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Are first-time episodes of serious LBP associated with new MRI findings?

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Abstract

BACKGROUND: Magnetic resonance (MR) imaging is frequently used to evaluate first-time episodes of serious low back pain (LBP). Common degenerative findings are often interpreted as recent developments and the probable anatomic cause of the new symptoms. To date no prospective study has established a baseline MR status of the lumbar spine in subjects without significant LBP problems and prospectively surveyed these subjects for acute changes shortly after new and serious LBP episodes. This method can identify new versus old MR findings possibly associated with the acute symptomatic episode.

PURPOSE: To determine if new and serious episodes of LBP are associated with new and relevant findings on MRI.

STUDY DESIGN: Prospective observational study with baseline and post-LBP MRI monitoring of 200 subjects over 5 years.

OUTCOME MEASURES: Clinical outcomes: LBP intensity (visual analogue scale), Oswestry Disability Index, and work loss. MRI outcomes: disc degeneration, herniation, annular fissures, end plate changes, facet arthrosis, canal stenosis, spondylolisthesis, and root impingement.

METHODS: 200 subjects with a lifetime history of no significant LBP problems, and a high risk for new LBP episodes were studied at baseline with physical examination, plain radiographs, and MR imaging. Subjects were followed every 6 months for 5 years with a detailed telephone interview. Subjects with a new severe LBP episode (LBP≥6/10,>1 week) were assessed for new diagnostic tests. New MR imaging, taken within 6 to 12 weeks of the start of a new LBP episode, was compared with baseline (asymptomatic) images. Two independent and blinded readers evaluated each baseline and follow-up study.

RESULTS: During the 5-year observation period of 200 subjects, 51 (25%) subjects were evaluated with a lumbar MRI for clinically serious LBP episodes, and 3/51 (6%) had a primary radicular complaint. These 51 subjects had 67 MR scans. Of 51 subjects, 43 (84%) had either unchanged MR or showed regression of baseline changes. The most common progressive findings were disc signal loss (10%), progressive facet arthrosis (10%), or increased end plate changes (4%). Only two subjects, both with primary radicular complaints, had new findings of probable clinical significance (4%). Subjects having another MR were more likely to have had chronic pain at baseline (odds ratio [OR]=3.19; 95% confidence interval [CI] 1.61–6.32), to smoke (OR=5.81; 95% CI 1.99–16.45), have baseline psychological distress (OR 2.27; 95% CI 1.15–4.49), and have previous disputed compensation claims (OR=2.35; 95% CI 0.97–5.69). Subjects involved in current compensation claims were also more likely to have an MR scan to evaluate the LBP episode (risk ratio=4.75, p<.001), but were unlikely to have significant new findings. New findings were not more frequent in subjects with LBP episodes developing after minor trauma than when LBP developed spontaneously.

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CONCLUSION: Findings on MR imaging within 12 weeks of serious LBP inception are highly unlikely to represent any new structural change. Most new changes (loss of disc signal, facet arthrosis, and end plate signal changes) represent progressive age changes not associated with acute events. Primary radicular syndromes may have new root compression findings associated with root irritation.

Keywords: Back pain; MRI; Minor trauma; Disc degeneration; Disc herniation; Annular fissure; High-intensity zone; Disability; Psychological distress; Chronic pain; Spinal stenosis

Introduction

Although classically considered a rheumatic and nontraumatic condition, low back pain (LBP) episodes are commonly ascribed today to minor traumatic injuries to the spine [1]. Clinical and population studies of subjects developing serious LBP illness demonstrate significant genetic [2–4], psychological, and social predisposing factors [5–8] and a high degree of nonspinal chronic pain co-morbidity (60–70%) [9,10] and mental disorders (35%) [11]. Despite such evidence, many LBP episodes are described as ''spinal injury'' apparently occurring in the absence of boney or ligamentous injury [1,12,13]. It is often postulated that minor trauma, while unlikely to injure a normal spinal segment, does cause serious structural injury to already degenerative components. The degenerative intervertebral disc is most commonly implicated as the ''injured'' structure, and many findings seen on imaging studies have been attributed to these ''injuries'' [1,12,13].

On the other hand, studies of asymptomatic subjects have shown that loss of disc signal, annular bulging, and facet arthrosis are frequently seen in subjects with no traumatic history nor serious back pain problems [14–17]. Similarly, these findings appear to be most clearly associated with aging [2,9,14,18]. Annular fissures, disc herniation, and end plate fractures have been more commonly attributed to acute events, although no cohort study to date has shown these findings as commonly developing in association with a new LBP episode. Studies that have followed magnetic resonance (MR) changes from a baseline examination to a set follow-up point (eg, 3 or 5 years later) have shown some progression of many types of degenerative changes and these changes are not highly correlated with interval LBP histories [9,19–22]. A limitation acknowledged in these studies is that findings such as annular fissures with bright signals and disc herniations may be transient phenomena occurring at the time of the LBP episode but resolving or evolving over time. Unless an imaging study was taken soon after the LBP episode, a bona fide structural change strongly associated with an acute event could be missed. No study has systematically looked for new structural findings after serious LBP episodes in subjects with known baseline MR findings.

In this study we have recruited a medium-sized (200 subjects) cohort of working persons, without any history of serious LBP problems, but with both an increased risk at baseline of spinal degenerative disease and co-morbid factors (neurophysiological and psychosocial) predisposing to the development of chronic disabling LBP problems. This cohort was examined in detail at baseline, lumbar degeneration documented by X-ray and MR imaging (MRI), and then followed for 5 years with detailed interval histories of LBP episodes and minor trauma events taken every 6 months. New MR images of the lumbar spine were examined in subjects developing persistent clinical LBP and compared with baseline studies. The incidence of LBP, whether associated with minor trauma or arising spontaneously, could then be correlated with the new structural findings (if any) on MR scan.

Our intention was that by recruiting this relatively high-risk cohort we would more closely simulate the subset from the general population with co-morbid chronic nonlumbar pain and psychological profiles, as identified by Von Korff et al. [11], Burton et al. [10], and others [7], who develop serious LBP illness. In this high-risk cohort, we could reasonably expect to observe sufficient serious events over a 5-year follow-up period and to record the new MR findings associated with these events.

