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ORIGINAL ARTICLE

Analysis of the clinical characteristics of thirteen patients with Weber-Christian panniculitis

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Abstract

Background Weber-Christian disease (WCD) is an uncommon, idiopathic disease that is challenging to diagnose and has an unclear treatment protocol. We reviewed thirteen patients with WCD and analyzed their clinical characteristics. The purpose of this article was to improve the understanding of this rare disorder.

Methods Thirteen cases of WCD were analyzed retrospectively regarding their clinical manifestation, laboratory results, misdiagnoses, therapy, and outcome.

Results Of the thirteen patients diagnosed with WCD, the majority were female (male to female ratio, 2:11), with a mean patient age of 50.1 years. Subcutaneous nodules were the most commonly reported symptom, followed by fever, arthralgias/arthritis, and myalgia. The laboratory results were typically nonspecific. In total, 61.5% patients were misdiagnosed before pathology confirmed the diagnosis of WCD. Most patients were treated with corticosteroids and/or immunosuppressants. Two patients were treated surgically. While long-term remission was successfully achieved in some patients, others had recurrent symptoms.

Conclusion WCD was predominantly observed in female patients in our cohort. Subcutaneous nodules and fever were the most common clinical characteristics. In addition, the patients' laboratory test results were nonspecific, which led to a high misdiagnosis rate. In this study population, corticosteroid and/or immunosuppressant treatments were efficacious therapeutic interventions for WCD.

Key Points
- *Subcutaneous nodules and fever were the most common clinical characteristics in Weber-Christian panniculitis.*
- *Misdiagnosis rate was higher in Weber-Christian panniculitis patient; tumors, bacterial infections and rheumatic diseases were the most common misdiagnoses.*
- *Corticosteroid and/or immunosuppressant therapy was effective in most Weber-Christian panniculitis patients.*

Keywords Clinical features · Corticosteroids · Diagnosis · Weber-Christian disease · Immunosuppressants

Introduction

Weber-Christian disease (WCD) is a chronic recurrent nonsuppurative disease characterized by the formation of painful dense nodules in the subcutaneous fat and accompanied by episodic fevers, chills, and myalgias. It is often characterized by involvement of the visceral organs [1]. In 1925, Weber [2] reported a case of relapsing nonsuppurative panniculitis showing phagocytosis of the subcutaneous fat cells by macrophages, while Christian [3] reported relapsing febrile nodular nonsuppurative panniculitis in 1928. Thereafter, Bailey [4] reported relapsing febrile nodular nonsuppurative panniculitis as WCD in JAMA in 1937. Because the biopsy of the nodules demonstrates lobular panniculitis, WCD is also called idiopathic lobular panniculitis. The clinical characteristics of WCD are complex

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and may involve the lungs, heart, intestines, spleen, kidney, adrenal glands, and even orbits, making early diagnosis difficult and misdiagnosis common [5]. Furthermore, while the prognosis of WCD is variable [6], patients with prominent visceral involvement typically have the worst prognosis. We analyzed 13 patients diagnosed with WCD at the Zhejiang Provincial People's Hospital in China to investigate the etiology, clinical manifestations, laboratory results, therapies, and prognosis to improve the diagnostic accuracy for WCD.

Methods

In a retrospective review, 14 patients were identified with a discharge diagnosis of WCD from the Zhejiang Provincial People's Hospital between 2000 and 2015. Among the 14 cases, 13 were confirmed by pathology, and 1 patient presented with no clinical symptoms, with only a CT scan revealing mesenteric panniculitis. Since no biopsy was performed, this case was excluded. In all, 13 eligible patients were identified. The clinical/laboratory results, misdiagnoses, therapies, and outcomes were analyzed. Because this study was based on the retrospective analysis of existing administrative and clinical data, the need to obtain informed patient consent was waived by the Ethics Review Committee of Zhejiang Provincial People's Hospital.

