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Review Article

Role of the vascular surgeon in the treatment of secondary Raynaud's phenomenon, literature review and endovascular management proposal

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ABSTRACT

Raynaud´s phenomenon is a very common condition, usually little investigated due to its benign nature, however, when talking about Secondary Raynaud's phenomenon, the situation can become very serious and even compromise the integrity of the extremities, it is then when it is important to know the multidisciplinary team that can and should intervene, as well as the treatment alternatives both medical, surgical and endovascular, this is where the extremely important role of the vascular surgeon in this condition comes in. A search was carried out in the main search engines of medical research (Pubmed, Ebsco host, Clinical key, Cochrane, and Springer Link) focused on the pathophysiology, diagnosis and treatment of Raynaud's phenomenon, finding most of the bibliography in dermatology, rheumatology, plastic surgery and to a lesser extent angiology and vascular surgery. And share the experience we have had in endovascular management and its long-term results. We select about 45 papers from different topics for made this recompilation trying to include the different approaches by specialty and emphasize in the management that the vascular surgeon could offer and present the experience obtained in some patients in our hospital. Secondary Raynaud´s phenomenon can be a mutilating condition that is usually managed exclusively by rheumatology and dermatology, however in selected (complex) cases the intervention of the vascular surgeon can make a difference in the integrity of the limbs and the quality of life of our patients.

Keywords: Raynaud's phenomenon, Secondary Raynaud´s phenomenon, Raynaud´s syndrome, Prostaglandin E1, PGE1, Intraarterial

INTRODUCTION

Raynaud's phenomenon is a widely identified condition, but little known by most medical specialties. Since most of the time it is considered as a relatively frequent, benign and self-limiting condition, however this is not always the case.

Raynaud's phenomenon is first described by the French physician Maurice Raynaud in his 1862 thesis "De l'asphyxie locale et de la gangrène symétrique des extrémités" where he describes the characteristic triad, assuming its origin in a dysregulation in the contraction of the precapillary arterioles caused by a hyperreactive neurological reflex.[1]

It is a condition that affects the microvasculature usually in the fingers, however it can occur in the nose, ears and nipples.[1] The classic three-phase presentation of pallor, cyanosis, and hyperemia occurs only in 33% of primary RF and up to 66% of secondary RF associated with systemic sclerosis.[2]

Defining primary RF

A phenomenon which is not related to any specific condition and is usually benign and self-limiting, generally

symmetrical, without the development of necrosis and patients who are seronegative to ANA.

Secondary RF

It is usually related to or participates in a systemic connective tissue disease, and although it can have a benign course, it can also present from stings, ulcerations to irreversible ischemic changes.

It is an equally distributed disease, with a prevalence of 3-5% of the world's population [1], increased by up to 21% in cold-climate countries.[2]

Its most frequent trigger is exposure to cold, however this reaction can be even with minimal changes (air conditioning) or even in situations of emotional stress, by drugs (b-blockers), chemotherapy (cylophosphamide, vincristine, vinblastine, doxorubicin, bleomycin, 5-fluorouracil), vibration injury, repeated trauma (typing), smoking, and vasculitis.[3]

In 10-20% of cases, Raynaud's phenomenon is the initial manifestation of an association with undiagnosed connective tissue pathologies such as scleroderma, dermatomyositis, systemic lupus erythematosus (SLE), Sjögren's syndrome, and rheumatoid arthritis (RA).

Table 1: Characteristics of primary and secondary Raynaud's phenomenon.

S. no.	Primary Raynaud's	Secondary Raynaud's
1	Symmetrical	Systemic connective tissue disease
2	Stimulus, temperature, or stress	Trophic changes (necrosis)
3	Respect thumbs	Sclerodactyly
4	Young people	Point-like scars on the fingertips
5	No trophic changes	Associated conditions, described below
6	Women 9:1 Men	
7	It can be presented in fingers, tongue, ears, nose, nipples	
8	Associated with migraine	
9	Family predisposition	

Conditions associated with secondary Raynaud's phenomenon

Connective tissue diseases

These include systemic sclerosis, systemic lupus erythematosus, mixed syndromes, sjögren's syndrome, dermatomyositis/polymyositis, and primary biliary cirrhosis.

Occupational

These include hand or arm vibration syndrome and hypothenar hammer syndrome, exposure to vinyl chloride monomer, silica and solvents (causing systemic sclerosis).

