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Delayed Management of Brachioradial Pruritis in a Primary Care Setting: A Case Report

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Abstract **Case Report**

Background: Brachioradial pruritus (BRP) is a neuropathic condition characterised by localised, intense pruritus in the dorsolateral aspects of the upper limbs, due to cervical spine disease. It is routinely mistaken for a primary inflammatory skin disorder in primary care settings, which results in delayed diagnosis & thus timely management, adversely affecting patients' quality of life. It is often linked aetiologically to mild cervical nerve root compression with or without significant foraminal stenosis in the literature. ***Methods:*** A 67-year-old Caucasian woman presented with a seven-month history of intense pruritus, numbness, and a tingling rash over the right shoulder corresponding to a C6 dermatomal distribution. The rash worsened in the summer & subsided in the winter months. Her GP had prescribed three different empirical treatments for eczema to no avail. She underwent haematological, biochemical, and autoimmune blood screen and a cervical spine MRI was subsequently performed. ***Findings:*** All inflammatory markers and the autoimmune screen came back as normal. The patient had independently self-identified her diagnosis & presented to the GP, prior to specialist consultation. She was then referred to the neurologist who then arranged a cervical MRI. This showed multilevel cervical degenerative disease at C5–C8 with osteophytes causing mild nerve root compression at C5 and C6, without significant foraminal stenosis. Gabapentin 300 mg twice daily was initiated alongside physiotherapy, topical capsaicin & she was given strict photoprotection advice. After only 6 weeks of targeted therapy, she demonstrated a 70% reduction in pruritus severity, with a significant resolution of the erythematous rash. ***Interpretation:*** BRP can arise from mild cervical nerve root compression without significant foraminal stenosis. A characteristic non-response to topical corticosteroids and antihistamines indicates the diagnosis. The clinical triad of dermatomal pruritus, neuropathic sensory symptoms, and summer exacerbation is sufficient to distinguish BRP from eczema clinically in primary care without the need for specialist investigation. For family physicians, BRP should be considered in any patient with unilateral dermatomal pruritus and neuropathic sensory symptoms which do not respond to standard dermatological treatment.

Keywords: Brachioradial pruritus; neuropathic pruritus; cervical spondylosis; misdiagnosis; primary care; family medicine; gabapentin; neuropathic itch.

INTRODUCTION

Brachioradial pruritus (BRP) is an anatomically localised sensory neuropathy presenting as persistent, intense pruritus of the dorsolateral aspect of the upper limbs, most commonly corresponding to the C5–C6 dermatomes. Early references in the literature since 1968 by Waisman as a sun-related disorder of the lateral elbow region termed 'brachioradial summer pruritis', [1] Miller *et al.,* established C5-C8 pruritis had radicular involvement secondary to cervical spinal stenosis in 85% of study patients, aggravated by ultraviolet exposure [2] A characteristic feature of the rash is short-lived relief by applying ice-cold packs to the affected skin, hence the term 'ice pack sign' [3] It predominantly affects fair-skinned middle/late aged women, with symptom flares in the summer periods due to high solar UV radiation. [4] This has been further proven in a larger retrospective study by Hiranput *et al.,* where women comprised 70% of the cohort, at a mean age at first presentation of 64 years, the symptoms were predominantly bilateral in 85% of patients, thus challenging the preconception of strict unilaterality.[5] Despite this recognisable presentation, BRP is frequently misattributed to inflammatory skin conditions in primary care such as eczema, with delayed diagnoses well established in the literature.[6] As BRP presents in an outpatient setting with cutaneous features that can resemble common dermatological conditions, it falls within the everyday

scope of family medicine. Despite this, the three features that reliably differentiate it from eczema: dermatomal localisation, associated neuropathic sensory disturbance, and worsening with sun exposure, are often overlooked. We report a 67-year-old woman with a seven-month period of misdiagnosis as eczema, subsequently found to have BRP associated with mild cervical nerve root compression at C5–C8 without significant foraminal stenosis, who had independently self-identified her diagnosis even prior to specialist assessment.

CASE REPORT

A 67-year-old Caucasian woman with no significant past medical history presented following seven months of pruritus, numbness, and tingling localised to the right shoulder in a C6 dermatomal distribution. Symptoms were consistently exacerbated during the summer months and with prolonged sunlight exposure. Over the preceding seven months, she had received three sequential empirical treatment trials: topical corticosteroids, topical antibiotics, and oral antihistamines, all under the working diagnosis of eczema, without clinical benefit at any stage.

Prior to her clinic attendance, the patient had independently researched her symptoms and accurately self-identified the condition as brachioradial pruritus, presenting with a well-informed account of its aetiology and characteristic features. She had no regular medications, no known allergies, and no personal or family history of atopy, psoriasis, or connective tissue disease.

