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Original Contribution

Topical antacid therapy for capsaicin-induced dermal pain: a poison center telephone-directed study

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Abstract

Purpose: The study aimed to assess the effects of topical antacids for treatment of capsaicin-induced dermal pain after exposure to capsaicin containing hot peppers, personal protection sprays, or topical creams.

Procedures: Participants of the study were California Poison Control System (CPCS) hotline callers 12 years or older with dermal pain from exposure to capsaicin-containing products or plants. Participants were instructed to apply a topical antacid and assessed for perceived pain (using a 0-10 scale) pre- and posttreatment. A positive response was defined as a sustained reduction of pain 33% or more within 30 minutes or achieving a pain score of 0 to 1.

Main findings: Of 93 eligible patients, 64 applied antacids and had outcome data available. Patients contacted the CPCS a median of 1 hour postexposure with a median initial pain score of 7.5/10. Thirty-six (56%) were exposed to unrefined (natural) peppers and 28 (44%) to refined capsaicin (eg, capsaicin-containing cream). Before calling the CPCS, 57 (89%) attempted at least one treatment. Forty-five (70%) reported positive response to antacid treatment as a 33% reduction in pain in 30 minutes (n = 17), a reduction in pain to a score of 0 to 1 (n = 3), or both (n = 25). A 33% reduction in pain within 30 minutes was associated with exposure to refined capsaicin (odds ratio, 3.37; 95% confidence interval, 0.98-11.66). Concomitant refined capsaicin exposure and early treatment (<1 hour of symptoms) was associated with even greater odds of response (odds ratio, 5.4; 95% confidence interval, 1.4-21.2).

The study findings were presented as a poster at the 2002 North American Congress of Clinical Toxicology conference in Palm Springs, Ca. In conjunction with this meeting, the findings were published in abstract form in the *Journal of Toxicology-Clinical Toxicology* [Kim SY, Anderson IB, Kearney TE. A prospective study evaluating the effectiveness of liquid antacid application for the treatment of capsaicin-induced dermatitis. (Abstract 56) *J Toxicol-Clin Toxicol*. 2002;40(5):621-622].

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Conclusion: Topical application of antacids for capsaicin-induced pain is effective, particularly in early treatment of exposure to refined capsaicin.

1. Introduction

Capsaicin, the pungent, active compound in the *Capsicum* plant genus, is found in several varieties of hot peppers. It is used in foods as a flavoring agent, in personal protection sprays and animal repellents, and in topical creams and ointments for the treatment of chronic pain. Dermal exposure of capsaicin affects cutaneous sensory neurons, inducing burning, redness, irritation, and pain that sometimes can be excruciating and last for hours to days after exposure [1].

Symptomatic exposures to capsaicin are common. In 2005, the American Association of Poison Control Centers reported 4377 exposures to "capsicum defense sprays" and 5305 exposures to "capsicum/peppers." [2] Capsaicin-containing topical treatments were not reported as a distinct category in these data, and thus, any adverse reports due to such products were presumably included in the latter group. A variety of topical treatments to alleviate the symptoms of capsaicin exposure have been suggested in the medical literature as well as on Internet sources [3-7]. These include milk, bleach, ice water, vinegar, and vegetable oil. A study of vegetable oil application vs cool water found that although cool water immersion initially provided more relief, vegetable oil provided better long-term relief from "chile burns" of the hands [5]. None of these treatments, however, effectively results in prompt, sustained pain relief. Local anesthetics offer relief for some patients but are rarely accessible in the home [3].

