Peripheral Disc Margin Shape and Internal Disc Derangement: Imaging Correlation in Significantly Painful Discs Identified at Provocation Lumbar Discography

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Key words: discography, discography, CT imaging, internal derangement, disc protrusion, disc bulge, disc degeneration, radial annular tear, radial annular fissure

Summary

Annular margin shape is used to characterize lumbar disc abnormality on CT/MR imaging studies. Abnormal discs also have internal derangement including annular degeneration and radial defects. The purpose of this study was to evaluate potential correlation between disc-margin shape and annular internal derangement on post-discogram CT in significantly painful discs encountered at provocation lumbar discography (PLD).

Significantly painful discs were encountered at 126 levels in 86 patients (47 male, 39 female) studied by PLD where no prior surgery had been performed and response to intradiscal lidocaine after provocation resulted in either substantial/total relief or no improvement after lidocaine administration. Post-discogram CT and discogram imaging was evaluated for disc-margin characteristics (bulge/protrusion), features of disc internal derangement (radial annular defect [RD: radial tear/fissure/annular gap], annular degeneration) and presence/absence of discographic contrast leakage.

In discs with focal protrusion, 50 of 63 (79%) demonstrated Grade 3 RD with 13 (21%) demonstrating severe degenerative change only. In discs with generalized-bulge-only, 48 of 63 (76%) demonstrated degenerative change only (primarily Dallas Grade 3) with 15 of 63 (24%) demon-

strating a RD (Dallas Grade 3). Differences were highly statistically significant ($p < 0.001$). Pain elimination with intra-discal lidocaine correlated with discographic contrast leakage ($p < 0.001$).

Disc-margin shape correlates with features of internal derangement in significantly painful discs encountered at PLD. Discs with focal protrusion typically demonstrate RD while generalized bulging discs typically demonstrated degenerative changes only ($p < 0.001$). Disc-margin shape may provide an important imaging clue to the cause of chronic discogenic low back pain.

Introduction

Most acute low back pain (LBP) resolves spontaneously with rest and anti-inflammatory medication^{1,2}. Significant LBP remains in a small percentage of people and more definitive treatment is typically sought. Sources of axial LBP include the facet joints, the sacroiliac joints and the intervertebral discs. In parallel with more advanced pain management, lumbar spine imaging is usually obtained in patients to exclude important causes of LBP that might require specific treatment (tumor, infection, trauma) as well as assess for specific spine degenerative changes that might be responsible for the LBP.

With advancing age, significant changes occur in the disc including alteration of proteoglycan/

water content (disc desiccation) and loss of anatomic integrity of the annulus fibrosus (concentric/lamellar separation, radial annular tears, annular fragmentation)^{3,4}. Features of spine degeneration may be present on lumbar spine Magnetic Resonance (MR) or Computer Tomographic (CT) imaging studies including disc bulge or protrusion, bony endplate changes, adjacent vertebral marrow degenerative changes (Modic changes) and accentuation of the age-related changes described above. These features are commonly present in the presence of or in the absence of symptoms⁵⁻⁸.

Disc margin shape is one of the primary features evaluated for when interpreting lumbar MR or CT imaging studies⁹. Four characteristic features are typically searched for when describing the abnormal disc including: diffuse bulge, disc protrusion, disc extrusion and free fragment (sequestration). Unfortunately, disc degenerative and age-related changes are nonspecific. The only MR imaging feature that to date has been equated with discogenic LBP as demonstrated at lumbar discography is the presence of an annular fissure or high intensity zone (HIZ)^{10,11}.

Provocation lumbar discography is a minimally invasive procedure that is commonly used in the assessment of patients with chronic LBP. Disc space injection, like physical examination, challenges the disc allowing assessment for potential discogenic source of the LBP. In addition to routine fluoroscopic imaging obtained with disc space contrast injection, post-discogram CT is commonly obtained to evaluate the features of disc internal derangement as well as assess for discographic contrast leakage. Axial CT imaging obtained following discography provides a unique opportunity to evaluate the shape of the disc margin in addition to the traditionally observed features of disc internal derangement.

The purpose of this study was to compare the peripheral disc margin shape to the features of internal disc derangement in significantly painful discs encountered at provocation lumbar discography.

Materials and Methods

During a 21 month period, 149 consecutive patients were studied with provocation lumbar discography. Patients typically presented with long-standing severe LBP unresponsive to conservative management and were referred by

spine-focused orthopedic or neurosurgeons for preoperative planning. Eight patients had previous fused levels with 14 patients having negative provocation discography and these patients were not included for analysis. In 127 of the 149 patients, one or more significantly painful discs were encountered.

Provocation discography was performed at 437 levels in these 127 patients. Significant pain (VAS $\geq$ 6-7) was encountered on provocation at 242 levels with 195 levels negative (no pain, pressure or minimal discomfort on disc injection). Intradiscal lidocaine was injected to attempt to eliminate the provoked pain at 211 disc levels with no lidocaine injected at 31 levels (intradiscal volume limitation, vascular identification, transient provoked pain). At seven of 211 painful levels and three of 195 negative levels prior discectomy had been performed and these were therefore not included in the assessment.

