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Case Report

Livedo of the Forearm in the Aftermath of Forearm Erysipelas

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Abstract

Livedo is an ischemic dermopathy caused by vasculopathies or prothrombotic states, and characterized by the violaceous lace-like mottling of the skin. We report on a patient who developed livedo reticularis – livedo racemosa overlap syndrome as a late sequel of erysipelas, the livedo being restricted to the limb segment affected earlier by erysipelas and devoid of systemic vasculopathy. Though erysipelas and livedo are common disorders, we could not find in the literature reports of an occurrence like that observed in this patient. In this case a favorable prognosis of livedo could be predicted. In a different context, livedo may be the alarming signal of an undiagnosed systemic disease.

Keywords: livedo reticularis; livedo racemose; erysipelas; broken rings; old woman, deoxygenated hemoglobin

Introduction

Livedo reticularis (LR) is an ischemic dermopathy characterized by the violaceous lace-like mottling of the skin forming complete dark rings surrounding a pale center that is caused by arteriolar vasospasm or flow disturbance as seen in polycythemia. LR typically occurs on the trunk and proximal segments of extremities. Livedo racemose (LRa), a related disorder, differs from LR by larger, irregular, asymmetrical, broken rings, often caused by an underlying vasculopathy and prothrombotic diathesis (1-3). There is a spectrum of livedo and livedo-like disorders. Congenital LR is limited to one extremity and often associated with limb asymmetry and neurologic abnormalities. LR can occur as a physiological response to cold exposure (known as cutis marmorata). Acquired LR and LRa may be an expression of systemic vasculopathies and prothrombotic disorders: hyperviscosity syndromes, chronic myeloid leukemia, multiple myeloma, heavy chain disease, cryoglobulinemia, cryofobrinogenemia, systemic lupus erythematous, antiphospholipid antibody syndrome, rheumatoid arthritis, systemic sclerosis, hyperhomocysteinemia, cholesterol embolism, factor Leiden mutation, deficiency of proteins C, S or antithrombin, elevated levels of plasminogen activator inhibitor type 1 (PAI-1), HIV, hepatitis B, hepatitis C, renal cell carcinoma, carcinoid, and intravascular lymphoma. Up to 20% of the cases of LV are idiopathic (3,4). At a difference, livedoid vasculopathy is characterized by punched out ulcers in the peri-malleolar area surrounded by lacy, reticular, streaks of LR. These ulcers heal forming white scars surrounded by telangiectasias and are remnants of infarction due to disturbed capillary

microcirculation. Retiform or stellate purpura is considered a hallmark lesion (5).

We report on the occurrence of LR-LRa in the aftermath of erysipelas, the livedo being confined to the segment previously affected by erysipelas and livedo persisting after recovery from erysipelas. Though both erysipelas and LR are frequent disorders, we could not find in the literature reports of a similar sequence of events.

Case History

An 88-year-old woman of Ashkenazi Jewish extraction was transferred to our institution under permanent mechanical ventilation and receiving enteral feeding. Her medical history was remarkable for end-stage Alzheimer's dementia, arterial hypertension, hypothyroidism, pulmonary aspirations and atelectasis. The medications were ramipril 1.5 mg/day, bisoprolol fumarate 1.25 mg/day, levothyroxine 100 mcg/day, and acetylcysteine 600 mg/day. The patient had normal levels of hemoglobin, white blood cells, platelets, chloride, bicarbonate, creatinine, blood urea nitrogen. The 28th of July an extensive erythema appeared on her left forearm: tense, red, hot, uniformly elevated, with a sharply defined, raised border. Her bodily temperature was 38.1°C. Erysipelas was diagnosed and ampicillin-clavulanate treatment was administered 875 mg b.i.d. for 10 days. There was no visible portal of entry of the infection. Improvement was witnessed within 5 days. However, a few days after completion and discontinuation of antibiotic treatment there was a flare of the erythema, involving the right forearm and distal area of the right arm. The white blood cell count was 5000/mm3 and the C reactive protein 3.1 mg/L.