In 1998, Herman and colleagues [8] described 7 patients who had severe dermal discomfort secondary to a "pepper-mace" exposure. Within minutes after the application to the affected area of Liquid Maalox (ingredients: magnesium hydroxide and aluminum hydroxide), the patients were completely pain-free. In a recent double-blind randomized controlled trial, 10 volunteers were sprayed with 10% capsaicin on both forearms simultaneously. One arm was treated with Maalox, the other arm with saline. The Maalox treatment resulted in significant reductions in perceived pain (10-point visual analog scale) at 10, 20, and 30 minutes; at 60, 90, and 120 minutes, pain in the treated arm was also reduced compared to the control arm, but these reductions were not statistically significant [9]. Another controlled study (presented as an abstract only), however, comparing Maalox, lidocaine, baby shampoo, milk, and water for treatment of pepper spray exposures found no differences in pain among the treatment groups [10]. We report our experiences with topical antacid application for capsaicin-induced dermal pain among a typical population with symptoms troubling enough to prompt a phone consultation with a poison control center.

2. Methods

2.1. Study design

We conducted an observational clinical study of topical antacid application for treatment of dermal capsaicin exposures using a convenience sample of cases serially contacting the telephone hotline of the California Poison Control System (CPCS), San Francisco, for consultation. Cases meeting a priori inclusion criteria were followed and analyzed prospectively. The study was approved by the Committee on Human Research at the University of California, San Francisco.

2.2. Setting

The CPCS is a 24-hour emergency telephone consultation service whereby licensed specialists in poison information or poison information providers evaluate poisonings and provide treatment advice to health care professionals and the lay public. The CPCS fields more than 200 000 calls related to human exposures annually. For each consultation, a computerized record is generated in accordance with criteria established by the American Association of Poison Control Centers. Study participants were recruited from callers to the CPCS who reported dermal exposure to capsaicin over the 15 month period between January 2001 and April 2002.

2.3. Selection of participants

Patients eligible for participation in the study met the following inclusion criteria: (1) self-reported history of dermal exposure to a capsaicin-containing material, (2) dermal symptoms consistent with contact of capsaicin on intact skin (ie, not abraded), (3) ability to assess and categorize discomfort using a 0 to 10 pain scale, (4) 12 years or older. We limited inclusion to persons 12 years old or older because we anticipated that persons younger than this would be unable to evaluate and report accurately their discomfort using the 0 to 10 pain scale, thus leading to unacceptable measurement error in a key study variable. Patients were excluded from the outcome analysis for the following reasons: (1) antacids were not used after contact with CPCS despite a recommendation to do so; (2) patients lost to follow-up (wrong phone number provided or unable to reach after repeated attempts); and (3) otherwise eligible patients not formally enrolled by the CPCS staff and were therefore missing key study variables.

2.4. Capsaicin source

We classified the capsaicin exposure according to its source: either unrefined capsaicin (peppers such as jalapeño, red chili, or habañero) or refined capsaicin (as contained in personal protection sprays (eg, "pepper spray," animal repellents, or a cream or ointment [eg, Zostrix cream]). We did not have any quantifiable measurements of capsaicin content available for most sources. For example, we did not attempt to collect the peppers and measure their capsaicin content in "Scoville Heat Units" nor did we solicit a subjective assessment from study participants as to the "hotness" of the pepper involved.

2.5. Treatment interventions

For the purposes of this study, antacids were defined as any divalent cation-containing product, such as antacids that contain calcium or magnesium (eg, Maalox, Mylanta, Milk of Magnesia [marketed as both a laxative and an antacid]) or antacid tablets (eg, Tums). Patients agreeing to participate in the study were advised to apply or soak the affected area liberally with a room-temperature antacid product as defined above. If only antacid tablets (eg, Tums) were available, the patient was advised to crush the tablets, create a slurry by mixing the crushed material with water, then apply to or soak the affected area with this slurry. Patients without access to, or unwilling to use, the antacids listed above were advised to apply other treatments cited in previous publications, such as ice water, milk, vegetable oil, baking soda, or local anesthetics. These non-antacid treatments were not included in the outcomes analysis.

