Abstract
Livedo reticularis can present with progressive ischemia and diffuse cutaneous involvement with or without any evidence of systemic diseases. Livedo reticularis (LR) is a livedoid discoloration of the skin in a reticular pattern. We report the case of a 30-year-old male who presented with an asymptomatic, red-colored, net-like rash all over the body for 4 years. Laboratory investigations were performed to rule out any systemic involvement. Biopsy showed perivascular mononuclear cell infiltrate and occasional arteriole showed thickening of the wall with obliteration of the lumen and extensive collagenization in dermis, suggesting a diagnosis of LR. The patient was advised oral pentoxifylline 400 mg thrice daily with oral nifedipine 10 mg twice daily, and mild improvement was seen after 6 weeks of therapy.

Key Words: Generalized, livedo reticularis, pentoxifylline

Introduction
Livedo reticularis (LR) is only a physical sign characterized by a persistent, reddish-blue, net-like cyanotic pattern.[1] LR without systemic associations may be differentiated into three distinct entities: physiologic LR, primary, and idiopathic.[2] Secondary LR is identified as LR with systemic associations. Most of the cases described in the literature belong to the category of secondary LR. However, a long-term follow-up is required for any future development of systemic associations.

Case Report
A 30-year-old married male presented with an asymptomatic, red-colored, net-like rash all over the body for 4 years. The patient was well 4 years back when he started developing such lace-like rash over the abdomen, which gradually spread to involve trunk, arms, and thighs within 6 months. There were no associated symptoms such as pain or pruritus. No other skin lesions such as ulcer, nodule, purpura, and atrophie blanche were present. The patient did not have any significant drug history. There was no history of headache, seizure, stroke, depression, confusion, syncope, dementia, chorea, sensory disturbances, hemiparesis, and limb pain. The patient did not give any history of surgery and trauma. A history suggestive of associated infections such as fever, vomiting, chronic diarrhea, cough, weight loss, and neck stiffness was absent. There was no history of oral ulcer, photosensitivity, joint pain, muscle weakness, skin thickening, and Raynaud's phenomenon. The patient did not give a history of extramarital sexual contact. A history suggestive of bleeding diathesis was not found. The patient did not have any previous episode of similar illness in the past. Family history was insignificant.

On cutaneous examination, a nonblanchable erythematous macular rash in a reticulate pattern was present over abdomen, trunk, both arms, thighs, and legs [Figures 1a and b]. Oral and genital mucosae, palms and soles, and scalp and nails were normal.

Routine investigations, such as complete blood count, erythrocyte sedimentation rate, liver function test, renal function test, lipid profile, fasting and postprandial blood sugar, urine analysis, human immunodeficiency virus enzyme-linked immunosorbent assay test, hepatitis B antigen, anti-hepatitis C virus antibodies, thyroid function test, antinuclear antibody, complement proteins C3 and C4, rheumatoid factor, cryoglobulins, Mantoux test, venereal disease research laboratory test, chest X-ray, and electrocardiography, were performed to rule out any systemic cause and to plan further treatment. All the investigations were found normal.
The differential diagnoses were LR, angioma serpiginosum, reticular erythematous mucinosis, and viral exanthem. The possibility of viral exanthem was ruled out because of chronic course of the present condition. Punch biopsy for histopathology was carried out to confirm the diagnosis. Histopathology revealed that epidermis was unremarkable. Upper dermis showed perivascular chronic mononuclear cell infiltrate, and occasional arteriole showed thickening of the wall with obliteration of the lumen. Deeper dermis showed extensive collagenization. Histopathological features were suggestive of LR [Figure 2].

The patient was advised oral pentoxifylline 400 mg thrice daily and oral nifedipine 10 mg twice daily. Mild improvement was seen after 6 weeks of therapy. However, later, the patient was lost to follow-up.

