Letters to the editor

Chemical Radiculopathy

To the editor:

I read with great interest the article by Slipman et.al. in the July issue of *Pain Physician* on chemical radiculopathy. While there have been many who have questioned the existence of discogenic pain, over the years this has become more universally accepted by both practicing physicians and insurance carriers. Chemical radiculopathy, the end result of a leaking disc syndrome, still remains controversial in many circles but is slowly gaining acceptance. Contributions such as those made by Slipman et.al. help bring more legitimacy to this clinical entity.

While most patients with radicular symptoms will demonstrate compressive pathology on their radiographic studies there is a sub-population that do not. Some of these patients may have pseudoradicular symptoms from referred pain syndromes as can be seen in facet syndromes, sacroiliac dysfunction, or even some myofascial pain syndromes. Others may have radicular appearing pain related to peripheral nerve compression (or irritation outside the spinal canal) such as in piriformis syndrome, thoracic outlet syndrome, or brachial plexopathy. Radicular pain can also come from chemical irritation of the nerve root.

Provocative discography has become the gold standard in the definitive diagnosis of discogenic pain. Not uncommonly one may find Grade V annular tears with free flow of contrast into the epidural space; this provides evidence for the possible communication between the nucleus of the disc and nerve roots. Patients in whom Grade V annular tears are seen at the time of discography may require surgical intervention after failing to respond to more conservative treatment. In those patients with prominent radicular symptoms, the surgeon may elect to explore the disc at that level looking for an unrecognized disc herniation not appreciated on preoperative radiologic studies. In my experience, at the time of surgery, these patients are often found to have extensive fibrosis (but no disc herniation) surrounding the associated nerve root that was clinically felt to be causing these radicular symptoms.

Many articles have pointed to the chemical mediators of the inflammatory response as the etiology of radicular pain in the presence of a disc herniation rather than the compression itself of the associated nerve root. Here in lies the rational for epidural steroid injections. While the existence of chemical radiculopathy is accepted by many pain management physicians, this area still requires greater research before it is considered dogma by all.

I include two discography figures from patients experiencing radicular pain in the absence of compressive pathology on MRI. Both patients had positive concordant responses to discography associated with Grade V annular tears and spread of contrast to the corresponding nerve root. I believe this type of finding provides further evidence for the existence of the clinical entity, "chemical radiculopathy".

Fig 1. C6,7 discography with spread of contrast to the C7 root.

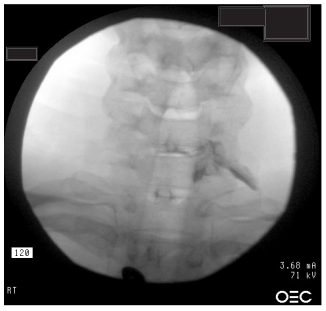
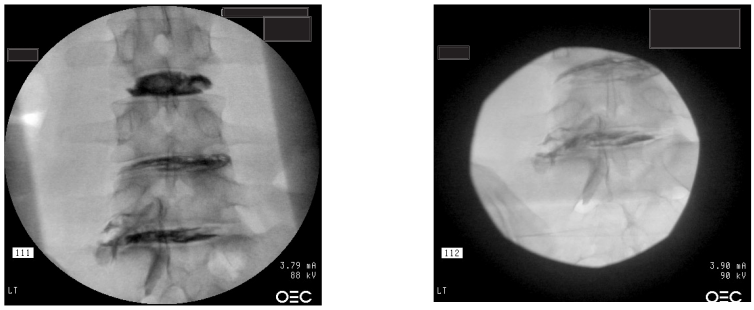


Fig 2. L5, S1 discography with spread of contrast to the S1 root.



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In Response:

Dr. Kloth's thoughts concerning the entity of radicular pain are quite similar to those of my colleagues and mine. Our mutual considerations emanate from undeniable facts. In particular, a plethora of literature has unequivocally demonstrated that a key element in the generation of radicular pain associated with a symptomatic focal disc protrusion is the liberation of phospholipids leading to the initiation of the inflammatory cascade. Observations using animal and human models have provided consistent confirmatory results. Given this indisputable and universally accepted notion, then it is not much of a conceptual leap to postulate that injury to discal material could generate chemical inflammogens that incite the spatially contiguous dorsal root ganglion (drg). A similar response could develop due to a synovitis associated with facet joint syndrome. While these are appealing notions, a severe lack of science has precluded our ability to definitively state that these chem-

ical inflammogens seep out and reach this target (the drg). By what scientific mechanism can we prove that such a process occurs? The answer to this question has been one of the challenges presented to the interventional spine/pain physician community. Our view was that if we could identify patients that presented with objective findings of a radiculopathy in the absence of a corroborative radiologic lesion and the symptoms were successfully abated following the injection of a small aliquot of corticosteroid, then we would have some supportive, though not definitive, evidence. As I have intimated this type of proof is limited in that it does not provide tangible data demonstrating the presence of inflammation. Using this clinical approach our conclusions are necessarily deduced from indirect evidence. Another limitation in our approach is that the development of florid radiculopathy and not just radicular pain is a rare occurrence. Consequently we could identify only four true cases during a two year interval. If the incidence of true radiculopathy were greater then our argument would be substantially stronger. So, at this juncture, we remain in the position of having to produce more compelling data or observations. Dr. Kloth points us to one of these potential facts. All experienced discographers have witnessed the egress of contrast from the nucleus to the outer annulus and along the traversing or exiting nerve root. Such a dye pattern is certainly a more common than the entity that we wrote about. Perhaps a careful analysis of patients with such a flow pattern would provide us with the requisite information to substantiate the entity of chemical radicular pain without a focal protrusion. It is this possible path that Dr. Kloth exposes for all of us and he should be applauded for opening our eyes.

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