Drugs

These include ergotamine-derived antimigraines, non-selective beta blockers (including ophthalmic), some cytotoxicants, cyclosporine (especially in transplant recipients), bromocriptine, interferon alfa and beta, abuse of cocaine, amphetamines or cannabis, estrogen replacement therapy without progesterone, ephedrine, including in otic, nasal and tracheal presentations.

Endocrine

Hypothyroidism and pheochromocytoma.

Paraneoplastic

Miscellaneous

These include Buerger's disease (thromboangiitis obliterans), low body mass index, post-bariatric surgery, complex regional pain syndrome, frostbite sequelae, and digital lesion sequelae.

METHODS

A search was carried out in the main search engines of medical research (Pubmed, Ebsco host, Clinical key, Cochrane, and Springer Link) focused on the pathophysiology, diagnosis and treatment of Raynaud's phenomenon, finding most of the bibliography in dermatology, rheumatology, plastic surgery and to a lesser extent angiology and vascular surgery. He also shares the experience we have had in endovascular management and its long-term results.

RESULTS

Physiopathology

Abnormalities occur in the density and structure of the arteries, arterioles, and capillaries that is associated with the loss of nutritional vasculature of the fingers.[4,5] In addition, larger digital arteries are involved with intimal hyperplasia and fibrosis caused by increased collagen deposition.[6] A histological study of the digital arteries showed a greater than 75% reduction in lumen diameter in 79% of the vessels studied. These vascular changes are not limited to the skin or digital vessels, but can take place throughout the microcirculation of the vascular system; especially in organs such as the heart, lungs, kidneys, and gastrointestinal tract. In addition to exaggerated vasospasm, microthrombi form in the blood vessels, leading to tissue damage. This process is likely related to both platelet activation and impaired fibrinolysis.[7]

Markers of platelet activation have been shown to be found at elevated levels in scleroderma, including thromboxane A2, β-thromboglobulin, serotonin, platelet-derived microparticles, and platelet-derived growth factor.[8]

Diagnosis

This will be based on the patient's clinical symptoms, if the classic three-phase presentation is available, it will be considered a primary Raynaud's phenomenon, however, if the symptoms persist, become chronic or deteriorate due to trophic changes in the patient's condition, the diagnosis of secondary Raynaud's phenomenon should be considered, so the coexistence of other conditions mainly affecting the connective tissue should be ruled out. as noted in the 2017 update of the EULAR, where he points out the consideration of systemic sclerosis as an association with the onset of secondary RF.[9] Within the characteristics or parameters for its diagnosis and clinical identification. Digital pressure and photoplethysmography are used to rule out the obstructive origin. Different publications comment on capillaroscopy of the nailfold as an objective study in the determination of the chronicity of the spastic event.[10-12] Capillaroscopy focuses on observing the morphology, density, and presence or absence of hemorrhages, as described by the study group on EULAR microcirculation.[13]

Diagnostic criteria

The criteria have varied, among those considered most relevant, the following are considered:

Classification criteria based on clinical evaluation; Brennan et al (1993).

Negative

Absence of episodes of color changes (pallor, cyanosis, erythema), or symptoms (paresthesia and numbness) in cold exposure

Positive

Episodes of uniphasic changes (one of; pallor, cyanosis, erythema) and or paresthesias or numbness.

Definitive

Repetitive episodes of biphasic color (at least two of them; pallor, cyanosis, erythema), both in cold and usual weather.

Severe

Repetitive episodes of biphasic color (at least two of them; pallor, cyanosis, erythema), as well as paresthesias or numbness in both cold and usual weather.[14]

Screening questions (Wigley (2002)): are your fingers usually sensitive to cold? do your fingers change color when exposed to cold temperatures? do they change to blue or white, or both? The diagnosis is confirmed if at least one affirmative answer is answered.

If you test positive for Raynaud's phenomenon; more criteria will be evaluated for distinction in primary or secondary.[15]

Criteria for the diagnosis of primary Raynaud's phenomenon (LeRoy et al (1992)): vasospastic attacks precipitated by cold or emotional stress, symmetrical attacks on both hands, absence of tissue necrosis or gangrene, no history or clinical findings of a secondary cause, normal nail fold capillaries, normal erythrocyte sedimentation rate, and negative serology; especially antinuclear antibodies.[46]

Sorting based on color charts and quizzes; Maricq et al (1988).[47]

Questionnaire

The questionnaire includes: are your fingers sensitive to cold? and do your fingers have unusual color changes, and if so, do they change to white, blue, red, or purple?