Cutaneous examination revealed an erythematous plaque with excoriations on the dorsolateral aspect of the right shoulder, with sharp borders conforming to the C6 dermatome. No other body sites were affected. Cervical spine examination demonstrated mildly restricted lateral flexion and extension without any upper limb neurological deficit, though the patient reported subjective paraesthesiae in the right shoulder distribution. No atopic features, flexural involvement, or bilateral distribution was identified.

Investigations

Haematological and biochemical investigations were performed to exclude systemic, inflammatory, and autoimmune causes of pruritus (Table 1). All results were within normal limits. Cervical spine MRI demonstrated multilevel degenerative disc disease with osteophyte formation at C5–C8, with mild nerve root compression at C5 and C6. No significant foraminal stenosis was identified at any level.

Table 1: Summary of investigations

Investigation	Result	Interpretation
C-reactive protein	< 5 mg/L	Normal
ESR	8 mm/hr	Normal
ANA	Negative	Normal
Anti-dsDNA	Negative	Normal
Full blood count	Within reference range	Normal
Thyroid function tests	Within reference range	Normal
Renal and hepatic profile	Within reference range	Normal
Cervical spine MRI	Multilevel spondylosis C5–C8 with osteophytes; mild nerve root compression at C5 and C6; no significant foraminal stenosis	See text

***ANA, antinuclear antibody; CRP, C-reactive protein; anti-dsDNA, anti-double-stranded DNA; ESR, erythrocyte sedimentation rate; MRI, magnetic resonance imaging.**

Differential Diagnosis

The following differential diagnoses were considered and systematically excluded:

1. ***Atopic or nummular eczema***:
   Excluded by dermatomal (not eczematous) distribution, absence of atopic history, associated neuropathic sensory symptoms inconsistent with eczema, and complete non-response despite three sequential treatment courses of topical steroids, topical antibiotics, Pimecrolimus & oral anti-histamines.

2. ***Contact dermatitis***:
   Excluded by dermatomal distribution, the absence of any identifiable contact allergen, and treatment non-response; patch testing was deferred due to the high probability of a neuropathic diagnosis.

3. ***Photodermatosis (polymorphous light eruption / chronic actinic dermatitis)***:
   Excluded by the strictly unilateral dermatomal distribution (not corresponding to sun-exposed anatomy), absence of vesicular or urticarial morphology, chronic unremitting course unresponsive to sun avoidance, and normal inflammatory and autoimmune serology.

4. ***Connective tissue disease***:
   Excluded by negative ANA, anti-dsDNA, normal CRP and ESR, and the absence of multisystem involvement.

5. *Brachioradial pruritus*:

Confirmed by the trifecta of dermatomal pruritus with concurrent sensory symptoms in the C6 distribution, summer exacerbation, and cervical spondylosis on MRI, in the absence of an alternative diagnosis.

Treatment

Following diagnosis, the patient was referred to neurology for further management. Gabapentin was selected as first-line pharmacological treatment. The use of transforaminal corticosteroid injections as well as gabapentin for BRP by Berger *et al.,* in 2022, who described a significant reduction in pruritus among patients receiving 300 mg administered in three divided daily doses, pointing to the evidence of centrally mediated neuropathic itch [7] In our patient, gabapentin was initiated at 300 mg twice daily and up-titrated as clinically indicated. Topical capsaicin cream was also prescribed to progressively reduce neuropeptide stores at peripheral sensory nerve endings, in order to achieve gradual desensitisation of the affected dermatome without systemic effects. Physiotherapy was commenced to improve cervical spinal mechanics and reduce ongoing irritation of the affected nerve roots, and strict photoprotection was advised, including broad-spectrum sunscreen (SPF ≥50) and UV-protective clothing for the affected shoulder. [12]

Outcome and Follow-Up

At the six-week review, the patient reported approximately 70% reduction in pruritus severity across all three symptom domains (pruritus, numbness, and tingling). The erythema had substantially resolved and excoriations were absent. Gabapentin was well tolerated. She continued physiotherapy and used topical capsaicin during exacerbations, with additional benefit. Neurological follow-up was ongoing.

