A case report and literature review: subcutaneous panniculitis-like T-cell lymphoma with liver failure as the main manifestation

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Background: Subcutaneous lipofuscinosis-like T-cell lymphoma (SPTCL) is a rare cutaneous lymphoma that often presents as recurrent subcutaneous nodules and may progress to hemophagocytic syndrome with fever and hepatic impairment in some patients. However, no cases of progression to hepatic failure have been reported. Here, we present a case of an adult diagnosed with fever and liver failure as the main manifestation, which was eventually confirmed as SPTCL by skin biopsy, but the patient eventually died due to disease progression.

Case Description: This study retrospectively reports a rare case of SPTCL in a 34-year-old female patient who was admitted with scleral jaundice for 1 month and fever for 20 days. Examination revealed multiple small subcutaneous nodules on the skin of the chest and tibial surface and an enlarged liver and spleen. Laboratory tests revealed hepatic impairment, and common causes of liver failure (viral infection, fatty liver, immune liver damage, etc.) were excluded. We continued to refine the positron emission tomography-computed tomography (PET-CT) examination, which revealed multiple flocculent and nodular hyperdense shadows with increased metabolism in the subcutaneous fat interstices. And then, we performed a skin biopsy and the final pathological diagnosis was SPTCL, but the patient died 1 month after diagnosis due to poor treatment outcome because the disease progressed too rapidly. With this case report, we hope to improve clinicians' understanding of liver injury caused by SPTCL. A review of the literature revealed that this is the first case report in the literature of SPTCL leading to severe liver failure.

Conclusions: For patients presenting with fever and liver injury, primary liver disease cannot simply be assumed, as this presentation may be a manifestation of some extra-hepatic diseases, including SPTCL. For this condition, early detection and early diagnosis may improve the prognosis of patients.

Keywords: Subcutaneous lipofuscinosis-like T-cell lymphoma (SPTCL); liver failure; hemophagocytic syndrome; case report

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Introduction

Subcutaneous panniculitis-like T-cell lymphoma (SPTCL) is a rare cytotoxic T-cell lymphoma, accounting for less than 1% of all non-Hodgkin's lymphomas and 1% of primary cutaneous T-cell lymphomas, mainly involving subcutaneous adipose tissue, with some patients having bone marrow invasion and concomitant phagocytic syndrome, the latter with rapid disease progression and poor prognosis (1). SPTCL was first reported as a new kind of T-cell

lymphoma in 1991 (1) and was officially named by the International Collaborative Group of Lymphoma Research (REAL Classification) in 1994 (2) and incorporated into the peripheral T-cell and natural killer cell tumors in 2001 (3). This disease is a rare type of lymphoma with specific clinical manifestations and histological changes. About 15–20% of patients with concomitant hemophagocytic syndrome may have elevated liver enzymes (4) but early presentation with severe jaundice is relatively rare.

Herein, we report the case of a patient with unexplained liver failure as the main manifestation who was diagnosed with SPTCL after several histopathological examinations and multidisciplinary consultations while being treated with aggressive hepatoprotective therapy and artificial liver support. The patient died 30 days after diagnosis. SPTCL can have high fever and liver function impairment, such as elevated transaminase and bilirubin levels, in some patients, but cases of progression to liver failure are more unusual and have not been reported. Given that the diagnosis and timely treatment of SPTCL remains a considerable challenge, this case may provide a different perspective regarding the approach taken when dealing with cases of jaundice and help to diagnose rare diseases. We present the following article in accordance with the CARE reporting checklist (available at https://atm.amegroups.com/article/view/10.21037/atm-22-5284/rc).

Case presentation

In this paper, we report the case of a 38-year-old female patient who was admitted to our hospital on May 9, 2022, due to scleral jaundice for 1 month and a fever for 20 days. She had started to experience systemic symptoms such as abdominal distension, weakness, and loss of appetite a month prior, which did not concern her at first, but developed skin-scleral yellowing and sunken edema of both lower limbs immediately thereafter. She then went to the local hospital for an examination. The examination revealed abnormal liver function and elevated bilirubin. The local hospital administered hepatoprotective treatment but the patient's liver function did not improve; her jaundice progressively worsened and her condition rapidly progressed to liver failure.

