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Case Report

Leukocytoclastic Vasculitis Associated with Rheumatoid Arthritis and Sjögren's Syndrome

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Abstract: Leukocytoclastic vasculitis (LCV) is a type of small vessel vasculitis that primarily affects superficial postcapillary venules. Histopathological examination reveals predominantly neutrophilic inflammation along with leukocytoclasia. The exact incidence of LCV is not fully understood. Clinically, LCV typically manifests as palpable purpura that can appear anywhere on the body, although it frequently affects the lower limbs. It can develop at any age, but it is more frequent in adults. LCV may be triggered by various factors, including infections, autoimmune disorders, neoplasms, or medications; in some cases, it may be idiopathic, accounting for up to half of all instances. Typically, the condition is mild to moderate, some patients may be asymptomatic, and can improve with supportive care measures. Diagnosis is made through clinical assessment, laboratory tests, and skin biopsy. Treatment should be dictated according to the etiology and most of the cases is symptomatic. We present the case of a 55-year-old woman with autoimmune conditions who presented with abdominal pain and disseminated palpable purpura.

Keywords: Dermatology, Rheumatology, vasculitis, leukocytoclasia, hypersensivity.

INTRODUCTION

LCV is also known as hypersensitivity angiitis; it refers to a histopathologic description of small vessel vasculitis, usually involving arterioles, capillaries, and post-capillary venules in which there is an inflammatory infiltrate composed of neutrophils and fibrinoid necrosis and disintegration of nuclei into fragments, a term known as leukocytoclasia [1-3]. These findings are not exclusive to LCV and may be found in other types of vasculitis that affect the skin and other internal organs; however, the term LCV refers to small-vessel vasculitis in the skin [1]. Incidence remains unclear due to variability of its definition; according to literature, it ranges from 15 to 38 cases per million per year; the prevalence varies from 2.7 to 29.7 per year per million. LCV affects both sexes equally; it may occur at any age. Some studies noted a predilection for the male sex and older age. This condition may be idiopathic in up to 50% of the cases; as mentioned, it also can be related to infections, malignancy, drugs, or autoimmune conditions. Of the identifiable causes, the most common trigger was infections [4]. Drug-induced vasculitis may be limited to the skin or be systemic; if systemic, arthritis, gastrointestinal or kidney involvement, and fever are reported in the literature; antibiotics (primarily beta-lactams) and non-steroidal anti-inflammatory drugs are the most commonly involved. Among infectious causes, upper respiratory tract infections were the most commonly implicated. LCV often develops seven to ten days after the exposure to a drug or infectious trigger. The most common clinical presentation of LCV is palpable purpura; lesions tend to merge with confluent aspects, and usually lesions resolve over two to three weeks, fading away and sometimes leaving post-inflammatory hyperpigmentation. Some patients may be asymptomatic, while some patients may have burning sensation, itching, or pain in the area of the injuries [1-4]. Clinical history and physical exam may suggest LCV, a workup should be performed. If a clear trigger (e.g., infection, drugs) is present, basic studies may be enough to conclude the diagnosis. In other cases, investigations should include infectious serologies, immunoglobulins, and antibody determination; skin biopsy is diagnostic. Treatment of LCV is guided by the underlying etiology and the extent of systemic involvement. If it is self-limited, treatment focuses on

symptomatic relief and trigger removal; if it is refractory, recurrent, or severe, systemic corticosteroids are given, and colchicine, dapsone, or immunosuppressants should be considered [1,6,8].

CASE PRESENTATION

A 55-year-old female with a past medical history of rheumatoid arthritis (AR), hypothyroidism, and Sjögren's syndrome presented with abdominal pain in the epigastrium, nausea, and vomiting, self-medicated with ciprofloxacin and antispasmodics with no improvement, and later presented with a disseminated dermatosis affecting the lower extremities, and in smaller quantities in the upper extremities, characterized by multiple palpable purpuric macules of variable sizes (the bigger ones of 4 cm and the smaller ones of 3 mm), well-defined edges; some of them tended to merge and develop tense blisters with hemorrhagic content; the skin surrounding the lesions was normal, as shown in Figures1-3. Antinuclear antibody (ANA), antineutrophil cytoplasmic antibody - cytoplasmic (ANCA-C) and antineutrophil cytoplasmic antibodies - perinuclear (ANCA-P), C3 complement and C4 complement determinations were normal, the viral panel was negative; endoscopy was performed, ruling out data of vasculitis in the gastrointestinal tract. A skin biopsy of one of the active lesions was taken that reported perivascular neutrophilic infiltrate in the dermis. A diagnosis of cutaneous small-vessel vasculitis was made. The patient started with oral prednisone 30 mg daily, tapered over 20 days, with the lesions evolving towards improvement and resolution of skin lesions.

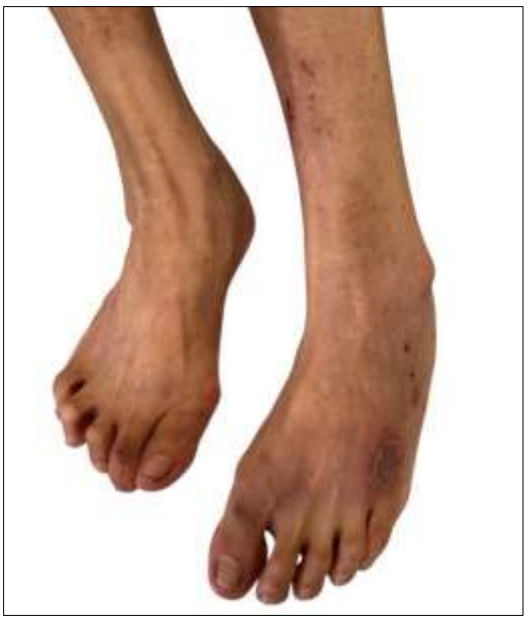

Figure 1: Multiple violaceous macules and a flaccid blister on the lateral metatarsal region.