2.6. Study measures

At the initial assessment, standard CPCS case data collection procedures recorded age, sex, product involved, time elapsed since exposure, and symptoms experienced. All data were entered as a case record, with a unique case identifier, in accordance with CPCS policies and protection of caller confidentiality. Once study eligibility was established, the CPCS staff informed the patient (or the parent/guardian of those 12-18 years of age) of the study and read a scripted verbal consent. Once consent for participation was obtained, each study participant was asked: (*a*) "What treatments have you already tried?" and (*b*) "How would you rank your pain currently on a scale of 0 (no pain) to 10 (worst pain ever experienced)?" The 0- to 10-point pain scale has been validated previously for assessment of pain through telephone interviews [11]. An initial follow-up telephone call was attempted within 2 hours of the initial consultation to allow time for procurement of an antacid if not readily available as well as time for treatment application and assessment of outcome. At follow-up, patients were asked: (*a*) "What treatments did you use after calling the CPCS?" (*b*) "How would you rank your pain after the treatment on a scale of 0 (no pain) to 10 (worst pain ever experienced)?" and (*c*) if the treatment was beneficial, "How long did the therapy take to work?"

Consistent with previously published pain assessments, a positive response to treatment was defined as a sustained reduction in the pain scale ranking of at least 33% within 30 minutes [12-15]. In addition, a positive response was also defined as "resolution of pain" based on a pain scale ranking of 0 or 1 post antacid therapy. If pain relief was not sustained, the treatment was classified as ineffective and excluded from the outcome analysis. For example, if a patient experienced at least a 33% reduction in the pain scale rating within 30 minutes of ice water immersion, but the pain returned promptly upon removal of the affected area from the ice water, the treatment was deemed ineffective.

2.7. Data analysis

We compared the characteristics of study subjects who were included in the analysis of the outcomes of antacid-treated patients to those who were excluded using the χ^2 test and unpaired *t* test or Wilcoxon rank sum test, as appropriate. For subjects included in the antacid outcome analysis, we compared pre- to posttreatment pain levels using the paired *t* test for pain score as a continuous variable. To reduce the potential confounding effects of multiple treatments, only those subjects who used antacids as the initial treatment after CPCS contact were included in the analysis for antacid treatment. We further studied pain response using 2 other measures derived from the continuous scores: a 33% reduction in pain within 30 minutes or achieving a posttreatment pain score of 0 to 1. The former measure of response (a 33% pain reduction) was considered the primary study outcome (dependent variable). Exposure and treatment variables as potential predictors (independent variables) associated with this outcome were investigated using logistic regression analysis. We chose these variables a priori and tested separately in a series of simple logistic models. Reported *P* values are from standard χ^2 analysis, except for the analysis of response according to baseline pain score for which we performed a χ^2 test for trend. After these bivariate analyses, we tested a multivariable logistic regression model by using a forward stepwise approach (chosen because of the relatively small sample size). We first entered into the model the strongest predictor of response from the bivariate analysis (capsaicin source). Additional variables were incorporated into the model, beginning with the next strongest predictor of response, and were retained in the model if (1) they showed a statistical association as an independent predictor of outcome using a $P < .10$ α value for inclusion and if (2) they were potential confounders of the effect of other variables (based on modification of the point estimate of the odds ratio [OR] in the previous bivariate modeling). In addition, we tested for interactions between source of capsaicin and time until contact with poison center

in 2 ways: by including an interaction term in the statistical model along with those 2 measures and by estimating the association between the risk factors combined relative to their absence. Finally, "survival data" (ie, time until resolution of pain) were analyzed using a Cox proportional hazards model including the same predictors as were used in the multivariate logistic regression. Follow-up time was censored at 2 hours.

3. Results

3.1. Study population

Of the 93 patients who were eligible for the study, 29 were excluded for analysis of antacid outcome according to the criteria previously described. Of the 29 excluded, 19 did not use antacids, 7 were unable to be contacted for the follow-up assessment (wrong telephone number or unable to reach despite repeated attempts), and 3 were not properly enrolled by the CPCS staff. This yielded a sample size of 64 participants (68% of the eligible population) for the antacid outcome analysis. Of the 64 subjects, 8 were treated at a health care facility. Table 1 describes the characteristics of the study participants, according to whether they were included in the analysis of antacid outcome. In both groups, most patients were female, and the mean age was approximately 35 years. Unrefined capsaicin (ie, natural peppers) was responsible for most of the capsaicin exposures (36/64, 56%) in the outcome analysis group, whereas subjects excluded from analysis were exposed to refined capsaicin more commonly (18/29, 62%), although this difference was not statistically significant (P = .10). The median pain score at baseline was higher in the included subjects compared to those who were excluded (7.5 vs 6.0, respectively; P = .03). Duration of pain, defined as elasped time between exposure and contact with the CPCS, was less than 1 hour for most callers in both groups and did not differ statistically on this basis.

