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CASE SERIES

Dupilumab, a Novel Treatment for Peripheral Neuropathy: A Case Series

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ABSTRACT

Peripheral neuropathy is a chronic neurological disorder that can present with a multitude of symptoms. It is observed in association with various disease states and organ systems. In the process of treatment for atopic dermatitis, we have observed that patients reported improvements in their previously diagnosed neuropathies, essentially, relief from neuropathic symptoms such as burning, itching, or tingling. On the basis of these observations, we conducted a single-arm trial to survey the qualitative changes in patients with peripheral neuropathy. Ten patients with atopic dermatitis with previously diagnosed peripheral neuropathies were treated with dupilumab (DP) and observed for symptom changes over 6 months. A paired *t*-test was used to determine whether DP might potentially be used as an off-label treatment for patients with peripheral neuropathy and dermatological conditions. Our patients showed significant symptom relief, thus suggesting that further investigation of the use of DP for patients with neuropathies is warranted.

Keywords: Dupilumab; Peripheral neuropathy; Biologicals

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Key Summary Points

- Patients in our clinic had longstanding symptoms associated with peripheral neuropathies, thus leading to difficulty in ambulating and functioning in daily life activities.
- We hypothesized that immune modulatory drugs might offset or dampen T-helper 2 (Th2) responses that induce symptoms associated with peripheral neuropathy.
- Many patients in this study gained ambulatory function as their symptoms started to subside.
- A specific population of patients diagnosed with peripheral neuropathies that are Th2-induced might benefit from mitigation of symptoms by dupilumab.

INTRODUCTION

Peripheral neuropathy is a condition involving damage to neurological pathways in the peripheral nervous system. Neurological pathways can be dysregulated, causing inappropriate gain or loss of function and/or leading to many types of peripheral neuropathies. According to the National Institute of Neurological Disorders and Stroke, neuropathy is classified by the nerves involved, including motor nerves (control of muscle movement), sensory nerves (e.g., transmission of pain, temperature, sensation), and autonomic nerves (control of internal organs and regulation) [1]. Symptoms of peripheral neuropathy involve these three neuronal types. Numbness, tingling, and itching may suggest sensory involvement; weakness and rigidity may suggest motor involvement; and autonomic involvement may include specific organs or changes in homeostasis, such as hyper/hypohidrosis, alterations in blood pressure, or impotence [2]. Peripheral neuropathies are also distinguishable by their length-dependent or non-length-dependent patterns. Patients with a length-dependent presentation, such as diabetic peripheral neuropathy, show paresthesia in the most distal nerve endings, such as the feet. In contrast, patients with non-length-dependent neuropathies show centrally presenting symptoms (e.g., in the torso), such as post-herpetic neuralgia.

Causes of peripheral neuropathy can be vast and complex to isolate, particularly in cases with rare origins. Neuropathies are known to be caused by traumatic, diabetic, vascular, autoimmune, hormonal, nutritional, oncological, chemotherapeutic, infectious, nutritional, or hereditary conditions [1]. Diagnosis is typically based on medical history, blood work, physical examinations, and possibly genetic testing. Further tests to aid in differential diagnosis include nerve conduction velocity, electromyography (EMG), nerve and skin biopsies, and imaging (magnetic resonance imaging (MRI) or computed tomography (CT)). Treatment and management typically involve addressing the root cause. For example, patients with carpal tunnel syndrome can be splinted, treated with inflammatory regulating medications, or treated with surgery to relieve symptoms.

Nerve dysregulation and damage in peripheral neuropathy are currently being studied to provide insights into the pathophysiology of poorly understood diseases. For example, prurigo nodularis, a chronic dermatological condition involving raised nodules (hyperkeratosis) that are incredibly pruritic (itchy), is strongly associated with peripheral neuropathy as an underlying etiology [3]. Herein, we investigated peripheral neuropathy and its potential treatment with DP. This human monoclonal antibody is an interleukin-4 (IL-4) receptor alpha antagonist that inhibits IL-4, interleukin-13 (IL-13), and interleukin-31 (IL-31) signaling, decreasing the Th2 inflammatory response [4]. DP is currently indicated for treating moderate to severe atopic dermatitis and is also Food and Drug Administration (FDA)-approved for treating prurigo nodularis in adults. Case studies have shown that DP effectively eliminates itching symptoms [5], thus expanding the discussion of whether DP might be a suitable off-label use for treating symptoms, such as itch, caused by peripheral neuropathies [6].

