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EXPERT OPINION

A Proposed Diagnostic and Treatment Algorithm for the Management of Lumbar Discogenic Pain

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Background: There is renewed interest in the intervertebral disc as a target for treatments aimed at ameliorating lumbar discogenic pain by restoring and preserving the natural structure and function of this component of the vertebral motion segment.

Methods: Using a modified Delphi methodology involving a panel of 11 experts, we developed a simple, understandable clinical algorithm to serve as a foundation for objective decision making regarding the diagnosis and treatment of lumbar discogenic pain throughout the entire continuum of care. A decision tree approach was utilized with "either/or" choices at each branch or node in the algorithm. Clinical activities in this algorithm were divided into examination procedures and corresponding treatment interventions. Corresponding treatment options were designated based on published degenerative disc disease (DDD)-specific clinical practice guidelines and/or meta-analyses.

Results: This algorithm recommends a systematic rule set for discogenic pain diagnostic and treatment options. Initially, the presence of lumbar discogenic pain is confirmed via assessment of a series of clinical signs including axial midline back pain (≥ 4 of 10), pain with flexion, sitting intolerance, positive pain provocation with sustained hip flexion, and absence of motor/sensory/reflex changes. Radiographic severity of DDD is graded by modified Pfirrmann grade (1 to 8). Treatment options are stratified by DDD severity to include conservative management (grades 1 and 2), minimally-invasive intradiscal therapies (grades 3 to 7), and more invasive surgical procedures (grade 8). Recognizing that the management program for patients with lumbar discogenic pain can be highly personalized, the treatment options recommended by this algorithm should be considered general guidance.

Conclusion: The proposed algorithm offers an easy-to-use clinical tool for identifying, evaluating and treating patients with lumbar discogenic pain. The successful implementation of this algorithm involves an important interplay between advanced practice providers, interventional pain physicians and spine surgeons.

Keywords: discogenic pain, disc degeneration, intradiscal, minimally-invasive, algorithm

Introduction

The recent issuance of specific International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM) codes for lumbosacral discogenic pain associated with degenerative disc disease by the National Center for Health Statistics (NCHS) underscores the importance of developing a clinical algorithm to enhance our ability to precisely diagnose and effectively treat patients suffering from discogenic low back pain.[1] Recognition of discogenic pain as a unique source of back pain represents the culmination of decades of extensive basic science and clinical research on the degeneration of the intervertebral disc and its role in precipitating widespread degenerative changes across the entire spinal motion segment.[2–4]

As our understanding of the sources and anatomical structures that contribute to chronic low back pain has evolved,[5,6] estimates of the prevalence of patients suffering from pain emanating principally from the anterior column of the spine

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has expanded to include as many as 70% of the cases of low back pain.[7] Indeed, anterior column pain has been differentiated further to involve not only the intervertebral disc but additionally the vertebral endplates.[8] Due to their anatomical intimacy,[9,10] it is recognized that there exists substantial pathoetiological interdependence of these structures in pain generation.[11]

Using an approach that integrates expertise in musculoskeletal imaging, interventional pain management, and spine surgery, we developed a pragmatic discogenic pain clinical algorithm to improve diagnostic efficiency and maximize clinical benefit. Successful implementation of this algorithm is optimized by utilizing a team approach to back pain management that can effectively address and personalize the myriad diagnostic and treatment options available to the patient throughout their continuum of care.[12]

Methods

Our primary objective was to develop a simple, understandable clinical algorithm that can serve as a foundation for physicians to confidently make objective decisions regarding the diagnosis, management and treatment of lumbar discogenic pain throughout the entire clinical course of the condition. In realizing and refining this algorithm, we utilized a modified Delphi methodology whereby a panel of 11 experts in this area were engaged to form a consensus around diagnostic components and characteristics, the full spectrum of treatment options as well as the temporal sequence of interventions with the objective of maximizing clinical benefit and efficiency of care.[13] The algorithm development process was administrated and coordinated by one panel member who aggregated panel member responses. The progression was iterative, commencing with the establishment of the clinical definition of lumbar discogenic pain in June, 2023,[4] followed by an in-person gathering of the expert panel in December, 2023 to further refine key clinical examination features and prioritize the range of interventions.[14] Final input was solicited and collated from the panel members at pain society conferences in July and October, 2024.

