Can Post-Op Recurrent Lumbar Disc Disease be Prevented?

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Abstract
Our purpose is to offer treating physicians guidelines, which can be proposed to their patients post-op, so that recurrent lumbar disc disease can be prevented. We have reviewed Medline and other possible resources. We have collected 714 relevant articles, which have been studied. The surgeon can try to close the annular defect or to keep it small, but the current relevant materials have not yet withstood the probation of time. Patients after lumbar discectomy or other similar techniques should be careful of trauma and other violent forces, avoid lifting weights for a few months post-op, try to lose weight if overweight, stop smoking, control diabetes mellitus, hypertension and increased cholesterol levels and discuss problems and treat psychological diseases and symptoms as necessary.

CORE TIP

During a lumbar discectomy the surgeon can try to close the annular defect or to keep it small. The current materials used for this purpose have not yet proved their adequacy in time.

Following a lumbar discectomy, we propose the following directions for the patient:
1. be careful of trauma and other violent forces.
2. Avoid lifting weights for a few months post-op.
3. Try to lose weight if overweight.
4. Stop smoking.
5. Control diabetes mellitus, hypertension and increased cholesterol levels.
6. Discuss problems and treat psychological diseases and symptoms as necessary.

INTRODUCTION

Our purpose is to offer the practicing clinicians guidelines on what they can advice their patients after a lumbar disc disease operation, so that they minimize the risk of recurrence. Recurrent postoperative disc disease is one of the main problems of lumbar discectomy. Nearly one-fourth of patients undergoing lumbar discectomy demonstrated radiographic evidence of recurrent disc herniation at the level of prior surgery, the majority of which were asymptomatic [1].

This recurrence appears clinically in 5-11% of patients [2]. Since the overall rate of unsatisfactory results after discectomy is between 5% and 20%, it is therefore a major cause of surgical failure [3].

We define it as a recurrent disc disease which appears at the same spinal level, classically at least six months after the operation. Some authors include only reherniations at the same side, others at the opposite side also. We believe that opposite side reherniations should be included, because it is the same disc that suffers. Despite this definition many authors include all disc reherniations, irrespective of postoperative time period. About 2 per thousand patients experience disc recurrence during the first week [4]. Some have even found that their mean time of recurrence was less than six months. Sixty percent of all pain relapses occurred within 1 year following the first operation; thereafter, the probability of a relapse declined steadily and was as low as 0.1% per year between 5 and 20 years [5].

Finally, many authors use the term recurrence more broadly, including almost all cases of surgical failure due to disc disease, irrespective of whether it is a real recurrence or not.

Recurrent disc disease is not the only cause of surgical failure in lumbar disc disease. We can find in the literature topics, where diminution of the number of cases of recurrence can possibly lead to worse results. For example, there is a problem of how much disc should be removed during operation: Discectomy or sequestrectomy? Extensive discectomy can lead to less disc recurrences but worse clinical outcomes.

In all cases where a surgical technique or device is proposed as a solution to the problem of disc recurrence, the added complications or costs from the technique or the device should be included in the analysis. For example, intervertebral spacers can be added and possibly reduce the rate of disc recurrence, but these materials can slip out of the disc space and cause pressure on neural elements.
Real disc disease recurrence is mainly due to the extrusion of another part of the nucleus pulposus through the preexisting opening of the annulus fibrosus, causing new clinical signs. The extent of disc removal affects the frequency of the event: More discs removed, less it remains to be extruded. On the other hand, extensive disc removal can lessen the height of the disc, straining the facets, can cause damage to the end plates and finally lead to worse clinical results. There have been efforts to close the annulus fibrosus opening, initially with a stitch. This can lead to increased intradiscal pressure, allowing the disc to continue its supportive role and creating better conditions for its regeneration [6].