Twenty days before hospital admission, she suddenly developed a fever with a temperature of up to 39 ℃, but without symptoms such as coughing, sputum, or sore throat. The patient's fever was thought to be combined with peritonitis, and thus, she received artificial liver treatment and ceftriaxone (2,000 g every 24 h) anti-infective treatment three times during her hospitalization. However, her fever was not well controlled and her temperature profile still fluctuated from 37.5 to 38.8 ℃. Also, her skin sclera yellowing did not improve. Ten days before admission to the hospital, some dark red rashes appeared on her skin surface; the lesions were distributed on the neck, anterior chest wall, and back, and varied in size and gradually increased in number (see *Figure 1*). She then underwent her first skin biopsy (right tibia) at the local hospital. She had been intermittently treated with oral hormones 4 years ago due to the discovery of subcutaneous nodules on the anterior tibia, diagnosed as erythema nodosum at another hospital. Apart from this, the patient denied recent travel, surgery, or trauma, and reported no history of infectious disease, diabetes, or alcohol or drug use.

At admission, the patient had a temperature of 36.2 ℃, a pulse rate of 98 beats/min, an increased respiratory rate of 24 breaths/min, a blood pressure of 99/65 mmHg, and oxygen saturation in ambient air of 98%. Physical examination revealed severe yellowing of the skin and sclera over the whole body. Skin examination revealed large dark red plaques of variable size and fusion in the anterior cervical region, anterior chest, and back, and several small dark red nodules (approximately 0.1–0.2 cm) in diameter were visible around the right breast, with no tenderness on pressure. A post-biopsy wound was seen on the anterior aspect of the right tibia, which was healing. Two well-defined infiltrative erythemas (size: ~5 cm × 5 cm) were visible on the dorsum. The patient's breathing sounds in both lungs were low and a few wet sounds could be heard. The abdomen was bulging in appearance, with mildly increased muscle tone, no pressure pain was found,

Highlight box

Key findings

- SPTCL is a rare cause of liver failure.

What is known and what is new?

- Some of the previous reports of SPTCL may be combined with liver function abnormalities, however, this is the first case of SPTCL with fever and liver failure as the main manifestations
- The possibility of SPTCL needs to be considered early in the screening of patients with clinical liver failure for its etiology.

What is the implication, and what should change now?

- Early skin pathology biopsy may help in the early diagnosis of SPTCL.

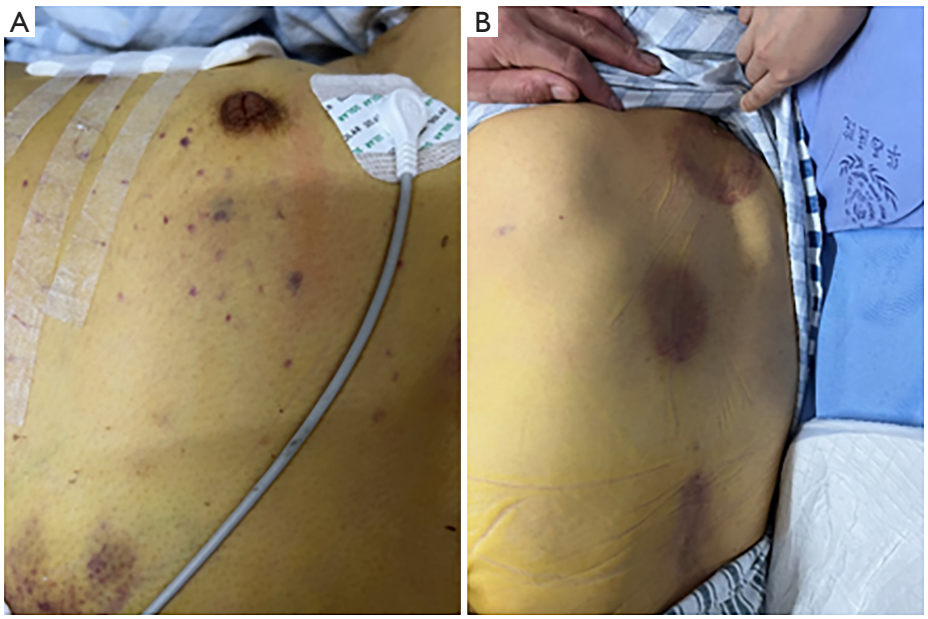

Figure 1 Patient's skin rash. (A) Chest skin rash; (B) back skin rash.