Negative

No whitening by hand photography or color scale.

Possible

Whitening by photograph and/or color scale, but insufficient to define it.

Definitive

At least 3 of the following: hand photography whitening, color scale whitening, yes to question (a), and yes to question (b).

The current criteria defined in the "International consensus criteria for the diagnosis of Raynaud's phenomenon", published in 2014 in the Journal of Autoimmunity. They promote a 3-step scheme to reach the diagnosis, in addition to the criteria for consideration of primary Raynaud's phenomenon, being by secondary rule out if these criteria are not met (Table 2).

A 3-step approach to diagnosing Raynaud's phenomenon.

Treatment

The therapeutic approach of these patients is mostly established by clinicians in the areas of rheumatology and dermatology, so the focus of most schemes is based on oral medical treatment aimed at controlling the acute event of both Raynaud's phenomenon and the concomitant

pathology. It is only when, despite conservative therapeutic measures, the cessation of the disease is not achieved that the participation of specialties such as vascular surgery, plastic surgery and interventional algology is requested.

Table 2: Diagnostic criteria for primary Raynaud's phenomenon.[1]

S. no.	diagnostic criteria for primary raynaud's phenomenon
1	Meets 3 criteria for Raynaud's phenomenon
2	Normal capillaroscopy (clusters 1 and 2 described as normal or perfectly normal by Ingegnoli et al)
3	Negative physical examination for findings suggestive of secondary causes (ulcerations, tissue necrosis or gangrene, sclerodactyly, calcinosis, or cutaneous fibrosis)
4	No history of connectivity tissue diseases
5	Negative or very low ANA titers (1:40 by indirect immunofluorescence)

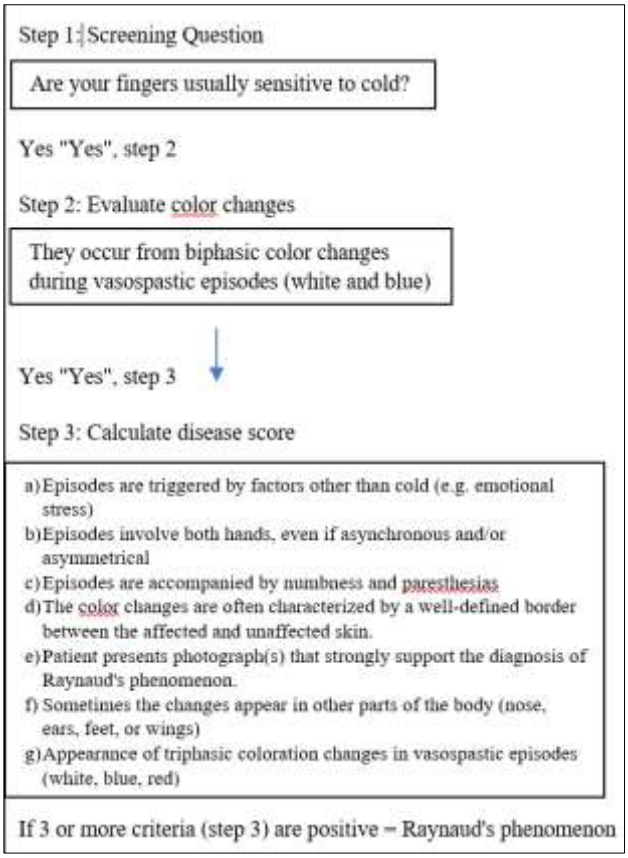

Figure 1: 3-Step approach to diagnosing Raynaud's phenomenon.

Medical treatment

It is the first-line treatment, the most studied and accepted, with a good therapeutic response in most cases. It consists of improving capillary permeability by reducing peripheral vascular resistance and promoting vasodilation.

Calcium channel blockers (dihydropyridine)

First line of treatment, specifically Nifedipine, which is the most commonly used, compared and accepted drug as an effective remedy. Taking advantage of its effect in reducing peripheral vascular resistance to improve distal permeability.[16,17]

The usefulness of Amlodipine, which also belongs to dihydropyridines, has been established. If fault is found with Nifedipine or Amlodipine, it is considered Nicardipine, Phelodipine, Isradipine or Nisoldipine.[10,18]

Reduce attack frequency by up to 66%.[19]

Dosage

Nifedipine 10-30 mg PO every 8 to 24 hours, and Amlodipine: 5-20 mg PO every 24 hours.