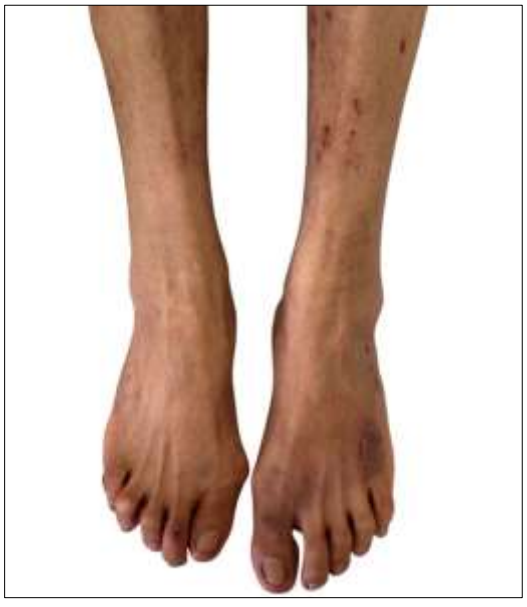

Figure 2: Disseminated violaceous macules.

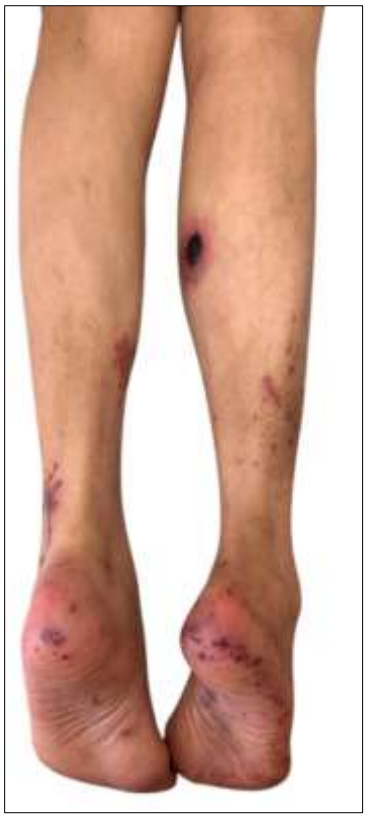

Figure 3: Necrotic purpuric lesion on the inner surface of the gastrocnemius, multiple purpuric lesions, active lesion on the heel.

DISCUSSION

LCV is an infrequent condition of the vessels, primarily of dermal capillaries and venules; it is often associated with other conditions, such as infections (firstly), but it may be associated with other autoimmune conditions [5], such as the case of our patient, who had prior diagnoses of AR, hypothyroidism, and Sjögren's syndrome.

LCV is characterized by a key pathological feature of small-vessel vasculitis-namely, the breakdown of neutrophil nuclei within the inflammatory infiltrate affecting the walls of arterioles, capillaries, and post-capillary venules [6]. This condition may occur without a known cause or be linked to other factors as mentioned previously. The principal presentation of LCV is palpable purpura that usually evolves over a few hours; it involves primarily the lower legs, but it may affect any other area of the body [2]. Commonly the lesions tend to be confluent and usually resolve over two to three weeks and slowly fade away, usually leaving behind post-inflammatory hyperpigmentation [2].

LCV is usually limited to the skin, and manifestations outside the skin are very rare, it is estimated that they occur in less than 30% of the cases [7]. This condition may be secondary to vasculitis, immune complex-mediated small vessel vasculitis; it can be seen in patients with AR, systemic lupus erythematosus, Sjögren's syndrome, Henoch-Schönlein purpura, cryoglobulinemic vasculitis, and others. Separate causes may include infections, drugs, and paraneoplasic vasculitis [7]. In the case of our patient, it was perhaps triggered by a previous history of AR, hypothyroidism, and Sjögren's syndrome; however, she also had consumption of fluoroquinolones for a short period of time.

Diagnosis is made from a combination of clinical findings, serologic, pathologic, and imaging studies; sometimes physical examination and history of the patients are sufficient to establish diagnosis [2,8]. Studies may include infectious serologies, serum protein electrophoresis, immunoglobulins, antinuclear antibody panel and rheumatoid factor, serum C3 and C4 complement levels, ANA, ANCA-C, ANCA-P and cryoglobulins. Skin biopsy should be performed to confirm the diagnosis of LCV [2].

This condition presents acutely, and it is self-limited in about 90% of the cases [3]. Once the diagnosis is made, the treatment is guided primarily by the underlying cause and the severity of the extent of the disease; if it is possible, identifying the underlying trigger treatments should be focused on the resolution of the same; if only skin is involved, the management focuses on relieving symptoms such as through rest, leg elevation, and wearing compression stockings. In

cases where the vasculitis is limited to the skin and it is severe, persistent, or recurrent, systemic corticosteroids may be required. Colchicine is another option for the management [2,3].

CONCLUSIONS

LCV is a rare inflammatory condition primarily affecting small dermal vessels. It can present without a clear underlying etiology, is frequently associated with infections and autoimmune diseases as well as certain medications; most often it is manifested as palpable purpura, especially on the lower extremities. Typically resolves within weeks, sometimes leaving residual post-inflammatory hyperpigmentation. Diagnosis involves a combination of clinical evaluation, laboratory testing, and skin biopsy. Management ranges from supportive care to systemic therapies such as steroids or colchicine when needed. The importance of identifying the etiology or triggering factor is important for the comprehensive management of the patient.

Conflict of Interest: The authors declare that there are no conflicts of interest at the time of publication of this article.

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