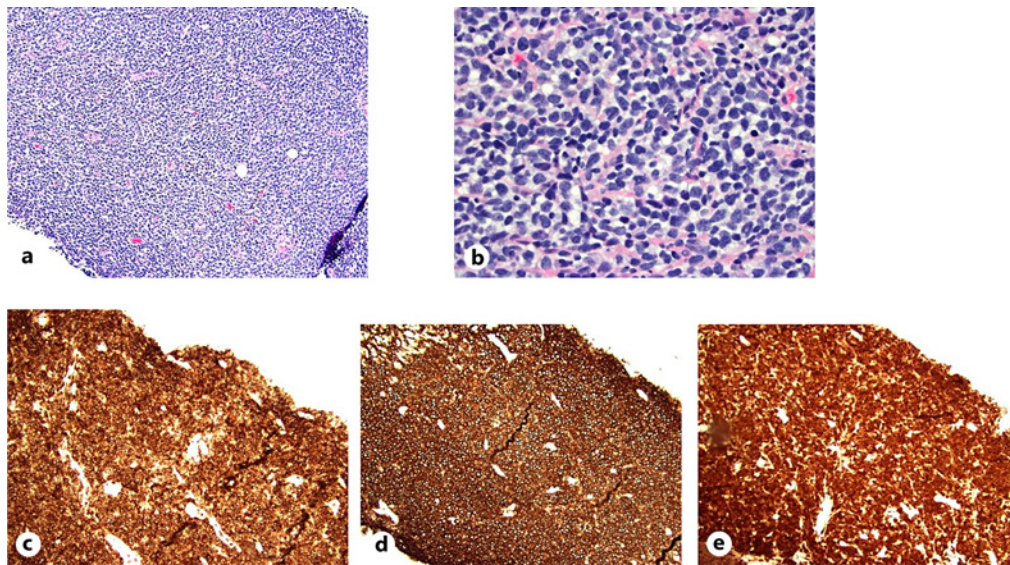


Fig. 2. Histopathology of MCL in the subcutaneous tissue. **a, b** Dense infiltrate of small- to intermediate-sized atypical lymphocytes with irregular nuclear contours, somewhat dispersed chromatin, inconspicuous nucleoli, and scant amphophilic cytoplasm (hematoxylin and eosin, $\times 100$ and $\times 400$). The neoplastic cells are strongly and diffusely positive for CD5 (**c**), CD20 (**d**), and cyclin D1 (**e**) (immunoperoxidase, $\times 100$).

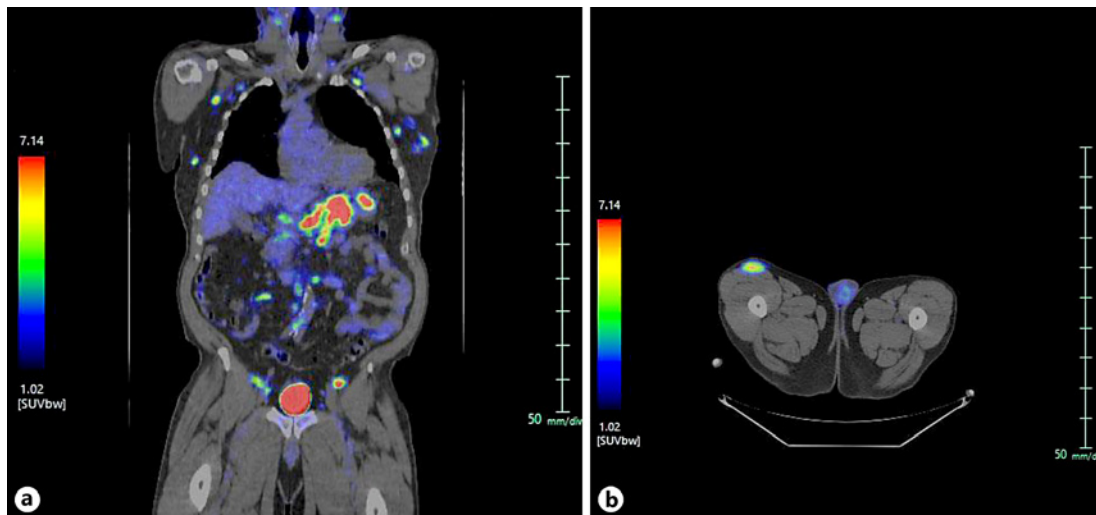


Fig. 3. PET/CT imaging demonstrating FDG uptake within cervical, axillary, gastro-hepatic, retroperitoneal, and pelvic lymph nodes (**a**). Uptake is also seen within the subcutaneous right thigh mass compatible with lymphoma (**b**).

