Post-Traumatic Raynaud’s Phenomenon Following Volar Plate Injury

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ABSTRACT
Post-traumatic Raynaud’s phenomenon following non-penetrating or non-repetitive injury is rare. We report a case of Raynaud’s phenomenon occurring in a single digit 3 months following volar plate avulsion injury. Daily episodes of painless pallor of the digit occurred for 1 month upon any exposure to cold, resolving with warm water therapy. Symptoms resolved after the initiation of hand therapy, splinting, and range-of-motion exercises.

KEYWORDS: Raynaud’s phenomenon, Volar plate injury, Raynaud’s syndrome

INTRODUCTION
Raynaud’s phenomenon is a condition in which a digit experiences episodic vasospasm, producing sharply demarcated pallor, coolness, paresthesias, and numbness in the digit distal to the affected vessels. The episodes are typically induced by exposure to cold temperatures and may represent an exaggerated cold response in the affected digit. Diagnosis of Raynaud’s relies solely on physical exam and history.

Raynaud’s phenomenon involves an increased vascular contractile response to sudden cooling and alpha-2-adrenergic agonists, which is particularly pronounced in the acral body parts because of their major thermoregulatory requirements. The condition may be idiopathic or secondary to an underlying pathology. In idiopathic Raynaud’s, also called primary Raynaud’s phenomenon, patients are typically female, in their second or third decade of life. When an underlying disorder is identified as the cause of Raynaud’s phenomenon it is called secondary Raynaud’s phenomenon.

The most common causes of secondary Raynaud’s phenomenon are systemic rheumatic disorders, such as scleroderma or systemic lupus erythematosus. Raynaud’s phenomenon secondary to scleroderma, which is the most studied presentation, involves a vasculopathy consisting of diffuse intimal fibrosis, activation of smooth muscle cells, and endothelial cell perturbations. Endothelial cell autoantibodies have been found in high frequency in scleroderma patients, and it has been postulated that activation or apoptosis of endothelial cells due to action of these antibodies could lead to the release of vasoconstrictors and decreased production of vasodilators.

Secondary Raynaud’s phenomenon also may occur after trauma, such as after digital replantation or after prolonged repetitive vibration trauma, as in the Vibration White Finger (VWF) syndrome typically seen in construction workers working with vibrating power tools. Raynaud’s phenomenon secondary to these types of trauma appears to be driven by perivascular nerve damage. Loss of calcitonin-gene-related-peptide (CGRP) nerve fibers in digital cutaneous perivascular nerves has been observed in patients with VWF. CGRP is a potent vasodilator and its loss leads to the vaso-regulatory imbalance characteristic of Raynaud’s phenomenon. In VWF, endothelial cells may play an additional role in further driving this misbalance, as it has been shown that oscillatory mechanical forces can induce increased expression and release of endothelin-1, a potent vasoconstrictor, from cultured endothelial cells.

While post-traumatic Raynaud’s phenomenon has been reported to occur after penetrating trauma or prolonged
Interestingly, the onset of symptoms in our patient had a delay of 2 months after the inciting injury, and it resolved promptly with range-of-motion exercises. In general, symptoms of Raynaud’s do not resolve quickly in the majority of patients and the etiology following blunt trauma may involve non-neurogenic factors as well.

Treatment of Raynaud’s syndrome using a dihydropyridine calcium-channel antagonist has been reported to be successful, depending on patient tolerability of the drug.12-14 However, it is preferable to first try conservative measures involving simple risk-factor avoidance, such as smoking cessation and avoidance of cold-exposure in an effort to reduce attack frequency and avoid the need for pharmacological treatment. These more conservative methods are often successful, and pharmacological intervention becomes unnecessary.15-17

In the case of our patient, our initial therapy involved conservative non-pharmacological methods. However, in addition to recommending risk-factor avoidance, we also initiated hand therapy to improve range of motion in the patient’s injured finger. The patient eventually regained full range of motion, as well as complete reduction in finger swelling, after completing the prescribed therapy regimen. Furthermore, at one year following initial presentation, the patient reported that he no longer avoids cold-exposure and was free of Raynaud’s symptoms. This case illustrates the value of a trial of conservative measures before prescribing pharmacological interventions in patients who present with secondary Raynaud’s syndrome from trauma.

References


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