



Livedo reticularis related to physical activity: A case report

Fiziksel aktivite ilişkili Livedo retikularis: Olgu sunumu

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Özet

Livedo retikularis; geçici veya kalıcı olabilen, maküler, morumsu renkte, birbirine bağlanan halkalardan oluşan bir cilt bulgusudur ve ağ şeklinde siyanotik bir desen oluşturur. Bu görünüm, venöz pleksusun dermal tabaka altında görünürlüğüne artmasıyla ilişkili herhangi bir durum tarafından tetiklenebilir. Yaşlılarda dermisin incilmesi, bu lezyonun alta yatan patoloji olmaksızın meydana gelme nedenlerinden biridir. Venodilatasyon (hipoksi veya otonom disfonksiyon nedeniyle) ve kanın deoksijenasyonu (venöz tromboz, vazospazm veya artmış kan viskozitesi) venöz pleksusun görünürlüğüne iki ana nedenidir. Genellikle livedo retikularis vakalarının çoğunda, bu durum soğuk maruziyetine fizyolojik bir tepki olarak ortaya çıksa da, antifosfolipid sendromu veya Sneddon sendromu gibi alta yatan bir hastalığın dermatolojik belirtisi olabilir. Bu makalede, fiziksel aktivite ile kötüleşen ve alta yatan patoloji veya sıcaklıkla ilişkisi ekarte edildikten sonra primer livedo retikularis tanısı alan 40 yaşında bir kadın hastayı sunuyoruz. Livedo retikularis tedavisi için dolaşımın genişlemesine yardımcı olması nedeniyle egzersiz önerilse de, bu olguda egzersizin bulguları artırdığı kontroversiyel bir durum görülmektedir.

Anahtar kelimeler: Livedo retikularis, fiziksel aktivite, egzersiz

Summary

Livedo reticularis is a skin finding consisting of transient or persistent, macular, violaceous, connecting rings that form a netlike cyanotic pattern. This appearance may be caused by any situation which is related to increase of visibility of the venous plexus under the dermal layer. Thinning of the dermis in elderly people is one of the causes of this lesion without any pathology. Venodilation (triggered by hypoxia or autonomic dysfunction) and blood deoxygenation (ie. venous thrombosis, vasospasm or increased blood viscosity) are two major causes of venous plexus visibility. Although, in most cases livedo reticularis is a result of a physiological response to cold exposure, it can also be a cutaneous sign of an underlying disease such as antiphospholipid syndrome or Sneddon's syndrome. In this paper, we present a 40 year old female patient having cutaneous manifestations exacerbated by physical activity and who got primary livedo reticularis diagnosis after the exclusion of any underlying pathologies and relation to temperature as in the case of physiological livedo reticularis. Albeit exercise is recommended for the treatment of livedo reticularis because increased circulation helps to dilate the blood vessels throughout the body it is controversial as in this case exercise was what exacerbated the situation.

Anahtar kelimeler: Livedo reticularis, physical activity, exercise

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Introduction

Livedo reticularis (LR), is a skin finding consisting of transient or persistent, macular, violaceous, connecting rings that form a netlike cyanotic pattern.⁽¹⁻²⁾ This appearance may be caused by any situation which is related to increase of visibility of the venous plexus under the dermal layer. Thinning of the dermis in elderly people is one of the causes of this lesion without any pathology. Venodilation (triggered by hypoxia or autonomic dysfunction) and blood deoxygenation (ie. venous thrombosis, vasospasm or increased blood viscosity) are two major causes of venous plexus visibility. Although, in most cases livedo reticularis is a result of a physiological response to cold exposure, it can also be a cutaneous sign of an underlying disease such as antiphospholipid syndrome or Sneddon's syndrome (Table 1).⁽²⁻⁵⁾

In this paper, we present a primary LR case exacerbated by physical activity. Due to its transient nature, and the absence of additional symptoms such as pain or itching, this is a condition that is particularly challenging to diagnose. Therefore, recognition of livedo reticularis and the detection of any underlying pathology through differential diagnosis become crucial in primary care settings serving patients of all age groups.

Case

A female, 40 year old patient referred to cardiology clinic with symptoms of palpitation, mild dyspnea and fatigue. After normal physical examination findings; a complete blood count, biochemical parameters, treadmill stress test and echocardiography were planned (Table 2).

