cephalosporins, such as cefoperazone in this case. Because the girl’s lobar pneumonia is very severe, we add dexamethasone into the treatment regimen as it can reduce the length of hospital stay when added to antibiotic treatment in non-immunocompromised patients with community-acquired pneumonia [2].

Based on the characteristic and distribution of pain, and it was totally relieved with the resolution of right upper lobe infiltrates, it is undoubted that the right brachial plexus root was irritated by the inflammation of the right lung. There have been three cases of Pancoast’s syndrome caused by infectious disease without Homer’s syndrome, and all of them have pain in the arm or shoulder [3–5].

It is rare for Pancoast’s syndrome to be caused by infectious disease, and there are only 35 cases reported in the English literature. The pathogenic organisms included Echinococcus sp. (11 cases), Staphylococcus aureus (8 cases), Mycobacterium tuberculosis (3 cases), Actinomycete (3 cases), Aspergillus (2 cases), Mucor (2 cases), Allescheria boydii (1 case), Nocardia asteroides (1 case), Cryptococcus neoformans (1 case), and unknown (1 case). Most of the cases were adults (33 cases); and there is only one case for teenager and infant each. Male patients (20 cases) outnumbered female patients (15 cases). Among the 35 cases, 31 cases have been reviewed by Heath D. White, who indicated that Pancoast’s syndrome secondary to infectious causes may be more common than previously reported [6].

To the best of our knowledge, this is the first reported Pancoast’s syndrome secondary to lobar pneumonia caused by Klebsiella pneumoniae in a school-aged child. Pancoast’s syndrome is a rare cause of arm and shoulder pain, which should be taken into consideration in clinical practice.

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References


Re: Are Peripheral Pain Generators Important in Fibromyalgia and Chronic Widespread Pain?

Dear Dr. Gallagher,

We appreciated Dr Gerwin’s editorial [1] reflecting on the interesting Albrecht et al. study “Excessive peptidergic sensory innervation of cutaneous arteriole–venule shunts (AVS) in the palmar glabrous skin of fibromyalgia patients: Implications of wide-spread deep tissue pain and fatigue” [2].

Dr. Gerwin summarizes the findings by Albrecht et al. [2]—“that changes in blood flow and increased thermal sensitivity may each contribute to local palmar pain”—and asserts that thereby “they contribute to peripheral nerve sensitization which gives rise to local pain as well as contributing to central sensitization and central pain.” In the article we could not find the evidence for this assertion, which is notable for proclaiming that peripheral nerve sensitization...
sensitization is the fundamental “driver” of all possible somatic mechanisms of pain in this syndrome.

Dr. Gerwin states, “It is the reduction in local and widespread pain in FM [fibromyalgia] in response to treatment of peripheral nociceptive sites that is most convincing in determining that peripheral nociceptive sites play an important role in initiating and maintaining pain.” We believe this is a circular argument: the conclusion is assumed before the evidence is presented. A circular argument is by definition self-perpetuating and cannot contribute to the explanatory function of any theory.

So what is the evidence for active peripheral nociception in the clinical phenomenon of chronic widespread pain?

The experimental studies performed by Kellgren [3,4] and replicated by others [5] showed that localized muscle pain and tenderness can be induced by noxious stimulation of distant deep tissues.

Although the intensity of pain did not diminish when Kellgren [4] anesthetized the areas of induced pain following saline injections into muscle, other investigators have reported that referred pain can be modulated by anesthetizing such areas [6].

These apparently contradictory results may be explained by the relative completeness of the anesthetic block. When all afferent input from the area of injection (experimental nociception) was interrupted (by sympathetic and somatic plexus blocks), the phenomenon of referred pain still occurred, implying that no peripheral input from the tissues in the region of experienced pain was necessary [5]. There is no need to postulate active peripheral nociception in these clinical syndromes.