Methods

Study design

This is a prospective cohort study designed to investigate whether new episodes of serious low back pain are associated with new structural pathology as detected by high-quality MR imaging. Baseline MR studies performed in asymptomatic or minimally symptomatic working subjects would be compared with new MR studies taken shortly after serious clinical LBP episodes. The development of new MR findings with serious LBP episodes would be considered in the context of perceived inciting events (minor trauma, usual activities, or spontaneous pain).

In order to obtain a wide spectrum of baseline MR findings and to increase the observed incidence of new LBP events over time, subjects at high risk were recruited to this study. All subjects recruited had known risk factors for degenerative lumbar disc disease but no history of clinical LBP episodes. In addition, the subject recruitment strategy was to recruit 50% of subjects with a history of chronic nonlumbar pain as this group is known to have high incidences of both psychological distress and presumed increased neurophysiological effects of chronic pain.

All subjects were then examined for structural pathology of the spine by physical examination, plain radiography, and MRI of the lumbar spine. Outcome measures were serious LBP episodes and occupational disability.

Primary hypothesis

Serious LBP episodes in subjects without LBP histories but with known risk factors for degenerative disc disease will commonly be associated with new MR findings (eg, annular fissures, disc herniation, or end plate fractures) associated with those new symptoms.

Two secondary hypotheses were considered: that minor trauma leading to LBP would be associated with specific new MR findings, such as annular fissures or disc herniation; and the new MR findings would occur with serious LBP episodes independent of chronic pain or psychological findings at baseline.

Subject recruitment

Consecutive patients seen for cervical disc disease at the Stanford University Hospital were assessed for concurrent LBP symptoms as part of a study of cervical disc herniations [15,16,23]. As described in previous publications, patients were screened and subjects without low back symptoms or those who described LBP symptoms as mild and not associated with any functional loss or medical treatment were recruited. For this cohort only working subjects were recruited, excluding some subjects in the original group who were occupationally disabled at baseline, to complete the full cohort of 200 subjects. In addition, potential subjects were identified who had any chronic nonlumbar pain syndrome (eg, chronic cervical pain syndrome, any chronic regional pain syndrome) by International Association for the Study of Pain (IASP) definition. Subjects were recruited by a stratification ratio of 1:1 with and without chronic nonlumbar pain, on a consecutive case basis. That is, one subject with chronic pain (nonlumbar) was selected for admission to the study for each ''pain-free'' (no current cervical or other chronic pain process) subject admitted.

Approval was obtained from the Institutional Review Board and the Administrative Panel of Human Subjects in Medical Research according to U.S. Department of Health and Human Services regulations at Stanford University School of Medicine. Informed consent according to University and Department of Health and Human Services guidelines was obtained from all prospective participants at the time of the original screening.

Potential subjects were excluded by the following criteria: structural spinal abnormalities (spondylolisthesis, scoliosis, Scheuermann's kyphosis, compression fracture, and the like) found on screening; subjects unable to undergo MRI scanning because of ferromagnetic implants, severe claustrophobia, or inability to tolerate positioning for MRI, not working more than 20 hours/week at the time of screening.

Screening for previous low back problems

A screening questionnaire and the Oswestry Disability Index for previous or current low back troubles were administered 6 to 9 months before the subsequent questionnaires in the present study. Patients were asked to evaluate the severity of any LBP using numeric rating scales for ''maximum'' and ''usual'' 0–10 pain over the last week. All subjects were confirmed to have reported being either asymptomatic or minimally symptomatic (<2/10) for all LBP history for screening complaints for at least 2 years before the study. A repeat screening for LBP problems was conducted again before the study start date and has been previously described [15,16,23]. For all subjects, they must have indicated never having sought medical attention for LBP troubles, never having restricted occupational or recreational activities due to LBP problems, and both the numeric rating scale score was <2/10 and the Oswestry Disability Index score must have been 15 or less on two repeated tests: one administered 6 to 9 months before and another within 2 weeks before the study start date.

Baseline physical examination

A physical examination was performed; documenting low back range of motion, the presence of any deformity or tenderness of the thoracolumbar spine, lower extremity neurological examination, and sciatic and femoral root tension signs.

Baseline imaging

All subjects meeting the entry criteria above were examined with plain radiographs and a lumbar spine MRI. Within 4 weeks before the start of the study, a standardized MRI and standing anterior and posterior radiographs were performed. The MRI protocol was a standard clinical examination protocol for lumbar disc pathology without contrast. The protocol used included a T1 sagittal sequence (4 mm thick), a second sagittal sequence (4 mm thick) with repetition and echo times (TR/TE) at 2400–3000/30, 70, and axial images at 4000/21. Two examiners blind to the clinical and demographic data graded degree of disc degeneration, annular disruption, herniation, and end plate status and followed previous reports' methodology [9,17,24,25]. When there was no agreement, a third examiner read the film in question and a modal score was used. Canal stenosis was graded mild to severe by subjective assessment and included all causes of stenosis (congenital, arthritic, or associated with disc pathology). Facet arthrosis and end plate changes (Modic changes) were only recorded if judged moderate or severe. For both of these findings our readers had poor reliability in distinguishing normal from mild changes. Canal stenosis was arbitrarily graded moderate

(touching or displacing nerves) or severe (compressing and distorting nerve). As previously described, baseline imaging of these asymptomatic or minimally symptomatic volunteers were graded in a mixed batch with MR imaging of 42 clinically symptomatic control subjects undergoing routine radiographic evaluation in the Orthopaedic Spine Clinic. Clinical control subjects with spinal deformity were excluded.

Standardized questionnaires

Pain intensity

Numeric rating scale scores of LBP intensity were scored on a 10 cm, 11-point scale with instructions indicating that 0 indicated ''no LB pain'' and 10 indicated the ''worst imaginable pain''.

Functional assessment

The Modified Oswestry Low Back Disability Questionnaire was completed as a measure of subjective functional assessment. The Oswestry Disability Index contains 10 items, each scored from 0 to 5, and the final score is expressed as a percent score (range, 0–100). A higher percent indicates a greater amount of disability [26].