Table 1 Blood test results

Inspection items	Reports	Comments
RBC	2.45×10^{12}/L	Decreased
Hb	75 g/L	Decreased
WBC	26.07×10^{9}/L	Increased
NEUT (%)	93.2%	Increased
LYMPH (%)	1.3%	Decreased
PLT	284×10^{9}/L	Normal
Blood ammonia	57.4 μmol/L	Increased
NK count	24/μL	Decreased
B count	14/μL	Decreased
CD3 count	240/μL	Decreased
CD4 count	194/μL	Decreased
CD8 count	43/μL	Decreased
CRP	43.20 mg/L	Increased
IL-6	61.40	Increased
IL-2R	3,845.0	Increased
PCT	0.60 ng/L	Increased
Complement C3	0.4870	Decreased
Complement C4	0.1430	Decreased

RBC, red blood cell; Hb, hemoglobin; WBC, white blood cell; NEUT, neutrophil granulocyte; LYMPH, lymphocyte percentage; PLT, platelet count; NK, natural killer cells; B, B cell; CRP, C-reactive protein; IL, interleukin; PCT, procalcitonin.

there were positive mobile turbid sounds on percussion, and severely depressed edema below the knee joint in both lower extremities was observed.

The patient remained recurrently febrile after admission, her red blood cell (RBC) count was decreased (2.45×10^{9}/L) and her hemoglobin value showed moderate anemia (75 g/L). Her neutrophil count was 10.26×10^{9}/ L (93.2%), and her C-reactive protein (CRP) and Procalcitonin (PCT) levels were elevated (43.20 mg/L and 0.6 ng/L, respectively). Liver function tests showed significantly elevated bilirubin levels [TBIL 244.3 μmol/L (normal range, 5.0–28.0 μmol/L)], normal alanine aminotransferase [40 IU (normal range, <50 IU/L)], low blood albumin level [24.4 g/L (normal range, 40–55 g/L)], and prolonged clotting time (PT 14.8s). Further blood tests revealed that the patient had high levels of inflammatory markers, including IL-6, ferritin, and IL-2 receptors. The tuberculosis interferon-gamma (IFN-γ) G [(1, 3)-β -D-polyglucan] test, galactomannan (GM) test, and autoantibodies and autoimmune liver disease antibodies were negative (the blood test results are displayed in *Table 1*; the liver and coagulation function comparisons are shown in *Table 2*). The patient tested negative for SARS-CoV-2 based on a Polymerase Chain Reaction (PCR) sample of a nasopharyngeal swab. Her abdominal computed tomography (CT) scan revealed a diffuse decrease in liver parenchymal density, an enlarged spleen,

Table 2 Liver and coagulation function comparison

Date	TB (umol/L)	DB (umol/L)	ALT (IU/L)	AST (IU/L)	ALB (g/L)	PT (s)	INR
5.9	244.3	191.2	40	211	24.4	14.8	1.35
5.12	248.5	230.7	30	186	21.9	15.6	1.46
5.15	274.3	251.7	25	130	18	16.9	1.59
5.19	288.6	260.5	30	190	20.5	17.4	1.64
5.25	314.6	273.8	31	120	26.9	16.3	1.52

TB, total bilirubin; DB, direct bilirubin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALB, albumin; PT, prothrombin time; INR, international normalized ratio.

