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CASE REPORT

Skin lesions and neutrophilic leukemoid reaction in a patient with angioimmunoblastic T-cell lymphoma: a case report and review of the literature

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Key Clinical Message

Here, we present a 53-year-old man with angioimmunoblastic T-cell lymphoma accompanied by skin lesions (vesicles, papulovesicles, and miliary papules symmetrically distributed on extremities and trunk, with more distal lesions increasing in severity). Routine blood tests showed a white blood cell count of 58.97 × 109/L (Neutrophils% 91.64%).

Keywords

Angioimmunoblastic T-cell lymphoma, neutrophilic leukemoid reaction, papulovesicular, skin lesions, vesicles.

Introduction

Angioimmunoblastic T-cell lymphoma (AITL) is categorized as a peripheral T-cell lymphoma and is clinically characterized by a sudden onset of constitutional symptoms, lymphadenopathy, hepatosplenomegaly, immune disease (hyperactivity of the immune system and immunodeficiency) and pleural effusion, ascites, and edema [1–3]. Up to approximate half of patients have skin lesions but vesicles directly caused by AITL are rare [1, 3–5]. Elevated white blood cells (usually eosinophilia) are also often observed in laboratory investigations but to our knowledge, neutrophilic leukemoid reaction caused by AITL was not reported, yet [1–3, 6, 7].

Here, we present an AITL case presenting with rare skin lesions (including vesicles, papulovesicles, and miliary papules) symmetrically distributed on the extremities and trunk, with more distal lesions increasing in severity and neutrophilic leukemoid reaction.

Case Report

A 53-year-old Asian man was referred to our hospital for evaluation of lymphadenopathy and skin lesions. The patient's symptom history was detailed as follows: a dry cough for 3 months, lymphadenopathy for 1 month, skin lesions (started from the extremities) accompanied by pruritus for 3 weeks, dyspnea, and a mild fever for 1 week. At the time of admittance to our hospital, the patient endured severe pain of vesicles on hands and feet. Upon examination, the skin lesions were symmetrically distributed, with more distal lesions increasing in severity. Some large vesicles containing dark reddish exudate were distributed on both fingers and feet (Fig. 1A and B). Some vesicles (large and small) and papulovesicles containing clear transparent exudate were distributed on the extremities (Fig. 1A and C). Miliary papules were distributed on the upper chest and lower abdomen. Few lesions were found in the face while skin lesions were not found in the palms, soles, genitalia, scalp, around the mouth, or oral mucosa. There were palpable superficial lymph nodes in the neck, axillary fossae, and inguinas. The temperature of the skin was between 37.0 and 38.5°C during his hospitalization. The routine blood test showed a white blood cell count of 44.43 × 10^9/L, red blood cell count of 5.02 × 10^{12}/L, and platelet count of 214 × 10^9/L. The cytological study of bone marrow showed hypergranulopoiesis. A computed tomography scan of the chest

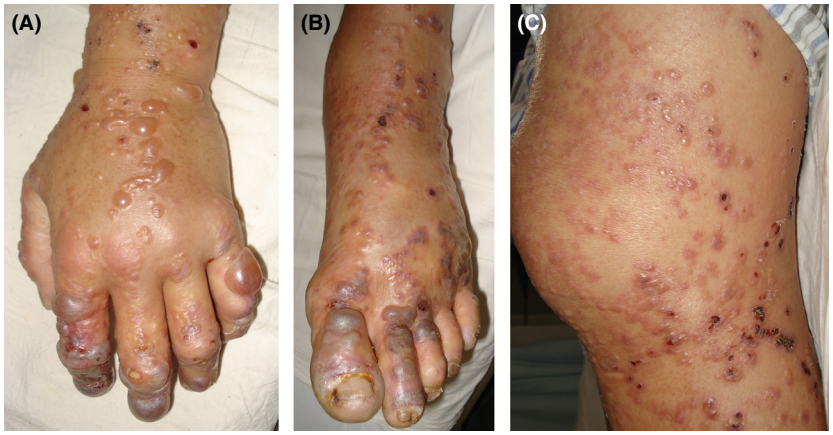

Figure 1. Skin lesions.