METHODS AND STANDARDIZED TREATMENT PLAN

In this study, ten patient cases were reviewed. Each patient had a prior diagnosis of peripheral neuropathy, with some cases confirmed through EMG or biopsy. Each patient involved in this study was evaluated in the clinic for dermatologically related symptoms, including atopic dermatitis. Interestingly, these patients exhibited additional complaints of tingling, allodynia, and numbness, symptoms more associated with neuropathy. When explicitly asked about neuropathy systems, some patients also complained of poor ambulation due to pain. Symptoms related to peripheral neuropathy were documented from each patient with a rating scale from one to ten, one being the lowest and ten being the highest exhibited level of that symptom prior to starting treatment. All cases are briefly summarized in Table 1.

All patients received DP with an initial loading dose of two 300 mg subcutaneous injections into the subcutaneous tissue on day 1 and a 300 mg dose every 2 weeks thereafter for 6 months. Patients were then reassessed on the same symptom scale from day 1 at the end of 6 months of treatment. Paired *t*-tests were conducted on the averages for each component of peripheral neuropathy symptoms, and groups were compared before and after DP treatment (Fig. 1).

Ethical Approval

Ethical approval was not required for this study in accordance with local or national guidelines. However, the study was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent was obtained from all participants prior to their inclusion in the study. No detailed identifying medical information of the patients is published in this study.

RESULTS

In our small observational study, our evidence shows that the DP regimen, with a loading dose of 600 mg and an additional 300 mg subcutaneous injections every 2 weeks for 6 months of total treatment, has been shown to reduce the symptoms exhibited by our patients in relation to peripheral neuropathy. Of note, none of the patients observed had any worsening symptoms

Table 1 Summary of all ten included patients with chronic pruritus and peripheral neuropathy referred for dermatologic evaluation

Case	Age	Sex	Diagnosis	Summary
1	94	F	PN (EMG+)	EMG-confirmed neuropathy with 10-year history of chronic pruritus and rash; also had SCC; treated with gabapentin without full relief
2	91	F	PN (EMG+)	EMG-confirmed neuropathy of unknown cause; 4 years of pruritus, weakness, burning, fatigue; diagnosed with MGUS; no prior treatments were effective
3	71	F	PN (Biopsy+)	Biopsy-confirmed small fiber neuropathy with erythromelalgia; severe burning, cold sensations, shock-like pain; unresponsive to multiple medications
4	74	M	PN (EMG+)	EMG-confirmed neuropathy; history of non-melanoma skin cancers; presented with weakness, instability, and mobility issues
5	36	M	PN (EMG+)	Developed EMG-confirmed neuropathy following severe COVID-19; experienced burning, numbness, itch, and generalized pruritus
6	79	F	PN (clinical)	Glove and stocking neuropathy with atopic dermatitis; worsening hand/foot pain disrupting sleep; gabapentin ineffective
7	77	F	PN (clinical)	Peripheral neuropathy and atopic dermatitis; minimal symptom improvement with gabapentin
8	84	F	PN (clinical)	Neuropathy with atopic dermatitis; progressive instability and sensory loss requiring walker; gabapentin ineffective
9	32	M	PN (clinical)	Severe, longstanding neuropathy with atopic dermatitis and type I diabetes
10	71	M	PN (clinical)	Severe lower extremity neuropathy with atopic dermatitis

EMG electromyography, *F* female, *PN* peripheral neuropathy, *M* male, *MGUS* monoclonal gammopathy of undetermined significance