During treadmill stress test; remarkable purple, netlike, macular lesions regionally appeared on her arms and lower legs (Figure 1). She mentioned that these lesions were appearing after any physical activity and were disappearing in approximately 15 minutes. The lesions did not blanch on applying pressure and extremities were neither cold nor painful.

We investigated the details of the medical history and broadened laboratory parameters to search a clue of an underlying disease. She mentioned that these lesions may appear especially when walking fast or engaging in intense household activities and disappear after a rest of 15-20 minutes in her daily life. She didn't notice any correlation between the lesions and hot or cold temperatures.

There was no history of any precipitating factors, drug intake, chronic disease or peripheral vascular disease. She had one spontaneous abortion and two children with no obstetric complications. She had past history of tuberculosis 20 years ago. There was



Figure 1

no similar condition or any known circulatory disorder in her family history.

Laboratory findings revealed decreased platelet count, elevated lactate dehydrogenase, erythrocyte sedimentation rate and fasting plasma glucose. Activated partial thromboplastin time and prothrombin were normal. Serum electrolytes, thyroid function parameters and renal function tests were within the normal range. Echocardiogram was reported as mild aortic and tricuspid regurgitation. Anticardiolipin antibody IgG, IgM and lupus anticoagulant tests were negative. After consulting with the dermatologist we excluded possible systemic causes of livedo reticularis and we concluded to primary LR.

Discussion

Livedo reticularis is a skin finding which may be classified in two major groups: related with a systemic condition or without any systemic association. If no underlying disease is found (such as hypercoagulability, hypothyroidism, connective tissue diseases, autoimmunity or neoplasia); the situation may be physiological, primary or idiopathic.⁽²⁾

Physiological LR (or cutis marmorata) is mainly a condition of young women and is a result of the impaired blood flow in cutaneous vessels due to cold exposure. Primary LR is also a transient skin colour change but it differs from physiological one with its lack of any connection to temperature. Idiopathic LR is persistent and unresolving form and the diagnosis is reached when no other pathological signs except LR are found.⁽¹⁻²⁾

To diagnose this situation, the most important part

of the workup is to exclude any underlying pathology. The laboratory studies directed by history and physical examination are sufficient in evaluation for most of the cases. This process involves a detailed anamnesis with information regarding the use of medication, time of onset, associated symptoms, current diseases and recent surgeries. The physical examination is extremely important, as well as laboratory tests and histopathological examination that may suggest the etiology.⁽⁶⁾

Although LR is a relatively common condition, what proved interesting about our case is that it was related to strenuous exercise. Exercise is recommended for the treatment of LR because increased circulation helps to dilate the blood vessels throughout the body.⁽⁷⁾ However, it is controversial as in this case exercise was what exacerbated the situation. In this context; in the absence of an underlying cause or trigger it would be useful to consider that the condition may be associated with exercise in individuals presenting with similar complaints to a primary care physician.

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Table 1: Etiology of Livedo Reticularis

1. LR without systemic association

a. Physiological LR (*cutis marmorata*)

b. Primary LR

c. Idiopathic LR

- i. Congenital - Cutis marmorata telangiectatica congenita (CMCT)
- ii. Transplacental transient vasculitis
- iii. With ulceration in winter
- iv. With ulceration in summer

2. LR with systemic association

a. Hematological/Hypercoagulable

- i. Stasis
 1. Paralysis
 2. Myocardial infarction
- ii. Occlusive Disease
 1. Thromboembolism
 2. The bends (nitrogen bubbles)
 3. Cholesterol embolism
 4. Oxalosis
 5. Thrombophilias
- iii. Antiphospholipid syndrome
- iv. Sneddon's syndrome
- v. Increased Blood Viscosity
 1. Cryoglobulinemia
 2. Cryofibrinogenemia
 3. Monoclonal gammopathy
 4. Cold agglutinins
 5. Polycythemia rubra vera