Results

General information and triggers

Our series included 2 men and 11 women (male to female ratio, 1:5.5) with an average age of 50.1 years. The mean time to diagnosis of WCD was 40 days, and the longest time was 11 years. Among the 13 patients, we found that some patients might have risk factors. One patient reported an upper respiratory tract infection 1 month prior to diagnosis, 1 patient received Lipodissolve treatment at a beauty parlor 2 months prior to diagnosis, and in 1 other patient, symptoms accompanied saphenous vein varicosity and gradually accelerated over the 2 weeks prior to diagnosis. The remaining patients had no identifiable triggers (Fig. 1 and 2).

Clinical manifestations (Table 1)

Subcutaneous nodules

In our series, all patients had subcutaneous nodules; among them, subcutaneous nodules were the initial symptom in 7 cases. In 1 case, subcutaneous nodules were accompanied by fever, while 2 other patients started to develop subcutaneous nodules after the onset of fever. The subcutaneous nodules

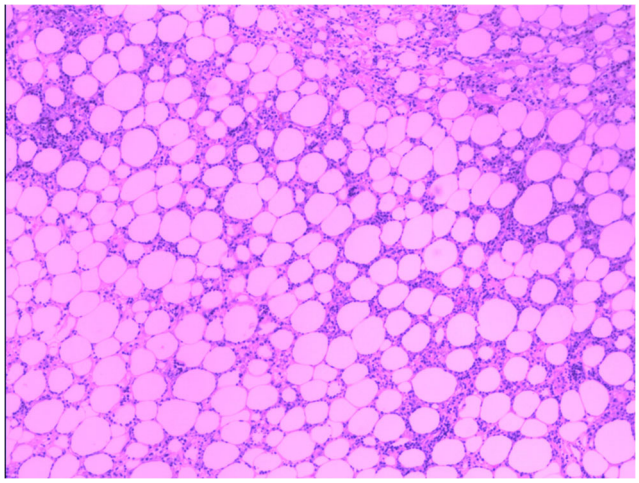

Fig. 1 The biopsy of the subcutaneous nodules revealed a lobular inflammatory lesion presenting acute and chronic infiltration with the obvious liquefaction of fatty cells

were in the lower extremities in 76.9% of cases and in the upper extremities in 46.2% of cases. The description of the subcutaneous nodules was as follows: hard, fixated, and with variable quantity, and the local skin was a burgundy or deep purple color. The nodules were tender and measured 0.5–3 cm in size. Four patients had partial skin pigmentation and dimples where nodules had regressed. In 1 case, the nodules were broken and emitted a yellow liquid, but bacterial cultures were negative.

Fever

In total, 8 patients had a fever. Fever was the first clinical symptom in 5 cases, whereas 2 patients developed fever in the days following the appearance of subcutaneous nodules. In 1 case, fever was accompanied by subcutaneous nodules. The fevers were irregular in 8 cases, with the body temperature ranging from 38 to 40 °C. Nonsteroidal anti-inflammatory drugs (NSAIDs) were effective against the fever.

Arthralgias/arthritis and myalgia

Eight patients had joint pain, with knee pain being observed most often (5 cases), followed by wrist pain (2 cases), and ankle pain (1 case). In all cases of joint pain, swelling and joint stiffness accompanied the pain. Joint deformity did not occur in any patients. The pain was persistent and symmetrical. Moreover, 5 patients had myalgia (muscle pain) that was described as bilateral low extremity muscular soreness.

Other symptoms and underlying disease

Abdominal pain was reported in 3 cases, pulmonary nodules were reported in 2 cases, and hepatosplenomegaly was reported in 1 case. In terms of underlying concurrent diseases, 3

