CLINICAL IMAGE

BILATERAL LOWER EXTREMITY RASH IN A 34-YEAR-OLD MAN

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CASE REPORT

A 34-year-old Caucasian male with no past medical history presented to the emergency department for a bilateral lower-leg rash that began 3 days prior. The rash was not painful or pruritic. The area of involvement had expanded and the rash had darkened since onset (see Figures 1 and 2). The patient denied any trauma to the area or contact with any irritants. No fevers were reported. He denied any similar rash in the past. He reported that he works at a large retailer and his job requires him to stand most of the day, in addition to walking, climbing, and performing physical work.

On examination, he was obese and hypertensive with a blood pressure level of 172/98 mmHg. His skin examination revealed a rash on his bilateral lower extremities that was nonpalpable, nonblanching, and nontender. It cut off abruptly at the sock line and spared the feet. It was not warm to palpation and there were no areas of open wounds or drainage. There was no mucous-membrane involvement or involvement of the palms or soles. There was no edema to the lower extremities.

FIGURE 1:

Right leg

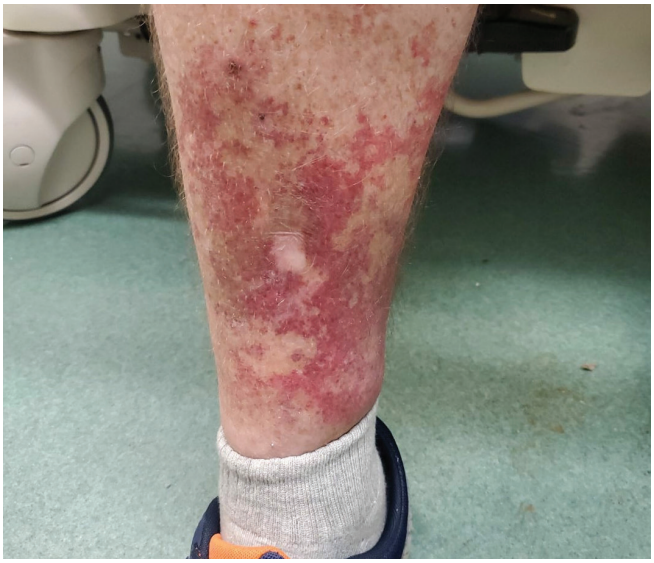
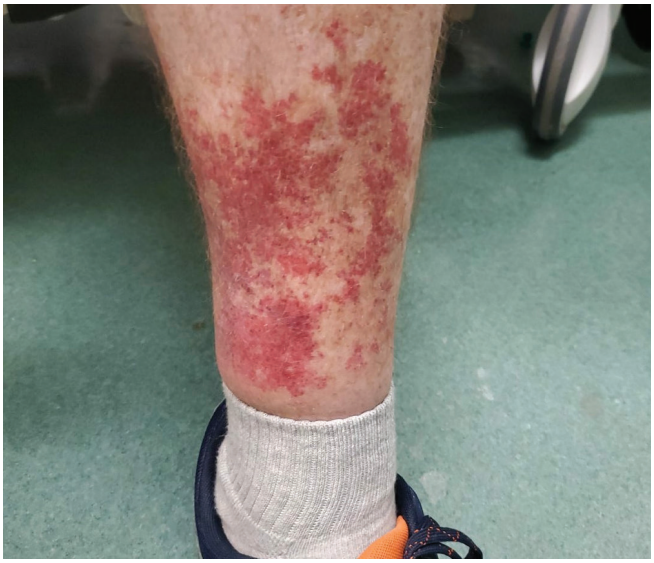


FIGURE 2:

Left leg



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QUESTIONS

1. This presentation aligns most with which diagnosis?

a. Cellulitis

b. Exercise-induced vasculitis (EIV)

c. Immunoglobulin A (IgA) vasculitis

d. Immune thrombocytopenia

Correct answer:
c. Exercise-induced vasculitis (EIV)

This patient presents with a lower-leg erythematous lesion, sparing below the sock line after prolonged standing, consistent with EIV.[1-5] Although EIV typically occurs in people over the age of 50 years who golf or hike in warm weather,[2,3] cases have been reported in younger patients.[6,7] The patient's history of prolonged standing with his job, possibly in combination with being obese, are risk factors for development of EIV.[8] Other types of vasculitis typically are not confined to the lower legs. IgA vasculitis would usually show signs of renal involvement. Immune thrombocytopenia presents with increased bleeding, and the palpable purpura is not typically present in crops in dependent body regions, like it is with EIV.[9] Cellulitis would present with erythema and tenderness to palpation, and the skin involved would be hot when palpated.[10]

2: What is the first-line treatment for the patient's diagnosis?

a. Intravenous (IV) antibiotics

b. Oral colchicine

c. Oral prednisone taper

d. Supportive care including leg elevation, compression stockings, nonsteroidal anti-inflammatory drugs (NSAIDs), and antihistamines

Correct answer:
c. Oral prednisone taper

Due to the clinical suspicion for metastases, a biopsy should be performed to characterize the histology and etiology of the tissue. Care should be taken when considering the region of the body that is being biopsied, history of present illness, and acuity of postbiopsy follow-up. In this case, a periauricular lesion was referred to an ENT for biopsy due to the sensitive nature of nearby neurovascular and bleeding risks, while the other lesions were referred to general surgery given the high suspicion for metastasis. There were no indications of infection in the area, so incision and drainage, antibiotics, or antifungals would not be appropriate. The lesions were painless, nonpruritic, and otherwise not impacting the patient at the time of presentation, so topical steroids are also not indicated.