3.2. Treatment characteristics

In both included and excluded subjects, approximately 90% (57/64 and 26/29, respectively) had attempted at least one treatment before calling the CPCS. The most frequently used pretreatments by the included subjects were cold water/ice (39), soap (24), milk (11), and baking soda (7). Among the CPCS-recommended antacid treatments included in the outcome analysis, 44 (68%) of 64 of the antacids contained aluminum and magnesium, with the remainder containing magnesium, aluminum, and calcium (either alone or in combination).

Table 1 Characteristics of capsaicin-exposed patients by analysis group

Participant characteristics	Included in analysis (n = 64)	Excluded from analysis (n = 29)	P
Demographics			
Sex (n [%])			.26
Female	49 (76.6)	19 (65.5)	
Male	15 (23.4)	10 (34.5)	
Age in years (mean [SD])	34.5 (14.4)	35.6 (12.9)	.73
Exposure and treatment			
Capsaicin source (n [%])			.10
Natural peppers	36 (56.3)	11 (37.9)	
Refined products	28 (43.7)	18 (62.1)	
Any pretreatment before call (n [%])			.93
Pretreatment	57 (89.1)	26 (89.7)	
No pretreatment	7 (10.9)	3 (10.3)	
No. of pretreatments (median [range])	1 (0-5)	1 (0-3)	.11
Antacid cations used (n [%])			
Both magnesium and aluminum	44 (68.7)	Not applicable	
Other cations	20 (32.3)	Not applicable	
Pain characteristics			
Duration of pain before call (n [%])			.94
≤1 h	36 (56.2)	16 (57.1) [a]	
>1 h	28 (43.8)	12 (42.9)	
Pain duration in hours (median [range])	1 (0.08-24)	0.6 (0.08-13)	.56
Baseline pain score (median [range])	7.5 (1.5-10)	6 (2.5-10) [b]	.03
Final pain score (median [range])	2 (0.5-5.25)	Not applicable	

[a] n = 28, data missing for 1 subject.
[b] n = 25, data missing for 4 subjects.

3.3. Outcomes

The median final pain score across the 64 subjects analyzed for outcomes was 2 of a maximum possible score of 10. The mean change in pain scores was a 4.2-point decrease from the initial pain score ($P < .0001$; 95% confidence interval [CI], 3.3-5.0). Overall, 45 patients (70%) reported a positive response as a 33% reduction in pain within 30 minutes (17 patients), a reduction in pain to a score of 0 to 1 (3 patients), or both (25 patients). For the 28 patients who reported a reduction in pain score to 0 to 1, the mean time to reach that pain level was 16 minutes, ranging from 1 minute (immediate relief) to 60 minutes.

3.4. Predictors of response

Table 2 represents an analysis of the response to antacid therapy, specifically the association between study participant characteristics and 33% reduction in pain within 30 minutes of treatment. As shown, exposure to refined capsaicin products (as opposed to unrefined material, eg, chili peppers) was associated with a statistically significant greater likelihood of pain reduction response, with a marginally statistically significant increased odds of response seen among those with pain duration of one hour or less before calling the CPCS. The duration of pain before calling the CPCS differed significantly by capsaicin source. For those exposed to unrefined capsaicin, 61% (22/36) waited an hour or more before contacting the CPCS, whereas, in contrast, of those exposed to refined capsaicin products, only 22% (6/28) waited an hour or more ($P = .002$). We identified a difference in pain relief among different antacid formulations. Of the 44 patients who used antacids containing magnesium and aluminum, 70.5% reported a 33% reduction within 30 minutes compared to 55% for those using other preparations (a nonstatistically significant difference).