Therefore 204 lidocaine-injected significant painful disc levels and 192 negative disc levels in 111 patients were available for analysis. These 396 disc levels are the focus of this report. Institutional Review Board approval was obtained for this retrospective study.

Lumbar discography technique

Discography was performed in a standard fashion as previously described^{12,13}. Back pain vs. leg pain contribution was clarified. The patient's most severe and immediate pre-procedure pain level was documented employing the 0-10 Visual Analog Scale (VAS; 0 = no pain, 10 = worst pain imaginable). Limited intravenous sedation (fentanyl 0.05 mg, Versed 1 mg [midazolam, Bedford Laboratories; Bedford, OH, USA]) was given before the procedure, occasionally supplemented during the study with fentanyl given at the end of the examination. Administration of additional fentanyl during the examination was necessary only on rare occasions when disc provocation resulted in extremely severe patient pain that was not responsive to intradiscal anesthetic and level of consciousness was never affected. Supplemental administration of additional conscious sedation during the discogram was generally avoided. Double needle technique using a 20 gauge guiding spinal needle followed by a long 25 gauge spinal needle accessed the center of the disc space with routine fluoroscopic guidance. All needles were placed concordantly opposite the side of leg pain pri-

or to disc evaluation. Anticipated normal/control disc was studied first.

Discs were provoked by a moderate/rapid hand injection of 1.5-4.5 cc Iohexol 240 mgI/cc (GE Medical Products, Milwaukee WI, USA) under direct fluoroscopic guidance. Injection volume depended on 1) disc volume end-point, 2) clearly established severe pain response or 3) exaggerated capacity in degenerative discs. Patients were kept unaware of whether a level was being provoked or which level was being studied. Initial injection response was observed by the operator and with a positive pain response, the features of the pain were clarified, VAS level of pain was established and these items recorded similar to Walsh et al. Concordant pain was recorded if the provoked pain was the patient's typical/familiar pain and non-concordant pain was recorded if the provoked pain was not their typical pain. Fluoroscopic spot-films were obtained for each disc level in anterior-posterior/lateral projections during and following the injections.

If a severely painful disc space (typically VAS $\geq$ 6-7; concordant/non-concordant) was encountered, preservative-free lidocaine (2% strength, 1-1.5 cc, Xylocaine-MPF, Astra-Zeneca, Wilmington DE, USA) was injected into the disc in an attempt to reduce the patient's provoked pain and allow response clarity in subsequently studied discs.

The patient was routinely questioned regarding any pain reduction after administration of the intradiscal lidocaine and their response was recorded either as: 1) complete/near-complete pain relief, 2) partial pain relief or 3) no significant pain relief *or* as a specific VAS grade reduction from the pain generated by disc provocation relative to base line pain - depending upon patient's ability to express the change. Responses reported with the VAS were converted to the three point scale by calculating % pain reduction relative to base line VAS pain level with: >66% pain reduction = complete or near-complete pain relief, 33%-66% pain reduction = partial pain relief and <33% pain reduction = minimal or no significant pain relief.

Post-discogram CT (General Electric, Milwaukee WI, USA) employing bone/soft tissue algorithm with either direct axial 3 mm slice acquisition or spiral technique, 3 mm axial/sagittal reformatting with isotropic voxels was obtained in all patients immediately following the discogram.

Imaging analysis

Discogram fluoroscopic images together with the post-discogram CT of all disc spaces were reviewed by two neuroradiologists experienced with lumbar discography and blinded to the pain response at provocation. Judgments were made by consensus agreement.

Post-discogram CT: annular internal derangement

Assessment of the post-discogram CT features followed the characteristics observed anatomically by Videman et al.¹⁴ and referred to in the Dallas discogram scale and subsequent reports^{15,16}. Fundamental observations and subdivisions included: 1) radial annular tear/fissure Dallas Grade 1-3, 2) degenerative disc features Dallas Grade 1-3 and 3) pain response at provocation. Wide radial annular defects (annular gaps [AG]) were also commonly encountered beyond simple expansion of a Grade 3 radial tear/fissure. Dallas grade 3 radial annular tear/fissure and annular gaps were tabulated together as radial annular defects (RD)¹⁶. Discs were categorized after the description of Videman et al.¹⁴ as: 1) RD with or without significant accompanying annular degenerative change, 2) Degenerative-change-only (Dallas Grade 1-3) without visualized RD or 3) normal. A central radial tear/fissure [cRT] was defined as full thickness Grade 3 tear/fissure located along posterior annulus between the medial aspect of the pedicles, lateral radial tear/fissure [latRT]) was located along the postero-lateral to lateral annulus from the level of the foramen to the far-lateral region. The observed degenerative features were typically complex including areas of concentric (lamellar) tears, annular fragmentation and peripheral annular tears with most painful discs demonstrating grade 3 degree of degeneration by Dallas criteria and previous reports.^{15,16}

Post-discogram CT: peripheral disc margin shape

Disc margin shape was assessed in a standard fashion as per nomenclature consensus definitions including: bulge, protrusion, extrusion, sequestration⁹. Disc extrusion and sequestrations were not encountered.

