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Meralgia paresthetica: Nerve stimulator-guided injection with methylprednisolone/lidocaine, a double-blind randomized placebo-controlled study

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Abstract

Background: Meralgia paresthetica is a mononeuropathy of the lateral femoral cutaneous nerve. A common therapy is injection with corticosteroids. The goal of this study was to analyze the effect of injection with methylprednisolone/lidocaine vs placebo.

Methods: After randomization, 10 patients received a nerve stimulator-guided injection with methylprednisolone/lidocaine, and 10 patients received saline. The primary outcome measure was pain (visual analogue scale, VAS).

Results: In the placebo group, there was a significant pain reduction (baseline VAS, 6.8; VAS week 12, 4.3; P = .014). The VAS score in the methylprednisolone group did not show a significant reduction (baseline VAS, 7.4; VAS week 12, 4.8; P = .053). There was no significant difference in pain reduction between the groups.

Conclusions: We found no objective evidence for benefit from nerve stimulator-guided injection with corticosteroids in meralgia paresthetica, although this study is limited by a small sample size. Future placebo-controlled studies using ultrasound-guided injection are warranted.

KEYWORDS

corticosteroid injection, lateral femoral cutaneous nerve, meralgia paraesthetica, nerve stimulator, pain, randomized controlled trial

Abbreviations: LFCN, lateral femoral cutaneous nerve; MP, meralgia paresthetica; VAS, visual analogue scale.

1 | INTRODUCTION

Meralgia paresthetica (MP) is a mononeuropathy of the lateral femoral cutaneous nerve (LFCN) characterized by pain, numbness, and tingling in the anterolateral region of the thigh. MP is often caused by compression of the LFCN near the anterior superior iliac spine,[1,2] passing under, through, or above the inguinal ligament. The majority of patients with MP have a good response to conservative therapy, such as weight loss.[3–5] Traditionally, the first choice of treatment after failure of conservative measures is a nerve block with an anesthetic and corticosteroids. This nerve block is often performed in a blind manner, although anatomical variability leads to a reported failure rate of this method as high as 60%.[6] Several authors have reported their experience with the use of guidance by ultrasound or a nerve stimulator for more accurate nerve localization.[7–9] Locating a nerve with a nerve stimulator is easy to perform and well tolerated by patients. This study aimed to analyze the analgesic effect of injection with methylprednisolone/lidocaine vs placebo, after localization with a nerve stimulator.

2 | METHODS

The study was designed as a double-blind randomized, placebo-controlled trial, that compared injection of 2 mL methylprednisolone/lidocaine (80 mg methylprednisolone, 20 mg lidocaine) with 2 mL saline 0.9%. Patients with a clinical diagnosis of MP, with symptom duration of 4 weeks or more, were eligible for inclusion. Exclusion criteria consisted of known allergies to steroids or lidocaine, pregnancy, coexisting conditions that may mimic MP, or a prior episode of MP treated with steroids. Informed consent was obtained.

Baseline characteristics, including pain severity, body mass index, and use of medication, were recorded by the primary investigator. Randomization and local injection were performed by a separate investigator. The syringe with the injectate was covered with aluminum foil, to ensure patient blinding to treatment. The patient and the primary investigator remained blinded until the end of follow-up.

2.1 | Procedure

The LFCN was located through electro-stimulation at 1 Hz (< 10 mA) with a nerve stimulator (Injection Needle, 27G Needle Electrode, Allergan Inc) until the patient reported an electric sensation in the area of the LFCN. At that time, the injectate was administered.

2.2 | Outcome measures

The primary outcome measure was pain, measured on a visual analogue scale (VAS), at baseline, weeks 4, 8, and 12. Secondary outcome measures were pain measured on an ordinal scale (pain worsening, no improvement, moderate improvement, or complete remission) and use of analgesics.

