Traumatic Disc Injury and the Acceleration of Disc Disease and Spondylosis

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Although clinicians have long made the connection between the development of spondylosis in the cervical spine and various forms of trauma, it was Mason Hohl, MD, who first analyzed the long term effects of trauma (whiplash in this case) on the cervical spine. In this classic work it was demonstrated that patients injured in such accidents develop spondylosis approximately six times more frequently than age and gender-matched controls, and in cases where a loss of consciousness was reported, these patients were 10 times more likely to develop such degenerative changes. Conversely, other authors have found that patients with pre-existing spondylosis generally fared worse in whiplash (cervical acceleration/deceleration) injuries. In 1993, Robinson and Cassar-Pullicino published a paper based on the long-term outcome of whiplash trauma finding that after more than 10 years, 86 percent of the subjects were still symptomatic. However, they concluded that neck sprain did not lead to cervical spondylosis. Unfortunately, their method for arriving at this conclusion was flawed. The patients’ radiographs were compared to normative data rather than their own radiographs.

Also in 1993, Hamer et al. provided further support for the connection between whiplash trauma and the progression of disc disease in their study in which they found that the incidence of whiplash in patients undergoing anterior cervical discectomy was twice that seen in the general population (p<0.05). They found that the mean age of the whiplashed surgical group was 45 +/- 12 years, whereas the mean age of the non-whiplashed surgical group was 55 +/- 14 years (p<0.001).

The exact mechanisms by which this degenerative process proceeds have not been clearly defined but probably include an initial destabilizing of the disc through direct injury from a high energy combination of shear, compression, axial stretch, and torsion forces. Mendel et al. have discovered intradiscal receptors in normal cervical discs. Damage to these receptors may result in an uncoupling of important biomechanical feedback loops operating through both intrinsic paraspinal and gross postural muscles. Loss of normal ligamentous integrity will allow a direct biomechanical destabilization of the spine and probably contributes significantly to the overall degeneration scenario.
References


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