Nucleus pulposus can regenerate after the operation. The problem is that the annulus fibrosus does not regenerate satisfactorily. Only fibrous closure of the defect can be expected [4]. Whatever we use for closing its opening must stand the probation of time. It is like using instrumentation without fusion for a lifetime.

Improvement of annulus fibrosus regeneration has been tried experimentally. Bioactive electrospun scaffold for annulus fibrosus repair and regeneration has been tried assayed [7]. The mRNA gene expression of recurrent disc cells were increased 1.47 times for aggrecan, 1.38 times for type I collagen, 2.04 times for type II collagen, 1.22 times for both Sox-9 and osteocalcin, and 1.31 times for alkaline phosphatase, respectively, compared with the primary herniated lumbar disc cells. These results indicate that the recurrent disc cells have similar chondrogenic and osteogenic gene expression compared to primary herniated disc cells [8].

Since disc regeneration can influence the rate of disc recurrence, bibliography on primary disc disease is useful for our review. We have studied this bibliography, but we must not forget that these articles use surrogate variables as far as it concerns our main discussion. A clear distinction is made concerning the population of each study.

MATERIALS AND METHODS

We have divided our topic in four parts: General and genetics, conservative measures, surgical techniques, surgical devices. Two authors were allocated to each part. The authors worked independently, so that the search of the relevant literature and its evaluation could be done independently. Medline was searched systematically. Relevant articles found in the references were also included. There were 714 relevant collected papers, which were read and are reviewed in this article. The main author collected and synthesized the whole data. No metaanalysis of the existing data has been tried. For the final form of the article a collective discussion was done. References were kept to a necessary minimum, because in such a topic there is the risk of allowing more pages to the references than to the main article. This also means that in subtopics where a systematic review existed, this review was used as the main reference. We have adopted the following grades of recommendation based on the North American Spine Society (NASS) Clinical Guidelines Levels of Evidence and Grades of Recommendation [9]:

A: Good evidence (Level I studies with consistent findings) for or against recommending intervention.

B: Fair evidence (Level II or III studies with consistent findings) for or against recommending intervention.

C: Poor quality evidence (Level IV or V studies) for or against recommending intervention.

I: Insufficient or conflicting evidence not allowing a recommendation for or against intervention.

GENETICS

Are there susceptible populations for increased risk of recurrent disc disease? The answer is yes, because there are populations with increased prevalence of disc disease, for example in tandem spinal disc disease. The encumbrances of 18-year-old or younger patients with lumbar disc herniation showed familial predisposition, with an odds ratio of 5.61 in comparison to the controls [10].

Genetic analysis has shown that pathological discs display different gene expression than their normal counterparts. There are a number of genes that have been associated with intervertebral disk degeneration in humans, including genes coding for collagen I, collagen IX (COL9A2 and COL9A3), collagen XI (COL11A1), IL-1, aggrecan, vitamin D receptor, MMP-3, and CILP. For specific genes and some environmental factors, gene-gene, gene-environment and gene-age interactions may exist [11]. We do not know if differences in gene expression are predisposing to disease or are adaptive to degeneration.

Homozygosity for the Arg allele of COL9A2 seems to be more frequent in the patient group with early recurrent disc disease [12]. It is suggested that (-511) T>C SNP in IL-1beta gene may be one of the susceptible alleles for lumbar disc disease (LDH) [13]. People with the mutation genotypes 5A of MMP-3 and/or A of VDR-Apa may have the increased risk of developing lumbar disc degeneration if they are exposed to whole-body vibration and/or bending/twisting [14]. The results of environment-gene interaction analysis revealed that, in LDH, the interaction of the FasL -844TT genotype and level III to IV lumbar load was consistent with the ultramultiplying model, and the interaction of the CASP9 rs4645978 GG genotype and level III to IV lumbar load was consistent with the submultiplicative model [15]. The findings in a Chinese military cohort indicated that CASP-9 (-1263A/G) and GDF5 (+104T/C) polymorphisms are associated with a susceptibility to low back pain (LBP) related to military training [16].

