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Idiopathic Lobular Panniculitis: Diagnosis, Symptoms and Current Treatment Progress

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Abstract. Panniculitis refers to the inflammation of the subcutaneous fat tissue. In this category of panniculitis, there is one type called idiopathic lobular panniculitis, which means the cause is unknown. The main symptoms of idiopathic lobular panniculitis include the presence of painful red nodules or plaques together with fever, pain of lower limbs and changes of skin pigmentation. Different people have different clinical manifestation characteristics. The age, sex, and background of the people affected are all different. Different subtypes exist. The diagnosis depends on the integration of all aspects. ILP essentially belongs to an exclusionary diagnosis, and it is necessary to rule out secondary factors such as infection, drugs, pancreatic diseases, α1-antitrypsin deficiency, autoimmune diseases, trauma and malignant tumors. The symptom mechanism is mainly related to inflammatory cell infiltration in fat lobules, fat necrosis and post-inflammatory pigmentation, and it can involve systems such as the liver, blood vessels, joints, intestines and lungs. In terms of treatment, glucocorticoids such as prednisolone are commonly used drugs. Immunosuppressants such as tacrolimus, thalidomide and biological agents such as infliximab and adalimumab can be used for refractory cases, but the latter two are still exploratory treatments.

Keywords: Idiopathic Lobular Panniculitis, diagnosis, symptoms, current treatment

1. Introduction

Idiopathic lobular panniculitis, also known as Weber-Christian disease, is a rare inflammatory disease and subtype of panniculitis that affects the subcutaneous fat beneath the skin. It commonly causes painful, red, and firm nodules or plaques under the skin, often followed by other symptoms such as fever, lower-extremity pain and skin discoloration. However, clinical presentation can vary significantly between patients. Panniculitis is a general term used to describe a group of conditions involving inflammation of this fatty tissue. Although panniculitis can have many different causes, idiopathic lobular panniculitis' exact cause remains unknown. This paper aims to focus on the diagnosis of Idiopathic lobular panniculitis (ILP) and systematically review the current progress in research on this disease.

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2. Diagnosis

The ILP diagnosis is dependent on a proper evaluation of the condition using the clinical features, lab tests, and pathological analysis. Among them, deep skin biopsy and histopathological examination are crucial for diagnosis. ILP usually presents as recurrent subcutaneous nodules, plaques or infiltrative skin lesions, accompanied by pain, fever and fatigue. The lesions often affect the limbs, trunk and buttocks, and some patients, the disease may also involve internal organs or mesenteric adipose tissue. Laboratory findings may include elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) results, and some patients may have anemia, changes in white blood cells or abnormal liver enzymes, but these indicators lack specificity. Histopathology is the core of diagnosis, typically presenting as panniculitis with fat lobules as the main feature, visible fat cell degeneration, necrosis and fat phagocytosis, accompanied by varying degrees of infiltration by lymphocytes, tissue cells, neutrophils, etc., and fibrosis may occur in the later stage of the disease; some cases may also be accompanied by vasculitis changes. The term "Weber-Christian disease" previously used to describe febrile, recurrent lobular panniculitis is often considered to lack specificity by modern research. Many cases previously diagnosed as this disease belong to nodular erythema, pancreatitis-related panniculitis, α1-antitrypsin deficiency-related panniculitis, artificial panniculitis, lymphoma or cell phagocytic panniculitis, etc. Therefore, ILP belongs to an exclusionary diagnosis: after histological confirmation of lobular panniculitis, further exclusion of secondary causes such as infection, drugs, pancreatic diseases, α1-antitrypsin deficiency, autoimmune diseases, trauma and malignant tumors should be carried out, and the diagnosis should ultimately be determined based on the clinical course and auxiliary examinations. Studies of 62 ILP patients have shown that morphological examination can clearly identify lobular panniculitis and exclude tumorous lesions; while re-evaluation of 30 patients previously diagnosed as Weber-Christian panniculitis revealed that most cases could eventually be classified into other definite diseases, suggesting that standardized etiological and histopathological diagnosis is very important [1, 2].