Psychometric studies

A Modified Zung Depression Test and Modified Somatic Pain Questionnaire were administered to each subject. From these measures a classification of subject indicating psychological distress was made according to Main et al. [27].

Follow-up interval assessment

Subjects were contacted every 6 months after baseline measures were complete. A scripted telephone interview was conducted by independent research assistants (TVT, GN, BY) who were blinded to patient baseline data and were not involved in the study design. The interview was conducted by telephone, including an interval medical history, interval lumbar imaging studies history, occupational history, medication usage, and accident or injury history. Subjects were asked specifically about their perceived cause of any new LBP episodes. These were classified as ''Major Injuries'' (defined as LBP episodes associated with high energy trauma resulting in serious visceral injury, proximal long bone, pelvic or spinal fracture or dislocation); ''Minor Injuries to the Low Back'' (defined as any perceived injury to the low back area with a back pain intensity $>2/10$ for at least 48 hours but not meeting the major injury definition *and* with specific instructions that this included such minor episodes as ''injuries'' occurring during lifting, sports, road traffic accidents, or slipping or minor falls); ''Activities of Daily Living'' (defined as LBP perceived to arise from usual and commonly performed activities which have otherwise been well-tolerated); and ''Spontaneous LBP'' episodes (defined as LBP developing with no apparent preceding event). The analysis of ''Major Injuries'' and their clinical associations have been previously reported and are not the subject of this study.

If a minor trauma was reported, an interview algorithm was used to describe the nature of the incident including: mechanism (lifting, fall, road traffic accident, sports injury, others); severity of the incident (weight lifted, awkwardness of lifting/twisting, height of fall, speed of traffic collision); associated injuries; perception of fault if a traffic accident; whether reported as a work injury; and whether a civil claim had been made.

Subjects who reported serious LBP episodes and who had a new MRI performed on clinical grounds were identified. The decision on whether to proceed to a new MRI was based on clinical symptoms and recommendations by the subjects' private physicians. This was not directed on any protocol by the investigators.

Follow-up imaging

Subjects reporting interval lumbar imaging with MR were identified; those images were retrieved, saved on optical discs, and the images were reviewed at the conclusion of the study. These follow-up examinations were graded in the same manner as the original MR images. The graders were blinded to both interval and baseline data. The new ''symptomatic'' MR images were mixed, 1 (new interval study) to 2 (controls), with MR images from both ''asymptomatic'' studies and clinically symptomatic control subjects. Not all interval symptomatic subjects were imaged at our facility: that is, some subjects had new ''outside'' MRI films. For this reason, ''outside'' films from new control subjects were added to the film review batch so that examiners at the final review would not be unblinded by the presence of ''non-university radiology'' films as indicating new interval symptomatic examinations. In addition to those MR findings graded at baseline, additional characteristics were also noted: fractures, pars defect, spondylolysis, and destructive lesions. As all of these abnormalities were exclusions at baseline, these additional possible findings were all considered newly developed although not specifically graded on the baseline intake forms. As stated above, the multiple radiographic and other imaging associated with ''Major [Traumatic] Injuries'' involving fractures and dislocations is not reported in this publication.

Outcomes data

Primary outcomes measures were:

1) MR findings at baseline and follow-up studies, including disc degeneration, annular disruption, herniation, moderate-severe end plate changes, spinal stenosis, neurologic compression.

2) ''Serious back pain episodes'' with a pain intensity defined by a numeric rating scale ≥6 for at least 1 week;
3) Compensation claims and disability from usual occupation due to LBP troubles (divided into ≤1 month work loss and >1 month work loss).

Statistical methods

Descriptive statistics were used to summarize patient socio-demographic and MRI characteristics measured at baseline, and adverse LBP events reported during follow up. Means, standard deviations, and medians were calculated for continuous variables; frequency distributions were generated for categorical variables, and these baseline analyses were performed for both the group as a whole (n=200) and for the chronic pain and nonchronic pain subgroups (100 subjects in each).

Incidence rates of LBP events according to trauma status (spontaneous LBP, activities of daily living LBP, minor trauma, major trauma) were computed for the 5-year follow-up period. Estimated effects of baseline structural findings on subsequent LBP were adjusted for age and sex.

The risk of proceeding to having a new MR with subsequent back pain was analyzed against both baseline variables and characteristics of the index LBP event (minor trauma vs. no minor trauma; compensation vs. no compensation; severity of trauma and radicular complaints vs. back pain alone). The risk of new MR findings was analyzed against the same baseline and LBP episode variables.

StatView statistical program (SAS Institute, Cary, North Carolina) was used for all analyses.

Power analysis

Current literature indicates that subjects with the selected risk factors have approximately 2–4% per year risk of LBP disability, and 20% risk of a lesser LBP episode [5,25,28–30]. If an MR were taken only in serious LBP events with disability, then a 1-year study of 100 subjects would be expected to yield 2–4 new MR in that cohort. Assuming 80% power and alpha=0.05, and a 20% detection of new MR findings, a conservative study design therefore required the recruitment of 200 subjects with a targeted 5-year follow-up (1000 man-years of observation and 20–40 new MRI taken to compare to baseline).

Results

Baseline characteristics and protocol compliance

The characteristics of subjects at baseline are given in Tables 1 and 2. As noted in previous studies, chronic nonlumbar pain was strongly associated with abnormal psychometric scores, smoking, and previous disputed compensation claims. All subjects completed the final 5-year follow-up evaluation. There were 23 missed interval observations (1.2%). One subject completed the final interview 3 months before the 60-month mark. Four subjects completed the final interview more than 30 days beyond the 5-year endpoint (63, 66, 67, and 67 months, respectively).

There was agreement on disc degeneration grade in 81% of the first two readings, 72% of high-intensity zone (HIZ)/annular disruption, 77% of end plate changes, and 79% disc herniations readings. The third reader in the event of disagreement agreed with one of the primary readers in all occurrences of disagreement.