In his editorial Dr. Gerwin moved the discussion from the “tender points” of FM to the “trigger points” of myofascial pain syndrome (MPS), the not unreasonable implication being that the peripheral phenomena are similar. However, the various studies quoted by Dr. Gerwin to support his contention that “myofascial trigger points were identified as sources of peripheral nociceptive pain that caused local tenderness” are all open to criticism on the grounds that the respective investigators begged the question by assuming that the trigger points they identified were in fact sites of damaged muscle tissue (i.e., regions of active nociception) [7–9]. This is conjecture for which we believe there is no supporting evidence [10].

Nonetheless, the “vicious circle” hypothesis formulated during the 1930s is still influential in terms of guiding treatment [11]. Kraus (as cited by Simons [11]) postulated that palpable muscle hardenings could cause a reflexly induced increase in muscle tension resulting in “a pain–reflex–pain self-perpetuating cycle that was disrupted by his spray (ethyl chloride) or injection therapy” (p. 299).

However, based on experimental animal research findings, Mense [12] concluded in a 1991 article that the proposed vicious-circle models “have to be considered as working hypotheses rather than explanations of known mechanisms.” The vast majority of studies published in this area over the 20 years since have continued to assume the “truth” of the theory of myofascial trigger points despite the absence of any demonstrable pathophysiological basis for them and the repeated failure to demonstrate interobserver reliability in identifying them [13].

Furthermore, within the constraints of MPS theory, well-performed systematic reviews have been unable to determine the effectiveness of the various modalities of therapy (physical and pharmacological) that have been advocated in pursuit of trigger points [14–16].

This raises the question: Why, in the face of such refutations, does adherence to MPS theory—and hence to the idea of active peripheral nociception in that syndrome—still pervade the pain medicine literature?

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References
Dear Editor,

I thank Drs. Quintner and Cohen for their interest in my editorial [1] about the study by Albrecht et al. [2]. Drs. Quintner and Cohen have raised several concerns about my discussion of the implications of that study, especially of the concept that peripheral nociception by myofascial trigger points initiates central sensitization. They object to the idea that peripheral nerve sensitization is the fundamental “driver” of, as they put it, all possible somatic mechanisms of pain in fibromyalgia, which they claim is found in the editorial. I agree that this idea is extreme and is incorrect. However, my editorial states only that “peripheral nociceptive sites play an important role in initiating and maintaining pain . . . Pain is the outcome of a complex interplay between . . . central modulation and peripheral input.” However, Albrecht et al. [2] in their article do discuss in detail the implications of their work on some of the many manifestations of fibromyalgia, specifically fatigue, sleep, and cognition, through the potential of reduced blood flow to skeletal muscle caused by shunting blood from muscle to glabrous skin during exercise. The central issue raised by Quintner and Cohen is the role that active peripheral nociception plays in chronic widespread pain. This is a role that they doubt exists.

Quintner and Cohen question the evidence that I presented in the editorial that demonstrates that trigger points are a peripheral nociceptive input leading to central sensitization. They believe that it is a circular argument to state that because inactivation of trigger points in fibromyalgia (and for that matter, in myofascial pain syndrome, as they have included that in their discussion) reduces pain and decreases manifestations of central sensitization, then trigger points (which are peripheral nociceptors) are important in the initiation and maintenance of central sensitization. They state that I presuppose the end (peripheral nociceptors are important in the initiation and maintenance of central sensitization), and therefore my argument does not contribute to the support of any theory.

My argument is linear, to the contrary, not circular. The trigger point can be isolated as a cause of local and referred pain. Pain can be reproduced by trigger point activation. Local and referred pain can be diminished or eliminated by inactivating the trigger point with or without local anesthetic. All these conditions are met in Giamberardino et al.’s studies of trigger points in migraine headache [3]. The pain can be caused again by activating the trigger point. This is more in keeping with Koch’s postulates for a bacterial cause of disease than a tautological, circular argument. In the case of muscle pain, if trigger point activation induces both local pain and referred pain (a manifestation of central sensitization), and inactivation of trigger points reduces local pain and also referred pain, then trigger points can logically be considered related in a causative way to central sensitization. The hypothesis, therefore, is that trigger points are capable of inducing central sensitization and referred pain. To perform experiments that show an effect of trigger point inactivation on central sensitization does not seem to