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Case report

Raynaud's phenomenon associated with nitric acid: Case report☆

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ABSTRACT

The Raynaud phenomenon is a clinical disorder, characterized by episodic attacks of vasospasm of peripheral arteries and arterioles, causing tissue ischemia of distal portions of the body, such as hands and feet, causing changes in skin color such as pallor, cyanosis and hyperemia. The occurrence of the Raynaud phenomenon in people who handle organic solvents or vibratory tools has been associated for a long time, and it has even been described that the solvents may be triggering some immune diseases such as scleroderma, however, as far as we know there are no reports of exposure of chemical compounds used in metallurgy, such as nitric acid and the development of scleroderma-spectrum diseases. This article presents a clinical case related to the appearance of Raynaud's phenomenon against exposure to chemical compounds used in metallurgy, especially nitric acid.

Fenómeno de Raynaud asociado a ácido nítrico: reporte de un caso

Palabras clave:
Fenómeno de Raynaud
Ácido nítrico
Solventes orgánicos
Capilaroscopia
Lecho ungueal
Enfermedades de espectro esclerodérmico
Patrón esclerodérmico

RESUMEN

El fenómeno de Raynaud es un trastorno clínico, caracterizado por ataques episódicos de vasoespasmo de arterias y arteriolas periféricas, causando así isquemia tisular de porciones distales del cuerpo, como manos y pies, produciendo cambios en la coloración de la piel como: palidez, cianosis e hiperemia. Se ha asociado la aparición del fenómeno de Raynaud en personas que manipulan solventes orgánicos o herramientas vibratorias por un largo período, incluso se ha descrito que los solventes pueden ser gatilladores de algunas enfermedades inmunológicas como la esclerodermia, sin embargo, hasta donde conocemos no existen reportes de la exposición de compuestos químicos utilizados en la metalurgia, como el ácido nítrico y el desarrollo de enfermedades de espectro esclerodérmico. En el presente

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artículo se presenta un caso clínico relacionado con la aparición de fenómeno de Raynaud frente a la exposición a compuestos químicos utilizados en la metalurgia, en especial al ácido nítrico.

Introduction

The Raynaud phenomenon (RP) was described for the first time in the thesis of Maurice Raynaud, in 1962, as local ischemia of the hands, feet, nose and tongue, that may be accompanied by pain and which was due to obliteration of the vessels; he also described that this mechanism was associated with the microvascular commitment due to a defective innervation that leads to a loss of vascular tone.[1]

Hutchinson described the difference between the primary and secondary RP in different diseases,[2] Brown and O'Leary demonstrated in 1965 the capillaroscopic abnormalities in patients with diagnosis of scleroderma who presented RP.[3]

Currently, the RP is defined as an episodic ischemic disorder in the fingers and toes, manifested by pallor, cyanosis and redness or the skin in response to stimuli such as cold or emotional stress.[4] The pathophysiology of RP is not well defined, however, it has been established that it may be primary or secondary to a number of underlying clinical entities such as scleroderma. Among the possible pathophysiological mechanisms of RP there are three: vascular, neural and intravascular, described below:

A. *Vascular*: it involves the structure and function.
   - Structural abnormalities: it is believed that there is a release of adhesion molecules, cytokines and growth factors, apoptosis of endothelial cells and activation of pericytes, which generate thickening of the vascular wall and decreased flow.[5]
   - Functional abnormalities: endothelial damage is responsible for the functional alteration, due to the imbalance between vasodilation and vasoconstriction.[6]
B. *Neural*: it involves the deficiency of vasodilators (calcitonin gene-related peptide), activation of alpha-2-adrenoreceptors and components of the central nervous system.[7,8]
C. *Intravascular*: it involves abnormal platelet activation, altered fibrinolysis, activation of white blood cells, increased blood viscosity and oxidative stress.[7]

The exposure to organic solvents has been associated with the development of diseases of scleroderma pattern.[9] The relationship with chlorinated solvents has been well established as a risk factor for the development of scleroderma and, generally, they are occupational exposures,[10] however, there is no evidence of the association between exposure to chemicals used in metallurgy (nitric, sulfuric, phosphoric, hydrofluoric acids, sodium chloride, etc.) and the development of scleroderma-spectrum diseases. For this reason, we want to make known the clinical case of a patient who presented diffuse manifestations of scleroderma spectrum, after a prolonged occupational exposure to chemicals used in the detection and screening of gold, also called Pickling.

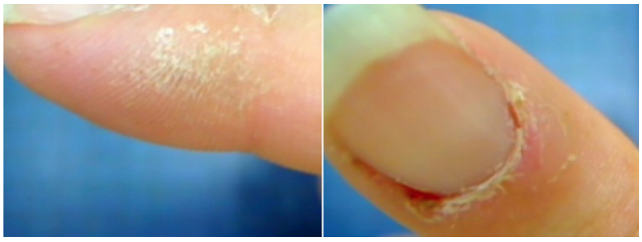

Fig. 1 – Scaly lesions on the lateral and distal part of the fingers and presence of cuticulitis.

Clinical case

A 36-year-old female patient who attends the outpatient clinic or Rheumatology for presenting poorly defined generalized pain, sleep disorders, RP and arthralgia in the hands. She does not report personal or family pathological antecedents, and she consumes tobacco and alcohol occasionally.

She stated that her occupation is metallurgy, focused on gold screening with nitric acid and sodium chloride; she has been carrying out this activity for approximately 24 months. The patient refers changes of coloration in the hands that were triggered since the beginning of the contact with these substances despite she was using latex gloves.

On physical examination she presented hypersensitivity of 18/18 fibrositic points, RP, hypersensitivity in proximal interphalangeal joints without articular tumefaction.

Scaly lesions were evident at the level of the fingers of the hands (Fig. 1), complementary laboratory studies (Table 1) and a videocapillaroscopy were requested. Two weeks later she returns to consultation because the generalized pain has increased; the physical examination showed tumefaction of the metacarpophalangeal and proximal interphalangeal joints (Fig. 2), accompanied by hypersensitivity in carpal joints and elbows, and also continued with all positive fibrositic points.

The patient reports that during the period between the first and the second visit she had palpebral edema on a trip to a cold weather area.

The videocapillaroscopy showed morphological alterations: decreased capillary density, criss-crossed and tortuous arborified capillaries, giant capillaries (72.05 μm, 80.43 μm), microhemorrhages, avascular zones and deposition of unidentified material; the capillaroscopy was interpreted as