Incidence of LBP events

There were 354 serious LBP episodes (≥6/10 visual analogue scale for ≥1 week) over the 5-year observation period (0.35 episodes/person/year). Of these, 126 were not associated with any specific event (spontaneous LBP), 102 were associated with the usual activities of daily living, 118 were associated with minor trauma events, and 8 were associated with major traumatic injury. Excluding the major traumatic episodes, the 346 serious LBP episodes described were associated with medical care visits in 73 cases (21.1%). There were 25 short-term occupational disability events (≤1 month) and 21 long-term disability events (>1 month) reported.

Baseline clinical and imaging data predicting serious LBP episodes

The association of new LBP with baseline clinical variables is the subject of a separate analysis and publication [31]. Serious LBP events were more commonly reported in the group with baseline nonlumbar chronic pain group (77 events) than those in the pain-free group (41 events) (odds ratio [OR]=4.26; 95% confidence interval [CI] 2.32–7.84). Adjusting for age and sex, an abnormal psychometric profile and smoking correctly identified 72 of 118 (61%) serious LBP events perceived to be associated with minor trauma (OR=3.97; 95% CI 2.19–7.22), p=.004. Adding a history of disputed compensation claim correctly identified 94 of 118 (80%) of the serious LBP events

Table 1
Distributions of baseline characteristics of subjects, by nonlumbar pain status

	All subjects	Chronic nonlumbar pain	No pain	p value
Number	200	100	100	
Age	39.4	38.2	40.8	.34
Sex (% male)	59.5%	62	57	.45
Baseline ODI	5.5	5.9	5.0	.10
Normal DRAM	100	29	71	<.0001
Previous disputed compensation claim	23	21	2	<.0001
Smoking	27.5%	44	11	<.0001
Heavy work	28.0%	25	31	.34

ODI=Oswestry Disability Index; DRAM=Distress Risk Assessment Method (Main et al. [27]).

(OR =10.6; 95% CI 5.50–20.68). Disability was predicted by abnormal psychological profile and previous disputed compensation claim, correctly identifying 41 of 44 (93%) disability events (OR=8.34; 95% CI 4.31–16.16), p<.0001.

There was no association of minor trauma to adverse LBP events: that is, the risk of subjects experiencing a serious LBP event was not higher with one or more minor trauma events (2.8–4.9%/year) compared with none (6.0% / year). There was also no appreciable trend toward more adverse events in subjects reporting a greater number (≥5) of minor trauma events.

The risk of developing a serious LBP episode was 4.2% per year unassociated with minor trauma and 4.8% per year after minor trauma (p=.59). The risk of disability when an LBP event arose with or without a preceding minor trauma event was not different. For a serious LBP episode associated with minor trauma there was a 15.7% risk of disability, and this rate was similar to the risk (15.2%) of disability unassociated with trauma (p=.62).

None of the baseline structural findings significantly predicted serious LBP episode. There was a trend toward more serious LBP associated with Grade 5 disc degeneration (disc collapse), OR 4.40 (p=.08); moderate-severe end plate changes, OR=2.5 (p=.1); or spinal canal stenosis, OR=2.9 (p=.09). These results are described in greater detail elsewhere [31].

Incidence and timing of new MRI for serious LBP symptoms

Fifty-three subjects were evaluated with an MR scan for clinically serious LBP during the 5-year observation period (5.3% per person per year). Of these 53 subjects, 16 had two MR scans to evaluate LBP after different episodes (69 total scans for 346 serious LBP events, 20%). No subject having two MR scans believed they had completely recovered between the two studies, but each felt new symptoms prompted the second MRI.

Subjects having clinical MR scans were different from the study cohort as a whole. Subjects having another MR were more likely to have had chronic pain at baseline (OR=3.19; 95% CI 1.61–6.32), to smoke (OR=5.81; 95% CI 1.99–16.45), have baseline psychological distress (OR 2.27; 95% CI 1.15–4.49), and have previous disputed compensation claims (OR=2.35; 95% CI 0.97–5.69). Within the group having another MR scan, the group having compensation claims tended to be younger and have the scan sooner after the LBP episode began (Table 3).

Table 3 shows subject and episode characteristics of those subjects having a new MRI. The mean time from the baseline (without clinical LBP) MRI to a second (symptomatic) scan was 2.2 (±0.82 SD) years, and 2.9 years to a third scan (±0.54 SD). The time from beginning of the new LBP episode and the second MR was 7.1 weeks (±2.2) and ranged from 1 to 12 weeks. In three cases the primary reason for the scan appeared to be radicular symptoms (leg pain greater than back pain), 21 cases had mixed back and leg pain, and 45 cases had predominant back pain (leg pain≤2 on visual analogue scale). Of 20 serious LBP episodes involving workers compensation or personal injury claims, 15 had at least one MRI scan (75%). Of 326 serious LBP episodes not involving a compensation claim, 38 had one MRI scan (12%). The difference in rates of MR being performed in compensation versus noncompensation settings was significant (risk ratio=4.75, p<.001). All but one of the subjects with two scans had compensation claims.

Comparison of baseline and new MRI findings

Although 53 subjects had 69 new lumbar MRI scans during the study period, two scans could not be retrieved from an outside facility. Therefore 67 MR scans were reviewed after the final 5-year interval and were compared with the baseline studies. Of the 51 subjects who had scans compared with baseline, 43 (86%) showed no new or progressive structural changes. As none of the 16 twice-repeated

Table 2
Baseline MR findings in subjects with no clinical low back pain

	All subjects (n=200)	Chronic nonlumbar pain (n=100)	No pain (n=100)	p value
Completely normal MR	24.5%	28	19	.13
DDD Grade 3–5 (most severe level)	76.5%	72	81	.13
Level of most severe MR DDD changes	L2/3=7	L2/3=3	L2/3=4	.44
	L3/4=28	L3/4=15	L3/4=13	
	L4/5=108	L4/5=49	L4/5=59	
	L5/S1=65	L5/S1=32	L5/S1=33	
Annular fissure or HIZ	19.5%	19	20	.86
Disc protrusion	30.5%	28	33	
Disc extrusion	7%	6	8	
End plate changes (moderate-severe)	21.5%	18	25	.23
Facet arthrosis (moderate-severe)	20%	17	23	.83
Spinal stenosis (moderate-severe)	13%	11	15	.40
Root touching	11%	9	13	.80
Root displacement or compression	3%	2	4	.54

MR=magnetic resonance; DDD=degenerative disc disease; HIZ=high-intensity zone.