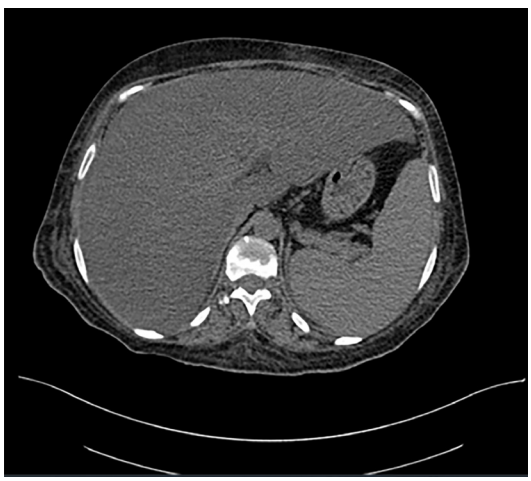

Figure 2 Abdominal CT. The density of the liver parenchyma diffusely decreased significantly, and the spleen enlarged. CT, computed tomography.

and abdominopelvic effusion (see *Figure 2*). Therefore, the patient was clinically diagnosed with the following: (I) subacute liver failure; (II) fever with rash pending diagnosis; and (III) severe fatty liver.

After admission, the patient received anti-infective treatment with Imipenem Cilastatin (500 mg every 8 hours) and hepatoprotective treatment with polyenyl phosphatidylcholine and magnesium isoglycyrrhizinate, as well as two artificial liver treatments on May 16 and May 20. Her bilirubin levels did not resolve significantly and she continued to experience recurrent fever. In addition, the cause of the patient's liver impairment was difficult to explain by infection or fatty liver alone, and we encountered difficulties in both diagnosis and treatment. To clarify whether she had hemophilia syndrome and hematologic tumors, we performed a bone marrow aspiration biopsy but found no relevant evidence to support this diagnosis. Thus, we continued to refine the PET-CT examination, which revealed multiple flocculent and nodular hyperdense shadows with increased metabolism in the subcutaneous fat interstices. On May 15, the patient underwent a second skin biopsy (right chest wall) on the advice of the dermatologist, and a skin biopsy section of the right tibia from a local hospital was consulted. Surprisingly, both pathologies suggested the same pathological diagnosis: subcutaneous lipofuscinosis-like T-cell lymphoma (aggressive) (*Figure 3*). Immunohistochemistry showed the following: aforementioned lymphocytes CD20 (negative), CD3 (positive), CD4 (sight positive), CD5 (positive), CD8 (positive), CD30 (negative), GranzymeB (positive), TdT (negative), Ki-67 (positive, 60%), EBER1/2-ISH: lymphocytes (negative) (*Figure 4*), and gene rearrangement (PCR & gene scan): TCRγ gene rearrangement undetected.

On May 24, blood and deep venous catheter cultures both yielded Staphylococcus epidermidis, and venous catheter-related infection was considered. Therefore, we removed the deep venous catheter and started anti-infective treatment with tigecycline (50 mg every 12 h) combined with the drug sensitivity results of the pathogen. Following anti-infective treatment, the patient's temperature showed an improving trend. However, we noticed that although the peak temperature decreased, it still fluctuated around 38 ℃, so the patient's fever was considered a possible co-infection based on lymphoma. After treatment, the patient's general condition gradually deteriorated, with poor diet and depression with persistent anemia and hypoproteinemia. After informing the patient's family of her condition, they refused to receive further treatment at our hospital, and the patient was discharged on May 28 and returned to her local hospital for further treatment. We followed up on the patient's condition by telephone and learned that the patient had died on June 23 due to persistent deterioration. The timeline of the patient with relevant data on the