A 10-year retrospective analysis conducted by Ramathibodi Hospital in Thailand included 169 patients diagnosed with erythema nodosum (EN) through biopsy, aiming to explore the relationship between their clinical and pathological characteristics and the etiology as well as recurrence. The results showed that the average age at diagnosis was 40.6 years, with 85.2% being female. Idiopathic EN accounted for 62.7%, and secondary EN accounted for 37.3%. Among them, tuberculosis (23.8%) and drugs (23.8%, mainly oral contraceptives) were the most common causes. Univariate analysis revealed that upper limb skin lesions ($p = 0.018$), fever ($p = 0.003$), and clinical lymph node enlargement ($p < 0.001$) were associated with secondary EN, while focal peripheral lobular panniculitis with eosinophilic infiltration suggested idiopathic EN ($p = 0.030$). However, multivariate analysis did not identify independent predictors. The recurrence rate was 46.6%, and there was no significant difference between the idiopathic and secondary groups. No clinical or pathological factors related to recurrence were found. From a histopathological perspective, acute EN accounted for 30.8%, and chronic EN accounted for 69.2%, but it was not related to the etiological classification. The study concluded that although most clinical features cannot reliably distinguish the causes of EN, the presence of eosinophils in focal lobular panniculitis is helpful in suggesting idiopathic EN. Future multi-regional prospective studies are needed to verify [3].

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3. Symptoms and effects on the human body

3.1. Painful nodules

Development of painful nodules is a significant clinical manifestation of Idiopathic lobular panniculitis. Nodules typically form on lower legs, although their development is possible on other body sites. Fat below the skin is arranged in small compartments known as lobules which consist of adipocytes, fat cells. Lobules are divided from each other by septa which consist of connective tissue as well as vessels – blood, lymphatic vessels and nerves. Panniculitis involves inflammation of the subcutaneous fat, fat lobules in the case of lobular panniculitis. Infiltration of the tissue with white blood cells causes the release of inflammatory mediators which destroy adipocytes. Damage results in fat necrosis which refers to cell death due to lack of blood. Inflammation and fat necrosis may result in thickening and hardening of the affected area, leading to formation of subcutaneous nodule [4].

3.2. Skin discoloration

Skin discoloration is yet another possible symptom of panniculitis. Post inflammatory hyper-pigmentation is one of the temporary or even permanent darkening of the skin that happens following an inflammation or trauma. As mentioned earlier, panniculitis affects mainly the subcutaneous fat that is under the epidermis and dermis, thus the inflammation may affect the skin above it. This way, the inflammation will trigger melanocytes, the cells which produce melanin, to create more melanin. The excess melanin will later be transported to the surrounding keratinocytes, which are the main cells of the epidermis, and this will lead to the darkening of the particular area. Moreover, in case of the trauma of the basal layer, the melanin pigment is produced and then it is captured by the macrophages in the papillary dermis. Overall, panniculitis may influence several organ systems. Some of the possible complications may include anemia, hepatomegaly, vasculitis, polymyalgia and polyarthralgia, bowel perforation and pleural effusion [5].

4. Treatment

4.1. Glucocorticoids

Prednisolone is a type of glucocorticoid corticosteroid medication that mimics the effects of the cortisol, which is a hormone naturally produced by the body. It works by inhibiting and regulating immune response, thus, leading to reduced inflammation and redness among other symptoms. Thanks to these properties of reducing inflammation, prednisolone can be used to manage inflammatory diseases such as panniculitis [6].