2: One proposed mechanism for the patient's pathology is:

a. Intravenous (IV) antibiotics

b. IgA-mediated tissue damage

c. Immune-mediated platelet destruction leading to dysfunctional clotting cascade

d. Local bacterial tissue infection

Correct answer:
a. Dysfunctional local tissue temperature regulation

Although the pathophysiology of EIV is not well understood, one proposed mechanism is dysfunctional local temperature regulation.[4] EIV is known to take place commonly in warm climates.[1-3] Some literature suggests that increased cutaneous blood flow in warm temperatures, in combination with dysfunctional venous return, can lead to extravasation of red blood cells resulting in purpuric or petechial lesions on the lower legs.[4,8] IgA-mediated destruction of tissues is the mechanism of IgA vasculitis that is seen on biopsy.[9] Local bacterial tissue infection is consistent with cellulitis.[10] Immune-mediated platelet destruction leading to a dysfunctional clotting cascade is consistent with a diagnosis of immune thrombocytopenia.[9]

DISCUSSION

EIV is a benign and self-limited cutaneous small-vessel vasculitis that has been found to occur after prolonged exercise and is especially prominent in warmer temperatures.[2,3,11] The vasculitis generally presents with nonblanching, purpuric, petechial, or erythematous lesions on the bilateral lower legs, although blanchable lesions have been reported.[4,5,9] In some patients, the lesions can be noted on the feet or thighs, although the sock area is usually spared, with clear demarcation.[3,6,7,13-15]

The lesions can be asymptomatic, but they are often pruritic, painful, and have a burning sensation.[8-10] Some lesions are seen with lower-leg edema.[4,8,13] EIV is typically a self-limited condition that resolves in less than 10 days, but recurrence is common.[1,2,8] It is commonly seen in golfers, hikers, and persons who participate in other activities that include prolonged walking or running.[1,3,5,13,14] It is reported most in women and people over the age of 50 years.[1-3,8]

The pathophysiology of EIV is not fully understood. Some studies suggest that diminished thermoregulation might contribute to development of EIV.[4] Raising the core body temperature results in increased cutaneous blood flow and vasodilation, causing an increase in venous blood volume to the extremities. If the venous system becomes overfilled due to the presence of edema or venous insufficiency, this might potentially lead to damaged vasculature and extravasation of red blood cells, causing EIV.[4] Increased adipose tissue in the legs may also alter thermoregulation causing an increased core temperature and precipitating EIV through thermal damage to blood vessels.[8] Additionally, venous filling time is reduced with prolonged exercise, subsequently decreasing the efficacy of venous return and also causing stasis and red blood cell extravasation.[8]

Although EIV is self-limited and benign, it can be difficult to differentiate its presentation from systemic etiologies of cutaneous small-vessel vasculitis. The differential diagnosis for cutaneous small-vessel vasculitis includes several autoimmune conditions including systemic lupus erythematosus and antinuclear cytoplasmic antibody vasculitis.[9] Infectious etiologies and adverse drug effects should also be ruled out. Pertinent review of systems should be performed to assess for systemic vasculitis, fever, weight loss, fatigue, arthralgias, hematuria, abdominal pain, paresthesias, weakness, shortness of breath, cough, hemoptysis, and sinusitis.[9] Although EIV can be diagnosed clinically using a patient's history and physical examination, some literature suggests that skin biopsy should be performed to rule out systemic pathologies.[9-12,14] If biopsy is performed, the histologic findings may vary depending on at what point in the disease the sample was obtained. The sequence of events leading to EIV suggested by timed biopsies is a multistep cascade that begins with immune complex deposition and results in neutrophil damage to vessels.[16,17] If underlying systemic disease cannot be ruled out with physical examination and review of systems, laboratory testing, including complete blood count, creatinine levels, erythrocyte sedimentation rate, liver function tests, and urinalysis, should be completed.[9]

The first-line treatment for EIV is supportive care, including rest and leg elevation.[9,11,12] If the patient's lesions are symptomatic, topical steroids and NSAIDs are indicated.[9,11,12] Some patients find that the use of compression stockings and lighter clothing decreases recurrence.[1-3,8,11] A short taper of systemic steroids, oral colchicine, or dapsone can be trialed for ulcerated or severe lesions.[2,8,9,11] If the vasculitis persists despite these treatments, immunosuppressants can be attempted.[9,11] If the patient has progressed to needing oral prednisone and immunosuppressants, there should be a full workup performed for systemic etiology of cutaneous small-vessel vasculitis, and specialist referral should be considered.[9,11]

Although EIV is a benign and self-resolving condition, awareness of the disease is important because it can be easily misdiagnosed as a more serious condition prompting unnecessary workup and treatment. The physician should rely on a thorough history and physical examination while looking for signs of systemic disease and considering more serious pathologies.

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