

SECONDARY RAYNAUD PHENOMENON IN WORKERS EXPOSED TO LOCAL VIBRATIONS

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ABSTRACT

Forty-one workers (28 males and 13 females) with secondary Raynaud phenomenon were studied. They were divided into two groups: group one - 28 workers exposed to vibrations and group two - 13 workers with overstrain and microtraumatism. The professional route, electroneurography, vegeto-vascular state, cryoglobulins, and erythrocytic antibodies were followed-up. The clinical diagnosis of vibration disease was proved in 24 workers (or 58,5 per cent of the cases). Immunohematologically, erythrocytic antibodies were found out in 13 of these patients. The relationship between the high percentage of erythrocytic antibodies and the secondary Raynaud phenomenon with the workers exposed to local vibrations was discussed.

Key words: Raynaud phenomenon, vibration disease, electroneurography, immunohematology, vibrational white fingers, workers

INTRODUCTION

Raynaud syndrome is characterized by episodic painful turning pale of the fingers because of a peripheral arterial spasm. It relates with an increased arterial constriction during exposure to cold or in emotional stress. This syndrome can also be caused by long-lasting action of vibrations, drugs inflicting vascular spasm such as ergotamin, cytotoxic drugs, and beta-blockers, and by the presence of circulating cryoglobulins or cold agglutinins. When Raynaud syndrome accompanies a connective tissue disease an immunomodulated local defect of the histaminergic vasodilatative system is suspected (5).

In 1911 Giovanni Loriga, an Italian physician, first describes cases with episodes of shivering and frozen and pale fingers and hands both in stonecarvers and drillers in quarries for marble production after usage of pneumatic hack-hammers.

In 1918, Alice Hamilton reports that 89,5 per cent out of a total of 38 stonemasons from Bedford, IN, USA experienced attacks of turning pale of their fingers defined as "spastic anemia affecting the arterioles of the fingers and hands" resembling the digital ischemic phenomenon described by the French physician Maurice Raynaud in 1862.

There exist specific differential-diagnostic criteria between the primary and secondary Raynaud phenomena.

Primary Raynaud phenomenon

- Periodic vasospastic attacks of turning pale or cyanosis

- 2. Capillaroscopically, nail-fold capillaries of normal shape
- 3. Negative ANA
- 4. Normal erythrocyte sedimentation rate
- 5. Absent trophic skin alterations such as wounds and ulcers or gangraena of fingers and toes.

Secondary Raynaud phenomenon

- 1. Periodic vasospastic attacks of turning pale or cyanosis
- 2. Capillaroscopically, nail-fold capillaries of abnormal shape
- 3. Positive ANA
- 4. Abnormal erythrocyte sedimentation rate
- 5. Present trophic skin alterations such as wounds and ulcers or gangraena of fingers and toes.

Occupational exposure to vibrating manual instruments can cause vasospastic disturbances of the fingers, the so-called vibration-evoked white fingers (VWF). Nosologically, VWF is considered a secondary form of Raynaud phenomenon. The paroxysmal turning pale of the fingers resulting from an episodic spasm of their blood vessels is commonly provoked by emotional stress and cold influences (4,6-9).

The pathogenesis of Raynaud phenomenon remains not clarified yet. The following mechanisms have been suggested: either the central sympathetic reflex (irritation of Pachini's bodies similar to the sensory receptor), the vasoconstrictive peptid endothelin 1, deficiency of the vasodilator calcitonin-liberating peptid, C- and S-proteins, enhanced sensitivity and/or concentration of alpha₂-adrenoreceptors, activity of serotonergic receptors, or the combination of them are used to explain the essence of the primary changes of VWF (1). The physiology of distal microcirculation is a complex of individual and professional peculiarities in the secondary Raynaud phenomenon.

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The purpose of the present study is to analyze the abnormalities of the immunological parameters in relation with the specific pathogenetic mechanisms in the professional secondary Raynaud phenomenon.

MATERIAL AND METHODS

The study covered 41 industrial workers from Varna divided into two groups. Group one consisted of 28 workers, 24 males and 4 females exposed to local vibrations, and group two - of 13 workers, 4 males and 3 females exposed to overstrain and microtraumatism. The following parameters were followed-up: professional route, subjective complaints, vegetovascular state, electroneurography, cryoglobulins, and erythrocytic antibodies.

RESULTS AND DISCUSSION

Concerning the subjective complaints it has to be noted that the paraesthetic pains in the palm and fingers aggravating by night and after cold exposure prevail in both workers' groups reaching up to 89,3 per cent of the cases in the first group but even to 92,3 per cent of the cases in the second one. The paroxysmal turning pale of the distal phalanges of one or several fingers is observed in 46,4 per cent and in 7,7 per cent of the cases in the first and second group, respectively.

Skin hypothermia and skin temperature restoration of vasoconstrictive type can be established in 93 per cent of the cases in the first group but in 76,9 per cent of the cases in the second one. There are electroneurographic data about a predominant lesion of *n. medianus* and *n. ulnaris* of demyelination type and prolonged distant latent time of M-response for both nerves in 78,6 per cent of the workers in group one but in 53,8 per cent of the workers in group two.

Positive cryoglobulins are detected in 17,9 per cent of the workers in group one but in 15,4 per cent of the workers in group two. There are positive iso- and autoerythrocytic antibodies at 4°C in 35,7 per cent of the cases in group one but in 61,5 per cent of the cases in group two.

The frequency of subjective complaints such as paraesthetic pains in the fingers corresponds to the factual skin hypothermia and vasoconstriction during the cold stress determined by the dominating central sympathetic mechanism. Low occurrences of "dead fingers" in the workers from the first group relate with slight and moderate forms of vibration disease. The damage is more manifested in this patients' contingent and consists of an abnormal vasoconstriction leading to peripheral nervous and skin dystrophias. The immunological changes presenting with positive cryoglobulins and iso- and autoerythrocytic antibodies in both groups are, probably, jointly involved in the pathogenetic mechanism.

These alterations seem to be mainly of functional nature in spite of the structural changes of the vascular wall described in histological investigations of finger arteries in patients with attacks of "dead fingers" (7). There are, possibly, no obstructive organic lesions of finger arteries in the slight and moderate forms of VWF. Epidemiological data about the reversibility of the symptom of "dead fingers" after cessation of the exposure to vibrations support the viewpoint that the attacks of "dead fingers" represent functional disorders only, at least at the early stages when a transitory increasing of the vascular tone can be found out (2-4,8). The long-lasting exposure to vibrations combined with other harmful factors such as muscular overstrain and cold influences could cause reiterating attacks of "dead fingers" resulting from abnormal vasoconstriction that leads to persistent vascular changes and irreversibility of the symptoms in some cases with VWF. Based on our results we could draw the following conclusions:

1. The skin hypothermia established in 93 per cent of the workers exposed to vibrations is an expression of generalized vascular spasm.
2. The effect of the occupational factors is accompanied by immunological disturbances involved in the mediatory pathogenetic mechanism of Raynaud phenomenon.
3. The low relative share of "dead fingers" is, probably, determined by the irreversible alterations in the vascular wall.
4. The presence of iso- and autoerythrocytic antibodies at 4°C is the dominating symptom in the patients with overstrain-induced diseases.

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