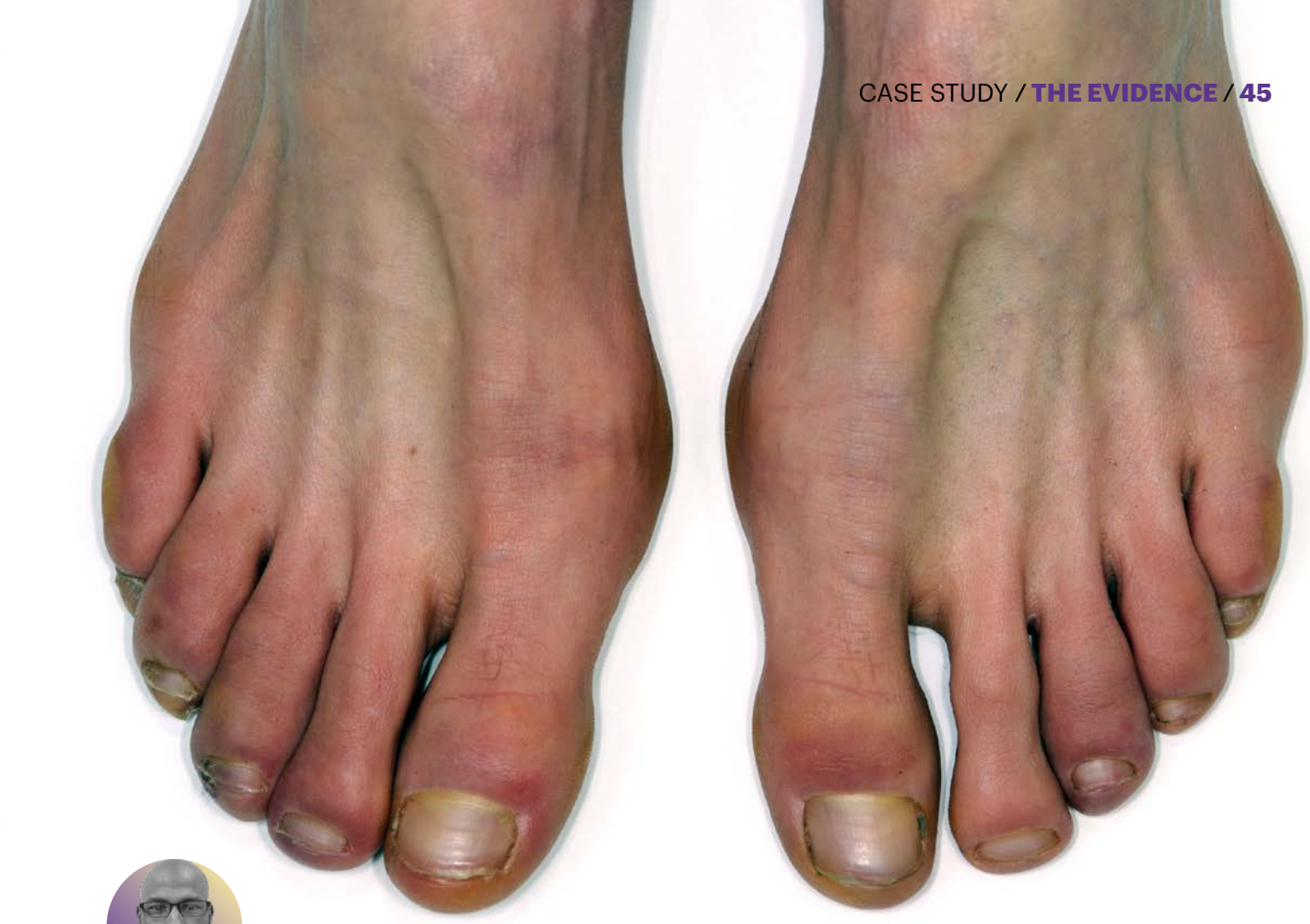

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FCPs and complex care management

First contact practitioner podiatrists (FCPs) could reduce GP and community podiatry workload: a case-based example of secondary Raynaud's phenomenon presentation in primary care.

Introduction

Winter is well and truly upon us, and amid the cold spells and sub-zero temperatures, podiatrists nationwide will undoubtedly be in consultation with patients presenting with new or worsening symptoms of Raynaud's phenomenon (RP). This condition affects approximately 1.6% to 7.2% of the general population (Garner et al, 2015). This case study will explore the consultation of a 49-year-old male seen in a primary care first contact podiatry clinic with suspected RP. Critical appraisal of the evidence for diagnosis and management of primary and secondary RP is presented, and consideration given to possible aetiologies of secondary RP. Subsequent investigative options, clinical management and multidisciplinary approaches to care are described.