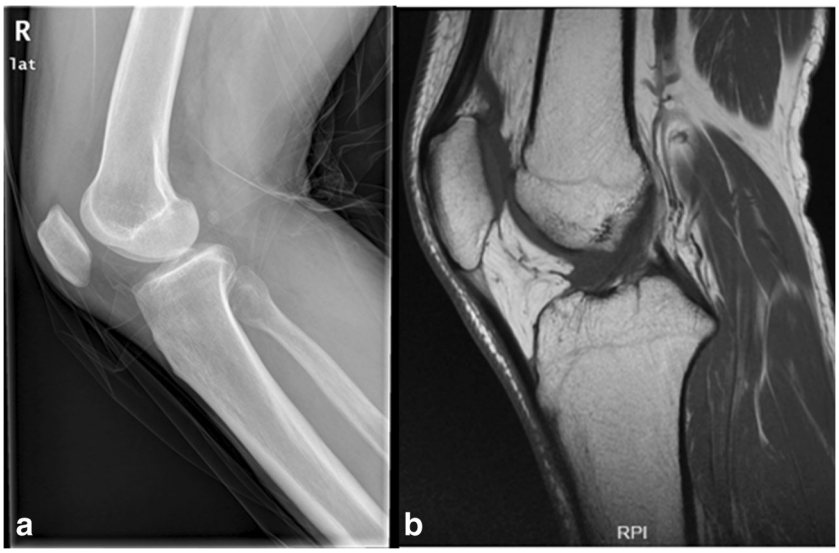

Fig. 2 A:X-ray showed no obvious deformity of right knee joint (lateral film);B: MRI show the thickness of right knee articular cartilage got close to normal and without

cases were complicated by systemic lupus erythematosus (SLE), 2 patients had fatty liver, 2 patients had biliary tract disease, 2 patients had hypertension, 1 patient had saphenous vein varicosity, 1 patient had interstitial pneumonia, and 1 patient had chronic hepatitis B (CHB).

Laboratory features (Table 2)

Five patients had elevated white blood cell (WBC) counts (leukocytosis), 2 patients had low WBC counts (leukopenia), 6 patients had an increased C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) values, 2 patients had elevated liver enzymes, 1 patient had compromised renal function, 3 patients had complement and immunoglobulin dysfunction, and 3 SLE patients were positive for antinuclear antibodies (ANA). Furthermore, the 7 patients who underwent bone marrow biopsy had no abnormal findings.

Therapy

Our therapeutic approach varied. Two patients underwent surgical treatment, and the remaining patients were treated with medications, including 4 patients treated with NSAIDs, 5 patients treated with dexamethasone (0.5–1 mg/day or a dose equivalent amount gradually tapered over 12 weeks), and 2 patients received hormone plus immunosuppressant therapy, with one patient receiving cyclophosphamide (CTX) (100 mg/day) plus dexamethasone (0.5–1 mg/day) therapy for 8 months

Table 1 The pathological features of thirteen patients with Weber-Christian panniculitis

Patient	Sex/age	Biopsy site	Pathological features
1	M/53 years	Lower limb	Predominantly lobular panniculitis with mixed inflammatory cell infiltration
2	M/35 years	Upper limb	Lobular panniculitis composed of mononuclear and foamy cells
3	F/61 years	Upper limb	Lobular inflammatory lesion presenting acute and chronic infiltration with abundant foamy histiocytes and giant cells
4	F/67 years	Lower limb	Inflamed fatty tissue with characteristic infiltration by mononuclear cells and lipid-laden macrophages
5	F/58 years	Lower limb	Lobular fat necrosis with inflammatory cell infiltration
6	F/22 years	Upper limb	Lobular panniculitis characterized by infiltration of inflammatory cells, mainly composed of lymphocytes and histiocytes
7	F/29 years	Lower limb	Predominantly neutrophilic infiltrate in the subcutaneous lobule
8	F/61 years	Upper limb	Lobular panniculitis with neutrophilic granulomatous inflammation
9	F/75 years	Lower limb	Lobular panniculitis with pronounced neutrophilic infiltrate and foamy histiocytes
10	F/52 years	Upper limb	Lobular panniculitis with infiltration of foamy histiocytes
11	F/53 years	Upper limb	Lobular infiltrate, foamy macrophages, and fibrosed septum
12	F/48 years	Upper limb	Infiltration of fatty tissue by inflammatory cells and considerable fibrosis
13	F/38 years	Lower limb	Lobular panniculitis composed of inflammatory and foamy cells

and the other patient receiving azathioprine (2 mg/kg/d) plus dexamethasone (0.5–1 mg/d) treatment for 6 months (Tables 3 and 4).