Isolated diffuse annular bulges were judged as either symmetric or asymmetric depending upon whether there was either symmetric uni-

Figure 1 'Type A' focal protrusions. A-D) Typical smaller focal disc protrusions were encountered including: A) central focal protrusion, B) paracentral focal protrusion, C) foraminal focal protrusion and D) far-lateral focal protrusion. Central and paracentral focal protrusions were most commonly encountered with central radial tears or annular gaps. Foraminal and far-lateral small focal disc protrusions were less frequently encountered but commonly associated with focal radial tears that projected into small focal peripheral annular tears.

Figure 2 'Type B' protrusions. A,B) A broad-based type of disc protrusion (arrowheads) varying in depth from thicker (A) to thinner (B) was also encountered. This appearance was typically present in discs with lateral radial tear that projected into a broad peripheral annular tear and seen to extend from the far-lateral region to lateral paracentral region or be primarily far-lateral and foraminal in location.

form or asymmetric smooth extension of the annular margin beyond the adjacent vertebral end plates. Focal disc protrusions were commonly identified, typically with some degree of associated disc bulge superimposed on the focal peripheral annular margin shape. Two types of disc protrusion were recognized (Figures 1 and 2). 'Type A' protrusions were usually seen as a focal protrusion of the annular margin projecting into the spinal canal centrally or in the paracentral region and less commonly within the neural foramen or far lateral region (Figure 1). These 'Type A' protrusions subtended less

than 45 degrees of the annular circumference and were most commonly seen in association with a central Radial Defect (RD) in the annulus being either a full thickness tear (Dallas grade 3) of the annulus or wider central annular gap with retained peripheral annular margin¹⁶. 'Type B' protrusions were seen as a focal but uniform broad-based protrusion of the annular margin extending from the far-lateral region into the foramen, occasionally projecting to the paracentral region (Figure 2). These 'Type B' protrusions usually subtended approximately 90 degrees of the annular circumfer-

ence and were typically associated with full thickness lateral radial annular tears (Dallas Grade 3).

Discographic contrast leakage

Identification of epidural discographic contrast leakage was primarily established by assessment of the fluoroscopic images with secondary inspection and correlation with the post-discogram CT. Significant leakage at the disc margin around the needle entry site was classified as true leak but minimal contrast identified at needle entry only after needle withdrawal was not classified as leakage. Discs were judged as either contained (no contrast leaking from the disc space) or *leaking* (epidural contrast leakage from the disc space) itemized and tabulated.

Imaging identification of discographic leakage in the severely painful/treated discs was compared to the response to lidocaine administration [1) complete/near-complete relief, 2) partial relief and 3) minimal/no relief].

Statistical analysis

Statistical analysis was performed by Chi squared comparison between 1) peripheral disc margin shape (bulge, protrusion) and internal derangement features (radial defect: radial tear, annular gap) and 2) complete response and no response to lidocaine in contained and leaking discs, SAS release 8.2 (SAS Institute, Cary NC, USA).

Results

Of 111 patients evaluated, 63 were male and 48 female with an average age 42.4 SD $\pm$ 11 years. Significantly painful discs were present at 204 levels in these 111 patients (L5-S1: 80, L4-5: 68, L3-4: 33, L2-3: 19, L1-2: 4; concordant pain: 166 levels, non-concordant pain: 25 levels). Of 192 negative disc levels, analysis could be performed on 190 discs with two normal disc levels demonstrating significant motion artifact on post-discogram CT.

Disc morphology and response to intradiscal lidocaine in the 204 significantly painful discs and 190 normal discs summarized in Tables 1-3. In the 204 significantly painful discs, complete/near-complete improvement after intradiscal lidocaine injection was present in 63 discs, par-

tial improvement in 65 discs with no significant improvement at 76 discs. The presence of a radial annular defect (RD) or degenerative change only was similar among all three pain-relief groups (Table 1). Normal disc morphology (no internal derangement and normal peripheral margin shape) was noted only in 13 of 204 significantly painful discs and all 13 had no improvement with intradiscal lidocaine administration and no leakage of contrast material. All discs with either complete response to lidocaine or partial response to lidocaine demonstrated annular abnormality on post-discogram CT.

Clinically normal discs

In the 190 clinically negative discs, no evidence of internal derangement was present in 94 (49%) discs with Grade 1-2 degeneration only in 24 (12.5%), Grade 3 degeneration only in 32 (17.3%) and the presence of a Grade 3 RD in 40 (21%) discs (Table 2). Disc margin shape appeared normal in 96 (50.5%) clinically negative discs with disc bulge in 69 (36.1%) and focal disc protrusion in 26 (13.6%) discs (Table 2).