Alpha1 adrenergic blockers

They seek their vasodilatory effect by competitive inhibition of sympathetic stimulation resulting from the release of norepinephrine. The usefulness of Prazocine in reducing the frequency of primary and secondary Raynaud's phenomenon attacks has been observed. However, hypotension is the main side effect.[20]

Dose

1 mg PO every 8 hours.

Selective serotonin reuptake inhibitors

Fluoxetine is the most widely known and with a proven vasodilatory effect by inhibiting the vasoconstrictor effect of serotonin, a decrease in the frequency and severity of attacks has been observed in both primary and secondary Raynaud's phenomenon.[21]

Dosage

20-40 mg PO every 24 hours.

5-Phosphodiesterase inhibitors

They cause myrco and macrovascular dilation by inhibition of cGMP. Drugs such as Silfenafil, Tadalafil and Vardenafil have been proposed as part of the management of Raynaud's phenomenon.[22]

Sildenafil

Dosage 50 mg PO every 12-24 hours or 100-200 mg (extended-release) PO every 24 hours.

Tadalafil

Dose 20 mg PO every 24 hours.

Vardenadil

Dose 10 mg PO every 12 hours.

Topical nitrates

Although the effectiveness of symptomatology was found in a randomized controlled study, the application of 2% Nirtroglycerin patches is not as widely accepted because of its side effects such as headaches and hypotension.[23]

Endothelin inhibitors

It seeks inhibition of the potent vasoconstrictor effect of endothelin. Bosentan was evaluated in the RAPIDS-2 study, where it showed a decrease in the incidence of new digital ulcers in patients with scleroderma, but did not demonstrate changes in ulcer healing.[24]

Dose

62.5 mg PO every 12 hours for 4 weeks, then 125 mg every 12 hours for 20 weeks.

Prostaglandins and their analogues

Prostaglandins

In various studies, several effects of medical interest have been demonstrated, such as vasodilation, improvement in cutaneous oxygenation, fibrinolytic activity, antiplatelet activity, and even a decrease in reperfusion injury.[25-29]

The use of epoprostenol has been studied in conjunction with vasodilators and antiplatelets, with significant improvement in temperature and distal flow, however, beneficial effects were observed only after 1 week.[30]

The use of intravenous prostanoids such as Iloprost with healing of ischemic ulcers after infusion for 6 hours for 5 days has also been proposed.[31]

Dosage

Epoprostenol 1-2 ng/kg/min IV.

Dose

Iloprost 0.5-2 ng/kg/min IV for 6 hours for at least 5 days.

The use of IV alprostadil has been described for use in Raynaud's phenomenon since 1983, with multiple assays mainly with lipid-bound molecules (lipo PGE1 or PGE1 cyclodextrin) to limit its degradation at the pulmonary level.[32] With multiple regimens from 10 mcg to 80 mcg for 14 to 21 days.[33]

Gardinali et al. describe the symptomatic improvement decrease in the frequency and severity of Raynaud's phenomenon "attacks" on the reapplication of prostaglandins (PGE1α-cyclodextrin, Prostavasin®) despite a 6-week interval between each "cycle".[34]

Botulinum toxin A

It inhibits the release of neurotransmitters by cleavage of receptor proteins from the soluble NSF-binding protein.

Botulinum toxin A can be injected intradigitally or into the palmar skin, it can reduce the severity of RP, after 6 weeks the temperature of the digital pulp injected with toxin was significantly higher than the control temperatures.[35]

At doses of 50 IU in 2.5 ml.

Surgical and endovascular management

As previously mentioned, after failure to achieve remission with conservative medical management, surgical treatment is currently being considered and endovascular management will be considered next.

