Does provocation lumbar discography cause premature disc degeneration or herniation?

Introduction

Lumbar provocation discography (LPD) is a commonly used diagnostic procedure. Yet, its purpose and effects have been contested. Stimulating an intervertebral disc via direct intranuclear injection of contrast media allows the evaluating clinician to assess for painful annular fissures. Such fissures are the tissue substrate for the most commonly painful structure explaining chronic low back pain. Chronic low back pain (CLBP) is challenging to diagnose and treat and is associated with significant costs. Therefore, LPD seemingly has a place in the diagnostic algorithm for CLBP.

Controversy has been generated by recent reports suggesting that LPD promotes premature degeneration and induces herniation of discs stimulated during LPD as control levels. A 10 year prospective investigation revealed higher proportions of disc degeneration and herniated nucleus pulposus (HNP) compared to age-matched control subjects. A 30% loss to follow-up and overlap of confidence intervals of these reportedly different proportions of degeneration and HNP are reasons to suspect these findings. Repeat LPD years after initial LPD has revealed no new morphologic abnormalities. Histologic studies have shown that disc puncture, as performed during LPD, is not associated with degradation of annular tissue.

Over a quarter of MRI scans of lumbar spines of asymptomatic subjects demonstrate abnormalities- 24% may demonstrate HNP, and 4% stenosis. In subjects less than 60 years old, 20% may have HNP, while 57% of those over 60 years of age may demonstrate HNP and stenosis, and the proportion of degenerative discs increases with advancing age. More specifically, lumbosacral MRI scans in asymptomatic subjects have revealed a disc bulge at one level in 52%, a protrusion in 27%, and an extrusion in 1% of subjects. Per level, regardless of age, there is approximately a 2-5% prevalence of a protrusion at L3-4, 11-19% at L4-5, and 8-11% at L5S1. In 40 year olds, these proportions were: 1%, 3-5%, and 4%.

The current study was undertaken to examine the association between LPD and disc degeneration and LPD and HNP at each specific level of disc stimulation.

Methods

MR imaging will be performed prospectively on 100 patients having undergone LPD 10 years prior. Routine sagittal T2 weighted sequences will be completed and are sufficient to determine disc abnormalities and contain costs. Two blinded evaluators will assess the degree of disc degeneration using the Pfirrmann grading disc degeneration scale. The presence/absence and the side and location and type of any HNP will be recorded. Subject age at the time of the MRI will be recorded.
The above data will be compared to historical data reported by Carragee et al and Jensen et al.

LPD protocols will have been performed per ISIS standards. Procedure notes will be reviewed to confirm that each studied patient underwent LPD and at what levels. Levels stimulated, side of annular puncture, and location of annular tear will be recorded.

MRI scans will be sent via secure CD, absent patient identifiers, to a central research nurse who will distribute the scans for interpretation and who will compile the MRI and LPD data.

Proportions (and 95% CI's) of discs demonstrating Pfirrmann scores I-II, III-IV, V, and HNP will be compared among LPD cases, and historical controls from Carragee and Jensen. Comparisons will be made between exact levels, when possible, rather than grouping levels together. Side of disc puncture and side of HNP, if present, will be compared per each disc level.