The highlight of another study was that in the same study population with degenerative disc disease (DDD), SNP associations completely changed when different radiographic features were used to define the DDD phenotype. This study therefore indicates that standardization of the phenotypes chosen to study the genetics of disc degeneration is essential and should be strongly considered before planning genetic association studies [17].

GENERAL PREDISPOSING FACTORS OF SPINAL DISC DISEASE

Gender

Besides a slower recovery rate, female gender was a strong
predictor of unsatisfactory outcome at one year for patients with sciatica [18].

Trauma

15-42% of patients with recurrent disc disease more than 6 months postoperatively had a history of trauma [19]. Initial disc herniation is due to trauma or injury in only 0.2-10.7% of cases.

Annulus fibrosus opening closes by fibrous tissue. This mechanism is relatively effective, because disc recurrence rate diminishes with time. Trauma can cause the extrusion of a new disc segment outside the confines of the annulus, causing new neurological symptoms. Even if its rate of provoking disc extrusion diminishes with time, its relative importance increases, because residual disc and other factors diminish with even a faster rate.

Repetitive lifting-Occupation and other activities

Frequent lifting of objects or children weighing 25 or more pounds with knees straight and back bent was associated with increased risk of herniated lumbar disc. Positive associations among confirmed case subjects were also seen for frequent lifting with arms extended and twisting while lifting [20].

A review uncovered 35 high-quality studies examining a relationship between occupational lifting and LBP, but these studies did not consistently support any of the Bradford-Hill criteria for causality. There was moderate evidence of an association for specific types of lifting and LBP [21].

A review of 9 selected papers failed to identify high-quality studies that supported any of the Bradford-Hill criteria to establish causality between occupational lifting and LBP. Based on these results, it is unlikely that occupational carrying is independently causative of LBP in the populations of workers studied [22].

A review of 24 studies failed to uncover high-quality data to support any of the Bradford-Hill criteria to establish causality between occupational sitting and LBP. Strong and consistent evidence did not support criteria for association, temporality, and dose response [23].

In another review, the 32 eligible studies included did not support a causal association between workplace manual handling or assisting patients and LBP in a Bradford-Hill framework. Conflicting evidence in specific subcategories of assisting patients was identified, suggesting that tasks such as assisting patients with ambulation may possibly contribute to LBP [24].

In another systematic review of eight studies, methodologically rated as high quality, with a total of 7023 subjects who were considered for risk analysis, different outcome measures for postural exposure was adopted making meta-analysis difficult to perform. The authors could not find a clear dose-response relationship for work posture exposures and LBP. Limited evidence was found for range of motion and duration of sustained flexed posture as risk factor for LBP. They found no evidence for frequency of trunk flexion as a risk factor for LBP [25].

Sedentary occupations increase the risk for herniated lumbar discs, particularly among those aged 35 years and older who have had sedentary jobs for several years. People in occupations requiring prolonged driving of motor vehicles, such as truck drivers, appear to be at particularly high risk [26].

The logistic regression analysis of another study demonstrated that the lack of regular physical exercise was a significant predictor for reoperation [27].

Review of 35 studies does not support the popular opinion that sitting-while-at-work is associated with LBP [28].

Prolonged motor vehicle usage

The greater the number of hours spent in a motor vehicle, the higher the risk. Use of Swedish and Japanese cars was associated with a lower-than-average risk, while use of other cars was associated with a higher-than-average risk. For each type of car, older cars were associated with higher risks than newer cars [29].

There is strong evidence that leisure time sport or exercises, sitting, and prolonged standing/walking are not associated with LBP [30].

Collectively, eight systematic review reports on occupational activities included 99 studies. None found strong evidence supporting a causal relationship between any occupational physical activity considered and LBP. Conflicting evidence was found between LBP and bending, twisting, lifting or pushing/pulling, but only for statistical association, not causation. Strong evidence against a causal relationship was found between LBP and manual handling/assisting patients, awkward postures, carrying, sitting, standing or walking [31].

