Scalp Dysesthesia

Diane Hoss, MD; Samantha Segal, MD

Background: Cutaneous dysesthesia syndrome is a disorder characterized by chronic cutaneous symptoms without objective findings. Patients complain of burning, stinging, or itching, which is often triggered or exacerbated by psychological or physical stress. These symptoms may be manifestations of an underlying psychiatric disorder or may represent a type of chronic pain syndrome.

Observations: Eleven women presented with chronic severe pain and/or pruritus of the scalp only without objective physical findings, a condition we term “scalp dysesthesia.” Five women described pain, stinging, or burning only; 4 women complained of pain and pruritus; and 2 women reported pruritus only. The patients ranged in age from 36 to 70 years. The duration of symptoms ranged from 9 months to 7 years. Five women had physician-diagnosed psychiatric disorders, including dysthymic disorder, generalized anxiety, and somatization. Seven women reported that stress triggers or exacerbates their symptoms. Eight women experienced improvement or complete resolution of symptoms with treatment with low-dose doxepin hydrochloride or amitriptyline hydrochloride. One patient responded completely to treatment with sertraline and hydroxyzine hydrochloride but then experienced a relapse.

Conclusions: We describe 11 patients with a new syndrome that we term scalp dysesthesia. Of 11 patients, 9 benefited from treatment with low doses of antidepressants.

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The chronic pain syndromes include the burning mouth syndrome (synonyms: glossodynia, stomatodynia, oral dysesthesia, glossopyrosis, and stomatopyrosis), vulvodynia, scrotodynia, and atypical facial pain (synonyms: oro-facial dysesthesia). Patients with 1 of these syndromes report localized cutaneous or mucosal debilitating pain or burning, sometimes without abnormal physical findings. We describe 11 women with chronic distressing scalp symptoms, a condition we term “scalp dysesthesia.”

REPORT OF CASES

The characteristics of the 11 women with scalp dysesthesia are presented in the Table.

COMMENT

In 1981, Cotterill described 28 patients with “dermatologic non-disease.” These patients reported significant facial, scalp, and genital burning and discomfort or itching often associated with a disturbed body image (body dysmorphic disorder). Koblenzer and Bostrom coined the term “chronic cutaneous dysesthesia syndrome” to refer to patients whose primary cutaneous complaint is dysesthesia. Dysesthesia can be defined as “a disagreeable sensation present with ordinary stimuli.” Chronic cutaneous dysesthesia is a separate clinical entity from body dysmorphic disorder or a circumscribed delusion, such as delusions of parasitosis.

Examples of chronic cutaneous dysesthesia include the burning mouth syndrome, vulvodynia, scrotodynia, and atypical facial pain. When confronted with a patient complaining of localized pain, one must consider possible underlying localized organic disease, systemic organic disease, or psychological disease. In the case of burning mouth syndrome, the physician must rule out local disease (geographic tongue, candidiasis, or contact stomatitis), systemic disease (diabetes, xerostomia due to Sjögren syndrome or medication use, or vitamin deficiencies), or psychological disease. Similar consideration must be given to women with vulvodynia and patients with oro-facial dysesthesia.

Cutaneous disease was present in only 1 of our patients. Patient 6 had biopsy-proven prurigo nodularis of the scalp. These lesions were caused by constant picking of a pruritic and burning scalp in a patient with known atopic dermatitis. Her prurigo nodularis lesions, which were present for 5 years, completely disappeared when her scalp symptoms resolved. None of our patients exhibited signs of psoriasis or seborrheic dermatitis that might have caused their symptoms.