Clinically painful discs with complete or no anesthetic improvement

Comparison of disc internal derangement and peripheral disc margin shape was performed in 126 significantly painful discs (L5-S1: 43, L4-5: 48, L3-4: 19, L2-3: 13, L1-2: 3) with either complete response to intradiscal anesthetic or no improvement after intradiscal anesthetic (Tables 4-10). Thirteen significantly painful discs injected with lidocaine were judged to be completely normal on post-discogram CT without bulge, focal protrusion, internal derangement and these had no improvement to intradiscal lidocaine.

Disc margin shape

Disc bulge was identified in 63 of 126 significantly painful discs with focal disc protrusion identified in 63 discs (Table 4; Figures 3-7). In discs with focal protrusion, Dallas Grade 3 radial defect (radial tear/annular gap) was identified in 50 of 63 (79%) with Grade 3 degenerative-change-only identified in 13 (21%). In diffusely bulging discs (symmetric, asymmetric), Dallas Grade 3 annular degeneration-only (concentric lamellar tear, attached/free annular fragments, peripheral annular tear) was identified in 48 of

Table 1 Features of internal derangement and anesthetic response in 204 significantly painful discs.

Internal Derangement	Lidocaine Response			Total
	Total Relief	Partial Relief	No Relief	
Normal (No Internal Derangement)	0	0	13	13
Degenerative Changes only (Typically Grade D3)	28	33	33	94
Radial Defect (Grade 3)	35	32	30	97
Total	63	65	76	204

Table 2 190 Discs clinically negative at discography: disc internal derangement and disc margin shapes.

Internal Disc Morphology	Disc Margin Shape			Total
	Normal	Bulge	Protrusion	
Normal (No Internal Derangement)	82	12	0	94 49.5%
Degeneration Only (Dallas Grade 1-2)	4	18	2	24 12.6%
Degeneration Only (Dallas Grade 3)	2	22	8	32 16.8%
Radial Defect (Grade 3: RT or AG)	8	16	16	40 21.1%
Total	96 50.5%	68 35.8%	26 13.7%	190

Table 3 65 significantly painful discs with partial response to intradiscal: disc internal derangement and disc margin shapes.

Internal Derangement	Bulge	Protrusion	Total
Degenerative Change only (Typically Dallas Grade D3)	20	13	33 50.8%
Radial Defect (Dallas Grade 3)	10	22	32 49.2%
Total	30 46.2%	35 53.8%	65

63 (76%) discs with a Grade 3 radial defect (radial tear/annular gap) identified in 15 (24%). The relation between peripheral disc margin shape (focal disc protrusion, bulge) and features of internal annular derangement (Grade 3 radial defect, Grade 3 annular degeneration-only) was highly statistically significant ($p < 0.001$).

Disc margin shapes encountered in the 65 discs with radial defects are summarized in Table 5 and Figures 3-7. In discs with radial tears, focal disc protrusion related to either: 1) out-pouching of the thinned residual annular margin or 2) out-pouching of the thin peripheral

margin of a connected peripheral annular tear (Figure 3). In discs with annular gaps, protrusion was related to out-pouching of the thinned residual annular margin present (Figure 4). Central radial tears and annular gaps typically demonstrated central or paracentral Type A focal disc protrusion. In lateral radial tears, the tear typically projected into a broad peripheral annular fissure which in most instances created the Type B protrusion shape (Figure 5) as compared with the smaller more focal Type A protrusion shape (Figure 3D).

In 15 of 65 (24%) of discs with RD, the pe-

Figure 3 ‘Type A’ focal protrusions in discs with radial tears. A,B) Patient is a 43-year-old male with a 1 year history of LBP after motor vehicle accident injury with twisting motion. Concordant LBP was provoked at L4-5 during discogram, completely eliminated by intradiscal lidocaine. Lateral fluoroscopic view from the L4-5 discogram (A) demonstrates typical appearance of discographic contrast leakage (arrows). Post-discogram CT demonstrates a central linear radial annular tear is noted (arrow) which projects into a small peripheral annular tear (arrowhead) with a small focal protrusion of the disc margin noted. C) Patient is a 15-year-old athlete with a 2 year history of severe LBP sustained after a foot ball injury. Concordant provoked pain was present at L4-5, completely eliminated by intra-discal lidocaine with discographic contrast leakage present. A central radial tear is present (arrows) that projects into a focal pocket along the peripheral annular margin (arrowheads) having the appearance of a focal disc protrusion. Slight bulge of the disc margin is also present. D) Patient is a 43-year-old female with a 9 year history of LBP following pregnancy. Concordant pain provoked at L4-5 and L5S1. At L3-4, non-concordant pain was provoked with no improvement after intradiscal lidocaine or discographic contrast leakage present. Lateral radial tear is present (arrow) that projects into a contained focal peripheral annular tear (arrowhead) with the appearance of a focal Type A far-lateral disc protrusion.