Our proposed algorithm recommends a systematic rule set of diagnostic and treatment decisions for lumbar discogenic pain based on unambiguous alternatives and clear stopping rules.[15] A decision tree approach was utilized with "either/or" choices at each branch or node in the algorithm which progresses logically in a temporal fashion (Figure 1). Actions and events are segregated into physical examination assessments, diagnostic procedures and corresponding treatment interventions. Diagnostic and clinical evaluation queries are represented in the algorithm as *diamonds*, with corresponding symptom severity grading and treatment options shown as *rectangles*.

Published validated standards were employed to grade symptom and radiographic severity as well as assessments of clinical change. Commensurate treatment choices were selected based on published discogenic pain-specific clinical practice guidelines and/or meta-analyses to support each option.

Results

Diagnostic Characteristics

The fundamental components of the patient physical examination were derived from the foundational article by Bogduk[16] and consolidated into a suite of clinical features characteristic of lumbar discogenic pain.[17] These features, when considered altogether, enhance the diagnostic accuracy for this condition.[2,18] During the initial patient encounter, a thorough clinical assessment should be conducted to determine the likelihood and duration of possible discogenic pain symptoms (Figure 1, Box 1).

To identify lumbar discogenic pain, the physical evaluation should consider five components. First, the prominent pain location should be axial midline back pain.[2,19] Clinical manifestation of non-discogenic low back pain due to painful facet or sacroiliac joints is not commonly midline.[20] The patient may also experience referred leg pain but it should be described as non-radicular/non-sciatic pain in a sclerotomal distribution.[16] Patient-reported low back pain severity should be ≥ 4 at the time of physical examination (0 to 10 scale).

Second, the pain is primarily exacerbated with forward flexion.[21] Lumbar flexion involves axial loading which increases annular strain and correlates with pain emanating from degenerated lumbar intervertebral discs.[22] Since the fulcrum of the anterior column is located posteriorly, a small percentage of patients may have pain with extension.