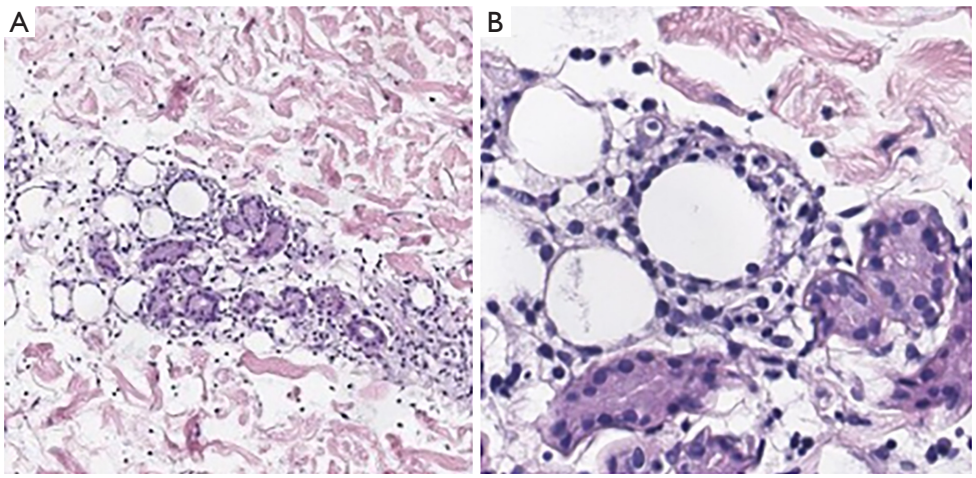

Figure 3 Hematoxylin-Eosin (HE) stain: A, 40×; B, 200×. A small number of lymphocyte infiltrates of moderate size and irregular nuclear shape were seen around the appendages of the dermis, and were also seen around the fat vacuole cells. The infiltrating lymphocytes were mainly T cells of the CD8 phenotype.

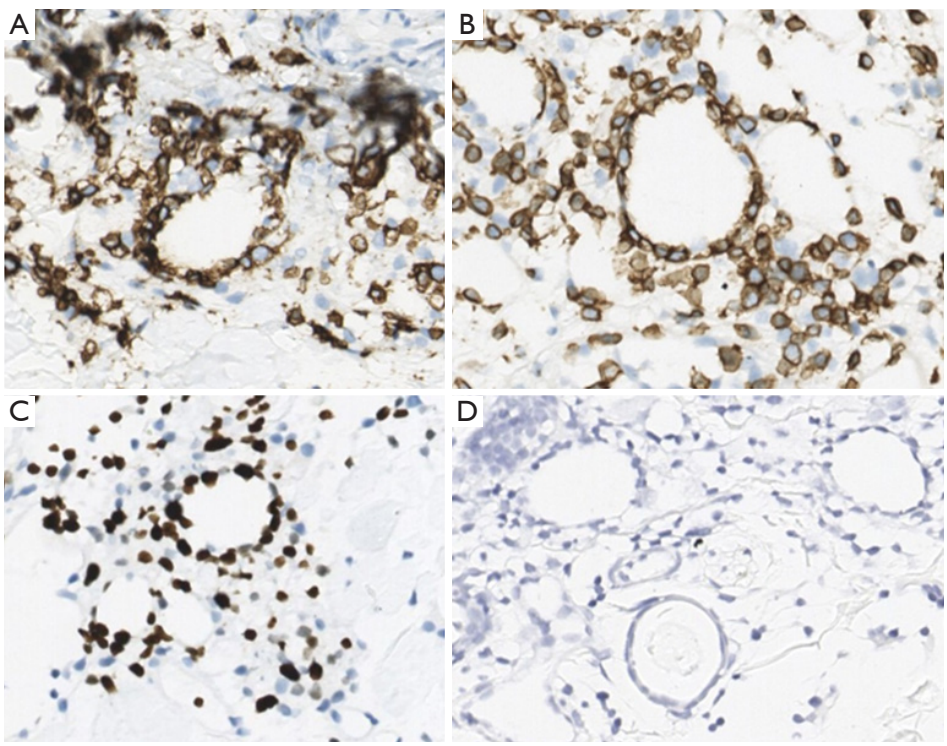

Figure 4 Immunohistochemical staining was used for the diagnosis of SPTCL, 200×. (A) CD8+; (B) CD3+; (C) Ki-67+; (D) EBER–. SPTCL, subcutaneous panniculitis-like T-cell lymphoma.

episodes and interventions is presented in *Figure 5*.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patient's families