Who are first contact practitioner podiatrists?

First contact practitioner (FCP) podiatrists work within primary care services and are integrated within the multidisciplinary primary care team. They work alongside GPs, advanced clinical practitioners, nurses, FCP physiotherapists and other primary care professions.

Based within primary care, FCP podiatrists can communicate with specialist practitioners based within community or secondary care services to help coordinate, integrate and personalise complex packages of care for people living with multimorbidity affecting the foot, ankle or lower limb (Cherry et al, 2023).

FCP podiatrists are experienced clinicians with demonstrable academic (master's level) training in the

clinical pillar of advanced practice, with additional primary care-specific training to allow them to navigate the complexity of consulting patients with undifferentiated and undiagnosed conditions affecting the foot, ankle and lower limbs (Health Education England (HEE), 2021).

Primary care-specific postgraduate training can be achieved either via a portfolio route or a taught FCP level 7 module via a university (RCPod, 2024a), plus extensive GP mentorship guided by the HEE (now NHS England) FCP podiatry roadmap in primary care (HEE, 2021).

This roadmap allows FCPs to provide holistic patient care by improving their skills and knowledge across different body systems, beyond training at initial registration, and work at the top of their clinical scope of practice by achieving competency in advanced diagnostic and decision-making such as:

- Advanced physical assessment
- History taking
- X-ray and blood test interpretation.

With additional training, FCPs can incorporate further skills in their clinical practice, such as non-medical prescribing and minor surgery as part of treatment planning and care coordination across integrated care services (RCPod, 2024b).

However, given the infancy of the FCP podiatrist role, further research is required to address the training needs and overall potential of FCP podiatrists in primary care (Biggerstaff et al, 2023).

Key messages

When based within primary care, FCP podiatrists are well placed to:

- Coordinate complex care across integrated care services and settings, including introduction of new diagnoses for people living with multi-morbidity.
- Optimise timely access to health reviews and holistic medication review, and implement changes to management planning or prescribing.
- Complement continuity of care within community podiatry services without duplicating service provision.
- Use primary care resources to act as complex care coordinators for lower-limb diagnostics across integrated care services where community podiatry services are not commissioned for this type of contact.
- Differentially diagnose underlying causes of digital ulceration that can be effectively and holistically managed within primary care, including primary and secondary Raynaud's phenomenon.

Clinical case study presentation

Our male patient presented with three-week duration of digital ulceration and ongoing history of cold extremities. His symptoms alternated between pale and cold skin with numbness to warm, erythematous skin with burning sensation or paraesthesia. Symptoms were reported as affecting both hands and feet, occurring year-round and aggravated by cold weather exposure. He denied intermittent claudication or rest pain and reported being 'otherwise well in himself'. Typically, he reported wearing waterproof leather boots when employed as a security officer. His occupation is mainly indoors and active.

Summary of patient demography and health status:

Patient: White British male, 49
Weight: 86.5kg
Height: 1.829m
BMI: 25.86
Clinical vital signs: temperature: 36.3°C; oxygen saturation: 98%; blood pressure: 143/96; heart rate: 60 beats per minute; pulse: regular
Medical history and medication: see Table 1.
Occupation: security officer
Social history: minimal alcohol intake, ex-smoker of approximately 30 pack years and started vaping instead in 2019.
Family history: aunt and cousin (blood relatives) both diagnosed with RP, uncle diagnosed with diabetes and father diagnosed with non-ischaemic cardiomyopathy.
Subjective clinical examination: visible superficial 2cm × 2.5cm ulceration on the apex of right hallux with 1mm depth, macerated border, no exudate, granulating tissue on examination of the wound bed (Table 1). All toes are cold to touch, pallor is noted. All pedal pulses are biphasic with Doppler examination. Reduced protective sensation to 10g monofilament (6/10 bilaterally). No joint pain or tenderness is noted on passive manipulation, negative MTP/MCP joint squeeze test, hands and feet capillary refill: 4-5 seconds. Palmar and digital erythema and rosacea are observed.
Working diagnosis and rationale: secondary RP. From the clinical examination and patient history, acute pernio (chilblains) was also a potential diagnosis. However, this tends to follow a seasonal pattern, making acute pernio less likely (Olin and AlMahameed, 2013) and warranting further investigation for secondary RP for an underlying cause of the symptoms.
Short- to long-term management plan and rationale:

1 Blood tests arranged to investigate for common autoimmune, vascular and endocrine disorders.
2 De-prescription of propranolol due to known unwanted side effects of RP.
3 Initiation of nifedipine 5mg three times daily to improve blood supply to peripheral tissue.
4 Onward referral to community podiatry services for wound care and localised management of foot health.
5 Follow-up consultation to discuss interpretation of blood test results, review of prescribed medication and coordination of onward referral alongside existing services already contributing to patient care.