Outcomes

No deaths occurred during follow-up. Of our 13 patients, the 2 surgical patients were lost to follow-up after hospital discharge. The remaining patients were followed up for 8 to 156 months, with a mean follow-up time of 63.1 months. There were 8 patients without recurrence during follow-up, and 3 female patients were still attending regular follow-up visits at our hospital at the time of article submission to this journal. Among the 3 female patients, one patient still had subcutaneous nodules and fever, one patient had subcutaneous nodules only, and one patient had fever only, without subcutaneous nodules. Additionally, during follow-up, we found that patients receiving hormone monotherapy were more likely to develop secondary hypertension (2 cases), osteoporosis (1 case), osteonecrosis (1 case), or other undesired consequences. Adverse reactions due to combined drug therapy were rare, as only one patient experienced elevated hepatic enzymes.

Misdiagnosis

In our study, before pathology confirmed the diagnosis of WCD, 61.5% (8/13) of the patients were misdiagnosed multiple times; the longest period of misdiagnosis was 2 years. The misdiagnosed diseases were as follows: 2 patients were misdiagnosed with tumors (1 with lymphoma and 1 with fibrocystoma); 3 patients were misdiagnosed with bacterial infections (1 with erysipelas, 1with lymphadenitis, and 1 with sebaceous cyst), and 3 patients were misdiagnosed with rheumatic disease (1 with rheumatic fever, 1 with erythema nodosum, and 1 with localized scleroderma).

Discussion

WCD is a rare autoinflammatory disease involving the adipose tissue, most commonly the subcutaneous fat; nonspecific symptoms of systemic disease, such as fever and arthralgias, are often observed. Active organ inflammation may also be detected, usually involving the liver, spleen, bone marrow, and mesenteric adipose tissue. However, the etiology and pathogenesis of WCD are still unclear and may be related to infections by *Streptococcus* and tuberculosis bacteria, drugs, immune deficiencies, and abnormal lipid metabolism [7]. In recent years, most scholars have concluded that the pathogenesis of WCD is related to dysfunctional fat metabolism and an abnormal immune response, which means that WCD usually coexists with other diseases rather than presenting as a solitary disease. The study by White and Winkelmann [8] revealed that 40% (12/30) of patients with WCD had another conditions at the same time, such as phlebitis or phlebitis after another syndrome, trauma, infections treated with penicillin, and even lymphoma and leukemia. Some scholars consider WCD to be associated with autoimmune diseases, such as SLE, myositis, Sjögren syndrome (SS), and vasculitis [9, 10] suggesting the possible role of an abnormal immune response. In our study, we found that abnormal immunoglobulin levels were detected in 3 patients, and abnormal complement was also detected in 3 patients, providing possible evidence for an abnormal immune response. Lastly, the disease courses of 3 patients were complicated by SLE; 1 patient had a history of injection lipolysis, and 1 patient had a history of CHB. Many of these diseases are associated with immune dysfunction.

The clinical manifestations of this disease were complex and varied. The most common clinical manifestations were subcutaneous nodules, erythema, and fever. Subcutaneous nodules were predominantly found in the limbs and abdomen. Fever, when present, was a high fever and was often accompanied by chills, joint pain, muscle soreness, abdominal pain, and skin rupture. WCD can involve not only the skin but also the internal organs. The most commonly affected organ was the liver, which manifested as fatty liver, diffuse hepatomegaly, multiple low-density shadows in the liver, and calcification of liver membranes. Liver pathology showed fatty degeneration and focal necrosis of liver cells, abnormal metabolism of adipose tissue, and immune injury as the main causes of associated liver disease [11]. Indeed, other organs such as the lungs, heart, and kidneys can also be affected, and WCD can present as lymphadenopathy and polyserositis as well. The majority of patients had no specific or consistent abnormalities revealed by laboratory investigations. They often showed leukocytosis and leukopenia, increased ESR and CRP levels, and complement and immunoglobulin abnormalities. Increased transaminase enzyme levels and abnormal lipid metabolism were found in patients with liver involvement. Among the patients we observed, 2 had associated fatty liver, 1 had interstitial pneumonia, and 1 had liver enlargement without specific etiology, which was similar to previous reports.