Surgical management

Thoracic sympathectomy, although it is rarely used, this sympathectomy has been considered in the surgical context itself (thoracoscopy). It has been considered effective in patients with digital critical ischemia, mainly for pain management.[36]

Distal periarterial sympathectomy

Also described as the stripping of the adventitia of the hand and fingers, it has allowed the epithelialization of digital ulcers and the delimitation in the processes of dry necrosis.[37]

As well as more recently described by Fabiani et al; the combination of distal periarterial sympathectomy (radial and ulnar) with the administration of interdigital botulinum toxin, with good results.[38]

Percutaneous sympathetic block

Buivacaine and mepivacaine blockades have been shown to be effective in the treatment of secondary Raynaud's phenomenon. Infusion at the T2 level showed progress in epithelialization of digital ulcers.[39]

Chemical sympathectomy

By administering botulinum toxin adjacent to digital arteries it has also been proposed as a therapeutic

alternative, as well as injection into the interstitial space around neurovascular junctions.[38,40]

Nerve stimulation

Transcutaneous, it has been observed to induce vasodilation with several results.[41]

Spinal, used for the treatment of chronic intractable pain in the upper extremities, has also been used for the treatment of severe or refractory Raynaud's phenomenon.[42,43]

Amputation

Despite being the worst-case scenario, it continues to be described as part of the treatment of refractory secondary Raynaud's phenomenon, mainly due to the presence of intense pain that is difficult to control, or in necrotic lesions, since, remembering that these are patients who, as part of the control of the underlying disease, are subjected to immunomodulation or immunosuppression that makes them susceptible to developing infection. However, it is also consistent to show that an early amputation (continuing in the acute phase) can condition the extent of ischemic or necrotic changes to the residual limb.

DISCUSSION

The aim of this review is to compile the different treatments available for the treatment of Raynaud's phenomenon, especially secondary Raynaud's phenomenon, which has already been described as having an uncertain and aggressive behavior, a situation in which the intervention of the vascular surgeon may be crucial.[1,2,4-6]

In previous reviews described in the areas of rheumatology, dermatology and vascular surgery, therapeutic management has practically not changed over time, probably due to good results in most cases.[1,2,4-6,16,33,34,44,45]

It is widely described and recognized that medical treatment should always be the initial phase of treatment.[2,4,10,11,16] However, apart from medical management, surgical management is scarce, which gives deficient levels of evidence (level of evidence 3), all management aimed at causing dilation by thoracic sympathectomy, digitalis sympathectomy, periarterial stripping with botulinum toxin, thoracic blocks, botulinum toxin, and nerve stimulation.[16,36-39,40-43]

Of all these surgical or interventional procedures, the vascular surgeon's contribution was only limited to peri-arterial stripping, botulinum toxin injection and amputations.

That is why the experience acquired in our vascular surgery service will be described below, as a new alternative in the intervention of the vascular surgeon in Raynaud's phenomenon.

Endovascular management proposal

The use of prostaglandins is well described for the management of Raynaud's syndrome, but in all studies intravenous administration is considered, with irregular results, intra-arterial administration has been described for other pathologies such as nonocclusive intestinal ischemia (NOMI) and in peripheral arterial disease, with intravenous and intra-arterial administrations being purchased where they found superiority in intravenous administration.[32,33,46,47] However, it should be considered that these studies were carried out in the context of very different etiologically different pathologies.

Next, we will present the experience observed through a series of cases presented in our tertiary center, Hospital General de México "Dr Eduardo Liceaga", where targeted intra-arterial administration was proposed in 14 patients with secondary Raynaud's syndrome.

Justification

Prostaglandins were discovered in 1935 when they were isolated from seminal fluid, have an eicosanoid molecular structure derived from 20-carbon fatty acids and a cyclopentane.[25] It is produced endogenously to cause relaxation of vascular smooth muscle, this agent produces relaxation of the body smooth muscle by binding to PGE (prostaglandin E) receptors, resulting in the activation of adenylate cyclase and the subsequent accumulation of 3.5'-cAMP and thus achieve vasodilation.[25,28] Several medically interesting effects have been demonstrated in several studies, such as vasodilation, improvement in skin oxygenation, fibrinolytic activity, antiplatelet activity, and even a decrease in reperfusion injury.[25,27-29,48]

Up to 80% of circulating alprostadil can be metabolized as it passes through the pulmonary circulation, by β and ω oxidation. Metabolites are mainly excreted by the kidneys, and excretion is essentially completed within 24 hours of administration.[25]

Biological half-life is around 5 to 10 minutes (after a single dose), in healthy adults and newborns. It binds in plasma mainly to albumin (81% bound) and, to a lesser extent, to the IV-4 fraction of alpha-globulin (55% bound)[49].

Considering the above, the administration of intraarterial prostaglandin E1 would prevent its pulmonary metabolization and under the same principle of minimum effective dose of directed thrombolysis; The targeted administration of prostaglandins will allow us to potentiate their vasoactive effects almost exclusively on the target vessels.