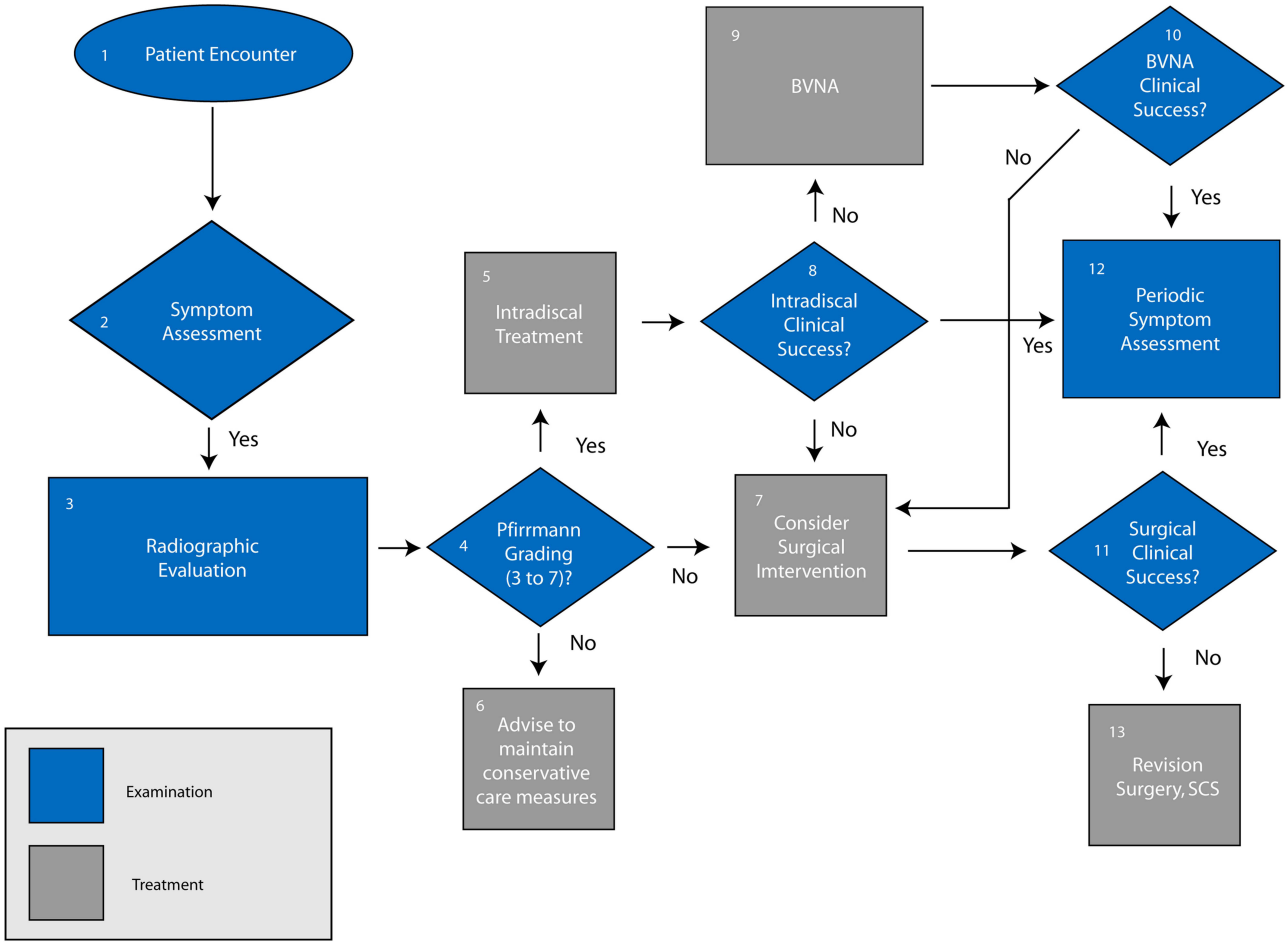

Figure 1 Diagnostic and Treatment Algorithm for Lumbar Discogenic Pain.

Notes: [1]The initial encounter with a health care practitioner (eg, physician, nurse practitioner, physician's assistant) used to evaluate by discussion and physical examination a patient's history and symptoms, and to provide advice, counseling, or treatment. [2]Comprehensive physical examination to determine and confirm presence of lumbar discogenic pain. Definitive features include axial midline back pain, pain with flexion, sitting intolerance, positive pain provocation with sustained hip flexion, absence of motor/sensory/reflex changes. Low back severity should be ≥ 4 (out of 10) for a period of ≥ 6 months. [3]Patients exhibiting chronic lumbar discogenic pain should undergo a magnetic resonance imaging (MRI) study using a standard lumbar spine imaging protocol. [4]Based on a T2-weighted sagittal view by MRI, disc degeneration of all lumbar levels should be graded using the modified Pfirrmann scale (1 to 8). [5]Patients exhibiting chronic clinical symptoms of lumbar discogenic pain with severity of ≥ 4, MRI evidence of Pfirrmann grades 3 to 7, with or without Modic type 1 and/or 2 changes. Treatment options consist of intradiscal injection of mesenchymal stromal cells, platelet rich plasma and nucleus pulposus tissue allograft. [6]Patients with lumbar discogenic pain and MRI evidence of Pfirrmann grades 1 and 2. Advise continued conservative care with a structured management regimen of manual physical therapy and individualized exercises. [7]Patients with lumbar discogenic pain and a modified Pfirrmann grade of 8 or patients who fail to achieve clinical success with basivertebral nerve ablation. Advise surgical consultation to consider discectomy with instrumented interbody fusion or disc arthroplasty. [8]Patients should report at least ≥ 30% improvement in low back pain severity by 6 months post procedure. [9]Patients with Modic type 1 and/or 2 changes who fail to achieve clinical success with first line intradiscal treatment should consider basivertebral nerve ablation (BVNA). [10]Patients should report at least ≥ 30% improvement in low back pain severity by 6 months post procedure. [11]Patients should report at least ≥ 30% improvement in low back pain severity by 12 months post procedure. [12]Patients should receive periodic monitoring (eg, annually) of their symptoms of lumbar discogenic pain to assess degree of improvement/worsening and/or to identify new onset symptoms. [13]If the patient has not achieved a clinically successful outcome and/or exhibits worsening or new onset symptoms, revision surgery, spinal cord stimulation (SCS) and/or facet denervation should be considered.