Table 4 126 significantly painful discs with either total or no response to intradiscal lidocaine: association between peripheral disc margin shape and features of annular internal derangement.

Internal Derangement	Disc Margin Shape		Total
	Diffuse Disc Bulge	Focal Disc Protrusion	
Degenerative changes only (typically D3)	48 (76%)	13 (21%)	61
Radial Defect: Radial Tear/Annular Gap	15 (24%)	50 (79%)	65
Total	63	63	126

Legend: D3: Dallas grade 3 degeneration ($\geq 50\%$ involvement of the annulus).

Table 5 Significantly painful discs with either total or no response to intradiscal lidocaine: disc shapes in 65 discs with radial annular defects (radial tears and annular gaps).

Disc Margin Shape	Internal Derangement Features			Total
	Central RT	Lateral RT	Annular Gap	
Protrusion	16	23	11	50
Diffuse Bulge	2	7	6	15
Total	18	30	17	65

Legend: RT: Radial Tear/Fissure.

ripheral disc margin demonstrated bulge only. In these instances the radial tear or annular gap typically extended into a peripheral annular tear which normalized or smoothed the shape of the outer annular margin (Figure 6).

In the 61 discs with degenerative change only, symmetric or asymmetric bulging of the annular margin was identified in 48 (79%) discs

with a focal protrusion of the disc margin identified in 13 (21%). In most instances complex degenerative features (Dallas grade 3) were encountered including annular lamellar (concentric) separation and tears, free or attached annular fragments and peripheral concentric annular tears (Figure 7A). In the degenerative discs, protrusion was occasionally observed

Figure 4 ‘Type A’ focal protrusions in discs with annular gaps. A) Patient is a 30-year-old female with a 1 year history of LBP after lifting injury at work. Concordant LBP was provoked at L4-5 completely eliminated by intradiscal lidocaine with discographic contrast leakage present. An annular gap is present (curved arrows) with a central focal disc protrusion (arrowheads) along the thin residual peripheral annular margin. B) Patient is a 25-year-old male with 4 year history of LBP after an injury. Concordant LBP was provoked at L5-S1 with no improvement after intradiscal lidocaine and no discographic contrast leakage identified. Moderate annular gap and annular debris are present (curved arrows) contained by the remaining annular margin with a slightly eccentric central disc protrusion (arrowheads) identified.

with focal, irregular/uneven thinning of the residual peripheral annular margin or a small focal peripheral annular fissure (Figure 7B).

Lidocaine response

Response to Lidocaine administration in the 126 discs with degenerative change or radial defect is summarized in Tables 6-8. Discographic contrast leakage was identified in 79 discs with 47 discs appearing contained. Complete provoked pain elimination was identified in 52 of 79 (66%) discs with discographic contrast leakage (cRT: 11; latRT: 13; AG: 7; degen only: 21) with 27 discs (34%) demonstrating no pain improvement after lidocaine injection (cRT: 3; latRT: 10; AG: 2; degen only: 12). Complete pain

elimination was identified in 11 of 47 (23%) contained discs (cRT: 0; latRT: 3; AG: 1; degen only: 7) with 36 discs (77%) demonstrating no improvement after lidocaine injection (cRT: 4; latRT: 4; AG: 7; degen only: 21). The presence of discographic contrast leakage correlated significantly with complete elimination of the provoked pain upon intradiscal lidocaine administration ($p < 0.001$). Leakage was typically seen emanating from characteristic locations including: 1) a small gap in the peripheral annular margin of a full-thickness central radial tear, 2) a tiny pinhole-like point in the annular margin of a central or lateral radial tear with peripheral annular tear or 3) in a permeative-like way along a thinned stretched annular margin (typically discs with either annular gap or diffuse

Figure 5 Patient is a 37-year-old male with a 3 year history of LBP sustained after a lifting injury. Concordant pain was provoked at L3-4, completely eliminated by intradiscal lidocaine with discographic contrast leakage present. Lateral radial tear is present (arrow) that projects into a broad peripheral annular tear that protrudes widely into both the foramen and far-lateral region (arrowheads) with the appearance of a Type B protrusion. Additional concentric lamellar tears are noted.