Pregnancy

The mean number of pregnancies was greater in women with low-back pain (2.6) than in those without (1.6) (P < 0.001) [32].

Obesity

A review of 56 articles has shown that thirty-two percent of all the studies report a statistically significant positive weak association between body weight and LBP. Studies that fulfilled the post hoc criteria never report a rate ratio above 2, but there is possibly a positive biological gradient. These studies had no information on temporality or reversibility [33]. In a single study, obese patients were 30 times more likely to require reoperation [34]. There is no evidence that overweight patients consistently lose weight as a result of lumbar decompression with or without fusion. Two small studies suggest that an overweight patient undergoing lumbar surgery is as likely to gain weight as to lose weight following surgery [35].

Spinal anatomy

Disc height index and sagittal range of motion showed a significant correlation with the incidence of recurrent lumbar disc herniation, suggesting that preoperative biomechanical conditions of the spine can be an important pathogenic factor in the site of lumbar disc surgery [36].

Thoracic flat back is a risk factor for lumbar disc degeneration after scoliosis surgery [37]. Smaller recurrences are symptomatic when canal stenosis is prominent.
Smoking

A review of 38 papers has concluded that the available data are consistent with the notion that smoking is associated with the incidence and prevalence of nonspecific back pain, but there are too few studies to make any conclusions for the other end points (e.g., sciatica, herniated discs) [38]. In a single study, relative risks for current smokers were 3 times for lumbar disc [39].

Some studies found a positive association between smoking and LBP; when present, the strength of this association was generally small. Some associations remained unchanged after multivariate analysis, whereas others became statistically nonsignificant. Contradictory results were also noted in studies which reported on the dose-response relationship and time of exposure in relation to time of onset of LBP. There was inconsistency of findings within and between studies relating to LBP. However, the evidence was consistently against a causal association between smoking and sciatica/discal hernia [40]. In eight of the thirteen studies that tested a preliminary positive association between smoking and LBP, this association remained after multivariate analysis, whereas it disappeared in after analysis in five [41].

Diabetes mellitus

Seven of the 25 (28%) diabetic patients had reoperation for recurrent disc herniation at the same level following the initial procedure compared with one of the 28 (3.5%) control patients [42].

Atherosclerosis and cardiac disease

In a relevant review, 25 papers were included. Post-mortem studies showed an association between atheromatous lesions in the aorta and disc degeneration, as well as between occluded lumbar arteries and lifetime low back pain. In clinical studies, aortic calcification was associated with low back pain, and stenosis of lumbar arteries was associated with both disc degeneration and low back pain. In epidemiological studies, smoking and high serum cholesterol levels were found to have the most consistent associations with disc degeneration and low back pain [43].

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In another review, the multivariate relative risk for diabetes was 1.52 (95% confidence interval [95% CI], 1.17 to 1.98); for hypertension it was 1.25 (95% CI, 1.11 to 1.41); for high cholesterol it was 1.26 (95% CI, 1.10 to 1.44), and for having a parent who had suffered a myocardial infarction before age 60 it was 1.13 (95% CI, 1.02 to 1.26). In these models, compared with never smokers, the relative risk for past smokers was 1.10 (95% CI, 1.00 to 1.20), for current smokers the risk increased with the number of cigarettes smoked per day. A decrease in risk occurred after cessation. Also in these models, the authors noted a significant trend of increasing risk with higher body mass index (p=.01) [44].

Considering sex, smoking and heavy works as predictors of recurrent LDH, surgeons should advise their patients to limit hard work and put away smoking especially in tall and male ones to prevent LDH recurrence [45].

Psychosocial factors

Patients reporting low-back pain also reported more episodes of anxiety (P < 0.001) and depression (P < 0.001) and had more emotionally stressful occupations (P < 0.001) [32].