**Discussion**

Ehrmann in 1907 distinguished two different patterns of livedo: the physiological LR where the reticular pattern comprises complete or unbroken circles and the pathological livedo racemosa which has an incomplete reticular pattern.[3] The livid rings in both the forms are caused by reduced blood flow and lowered oxygen tension at the peripheries of the skin segments.[6-5] LR is a livedoid discoloration of the skin in a reticular pattern.[3] Barker et al. described it as a circulatory phenomenon and not a disease.[6] LR without systemic associations may be differentiated into three distinct entities: physiologic, primary, and idiopathic.[2]

Physiologic LR is known as cutis marmorata which appears in response to cold exposure, resolving completely with warming of the affected limb. It is most commonly found in preterm infants, neonates, and fair-skinned girls and women, and it is usually confined to the lower extremities.[2]

Primary LR is a diagnosis of exclusion. It is defined by the appearance and resolution of LR independent of ambient temperature and in the absence of underlying disease.[2]

Idiopathic LR is persistent LR without an underlying cause. While it clearly shares many features with primary LR, it differs by the persistence of the livedo pattern.[3] Although generalized LR has been reported, in our case, persistent generalized LR has not been found to be associated with other cutaneous changes and systemic involvement till now.

LR with systemic associations is called secondary LR. Among causes of secondary LR, one of the main causes mentioned is amantadine-induced LR. The incidence of LR in patients treated with amantadine (mostly parkinsonism patients) is still unclear.[5] Other important causes of secondary LR are hematological conditions such as antiphospholipid syndrome, cryoglobulinemia, polycythemia vera, Sneddon’s syndrome, and thrombotic thrombocytopenic purpura; autoimmune disorders such as small and medium vessel vasculitis; connective tissue diseases such as systemic lupus erythematosus and systemic sclerosis; infections such as syphilis, tuberculosis, brucellosis, mycoplasma pneumonia, parvovirus B19, meningococcemia, and viral infection; neurological disorders such as encephalitis, polyomyelitis, Parkinson’s disease; and medications such as minocycline, catecholamines, quinidines, and gemcitabine.[1,2,5]

No ideal and effective treatment for LR has been described till date. Avoidance of cold must be there in case of idiopathic LR. Various modalities that had been tried were antiplatelet drugs such as low-dose aspirin and clopidogrel and vasodilators such as nifedipine and pentoxifylline. Other modifications that had been advised for lowering the risk were blood pressure control, diabetes management, smoking cessation, and weight loss. However, the effects of therapies are not consistent.[7]

LR is really a diagnostic challenge as we must rule out a large bagful of associated systemic diseases. Further, our case might be a quite rare case of generalized LR without any features of ischemia, ulceration, and
other systemic involvement. However, the patient needs to be followed up for future development as a large number of patients develop systemic associations including neurovascular and cardiovascular complications several years after the onset of livedoid reticularis. The rarity of such entity and the paucity of reports in the literature prompted us to report this case.

**Declaration of patient consent**
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**
There are no conflicts of interest.

**References**
1. Sajjan VV, Lunge S, Swamy MB, Pandit AM. Livedo reticularis: A review of the literature. Indian Dermatol Online J 2015;6:315-21.
2. Gibbs MB, English JC 3rd, Zirwas MJ. Livedo reticularis: An update. J Am Acad Dermatol 2005;52:1009-19.
3. Sangle SR, D'Cruz DP. Livedo reticularis: An enigma. Isr Med Assoc J 2015;17:104-7.
4. Lubach D, Schwabe C, Weissenborn K, Hartung K, Creutzig A, Drenk F, et al. Livedo racemosa generalisata: An evaluation of thirty-four cases. J Am Acad Dermatol 1990;22:633-9.
5. Kraemer M, Linden D, Berlit P. The spectrum of differential diagnosis in neurological patients with livedo reticularis and livedo racemosa. A literature review. J Neurol 2005;252:1155-66.
6. Barker NW, Hines EA, Craig W. McK. Livedo Reticularis: A Peripheral Arteriolar Disease. Am Hear J 1941;21:592–604.
7. Maessen-Visch MB, Koedam MI, Hamulyák K, Neumann HA. Atrophie blanche. Int J Dermatol 1999;38:161-72.