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Blood cultures were negative. There was no evidence of extension of the infection to deeper tissues (abscess, septic phlebitis, deep vein thrombosis, necrotizing fasciitis or myonecrosis). Treatment with piperacillin-tazobactam was started followed within a week by resolution of the erythema. After the infection remitted a violaceous netlike mottling of the skin became apparent on the patient's right forearm distinctive to the normal skin color elsewhere. Complete violaceous rings and broken

rings were surrounding pale centers. Under slight pressure, blanching of the violaceus areas occurred. There was no change of the livedo in a warm ambience. The diagnosis was livedo reticularis – livedo racemosa overlap (**Figure 1**). During three months of observation the livedo persisted and remained limited to same area. There was no alteration in the patient's general state, temperature, blood pressure, heart rate and SpO₂.



Figure 1: Complete violaceous rings of livedo reticularis (arrows) and broken rings of livedo racemosa (in the more distal areas).

Discussion

The cutaneous vasculature consists of 1-3 cm cones with the apex of the cone situated deep in the dermis at the position of an ascending arteriole. At the margins of each cone the density of the arterial bed is decreased and the superficial venous plexus becomes prominent. Any process that impedes blood flow to the skin can result in an increased proportion of deoxygenated hemoglobin and thereby a prominent livid coloration in the venous areas at the margins of the cones, i.e. livedo (6). Livedo has been divided into a) LR which presents as symmetrical, lace-like, patches forming complete dark rings surrounding a pale center, generally associated with cold-induced cutaneous vasoconstriction or vascular flow disturbances such as seen in polycythemia and, and b) LRa characterized by larger, irregular, and asymmetrical rings, more frequently associated with focal impairment of blood flow (2,3,7). The differential diagnosis of livedo, either LR or LRa, includes some viral exanthems and erythema ab Inge, i.e. a reticulated hyperpigmentation induced by continued heat exposure. Further, mycosis fungoides, dermatomyositis, parvovirus B19 infection, and reticular erythematous mucinosis may present with a reticulate pattern but the presence of epidermal changes and telangiectasias helps to distinguish these conditions from LR (8).

Erysipelas was diagnosed in the proposito as usually, built on the eruption's appearance and the clinical context. Bacteriologic cultures are useless because positive results are rare. The prognosis of erysipelas with antibiotic treatment is generally good, but recurrences are common. A flare of erysipelas may be due to unusual organisms, resistant strains of staphylococcus or streptococcus, or to extension of the infection to deeper tissues (abscess, septic phlebitis, deep vein thrombosis, necrotizing fasciitis or myonecrosis). There was no evidence of either in the present case. Eventually, under antibiotic treatment, recovery was complete and uncomplicated except for emergence of livedo.

Occurrence of livedo during infections has been noticed sporadically (3,4) but came into focus recently during the 2019-2021 Covid-19 epidemic. Among Covid-19 associated cutaneous manifestations - urticarial, morbilliform rash, papulovesicular exanthem, chilblain-like - livedo is a small minority (9,10). LR occurring during Covid-19 is usually mild, transient, and not associated with thromboembolic complications (11). On the contrary, LRa-like and retiform purpura occurring during Covid-19 was associated with severe coagulopathy (12). The proposito had received the second dose of the BNT162b2 (Pfizer–BioNTech) vaccine late in February 2021. There were no symptoms and signs of a corona disease and there was no deterioration of her stable respiratory status. In the

proposito livedo occurred in the aftermath of erysipelas. Livedo was confined exclusively to the limb segments that were shortly before affected by erysipelas. Livedo persisted long after recovery from erysipelas. By all these features livedo in this patient differs from livedo which complicates Covid-19 disease.

Conclusion

Livedo in this patient occurred as a late sequel of erysipelas and was restricted exclusively to the limb segment affected by erysipelas. It was devoid of symptoms of a systemic vasculopathy of thrombotic diathesis. In this context a favorable prognosis of livedo could be predicted. In distinction, in a different context, livedo may be the alarming signal of an undiagnosed systemic disease.

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