Gabapentin Therapy for Vulvodynia

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Vulvar pain may be the presenting symptom of a variety of unrelated disorders (1). “Essential” vulvodynia or “burning vulva syndrome” is a chronic, idiopathic pain syndrome characterized by unremitting, diffuse burning of the vulva. Allodynia and hyperpathia are present despite a lack of clinical findings. The condition is accompanied by psychological disability, severe preoccupation with the pain, and limitation of daily activities. Its diagnosis is one of exclusion, particularly of chronic vestibulitis (2). Of the various therapies that have been recommended, none has been proven successful.

Recently, the novel anticonvulsant gabapentin (GBP) has been reported effective in the treatment of a variety of chronic pain conditions, particularly those of neuropathic pain (3). Based on these encouraging reports and our conviction that vulvodynia represents an unexplained neuropathic process, we chose to try gabapentin treatment in a group of patients. This is the first published report of its use in vulvodynia.

Case Report

Patients suffering from vulvar pain were screened for dermatoses, infection, and other gynecologic or systemic causes by history, physical examination, and relevant testing including pap smear, wet mount, vaginal cultures, and biopsy. Atrophy as a primary cause was ruled out by a trial of hormone replacement therapy.

GBP dosage was begun at 300 mg per day and continued for 1 wk. In the absence of relief or side effects, the dosage was increased weekly to 300 mg twice a day, 300 mg three times a day, then 300 mg four times a day. If relief was achieved, the dosage was not increased. Once relief was achieved or a daily dosage of 1200 mg/day was reached, the dosage was maintained for at least 12 wk. Thereafter, dosage was tapered on an individual basis.

Patients were asked to categorize their relief as excellent, good, poor, or none. Excellent relief meant the complete absence of pain; good relief, the quality of life was measurably improved; poor relief, pain relief was inadequate to improve quality of life; none, no reduction in pain level was detectable. None or poor relief was deemed treatment failure.

Seventeen consenting patients, ages 26 to 82 yr (median 62) were treated. Twelve (70%) were postmenopausal. All except one patient had been symptomatic for more than 1 yr (median, 4 yr; range, 10 mo to 11 yr). Most patients had failed to gain relief with multiple other treatments. Twelve patients experienced failed prior treatment with amitriptyline. Fourteen patients (82%) had either partial or complete relief with GBP therapy. Seven had complete relief and seven had significant pain relief. There were three failures of treatment. Patients typically reported symptom relief at between 2 and 4 wk from onset of treatment.

Although side effects (headaches, nausea, vomiting, fatigue, and dizziness) were reported in more than half the patients (9/17), these were in all cases, except one, transient and/or mild. Follow-up extended between 26 and 32 wk for all patients. There were no cases of late failure of therapy. Four patients were able to terminate treatment without recrudescence of symptoms.

Discussion

Vulvodynia has features which are characteristic of other chronic neuropathic pain conditions. These include the persistent and burning quality of the pain, the allodynia and hyperpathia, the absence of physical findings on examination, and the patient’s obsession with the pain.

Amitriptyline has been recommended as the treatment of choice for vulvodynia (4). Our experience with the drug, however, has been disappointing. In a prospective, placebo-controlled trial, we found limited treatment success (5). Most of the patients in this trial, in fact, had had no relief from previous amitriptyline treatment. Moreover, amitriptyline has a number of side effects (e.g., sedation, lethargy, weakness, dizziness, dry mouth, visual disturbance, tinnitus, and palpitations), which are frequently distressing to patients and promote noncompliance.

Recently, GBP has been shown to be highly effective in the treatment of neuropathic pain (3) and in states of spinally facilitated nociceptive and afferent processing (6). Its use has been demonstrated in the treatment of a variety of chronic pain conditions (7–12). Corroborating these early clinical reports, animal studies
have shown a GBP reversal of tactile allodynia (13,14), thermal hyperalgesia (15,16), and Phase II of the formalin test (17). GBP does not, however, alter response levels or thresholds to acute pain (15,17,18).

The mechanism of action of GBP remains unknown. It is structurally related to -aminobutyric acid (GABA), but neither binds to GABA-A or GABA-B receptors, nor inhibits reuptake or metabolism of synaptic GABA (19). Its action is unaffected by antagonists of either GABA-A or GABA-B (13). However, GBP does cause accumulation of GABA in many areas of the central nervous system through increased either production or release (19,20). An increase in spinal GABAergic tone could explain the effectiveness of GBP, because both GABA-A and GABA-B receptor agonists reduce the second phase of the formalin test (21) and the allodynia and thermal hyperalgesia of experimental neuropathy models (22).

The benefits of GBP include its low side effect profile and its minimal interaction with other medications. Ness et al. (11) reported on the use of GBP in 260 patients suffering from chronic pain of multiple etiologies. Only 14% of their patients quit the trial as a result of side effects of the medication (primarily drowsiness-confusion, dizziness-imbalance, and nausea). Seventy-three percent of their patients reported less pain, and of these, 54% maintained a long-term benefit. Of those patients suffering from peripheral neuropathic pain, 87% had improvement with GBP.

The results of this therapeutic trial parallel other reports in the literature of minimal side effects and dramatic relief of chronic pain with GBP. Our 82% response rate corresponds closely to the 87% response rate reported by Ness et al. (11). Unlike that report, however, we did not see late failures in maintenance therapy. It is possible, though, that our follow-up period did not extend long enough to note this phenomenon.

This is the first published report of the use of GBP in the treatment of vulvodynia. It demonstrates a high success rate in the treatment of these patients and a dramatic improvement in their quality of life. Because vulvodynia is a diagnosis of exclusion, we emphasize the caveat of the importance of correct diagnosis. Based on these findings, we believe consideration of GBP in the treatment of vulvodynia is warranted.

References