6. Hypergammaglobulinemia

7. Thrombocytemia

- vi. Protein C/S deficiency
- vii. Antithrombin III deficiency
- viii. Thrombotic thrombocytopenic purpura
- ix. Disseminated intravascular coagulation
- x. Hemolytic uremic syndrome

b. Autoimmune

- i. Small vessel vasculitis
- ii. Medium vessel vasculitis
- iii. Cutaneous polyarteritis nodosa
- iv. Rheumatoid vasculitis
- v. Wegener's granulomatosis
- vi. Polyarteritis nodosa
- vii. Microscopic polyangiitis
- viii. Nodular vasculitis
- ix. Thromboangiitis obliterans
- x. Large vessel vasculitis
- xi. Takayasu arteritis
- xii. Temporal arteritis
- xiii. Livedoid vasculitis
- xiv. Arteriosclerosis
- xv. Arteritis of pancreatitis

c. Connective tissue disease

- i. Dermatomyositis
- ii. Systemic lupus erythematosus
- iii. Systemic sclerosis
- iv. Still's disease
- v. Sjögren's disease

Table 1: Etiology of Livedo Reticularis

- vi. Sharp's syndrome
- d. Neoplasia**
 - i. Renal cell carcinoma
 - ii. Inflammatory breast cancer
- e. Lymphoma**
 - i. Acute lymphocytic lymphoma
 - ii. Mycosis fungoides
 - iii. Angiotrophic lymphoma
- f. Infections**
 - i. Tuberculosis
 - ii. Syphilis
 - iii. Leprosy
 - iv. Hepatitis C
 - v. Mycoplasma pneumonia
 - vi. Brucella
 - vii. Coxiella burnettii
 - viii. Parvovirus B19
 - ix. Meningococemia
 - x. Streptococemia
 - xi. Rickettsia
 - xii. Rheumatic fever
 - xiii. Typhus fever
 - xiv. Endocarditis
 - xv. Nonspecific viral infection
- g. Neurological**
 - i. Reflex sympathetic dystrophy
 - ii. Diabetes mellitus
 - iii. Pernicious anemia
 - iv. Apoplexy
- v. Multiple sclerosis
- vi. Encephalitis
- vii. Poliomyelitis
- viii. Parkinson's disease
- ix. Brain injury
- x. Divry-Van Bogaert syndrome
- h. Endocrinological**
 - i. Hyperparathyroidism (hypercalcemia)
 - ii. Calcifixia
 - iii. Hypothyroidism
 - iv. Cushing's disease
 - v. Carcinoid syndrome
 - vi. Pheochromocytoma
- i. Nutritional**
 - i. Pellegra
- j. Medications**
 - i. Amantadine
 - ii. Bromocriptine
 - iii. Minocycline
 - iv. Pentazocine
 - v. Gemcitabine
 - vi. Pyridylidione/diphenhydramine
 - vii. Catecholamines (phenylephrine)
 - viii. Quinidine
 - ix. Bismuth
 - x. Erythromycin-lovastatin interaction
 - xi. Aspheramine
 - xii. Interferone beta
 - xiii. Thrombocytic
 - xiv. Nonsteroid antiinflammatory drugs

Table 2: Laboratory findings

Puan		
Complete Blood Count	WBC	7370/ μ L
	Hemoglobin	12,4 g/dL
	Hematocrit	37,2 %
	MCV	80 fL
	MCH	26,6 pg
	PLT	182000/ μ L
Biochemical Parameters	Na	141 mg/dL
	K	4,1 mg/dL
	Ca	9,2 mg/dL
	ALT	18 IU/L
	AST	16 IU/L
	ALP	69 IU/L
	BUN	13 mg/dL
	Cr	0,83 mg/dL
	LDL	111 mg/dL
	VLDL	25 mg/dL
	HDL	60 mg/dL
	Total cholesterol	196 mg/dL
Coagulation	Prothrombin time	11,9 sec
	Activated partial thromboplastin time	34,7 sec
Hormones	TSH	1,7852uIU/mL
	T4	1,06 ng/dL
Romathological Parameters	Antiphospholipid IgG	9,29 U/mL
	Antiphospholipid IgM	5,83 U/mL
	RF	8 IU/mL
	CRP	1,02 mg/dL

*WBC: White blood cell, MCV: Mean corpuscular volume, MCH: Mean hemoglobin volume, PLT: Platelet, Na: Sodium, K: Potassium, Ca: Calcium, ALT: Alanine aminotranferase, AST: Aspartate transaminase, BUN: Blood urea nitrogen, Cr: Creatinine, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein, HDL: High density lipoprotein, TSH: Thyroid stimulating hormone, T4: Thyroxine, IgG: Immunglobulin G, IgM: Immunglobulin M, RF: Rheumatoid factor, CRP: C-reactive protein



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