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STUDY OF PERIPHERAL NERVE AFFECTION IN RAYNAUD'S PHENOMENON

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By

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INTRODUCTION

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RAYNAUD'S PHENOMENON (RP)

In 1862, Maurice Raynaud, a French clinician recognised that some people who were exposed to cold temperature had transient digital ischaemia, that he ascribed to an exaggerated response of central nervous system.[1,2]

The term "Raynaud's phenomenon" is now used to describe these episodic events, which represent vasoconstriction of digital arteries, precapillary arterioles, and cutaneous arteriovenous shunts.[3]

The ischaemic phase of the attack is evidenced by demarcated pale or cyanotic skin limited to the digits. It typically starts in one or several digits after exposure to the cold or a stressful situation and then spreads symmetrically to all fingers of both hands, the attack usually ends with a rapid reflow of blood into the digit, which is manifested by erythematous skin (reactive hyperemia).[4]

In over 80% of patients with Raynaud's phenomenon who are seen, the condition is simply an exaggeration of physiologic response to cold temperature[5]. However, it can also represent a clinical manifestation of a serious underlying disease or be the first sign of critical ischaemic of a digit or limb. Depending on the severity of the vascular insult and the size of the vessel involved, superficial ulceration or deep tissue necrosis with gangrene and amputation can result[6].

Patients who have had Raynaud's phenomenon alone for more than two years and have not developed any additional manifestations are at low risk for developing an autoimmune disease. Most of these patients are considered to have primary Raynaud's phenomenon or Raynaud's Disease.[7]

Classification of Raynaud's Phenomenon[8]

I)According to cause

1) Raynaud's disease "primary Raynaud's phenomenon" occurs as a primary disease of unknown cause now the term of primary Raynaud's phenomenon is more convenient to rule out confusion.

2) Raynaud's syndrome "secondary Raynaud's phenomenon" occurs secondary to connective tissue disease, such as scleroderma, medication or vasospastic diseases.

II) Mechanistic classification of Raynaud's phenomenon

A. Vasospastic:

1. Primary Raynaud's phenomenon.
2. Drug induced:

- Ergot.
- Methysergide .
- β-adrenergic blockers.

3. Pheochromocytoma .
4. Carcinoid syndrome.
5. Primary with other vasospastic syndromes (Migraine, variant angina).

B. Structural

1. Large and medium sized arteries.

- Thoracic outlet syndrome.
- Crutch pressure .
- Brachiocephalic trunk disease (Takayasu's, atherosclerosis).

2. Small arteries and arterioles.

- Vibration disease .
- Arteriosclerosis and thromboangitis obliterans.
- Cold injury (frost bite).
- Polyvinyl chloride disease.
- Chemotherapy (bleomycin, vinblastine).
- Connective tissue disease (systemic sclerosis, systemic lupus erythematosus, inflammatory myopathy, overlap syndromes).

C. Hematologic and haemorrheologic causes:

- Cryoglobulinemia
- Cryofibrinogenemia
- Paraproteinemia and hyperviscosity syndromes.
- Cold agglutinin disease.
- Polycythemia (essential thrombocythemia-polycythemia rubra vera).

Vascular biology and pathophysiology of Raynaud's phenomenon

Vasomotor homeostasis is maintained by complex interactions between endothelium, smooth muscles, autonomic and sensory nerves that innervate the vessels.[9]

Regulation is mediated via

1. Endothelium-dependent vasoregulation.
2. Endothelium-independent vasoregulation.
3. Non vascular factors.

I.Endothelium dependent vasoregulation

It is now clear that the vascular endothelium and platelets are major contributors to the control of vascular tone. Endothelial cells are intimately involved in the regulation of vascular tone via the synthesis and release of cytokines, growth factors, prostaglandins, and other bioactive macromolecules.[10,11]

These include mediators of vasodilation (endothelin -1). Furthermore, endothelial cells release neurotransmitters, including substance P and acetylcholine.[11]

In Raynaud's phenomenon varied plasma endothelin concentrations and reduced endothelial release of nitric oxide suggest that endothelial-dependent vasoregulation could play an important role in its pathogenesis