In our angiology, vascular and endovascular surgery service of the General Hospital of Mexico "Dr Eduardo

Liceaga", a total of 14 patients were treated without surgical or endovascular treatment, finding significant improvement in the 4 young patients (<30 years old) with underlying rheumatological disease (systemic lupus erythematosus and triple positive antiphospholipid antibody syndrome), who presented ischemic ulcers in the fingers of the upper and lower extremities. Next, the characteristics of 2 patients will be described.

Patient 1

A 24-year-old female odontology student with a previously known diagnosis of systemic lupus erythematosus (SLE) was being monitored and treated by the rheumatology department. Suddenly, she presented cyanotic discoloration changes in the distal phalanges of both hands (acrocyanosis) accompanied by pain, so she came to our hospital, was admitted to the rheumatology service to manage the acute activation of her autoimmune condition with cyclophosphamide and steroids, and they started conventional therapy for the management of secondary Raynaud's phenomenon with nifedipine, sildenafil and physical media (thermal gloves).

The ischemic changes progressed despite the aforementioned treatment, so she was consulted to our department for evaluation and therapeutic complementation, finding irreversible ischemic changes at the cutaneous level, with necrotic areas with cyanotic borders without the presence of capillary filling. The case was convened and hemodynamic room, alprostadil (PGE1) and alteplase was requested. At hemodynamics room, a right femoral puncture was performed and a 0.035" guide was advanced and a multipurpose catheter was advanced to the right subclavian where an initial arteriography was performed, evidencing a decrease in the caliber of the vessels posterior to the wrist joint, a 0.035 guide was changed and a 0.014" guide was advanced with a microcatheter which was advanced to the distal radial artery at the level of the wrist. where 5 mg of alteplase is administered, with subsequent administration of 10 mcg of alprostadil (PGE1) diluted in 20 cc of isotonic saline solution, causing intense pain upon its administration, and immediately evidencing predominant coloration changes in the thenar region, observing a fluctuating marble image at the level of capillary circulation as shown in Figure 2, The same procedure is repeated in the contralateral thoracic limb. Upon leaving the hemodynamics room, he was discharged painlessly, persisted on the floor with intravenous infusion of prostaglandin E1 at a dose of 40mcg in 250cc of isotonic saline solution for 4 hours every 12 hours, for 14 days, with analgesia, anticoagulation with low molecular weight heparin at a prophylactic dose calculated by weight and was discharged home with nifedipine 30 mg every 12 hours. In order to avoid physical means and to avoid temperature changes, follow-up was given by outpatient clinic 1 month after the procedure, finding the complete delimitation of the ischemic process as shown in Figure 3, and in the last follow-up 1 year after the procedure it was observed with an exceptional progression with practically imperceptible scars and functionally without any sequelae as observed in Figure 4.

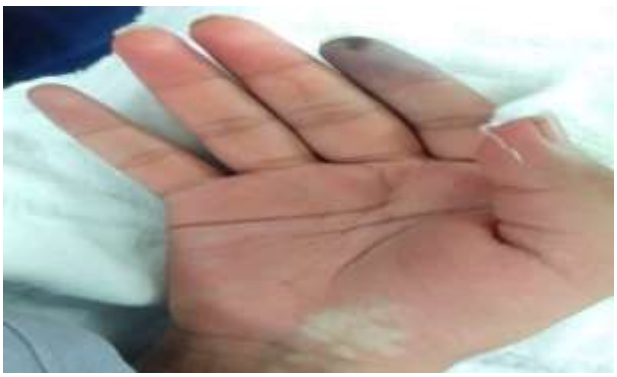

Figure 2: Patient 1 day of targeted PGE1 administration in right radial artery, original unpublished photo (Dr. Carlos Achurra).

Figure 3: Patient 1, 1-month post-treatment, original unpublished photo (Dr. Carlos Achurra).

Figure 4: Patient 1, 1-year post-treatment, original unpublished photo (Dr. Carlos Achurra).