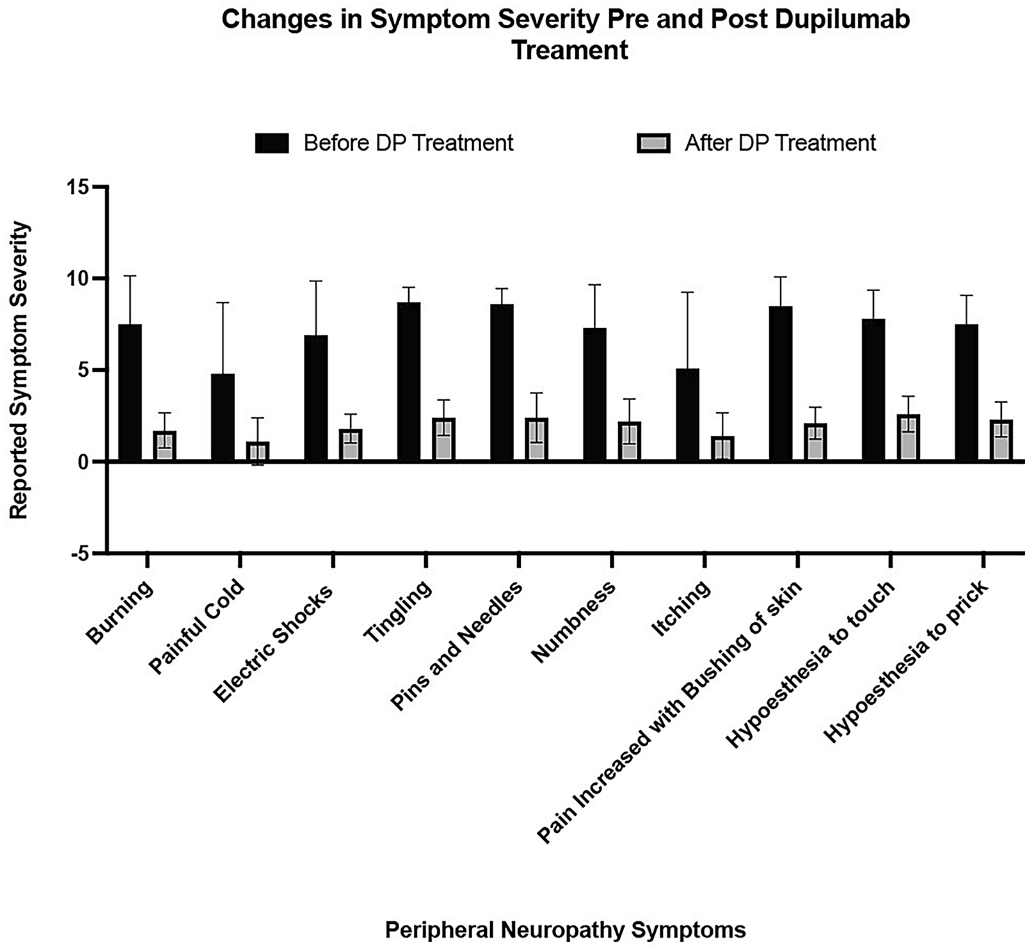

Fig. 1 Bar graph representing paired *T*-test results comparing the average reported symptoms present in each patient before and after DP treatment within every symptom

when on DP. Paired *t*-tests were selected owing to highly sensitive comparisons of patients before and after DP treatment. The tests were conducted on the basis of average results for each peripheral neuropathy symptom observed, and participants were compared before and after DP treatment (Fig. 1). All ten patients showed sustained improvements based on decreased symptoms after DP administration. However, post-treatment EMG was not performed in this study to objectively confirm neurological improvement. Prior to treatment, tingling, pins and needles sensation, increased pain with brushing of skin (allodynia), and hypothesia generally had higher symptom ratings, alongside painful cold, electric shock, and itching having lower ratings. Evaluation of patient reported reduction in symptom severity, with an average decrease of −5.063 in VAS scores (0–10) [95% CI: −5.754, −4.361], indicating a clear improvement in patient-reported symptoms of peripheral neuropathy following DP treatment. Of all the symptoms evaluated, tingling ($p<0.000001$), pins and needles sensation ($p<0.000001$), and hypothesia to prick ($p<0.000001$) were revealed to have the most remarkable improvements observed with DP treatment as reported by each patient. Second to that, hypothesia to touch ($p<0.000001$), increased pain with brushing of skin (allodynia) ($p<0.000002$), numbness ($p<0.000005$), burning ($p<0.000019$), and electric shock ($p<0.000081$) were also observed to be significant. Notably, the symptoms that showed the least improvement were found to be painful cold ($p < 0.007778$) and itching ($p < 0.004628$) after DP treatment.

