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Leukocytoclastic Vasculitis Revealing Crohn's Disease: An Unusual Case Report

S. Boumadiane[1]*, M. Aouroud[1], A. N'Khaili[1], H. Aouroud[2], O. Nacir[1], F. Lairani[1], A. Ait Errami[1], S. Oubaha[2], Z. Samlani[1], K. Krati[1]

[1]Department of Gastroenterology, Mohammed VI University Hospital, Marrakech
[2]Department of Physiology, Cadi Ayyad University, Mohammed VI University Hospital, Marrakech

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***Corresponding author:** S. Boumadiane
Department of Gastroenterology, Mohammed VI University Hospital, Marrakech

Abstract | **Case Report**

Crohn's disease (CD) is a chronic inflammatory bowel disease (IBD) that can affect the entire gastrointestinal tract. Leukocytoclastic vasculitis (LCV) represents one of the rare extra-digestive cutaneous manifestations encountered in this condition, whose therapeutic management remains nonstandardized and generally responds favorably to treatment of the digestive disease flare. We report the case of a 19-year-old female patient diagnosed for Crohn's disease, in whom the diagnosis of leukocytoclastic vasculitis was established via skin biopsy during the initial presentation of Crohn's disease.

Keywords: Crohn's disease - Inflammatory bowel disease - Leukocytoclastic vasculitis - Cutaneous manifestations - Skin biopsy.

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INTRODUCTION

Crohn's disease (CD) is a chronic inflammatory bowel disease, evolving in flares interspersed with remission phases, characterized by transmural involvement and potentially associated with anoperineal and extra-digestive manifestations. Cutaneous manifestations are among the most frequent extra-digestive manifestations in inflammatory bowel diseases (IBD) [1]. Commonly encountered lesions include aphthous ulcers, erythema nodosum, neutrophilic dermatoses, pyoderma gangrenosum, and granulomatous lesions specific to Crohn's disease [2,3].

Leukocytoclastic vasculitis (LCV) constitutes one of the rare cutaneous manifestations of IBD, encountered primarily in the context of Crohn's disease [4,5]. The purpose of this work is to present an unusual case of a young patient followed for Crohn's disease, in whom clinical examination revealed the presence of leukocytoclastic vasculitis (LCV), along with a review of the literature.

CASE REPORT

We report the case of Ms. R.Z., a 19-year-old woman with no notable past medical history, no prior appendectomy, and no active or passive smoking history. She was hospitalized in our department of gastroenterology for management of atypical abdominal pain, predominantly epigastric, evolving over 4 months prior to admission, refractory to analgesic treatments, with intractable postprandial food vomiting and no other associated digestive or extra-digestive manifestations. The symptoms evolved in flares interspersed with remissions, in the context of general deterioration including unquantified weight loss, anorexia, and asthenia.

Physical examination revealed a hemodynamically and respiratorily stable patient with a BMI of 27.6 kg/m². Abdominal examination showed diffuse abdominal tenderness, maximal at the epigastric region, without guarding or rigidity. Dermatological examination noted the presence of a lenticular petechial purpura over both lower limbs, non-blanching on diascopy, with no other detectable lesions, and peripheral pulses were well-palpated.

Laboratory findings revealed iron-deficiency anemia with hemoglobin at 11.6 g/dL, leukocytes at 10,200/µL with neutrophils at 5,540/µL, and an inflammatory syndrome with C-reactive protein (CRP) at 43.4 mg/L and erythrocyte sedimentation rate (ESR)

at 20 mm at the first hour. Platelet count was 354,000/mm³ and prothrombin time (PT) was normal at 86%. Electrolytes, liver function tests, and renal function were all within normal limits. Stool parasitological examination was negative; tuberculosis workup and HIV serology were negative.

Endoscopic evaluation included an upper gastrointestinal endoscopy (UGIE) revealing erythematous, petechial, and erosive pangastritis. Total colonoscopy with cannulation of the terminal ileum demonstrated erythematous, granular mucosa with erosions and superficial ulcerations, with intervening areas of healthy mucosa. Ileo-colonic biopsies showed findings consistent with IBD, including architectural disorganization, inflammatory infiltrate, cryptitis lesions and rare crypt abscesses, as well as a giant-cell granuloma without caseous necrosis.

Complementary CT enterography demonstrated circumferential and regular parietal thickening of several jejunal loops without signs of stenosis. Based on this constellation of findings, a diagnosis of Crohn's disease was established. The patient was initiated on oral corticosteroid therapy as induction treatment, with indication for biologic therapy as maintenance treatment following a negative prebiotherapy workup.