Figure 6 Patient is a 46-year-old male with 2 year history of progressive LBP. Concordant pain was provoked at L45, completely eliminated by intradiscal lidocaine, discographic contrast leakage was present. A large posterior annular gap (AG) is present (curved arrows) which connects laterally on the right to a peripheral annular tear (arrowheads) normalizing the peripheral annular margin rendering a final appearance of diffuse disc bulge.

disc bulging only). In RD discs, 31 of 46 (67%) with discographic contrast leakage demonstrated complete response to lidocaine with 15 (33%) demonstrating no response (Table 7, $p=0.002$). In this group, 37 of 46 leaking discs demonstrated protrusion with 24 of these 37 discs (65%) demonstrating complete response to intradiscal lidocaine ($p=0.006$). Leakage typically occurred at the annular gap near a focal radial tear, at the edge of a peripheral annular tear or along the thinned stretched residual annular margin. In four of 19 contained RD discs (21%), complete response to intradiscal lidocaine was noted with two of four demonstrating disc protrusion. In discs with degenerative-change-only, complete response to intradiscal lidocaine was noted primarily in discs with con-

trast leakage (Table 8) and this was statistically significant ($p=0.006$). In 48 of 61 discs with degenerative-change-only, disc margin shape was judged as bulge and in these discs complete response to lidocaine was also seen primarily in discs with contrast leakage ($p=0.009$).

Results are re-tabulated to reflect the observed disc margin features of focal protrusion or bulge only (Tables 9-10). Discographic contrast leakage was present in 46 of 63 (73%) significantly painful discs with focal protrusion, and 29 of these 46 discs (63%) responded completely to intradiscal lidocaine administration ($p=0.004$). In discs with bulge only, complete response to intradiscal lidocaine was again primarily encountered with contrast leakage ($p=0.002$).

Figure 7 Discs with degeneration only. A) Patient is a 56-year-old male with a 30 year history of long-standing progressively developing LBP. Concordant pain was provoked at L3-4 without change after intra-discal lidocaine, discographic contrast leakage was present. Extensive (Grade 3) degenerative changes are noted (curved arrows) with only diffuse bulge of the disc margin present (arrowheads). B) Patient is a 57-year-old male with 25 year history of progressive LBP beginning after a motor vehicle accident. Concordant pain was provoked at L5S1 with no improvement after intradiscal lidocaine and no discographic contrast leakage identified. Extensive degenerative changes of the disc are present (Grade 3) with concentric or lamellar tears of the annulus (curved arrows), some of which resemble a bucket handle tear of the meniscus. A central and leftward projecting Type-A focal disc protrusion (paracentral) is noted on the left (arrowhead) in addition to diffuse bulge of the disc margin.

Discussion

Our data in patients with chronic LBP studied with provocation discography demonstrate that focal disc protrusion in significantly painful discs was typically associated with a Dallas Grade 3 radial defect in the annulus (radial tear/annular gap) with retained peripheral annular margin while generalized disc bulging was associated with Dallas Grade 3 degenerative-change-only and this correlation was highly statistically significant ($p < 0.001$). Clinically negative discs generally appeared normal on post-discogram CT with mild to severe degenerative change or Radial Annular Defects uncommon. Similar to previous reports, complete improvement of provoked pain was identified most typically in discs with discographic contrast leakage and this observation was also statistically significant ($p < 0.001$). The features of disc margin shape and internal derangement reflect impor-

tant parts of the morphological state present in chronically painful discs. In addition, the features of disc margin shape, internal derangement and response to anesthetic may help relate to the cause of discogenic LBP in these discs and in these patients with chronic LBP.

Under most circumstances, acute LBP recovers spontaneously or with conservative medical management¹. In a study of work-related acute LBP, pain resolved in two weeks in approximately 50% of patients with 85% experiencing functional recovery in four to six weeks and 95% developing significant improvement and return to work by three months². In approximately 5% of patients LBP persists and becomes chronic^{1,2}. Acute-onset sciatica is less common but symptom resolution is often incomplete with persistent radiculopathy in approximately 50% of patients after four to six weeks^{1,2}.

Patients with acute LBP or sciatica are occasionally operated upon emergently in the set-

Table 6 Significantly painful discs with either total or no response to intra-discal lidocaine: intradiscal lidocaine response in 126 significantly painful discs.

Disc Features	Pain Response to Intradiscal Lidocaine		Total
	Total Elimination of Provoked Pain	No Pain Improvement	
Leak	52 (RD: 31; Deg: 21) 66%	27 (RD: 15; Deg: 12) 34%	79
Contained	11 (RD: 4; Deg: 7) 23%	36 (RD: 15; Deg: 21) 77%	47
Total	63	63	126

Legend: RD: radial defect (radial tear/fissure or annular gap); Deg: degenerative change only; Leak: disc demonstrating discographic contrast leakage; Contained: disc without leakage identified.

Table 7 Significantly painful discs with either total or no response to intradiscal lidocaine: intradiscal lidocaine response in 65 discs with radial defects.

Disc Features	Pain Response to Intradiscal Lidocaine		Total
	Total Elimination of Provoked Pain	No Pain Improvement	
Leak	31 (Prot: 24; Bulge: 7) 67%	15 (Prot: 13; Bulge: 2) 33%	46
Contained	4 (Prot: 2; Bulge: 2) 21%	15 (Prot: 11; Bulge: 4) 79%	19
Total	35	30	65

Legend: Leak: disc demonstrating discographic contrast leakage; Contained: disc without leakage identified; Bulge: bulging disc margin only; Prot: disc margin protrusion.