We also considered whether alopecia could have caused or contributed to the scalp dysesthesia in our patients. Of our 11 patients, 7 (2 premenopausal and 5...
<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Complaint/Duration, y</th>
<th>Scalp Conditions</th>
<th>Medical History</th>
<th>Associated Life Events</th>
<th>Psychiatric Disorders</th>
<th>Patient-Identified Exacerbating Factors</th>
<th>Type of Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/35</td>
<td>Burning, initial transient pruritus/2</td>
<td>Telogen effluvium</td>
<td>Premenopausal</td>
<td>Marital stress, new job</td>
<td>Dysthymic disorder (P)</td>
<td>“Worsens with stress”</td>
<td>Doxepin hydrochloride, 50 mg/d</td>
<td>CR, then recurred 3 times when doxepin use stopped; CR when restarted use</td>
</tr>
<tr>
<td>2/66</td>
<td>Pain, burning, stinging/7</td>
<td>Mild AGA</td>
<td>PUD, arthritis, postmenopausal using ERT</td>
<td>None</td>
<td>Hypochondriasis, anxiety disorder (PCP)</td>
<td>Denies</td>
<td>Doxepin hydrochloride, 30 mg/d; amitriptyline hydrochloride, 10 mg at bedtime</td>
<td>Improved symptoms; CR initially with mild recurrence</td>
</tr>
<tr>
<td>3/70</td>
<td>Tightness/4 (“like a helmet on my head”) worsened/1</td>
<td>Mild AGA</td>
<td>Family history of depression, postmenopausal with no ERT</td>
<td>Retired for 2 y</td>
<td>Refuses psychiatric evaluation</td>
<td>“I’m a very intense person”</td>
<td>Doxepin hydrochloride, 50 mg/d</td>
<td>Marked improvement</td>
</tr>
<tr>
<td>4/36</td>
<td>Pain with combing and cold, formication, tight feeling/3</td>
<td>Mild AGA</td>
<td>Premenopausal</td>
<td>Immigration</td>
<td>“Worsens with stress”</td>
<td>Doxepin hydrochloride, 50 mg/d; hydroxyzine hydrochloride, 50 mg/d</td>
<td>Improved symptoms, NR</td>
<td></td>
</tr>
<tr>
<td>5/40</td>
<td>Pain and burning/2</td>
<td>Mild AGA</td>
<td>Premenopausal</td>
<td>Marital stress</td>
<td>Anxiety disorder (PCP)</td>
<td>“Worsens with stress, anxiety”</td>
<td>Alprazolam, 0.25 mg/d</td>
<td>Mild relief of symptoms; (alprazolam) Noncompliant (doxepin); mild relief of symptoms (lorazepam)</td>
</tr>
<tr>
<td>6/58</td>
<td>Constant soreness, occasional burning/9 mo</td>
<td>Mild AGA</td>
<td>Postmenopausal using ERT</td>
<td>. . .</td>
<td>Worsens with wind, shower, and grooming, not worsened by stress, onset associated with “free-floating anxiety”</td>
<td>Amtriptyline hydrochloride, 20 mg at bedtime</td>
<td>90% resolution of symptoms 3 weeks after taking medication; CR after 10 months using medication; symptoms recur if amitriptyline use is discontinued</td>
<td></td>
</tr>
<tr>
<td>7/61</td>
<td>Pruritus/5; burning/2</td>
<td>Prurigo nodularis</td>
<td>Atopic dermatitis, postmenopausal using ERT</td>
<td>Generalized anxiety disorder (P), acute panic disorder (P), dysthymic disorder with obsessive and somatic components (P)</td>
<td>“Worsens with stress”</td>
<td>Intral Addison triamcinolone; Acetone hydroxyzone hydrochloride, 50 mg; topical doxepin; Diprolene gel, eclocon lotion, 1% hydrocortisone foam with 1% pramoxine, temovate cream; Doxepin hydrochloride, 40 mg/d</td>
<td>Combination of sertraline, 50 mg/d; hydroxyzine hydrochloride, 50 mg/d; intral Addison triamcinolone acetone, 10 mg/mL; and psychiatric consultation</td>
<td>NR</td>
</tr>
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(continued)
Distribution of scalp pain and the presence of normal erythematousness are the diffuse scalp pain or tightness. Features that militate against a diagnosis of temporal arteritis in our patients are the diffuse scalp burning associated with increased daily hair loss. In 1960, Sulzberger et al reported that women with “diffuse alopecia” complained of associated scalp symptoms, including “spotty tenderness, tingling, crawling, itching, burning and uncomfortable awareness of the scalp.” Mild AGA is common in women. Venning and Dawber noted mild AGA in 220 (87%) of 254 premenopausal women seen in a dermatology clinic for reasons other than hair loss. Thus, our patients do not have an increased incidence of AGA compared with the general population. It is unlikely that mild AGA is the sole cause of the scalp symptoms in these 7 women. Even if mild AGA is the cause, control of scalp symptoms can be achieved without changing the clinical finding of AGA.