2.3 | Sample size

Sample size calculation was based on results from a previous study,[7] which showed a mean reduction of 6 points on the VAS 2 months after injection (mean ± SD; 8.1 ± 2.1 at baseline; 2.1 ± 0.5 at 2 months). Sample size was calculated using Lehr's formula, using a power of 80% and a two-sided significance level of 0.05. Assuming an SD of 2.1 and a difference in VAS score of 4 points between treatment modalities, this resulted in 5 patients in each group. However, because the calculation is based on results of a study with no control group, we aimed to include 10 patients in each group.

2.4 | Statistical analysis

Analysis was based on intention to treat. Statistical analyses were performed using IBM SPSS v 24.0. Descriptive statistics were used to compare baseline characteristics. The VAS scores at baseline, weeks 4, 8, and 12 were compared between treatment groups, using generalized estimating equations for repeated measures, with exchangeable correlation structure, with adjustment for age, gender, body mass index, VAS score at baseline, follow-up, and interaction between follow-up and treatment. All statistical tests were two-tailed with significance set at P-value < .05.

3 | RESULTS

From October 2015 through August 2017, twenty patients were randomly allocated to injection with methylprednisolone/lidocaine or with saline. Baseline characteristics are shown in Table 1. There were no significant differences in baseline characteristics between groups. Several patients had one or more diagnostic tests before or during the course of their treatment to exclude other underlying pathology. Eight patients in the methylprednisolone group and four patients in the placebo group underwent magnetic resonance imaging of the lumbar spine. Six patients in the methylprednisolone group underwent radiological examination of the hip region. In one patient, treated with placebo, electromyography was performed to exclude other causes. These tests did not reveal clinically relevant findings.

3.1 | Primary and secondary endpoints

In the placebo group, there was a statistically significant pain reduction in uncorrected VAS score after 12 weeks (VAS at baseline, 6.8; VAS at week 12, 4.3; paired t-test P = .014) (Figure 1). The uncorrected VAS score in the methylprednisolone group did not show a significant pain reduction (VAS at baseline, 7.4; VAS at week 12, 4.8; paired t-test P = .053). Statistical analysis showed no significant difference in reduction in VAS score between methylprednisolone/lidocaine and placebo, with a 95% confidence interval of the difference

TABLE 1 Baseline characteristics

	Methylprednisolone (n = 10)	Placebo (n = 10)
Age in years (SD)	56.6 (8.9)	52.3 (8.9)
Sex, female (n)	4	3
BMI, kg/m2 (SD)	27.7 (3.0)	29.7 (3.7)
Duration, months (median)	11.5	7
Side, left (n)	5	6
VAS score baseline (SD)	7.4 (1.8)	6.8 (1.3)

Abbreviations: BMI, body mass index; VAS, visual analogue scale.

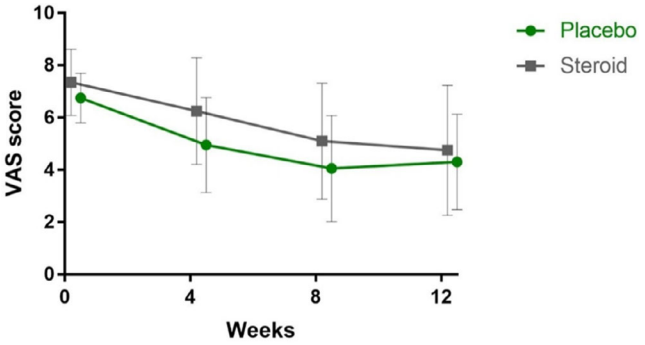

FIGURE 1 Visual analogue scale (VAS) score at baseline, weeks 4, 8, and 12 (uncorrected VAS score, error bars indicate 95% confidence interval for the mean) [Color figure can be viewed at wileyonlinelibrary.com]

(difference between groups in VAS reduction baseline/week 12) ranging from −2.8 to 3.1.

Analysis of pain on an ordinal scale (Supporting Information Table S1) and use of analgesics did not reveal any meaningful findings. There was no loss to follow-up.