Table 8 Significantly painful discs with either total or no response to intradiscal lidocaine: lidocaine response in 61 discs with degenerative changes only.

Disc Features	Pain Response to Intradiscal Lidocaine		Total
	Total Elimination of Provoked Pain	No Pain Improvement	
Leak	21 (Prot: 5; Bulge: 16) 64%	12 (Prot: 4; Bulge: 8) 36%	33
Contained	7 (Prot: 1; Bulge: 6) 25%	21 (Prot: 3; Bulge: 18) 75%	28
Total	28	33	61

Legend: Bulge: bulging disc margin only; Prot: disc margin protrusion.

ting of disc herniation with cauda equina compression and loss of bowel/bladder function or severe unrelenting pain unresponsive to medication^{1,17,18}. In most instances patients with LBP are managed conservatively including oral pain medication, bed rest, physical therapy and minimally invasive targeted injections after appropriate clinical targeting (epidural steroid, nerve root, facet/SIJ). When surgery is performed due to persistent radiculopathy, the radiating pain distribution, objective signs of root compression and correlative MR/CT/myelogram imag-

ing are traditionally used to target which disc level should be operated upon^{19,20}.

When chronic LBP is present without specific clinical/imaging clue to the responsible disc level, or, if significant LBP persists after laminectomy/discectomy, the intervertebral disc is often considered a potential source of the pain and fusion is contemplated as advanced surgical treatment. Confirmation of a discogenic source of LBP is difficult clinically and most imaging features are non-specific when used prospectively. Provocation discography remains the only ob-

Table 9 Significantly painful discs with either total or no response to intradiscal lidocaine: lidocaine response in 63 discs with protrusion.

Disc Features	Pain Response to Intradiscal Lidocaine		Total
	Total Elimination of Provoked Pain	No Pain Improvement	
Leak	29 (RD: 24; Deg: 5) 63%	17 (RD: 13; Deg: 4) 37%	46
Contained	3 (RD: 2; Deg: 1) 18%	14 (RD: 11; Deg: 3) 82%	17
Total	32	31	63

Legend: RD: Radial defect (radial tear/fissure, annular gap); Deg: Degenerative change only.

Table 10 Significantly painful discs with either total or no response to intra-discal lidocaine: lidocaine response in 63 discs with bulge only.

Disc Features	Pain Response to Intradiscal Lidocaine		Total
	Total Elimination of Provoked Pain	No Pain Improvement	
Leak	23 (RD: 7; Deg: 16) 72%	10 (RD: 2; Deg: 8) 28%	32
Contained	8 (RD: 2; Deg: 6) 26%	22 (RD: 4; Deg: 18) 74%	31
Total	31	32	63

Legend: RD: Radial defect (radial tear/fissure, annular gap); Deg: Degenerative change only.

jective test that can confirm a discogenic source for axial LBP²¹ and is typically used for preoperative planning in order to determine which disc or discs are responsible in these difficult patients with longstanding LBP.

The findings at provocation discography and accompanying post-discogram CT offer a unique opportunity to correlate discogenic LBP with axial imaging features of the responsible disc in patients with non-acute more chronic pain. Features of the peripheral disc margin are fundamental observations made on lumbar MR/CT imaging studies and post-discogram CT affords the ability to equate the disc margin shape with the underlying features of disc internal derangement.

In our painful discs with radial defects, a gap in the annulus was present, but the peripheral annular margin remained substantially intact. Disc protrusion was seen in the majority of these discs, generally related to out-pouching and stretch of the thin residual annular margin present. A variable degree of generalized disc bulging was present in these internally deranged discs, likely contributing to stretching and thinning of the annular margin. In spite of the radial annular defect, disc extrusion was

not observed due to presence of the residual annular margin. Given the focal location of the annular derangement in these discs, the source of discogenic pain most likely relates to the focal annular defect zone. Identification of the focal protrusion therefore, likely relates to the cause of discogenic LBP in these discs.

The normal disc is innervated in up to the peripheral third annulus only²². In abnormal discs, nociceptors have been identified deep within the annulus or nucleus²³⁻²⁶. Inflammatory byproducts leaking from the disc can promote the in-growth of local pain fibers and granulation tissue²⁷. In addition, inflammatory substances or direct injury can accentuate the nociceptive response (lower nociceptor stimulation threshold, prolong nociceptor response) and increasing the number of nociceptors present, as has been demonstrated in the facet joint (where explicit models exists)^{28,29}. Provoked pain in discs with radial defects could certainly be related to the observed anatomic features of internal derangement. Local pain fibers may be present in the peripheral annular margin or along the annular margins of the radial defect and become sensitized. Radial tears that project to the annular margin (without or

with associated peripheral annular tear) correlate with pain provoked at discography^{15,30}. Alternatively, pain might be related to leakage of inflammatory byproducts present in these discs²¹. Response to intra-discal lidocaine in painful discs correlates with discographic contrast leakage, radial defects and radial tears projecting into peripheral annular tears¹⁶.

