

Identity Loss Due to Chronic Fingertip Ischemia

JOSE C. CRISPIN, MARGARITA FALLENA, and ARNOLDO KRAUS

ABSTRACT. We describe a young woman with systemic lupus erythematosus and secondary Raynaud's phenomenon who lost her fingerprints as a consequence, we suspect, of chronic ischemia. This is the first time this unusual manifestation of Raynaud's phenomenon is reported. We discuss the hypothetical etiopathogenic mechanisms that underlie this condition and place special emphasis on the psychological and philosophical effect it had on our patient. (*J Rheumatol* 2004;31:1222-4)

Key Indexing Terms:

SYSTEMIC LUPUS ERYTHEMATOSUS RAYNAUD'S PHENOMENON FINGERPRINT

Raynaud's phenomenon (RP) is a clinical disorder that includes recurrent, episodic vasospasms of fingers and toes, often associated with exposure to cold. In most instances it appears unrelated to any underlying disease and is labeled primary RP (formerly Raynaud's disease). When part of a systemic illness, it is regarded as secondary^{1,2}. The pathophysiologic events that lead to this disorder are poorly understood. Multiple vascular and non-vascular factors have been proposed to play a role³. Treatment focuses on the underlying process (when possible), and on supportive measures. The latter include non-pharmacological lifestyle modifications and vasodilator therapy⁴.

When RP accompanies a systemic rheumatic disorder, it is usually more severe than the primary form. In these instances, it may be associated with mutilation of fingertips or even distal phalanges due to intense and sustained vasoconstriction.

This report describes an interesting and uncommon situation: a patient with systemic lupus erythematosus (SLE) and secondary RP who lost the digital skin ridges, i.e. her fingerprints, as a consequence, we suspect, of persistent RP.

CASE REPORT

This patient's disease began in 1988 when she was 18 years old with the appearance of arthritis, fever, serositis, leuko-lymphopenia, and mucocutaneous manifestations. Antinuclear antibodies were positive. Disease remission was achieved after 4 months of treatment with prednisone and nonsteroidal antiinflammatory drugs. She remained in good health until 1993 when she developed vasculitis in her hands and severe RP. Prednisone was prescribed, and azathioprine, nifedipine, and transdermal nitroglyc-

erine were added; nevertheless, she lost a distal phalanx. Disease activity was controlled and prednisone was gradually tapered.

Since then, no disease activity was detected but mucocutaneous manifestations and occasionally low intensity flares of vasculitis (always in the hands) occurred. Her RP, although not very severe, has never been completely controlled, independent of climate and the use of gloves and in spite of treatment with cyclophosphamide, hydroxychloroquine, and low dose prednisone.

In March 2002, she tried to renew her Mexican voters' registration card, which requires the fingerprint of the index finger (Figures 1 and 2). The computer was unable to obtain any of her dactylograms. Her previous identification was issued in 1996. At that time, no abnormalities were found.

DISCUSSION

When RP presents as a manifestation of systemic sclerosis and related conditions (such as SLE or mixed connective tissue disease) it has a worse prognosis¹. In scleroderma, in addition to RP, vascular abnormalities that produce chronic tissue ischemia play an important role in the development of acral cutaneous manifestations (sclerodactyly, digital ulceration, ischemic digital amputation). Although our patient has SLE, she has had vasculitis and severe RP that have produced acral ischemic changes similar to those observed in patients with systemic sclerosis. As far as we know, there is no information about fingerprint loss due either to vasculitis, RP, or SLE⁵⁻⁸. It is reasonable to postulate that atrophic skin changes secondary to chronic ischemia caused by the protracted RP, in the setting of vascular damage due to repetitive vasculitic events, were the cause of fingerprint loss.

Our patient lost a distal phalanx and is dependent on medicines. Nevertheless, the loss of her fingerprints worries her most. She feels something personal and unique has been taken away. This is an awkward and perhaps contradictory circumstance: while we consider the vanished fingerprints an uncommon yet irrelevant finding, our patient feels mutilated.

Disease awaits. Any given day it appears revealing our weakness and fears. Some fade away; most do not. Unfortunately, we lack curative therapy for most diseases

From the Departamento de Inmunología y Reumatología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubiran, Mexico City, Mexico.

J.C. Crispin, MD; M. Fallena, MD; A. Kraus, MD.

Address reprint requests to Dr. A. Kraus, Departamento de Inmunología y Reumatología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubiran, Mexico 14000, Tlalpan D.F. Mexico City.

E-mail: akraus@quetzal.innsz.mx

Submitted February 21, 2003; revision accepted September 11, 2003.



Figure 1. Fingers of the patient's right hand. Absence of skin ridges, along with other atrophic skin changes due to chronic ischemia are prominent.



Figure 2. A closer view of the left hand fingers shows absence of the distal phalanx of the fourth finger along with atrophic skin changes.

and commonly our goals are modest: control of progression, treatment of complications, etc. Hence, chronic illnesses become a part of the life of our patients. Uninvited guests, they slowly modify behavior, lifestyle, and physical aspect. How deep can they reach and how much can they modify?

In the clinical setting, the subjective point of view of the patient may become an uncomfortable and conceivably even annoying element. Sometimes it is difficult to know how much weight to give to their complaints. The mind is a private, hidden, internal, unequivocal subjective entity.⁹ Sense of self is extremely important in a person's life. Losing it may be devastating: it can lead to serious consequences, even suicide. It is simple to imagine this scenario in a person who has been mutilated. However, a careful look reveals that patients with subtle physical modifications, with common diseases, live through similar experiences, perhaps to a lesser degree, all the time.

What must we do, as physicians, concerning these issues? Unfortunately, the magic of the patient-physician relationship is menaced and many doctors treat only the disease and leave its psychological consequences to the patients to deal with. Although there are no straightforward answers to these issues, we should at least think about them

and keep in mind that sometimes patients worry about different things, about aspects of their disease that may pass totally unnoticed by us.

REFERENCES

1. Block JA, Sequeira W. Raynaud's phenomenon. *Lancet* 2001;357:2042-8.
2. Wigley FM, Flavahan NA. Raynaud's phenomenon. *Rheum Dis Clin N Am* 1996;22:765-81.
3. Turton EPL, Kent PJ, Kester RC. The aetiology of Raynaud's phenomenon. *Cardiovasc Surg* 1998;6:431-40.
4. Belch JFF, Ho M. Pharmacotherapy of Raynaud's phenomenon. *Drugs* 1996;52:682-5.
5. Yell JA, Mbuagbaw J, Burge SM. Cutaneous manifestations of systemic lupus erythematosus. *Br J Dermatol* 1996;35:355-62.
6. Cardinali C, Caproni M, Bernacchi E, Amato L, Fabal P. The spectrum of cutaneous manifestations in lupus erythematosus. The Italian experience. *Lupus* 2000;9:417-23.
7. Kapadia N. Cutaneous manifestations of systemic lupus erythematosus: study from Lahore, Pakistan. *Int J Dermatol* 1996;35:408-9.
8. Vormittag W, Weninger M, Scherak O, Kolarz G. Dermatoglyphics and systemic lupus erythematosus. *Scand J Rheumatol* 1981;10:296-8.
9. Damasio AR. How the brain creates the mind. *Scientific Am* 1999;280:74-9.