Mesenteric panniculitis, which is a rare form of panniculitis associated with the fatty tissue in the abdomen, is described in a case report of a 13-year-old boy treated with prednisolone. Among the symptoms of the patient, upper abdominal pain, vomiting, and diarrhea can be mentioned. Laboratory data indicated signs of inflammation, including highly increased WBC of 15,100/μL. Moreover, the C-reactive protein level was 5.45 mg/dL. C-reactive protein is the name of a protein produced in the liver during inflammation. Prednisolone therapy at the dose of 30 mg/day was prescribed on the sixth day of the hospitalization. Abdominal pain and inflammatory indices have been gradually decreasing in four days. There has been a decrease in the WBC level from 15,100/μL to 7,320/μL. Besides, CRP decreased from 5.45 to 1.65 mg/dL. With time, the prednisolone dose

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was slowly decreased to 10 mg/day in one month and then maintained at the same dose for seven more months. Follow-up examinations after eight months demonstrated significant improvement and no problems during the next two years [7].

On the downside, Prednisolone being an immunosuppressive drug, it can lower the body's ability to resist infections. Thus, people who use prednisolone may be more likely to suffer from infections, especially those taking larger dosages and for long periods of time.

4.2. Immunosuppressive medications

4.2.1. Tacrolimus

The primary use of tacrolimus capsules is that of an immunosuppressive agent in order to prevent rejection of a transplanted organ by the body. The mechanism of action of tacrolimus is through inhibition of the function of T cells, which are major constituents of the immune system. It binds to a protein FKBP-12 and forms a complex that will inhibit the function of enzyme calcineurin. Consequently, there will be decreased production of interleukin-2 (IL-2) which is vital for activation of T cells [8].

The above mechanism also explains the application of the drug in the management of some types of panniculitis. The signs and symptoms of panniculitis include painful nodules, and inhibition of T-cell actions might reduce the level of inflammation in the inflamed tissues. A clinical trial was carried out to determine the effectiveness of tacrolimus in the management of delayed adverse reactions to medical grade silicone injections. Patients who had been enrolled in this study were selected from those individuals that could not respond to at least six drugs. The time span of development of signs and symptoms was found to be 65 months, which commonly develop in the form of nodules, plaques, and panniculitis. Tacrolimus therapy for about 18 months gave mild symptoms with rapid responses in five patients and complete relief in two patients without any side effects.

4.2.2. Thalidomide

Thalidomide is another example of anti-inflammatory drugs. Thalidomide is an immunomodulatory drug with anti-inflammatory properties, even though its mechanism of action has not been fully discovered yet. One of the major properties of Thalidomide is suppressing the formation of tumor necrosis factor alpha (TNF-α), a substance which is crucial for inflammatory response. In addition, Thalidomide is capable of inhibiting NF-κB, a protein that regulates the genes involved in the immune response. With its effect on such pathways, thalidomide may help in decreasing the amount of inflammation mediators, like TNF-α, IL-6, and IL-12. It may even have an influence on caspase-1.

The anti-inflammatory properties of this drug might be able to make thalidomide applicable in treating certain kinds of panniculitis. With its ability to suppress inflammatory reactions, thalidomide might be able to lower down the inflammation as well as the symptoms of the disease. As an illustration, a pilot study on four out of five patients who have mesenteric panniculitis proved to react positively to thalidomide therapy. But since this was done on few patients, further studies should be done to confirm the efficacy of thalidomide on panniculitis [9].

Furthermore, Barthel et al. described a case of idiopathic nodular panniculitis (Weber-Christian disease) that did not respond to several medications. The use of therapies including glucocorticoids, methotrexate, hydroxychloroquine, azathioprine, cyclosporine, colchicine, and doxycycline failed to

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bring any positive effect; however, with administration of thalidomide 100 mg daily, the patient entered into remission and remained in it for three years [10].

4.3. Biological agents

While assuming ILP might be an Immune-Mediated disease, biologics have entered a field of refractory cases, such as TNF-α treatments. In a case study, a 42-year-old woman with Weber-Christian Disease experienced symptoms with periorbital, mesenteric, and ileocecal involvement, mimicking Crohn's disease. This study was designed as a case report. The results showed that the patient underwent a right hemicolectomy and ileal resection because of fever, abdominal pain, and an ileocecal stricture. She experienced postoperative recurrence, and histological examination confirmed lobular panniculitis with granulomas. After thalidomide was discontinued because of neurotoxicity, she was switched to infliximab (5 mg/kg at weeks 0, 2, and 6, followed by every 8 weeks), which successfully induced and maintained clinical and endoscopic remission. No recurrence was observed during the two-year follow-up period. The authors concluded that infliximab may be used for intestinal involvement in refractory WCD. In another case, the patient discontinued infliximab because of a hypersensitivity reaction and was subsequently switched to adalimumab, which provided long-term disease control. However, anti-TNF-α therapy is currently considered an exploratory treatment for refractory ILP rather than a standard first-line treatment.