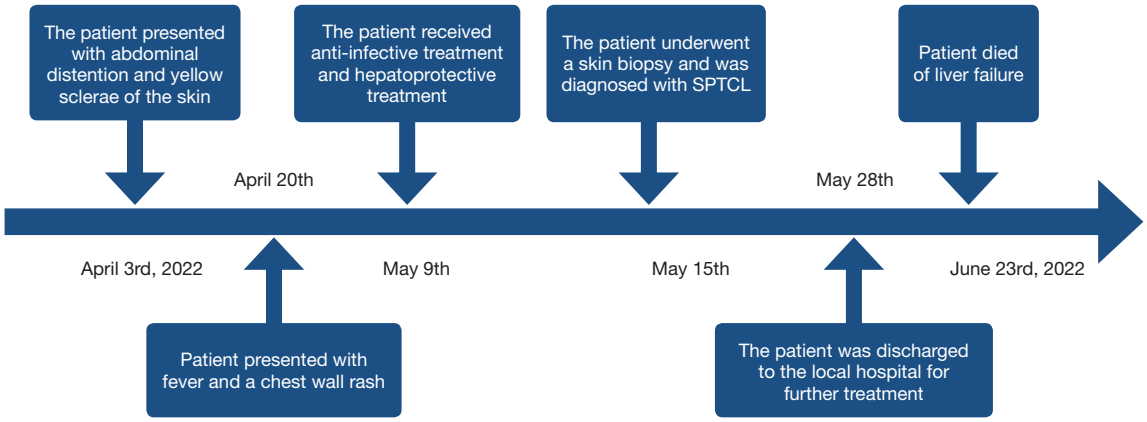

Figure 5 Timeline of the patient with relevant data of the past episodes and interventions. SPTCL, subcutaneous lipofuscinosis-like T-cell lymphoma.

for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

SPTCL is a rare primary cutaneous lymphoma composed of cytotoxic alpha-beta T-cells that mimics panniculitis. It accounts for less than 1% of all non-Hodgkin lymphomas (4), and is rare in Caucasians (5,6) but more common in Asians (7,8). Regarding incidence, there are no significant sex differences and it can occur at any age, with an average age of onset of 36 years (4,9). The primary clinical manifestation is non-specific subcutaneous nodules, which are easily misdiagnosed. The cutaneous lesions are usually on the trunk and extremities, and can sometimes be observed on the face, neck, axilla, groin, and hip (4,9-12). SPTCL predominantly presents as melasma to reddish subcutaneous plaques and nodules without tenderness, which can vary in size (diameter, 0.5–0.9 cm) (4,10,13-16). Furthermore, there is no obvious lymph node involvement as well as little ulceration and skin disease (10,17).

In the case of our patient, skin erythemas (of various sizes) were distributed on the forebreast, back, and calf, which is a typical skin change of SPTCL. However, our patient's other clinical manifestations were very special. Firstly, she began with prominent and severe liver function damage, including bloating, fatigue, anorexia, and yellow staining of the skin sclera, combined with ascites, hypoproteinemia, and coagulation dysfunction, which rapidly developed into liver failure, and the causes of liver failure exclude viral, immune, alcohol, drug and other factors. Although our patient had a fatty liver, it did not explain her severe liver failure, and SPTCL was considered more likely to be the cause of her liver failure. This phenomenon is inconsistent with the mild liver injury common to SPTCL, such as elevated liver enzymes (10,12,18). A trans-jugular vein hepatic puncture test can help us to identify the cause of liver failure. Artificial liver therapy is not effective in liver failure due to neoplastic etiology, which eventually leads to the death of the patient.

Secondly, in addition to liver damage, patients also have a fever, peripheral blood shows a significant elevation of white blood cells and anemia, and antibiotic therapy is not effective. So, in addition to considering lung and abdominal infection, the possibility of SPTCL combined with hemophagocytic syndrome (HPS, also known as hemophagocytic lymphocytosis, HLH) should also be considered as the potential cause of fever. SPTCL is often complicated by HPS (around 20% of cases) (4,19), which is mainly characterized by the following clinical manifestations: hepatosplenomegaly, persistent high fever, and abnormal coagulation function (20-22). A lymphokine, possibly produced by malignant cells, activates monocyte-macrophages, causing macrophages in the bone marrow, liver, spleen and lymph nodes to engulf blood cells (20,23). Our patient exhibited a partial basis for inflammatory activation combined with hepatosplenic growth according to the current consensus/guidelines for the diagnosis and treatment of hemophilic syndrome (24,25); however, there was no significant decline in white blood cells and platelets, and no blood-thirsty cells were found in the bone marrow. At present, it is not possible to fully diagnose hemophagocytic syndrome, but it may also be

an early stage of hemophagocytic syndrome and therefore underrepresented.