D1, positive for BCL2, and negative for CD3, CD10, CD23, and BCL6. The proliferation index was estimated at $>95\%$ based upon Ki-67 expression. A subsequent biopsy of an enlarged celiac lymph node showed similar morphology and demonstrated kappa-restricted B cells strongly positive for CD20 and CD5 by flow cytometry. These results were consistent with MCL. TP53 sequencing was not performed.

A post-biopsy fluorodeoxyglucose-positron emission tomography (FDG-PET) scan (Fig. 3) revealed abnormal uptake within lymphadenopathy in the neck, bilateral axilla, mesenteric, retroperitoneal, and bilateral pelvic regions. There was also diffuse uptake in the spleen and abnormal uptake in the known right anterior thigh lesion. This is consistent with MCL involvement.

A bone marrow aspirate demonstrated abundant bone marrow particles for review. Numerous megakaryocytes were noted, most demonstrating normal lobulated nuclei. There was trilineage hematopoiesis with maturation and progression of the myeloid series through the segmented neutrophil. No significant dysgranulopoiesis was seen. There was no morphologic increase in blast cells. Numerous erythroid precursors were noted demonstrating normoblastic maturation. No significant dyserythropoiesis was seen. Scattered small- to intermediate-sized lymphocytes were noted and did not appear to be increased in quantity. Occasional plasma cells were seen, not increased in quantity. Flow cytometry showed 2 small monoclonal B-cell populations, one with CD5-positive B cells (2% of lymphoid cells) and a separate one with CD10-positive B cells (5% of lymphoid cells) both showing kappa light chain restriction. CD34+ cells (2.4% of total cells) were not increased. In conclusion, the bone marrow aspirate presented no morphologic or immunohistochemical evidence for bone marrow involvement by MCL. A bone marrow biopsy demonstrated cellularity averaging approximately 50% with areas of intramedullary hemorrhage. Scattered megakaryocytes were adequate in quantity with most appearing to demonstrate normal lobulated nuclei. Trilineage hematopoiesis with maturation was present. There appears to be a mild erythroid hyperplasia. The myeloid to erythroid ratio appears to be decreased and was estimated at approximately 1:1. There was no obvious increase in blast cells. There was no obvious increase in plasma cells. No lymphoid aggregates or atypical lymphocytes were seen. No morphologic evidence for bone marrow involvement by MCL was seen.

The patient went to UC Davis' Comprehensive Cancer Center for therapy that consisted of R-CHOP (rituximab plus cyclophosphamide, doxorubicin, vincristine, and prednisone) alternating with R-DHA0x (rituximab plus dexamethasone, high-dose cytarabine, and oxaliplatin), a slight modification of standard R-CHOP alternating with R-DHAP (rituximab plus dexamethasone, high-dose cytarabine, and cisplatin) as cisplatin was substituted with oxaliplatin due to the patient's age [4]. After 6 courses of chemotherapy, a PET-CT showed complete full metabolic remission. He proceeded to receive high-dose chemotherapy consisting of R-BEAM (Rituxan, BCNU, etoposide, cytosine arabinoside, and melphalan) followed by autologous stem cell transplantation/rescue as consolidation. At present, he is alive with no evidence of disease 12 months following diagnosis.

Discussion

While rare, MCL, which typically presents as advanced-stage disease, is prevalent enough to have a common and therefore recognizable presentation. Most patients will present with multiple enlarged lymph nodes and constitutional symptoms. However, in a few instances the initial presentation will be primarily extranodal, not involving the commonly reported (bone marrow, peripheral blood, gastrointestinal tract, Waldeyer's ring, and/or liver) extranodal sites. Since a full picture of MCL relies on larger series of patient as well as on distinct case reports, we believe that our case further broadens our understanding of how MCL can present clinically.

Over the past few decades, numerous reports have highlighted that MCL can present, albeit infrequently, in the skin. An extensive review of the English literature revealed 42 cases [3, 5–21] (Table 1), including our patient, with MCL skin presentation. Of them, 30 patients

(71%) actually presented to their physician with skin anomalies that led to the diagnosis (section A of Table 1), in 10 patients skin involvement either followed other organ involvement or was found at initial diagnosis but was not the presenting symptom (section B of Table 1), while in 2 cases (section C of Table 1) this feature was not known (these 2 cases are part of a retrospective pathology registry review [3]).