Because WCD lacks disease specificity, many patients visit different departments to establish a diagnosis. In our study, WCD patients were mainly treated in the department of rheumatology (5 cases), followed by the departments of general surgery (3 cases), infectious diseases (3 cases), and dermatology (3 cases). WCD should be differentiated from the following diseases: (1) erythema nodosum. This disease can also present with subcutaneous nodules with symmetrical distribution; however, the nodules are mostly confined to the extensor surface of the lower legs and do not collapse. After 3–4 weeks, the nodules subside spontaneously, and no atrophic scar remains. The symptoms are mild, and no visceral damage occurs. Patients with secondary systemic diseases (such as Behçet's disease) have symptoms of the related disease.

Table 2 The clinical characteristics of thirteen patients with Weber-Christian panniculitis

Patient	Sex/age	Potential causes	Clinical features	Laboratory findings	Concomitant disease	Therapy
1	M/53 years	Unclear	Subcutaneous nodule, fever, myalgias, abdominal pain	Elevated CRP and ESR	Pulmonary nodules	Prednisone
2	M/35 years	Unclear	Subcutaneous nodules, myalgias, arthralgias	Elevated WBC, CRP, ESR, and ANA	SLE	Prednisone, NSAIDs
3	F/61 years	Unclear	Subcutaneous nodules, myalgias, arthritis	Elevated ESR and Ig, decreased complement	Biliary tract disease	Prednisone
4	F/67 years	Unclear	Subcutaneous nodule, abdominal pain	No abnormal	Saphenous vein varicosity	Surgical excision
5	F/58 years	Unclear	Subcutaneous nodule, fever, arthralgias	Elevated WBC, CRP, ESR, Scr, ANA, and Ig	SLE	Prednisone, NSAIDs
6	F/22 years	Unclear	Subcutaneous nodule, fever, hepatosplenomegaly	Elevated ESR, decreased complement	Pulmonary nodules, interstitial pneumonia	Prednisone
7	F/29 years	Lipodissolve treatment	Subcutaneous nodule, fever, arthritis, myalgias	Leukopenia, elevated ALT	Fatty liver	Prednisone
8	F/61 years	Unclear	Subcutaneous nodule, fever, arthritis	Elevated CRP	Biliary tract disease	Prednisone, NSAIDs
9	F/75 years	Unclear	Subcutaneous nodule, abdominal pain	Elevated WBC, ALT, and ANA	SLE	Prednisone Cyclophosphamide
10	F/52 years	Unclear	Subcutaneous nodule, arthralgias	No abnormal	Fatty liver	Surgical excision
11	F/53 years	Unclear	Subcutaneous nodule, fever, myalgias, arthralgias	Leukopenia, elevated Ig	Hypertension	Prednisone
12	F/48 years	Unclear	Subcutaneous nodule, fever, arthritis	Elevated WBC and CRP, decreased complement	Chronic hepatitis B	Prednisone, azathioprine
13	F/38 years	Unclear	Subcutaneous nodule, fever	Elevated WBC, CRP, ESR, and ALT	Hypertension	Prednisone, NSAIDs

WBC, white blood cell; *CRP*, C-reactive protein; *ESR*, erythrocyte sedimentation rate; *ALT*, alanine aminotransferase; *Scr*, serum creatinine; *ANA*, antinuclear antibodies; *Ig*, immunoglobulin; *SLE*, systemic lupus erythematosus