DISCUSSION

Medical literature describing the association between atopic dermatitis and neuropathy is relatively sparse. While not often observed, particularly in Western cultures, atopic myelitis is a well-documented condition linking atopy and neuropathy. Given that neurologic symptoms such as pruritus and dysesthesia are commonly encountered in atopic conditions, their evaluation remains relevant to dermatologic practice. DP is an approved treatment option for controlling inflammatory disorders such as atopic dermatitis and prurigo nodularis. This subjective study has indicated amelioration of patient-reported peripheral neuropathy symptoms. Herein, among all associated symptoms of peripheral neuropathy, tingling, pins and needles sensation, and hypothesia to prick showed the most significant improvement, whereas itching and painful cold showed the least improvement.

In contrast, itching—commonly associated with peripheral neuropathy and atopic dermatitis—showed the least improvement. However, this outcome may be influenced by the lower baseline severity of itching prior to treatment, though some degree of symptom relief was still observed following DP administration. Notably, no symptom exhibited a worsening effect, further supporting the potential therapeutic benefit of DP in this context. Interestingly, several patients were unable to walk prior to therapy and were able to report that, with neuropathic symptom relief, daily ambulation was achieved with the DP regimen.

Our research suggests that Th2 modulation by DP may be attributable to its therapeutic benefit. However, the precise mechanisms underlying these effects remain incompletely understood, preventing a definitive explanation of the mode of action. Nonetheless, this study represents an initial step toward understanding the relationship between immune responses, biological mechanisms of action, and peripheral neuropathies.

DP is an IL-4R alpha inhibitor that targets the Th2 pathway implicated in highly pruritic inflammatory diseases such as atopic dermatitis, prurigo nodularis, and urticaria [7]. The primary cytokines involved in this pathway include IL-4, interleukin-5 (IL-5), IL-13, and IL-31 [8]. Upregulation of these cytokines contributes to the various symptoms observed in these conditions. Specifically, IL-5 is associated with edema and blister formation, whereas upregulation of IL-4 and IL-13 impairs the barrier function of the epidermis, thereby leading to xerosis. Moreover, these cytokines inhibit cutaneous innate immunity and facilitate skin colonization by common microorganisms such as *Staphylococcus aureus* [9].

Pruritus in the Th2 pathway is associated predominantly with the upregulation of IL-31. IL-31 mediates pruritus by activating the IL-31 receptor and oncostatin M receptor (OSMR) receptors in keratinocytes and the dorsal root ganglia of cutaneous sensory nerves [10]. Activation of IL-31R receptors on the dorsal root ganglia leads to the sensation of itch [11], by triggering transient receptor potential cation channels (transient receptor potential cation channel subfamily V member 1 (TRPV1)/transient receptor potential cation channel subfamily A member 1 (TRPA1)) [11]. An in vivo study on mice has demonstrated significantly diminished IL-31R-induced pruritus in TRPV1+-deficient mice, thus developing a strong correlation between IL-31 and pruritus [9]. Notably, IL-31 upregulation is not confined to the Th2 pathway but also occurs in T-helper 1 (Th1) reactions in the presence of IL-4. IL-4 induces the upregulation of IL-31, thus suggesting a direct relationship between these cytokines. Genetic studies have shown that inhibiting IL-4 decreases IL-31 levels within 48 h [12].

Our literature search did not yield specific studies on the modus operandi of DP in PN and the Th2 pathway. This is partly because peripheral neuropathy is an umbrella term encompassing multiple pathological subtypes with distinct and, in some cases, unknown pathophysiologies. Interestingly, several reports and studies have already explored the association between the development of peripheral neuropathy and Th2-associated interleukins such as IL-4, IL-5, IL-13, and IL-31.

A commonality among studies is that increased atopy and eosinophilia have been found to lead to mast cell destruction of the blood–brain barrier and the induction of spinal cord lesions [13]. Th-2 mediates this response, thus potentially explaining why DP ameliorated the neuropathies in our patients. Another prospective observational study on 13 subjects showed that using mepolizumab, an IL-5 inhibitor, significantly improved treatment-resistant peripheral neuropathy for pain and numbness [14].

