A novel pharmacological treatment strategy for chronic itching associated with notalgia paresthetica. A case report

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ABSTRACT

Background: Notalgia paresthetica is a chronic sensory neuropathy characterized by localized pruritus and associated dysesthesias. There exists no definitive therapy for the disease to date. Here we report a novel pharmacological treatment strategy with levodopa/benserazide in a female patient with chronic itching due to a probably hereditary form of notalgia paresthetica.

Case Presentation: We describe a 76-year-old female patient suffering from severely distressing notalgia paresthetica. Here, itching occurred mainly during the cold season in the area medial to the left scapula. Notably, the patient's mother, uncle, and cousin were also affected by the disease. Low-dose treatment with levodopa 50 mg combined with benserazide 12.5 mg per day significantly reduced itching and additionally alleviated the compulsive urge to scratch.

Conclusion: We present a hypothesis-generating observation describing low-dose levodopa associated with sustained symptomatic improvement in a patient with chronic itching due to notalgia paresthetica. We propose further controlled investigations to prove a novel treatment concept with low-dose levodopa and the suspected neurobiological difference between A11 and A10 dopaminergic neurons.

Keywords: Notalgia paresthetica, itching, levodopa, dopamine, dopamine transporter, A10/A11 neurons.

Type of Article: Case Report **Specialty:** Neurology

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Background

Notalgia paresthetica, relatively common yet underdiagnosed and frequently resistant to therapy, is a sensory, usually unilateral neuropathy [1,2]. For review, see also Robinson et al. [3]. It presents as chronic itching and burning with paresthesia in the area of the shoulder blades and upper back. The skin may undergo lichen-like changes and hyperpigmentation. Symptoms almost invariably persist for years. The lack of established treatment guidelines makes treating notalgia paresthetica complicated, as it dramatically affects patients' quality of life [3,4].

Itching is an unpleasant sensation that triggers the urge to scratch in order to alleviate the symptom. In contrast, scratching itself is usually perceived as pleasant. This pleasurable aspect, in particular, suggests the involvement of the dopaminergic reward pathway. The pathway extends from the ventral tegmental area (VTA) to nuclei within the limbic system [5,6]. The A10 dopamine (DA) cell group constitutes the main component of the VTA [7]. Itching sensations perceived in the skin reach the spinal cord via C-fibers and are transmitted to the central nervous system within the dorsal horn, where dopaminergic projection neurons may modulate the encoded itch signal. These projection neurons extend from the diencephalic A11 cell group to the dorsal horn [8].

These two distinct forms of dopaminergic neuromodulation via A10 and A11 neurons, respectively, appear to be promising targets for pharmacotherapy. Here we report a novel strategy of dopaminergic treatment in a female patient with chronic itching due to a probably hereditary form of notalgia paresthetica.

Case Presentation

A 76-year-old female patient presented in 2025 to one of the authors (TJF) with a long history of itching. Since the age of 13, the patient had experienced severe

itching, particularly medial to the left scapula, but occurring mainly during the cold season. During the winter months, the patient endeavored primarily to limit external influences on the itchy areas. Medical consultations took place first during childhood, and diabetes mellitus could be excluded. Dermatological and neurological assessments were repeatedly performed without any abnormalities. Especially, no focal motor or sensory deficits and no pyramidal tract signs were found. Local skin changes were not seen on the back. Spinal imaging (CT) was also normal. Family history was indeed remarkable: The same symptoms were present in the patient's mother, uncle (brother of her mother), and cousin (daughter of her uncle), suspecting a familial clustering. However, genetic or systematic clinical confirmations in relatives were not available. The patient herself gave birth to two healthy children. No further neurological disorders were known in the family. Due to the family history of the condition, symptoms were seen as an inevitable fate. Consequently, further therapy did not take place during life. In 2025, notalgia paresthetica was first diagnosed. No clinical evidence for multiple endocrine neoplasia in the history could be identified.

As an initial therapeutic approach, treatment with the DA receptor agonist pramipexole was initiated, but was ineffective at low doses of 0.09 mg/d or 0.17 mg/d when given for two weeks. It especially failed to alleviate the severe itching during the night that was preventing the patient from sleeping. A second therapeutic trial was therefore started with a daily dose of levodopa 50 mg plus the peripheral decarboxylase inhibitor benserazide 12,5 mg. After four days, medication showed clear success: itching was significantly alleviated, and the patient was now able to sleep through the night after taking the single dose in the evening. Itching-related treatment efficacy was validated using the visual analogue scale (VAS): VAS value declined from 8 before to 0–1 after treatment. This therapeutic effect persisted for six weeks. Thus, the levodopa dose could be reduced by half, which was sufficient to keep symptoms under control for the other four weeks. During treatment, adverse effects were monitored at one-week intervals and did not occur. Following a trial discontinuation of therapy for about 10 weeks, symptoms recurred, prompting the successful re-initiation of levodopa with symptom improvement five days after restarting, with a current dose of levodopa 50 mg and benserazide 12,5 mg/d until this day.