Multivariable logistic regression was used to analyze the independent effects of these variables on outcome. Type of cation administered and baseline pain score did not have statistically significant independent associations with outcome or act as substantial confounders of the relationship between capsaicin type and outcome and were thus excluded from the model. Duration of pain less than an hour before treatment did not have an independent effect on outcome but attenuated the effect of capsaicin type. Exposure to refined capsaicin was marginally associated with increased likelihood of response after adjustment for pain duration ($P = .05$; OR, 3.37; 95% CI, 0.98-11.66). There was no evidence for statistical interaction between capsaicin type and duration of pain before calling CPCS when an interaction term was included in the model (interaction term $P = .54$). Using a separate analytic approach, odds of response were similar in the groups exposed to refined capsaicin products whether they experienced greater than (OR, 6.0; 95% CI, 0.6-60) or less than an hour of pain before calling (OR, 5.4; 95% CI, 1.4-21.2) relative to the group exposed to unrefined products with greater than an hour of pain (the reference group). In the group exposed to unrefined capsaicin for less than an hour before calling, odds of a positive response were 2.2 times (95% CI; 0.5-8.6) the odds of response in the reference group.

The time to reduction in pain to a score of zero to one was further analyzed using a Cox proportional hazards model. No patient or treatment characteristics were statistically significant predictors of response in bivariate analyses (data not shown) or in a multivariable analysis using the same

Table 2 Response to topical antacid therapy among 64 treated cases followed for pain response: results of logistic regression analysis

Predictors of pain reduction	33% Pain reduction within 30 min		OR (95% CI)	*P*
	Yes	No		
All participants	42 (65.2%)	22 (34.8%)	N/A	N/A
Capsaicin source				.01
Unrefined	19 (52.8%)	17 (47.2%)	1.0 (Referent)	
Refined	24 (82.1%)	5 (17.9%)	4.11 (1.28-13.2)	
Pain duration before call				.07
>1 h	15 (53.5%)	13 (46.5%)	1.0 (Referent)	
≤1 h	27 (75.0%)	9 (25.0%)	2.6 (0.90-7.49)	
Baseline pain score quartile				.12
Lowest (1.5-5)	10 (58.8%)	7 (41.2%)	1.0 (Referent)	
Moderate (5.5-7.5)	10 (55.6%)	8 (44.4%)	0.88 (0.23-3.34)	
High (8-8.5)	9 (69.2%)	4 (30.7%)	1.56 (0.34-7.22)	
Highest (9-10)	13 (81.2%)	3 (18.8%)	3.03 (0.62-14.78)	
Any pretreatment before call				.73
No pretreatment	5 (71.4%)	2 (28.6%)	1.0 (Referent)	
Pretreatment	37 (64.9%)	20 (35.1%)	0.74 (0.13-4.01)	
Antacid cation				.23
Other cations	11 (55.0%)	9 (45.0%)	1.0 (Referent)	
Both magnesium and aluminum	31 (70.5%)	13 (29.5%)	1.95 (0.65-5.82)	

predictors as tested in the previous multivariate logistic regression (male sex, exposure to refined capsaicin, baseline pain score).

4. Discussion

Capsaicin is an excitatory substrate for the nociceptors (noxious stimuli detectors) of sensory neurons. Stimulation of these primary afferent fibers results in an influx of cations, which initiates action potential firing and subsequent release of neurotransmitters from central terminals in the spinal cord to generate acute pain. Activation also leads to release of proinflammatory peptides such as substance P and calcitonin gene–related peptide from the peripheral terminals of these afferent neurons, promoting vasodilation, vascular leakage, and inflammation [16,17]. An important hallmark of this cascade of events is local tissue acidosis. After repeated exposure to capsaicin, the receptors become desensitized, resulting in a lack of excitatory response to capsaicin and certain other painful stimuli. This desensitization is the basis for using topical capsaicin for treatment of chronic pain. The onset and extent of desensitization depends on many factors, including the dose and intervals between applications [17].