In our discs with radial defects, complete elimination of the provoked pain occurred in 31 of 46 (67%) discs which demonstrated contrast leakage. In 37 of these 46 discs, focal protrusion was present and complete elimination of provoked pain occurred in 24 of these 37 discs (65%). The response to intradiscal lidocaine in these discs could be related to anesthetic effects on activated nociceptors at several locations including: along the course of the radial defect, at the peripheral annular margin, within the peripheral annular tear (fissure) or in the epidural space. Therefore, identification of focal disc protrusion, with its implication of underlying radial annular defect, has potential implication relating morphology of disc internal derangement to the cause of discogenic LBP. In discs with RD but without response to intra-discal lidocaine, pain might be related to stretch of the intact annulus or other parts of the functional spinal unit and anesthetic might not reach the responsible nociceptors.

Approximately half of the disc protrusions observed in our patients were central (Type A) with approximately half occurring broadly across the foramen and far-lateral region (Type B) and associated with lateral radial tear and peripheral annular tear. These disc protrusion shapes, including the Type B shape, are commonly observed on axial MR/CT imaging and appear to have a distinct morphologic basis as defined by features of internal derangement.

In our significantly painful discs with generalized bulge of the disc margin, complex features of internal disc degeneration (Dallas grade 3) were typically encountered with variable presence of lamellar (concentric) annular tears, attached annular fragments, loose annular fragments and circumferential peripheral annular tears. In these discs, the degenerative changes likely relate to the pain encountered at provocation but it is more difficult to accurately target the focal location of the discogenic pain source. Response to intradiscal lidocaine with elimination of provoked pain was typically identified in bulging degenerative discs with discographic contrast leakage as compared

with contained discs and this observation was statistically significant (Table 5). This might suggest that leakage of degenerative disc byproducts is an important contributor to pain in a subset of severely degenerative discs.

Age-related effects or disc degeneration are features typically observed when assessing CT/MR imaging for the presence of disc disease. Reduced disc signal on T2-weighted MR images has been incorporated into many population studies that have evaluated for the presence of disc degeneration and is typically equated to altered proteoglycan content and reduced disc hydration^{3,4,6-8,31,32}. Age-related/degenerative changes, including disc bulge and disc protrusion, are observed in asymptomatic individuals as well as symptomatic patients⁵⁻⁸. While altered biochemistry/hydration is undoubtedly present in these discs, significant annular internal derangement is also present including radial annular defects and annular degeneration/fragmentation^{3,4}. This age-related/degenerative internal derangement likely parallels the observations in our patients with LBP (annular fragmentation, RD with thin residual annular margin) and may directly equate to the features of disc bulge or protrusion observed in asymptomatic individuals. The features of annular derangement will also relate to pain in symptomatic patients. Peripheral annular tears (fissures) are commonly observed with current sagittal and axial T2-weighted imaging techniques. Advanced MR imaging, in particular higher field strengths, might make detailed assessment of the internal annular architecture even more feasible.

In a previous study comparing disc margin shape with annular internal derangement, observations similar to our own were made with common identification of full thickness radial annular fissure or tear (radial defect) in association with disc protrusion³³. Pain duration was not stated in this study with disc extrusion and sequestration frequently noted and severe annular degeneration (Dallas grade 3) not common suggesting their population included patients with acute disc herniation. Disc morphology in our patients with chronic LBP more closely parallels the observations of Videman et al. with near-equal identification of severe disc degeneration and radial defects in severely painful discs¹⁴. The features of internal annular derangement that underlie the observation of disc protrusion or bulge might have implications for surgical management. Targeting the painful radial defect as-

sociated with protrusion could be important in correcting disc-origin pain. Discogenic pain related to bulge and severe annular degeneration might require different management.

Limitations

Discography remains a complex and often controversial test. The method of disc injection/provocation and interpretation of the patient's response requires an interventionalist experienced in the technique. Both false negative and false positive results can occur. Injection of the disc can distort or challenge other portions of the functional spinal unit thereby affecting specificity. Alternatively, the pain-ablation response to intradiscal anesthetic (total, partial pain ablation) as occurs in other diagnostic spine blocks

(nerve root, facet, SIJ) might augment aspects of discography's accuracy and specificity. Additional limitations include post-discogram CT interpretation that could be affected by patient-related image quality effects.

Conclusion

In patients with chronic LBP, peripheral disc margin shape correlates with the features of internal annular derangement in significantly painful discs identified at provocation lumbar discography. Disc protrusion is seen in the presence of underlying radial defect (radial tear, annular gap) with thin overlying peripheral annular margin. Disc bulge suggests the presence of complex disc degeneration with annular fragmentation ($p < 0.001$).

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