Case Report

A Novel Treatment of Postherpetic Neuralgia Using Peppermint Oil

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Abstract:

Background: Postherpetic neuralgia remains a difficult problem to treat. A number of therapies have been shown to be effective, but some patients have intractable pain.

Patient: The case of a 76-year-old woman whose pain had been resistant to standard therapies is described. The pattern of quantitative sensory testing results for this patient led the authors to believe that she had an “irritable nociceptor” type of pathophysiology.

Intervention: The patient was instructed to apply neat peppermint oil (containing 10% menthol) to her skin, resulting in an almost immediate improvement in her pain. This pain relief persisted for 4–6 hours after application of the oil.

Results: The patient was successfully treated with topical peppermint oil. During 2 months of follow-up she has had only a minor side effect, with continuing analgesia. The authors believe this is the first evidence of peppermint oil (or menthol) having a strong analgesic effect on neuropathic pain. The possible mechanisms of action of peppermint oil are discussed.

Key Words: Menthol—Neuropathic pain—Peppermint—Postherpetic neuralgia—Treatment.

Postherpetic neuralgia (pain persisting in the dermatomes affected by herpes zoster infection for >3 months) remains a difficult and often intractable pain problem. Current treatments that are effective in many patients include tricyclic antidepressants,1 anticonvulsants,2 topical capsaicin,3 and local anesthetics.4 A subpopulation of patients, however, do not gain any relief from these treatments or find the side effects intolerable.

We describe the novel use of topical peppermint oil in the treatment of a patient for whom the aforementioned therapies were unsuccessful.

CASE REPORT

A 76-year-old woman had been under the care of the University College London Hospitals Pain Management Center for 14 months for postherpetic neuralgia. Three years previously she had had a herpes zoster infection, affecting her right breast and the inner aspect of her upper arm (T2 dermatome). She described her current pain as a constant burning, with intermittent shooting pains that were rated as severe on a short-form McGill pain questionnaire, giving a total score of 12. She rated her pain in previous weeks on a visual analogue scale (0–100 points) with a score of 73. Her pain was exacerbated by heat, and she gained some relief from cold. She exhibited marked allostyrinia, especially from clothes, and would avoid wearing underwear and heavy clothing. On quantitative sensory testing, performed a month before the start of treatment, she displayed marked dynamic
mechanical allodynia (to a soft brush stroke) and static mechanical hyperalgesia and hyperpathia (to von Frey hairs). Detection and pain thresholds to hot and cold stimuli, as measured by means of Peltier principle thermodes (Thermotest; Somedic AB, Hörby, Sweden), were normal.

The patient had tried numerous drug treatments over the previous 3 years but experienced little benefit. She had been prescribed the tricyclic antidepressants amitriptyline and dosulepin and the anticonvulsants carbamazepine and gabapentin, but these yielded no relief. She had also reported that systemic sodium channel blockade, in the form of intravenous lidocaine, was unhelpful. Topical local anesthetics in the form of EMLA cream (Astra Pharmaceuticals, Wayne, PA, U.S.A.) had also been used, but she found that the plastic film used to hold the cream in place exacerbated the allodynia. Capsaicin cream exacerbated her burning pain, so she had stopped using it. A thoracic epidural (with steroids) and acupuncture gave no relief. Further therapeutic options under consideration included an intercostal nerve block and a cognitive behavioral approach to her pain management.

It is now widely recognized that postherpetic neuralgia is not a single disorder in terms of the underlying neuropathophysiology. The pattern of distinct tactile allodynia without thermal hypesthesia in our patient fits the "irritable nociceptor" picture. Patients with irritable nociceptor have sensitization and ectopic activity in intact C fibers, resulting in spontaneous pain. The tactile allodynia is considered to be due to rewiring of the dorsal horn so that Aδ input is received and processed by nociceptor-specific neurons.

Menthol has been shown to reduce thermal C-fiber activity, and with this in mind we recommended that the patient use peppermint oil as a putative topical analgesic. She had a patch test performed in the outpatient clinic to detect any acute adverse reaction to the oil, but the results were negative. She was then instructed to purchase some peppermint oil from a local health food shop. She bought peppermint oil (Perfectly Pure; Holland and Barrett, Warwickshire, U.K.), containing 10% menthol, and massaged a small amount of the neat oil (2 or 3 drops) into the affected skin 3 or 4 times a day. The patient telephoned us 3 days after starting the treatment to say that she obtained dramatic pain relief after the first application. At a 2-week follow-up, the patient stated that she was continuing to gain considerable relief from the postherpetic neuralgia and that she had experienced no systemic side effects from the oil. However, she reported that when she applied the oil to her skin she felt an immediate stinging sensation, lasting approximately 2 minutes, followed by almost complete pain relief. This relief would usually last for approximately 6 hours. She believed that she had been able to sleep more easily (possibly because of the soothing smell of the peppermint as well as the analgesia, she said), but she did still feel pain to light touch. She reported a reduction in both the intensity of burning pain and the frequency and intensity of shooting pains. When asked again to rate on a visual analog scale the pain she had felt in the previous week, she gave a score of 19. A short-form McGill pain questionnaire was completed again and yielded a score of 5. The patient appeared happier and more relaxed and stated that this was the first time that she had gained any relief from the pain of her postherpetic neuralgia. Quantitative sensory testing was repeated and showed a reduction in hyperpathia, with no change in the intensity of dynamic mechanical allodynia or static mechanical hyperalgesia. Thermal detection and pain thresholds remained unchanged.