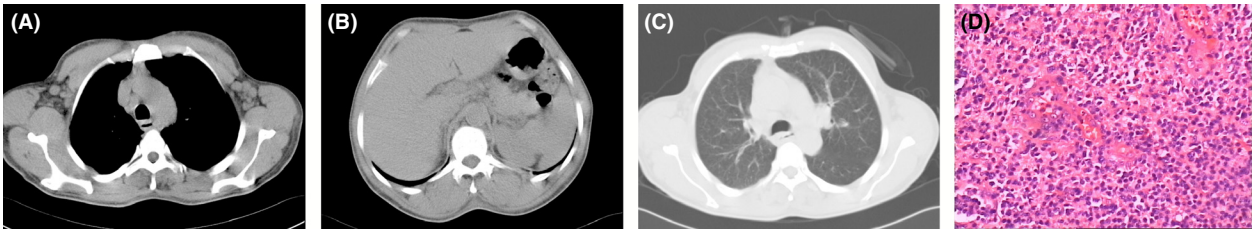

Figure 2. Computed tomography (A–C) and hematoxylin & eosin staining of lymphadenopathy (D). Diffuse infiltration of immunoblasts, abnormal lymphoid cells, clear/pale cells, and proliferation of high endothelial venules are observed in hematoxylin & eosin staining of lymphadenopathy.

revealed lymphadenopathy in the mediastinum and axillary fossae (Fig. 2A–C). Immunochemistry of biopsy from cervical lymphadenopathy showed CD3 was diffuse strong positive, CD20 and CD8 was scattered positive, CD21 and Bcl-2 were focally positive, and Ki67 positive cells ratio was higher than 80%. Based on the cytological study of bone marrow, peripheral blood, immunochemistry and hematoxylin & eosin staining of lymphadenopathy (Fig. 2D), the pathologic diagnosis was AITL.

Both morphine and tramadol were used to control pain. After 4 days of intravenous latamoxef sodium and isepamicin treatment, the routine blood test showed a white blood cell count of 58.97×10^9/L (neutrophils% 91.64%, lymphocytes% 5.24%, monocytes% 2.44%, and eosinophils% 0.42%), red blood cell count of 4.50×10^{12}/L, and platelet count of 177×10^9/L. Manifestations progressively aggravated. Then chemotherapy consisting of cyclophosphamide, epirubicin, vincristine, and prednisone (CHOP-like therapy) was administered. After chemotherapy, there was abatement of clinical manifestations. The number of skin lesions increased during his 9 days of hospitalization. Some vesicles were blisters in the beginning, without erythematous or hemorrhagic base. Some papules on extremities slowly became papulovesicles and it usually took 1 week. None of papules on the trunk became papulovesicles or vesicles. The patient abandoned further treatment.

Discussion

AITL is a rare malignancy accounting for about 2% of all non-Hodgkin lymphoma and a highly aggressive neoplasm of the elderly [1–3, 8]. The median patients' age is approximate 65 years [1–3, 8, 9]. It generally presents with lymphadenopathy and is almost always accompanied by concomitant symptoms, including B symptoms, skin lesions, splenomegaly, hepatomegaly, effusion/edema/ascites, anemia, thrombocytopenia, elevated LDH, hypergammaglobulinemia and so on (Table 1).

Nearly half of AITL patients have skin lesions [1, 3–5, 8]. Skin lesions can precede, follow or be concurrent to lymphadenopathy [5, 10, 11]. AITL skin lesions do not have characteristics to enable it to be distinguished from other skin eruptions, especially from drug eruptions. Therefore, misdiagnosis is not rare [5, 12–14]. Since AITL is almost always accompanied by concomitant symptoms (Table 1), to be

Table 1. Clinical manifestations of AITL (numbers are presented as % except Patient number and Age).