In a systematic review of the subject, 25 studies were identified, of which none were randomized controlled trials and only four SCS studies met inclusion criteria. The methodological quality of the studies varied and some important shortcomings were identified. A positive relationship was found between one or more psychological factors and poor treatment outcome in 92% of the studies reviewed. In particular, presurgical somatization, depression, anxiety, and poor coping were most useful in helping to predict poor response (i.e., less treatment-related benefit) to lumbar surgery and SCS. Older age and longer pain duration were also predictive of poorer outcome in some studies, while pretreatment physical findings, activity interference, and presurgical pain intensity were minimally predictive [46].

Psychological factors can possibly convert an otherwise rather asymptomatic recurrence to a symptomatic one.

Surgery

Surgical discectomy for carefully selected patients with sciatica due to lumbar disc prolapse provides faster relief from the acute attack than conservative management, although any positive or negative effects on the lifetime natural history of the underlying disc disease are still unclear.

Generally speaking the results of standard discectomy and microdiscectomy are equivalent. Standard discectomy in first-time lumbar herniation of nucleus pulposus (HNP) may increase the risk of subsequent same-level lumbar disc degeneration compared with microdiscectomy as seen in one low-quality study. However, disc degeneration is likely a natural, temporal consequence following HNP, as demonstrated in a second low-quality study. The overall strength of evidence for the conclusions is very low [47].

Total removal of disc vs. removal of its protruding part

In a relevant review, there is fair evidence that conservative discectomy will result in shorter operative times and a quicker return to work despite similar lengths of hospital stay, similar pain levels at discharge, similar 6-month functional status, and a similar 2-year incidence of persistent/recurrent back and leg pain. There is poor quality evidence that conservative discectomy will result in a lower incidence of recurrent back pain beyond 2 years postoperatively. There is fair quality evidence that conservative discectomy will result in a higher incidence of recurrent disc herniation [48].

Review of 54 eligible studies has shown a greater reported incidence of long-term recurrent back and leg pain after aggressive discectomy but a greater reported incidence of recurrent disc herniation after limited discectomy [49].

Annular defect

Subjects with larger annular defects were associated with an increased risk of recurrent disc herniation (P = 0.019) [50].
Intervertebral disc lavage

There is a possible trend towards intervertebral disc lavage reducing the rate of recurrence [51].

Fibrosis inhibitors

Fibrosis inhibitors are not discussed in this article, because they do not address the problem of disc recurrence.

Other surgical techniques

The current evidence suggests that both open discectomy and minimally invasive discectomy (MID) lead to a substantial and equivalent long-term improvement in leg pain. Adequate decompression, regardless of the operative approach used, may be the primary determinant of pain relief-the major complaint of many patients with radiculopathy. Incidental durotomies occurred significantly more frequently during MID, but total complications did not differ between the techniques [52].

Higher risk of dural tears and recurrent herniation has been detected with lumbar micro-endoscopic discectomy [53].

The evidence for other minimally invasive techniques remains unclear except for chemonucleolysis using chymopapain, which is no longer widely available [54].

Injectable nucleus replacement

There is no adequate evidence of effectiveness in prevention of disc recurrence for injectable nucleus replacement.

Nucleoplasty with coblation

The indications are not the same with discectomy, because nucleoplasty is generally indicated only for contained discs. Anyhow, a higher recurrence rate has been reported, probably due to more conservative disc removal or coexisting lumbar stenosis.

Interspinous elastic stabilization

The implant does not significantly change the intradiscal pressures at the adjacent levels, yet it significantly unloads the intervertebral disc at the instrumented level in the neutral and extended positions [55]. Interspinous fusion devices have a high complication rate, reoperation rate, poor outcome and high cost [56]. The current Wallis implant is probably incapable of reducing the incidence of recurrent herniations [57]. Other possible actins of these devices, such as alleviation of the symptoms of spinal stenosis or prevention of facet degeneration are not discussed in this article.