Patient had documented telogen effluvium on 2 separate occasions. No cause was determined for the telogen effluvium. The results of a complete blood cell count, thyroid function tests, and iron studies were all normal. Treatment of her scalp burning with doxepin hydrochloride resulted in cessation of burning and decreased daily hair loss on both occasions. A recent review of telogen effluvium does not mention scalp burning associated with increased daily hair loss.

Temporal arteritis and tension headaches are underlying medical conditions with symptoms that may include scalp pain or tightness. Features that militate against a diagnosis of temporal arteritis in our patients are the diffuse distribution of the scalp pain and the presence of normal erythrocyte sedimentation rates in those patients tested. When queried, all patients stated that their pain did not resemble a headache. The 2 women who reported scalp pruritus without pain had negative or normal results of a laboratory workup for pruritus (complete blood cell count, liver function tests, and measurement of levels of blood urea nitrogen, creatinine, glucose, and thyroid stimulating hormone).

A psychiatric cause or overlay has been reported in patients with chronic pain syndromes or cutaneous dysesthesias. In fact, chronic pain has been referred to as a “depressive equivalent.” Many of the chronic pain syndromes were initially thought to represent a psychiatric disorder. However, one must be cautious when reviewing data describing a personality profile or a psychiatric disorder only. However, one must be cautious when reviewing data describing a personality profile or a psychiatric disorder only. Particularly a painful one. Is a psychiatric disturbance (i.e., depression) the cause of chronic pain or does experiencing chronic pain result in symptoms of depression? A subset of chronic pain sufferers probably do have an underlying psychiatric illness causing their symptoms. However, in the experience of Bowes, “most of the cutaneous/mucosal pain syndromes are not caused by psychological pathology.” Continued research into the chronic pain syndromes now suggests that they may represent a neurologic dysfunction that in some cases is associated with a secondary psychiatric component.

It is interesting that 5 of our patients had 1 or more physician-diagnosed psychiatric disorders. Dysthmic dis-

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</thead>
<tbody>
<tr>
<td>8/59</td>
<td>Pain and pruritus/4</td>
<td>Mild AGA</td>
<td>Postmenopausal using ERT</td>
<td>Mother's illness 4 years ago; mother's death 1 year ago; elderly widowed father</td>
<td>None</td>
<td>“I’m a nervous person,” “I thought it was due to stress”</td>
<td>Amitriptyline hydrochloride, 20 mg/d</td>
<td>CR after 3 months of treatment; symptoms recur if treatment discontinued</td>
</tr>
<tr>
<td>10/52</td>
<td>Pruritus/3</td>
<td>None</td>
<td>Postmenopausal using ERT</td>
<td>Father’s death; family stress</td>
<td>None</td>
<td>“Worse with stress, sweating, warmth, wearing hats”</td>
<td>Topical steroid, 30 mg/d; Nortriptyline hydrochloride, 25 mg at bedtime</td>
<td>NR, 40% improvement unknown</td>
</tr>
<tr>
<td>11/67</td>
<td>Tenderness, burning, and pruritus/1</td>
<td>Mild AGA</td>
<td>Postmenopausal without ERT</td>
<td>. . .</td>
<td>. . .</td>
<td>Worse in morning and combing or washing hair</td>
<td>Doxepin hydrochloride, 20 mg at bedtime</td>
<td>CR (tenderness and burning) in 6 months; CR (pruritus) in 10 months</td>
</tr>
</tbody>
</table>