The activation of capsaicin-sensitive nociceptors is potentiated by high temperature and low pH [16]. Cool temperature may attenuate the calcitonin gene-related peptide–mediated vasodilation. Consistent with this mechanism, some of our hotline callers reported that soaking the affected skin area in cold liquids provided immediate pain relief, but only as long as they continued this treatment. The increased stimulation of nociceptors by protons has obvious implications for antacid therapy. In vitro studies suggest that extracellular protons increase the probability of receptor channel opening [16]. It is possible that antacids provide pain relief by raising extracellular pH, thereby decreasing receptor sensitivity.

The effect of antacids might also be due to the presence of divalent cations, particularly Ca^{2+} and Mg^{2+}. The permeability of the capsaicin-operated inward current during depolarization of the afferent neurons is greatest for Ca^{2+} and Mg^{2+}. In in vitro studies, removal of extracellular Ca^{2+} enhances the excitatory action of capsaicin [17]. Thus, providing Ca^{2+} and Mg^{2+} externally may suppress the actions of capsaicin. Nonetheless, whether these cations can interact with capsaicin-sensitive nociceptors across intact skin is unclear. In our study, of those who used antacids, 69% used a combination product containing magnesium and aluminum. Although not statistically significant, 70.5% of those using this combination reported a 33% reduction within 30 minutes compared to 55% for those using other preparations. Whether trivalent cations such as aluminum have a role in reducing pain from capsaicin can be a subject for further study.

We observed a greater likelihood of pain improvement measured as a 33% reduction in 30 minutes or less (although not a more rapid time until complete resolution) among those exposed to refined as opposed to unrefined capsaicin. This may be explained by a higher exposure level or skin penetration of capsaicin from unrefined sources (ie, from hot peppers rather than capsaicin containing sprays or creams), but we cannot formally test these hypotheses in this analysis.

As discussed in the Introduction, there are conflicting reports in the medical literature regarding the utility of topical antacid treatment of capsaicin exposures [8-10]. To our knowledge, ours is the largest study to examine the therapeutic effect of antacids for dermal capsaicin exposure. This study setting provides a feasible methodology and "real-world" experience base. However, this methodology placed several constraints on our experimental study design, including the inability to blind observers and study participants (thus being unable to control for placebo effects), randomize subjects to treatment arms, independently confirm treatment applications (because the study was dependent on subject self-reporting), or assess pain using a visual analog scale. Other limitations to the study include a lack of a "wash-out period" between the treatment methods, many of which were initiated by patients before calling the CPCS. Furthermore, we did not document dermal areas of exposure, address chronic skin conditions, or ascertain the concentrations of capsaicin in products, all of which could have affected responsiveness to antacid treatment. In regard to capsaicin content specifically, even if it had been possible to identify the specific species of peppers, this would have provided qualitative data at best. The "hotness" in peppers is measured by Scoville heat units, a subjective taste test that depends on the individual taste sensitivity. Although capsaicin concentrations can be determined by sophisticated laboratory analysis techniques, such methods are limited to the food industry [18]. Quantification for personal defense sprays presents similar difficulties: the oleoresin capsicum (OC) concentration reported does not measure the actual concentration of capsaicin in the formulation, which varies with the species of the pepper used in the product (eg, a product containing 5.5% OC can contain 5 times that amount of capsaicin, as assayed by high-performance liquid chromatography, as the amount found in a 10% OC product) [19]. Despite these limitations noted above, our findings do indeed support the use of an easily available self-treatment with a negligible risk-profile to alleviate pain due to a very common source of household exposures.

Application of topical antacids for capsaicin-induced dermal pain is a safe, inexpensive, and reasonable treatment approach of a frequent and often excruciating problem. Antacids may be particularly useful for early treatment of dermal exposure to refined capsaicin products.

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