Table 3
Clinical characteristics of subjects with new MRI scans

		Minor trauma		
	No trauma	Compensation	No compensation	p value
n	21	30	16	
Age	42.2	39.0	45.4	.10†
Men (%)	10 (48%)	17 (57%)	13 (56%)	.32
Time to scan* (range)	7.1 (2–11)	4.2 (1–12)	6.5 (2–12)	.06†
Chronic pain (%)	16 (76%)	19 (63%)	9 (81%)	.15†
Distress on DRAM	9 (43%)	9 (30%)	7 (44%)	.34
Smoking (%)	6 (28.5%)	11 (36.6%)	6 (37.5%)	.21
Heavy work (%)	6 (28.5%)	10 (33%)	5 (31.2%)	.56
Preceived event				
ADL	10	—	—	—
Spontaneous	11	—	—	—
Fall	—	7	3	
MVA	—	9	0	
Sports	—	0	9	
Lifting	—	13	2	
Miscellaneous	—	1	2	
New MRI findings compared with baseline				
No change	17	27	15	
New finding	2	0	1	
Progressive finding	2	3	0	

DRAM=Distress Risk Assessment Method (Main et al. [27]; ADL=activities of daily living; MVA=motor vehicle accidents.
* Time in weeks from low back pain episode to magnetic resonance scan.
† Compares Compensation to No Compensation.

MR scans showed new changes, only 8 of 67 reviewed scans had new or progressive findings.

There were 23 individual new or progressive findings (Table 4). There were 122 findings at baseline, of which 9 were seen to resolve or improve (below). Progressive signal loss in the disc occurred most frequently (9%), followed by progressive facet arthrosis (6%). There were two new annular fissures and only one of these had a high intensity signal (HIZ). There did not appear to be more new findings in subjects with minor trauma LBP episodes compared with those with no traumatic antecedents. Of eight new or progressive findings found in 38 subjects who perceived their LBP to be the result of traumatic injury to the spine, seven were common degenerative processes highly unlikely to have evolved in the short time from ''injury'' to MR scanning. Furthermore, the time from baseline to MR scan was significantly longer than in subjects showing new or progressive changes (3.6 years), compared with those with no new changes (2.1 years).

Table 5 shows clinical and MR imaging details in the eight subjects with new or progressive findings. Many of the findings clustered in two subjects (Cases 1 and 8), who accounted for 11 of the 23 additional findings. Both of these subjects had primary radicular symptoms and

Table 4
Comparison of baseline prevalence and new or progressive findings on MR studies taken for clinical back pain episodes

	Baseline Prevalence (n=51)	New or progressive findings		
		Adtnl findings (n=67)	Minor trauma (n=38)	No trauma (n=29)
Annular fissure/HIZ	13 (26%)	2 (3%)	0	2
Disc protrusion	17 (33%)	2 (3%)	1	1
Disc extrusion	5 (10%)	1 (1.5%)	0	1
DDD Grade 3–5	43 (84%)			
Advanced DDD 1 grade	—	4 (6%)	2	2
Advanced DDD 2 grade	—	2 (3%)	1	1
End plate changes (mod-severe)	13 (26%)	2 (3%)	1	1
Facet arthrosis (mod-severe)	13 (26%)	4 (6%)	3	1
Spinal stenosis (mod or severe)	9 (18%)	1 (1.5%)	0	1
Spondylolisthesis	0	1 (1.5%)	0	1
Root touching	6 (12%)	2 (3%)	1	1
Root displacement/Compression	3 (6%)	2 (3%)	0	2

DDD=degenerative disc disease; mod=moderate HIZ=high-intensity zone; Adtnl=Additional.

Table 5
Characteristics of subjects with new or progressive findings

	Age	Sex	Chronic pain	Time* (years)	Cause	Occupational disability	Compensation	Symptoms	New findings
1	28	M	No	2.8	SP	Yes	No	R- LP (VAS 8/10) LBP (VAS 2/10) ODI=54	New HIZ L4/5 (Previous Grade 3 DDD) New 11 mm R-Extrusion L5/S1 (Previous Grade 3 DDD) New root compression L5/S1 (right) Progression of facet arthrosis L5/S1
2	31	F	Yes	4.1	Fall	Yes	Yes	LBP (VAS 10/10) B−LP (4/10) ODI=60	Progression L4/5 DDD Grade 3 → 4
3	35	M	Yes	3.5	MVA	Yes	Yes	LBP (VAS 8/10) B–LP (VAS 4/10) ODI=56	Progressive DDD L5/S1 L3 → L5 Progression of facet arthrosis L4/5 (Previous Grade 4 DDD only)
4	40	F	Yes	2.8	Lifting	Yes	Yes	LBP (VAS 8/10) L–LP (3/10) ODI=38	Progression of DDD L4/5 (Grade 4 → 5) Progression of facet arthrosis L4/5 Progression end plate changes (mild → mod) L4/5
5	41	M	No	4.0	ADL	No	No	LBP (VAS 8/10) LP (VAS 0/10)	New disc protrusion R–L3/4, No root contact, New annular fissure (not bright signal) L4/5.
6	44	F	Yes	4.4	Lifting	No	No	LBP (VAS 7/10) R LP (VAS 3.10) ODI=26	New disc protrusion L−L4/5 (previous DDD Grade 4) New root touching Resolved R L5/S1 8 mm extrusion to 3 mm protusion. Resolved root displacement Progressive facet arthrosis L4/5
7	51	M	No	3.2	SP	No	No	LBP (VAS 6.10) B LP (VAS 1/10) ODI=24	Progression DDD L3/4 (Grade 2 → 3) Progression DDD L4/5 (Grade 3 → 4)
8	56	F	Yes	4.5	ADL	Yes	No	LBP (VAS 6/10) LP (VAS 4/10) ODI=40	New Grade I spondylolisthesis L4/5; Progression stenosis (mild → moderate); New root displacement; New root touching; Progressive facet arthrosis L4/5, Progressive DDD Grade 3 → 5 L4/5 Progression end plate changes mild → moderate L5/S1

* Time after baseline MR in years; DDD=degenerative disc disease; mod=moderate; HIZ=high-intensity zone; ODI=Oswestry Disability Index; B=bilateral; R=right; L=left; LP=leg pain; LBP=low back pain; ADL=activities of daily living; SP=spontaneous; VAS=visual analogue scale.

new nerve root compression or displacement. Case 1 had an 11 mm L4–L5 disc extrusion and Case 8 had a new degenerative spondylolisthesis and progressive stenosis. In neither case did the symptoms appear to begin with specific trauma. There were two new disc protrusions; one appeared on the opposite side of the current leg symptoms and is of questionable relevance. The other disc protrusion was not associated with any leg pain.