However, in studies on chemotherapy-induced peripheral neuropathy, IL-13 administration has been reported to induce IL-10 production and to cause a shift from inflammatory M1 macrophages to reparative M2 macrophages in patients with neurological damage from cisplatin [15]. Similarly, studies in mice models have shown that Il-13 decreases tactile allodynia after partial sciatic nerve ligation [16]. Another notable finding is that IL-4 is the primary cytokine that stimulates immune cells, including Schwann cells, macrophages, fibroblasts, and neurons, to adopt a healing phenotype in nerve regeneration. This makes IL-4 a promising target for the treatment of peripheral nerve injuries [17]. An additional study demonstrates that IL-4 can alleviate neuropathic pain by promoting the accumulation of macrophages, which exert a therapeutic effect [18].

DP additionally acts as an indirect IL-31 modulator through the IL-4 pathway, demonstrating significant effects on inflammation. Given that IL-31 is considered the primary cytokine driving pruritic conditions, IL-31 targeted therapy might also offer substantial benefits for patients with peripheral neuropathy. However, the potential risk of neuromodulation warrants further investigation; incorporating a direct IL-31 modulator such as nemolizumab might potentially decrease these risks [19]. The results across many studies have led us to understand that further investigation of biologics as neuropathic therapies is warranted to develop new treatment protocols for patients with peripheral neuropathies or subtle signs of neuropathy.

Although our study demonstrated that DP might serve as a possible novel therapy for peripheral neuropathy, it is important to highlight the potential risks of immune modulation for neurological disorders, particularly in patients without atopic root causes. Because DP decreases Th2-mediated inflammation, it might shift toward a Th1-interferon gamma (IFN-γ) response, thus further damaging neurological pathways and potentially leading to permanent damage. Two cases have been described in which IL-13/IL-4 modulation has led to either chronic inflammatory demyelinating polyneuropathy or acute inflammatory polyneuropathy after DP treatment onset [20, 21]. After discontinuing DP, the numbness resolved in the latter case after 6 months. Furthermore, DP has also been associated with Th1/17 immune dysregulation in a patient with multiple sclerosis (MS) [22]. In contrast, reports have indicated amelioration of MS lesions in a single patient receiving DP and teriflunomide therapy [23].

Adverse reactions to DP specifically are relatively uncommon and usually mild. An adverse event of interest is the development of arthritis after DP therapy. One possible mechanism is a shift from a Th2-heavy immune response to a Th1 response, leading to inflammatory arthropathies. In some patients, underlying arthralgias masked by chronic and longstanding neuropathy may become unmasked by DP therapy.

Our study provides insight into the potential off-label use of DP for patients with peripheral neuropathy. Given our smaller sample size and subjective measurements of improvement, observing the effects of DP therapy and the amelioration of peripheral neuropathy in our patients has provided the notion to gather quantitated studies as to the mechanism DP is a possible treatment to PN. We propose that further investigation of the use of biologic agents in neuropathic therapies should be encouraged further to elucidate the therapeutic potential of DP in this context. We propose a study incorporating a larger sample size and standardized EMG throughout the treatment course to monitor symptomatic changes objectively. Skin and nerve biopsies should also be performed to assess histopathological alterations in response to DP therapy. In patients with comorbid atopic disorders, further investigations—including IgE levels, cerebrospinal fluid (CSF) analysis, and spinal cord MRI—may also provide critical evidence supporting the role of biologic agents in the management of peripheral neuropathies. We encourage the discussion to develop new treatment protocols for patients with peripheral neuropathies to recognize, treat, and/or prevent secondary damage, further improving patient prognosis.

CONCLUSIONS

We observed a substantial decrease in symptoms associated with peripheral neuropathy after DP treatment, with clinical improvement as reported by each patient. We want to clarify that our study does not suggest that DP treatment repairs nerve damage but rather reports that patients exhibited significant clinical improvement with therapy and implements quantification of the subjective findings. Because peripheral neuropathy can involve various symptoms, have a wide array of etiologies, and be potentially managed with multiple modalities, expanding the options to treat dermatologic and neurologic symptoms will be crucial for patients who are afflicted by these chronic diseases.

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Data Availability. All data generated or analyzed during this study are included in this published article.

Declarations

Conflict of Interest. Sunny B. Patel, David R. Roy, Bart W.B. Sweers, and Michael K. Coffin all have nothing to disclose.

Ethical Approval. Ethical approval is not required for this study in accordance with local or national guidelines. This study was performed in accordance with the Helsinki Declaration of 1964, and its later amendments. Informed consent was obtained from the patient for participation in this study. No detailed identifying medical information of the patients is published in this study.

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