Dynamic Chiropractic

BACK PAIN

Lumbar Vertebral Compression, End Plate Fracture, and Disc Degradation

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Clinical Anatomy of the Lumbar Spine is one of the great books in print regarding the lumbar spine. It is written by Drs. Bogduk and Twomey, and published by Churchill Livingstone. The book gives information about the structure and function of the lumbar spine as it relates to the most common source of low back pain, mechanical low back pain. In the chapter on the pathology of mechanical lumbar back pain, the various body movements are discussed. I would like to visit the topic of lumbar compression.

When excessive lumbar compression occurs, the lumbar discs do not typically herniate, but rather the vertebral end plate may fracture. This can occur with heavy axial loads, such as falling into a standing or seated position, or during heavy lifting when the back muscles exert a large compressive load on the discs and vertebrae. Just as muscles become conditioned with use to loading stress, so do bones, such as the lumbar vertebrae.

While the end plate fracture can occur with a fall into a standing or seated position, it also occurs in poorly conditioned individuals who undertake heavy lifting that their bodies are not accustomed to handle.

Take for example the person who works at a desk all week, then decides on the weekend to move furniture or lift some other heavy object with a maximum pull. Proper lifting techniques would help in this situation, but the risk of an end plate fracture still remains. Obviously, persons with osteoporosis or osteomalacia would be significantly more susceptible to this.

Because the end plate is not innervated, this end plate fracture is not typically painful. A person could heal and not even know that they had such an injury.

Another possibility, however, is disc degradation via an inflammatory repair process, or from an autoimmune response. Autolytic enzymes can become activated by changes in the pH, secondary to the disturbance caused by this end plate fracture. Another theory following end plate fracture is that the nucleus pulposus elicits an autoimmune response. The nucleus pulposus does not have a blood supply and, in the presence of an end plate fracture, the nucleus is exposed to circulation within the vertebral spongiosa. This elicits an immune response.

Whether the mechanism is autoimmune or an inflammatory process, the nucleus pulposus begins the process of progressive degradation. This process is a consequence of trauma, not age-related degeneration. There is a progressive loss of the water binding capacity and a deterioration of nuclear function. Since the nucleus is less able to sustain pressure because it is not able to bind water, greater loads are placed on the annulus fibrosus. Eventually the annulus is unable to sustain the load; the disc loses height; and this changes the function of all the joints in the affected area. This can lead to osteophyte formation around the zygapophyseal joints and vertebral bodies.

This nuclear degradation eventually extends peripherally to affect annular fibers, typically along radial fissures that have formed. This condition is known as an internal disc derangement or

internal disc disruption. At this point, there is no disc bulge or disc herniation. It is possible for the internal disc derangement to progress to a disc herniation because the nucleus pulposus is no longer normal, coupled with the radial tears that occur in the annulus, setting up a situation that allows for herniation of the nucleus, typically during flexion or flexion and rotation movements.

Before a disc herniation occurs, and before any osteophyte formation and subsequent inflammatory response causing radicular type pain can occur, the internal disc derangement can become painful as a result of mechanical or chemical irritation of the nerve endings in the annulus fibrosis. More stress is imparted on the annular fibers and, since these annular fibers are innervated, pain can result. If there are radial fissures in the annular fibers extending into the outer third, inflammatory chemicals can be brought into the area and pain can occur.

This chemical stimulation of nociceptive fibers can explain the constant pain that is unrelated to activities. Certainly, they can have both chemical and mechanical pain, so that the pain is increased with activity on top of their ongoing constant pain. With this internal disc derangement, there would be no neurologic deficit, as the problem is centrally located within the disc, so CT scans, MRI, and myelography would be normal, as would an electrodiagnostic evaluation.

Discography, an expensive and invasive procedure, would help identify this problem. Over time, other imaging that is less invasive can be used to help explain some of these patients with ongoing low back pain, despite the absence of neurologic deficit.

While not always visible on plain film radiography, Schmorl's nodes often form secondary to these end plate fractures. Excellent pictures and radiographs are given in the textbook Essentials of Skeletal Radiology, second edition, by Yochum and Rowe.

One of the problems with this model for low back mechanical and/or chemical pain secondary to stimulation of nociceptive fibers is the annulus fibrosis in the clinical setting, with the current health care environment, is the need for documentation. As discussed, outside of discography, there is not a great deal that we can do that I know of to document these lesions. I do not believe, as some physicians do, that everyone with long-term low back pain is simply malingering. The theories discussed above offer some possible explanations. I feel we owe it to our patients to learn more about these possibilities to explain their pain syndromes. Chiropractic care can help many of these people via chiropractic adjustments and the resultant firing of mechanoreceptors and inhibition of nociceptors.

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