Third, the patient should exhibit significant functional limitation in sitting duration and tolerance.[23] The pain may become worse when patients sit without support, especially when sitting forward. A sitting intolerance limit of 30 minutes is considered the standard threshold for discogenic pain.[24]

Fourth, the pain should be provoked and reproduced with sustained passive hip flexion.[20] This maneuver is undertaken with the patient in the supine, straight-leg position. Both legs are elevated simultaneously to approximately 45 degrees and then both are allowed to come down slowly. As with forward flexion, biphasic straightening from flexion produces strain across the disc with pain provocation indicating that both or either leg activates back pain.[19] This clinical assessment should not be confused with the straight leg raising test (Laseque's sign) which is a neurodynamic examination to evaluate nerve root irritation or compression associated with radiculopathy secondary to disc herniation.[25]

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Fifth, the foregoing conditions should exist in the absence of motor/sensory/reflex changes. The patient should exhibit a normal lower extremity neurologic exam without marked motor weakness or other deficits.[18,21] Dermatomal radiation and other neurologic symptoms such as motor weakness and numbness are absent with discogenic pain.

Patients demonstrating the foregoing constellation of signs and symptoms for greater than six months can be classified diagnostically as exhibiting lumbar discogenic pain (Figure 1, Box 2). We provide the following acronym, *DISCS*, as a helpful cue in the initial assessment of the back pain patient:

D - Duration of symptoms: Chronic pain persisting for more than six months.

I - Increased pain with flexion: Pain is aggravated with forward flexion due to annular strain.

S - Sitting intolerance: The patient shows limited sitting tolerance, typically less than 30 minutes.

C - Central back pain: Axial midline pain without radicular symptoms.

S - Sustained hip flexion test: Pain is reproduced with sustained hip flexion in the supine position, distinguishing discogenic pain.

Imaging Assessment

Imaging of the lumbosacral spine is essential in the workup of patients with the heretofore-described clinical features of discogenic pain.[26–28] Specifically, the minimum imaging to classify the degree of degenerative changes in the disc requires a sagittal T2-weighted magnetic resonance imaging (MRI) view (Figure 1, Box 3). This imaging sequence allows for the grading of intervertebral disc degeneration based on MR signal intensity, disc structure, distinction between nucleus and annulus, and disc height.[28] Originally devised by Pfirrmann et al[29] and later modified to an eight-category rating scale, this system describes the progression from a normal disc to severely degenerated disc. Grade 1 corresponds to no disc degeneration while Grade 8 corresponds to end-stage degeneration.[30]

Grades 1, 2, and 3 are based on the signal intensity of the nucleus (ie, hyperintense ranging from cerebrospinal fluid [grade 1] to presacral fat [grade 3]) with a distinct junction between the inner and outer fibers of annulus posteriorly and normal disc height. For Grade 4, the margins between the inner and outer fibers of the annulus at the posterior margin of the disc are indistinct with normal disc height. For Grade 5, the disc is uniformly hypointense (ie, equal to outer annulus fibers) with an indistinct junction between inner and outer annular fibers and no loss of disc height. For Grades 6, 7, and 8, there is a hypointense MR signal and progressive loss of disc space height. These are broadly classified as mild (< 30%), moderate (30–60%), to severe (>60%) loss of disc height, respectively (Figure 1, Box 4).

Sagittal MRI views should also be evaluated for the presence or absence of Modic changes.[31] These vertebral bone marrow signal intensity changes are graded as absent, 1, 2 or 3. Briefly, type 1 changes represent bone marrow edema and inflammation; type 2 changes represent normal red haemopoietic bone marrow conversion into yellow fatty marrow as a result of marrow ischemia; and, type 3 changes represent subchondral bony sclerosis.[32]

First-Line Interventions

Several first-line interventions are now available to the interventional pain physician to treat lumbar discogenic pain.[14,33,34] In this treatment algorithm, these therapeutic options should be reserved for patients exhibiting the previously specified cluster of clinical symptoms with low back pain severity of ≥ 4, MRI evidence of Pfirrmann grades 3 to 7, and with or without Modic type 1 and/or 2 changes (Figure 1, Box 5).

Without disrupting the normal anatomical structures of the vertebral motion segment, these minimally invasive therapies can be delivered intradiscally via cannula directly into the target degenerated disc(s) with the objective of supplementing, restoring and preserving native disc structure and function. In the US, these intradiscal therapies are classified as either exempt from regulatory oversight and demarcated as a tissue (section 361 of the Public Health Service (PHS) Act) or as a device, biologic or drug product (section 351 of the PHS Act).[14] This latter group of interventions requires Food and Drug Administration (FDA) oversight and adherence to strict regulatory guidelines for market approval.[35,36] There are several types of intradiscal therapies available commercially or in clinical development including autologous bone marrow concentrate (BMC), platelet rich plasma (PRP), nucleus pulposus (NP) allograft as well as autologous and allogeneic mesenchymal stromal cells (MSCs).[33,34,37–43] NP allograft is available for commercial use as a 361 tissue product whereas autologous BMC and PRP are exempt from regulatory oversight. Regulatory classification

of MSC products is variable.[44] To date, no 351 products have been approved by the FDA specifically indicated for use to treat lumbar discogenic pain (Figure 1, Box 4).