In the entire group of 67 reviewed MR scans, three disc extrusions at baseline were seen to resolve, one disc protrusion resolved, two root displacement resolved to touching only; two root compression decreased to touching; one HIZ resolved, and one disc signal appeared to improve from a Grade 4 to Grade 3.

Of the 21 subjects with disability lasting more than 1 month, there were only three (14%) new findings: one subject (Case 8) had a new spondylolisthesis and progression advanced stenosis; one (Case 1) had an extruded disc herniation with root compression; one subject (Case 2) had an advance of one grade of degenerative disc disease scoring (Grade 3 to 4).

Discussion

It is not clear what causes LBP in most people. The serious structural lesions such as tumors, infection, fractures, and severe deformities are frequently painful and fortunately can be diagnosed with modern imaging studies. However, these patients with serious structural problems are uncommon. Much more commonly people have back pain episodes of varying degrees and either do not seek care or are treated symptomatically without a pathoanatomic diagnosis.

Why some people with common backache become patients with serious disability is of enormous clinical and public health importance. A common hypothesis in the last century has supposed that minor trauma causes additional structural injury to the degenerative spine [1]. Some have hypothesized that the spinal injury is an acute annular tear extending into the innervated outer annulus [32]. Alternatively an existing annular fissure may become inflamed and appear as a bright annular signal on MR imaging [32–34]. Others have suspected common minor trauma leads to minor end plate failures and causes rapid structural failure of the disc [13,35,36]. Alternatively injury may be suspected of causing disc herniation and either pain distension of the annulus or compression of neural elements.

However, it has been interesting that all of these findings can be seen in subjects with no back pain or only minor problems [3,14,16,17,19–22,37,38]. Also, significant LBP problems can develop without any unusual activity or trauma. Progression of subclinical common backache or acute back pain to serious disabling LBP illness appears to be associated with various nonstructural issues such as emotional distress, poor coping strategies, compensation disputes, and other chronic pain problems [39]. These associative conditions make determining clear structural causes of serious LBP illness problematic.

In this study we attempted to isolate structural findings associated with first-time serious LBP episodes. We hypothesized that when subjects with no significant LBP problems develop acute and serious symptoms, MR imaging will commonly demonstrate new findings. This close temporal association would support a causative relationship between specific structural changes and the development of serious LBP illness. We also were interested in determining if such new findings were more likely to be seen after minor trauma, supporting an ''injury'' model of LBP development. Our results, however, do not support either hypothesis.

Subjects having MR imaging within 12 weeks of a serious LBP episode uncommonly had new findings or progression of old findings. The most common new finding, a progressive loss of disc signal intensity, has been shown to be primarily an aging phenomenon poorly correlated with symptoms [2,9,14,35]. Similarly, progressive facet arthrosis, which was our next most common finding, is a slowly evolving process unlikely to be related to any recent specific event.

Our results can be compared with three other studies, which examined serial lumbar MR images performed at fixed intervals (that is, these studies did not time the follow-up study to any specific clinical event). Elfering, Boos and associates [9,20] after a 5-year follow-up of 46 subjects found 22% of subjects had an increase in degenerative disc disease grade and 6% had new HIZ. These findings compare closely with our own, 9% degenerative disc disease and 3% HIZ after a mean of approximately 2.5 years. Jarvik et al., following an older cohort of veterans over 3 years, found 9% of subjects had disc signal loss, 8% end plate changes, 7% new disc protrusion and 5% new annular fissures [21]. These results are also very similar to our own in younger subjects with a predisposition to lumbar degenerative disc disease. Borenstein et al., following a small and somewhat older group (n=31 and mean age 53), found 70% of subjects with 1 week or more of interval LBP had either no change on serial MRI after 7 years or simple disc bulging [19].

Other studies attempting to predict LBP symptoms with baseline MR findings have also found only weak correlations. The best correlations found by Jarvik et al. involved subjects with baseline stenosis or root involvement [21,22]. Elfering et al. found a marginal statistical significance between progressive degenerative disc disease or end plate changes and work loss caused by back pain [20]. Both of these studies found psychosocial characteristics to be better predictors of significant LBP problems. Borenstein et al. did not find any association between either canal stenosis, disc protrusion or extrusion, and new symptoms, although their numbers were small [19].

A potential weakness in these previous studies has been a concern that acute structural changes which may have

occurred with serious LBP episodes, for instance a disc extrusion or bright annular fissure, may well have resolved when the follow-up MR was taken several years later. In the present study where imaging was performed at a mean of approximately 6 weeks after the LBP episode began, we still did not find the suspected acute changes with any frequency. In fact, there were only two new annular fissures seen, neither associated with trauma, and one of these was apparently a serendipitous finding in a patient with a large disc herniation at another level. The two patients who had well-correlating new pathoanatomic findings both had root involvement and primary radicular symptoms. In fact our results, taken as a whole, appear quite similar to those of the previous work with follow-up MR scans taken at an arbitrary point unrelated to clinical symptoms.

It may be argued that taking an MR within 12 weeks of the LBP episode may not be enough time for some findings to develop, such as the high signal intensity of a presumably inflamed annular fissure. However, we did have data from yet another MR scan taken in 16 subjects at about 6 months after the first clinical MR scan. Not one of these demonstrated a new HIZ or any other new but delayed MR finding.