3.2 | Adverse reactions

In the methylprednisolone group, one patient reported mild muscle fatigue, which lasted for several days after the injection. In the placebo group, no adverse events were reported.

4 | DISCUSSION

This study did not show evidence for a difference in treatment effect between methylprednisolone/lidocaine and placebo.

A Cochrane review[5] from 2012 pointed out the weak objective evidence base for treatment choices in MP, due to the lack of randomized placebo-controlled studies, prompting the need for such studies. There are, however, several other more basic clinical questions with regard to MP and its treatment. First, there is insufficient knowledge about the natural disease course of MP. Any reported treatment effect might reflect a spontaneous recovery. Second, there is no consensus regarding dose, type of medication or injection volume. Different treatment regimens are applied throughout the world, most often a combination of lidocaine or bupivacaine with methylprednisolone, hydrocortisone, or triamcinolone.

There are several possible explanations for the lack of difference between methylprednisolone/lidocaine and placebo in our study. First, the use of nerve stimulator-guided injection: although easily performed, it is not possible to directly visualize the spreading of the injectate around the compressed nerve with nerve stimulator-guided injection (as opposed to ultrasound-guided injection), in the open anatomy of the abdominopelvic cavity. Second, we used a relatively low injection volume (2 mL, as compared to 9 mL in the study by Tagliafico et al.[7]). A larger injection volume might positively influence treatment outcome, perhaps partially explained by an effect through "hydrodissection," the effect of introducing a fluid around an entrapped nerve. Mulvaney presented[9] a patient with long-term pain relief after ultrasound-guided injection of 1.5 mL of 0.5% lidocaine, termed hydroneuroplasty. The author hypothesized relief of ischemia as a plausible mechanism behind this therapeutic effect. The injection of saline in our placebo group might have resulted in the same therapeutic effect in some of our patients, although not visually confirmed by sonography.

A possible limitation of our study is the inclusion of patients based on clinical diagnosis only. This is not unique,[1,8] and in a patient with a typical history and neurological examination, confirmation is rarely needed. Furthermore, nerve conduction studies for the LFCN in this overweight population are difficult to perform,[10,11] and sonography as a diagnostic tool lacks consensus about normal and abnormal values of the cross-sectional area of the LFCN.

A further limitation of this study is the small sample size, with large confidence intervals: a different study population or a larger sample size could give different results. We, however, propose that future placebo-controlled studies focus on ultrasound-guided injection, using a larger volume (combination of corticosteroid and anesthetic) and a larger sample size.

5 | CONCLUSION

This study did not show a significant difference in treatment effect between methylprednisolone/ lidocaine and placebo, after localization of the lateral femoral cutaneous nerve with a nerve stimulator. Future randomized placebo-controlled trials are needed to explore the possible benefits of ultrasound-guided injection, using a standardized dose and injection volume.

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The trial was approved by the Medical Ethics Committee of the Haga Hospital, The Hague.

CONFLICT OF INTEREST

None of the authors has any conflict of interest to disclose.

ETHICAL PUBLICATION STATEMENT

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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Small-fiber neuropathy associated with autoinflammatory syndromes in children and adolescents

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Abstract

Introduction: Small-fiber neuropathy is rare in children. It has been associated with several autoimmune disorders, but there are no reports of an autoinflammatory etiology.

Methods: The data of four children/adolescents presenting with erythromelalgia and neuropathic pain from 2014 to 2019 were collected retrospectively from the electronic database of a pediatric medical center.

Results: Results of clinical and/or electrophysiological evaluation excluded large nerve fiber involvement. Skin biopsy results confirmed small-fiber neuropathy. According to genetic analysis, two patients were heterozygous and one was homozygous for mutations in the familial Mediterranean fever (*MEFV*) gene. Behcet disease was

Abbreviations: FMF, familial Mediterranean fever; IENFD, intraepidermal nerve fiber density; IL, interleukin; SFN, small-fiber neuropathy.