Mesenchymal Stromal Cells

The core evidence supporting the clinical efficacy of MSCs in the treatment of discogenic pain consists of a number of feasibility investigations as well as a pivotal trial.[14,42,45] Specifically, Noriega et al[46,47] conducted a randomized sham controlled trial of allogeneic bone marrow derived MSCs in 24 patients with over 3 years of post procedure followup. Infiltration of the paraspinous musculature with an anesthetic agent served as the sham control. Significant differences in favor of MSC treatment were demonstrated between study groups in back pain and functional improvement throughout the followup period. Sham treated patients, in contrast, exhibited progressive degradation in all outcomes including structural disc degeneration with increasing Pfirrmann grades on MRI over baseline, whereas the MSC treated patients maintained the improved Pfirrmann grades achieved during the initial year after injection.[46]

Aggregating the pain severity findings over all published MSC studies of discogenic pain based on the proportion of individuals achieving substantial clinical benefit (≥ 50% pain relief) at 6 months after intradiscal administration, the aggregate success rate was 54% (95% Confidence Interval (CI): 39%-68%).[34] Society guidelines indicate Level III evidence for intradiscal MSC therapy.[48]

Platelet Rich Plasma

Intradiscal autologous PRP injection has also shown benefit in patients with chronic lumbar discogenic pain.[49–51] A meta-analysis of all published studies of PRP for disc degeneration estimated the aggregate 6-month success rate for substantial clinical benefit in back pain at 55% (95% CI: 40%-70%).[34] Similar efficacy findings were confirmed in additional meta-analyses conducted by Peng et al and Muthu et al[39,40] Post procedure MRI studies failed to show structural improvements within the intervertebral disc associated with intradiscal PRP treatment. Society guidelines indicate Level III evidence for intradiscal injections of PRP.[48]

Nucleus Pulposus

There is a growing body of evidence indicating clinically significant and durable improvements in low back pain and function after intradiscal treatment with NP allograft.[14] The commercially available 361 NP product is known as VIA Disc NP (Vivex Biologics, Miami, Fl, USA).

The most recent clinical results are summarized from three studies and include patients spanning a large age-range. Beall et al[52] reported on 29 patients (mean age, 44 ± 13 years) with symptoms of lumbar discogenic pain and corresponding imaging evidence of disc degeneration treated with a single intradiscal administration of NP allograft. The average back function and pain severity improvements between baseline and 6 months were 55%, and 53%, respectively ($p<0.001$). A minimal clinically important difference (MCID) of ≥ 30% improvement over baseline was achieved in 79% and 68% of patients for back function and pain, respectively. At 6 months, 64% of patients had a pain score ≤ 3.

In an older population of 21 patients (age range: 65–76 years) with lumbar discogenic pain, Azeem et al[53] reported a 76% and 66% improvement in back pain and function, respectively, at 6 months following a single NP allograft procedure. Corresponding responder rates for substantial clinical benefit for back pain and function were 86% and 71%.

Evaluating the back pain and functional outcomes in a heterogeneous population of 21 patients (age range: 41–73 years) with lumbar discogenic pain treated with NP allograft in a real world clinical setting, Lin et al[54] reported that 86% of patients reported some level of satisfaction with the procedure, of which 71% reported "extreme satisfaction". Additionally, there was an average 58% improvement in back pain severity at the latest followup assessment (range: 4–24 months).

For patients diagnosed with lumbar discogenic pain and MRI evidence of Pfirrmann grades 1 and 2, continued conservative care should be considered. If not undertaken previously, these patients should be offered a structured and supervised conservative management regimen consisting of a combination of manual physical therapy and individualized exercises such as the McKenzie extension program and behavioral posture changes.[55] Additional diagnostic modalities

such as magnetic resonance spectroscopy (MRS) may be considered for patients that report bothersome pain symptoms without structural changes on MRI (Figure 1, Box 6).