DISCUSSION

Mild cervical nerve root compression as a predominant cause

Numerous articles in the literature point to cervical disc degeneration being a primary cause of BRP mainly due to nerve root sensitisation leading to aberrant sensory signals in the upper limbs. Shields *et al.,* examined with electromyography & cervical MRI a total of nine patients, all which had structural cervical spine abnormalities on imaging [8]. Nerve conduction and electromyographic findings revealed C6 cervical radiculopathy as the main pathological finding as well as bilateral C5 and C6 involvement in one patient. Clear objective examination findings were demonstrated in the majority of study patients, such as hyperesthesia and abnormal upper-limb tendon reflexes [8]. This was further re-iterated by Marziniak *et al.,* who demonstrated cervical changes in the C5-C6 segment on MRI in 38 patients with BRP symptoms [9]. However, our case is among a small number of reported instances in a complete BRP clinical picture arose in the setting of only mild nerve root compression at C5 and C6, with no significant foraminal stenosis and no objective motor deficit. This indicates that even low-grade nerve root irritation may be adequate to initiate the neuropathic sensitisation that underlies this condition.

A diagnostic non-response to conventional dermatological treatments

We found a consistently observed pattern in the literature of failure to benefit from topical corticosteroids and oral antihistamines, proven by Mirzoyev and Davis in their 10 year Mayo Clinic review of 111 patients. Neither topical steroids nor antihistamines improved the associated hyperaesthesia & pruritic symptoms, due to the pathophysiological mechanism involving sensitised nerve roots rather than cutaneous inflammation or histamine-release. [10]

Only less than half of the patients underwent cervical imaging as part of their diagnostic workup at any stage, as cervical spinal pathology is rarely considered as part of routine primary care evaluation. Among those who underwent cervical MRI, varied structural findings were found: neural compression & narrowing of the neural foramina in a few, others showed intervertebral disc displacement, but in many, no compressive pathology was found, similar to our patient.

In 2017, Pereira *et al.,* compared BRP and notalgia paraesthetica in 58 consecutive patients with burning and stinging symptoms frequently reported. Cervical spinal imaging demonstrated corresponding dermatomal correlation with the symptomatic area described by the BRP group versus the NP group, which renders cervical MRI as the key differentiating diagnostic investigation in BRP [11]. In the present case, three consecutive treatment failures over seven months failed to prompt any reconsideration of the working diagnosis, representing a substantive clinical error, as persistent non-response to standard dermatological therapy in the context of a dermatomal itch pattern should prompt *redirection* of clinical evaluation rather than continuation of empirical prescribing.

On reflection, initially at first presentation, there were *three* distinguishing clinical features which should have prompted the consideration of neurological involvement as a precursor to BRP symptoms prior to specialist referral. Firstly, the unilateral dermatomal localisation of symptoms to the right shoulder cannot be attributed to eczema, as it does not involve a single dermatome with isolated unilateral shoulder pain. Secondly, the associated sensory symptoms of numbness and tingling in the same C6 area are absent in eczema. This was demonstrated by Shields *et al.,* where more than half of the participants had cutaneous sensory symptoms with diminished upper limb reflexes, [8] highlighting how objective neurological signs occur simultaneously with pruritus in BRP. Thirdly, the

summer/heat exacerbation of the rash, reflecting the role of ultraviolet radiation as a peripheral sensitising stimulus in BRP, [12] in contrast to eczema, which characteristically deteriorates in colder months due to reduced ambient humidity, causing compromise of epidermal barrier function. Although there is no set diagnostic work-up framework for BRP, the clinical pattern is well-described in the literature in order to be recognised in a primary care setting.

Patient self-identification

The patient's accurate independent identification of BRP, following seven months of ineffective management, prompted appropriate specialist referral. Published guidance on BRP identifies two primary obstacles to timely diagnosis: the absence of agreed-upon diagnostic criteria, and insufficient awareness of the condition among primary care clinicians. This calls for greater collaboration between dermatology and neurology services in order to practically reduce these delays. This recommendation is highlighted in our patient who bore this consequence. Despite no medical training, she was able to identify her own condition through pattern recognition and online research when primary care physicians failed to deliver. Systematic evaluation is warranted as the patient presented with a self-generated diagnosis after several failed treatment trials. Her self-identification was not only accurate but was the sole driver of appropriate onward referral.

CONCLUSION

To our knowledge, this case is among the few reported examples of BRP attributable to mild cervical nerve root compression at C5 and C6 in the absence of significant foraminal stenosis, extending the recognised radiological threshold for this diagnosis. For family physicians, the triad of unilateral dermatomal pruritus, accompanying neuropathic sensory disturbance, and predictable worsening with summer sun exposure should raise suspicion of BRP and prompt cervical spine imaging regardless of whether the patient has visible skin changes. Persistent lack of response to standard dermatological therapy is a further pointer towards a neuropathic rather than inflammatory diagnosis and should trigger earlier referral rather than ongoing empirical treatment. Multimodal management combining gabapentin, topical capsaicin, cervical physiotherapy, and sun protection achieved substantial symptomatic benefit in this case. Increased awareness of BRP among primary care clinicians is essential to shorten the diagnostic intervals that continue to characterise this condition.

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