In this case, there was a need to differentiate our patient's condition from immune system disorders before a definitive diagnosis could be made. First, the patient is a woman with a previous diagnosis of erythema nodosum, for which hormonal therapy was effective. Also, her clinical presentation included fever and rash, but her immune markers were normal for her autoantibodies, ANCA, and IgG4, except for a mild decrease in complement. Furthermore, apart from liver damage, our patient exhibited no other multi-system damage and thus, a diagnosis of immune system disease was not supported. Finally, the diagnosis of SPTCL was confirmed through a skin biopsy. In addition to her skin damage, the enlargement of multiple lymph nodes and significant peritoneal thickening may also have been caused by tumor cell infiltration.

SPTCL is a special type of T-cell lymphoma, and the gold standard for diagnosis still relies on pathology, which is characterized by primary small, medium, or large polymorphic T cells in the subcutaneous adipose tissue, arranged in a garland of flowers around fat cells, as well as tumor cells with significant nuclear fragmentation and nuclear necrosis (12,18). Extensive adipose necrosis can be seen in tumor tissue tissues, which are formed after histiocytes engulf erythrocytes. Also, necrotic detritus and reactive histiocytes (often with adipose necrosis and coagulant necrosis) can be seen, especially when there is considerable damage, and necrotic fat often leads to histiocytic reactions, including multinucleated giant cell or granulomatosis (12,26,27).

SPTCL tumor cells are more aggressive, which results in rapid disease progression, and the natural course of the disease is dangerous. At present, there is no standard treatment for SPTCL (10), but it is sensitive to systemic combination chemotherapy such as cyclophosphamide + doxorubicin, vincristine + prednisone regimens (CHOP regimens) (13,28-30), and topical radiotherapy is sensitive and effective (16,31,32). Despite the diagnosis of SPTCL in this case, the patient could not receive chemotherapy due to severe liver failure and secondary infection, which eventually led to her unfortunate death in February following the onset of the disease. Clinically, partial relapse-refractory SPTCL has gradually emerged, and the treatment mode of combination therapy is currently the first choice for patients with relapsed refractory SPTCL (4,16,33). Immunosuppressants are more effective for SPTCL patients with TIM-3 deficiency (9,34). Some studies have suggested that allogeneic hematopoietic stem cell transplantation is justified for the treatment of refractory SPTCL (35,36). SPTCL generally has a good prognosis and treatment outcomes, with a 5-year overall survival rate of 85–91% (16,26). However, further research involving large numbers of samples, multi-center studies, and more clinical observation are still required.

However, there are some limitations in our study that should be noted. Due to our lack of understanding of the functional impairment of the liver due to such rare diseases. The etiology of liver function impairment was not clarified early, and the patient experienced progressive deterioration of liver function and eventually died. Therefore, the possibility of SPTCL needs to be considered early in clinical work-up for screening the etiology of fever with liver function impairment.

Conclusions

In summary, this case report illustrates that although SPTCL usually causes mild liver damage, it can also sometimes result in severe liver damage and even liver failure. Moreover, it can be the main manifestation of SPTCL, often leading to rapid progression of the disease and affecting further treatment, with a poor prognosis. Therefore, for patients with rashes, it is recommended to perform a skin biopsy as soon as possible and to determine whether the diagnosis is a malignant disease at an early stage, so as to start treatment promptly, improve the prognosis of patients, and enhance the survival rate.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://atm.amegroups.com/article/view/10.21037/atm-22-5284/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://atm.amegroups.com/article/view/10.21037/atm-22-5284/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Declaration of Helsinki (as revised in 2013). Written informed consent was obtained from the patient's families for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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