*P indicates diagnosed by a psychiatrist or psychologist; CR, complete resolution of symptoms; AGA, androgenetic alopecia; PUD, peptic ulcer disease; ERT, estrogen replacement therapy; PCP, diagnosed by patient’s primary care physician; NR, no response; and ellipses, not applicable.
order, a mood disturbance characterized by a chronically depressed mood, was present in 2 patients. Prior somatization was present in 3 patients. Generalized anxiety was present in 4 patients. Of these 5 patients, 4 had psychiatric problems that were present prior to the onset of scalp dysesthesia. Of the 11 patients we describe herein, the symptoms in 7 patients intensified with psychological stress.

A common denominator of many chronic pain syndromes is improvement or complete resolution with treatment with low-dose antidepressants. Three possible mechanisms have been proposed: first, antidepressant use may relieve depression associated with or caused by chronic pain and thus improve symptoms; second, the tricyclic antidepressants may have analgesic properties; and/or third, depression and pain may share a similar underlying biochemical mechanism.

Several placebo-controlled studies have documented the efficacy of low-dose antidepressants for treating chronic pain. Postherpetic neuralgia responds to treatment with amitriptyline hydrochloride as demonstrated in a double-blind, placebo-controlled, crossover study. There was no significant antidepressive effect with the low doses used (median dose, 75 mg). A 1992 placebo-controlled study compared the efficacy of desipramine hydrochloride, amitriptyline, and fluoxetine hydrochloride for treating chronic pain in diabetic neuropathy. Desipramine and amitriptyline were equally effective and superior to placebo in both depressed and nondepressed patients with diabetic neuropathy. Fluoxetine had no greater analgesic effect than placebo in depressed patients. Patients who were depressed benefited from fluoxetine therapy. In a retrospective review, McKay reported that dysesthetic (essential) vulvodynia responds to treatment with low-dose amitriptyline. Gabapentin, a newly released anticonvulsant medication, has been effective in treating chronic pain syndromes (erythromelalgia and reflex sympathetic dystrophy) in uncontrolled studies. In the future, this drug may prove to be a useful adjunctive therapy for managing a variety of chronic pain syndromes, including scalp dysesthesia.

Of the 11 patients, 9 experienced improvement or complete resolution of their scalp symptoms with low-dose antidepressant treatment. Patient 9 (with pruritus only) was not responsive to numerous therapies including several antidepressants. Patient 5 was unavailable for follow-up. Patient 7 responded to therapy for 1 year but then had a relapse. Patients 1, 6, and 8 reported that symptoms recur if use of the medication is stopped. The positive response to therapy supports our belief that scalp dysesthesia represents a chronic pain syndrome or a subset of the cutaneous dysesthesia syndrome.

It might be argued that patients with only scalp pruritus may have a different condition than those complaining of scalp pain. We included those patients with only pruritus for several reasons: first, several patients with pain or burning reported coexisting pruritus or initial transient pruritus; second, there appeared to be a spectrum of complaints ranging from pain only, to pain and pruritus, to pruritus only; and third, the degree of interference with daily life seemed similar in all patients regardless of their specific symptom. The sensations of both pain and itch are carried on the same unmyelinated (slow) afferent, group C nerve fibers. However, patients with only pruritus were less responsive or unresponsive to therapy with low-dose antidepressants.

**CONCLUSIONS**

We describe 11 women with scalp dysesthesia, a condition that we believe is a type of chronic cutaneous dysesthesia. These women experienced debilitating scalp pain and/or pruritus. We are not certain if our patients have pain secondary to underlying psychiatric conditions, such as anxiety, dysthymic disorder, and somatization, or if these psychiatric problems are unrelated or caused by the scalp dysesthesia. More studies are needed to determine the cause of scalp dysesthesia and define any role psychiatric diseases may play in causing or enhancing the pain experienced by these patients. Further studies should also formally evaluate the efficacy of treating scalp dysesthesia with low-dose antidepressants.

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**REFERENCES**