She was interviewed by telephone for follow-up 8 weeks after the initiation of treatment. She reported continuing analgesia from application of the oil but also the appearance of a local side effect: the treated skin had started reddening after 4 weeks of use. As directed, she stopped applying neat oil and instead used a 1:5 dilution with almond oil (to give a menthol concentration of approximately 2%). The diluted oil prevented reddening and produced adequate pain relief, which she felt was slightly less than that produced by neat peppermint oil. The patient said she still used undiluted oil when the pain was particularly bad. She stated that when the effect of the oil had worn off, the returning pain was of the same intensity as that before she started the treatment.

**DISCUSSION**

We believe this is the first evidence of a strong analgesic effect of peppermint oil on neuropathic pain. In a letter to the editor of *The Lancet* in 1870, Dr. A. Wright said that he had witnessed peppermint oil being used to treat "facial neuralgia" in China and that he had been able to repeat this success in his own practice. Peppermint oil or menthol, its key ingredient, is often included in varying concentrations in over-the-counter topical remedies for musculoskeletal pain, and peppermint oil is prescribed clinically as pure oil in capsules for the treatment of irritable bowel syndrome. Little has been published on its analgesic effect on any condition other than irritable bowel syndrome, for which its use is still controversial.

The exact mechanism of action of peppermint oil is unknown. Menthol is the principal ingredient of the oil,
at concentrations of up to 55%, and is presumed to be the key active ingredient. Menthol selectively affects subpopulations of thermoreceptors characterized by conduction velocity as either C or Aδ fibers, depending on dose.

Exactly how peppermint oil relieves the burning pain of postherpetic neuralgia we can only postulate. One explanation is that it has a direct inhibitory effect on the sensitized nociceptors. A second putative explanation is that central gating mechanisms underlie the analgesic effect of menthol. It is believed that in normal skin, Aδ input produces a tonic inhibition of C-fiber input. If this central control is preserved in a neuropathic pain patient, menthol stimulation of Aδ fibers may result in central inhibition of afferent inputs from the ectopic C fibers thought to mediate burning pain.

The main side effect of topically applied peppermint oil is that of contact dermatitis. We have been unable to determine the incidence of this rare side effect, but there have been a number of case reports in the dermatologic literature, usually describing patients who have a reaction to peppermint oil or menthol contained in toothpaste. The reddening that our patient saw was possibly the start of this; fortunately, a reduction in the strength of the oil appeared to cure the problem, but this is something that would require close attention in other patients. When given orally, peppermint oil has been shown to have only minor adverse effects.

It is interesting to note that after 6 weeks of regular use of the oil, the patient reported that when its effect wore off the pain returned with the same intensity as that incurred before treatment. This implies that the oil provides only temporary relief; permanent treatments, therefore, should still be sought during its use. Irritable nociceptor pain may be controlled by capsaicin creams, but because their nociceptors are sensitized, patients with this condition are least likely to be able to tolerate the treatment. Menthol has been shown to have an antinociceptive effect on the burning pain produced by capsaicin in healthy volunteers. If this desensitization is shown clinically, then menthol may have an important role as an adjunct to capsaicin treatment.

In summary, we have shown evidence in this single case study of a remarkable analgesic effect in response to topical application of peppermint oil, an effect not previously described in the recent scientific literature (MEDLINE search for the period 1966–2000). Because of the patient’s negative response to all other treatments given, we believe that it is highly unlikely that a placebo effect was responsible for the patient’s analgesia; however, this possibility cannot be completely discounted.

We are currently in the process of setting up a double-blind, randomized, placebo-controlled trial to scientifically evaluate the analgesic effectiveness of peppermint oil in patients with neuropathic pain and to establish whether treatment with peppermint oil could be a useful clinical tool in pain management.

REFERENCES

8. Wright A. Oil of peppermint as a local anaesthetic. Lancet 1870; 2464:726.