Patient 2

A 29-year-old female with a previous diagnosis of systemic lupus erythematosus was hospitalized for acute attacks and serositis. She presented sudden changes in the

coloration of the left foot, was managed by an external physician with nifedipine, nitroglycerin patch and antiplatelet agents, persisting with painful symptoms and progression of ischemic lesions, she came to our hospital where she was approached by rheumatology and began treatment with steroids and continued medical management for secondary Raynaud's phenomenon with nifedipine and sildenafil and nitroglycerin patch (Figure 5 and 6). who, in view of the progression of the lesions presented, is consulted at our service, a case session is held and is considered a candidate for directed intra-arterial administration of prostaglandin E1, a hemodynamics room is requested, alprostadil and is subjected to diagnostic arteriography by ipsilateral femoral approach, observing a decrease in the caliber of the arteries in the distal third of the anterior tibial artery with little passage of contrast medium in the plantar and interdigital arches, Multipurpose catheter is advanced to the middle third of the anterior tibial artery and 10 mcg of alteplase is infused.

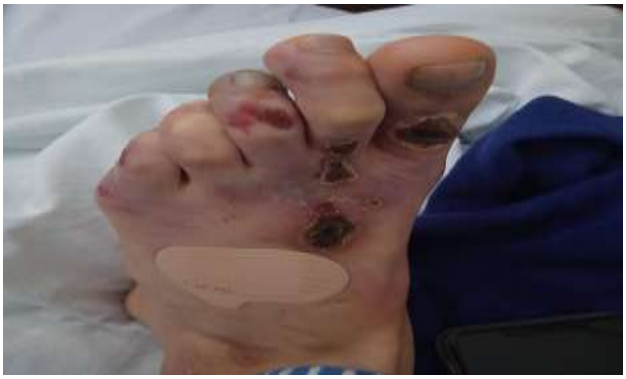

Figure 5: Patient 2, day of hospital admission, original unpublished photo (Dr. Carlos Achurra).

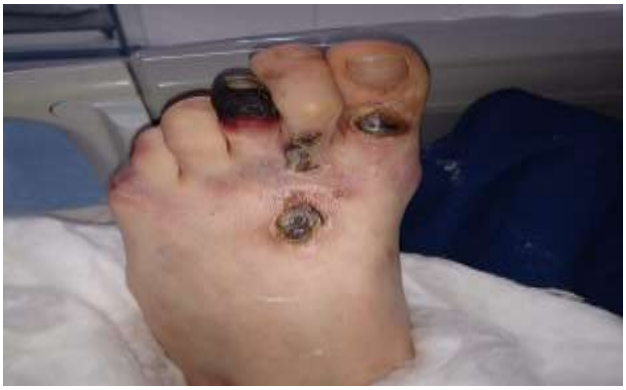

Figure 6: Patient 2, initiation of PGE1 directed to anterior tibial artery, original unpublished photo (Dr. Carlos Achurra).

The patient was discharged to the floor where he persisted on the floor with intravenous infusion of prostaglandin E1 at a dose of 40 mcg in 250 cc of isotonic saline solution for 4 hours every 12 hours, for 14 days, with analgesia, anticoagulation with low molecular weight heparin at a prophylactic dose calculated by weight and was discharged home with nifedipine 30 mg every 12 hours. In order to avoid physical means and to avoid temperature changes, follow-up was given by outpatient clinic with progressive improvement of the symptoms, finding the complete delimitation of the ischemic process and self-amputation of the third finger as shown in Figure 7.

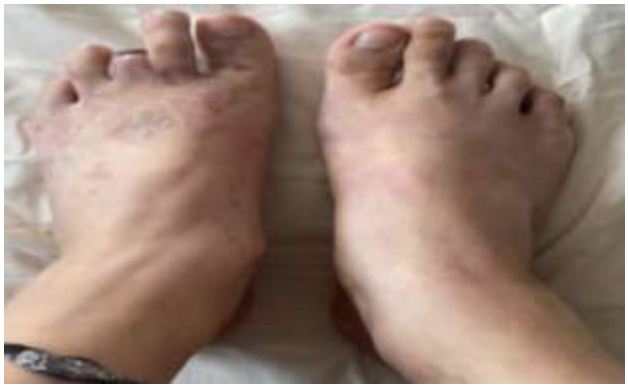

Figure 7: Patient 2, 1 year after treatment, original unpublished photo (Dr. Carlos Achurra).

What was proposed in theory and with few reports on doses and results, achieved excellent results in practice, by achieving pain control, in addition to the limitation of the ischemic process, with excellent results, which has not even recurred 2 years after its treatment.

CONCLUSION

Secondary Raynaud's phenomenon can be a mutilating condition that is usually managed exclusively by rheumatology and dermatology, however in selected (complex) cases the intervention of the vascular surgeon can make a difference in the integrity of the limbs and the quality of life of our patients.

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