Review: Tricyclic antidepressants, capsaicin, gabapentin, and oxycodone are effective for postherpetic neuralgia


Q U E S T I O N
In patients with postherpetic neuralgia (PHN), are any treatments effective in reducing pain or disability?

D A T A S O U R C E S
Studies were identified by searching MEDLINE (1966 to October 2000) and the Cochrane Controlled Trials Registry with the terms postherpetic neuralgia, neuropathy, and pain; searching Current Contents, bibliographies of relevant studies, and the U.S. Food and Drug Administration Web site; and contacting authors and content experts.

S T U D Y S E L E C T I O N
English-language studies were selected if they were full reports of randomized controlled trials that included patients with PHN (history of herpes zoster, pain in the dermatomal distribution of the zoster rash, and pain persisting after resolution of the rash) and addressed relevant outcomes (pain resolution, pain severity, or quality of life).

D A T A E X T R A C T I O N
2 reviewers independently reviewed trials for quality and extracted data on patient age and duration of PHN, type of therapy, treatment dosage and duration, results, and adverse effects.

C O M M E N T A R Y
The initial treatment choice for PHN usually lies between tricyclic antidepressants and anticonvulsants (the most widely studied being gabapentin). Although some have argued for making the choice based on the character of the pain (e.g., antidepressants for burning pain and anticonvulsants for shooting pain), no strong evidence exists for this distinction. In the review by Alper and Lewis, the authors conclude that the strongest evidence for effectiveness in PHN exists for tricyclic antidepressants.

Approaching PHN as just one subset of neuropathic pain leads to a slightly different conclusion. In a previous systematic review that included trials of both PHN and diabetic neuropathy, Collins and colleagues (1) found that the numbers needed to treat to reduce neuropathic pain by 50% were identical for antidepressants and anticonvulsants (2.9 for both). Both types of drugs had a similar incidence of minor adverse effects (number needed to harm [NNH] of 2.7 for both). They defined a major adverse effect as one leading to withdrawal from treatment. Using this definition, they found an NNH of 17 for antidepressants. For anticonvulsants, no significant difference in major adverse events existed between active treatment and placebo.

Approaching pain from this perspective provides a counterpart to the approach used by Alper and Lewis: It gives us an interesting example of the “lumping versus splitting” debate in research synthesis. By focusing on the type of pain rather than the cause, the review by Collins and colleagues drew on a larger data set. This led to more precise estimates of benefit and harm for the different classes of drugs. The clinical implications of the review by Alper and Lewis are that tricyclics are the first-line treatment and anticonvulsants the second. The implication of the review by Collins and colleagues, on the other hand, is that anticonvulsants may be the first choice on the basis of a better side-effect profile. The limitation of that conclusion is that most trials report the number of side effects but not their severity. Many clinicians perceive the central nervous system side effects of anticonvulsants (giddiness, dizziness, and gait and visual disturbance) to be more severe than those of moderate-dose tricyclics (dry mouth and hangover). In the absence of a head-to-head comparison, the choice of which to use first remains a matter of clinical judgment.

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R e f e r e n c e