

Cutaneous Leukocytoclastic Vasculitis Associated With A Fast Food Diet

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Citation

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Abstract

Leukocytoclastic vasculitis (LCV) is the inflammation and necrosis of the wall of small blood vessels exhibited by endothelial swelling, perivascular polymorphonuclear cellular infiltrate, neutrophil fragmentation and nuclear debris and is typically localized to the skin. Many medications, diseases and infections have been implicated in the development of leukocytoclastic vasculitis. A review of available literature reveals case reports of leukocytoclastic vasculitis induced by medications such as infliximab, rituximab, etanercept, cocaine and numerous others and infections such as HIV and Streptococcal pneumonia as well as malignancies and chronic inflammatory conditions such as ulcerative colitis and collagen vascular diseases. Leukocytoclastic vasculitis induced by food has also been reported. Here, we report an interesting case of cutaneous leukocytoclastic vasculitis that developed in a patient after months of consuming a fast food diet for breakfast, lunch and dinner, which would improve only after abstaining from such a diet. The proper diagnosis and management of leukocytoclastic vasculitis remains a challenge for many clinicians and this report serves to give a brief overview of this condition, its causes, epidemiology, diagnosis and management.

INTRODUCTION

Leukocytoclastic vasculitis is characterized by necrotizing inflammation around blood vessels in the dermis which is composed mainly of neutrophils and their debris. It has been associated with numerous conditions such as autoimmune diseases, infections, malignancies, allergic or adverse reactions to medications or food, although it may also be idiopathic. Cutaneous lesions may vary from purpura, macules, papules, urticarial, vesicles, ulcers, livedo reticularis to necrosis. Systemic manifestations may include pulmonary, gastrointestinal, renal or central nervous system involvement. We describe an unusual case in which isolated cutaneous leukocytoclastic vasculitis was induced after months of consuming a fast food diet associated with the development of hyperlipidemia and hypertriglyceridemia.

CASE

The patient is a 59 year old male with remote history of intravenous drug abuse (IVDA), psoriasis, nephrolithiasis, who reported to his physician that he was consuming a fast food diet for the last 2-3 months and was feeling unwell and developed a rash. On examination were areas of reticular erythema surrounding a pale central area consistent with livedo reticularis on his upper extremities. On the lower extremities were multiple scattered maculopapular

erythematous lesions, some blanchable, and some ulcerative nodular lesions at different stages of healing. (Figure 1) The patient was then evaluated by a dermatologist whose clinical impression was consistent with erythema nodosum. A punch biopsy at the time showed a perivascular infiltrate of lymphocytes in the dermis, becoming denser in the deeper part of the reticular dermis with hints of an infiltrate in the subcutaneous fat at the base of the biopsy. The diagnosis by the pathologist was probable panniculitis which 'could be the surface of erythema nodosum. It was discovered that the patient's cholesterol level was greater than 900 and he was started on statin therapy. After becoming compliant with a balanced diet, these lesions disappeared though the livedo reticularis persisted.

Three years later, when the patient became non-compliant with his diet, the painful nodules recurred would later ulcerate, heal and ultimately disappeared after 6 weeks once he stopped eating at fast food restaurants. In August 2010, he resumed poor eating habits once again with recurrence of a rash, nodules, and ulcers after 2 months. A punch biopsy of an ulcer on his right leg was consistent with leukocytoclastic vasculitis as it had scattered perivascular infiltrates composed of neutrophils with leukocytoclastic debris within the superficial and deep dermis. Several vessels also had

neutrophils within their walls and were occluded by fibrin thrombi. The patient was then referred to rheumatology and a review of systems was negative for signs or symptoms of systemic disease.

On physical examination, the patient was afebrile, normotensive with livedo reticularis on his extremities and trunk, and a series of ulcerated lesions on his extremities and the remainder of the exam was otherwise unremarkable. A serological workup revealed a negative ANA, ANCA, Rheumatoid factor, Anti-cardiolipin antibodies, lupus anticoagulant, beta 2 glycoprotein, and Hepatitis C antibodies. Cryoglobulins and cold agglutinins were negative on two separate occasions. He tested positive for hepatitis B core antibody, but the DNA PCR for viral load was negative. Protein C and protein S levels were normal and the factor V Leiden mutation test was negative. He also tested positive for the heterozygous form of the methylenetetrahydrofolate reductase (MTHFR) gene polymorphism.

A complete metabolic profile, liver function, complete blood count, urinalysis, complement levels, serum and urine electrophoresis, homocysteine, folate and cholesterol levels were all within normal limits. The ulcers on his lower extremities became infected which precluded the use of glucocorticoids or immunosuppressant therapy.

DISCUSSION

The incidence of cutaneous vasculitis is reported to range from 15.4 to 29.7 cases per million per year, and it affects all ages, slightly fewer males than females, and adults more often than children, with 90% of the latter having Henoch-Schonlein Purpura (HSP) (2). It has been further reported that almost half of all patients presenting with cutaneous vasculitis have self-limited disease localized to the skin without any attributable cause, trigger or associated systemic disease (idiopathic). The remainder of localized cutaneous vasculitis cases is usually either due to infection or drugs and a minority of cases are ANCA-associated (2). The presence of Anti-Neutrophil Cytoplasmic Antibodies (ANCA) is not diagnostic of systemic vasculitis, as some cases of cutaneous LCV have a positive ANCA and limited skin disease, and can be found at low levels in many systemic inflammatory and pulmonary disorders that mimic vasculitis (2). LCV is part of a spectrum of diseases (4); and as such the mechanism by which it presents may vary depending on the inciting agent. For instance, in hypersensitivity vasculitis, immune complex formation, deposition, and complement

activation may be important early events, in at least a subset of patients (4).

At a microscopic level, cutaneous leukocytoclastic vasculitis affects the upper to middle dermis while cutaneous polyarteritis nodosa tends to also affect the deeper dermis. An incisional biopsy is essential in distinguishing between these two diagnoses. The timing of the biopsy is also important as histological findings of vasculitis can vary. As suggested by Carlson et al., the optimal time for the skin biopsy is less than 48 hours after the appearance of a vasculitic lesion (2). It is therefore recommended to obtain incisional biopsies at an early stage in order to increase the diagnostic yield.

Our patient was diagnosed with cutaneous polyarteritis nodosa (PAN) given the results of the biopsy, history of hepatitis B exposure, presence of livedo reticularis and painful subcutaneous nodules which would ulcerate. This condition is typically not associated with significant internal disease but its course may be chronic and relapsing, and is usually treated relatively conservatively. Patients with mutations in the MTHFR gene, which result in reduced enzymatic activity in the metabolism of homocysteine are at increased risk for arteriosclerotic coronary disease and venous thrombosis. This mutation has been associated with cutaneous ulcers due to elevated homocysteine levels. The temporal relationship between the diet of the patient and development of this condition has not been reported in the English literature. In our patient, dietary modification led to the resolution of the rash. It remains unclear however, whether the underlying cutaneous PAN was unmasked by the diet and dyslipidemia or whether it was an unrelated manifestation at that time.

Figure 1

Figure 1: Lesions on the lower extremities of the patient



In summary, depending on the type of cutaneous vasculitis, various degrees of treatment success have been reported with the use of cromolyn, a mast-cell stabilizer, methotrexate, mesalamine, infliximab, and other immune-modulating agents, as well as steroids. A detailed history, physical examination and investigations to rule out systemic involvement are essential to establishing a diagnosis along with an incisional biopsy. Close follow-up and monitoring is imperative to allow for early detection should systemic involvement occur.

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