The mean age of the 30 patients presenting with skin involvement was 67 years (range 22–89), and all but 6 (80%) were male; this is similar to what has been reported for patients with MCL in general [2]. It would seem that any area of the skin can be affected, including the scalp [7] and earlobes [9]. Also, the presentation of skin involvement varies greatly and is not restricted to nodules, but can present as erythematous (infiltrated) plaques, a macular rash, papules, petechiae, ecchymosis, or even a subcutaneous mass. This observation suggests that any skin lesion in an older male patient, who also has systemic (B) symptoms, should be biopsied. Moreover, for patients presenting with a skin lesion of the leg, consideration should be given to primary cutaneous B-cell lymphoma, pcDLBCL-lt, which in fact shares many of the immunohistochemical features of MCL. Using the guidelines identified in the National Comprehensive Cancer Network (NCCN) version 1.2020 is paramount in arriving at the correct diagnosis. Adequate immunophenotyping includes CD5, CD20, CD43, CD23, CD10, and cyclin D. The latter is pathognomonic for MCL and allows distinguishing these 2 entities. Cell surface marker analysis by flow cytometry with peripheral blood and/or tumor tissue should also be performed for kappa/lambda, CD19, CD20, CD5, CD23, and CD10. We did not perform TP53 sequencing but recommend this to be done routinely, especially for those patients with an expected aggressive clinical course or if transplantation is considered upfront at diagnosis. Additional testing that can be of use includes LEF1 expression, SOX11 or IGHV sequencing, karyotyping or FISH to look for t(11; 14), t(14; 18), and cell surface marker analysis by flow cytometry with peripheral blood and/or biopsy specimen for CD200.

Based on the occasionally subtle skin manifestations of MCL, that is, cases 1 and 3, we suggest that all patients with MCL would benefit from a detailed skin exam at initial presentation, to ensure appropriate staging.

With regard to staging, we reviewed all case reports and recorded staging if documented or, alternatively if possible, assigned a stage based on the data provided (see Table 1). Based on this, 7 patients presented with stage 1 disease, 1 with stage 3 disease, and 19 with stage 4 disease, while for the remaining 3 patients a definitive stage could not be determined. One with late-stage disease had stage 3 or 4 disease (as determined by us), one had stage 2 or 3 disease, and the last one had either stage 1 or 2 disease. This means that of those for whom staging was clear ($n = 27$), 74% presented with advanced-stage disease, similar to what has been reported for MCL in general.

In terms of treatment, data available from the cases presented do not suggest that patients presenting with cutaneous MCL should be treated any different from others with MCL. In general, patients with advanced-stage disease benefit from adjuvant chemotherapy. Since MCL is clinically heterogeneous, treatment should be adjusted to the patient's age and any prevalent comorbidities. Excellent suggestions for consideration were recently provided by Fakhri and Kahl [22].

In summary, MCL is relatively rare but usually presents with typical symptoms associated with lymphoma. Patients primarily presenting with either cutaneous or subcutaneous lesions are exceedingly rare, but they do form part of the clinical presentation of MCL. These lesions can occasionally be present for months, as in our patient. Immediate adequate investigation is paramount to provide the patient with the best possible outcome.

Statement of Ethics

Written consent to publish all data presented in this manuscript was obtained from the patient.

Conflict of Interest Statement

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Author Contributions

Drew A. Fajardo identified the patient as of interest, collected all relevant information, participated in the literature search, wrote the first draft, and edited subsequent drafts. He approved the finalized manuscript.

Joel France collected and interpreted all relevant laboratory information (he was not the primary pathologist for all studies performed), participated in the literature search, specifically with regard to immunohistochemistry, edited drafts, and approved the finalized manuscript.

Bogna I. Targonska collected and interpreted all relevant imaging information (she was not the radiologist on any of the imaging studies performed), edited drafts, and approved the finalized manuscript.

H. Bobby Kahlon is the patient’s primary care physician. He helped Drew A. Fajardo obtain all clinical information and obtained consent from the patient to publish a manuscript that includes medical information of the patient. He approved the finalized manuscript.

Max J. Coppes actively mentored Drew A. Fajardo in all aspects of the manuscript, connecting him with the right pathologist and radiologist that helped put the manuscript together. He reviewed the drafts in detail, helped to expand literature search, and helped selecting the right Figures and Table. He approved the finalized manuscript and is the corresponding author.

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