Brachioradial Pruritus: A Case Report and Review of the Literature

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GOAL

To understand brachioradial pruritus to better manage patients with the condition

OBJECTIVES

Upon completion of this activity, dermatologists and general practitioners should be able to:

1. Describe the presentation of brachioradial pruritus.
2. Discuss possible causes of brachioradial pruritus.
3. Identify treatment options for brachioradial pruritus.

CME Test on page 70.

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Brachioradial pruritus is an enigmatic pruritic sensation that characteristically involves the proximal lateral forearm of middle-aged women residing in tropical to temperate climates. There are often no associated cutaneous signs. The pathophysiology has been debated but is believed to involve UV radiation and/or cervical spine disease. We present a patient with brachioradial pruritus and a review of the literature. Brachioradial pruritus should be suspected in patients with intractable pruritus overlying the brachioradialis muscle of the forearm that is recalcitrant to standard therapies. These patients commonly report a history of chronic solar damage and/or cervical spine disease.

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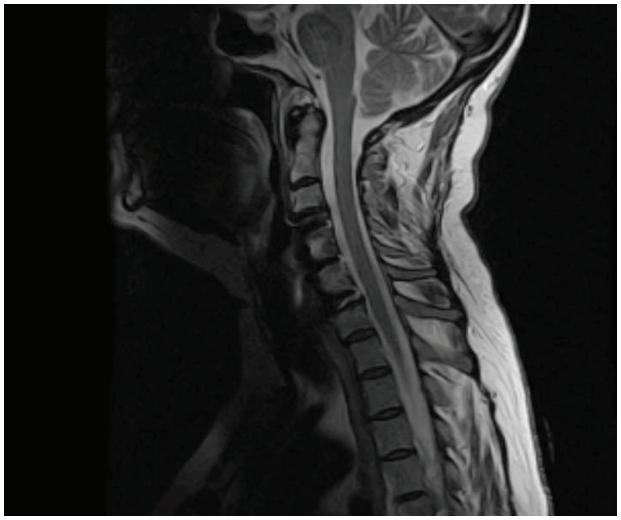
Brachioradial pruritus is an enigmatic entity characterized by pruritus localized to the skin overlying the proximal heads of the brachioradialis muscle, with few other clinical symptoms.[1-5] It was first described by Waisman[6] in 1968. Brachioradial pruritus characteristically affects middle-aged women residing in tropical to temperate climates, though it may occur in males and



Magnetic resonance imaging of the cervical spine revealed a left-sided osteophyte causing minimal left neural foraminal stenosis at the level of C3 to C4.

individuals residing in other climates.[1-8] It often presents as tingling, burning pruritus on the dorsal aspect of the forearm. Pruritus may be unilateral or bilateral and may extend up the arm and across the upper back. It often is described as burning, stinging, or painful, and is seasonal in nature, often presenting in late summer and lasting into December.[1-5]

Brachioradial pruritus was originally associated with solar exposure and UV radiation. Later, Heyl[9] proposed that cervical nerve root impingement or injury secondary to cervical trauma was causative. Although a true cause remains elusive, both actinic damage and cervical spine disease are likely involved. We discuss an otherwise healthy woman who presented with nonspecific pruritus consistent with brachioradial pruritus.

Case Report

A 46-year-old woman presented with persistent pruritus of the left proximal lateral forearm. The pruritus had been present for several years and was recalcitrant to a multitude of therapeutic modalities, including topical corticosteroids and oral antihistamines. Application of heat did not affect her symptoms; however, application of cold compresses did alleviate some of the pruritus. Her medical history was noncontributory. Her surgical history was pertinent for prior anterior cervical discectomy and fusion from C4 to C6. Physical examination revealed no obvious cutaneous lesions in the site other than mild erythema secondary to scratching. Magnetic resonance imaging of her cervical spine revealed a left-sided osteophyte causing minimal left neural foraminal stenosis at the level of C3 to C4 (Figure). Electromyography and nerve conduction velocity studies of the left upper extremity revealed a mildly prolonged left median sensory latency. The patient was treated with topical capsaicin cream with moderate success. She also was advised to avoid sun exposure to the affected area.

Comment

Brachioradial pruritus affects a focal circumscribed area of the proximal lateral forearm. The area typically involves a sun-exposed region and may include the forearm, arm, shoulder, neck, and/or upper thorax.[2] Middle-aged women are most commonly affected, with tingling, burning, and pruritus of the affected area.[1] Symptoms may last from August to December and may subside during the winter.[1-5] Pruritus may be unilateral or bilateral. The nature of the pruritus is often described as burning, stinging, or painful, and manifests a seasonal relation, with high incidence in late summer corresponding with the highest UV exposure.[1]

Patients may report intensification of pruritus with scratching, which may be related to damage of peripheral nerve fibers.[2,10] This intensification may, in part, be secondary to chronic UVA irradiation.[11] Involvement of C fibers containing neuropeptides responsible for pruritus explains the rationale for treatment with topical capsaicin.[2,5] Application of ice often is the only means of alleviating pruritus.[10,12] The consistency of relief with ice is useful in the diagnostic screening of this entity.[10]