Pathologically, erythema nodosum manifests as septal panniculitis accompanied with vasculitis. (2) Erythema induratum. The nodules are dark red, located in the middle and lower part of the flexor side of the lower leg, and perforate after ulceration. Tuberculous granulomas are observed on histopathology. (3) Localized scleroderma. This disease manifests as localized skin swelling initially, and then, the areas become hard and atrophic. The condition often occurs on the scalp, forehead, waist, abdomen, and limbs. Generally, this disease has no systemic symptoms; however, patients may complain of mild pruritus or tingling, arising gradually. Patients do not have obvious systemic symptoms, and limited scleroderma does not generally affect the viscera. (4) Rheumatoid arthritis (RA). Subcutaneous nodules and joint pain can also occur in RA patients, but the subcutaneous nodules in RA patients are hard, round/oval, and painless. They are usually located in susceptible areas, such as the elbow, Achilles tendon, scalp, sciatic tubercle, lower back, wrist, or finger joints. WCD usually does not cause joint destruction; thus, distinguishing it from RA becomes easier with disease progression. (5) Other diseases. WCD should also be distinguished from other diseases that can cause subcutaneous fat necrosis, such as lipogranulomatosis subcutanea, subcutaneous panniculitis-like T cell lymphoma, pancreatic panniculitis, pancreatitis, pancreatic carcinoma, leprosy, trauma, and foreign bodies. The diagnosis of WCD mainly depends on the biopsy of the lesion. In addition, this disease can coincide with other (immune) diseases, and it may appear subsequent to the occurrence of a primary disease or as a primary disease in which another disease that follows WCD is secondary. WCD should be carefully identified.

There is no specific treatment for this disease. Glucocorticoid therapy is currently the first-line treatment, as it can inhibit the inflammatory reaction and immune cell dysfunction. According to the analysis of the curative effect of our 13 patients, the routine dose of glucocorticoid therapy (0.5–1 mg/day of prednisone or an equivalent dose of dexamethasone) had a curative effect for most patients; however, during the treatment process, it was necessary to pay close attention to medication reduction and adverse reactions. For patients with recurrent disease and those who did not respond to initial glucocorticoid therapy, glucocorticoid combined with an immunosuppressant (such as methotrexate (MTX), CTX, or azathioprine) was a good choice. In some cases, cyclosporine A (CsA) has been used for the treatment of WCD [12, 13]. Unfortunately, the long-term effect and withdrawal reaction need to be further observed. In addition, CsA is expensive, which limits its availability.

Table 3 Laboratory features of thirteen patients with Weber-Christian panniculitis (part 1)

Patient	WBC (3.5–9.5 × 10^9/L)	CRP (0–10 mg/L)	ESR (0–21 mm/h)	ALT (9–50 U/L)	Scr (44–133 μmol/L)	ANA (1:32)
1	5.2	35	28	45	79	(−)
2	11.3	65.4	61	31	102	1:320
3	9.1	9.8	27	22	97	(−)
4	6.2	5.2	14	21	82	(−)
5	13.6	101.3	85	47	159	1:160
6	4.5	6.1	33	19	77	(−)
7	3.1	7.2	11	82	117	(−)
8	7.8	29.3	14	38	69	(−)
9	3.3	5.3	17	78	62	1:320
10	4.8	4.8	15	30	90	(−)
11	10.5	10	8	19	70	(−)
12	12.6	45.1	20	42	83	(−)
13	12.8	70.4	59	64	110	(−)
Means ± SD	8.1 ± 3.8	30.4 ± 31.6	30.2 ± 23.6	41.4 ± 21.6	92.1 ± 25.9	–

WBC, white blood cell; *CRP*, C-reactive protein; *ESR*, erythrocyte sedimentation rate; *ALT*, alanine aminotransferase; *Scr*, serum creatinine; *ANA*, antinuclear antibodies

The prognosis of our WCD patients varied considerably. Only a few patients were healed or cured within several months. Many patients gradually improved and achieved long-term remission after glucocorticoid treatment, with or without an immunosuppressant. Some patients' disease courses were quite prolonged, with recurrent inflammation of subcutaneous fat. In our study, 11 patients were followed up for an extended period of time. Most patients exhibited long-term remission (8 cases) during the follow-up process, but some still required medication to control recurrent symptoms. In addition, for the patients with concurrent autoimmune diseases, especially RA and SLE, good control needs to be established with aggressive treatment of the primary disease. Patients who have symptoms and signs of WCD that cannot be differentiated from those of other autoimmune diseases should be followed up until the best treatment for a good prognosis can be offered while minimizing adverse reactions.