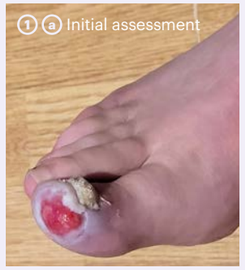
TABLE 1

Summary of case study patient medical history and medication use

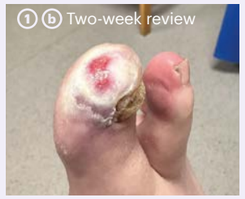
Medical history	Prescribed medication
Lumbar spondylosis (Jan 2009)	**Amitriptyline 50mg** one at night **Gabapentin 300mg** four times a day **Naproxen 500mg** twice a day
Migraine with aura (Sep 2015)	**Propranolol 40mg** three times a day
Hypertension (Jan 2018)	**Ramipril 2.5mg** once daily
Pre-diabetes (Aug 2021)	**Nil**

FIGURE 1

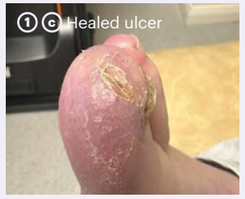
Secondary Raynaud's phenomenon with superficial digital ulceration



Nifedipine 5mg three times a day, prescribed for Raynaud's phenomenon.
Propranolol 40mg de-prescribed.
Referral made to community podiatry.
Blood tests arranged.
Blood pressure monitored.



Nifedipine 10mg three times a day.
Blood pressure monitored.
Under community podiatry for wound care management.



Blood pressure monitored.
Follow-up with community podiatry for review.

Photographs provided with consent to use and reproduce for educational purposes.

Key learning points

- RP can be idiopathic or secondary to an underling aetiology.
- Iatrogenic causes, connective tissue disorders or other rheumatic conditions and vascular disease can contribute to secondary Raynaud's-related digital ulceration.
- Clinical presentation may be phasic, therefore rapid access for health professional review is helpful in aiding timely diagnosis and improving prognosis.

Diagnosing Raynaud's phenomenon

RP was first described in 1862 by Auguste Gabriel Maurice Raynaud (1834-81). It is a common vasospastic disorder with disruption of the peripheral vasculature that affects normal tissue nutrition and body thermoregulation (Wigley and Herrick, 2015). There are several triggers for RP and exposure to cold temperatures or emotional stressors are common ones (Haque and Hughes, 2020). RP can be primary and seen in most cases with no known causes (80-90%) or it can present secondary to an underlying aetiology (Pauling et al, 2019). The prevalence of RP within the general population is approximately 5%, but some studies have found greater prevalence of primary RP in women (2-20%) compared to men (1-12%) and increasing prevalence with age (Maundrell and Proudman, 2015).

Primary versus secondary RP

When RP is suspected, it is important for an FCP podiatrist to distinguish between primary and secondary RP to guide investigation and a management plan. This relies on comprehensive history-taking, which includes the patient's past medical history, family history, current medication, socioeconomic factors, occupation and onset and severity of presenting symptoms. Primary RP has no known causes and is defined as idiopathic; it has an earlier onset (15-30 years old) and is characterised by milder symptoms (Chikura et al, 2010), with an estimated prevalence of 5% within the general population (Garner et al, 2015). Diagnosis of primary RP is usually made clinically and based on patient history and a thorough evaluation to rule out the presence of underlying causes.

Typical manifestations in both primary RP and secondary RP include a characteristic 'triphasic' colour pattern and numbness and swelling in the affected digits, which are considered mild and not highly problematic to the patient (Block and Sequeira, 2001). However, in secondary RP, patients can present with more severe symptoms such as irreversible ischaemia, digital ulcers and necrosis leading to amputation – this is usually related to other underlying health issues. Secondary RP can be a syndrome of underlying vascular disorders, iatrogenic causes (drug-induced) or occupational, haematologic or endocrine disorders (Haque and Hughes, 2020). It is most often associated with autoimmune diseases such as systemic lupus erythematosus or scleroderma (Musa and Qurie, 2023). Therefore, prompt identification and diagnosis of the underlying cause of secondary RP is vital to positively improve prognosis (Maciejewska et al, 2022).