It is interesting, but perhaps not surprising, that new MR examinations seem to be much more frequently performed when subjects were involved in compensation claims. Of 20 subjects with compensation claims after a serious LBP episode, 15 had at least one MRI scan (75%), compared with only 38 of 326 serious LBP episodes not involving a compensation claim (12%). No patient with a compensation claim had a clear new finding of significant pathology. It is also remarkable that new MRI studies were much more frequently performed in subjects with baseline psychological distress, chronic pain issues, or smoking history. While it may be argued that MR scanning in these more vulnerable subgroups may serve to reassure and help recovery, recent work by Modic et al. suggests that early MR imaging may be associated with a lesser sense of well-being despite benign findings [40].

As a matter of clinical practice, it is important to consider how these MR scans would be interpreted without knowledge of the original baseline studies. In that case, there would appear to be some potentially serious pathology on most studies: nearly 50% have either disc protrusion or extrusion; nearly 30% have annular fissures; and there is potential root irritation in 22% of the studies. If this were the only imaging available, as it most certainly is in clinical practice, then it is easy to see how a clinician may suppose that these findings developed, de novo, with this first serious LBP episode. Furthermore, if the patient suggests a history of minor trauma and no pre-trauma MR is available, all or many of these preexisting findings may be considered evidence of serious structural ''injury''. It is only when viewed from the perspective of knowing that more than 90% of these findings were already present, years earlier, when the subjects were without any significant low back symptoms, that the association of these findings to specific symptoms or events becomes untenable.

This study has certain limitations. The subjects were recruited from a patient pool with a known predilection to disc disease (previously diagnosed cervical disc herniation) and were not a random population sample. This strategy should inflate the prevalence of MR findings in this relatively young group. In fact the findings are most similar to the work of Jarvik et al. on an older random sample of veterans [21,22]. The subject selection did however successfully enroll subjects with a wide spectrum of baseline MR findings with approximately 25% being normal and 25% having severe findings. This would not likely have been seen in a random population sample of the same age. Another limitation of design may be that we did not rescan all 200 subjects at the end of the 5-year study. This would of course greatly increase both cost and logistical burdens. And while this data would have been interesting and would have added comparability to other studies of that design, none of the previous work found high correlations with specific symptoms, and we suspect our results would have been similar. Finally, the use of a modal agreement scoring for the MR findings has both advantages and disadvantages.

Despite these design constraints, we believe the substance of the findings is real and can be reproduced. That less than 5% of follow-up MR scans showed clinically relevant new findings is new and intriguing data. That minor trauma, while very commonly reported, did not correlate with serious LBP episodes or cause any clinically significant structural changes is incompatible with the common ''injury'' model of LBP disability. These findings rather support an alternative hypothesis that disc and spinal degeneration begin very early in life [37], primarily on the basis of nutritional [41,42], developmental [2,37,43], and genetic factors [2,3,18,44]. Later minor traumatic or repetitive occupational events play a minor role, if any, in eventual structural changes and serious disability [2,3,20,37]. Rather, we found the primary predictors of serious LBP disability, as has been repeatedly shown in previous work by many investigators [7,20–22,45–49], were once again psychosocial factors. Most common findings on MR images taken after acute, serious LBP episodes should not, in our opinion, be considered explanatory of either the new event or the severity of symptoms.

Conclusion

Findings on MR imaging within 12 weeks of new and serious LBP development are highly unlikely to represent any new structural change. Most new changes (loss of disc signal, facet arthrosis, and end plate signal changes) represent progressive age-related changes not associated with acute events. Primary radicular syndromes may have new root compression findings associated with root irritation.

References

[1] Allan DB, Waddell G. An historical perspective on low back pain and disability. Acta Orthop Scand Suppl 1989;234:1–23.

[2] Battie MC, Videman T, Parent E. Lumbar disc degeneration: epidemiology and genetic influences. Spine 2004;29:2679–90.

[3] Videman T, Battie MC, Gibbons LE, Maravilla K, Manninen H, Kaprio J. Associations between back pain history and lumbar MRI findings. Spine 2003;28:582–8.

[4] Videman T, Nurminen M, Troup JD. 1990 Volvo Award in clinical sciences. Lumbar spinal pathology in cadaveric material in relation to history of back pain, occupation, and physical loading. Spine 1990;15:728–40.

[5] Bigos S, Battie M, Spengler D, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. Spine 1991;16:1–6.

[6] Klenerman L, Slade P, Stanley I, et al. The prediction of chronicity in patients with acute attack of low back pain in a general practice setting. Spine 1995;20:478–84.

[7] Pincus T, Burton AK, Vogel S, Field AP. A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. Spine 2002;27:E109–20.

[8] Valat JP, Goupille P, Vedere V. Low back pain: risk factors for chronicity. Rev Rhum [Engl Ed] 1997;64:189–94.

[9] Boos N, Semmer N, Elfering A, et al. Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging: predictors of low back pain-related medical consultation and work incapacity. Spine 2000;25:1484–92.

[10] Burton A, Tillotson K, Main C, Hollis S. Psychosocial predictors of outcome in acute and subacute low back trouble. Spine 1995;20: 722–8.

[11] Von Korff M, Crane P, Lane M, et al. Chronic spinal pain and physical-mental comorbidity in the United States: results from the national comorbidity survey replication. Pain 2005;113:331–9.

[12] Adams MA, Dolan P, Hutton WC. The stages of disc degeneration as revealed by discograms. J Bone Joint Surg [Br] 1986;68:36–41.

[13] Adams MA, Freeman BJ, Morrison HP, Nelson IW, Dolan P. Mechanical initiation of intervertebral disc degeneration. Spine 2000;25:1625–36.

[14] Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation. J Bone Joint Surg Am 1990;72: 403–8.

[15] Carragee EJ, Alamin TF, Miller J, Grafe M. Provocative discography in volunteer subjects with mild persistent low back pain. Spine J 2002;2:25–34.

[16] Carragee EJ, Tanner CM, Khurana S, et al. The rates of false-positive lumbar discography in select patients without low back symptoms. Spine 2000;25:1373–80. discussion 1381.