Because the clinical characteristics of WCD are complex, it is easy to misdiagnose. In our study, the misdiagnosis rate was 61.5%, and tumors, bacterial infections, and rheumatic diseases were the most common misdiagnoses. We concluded that the main reasons for these misdiagnoses are as follows: (1) Clinicians have an insufficient level of awareness of this disease. Patients might present at many different clinical

Table 4 Laboratory features of thirteen patients with Weber-Christian panniculitis (part 2)

Patient	Complement 3 (0.79–1.52 g/L)	Complement 4 (0.16–0.38 g/L)	IgA (0.82–4.53 g/L)	IgG (7.51–15.60 g/L)	IgM (0.46–3.04 g/L)
1	0.93	0.26	3.46	12.88	2.73
2	0.82	0.19	4.22	11.73	1.21
3	0.67	0.15	5.08	13.2	2.98
4	1.03	0.21	3.39	9.69	2.55
5	1.09	0.27	4.65	13.88	5.09
6	0.59	0.11	3.71	9.02	2.58
7	1.20	0.23	3.88	10.56	2.09
8	1.19	0.34	4.06	8.89	1.18
9	1.44	0.25	3.09	14.82	0.98
10	0.87	0.29	1.44	8.93	3.41
11	1.31	0.30	4.82	16.12	3.01
12	0.48	0.12	4.01	13.82	0.92
13	1.39	0.28	0.94	10.43	1.04
Means ± SD	1.0 ± 0.3	0.23 ± 0.07	3.60 ± 1.21	11.84 ± 2.44	2.29 ± 1.23

Ig, immunoglobulin

departments because of the diverse manifestations of WCD. At the same time, physicians may focus on prominent symptoms and ignore other less obvious symptoms. In our study, 3 patients were misdiagnosed with erysipelas, lymphadenitis, and sebaceous cysts, because of the recurrent tender localized nodules. Repeated anti-inflammatory treatments failed. The final diagnoses were confirmed through skin biopsies. Some patients manifested subcutaneous nodules, but doctors did not find them or disregarded them. In this cohort, 3 patients had obvious joint swelling and pain. However, the nearby subcutaneous nodules and irregular low-grade fever were not detected, which led to the misdiagnosis of rheumatic fever or rheumatoid arthritis. Continuous antirheumatic treatment was ineffective, and the number of subcutaneous nodules increased; WCD was finally confirmed by skin biopsies. (2) WCD often coexists with other diseases; the study by White and Winkelmann [8] revealed that 40% (12/30) of patients with WCD had another diseases at the same time, such as phlebitis or phlebitis after another syndrome, trauma, infections treated with penicillin, lymphoma, leukemia, connective tissue diseases like SLE, and tuberous polyarteritis. (3) There is an excessive reliance on examination results. In our study, the presentation of WCD in 1 patient was only a local painless nodule, and he was diagnosed with fibromyoma after cytological biopsy. However, the nodules continued to develop, and he finally underwent a second biopsy, which led to the accurate diagnosis of WCD. Therefore, clinicians need to deepen their understanding of this disease. For the patients with recurring fever of unknown origin and nodules or masses, histopathological examination might be a good choice to confirm the diagnosis as soon as possible.

Conclusions

In summary, we reported and analyzed the clinical characteristics of 13 patients with WCD. Prominent clinical features included female predominance, subcutaneous nodules, fevers, arthralgias/arthritis, and myalgias. The laboratory examination results were nonspecific, leading to a high misdiagnosis rate; the final diagnosis of WCD depended on pathological biopsies. We suggested that histopathological examinations should be performed as soon as possible if patients presented with unexplained recurrent fever and nodules or masses.

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Author contributions Wei Zheng had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Wei Zheng, Wen yuan Song, and Hongying Pan were involved in the design of the study; authors Wei Zheng and Wen yuan Song jointly completed the writing, and Qingqing Wu, Qiaoqiao Yin, and Chaolan Pan were substantially involved in revising the article.

Compliance with ethical standards

Because this study was based on the retrospective analysis of existing administrative and clinical data, the need to obtain informed patient consent was waived by the Ethics Review Committee of Zhejiang Provincial People's Hospital.

Disclosures None.

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