Raynaud's symptoms and ulceration

The patient was presenting with Raynaud's symptoms as well as digital ulceration and therefore investigated for secondary RP. Secondary RP often has a later onset, usually after 40 years old, and accounts for >

approximately 10% to 20% of RP cases (Maundrell and Proudman, 2015). The pathogenesis of RP is not well understood, but it is theorised that the imbalance between vasoconstrictors and vasodilators, abnormalities in blood vessels, neural control and intravascular mediators, such as platelet activation and oxidation stress, may be minimal in primary RP and more severe in secondary RP, leading to more severe symptoms (Herrick, 2005; Wigley, 2002).

Autoimmune disorders: systemic sclerosis and systemic lupus erythematosus

Systemic sclerosis (SSc): It is important to consider autoimmune disorders such as systemic sclerosis and systemic lupus erythematosus as a potential cause of secondary RP. SSc is an autoimmune disorder characterised by inflammation, fibrosis and vascular abnormalities that can affect several organs of the body including the heart, lungs and skin. SSc has strong correlation with secondary RP: 90% of people with diagnosed SSc present with Reynaud's symptoms (Maundrell and Proudman, 2015; Levien, 2010). It is typically the initial manifestation of the disease and can precede the involvement of other organs by many years (Mostmans et al, 2017).

Systemic lupus erythematosus (SLE): A chronic autoimmune disorder that causes inflammation in the body's tissue and can manifest in the whole body, including the skin, joints and internal organs. Symptoms of SLE are often general but can include muscle, joint pain and skin rash (NICE, 2021). RP appears in 18% to 46% of people with SLE (Pavlov-Dolijavonic et al, 2013; Block and Sequeira, 2001). Nonetheless, RP is considered a non-specific and more benign clinical manifestation for SLE (Heimovski et al, 2015).

Vascular disorders

In addition to autoimmune disorders, vascular conditions should be considered in the differentials of secondary RP and distinguished from disorders that can cause digital discolouration including acrocyanosis, pernio and livedo reticularis, which can share similar clinical characteristics with RP (Choi and Henkin, 2021). In older patients, obstructive vascular disease is a common cause of RP, which includes thromboangiitis obliterans (Buerger disease), microemboli, diabetic angiopathy or atherosclerosis. When a vascular aetiology is suspected, vascular opinion should be sought for further investigation and a management plan.

Iatrogenic causes of RP

Certain medications can lead to secondary RP as an unwanted side effect. Our patient was taking propranolol to prevent migraines. Propranolol is a non-selective B1 and B2-adrenoceptor antagonist (beta blocker) and is commonly used as first-line migraine prophylaxis (NICE, 2023). However, RP is a known common undesirable effect of propranolol (Electronic Medicines Compendium, 2023), with RP having a higher prevalence in patients receiving beta blockers compared to the general population (Mohokum et al, 2012). Beta blockers can induce peripheral vasoconstriction and have been identified by the Framingham Heart Study as a common (34.2%) cause of secondary RP (De Angelis et al, 2006).

While exact pathophysiology of how beta blockers lead to peripheral vasoconstriction is not completely understood, the antagonism of B2-adrenoreceptions, which are responsible for peripheral arteriolar vasodilation, has been identified as the main mechanism (Khouri et al, 2016).

The role of diagnostic investigations

Blood tests are not carried out in primary RP and no specific blood test can diagnose it. However, a panel of blood tests are available in primary care to differentiate between primary and secondary RP (Belch et al, 2017). This includes:

- Full blood count
- Erythrocyte sedimentation rate
- Haematinics (B12, folate, ferritin)
- Renal and liver function tests
- Thyroid-stimulating hormone
- Urinalysis
- Rheumatoid factor
- Vasculitis screen
- Immunological tests for antinuclear (ANA), anti-Ro (SS-A) and anti-La (SS-B) antibodies.

TABLE 2

Case study blood test results

Blood test	Result	Interpretation
Full blood count	Within normal range	Normal
ESR	16mm/hr	Satisfactory
Vitamin B12	366ng/L	Normal
Ferritin	90ng/ml	Normal
Folate	1.5ng/ml	Low – needs replacement therapy
CRP	2mg/L	Normal
eGFR	>90	Normal
Liver function test	Serum alkaline phosphate: 130iu/L Serum alanine aminotransferase: 37iu/L Serum total protein level: 75g/L Serum albumin level: 44g/L	Normal
Serum TSH level	2.17miu/L	Normal
Rheumatoid factor	<10 igM RF	Normal
ANA screen	Negative for antibodies: Ro, La, Sm, RNP, Jo-1, Scl-70, dsDNA, histones, PMScl-100, centromere, nucleosomes	Normal
Complement C3 Complement C4	1.56g/L 0.39g/L	Normal
HbA1c	41	Normal

ANAs are antibodies that attack their own cells and tissues and are seen in autoimmune disorders such as SLE. Around 95% of people with SLE have positive ANA (Zanussi et al, 2023; Avery et al, 2014). In primary RP, ANA is negative or normal. However, if a positive or abnormal ANA result is found this may suggest an underlying autoimmune disorder; therefore, rheumatology input should be sought to exclude rheumatological or autoimmune conditions (Maciejewska et al, 2022; Ratchford and Evans, 2015).