[17] Jensen M, Brant-Zawadzki M, Obuchowski N, Modic M, Malkasian D, Ross J. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 1994;331: 69–73.

[18] Videman T, Battie MC. The influence of occupation on lumbar degeneration. Spine 1999;24:1164–8.

[19] Borenstein DG, O'Mara JW Jr, Boden SD, et al. The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects: a seven-year follow-up study. J Bone Joint Surg Am 2001;83-A:1306–11.

[20] Elfering ADP, Semmer N, Birkhofer D, Zanetti M, Hodler J, Boos N. Young Investigator Award 2001 Winner: Risk factors for lumbar disc degeneration—a 5-year prospective MRI study in asymptomatic individuals. Spine 2002;27:125–34.

[21] Jarvik JG, Hollingworth W, Heagerty PJ, Haynor DR, Boyko EJ, Deyo RA. Three-year incidence of low back pain in an initially asymptomatic cohort: clinical and imaging risk factors. Spine 2005;30:1541–8; discussion 1549.

[22] Jarvik JJ, Hollingworth W, Heagerty P, Haynor DR, Deyo RA. The Longitudinal Assessment of Imaging and Disability of the Back (LAIDBack) Study: baseline data. Spine 2001;26:1158–66.

[23] Carragee EJ, Chen Y, Tanner CM, Truong T, Lau E, Brito JL. Provocative discography in patients after limited lumbar discectomy: a controlled, randomized study of pain response in symptomatic and asymptomatic subjects. Spine 2000;25:3065–71.

[24] Carragee EJ, Paragioudakis SJ, Khurana S. 2000 Volvo Award winner in clinical studies. Lumbar high-intensity zone and discography in subjects without low back problems. Spine 2000;25:2987–92.

[25] Weishaupt D, Zanetti M, Hodler J, et al. Painful lumbar disk derangement: relevance of endplate abnormalities at MR imaging. Radiology 2001;218:420–7.

[26] Fairbank JC, Couper J, Davies JB, O'Brien JP. The Oswestry low back pain disability questionnaire. Physiotherapy 1980;66:271–3.

[27] Main C, Wood P, Hollis S, Spanswick C, Waddell G. The distress and risk assessment method (DRAM): a simple patient classification to identify distress and evaluate the risk of a poor outcome. Spine 1992;17:42–52.

[28] Adams MA, Mannion AF, Dolan P. Personal risk factors for first-time low back pain. Spine 1999;24:2497–505.

[29] Jarvik JJM, Hollingworth W, Heagerty P, Haynor D, Deyo RA. The Longitudinal Assessment of Imaging and Disability of the Back (LAIDBack) Study—baseline data. Spine 2001;26:1158–66.

[30] Weishaupt D, Zanetti M, Hodler J, Boos N. MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers. Radiology 1998;209: 661–6.

[31] Carragee EJ, Alamin TF, Cheng I, Franklin T. Does minor trauma cause serious low back illness? Spine 2006; submitted.

[32] Aprill C, Bogduk N. High-intensity zone: a diagnostic sign of painful lumbar disc on magnetic resonance imaging. Br J Radiol 1992;65: 361–9.

[33] Lam KS, Carlin D, Mulholland RC. Lumbar disc high-intensity zone: the value and significance of provocative discography in the determination of the discogenic pain source. Eur Spine J 2000;9: 36–41.

[34] Saifuddin A, Mitchell R, Taylor BA. Extradural inflammation associated with annular tears: demonstration with gadolinium-enhanced lumbar spine MRI. Eur Spine J 1999;8:34–9.

[35] Adams MA. Biomechanics of back pain. Acupunct Med 2004;22: 178–88.

[36] Adams MA, McNally DS, Dolan P. 'Stress' distributions inside intervertebral discs: the effects of age and degeneration. J Bone Joint Surg Br 1996;78:965–72.

[37] Boos N, Weissbach S, Rohrbach H, Weiler C, Spratt KF, Nerlich AG. Classification of age-related changes in lumbar intervertebral discs: 2002 Volvo Award in basic science. Spine 2002;27: 2631–44.

[38] Videman T, Nurminen M. The occurrence of anular tears and their relation to lifetime back pain history: a cadaveric study using barium sulfate discography. Spine 2004;29:2668–76.

[39] Klenerman L, Slade PD, Stanley IM, et al. The prediction of chronicity in patients with an acute attack of low back pain in a general practice setting. Spine 1995;20:478–84.

[40] Modic MT, Obuchowski NA, Ross JS, et al. Acute low back pain and radiculopathy: MR imaging findings and their prognostic role and effect on outcome. Radiology 2005;237:597–604.

[41] Urban JP, Roberts S. Degeneration of the intervertebral disc. Arthritis Res Ther 2003;5:120–30.

[42] Urban JP, Smith S, Fairbank JC. Nutrition of the intervertebral disc. Spine 2004;29:2700–9.

[43] An HS, Anderson PA, Haughton VM, et al. Introduction: disc degeneration: summary. Spine 2004;29:2677–8.

[44] Battie MC, Videman T, Gibbons LE, Fisher LD, Manninen H, Gill K. 1995 Volvo Award in clinical sciences. Determinants of lumbar disc

degeneration. A study relating lifetime exposures and magnetic resonance imaging findings in identical twins. Spine 1995;20: 2601–12.

[45] Allan DB, Waddell G. An historical perspective on low back pain and disability. Acta Orthop Scand Suppl 1989;234:1–23.

[46] Bigos SJ, Battie MC, Spengler DM, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. Spine 1991;16:1–6.

[47] Burton A. Spine update: back injury and work loss: biomechanical and psychosocial influences. Spine 1997;22:2575–80.

[48] Rainville J, Sobel JB, Hartigan C, Wright A. The effect of compensation involvement on the reporting of pain and disability by patients referred for rehabilitation of chronic low back pain. Spine 1997;22: 2016–24.

[49] Waddell G. 1987 Volvo award in clinical sciences. A new clinical model for the treatment of low-back pain. Spine 1987;12:632–44.