The neurophysiology of pruritus is not fully understood. However, it is known that Aδ and C fibers are responsible for the transmission of pruritus.[9] Pruritus can be categorized as pruritoceptive (originating in the skin) or neurogenic (originating in the central nervous system), or a combination of both. The Aδ and C fibers are co-responsive to temperature change as well as pruritus. Increases in skin temperature lower the threshold of cutaneous pruritus receptor units.[13] The neural pathway for pruritus has been established. Nociceptor C fibers transmit impulses to the dorsal horn of the spinal cord and then to the thalamus via the spinothalamic tract.[14] Scratching typically relieves the sensation of pruritus.[15] The gate-control theory postulates that afferent sensory input from cutaneous C fibers is modulated by a gate-control system at the level of the spinal cord.[5,16,17] A subsequent Aδ impulse may act in a negative feedback method to shut off C-fiber stimulation. However, stimulation of Aδ fibers in brachioradial pruritus paradoxically seems to potentiate the sensation of pruritus.[5] Wallengren[5] suggested that local damage to peripheral nerve fibers could be responsible for this potentiation of pruritic sensation. Kumakiri et al[18] demonstrated ultrastructural damage to dermal nerve fibers following UVA

irradiation. UV radiation has been shown to cause a sensitizing effect on sensory nerve fibers and lower the threshold for sensory nerve stimulation, which may occur via direct effects of UV exposure or by release of neural mediators.[19]

The pathophysiology of brachioradial pruritus is controversial. The 2 proposed associations are UV exposure and cervical spine disease.[20] Wallengren and Sundler[2] proposed that solar-induced nerve injury is responsible for brachioradial pruritus. UV radiation may be an eliciting factor, while cervical spine disease may be a predisposing factor.[2] On the contrary, Fisher[21] reported on the association of cervical nerve root impingement and involvement of 1 or more of the C5 to C8 cervical nerve root segments. Brachioradial pruritus has been reported in association with an ependymoma and secondary to cervical nerve compression.[9,22] In a report by Heyl,[9] 4 of 14 patients demonstrated evidence of degenerative changes and osteoarthritis between C4 and C7. In addition, Heyl[9] noted that brachioradial pruritus may be caused by compression of structures not identified by cervical radiographs. In a series of 11 patients with brachioradial pruritus who underwent radiography of the spine, Goodkin et al[12] found that all patients demonstrated radiographic abnormalities of the cervical spine. Magnetic resonance imaging is the most reliable diagnostic method for cervical nerve root compression.[20,22] Recently, Crevits[20] suggested that neural damage from peripheral nerves by solar radiation or local injury or from central sensory pathways, such as cervical spine disease, is a nonspecific cause. In view of the literature, a definitive pathophysiology for brachioradial pruritus does not exist. It is clear that it is a unique clinical entity, but the pathophysiologic basis is not known. While the association between cervical spine disease and brachioradial pruritus is not fully understood, the prevalence of cervical spine disease is higher in patients with brachioradial pruritus.[12] It has not been determined if this association is causal or causative.[20]

A similar localized itch syndrome, notalgia paresthetica (NP), also has been examined with respect to cervical spine pathology.[12,23-25] NP is similar to brachioradial pruritus but involves the dorsal spinal nerves. Savk et al[23] found the presence of vertebral column pathology in 7 of 10 patients with NP. A subsequent larger study involving 43 patients with NP reported that in 34 patients (79%), spinal pathology had been detected by radiographs. Additionally, in 65% of patients (28/43), changes were most prominent in the vertebrae and corresponded to clinically involved dermatomes.[24] However, not all pathologies are detectable via radiographic imaging.[25] Thus, Savk and Savk[25] reinforced the necessity of clinical and radiographic examination.

Histologic examination is not necessary for the clinical diagnosis of brachioradial pruritus. Cutaneous biopsies most often demonstrate nonspecific findings and/or chronic solar damage. Wallengren and Sundler[2] compared cutaneous biopsies in afflicted and control patients utilizing antibodies against a pan-neuronal marker, protein gene product 9.5, calcitonin gene-related peptide, and vanilloid receptor subtype 1 (VR1) for capsaicin-sensitive nerve structures. The number of protein gene product 9.5 immunoreative nerve fibers was reduced in pruritic skin by 23% to 43%.[2] These findings suggest that brachioradial pruritus may be elicited by exposure to UV radiation and/or heat. Wallengren and Sundler[2] noted one patient who relapsed with pruritus during the winter following use of a heating pad to relieve neck pain, which supports the notion that UV radiation and/or heat may be causative. Immunohistochemical studies demonstrated protein gene product 9.5 immunoreactive nerve fibers in a dense population in the epidermis and the dermis and calcitonin gene-related peptide immunoreactive nerve fibers located primarily in the dermis. Capsaicin-sensitive VR1 immunoreactive nerve fibers were located as free nerve fibers.[2] Wallengren and Sundler[2] proposed that the VR1 structures are associated with thermoreception. The reduction in nerve fibers via all markers in patients with brachioradial pruritus implicates their role. Furthermore, these histologic changes resemble those found in patients after serial phototherapy.[2,26] Underlying cervical spine disease may amplify this pruritus.

Treatment of brachioradial pruritus includes a multitude of possible topical and oral modalities, such as topical capsaicin, gabapentin, carbamazepine, oxcarbamazepine, cervical spine manipulation, anti-inflammatory medications, surgical rib resection, avoidance of sun exposure, and lamotrigine.[1,3,5,12,20,21,27-32]

Pruritus is a common symptom with a multitude of potential causes, some of which are never diagnosed. The absence of cutaneous signs makes diagnosis more difficult; however, the consistency of anatomic location and historical characteristics make the diagnosis of brachioradial pruritus possible. In addition, relief of pruritus with application of ice helps to confirm the diagnosis.[10] A familial form of brachioradial pruritus was reported with a dominant and possible X-linked inheritance pattern.[33] While an association between brachioradial pruritus and cervical spine disease is present, cervical spine disease alone cannot explain the pathophysiology of brachioradial pruritus.

Furthermore, cervical spine disease undetectable by radiography may hinder a definitive understanding of this association. It is likely that both UV exposure and cervical spine disease contribute to this entity.

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