Interpretation of history and diagnostic investigation

For the presenting case, blood tests (see Table 2) and other investigations were unremarkable, with negative ANA, <10 rheumatoid factor, normal full blood count, normal complement C3/C4 results and normal urinalysis.

Therefore, the presence of underlying autoimmune and vascular disorders was considered unlikely, and alternate causes for secondary RP such as iatrogenic attribution were considered, in this case with relation to propranolol. The patient was advised and agreed to stop taking propranolol, and a review was made with his GP to consider other suitable medication for migraine prophylaxis. He was simultaneously referred to the local NHS podiatry service for further management of the ulcerated digit.

Management plan

In a few cases, pharmacological intervention may be necessary to manage RP symptoms (Rirash et al, 2017). While many randomised controlled trials have investigated several pharmacological treatments of both primary and secondary RP, calcium-channel blockers (CCBs) have become the first line of treatment for RP due their effectiveness and tolerability (García-Carrasco et al, 2008). Nifedipine and amlodipine are among the most widely prescribed.

Nifedipine was prescribed and monitored by the FCP podiatrist, reducing the need for GP input.

Both nifedipine and amlodipine are dihydropyridine calcium-channel blockers (D-CCB), which work by blocking calcium channels located in the cell membrane of vascular smooth muscle in the wall of arteries. This causes vasodilation and improved blood supply to tissues while having less effect upon the function of the heart compared to non-dihydropyridine CCBs (Elliott and Ram, 2011). D-CCB has been found to have a similar effect on both primary and secondary RP in reducing the frequency of attacks when compared to placebo; however, nifedipine has been shown to be slightly more effective in reducing the frequency of attacks in primary RP than in secondary RP. This may be due to secondary RP being more severe and potentially less reversible, with fixed vascular changes and not just vasospasm – but this is dependent on the aetiology of secondary RP.

A shared decision was taken with the patient to trial nifedipine, initially at low dose, to improve the Raynaud's symptoms. This was prescribed and monitored by the FCP podiatrist, a non-medical prescriber, reducing the need for GP input.

Dosing is also an important factor. Comparison of CCBs vs placebo revealed that higher CCB doses were superior to lower doses in reducing the frequency, duration and severity of attacks (Rirash et al, 2017). However, it is important for the prescriber to monitor blood pressure before initiation and after, due to CCBs' hypotensive effects, such as light-headedness or dizziness, weakness and syncope (Krasowska et al, 2017). Therefore, verbal safety-netting advice was given to the patient should he experience any unwanted side effects or adverse reactions to ensure safe prescribing practice (Silverston, 2014).

For the presenting case, nifedipine 5mg three times a day was prescribed and a follow-up appointment with the FCP podiatrist was arranged to revise the dosage. On follow-up, nifedipine was being well tolerated and consequently up titrated to 10mg three times a day, which reached maximum effectiveness. Great improvement in symptoms was reported by the patient.

In the general management of RP, every clinician can provide simple but invaluable education to the patient, such as avoiding triggering factors such as exposure to cold, sudden changes in temperature, stress and smoking cigarettes. Patients should keep their body and peripheries warm by wearing extra layers of clothing, thermal socks and gloves and appropriate non-restrictive footwear, and should be encouraged to exercise to improve circulation. The patient's occupation is an important factor for clinicians to consider, as vibration tools can induce RP and patients should be advised to avoid these where possible (Cooke et al, 2022).

Scan to view references

In conclusion

It is noteworthy that the FCP podiatry role is in its infancy, and more research is required to evaluate the role's effectiveness in primary care and on patient outcome. This case study aims to highlight how FCP podiatrists are well placed in primary care to manage complex clinical care, utilising their specialist knowledge of the foot, ankle and lower limb, postgraduate training and primary care resources to consider possible differential diagnoses and initiate investigations and treatment. These may include blood test interpretation, non-medical prescribing and holistic care plan formulation with a multidisciplinary team approach that includes input from GPs, community podiatry and other integrated care services.