Authors	Federico [1]	Tokunaga [2]	Mourad [3]	Lachenal [9]	Siegert [4]	Aozasa [25]
Patient number	243	207	157	77	62	44
Age, years						
Mean	65	67	62	64.5	64	64
Range	20–86	34–91	20–89	30–91	21–87	25–84
Male sex	56	64		56	58	55
Lymphadenopathy						
Generalized	76			90		84
Localized	24			9		16
Skin rash	21		44	45	49	27
B symptoms	69	60	72	77	68	
Splenomegaly	35			51		39
Hepatomegaly	26			26		52
Effusion/edema/ascites		14	26	25	>38	
Bone marrow involvement	28	29	47			
Extranodal sites, >1	27	23	46			
Anemia	33	61	65	51	57	20
Platelet count $<150 \times 10^9$/L	25	34		20	20	
Elevated LDH	60	75	66	71	70	
Elevated C-reactive protein	35	46		67		
Hypergammaglobulinemia	30		50	51	51	64
Positive Coombs test	13	46	33	58	32	

LDH, Lactate dehydrogenase.

Table 2. Uncommon skin lesions of AITL.

Authors	Age/Gender	Skin lesions	Duration of skin lesions before/after onset of lymphadenopathy	Prognosis
Wechsler [29]	53/F	Erythematous macules; petechiae; purpura	1 month after	Died (23 month)
Matloff [30]	77/M	Macules; petechiae; purpura	1 year before	Alive (4 month)
Seehafer [10]	74/M	Petechiae	Concurrent	Died (3 month)
Seehafer [10]	61/M	Erythroderma and purpura	Concurrent	Alive (48 month)
Seehafer [10]	57/M	Petechiae	10 month before	Alive (48 month)
Schmuth [31]	73/F	Macules; petechiae; purpura	4 week before	Alive (4 month)
Martel [15]		Necrotic purpura, maculopapules and urticaria		Died (26 day)
Martel [15]		Pruritic papulovesicular (prurigo-like) lesion		Alive (96 month)
Hashefi [32]		Maculopapules, petechiae	3 month before	Died (23 month)
Suarez-Vilela [33]	67/F	Sarcoidosis	1 month before	
Huang [34]	62//M	Erythroderma; plaques; nodules	3 year after	Died (3 year)
Jones [35]	67/M	Erythroderma, toxic epidermal necrolysis	Concurrent	Died (5 month)
Tsochatzis [36]	50/M	Polyarthritis, subcutaneous nodules	Concurrent	Died (2 month)
Jayaraman [11]	61/M	Macules, papules, plaques, and nodules	Concurrent	Alive (5 year)
Ortonne [37]	63/F	Nodules, gingival ulceration		
Ortonne [37]	54/M	Maculopapules, hemorragic/necrotic nodules		
Nassar [5]	M/47	Erythematous eruption; violaceous plaques with bullae containing pale yellow exude	3 month before	Alive (4 month)
Smithberger [38]	79/F	Cutaneous tumors and ulcerated nodules	No lymphadenopathy	
Ponciano [39]	36/M	Erythematous plaques, sometimes annular	5 year before	Alive (2 year)

familiar with the common clinical characteristics and types of skin lesions is greatly helpful to avoid misdiagnosis.

Skin lesions in AITL usually accompany pruritus [1–5, 8]. Typical lesions are usually a generalized morbilliform or maculopapular eruptions on the trunk mimicking toxic erythema [2, 4, 5, 8]. In the literature, uncommon skin lesions of AITL are mostly described in case reports or review articles (Table 2). It is not rare that the patient has a generalized pleomorphic rash composed of several types of rashes, such as macula, papules, maculopapules,

Table 3. VZV infection in AITL patients.

Authors	Age/Gender	Skin lesions of AITL	Duration[1]	Manifestations of VZV infection	Duration[2]	Prognosis
Kaneko [14]	49/F	Maculopapule	Concurrent	Disseminated herpes zoster	13 month	Died (15 month)
Zelickson [16]	73/F	Hyperkeratotic papules	1 week after	Hemorrhagic vesicles and bullae	Concurrent	Alive (4 month)
Boni [17]	81/M	Maculopapule	2 month before	Unilateral necrotizing herpes zoster	2.5 month	Died (16 month)
Kanzaki [13]	67/M	No	Not applied	An pharyngeal wall ulcer[3]	Concurrent	Alive (18 week)
Imafuku [12]	67/M	Maculopapule	40 day before	Varicella	1 month	Alive

[1]Duration of skin lesions before/after onset of lymphadenopathy.
[2]Duration of herpes zoster infection after onset of AITL.
[3]Elevation of antibodies against HSV and VZV were observed in the patient but the ulcer was unlikely caused by VZV.

nodules, erythroderma, urticaria, petechiae, purpura and so on. Necrotic purpura, polyarthritis, gingival ulceration, erythematous plaques (sometimes annular), toxic epidermal necrolysis, and hemorragic/necrotic nodules are also reported. Two cases of vesicles (one is pruritic papulovesicular (prurigo-like) lesion and the other is a pale erythematous eruption and violaceous plaques with bullae containing pale yellow exude.) were reported but they are apparently different from our case [5, 15].