5. Conclusion

Idiopathic Lobular Panniculitis is a kind of inflammatory disease that impacts the subcutaneous fat tissue. Because of overlapping symptoms with other diseases, it is still hard to diagnose ILP. The deep biopsy of the skin is necessary for the diagnosis. Current therapy is mostly aimed at the alleviation of the symptoms but not the cure of the disease. Therefore, there is an urgent need for further research for better diagnosis and new effective and safe treatment options for the condition. In the future, multicenter prospective studies should be carried out for deep investigation of the disease pathogenesis and immune mechanism as well as finding diagnostic and prognostic factors. As for the treatment, it is crucial to reinforce clinical research in the field of biological treatment, develop stratification protocol and create an international consensus for providing the refractory patients with better prospects. Moreover, long-term follow-up studies are necessary for estimation of the risk of recurrence and systemic affection of ILP.

References

[1] Radenska-Lopovok, S. G., Egorova, O. N., Belov, B. B., Glukhova, S. I., Musatov, I. D., & Nagornaya, D. S. (2019). Idiopaticheskiĭ lobuliarnyĭ pannikulit: Kliniko-morfologicheskie korreliatsii [Idiopathic lobular panniculitis: Clinical and morphological correlations]. *Arkhiv Patologii, 81*(3), 37–44.

[2] White, J. W., Jr., & Winkelmann, R. K. (1998). Weber-Christian panniculitis: A review of 30 cases with this diagnosis. *Journal of the American Academy of Dermatology, 39*(1), 56–62.

[3] Limtong, P., Suchonwanit, P., Chanprapaph, K., & Rutnin, S. (2021). Clinicopathological characteristics related to etiologies of erythema nodosum: A 10-year retrospective study. *Clinical, Cosmetic and Investigational Dermatology, 14*, 1819–1829. https://doi.org/10.2147/CCID.S343351

[4] Panniculitis. (n.d.). *Cleveland Clinic*. https://my.clevelandclinic.org/health/diseases/panniculitis

[5] Ngan, V. (2015). Postinflammatory hyperpigmentation. *DermNet NZ*. https://dermnetnz.org/topics/postinflammatory-hyperpigmentation

[6] Puckett, Y., Gabbar, A., & Bokhari, A. A. (2025). Prednisone. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK534809/

DOI: 10.54254/2753-8818/2026.HZ38856

[7] Mesenteric panniculitis in a thirteen-year-old Korean boy treated with prednisolone: A case report. (2016). *Pediatric Gastroenterology, Hepatology & Nutrition, 19*(2), 143–146. https://doi.org/10.5223/pghn.2016.19.2.143

[8] Thomson, A. W., Bonham, C. A., & Zeevi, A. (1995). Mode of action of tacrolimus (FK506): Molecular and cellular mechanisms. *Therapeutic Drug Monitoring, 17*(6), 584–591. https://doi.org/10.1097/00007691-199512000-00007

[9] Majumder, S., Sreedhara, S. R. C., Banerjee, S., & Chatterjee, S. (2012). TNF-α signaling beholds thalidomide saga: A review of mechanistic role of TNF-α signaling under thalidomide. *Current Topics in Medicinal Chemistry, 12*(13), 1456–1467. https://doi.org/10.2174/156802612801784443

[10] Barthel, H. R., Charrier, U., Kramer, M., & Loch, C. (2002). Successful treatment of idiopathic febrile panniculitis (Weber-Christian disease) with thalidomide in a patient having failed multiple other medical therapies. *Journal of Clinical Rheumatology, 8*(5), 256–259.