Discussion

In our patient, notalgia paresthetica was diagnosed. Although the etiology of the disease is still unclear, irritation of the dorsal branches of upper thoracic spinal nerves (T4–T6) is suspected [4]. Notalgia paresthetica commonly occurs in middle-aged patients, with an incidence of two to three times higher in women than in men [9,10]. Although the mean age of patients is between 50 and 60, notalgia paresthetica can occur in people during childhood [9,11], as in our patient. However, especially in younger patients, an association with multiple endocrine neoplasia 2B (MEN-2B) has been identified [9,10] and is, additionally, thought to be inherited [10]. In our patient, a hereditary form of the disease is, in principle, possible. However, genetic analysis was not performed.

As a therapy-related hypothesis, we would like to propose an underlying A10/A11 DA mechanism. A11 dopaminergic neurons projecting to the spinal dorsal horn lack the DA reuptake mechanism by the DA transporter (DAT) system [8]. In contrast to A11 neurons, A10 DA neurons from the ventral tegmental area (VTA) do possess this transporter system [7]. Both dopaminergic systems play a crucial role in chronic itch. The centripetal transmission of itch signals is modulated by inhibitory A11 dopaminergic neurons in the spinal cord through especially D2 and D3 receptor subtypes on one hand, by the DA-mediated pleasurable sensation associated with scratching via the A10 VTA neurons on the other. The shared dopaminergic basis underlying both the modulation of centripetal itching signals and the pleasurable sensation highlights DA-based therapeutic concepts for chronic itching.

Due to the absence of DAT in A11 neurons, even low-dose levodopa would significantly increase extracellular DA amounts and thus also the duration of postsynaptic inhibitory action of DA in the dorsal horn of the spinal cord, as described, for example, in DAT knockout mice [8]. Due to the absence of DAT and partly because DA is stored in vesicles, intracellular cytosolic DA concentrations would remain low. This would imply, additionally, low feedback inhibition of tyrosine hydroxylase activity in the cytoplasm, with further ongoing endogenous levodopa and DA synthesis, respectively, supplementing the therapeutically administered medication and inhibiting spinal itch transmission.

Since the DAT system is present in A10 neurons, extracellular DA increase would not simultaneously take place in these neurons to the same extent as in A11 neurons, and enhancement of reward-related mechanisms would not be induced. Here, intracellular DA inhibits tyrosine hydroxylase, and DA synthesis probably decreases. These biochemical effects in A10 neurons would not facilitate the sense of pleasure in the context of scratching following chronic itch, despite levodopa treatment. Systemic levodopa administration would therefore need to be titrated to achieve an optimal relation between A10 and A11 neuronal activity.

Conclusion

Taken together, we present a hypothesis-generating observation describing low-dose levodopa associated with

sustained symptomatic improvement in a patient with chronic itching due to notalgia paresthetica. We propose further controlled investigations to prove a novel treatment concept with low-dose levodopa and the suspected neurobiological difference between A11 and A10 dopaminergic neurons.

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Both authors contributed equally to the work.

What is new?

Notalgia paresthetica, relatively common yet underdiagnosed and frequently resistant to therapy, is a sensory, usually unilateral neuropathy. The lack of established treatment guidelines makes treating notalgia paresthetica complicated, as it dramatically affects patients' quality of life. Here, the authors report a novel concept of dopaminergic treatment in a female patient with chronic itching due to a probably hereditary form of notalgia paresthetica.

List of Abbreviations

DA	dopamine
DAT	dopamine transporter
MEN-2B	multiple endocrine neoplasia 2B
VTA	ventral tegmental area

Conflict of interest
The authors declare that there is no conflict of interest regarding the publication of this article.

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Consent for publication
Written informed consent was obtained from the patient.

Ethical approval
Ethical approval is not required at our institutions for anonymous case reports.

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Summary of the case

1	Patient (gender, age)	Female, 76 years
2	Final Diagnosis	Notalgia paresthetica
3	Symptoms	Chronic itching
4	Medications	Levodopa
5	Clinical Procedure	Novel pharmacological treatment concept
6	Specialty	