Vesicles caused by virus infection (such as HSV, VZV) in AITL patients were sporadically reported [12–14, 16, 17]. Five AITL patients with VZV infection were reported in the literature and are summarized in Table 3 [12–14, 16, 17]. The skin lesions of varicella appear on the trunk and face, and rapidly spread centrifugally to involve other areas of the body. Manifestations consist of maculopapules, vesicles, and scabs in varying stages of evolution. Skin rashes become papules within 12 h then become vesicles within 2 days. Over a very short period of time they scab. This rapid progression from stage to stage characterizes the clinical syndrome of varicella and enables it to be distinguished from certain other vesicular eruptions [12, 18, 19]. Each skin vesicle appears on an erythematous base and immunocompromised patients have more numerous lesions, often with a hemorrhagic base [18, 19]. Since lesions in this patient were not symptoms of herpes zoster because of distribution of lesions and were quite unlike varicella [12, 14, 16–19], lesions are more likely caused by AITL than VZV infection. Further laboratory investigations (assessing for the presence of Epstein–Barr virus infected B cells, VZV in the lesional tissue, pathological examination of the lesional tissue, serological test and so on) can confirm the diagnosis but regretfully, the patient abandoned them.

As mentioned before, hematological changes including anemia, lymphopenia, leukocytosis, neutrophilia, eosinophilia, and thrombocytopenia, are often observed on laboratory investigations but leukemoid reaction is rare. Several cases mimicking plasma cell leukemia and one case of eosinophilic leukemoid reaction were reported by computer-based searches in PUBMED [6, 7, 20]. The routine blood test of this case showed a white blood cell count of 58.97×10^9/L (neutrophils% 91.64%, lymphocytes% 5.24%, monocytes% 2.44%, eosinophils% 0.42%). To the best of our knowledge, this is the first report of neutrophilic leukemoid reaction in AITL.

Treatments to AITL are similar to other noncutaneous peripheral T-cell lymphoma but results are often unsatisfactory. The majority of cases are treated with chemotherapy (CHOP, CHOEP, and CHOP followed by ICE, CHOP followed by IVE, dose-adjusted EPOCH and HyperCVAD are the first line chemotherapies), stem cell transplantation, radiotherapy, and molecular targeted therapy [3, 8, 21]. Folate antagonists (methotrexate, pralatrexate), purine analogs (fludarabine, azathioprine), prednisone, cyclosporine A, lenalidomide/thalidomide, interferon, and denileukin diftitox are reported to be effective in treating AITL [1, 8, 22]. Alemtuzumab and bortezomib are promising therapy options that have been explored recently and warrant [8, 22].

Though about two thirds of patients can achieve a complete remission, only half survive longer than 2 years [3, 8]. International prognostic index (based on age, stage, serum LDH, ECOG/Zubrod performance status, and extranodal site) and prognostic index for peripheral T-cell lymphoma (based on age, performance status, serum lactate dehydrogenase (LDH) levels, and bone marrow involvement) are widely accepted and used [2]. Skin rashes imply poor prognosis [4, 23, 24]. Other factors, such as male sex, B symptoms, edema, ascites, mediastinal lymphadenopathy, anemia, thrombocytopenia, lymphocytopenia, elevated white blood cell, decreased hemoglobin, elevated IgA levels, lymph node eosinophilia, the presence of clear and convoluted cells, high microvessel density measured in the microenvironment, higher ratio of M2 macrophages, failure to achieve complete remission, and drug exposure are reported to correlate with poor prognosis [2, 3, 23–28]. So far, due to limited data available, prognostic factors in AITL sometimes yield controversial results.

Conflict of Interest

The authors have no competing interest.

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