Closing the annular defect techniques

The primary outcome measure, reherniation surgery rates at 3 months, 6 months, and 2 years, did not differ statistically between the experimental and control groups. However, a post hoc subgroup analysis was conducted for individuals presenting with predominant leg pain as indicated by VAS leg and back pain scores. For this subgroup, the frequency of reoperation due to reherniation was reported as lower in the Xclose group at 3-month follow-up and 6-month follow-up [58].

The conclusion of the American Pain Society (Chou et al, 2009) seems well balanced and fair: They concluded that there is fair evidence of effectiveness and a moderate net benefit through to two years but longer term outcomes were unknown [59].

DISCUSSION

We should not be restrictive with our guidelines. The operation is done with the expectation of normal life in the future. We believe that only fair evidence or above justify restrictions in everyday life postoperatively. It would be good if these guidelines are in accordance with existing general medical health advices. There is a rich bibliography concerning genetic mutations and lumbar disc disease (LDD). Few specifically address the problem of recurrent disc. Their ratio of appearance in diseased disc is almost always less than 4. As far as it concerns studies of gene expression we are not sure if the appearing differences are causal or adaptive. Different definitions of disc disease can lead to different correlations. Finally, we cannot suggest populations which should be excluded from difficult occupations. Trauma is a major factor of lumbar disc recurrence, the main factor in the time period after six months post-op.

There is fair evidence that lifting weights can cause LDH, especially when the back is bent or the torso is twisted. There is fair evidence that sedentary life and occupations can increase the risk of LBP. Especially people in occupations requiring prolonged driving of motor vehicles, such as truck drivers, are at high risk. Lack of regular physical exercise is a significant predictor of reoperation. Poor or conflicting evidence exists for the other usual physical activities in work or leisure. There is fair evidence that there is a weak correlation between obesity and LBP. There is no evidence that overweight patients consistently lose weight post-op. There is fair evidence that smoking is associated with LBP. Cessation of smoking reduces the risk. There is fair evidence that diabetes mellitus can lead to lumbar disc recurrence. There is fair evidence that hypertension, atherosclerosis, especially of the aorta, and increased cholesterol levels are associated with increased risk of LBP and LDD.

Psychological diseases, especially anxiety, depression and somatization predict a worse outcome post-op. This correlation is not causative, but these diseases can possibly convert a rather asymptomatic disc recurrence to a symptomatic one.

Discectomy is the standard treatment offered to patients with LDD and appropriate clinical symptoms and signs. Conservative discectomy can lead to better post-op results, but the evidence is poor. There is fair evidence that more radical discectomy can lead to less recurrent disc herniations, but this result is probably clinically insignificant. No recommendation can be made concerning the quantity of disc removal. There is fair evidence that larger annular defects are associated with an increased rate of recurrent disc herniations. There is poor evidence that intervertebral lave can reduce the rate of recurrence. Minimally invasive techniques have not been shown to reduce the risk of disc reherniation. Some of them increase it, possibly because of reduced disc removal and reduced bone removal in cases of coexisting lumbar stenosis. There is inadequate evidence for the use of various types of intervertebral spacers for reduction of incidence of disc recurrence.
There is poor evidence that interspinal elastic stabilization can reduce the rate of disc recurrence.

There is fair evidence that techniques for closure of annular defect, especially in susceptible subpopulations, can protect from disc recurrence. The main problem is that longer follow-ups are necessary.

CONCLUSIONS

During a lumbar discectomy the surgeon can try to close the annular defect or to keep it small. The current materials used for this purpose have not yet proved their adequacy in time.

Following a lumbar discectomy, we propose the following directions for the patient:

1. be careful of trauma and other violent forces.
2. Avoid lifting weights for a few months post-op.
3. Try to lose weight.
4. Stop smoking.
5. Control diabetes mellitus, hypertension and increased cholesterol levels.
6. Discuss problems and treat psychological diseases and symptoms as necessary.

REFERENCES