Regarding the cutaneous findings, a skin biopsy was performed. Histopathological analysis showed a normoacanthotic epidermis covered by an orthokeratotic stratum corneum. The dermo-epidermal junction was preserved. The papillary and superficial reticular dermis showed a moderate perivascular inflammatory infiltrate in a strict perivascular cuff, associated with vasculitic lesions involving intact and altered polymorphonuclear neutrophils, focal fibrinoid necrosis, and a lymphoplasmacytic infiltrate, consistent with leukocytoclastic vasculitis.

Etiological workup for the dermatological lesions included an immunological panel (antinuclear antibodies, anti-DNA, anti-cardiolipin antibodies, p-ANCA, ASCA), which returned negative, as well as 24-hour urinary protein and viral serologies (HBV, HCV, EBV, CMV), all of which were unremarkable. The therapeutic decision was therefore to maintain the patient on the same regimen (tapering oral corticosteroids and anti-TNF alpha biotherapy with Infliximab) along with analgesic treatment and monitoring of the evolution of the skin lesions.

Clinical evolution was favorable, with resolution of abdominal pain, improvement in general condition, and regression of purpuric lesions on both lower limbs.

DISCUSSION

Together with ulcerative colitis (UC), Crohn's disease (CD) constitutes an inflammatory bowel disease (IBD) of unknown etiology. It can affect all segments of the gastrointestinal tract and is associated with various extra-intestinal manifestations involving multiple organs (joints, skin, eyes, kidneys, bones, etc.). It is generally diagnosed in young adults and is characterized by a prolonged course with alternating flares of variable intensity and remission periods of varying duration, without spontaneous resolution, manifesting as chronic diarrhea, abdominal pain, weight loss, and vomiting [1].

Among the extra-digestive manifestations commonly encountered in Crohn's disease, mucocutaneous lesions are particularly noteworthy. These can be classified according to their pathophysiology into: "reactive" lesions, which generally evolve in parallel with digestive flares (e.g., oral or genital aphthous ulcers); granulomatous lesions observed exclusively in CD and not correlated with disease activity; autoimmune-mediated dermatoses associated with IBD (e.g., psoriasis); and finally, manifestations secondary to various deficiencies (zinc, iron, etc.) [3,4,6].

Diagnosis of cutaneous manifestations is most often made during a thorough clinical examination, prompting the treating physician to perform skin biopsies, particularly in cases of unusual involvement, to establish the diagnosis and guide management.

Leukocytoclastic vasculitis (LCV) is a frequent type of small-vessel cutaneous vasculitis but constitutes one of the rare cutaneous manifestations of IBD, more commonly encountered in the context of ulcerative colitis (UC). It is an inflammation of small-caliber vessels characterized by an inflammatory infiltrate associated with leukocytoclasis (fragmentation of polymorphonuclear neutrophils which, following degranulation, leads to deposition of nuclear debris) and fibrinoid necrosis at the level of post-capillary venules of small vessels [7]. It most often manifests clinically as palpable purpura, which can occur on any part of the body, but with a predilection for the lower limbs [8].

The positive diagnosis of LCV is based on skin biopsy, which should be performed within the first 48 hours after the appearance of the lesion [8]. The biopsy must be sufficiently deep to allow examination of the hypodermis; histological study demonstrates the existence of fibrinoid deposits at the level of capillaries and/or post-capillary venules, inflammatory cells (particularly polymorphonuclear neutrophils penetrating the vessel wall), and perivascular nuclear dust: this constitutes leukocytoclasis [9].

For the therapeutic management of LCV in the context of IBD, the decision depends on the extent and severity of the lesions. LCV generally responds to treatment of the IBD flare, as was described and adopted in our case. It can be treated with salicylate derivatives, systemic corticosteroids, immunosuppressants, or

colchicine (which has proven very effective in LCV through its action on polymorphonuclear neutrophils), and intravenous immunoglobulins for very severe or treatment refractory cases, combined with standard symptomatic treatments such as analgesics or antihistamines [4,8]. In advanced stages, development of necrosis or gangrene of the affected limb may be observed, which can compromise functional prognosis and necessitate amputation [4].

It is equally important to counsel affected patients regarding hygiene measures to be followed for optimal management, including reduction of vasculitis-exacerbating factors such as prolonged standing, cold exposure, and wearing of tight-fitting clothing, as well as rest with leg elevation while keeping the limbs warm [4,10,11].

CONCLUSION

Leukocytoclastic vasculitis is a rare and unusual cutaneous manifestation of IBD, particularly Crohn's disease. Unlike other common dermatological manifestations, LCV requires skin biopsy to confirm the diagnosis.

The management of this type of lesion remains non-standardized and and depends on the severity and extent of the lesions. Generally, it responds favorably to treatment of the digestive disease flare. This underscores the importance for gastroenterologists and dermatologists to be well acquainted with this rather specific form of vasculitis; in order to diagnose and treat it correctly and thereby avoid the deleterious consequences that may arise in advanced stages of the disease, notably cutaneous necrosis.

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