For patients with functionally disabling lumbar discogenic pain and a Pfirrmann grade of 8, surgical consultation should be considered (Figure 1, Box 7).[56]

Clinical Success Criteria

To evaluate the clinical effectiveness of first line intradiscal therapies, we recommend the periodic evaluation of all patients to assess the magnitude of symptom change and whether additional tests or procedures are warranted. The treatment goal should determine whether patients have achieved a clinically significant outcome within 6 months of the index intervention (Figure 1, Box 8). At a minimum, patients should be queried regarding the current severity of their low back pain. Comparison of baseline pre procedure pain severity status with scores reported at clinical followup should employ the MCID and optionally, substantial clinical benefit (SCB), and the patient acceptable symptomatic state (PASS).[57] For discogenic low back pain, the validated MCID reflects a $\geq$ 30% improvement over baseline,[58] the SCB represents $\geq$ 50% improvement,[59] and the PASS threshold is $\leq$ 3.[60] In this algorithm, the panel recommends that, at a minimum, the MCID be used to determine patient success and guide subsequent therapeutic interventions.

Secondary Interventions

We recommend additional multidisciplinary team consultation for patients who fail to realize a clinically significant improvement and/or exhibit worsening or new onset symptoms following first line intradiscal therapy. For patients with Modic type 1 and/or 2 changes evident on MRI, basivertebral nerve ablation (BVNA) should be considered for L3 to S1 involvement (Figure 1, Box 9). For patients without Modic changes, surgical consultation should be considered (Figure 1, Box 7). Surgical options for chronic lumbar discogenic pain consist of discectomy with instrumented interbody fusion or disc arthroplasty.[27,61] Both approaches remove the painful disc and either stabilize the motion segment, as in the case of fusion,[62,63] or preserve motion, as in the case of disc arthroplasty.[64] While there is ongoing debate regarding choice of surgical treatment for symptomatic disc degeneration, several meta-analyses have concluded that lumbar fusion provides improvement in back function that is no better than that achieved with nonoperative care alone.[65–67] The reimbursement barriers to lumbar disc arthroplasty notwithstanding,[68,69] our panel concluded that it remains preferable to preserve natural motion across the joint if at all possible.

Similar clinical success criteria as noted previously should be employed to evaluate the effectiveness of secondary interventions for disc degeneration and discogenic back pain with the caveat that assessment of clinical success should be undertaken within 6 months of BVNA (Figure 1, Box 10) and within 12 months for surgical interventions (Figure 1, Box 11). For all interventions deemed a treatment success, periodic symptom assessment should be undertaken at least annually (Figure 1, Box 12). For patients who fail to experience clinical success post surgery (ie, failed back surgery syndrome), additional interventions may be considered such as revision surgery, spinal cord stimulation and/or facet denervation (Figure 1, Box 13).

Discussion

Using a modified Delphi methodology, we engaged a panel of experts in musculoskeletal imaging, interventional pain management, and spine surgery to develop a pragmatic discogenic pain clinical algorithm that provides a foundation for making procedural decisions related to the management of patients with lumbar discogenic pain. The advantage of this Delphi-based approach is the ability to develop consensus around this topic by involving experts with substantial experience in treating such patients and with significant contributions to the existing literature on this issue.

We recognize that many decisions regarding the diagnosis and treatment of patients with disc degeneration require personalized treatment plans that cannot be addressed via binary choices in an algorithm. Indeed, many patients with chronic low back pain have multiple contributory spinal conditions that can act as pain generators. That said, the current algorithm specifically focuses on identifying those patients where the primary complaint is axial midline back pain without radicular symptoms, a symptom set highly specific to disc associated pain.

Although not mandatory components of this algorithm, there are several additional imaging assessments that serve to further characterize the degree of intervertebral disc degeneration for an individual patient with lumbar discogenic pain. The standard MRI evaluation includes the presence of high-intensity zones (HIZ), nuclear signal, disc height, disc contour, and bone marrow intensity changes.[70] Specifically, Modic changes on MRI aid in the diagnosis of discogenic or vertebrogenic pain as signal intensity changes on T2- and T1-weighted sequences are useful in classifying scans of vertebral endplates and subchondral bone into categories describing inflammatory changes, fatty degeneration of bone marrow, and sclerosis of subchondral bone.[26,71,72]

Historically considered a *sine qua non* for the diagnosis of discogenic pain, positive provocation of back pain by discography can accurately reproduce the patient's pain at the target disc and serves as a sensitive and specific diagnostic tool.[73] Pain reproduction is attributed to contrast extravasation into annular fissures or defects in the endplate associated with disc degeneration.[74] A positive discogram requires reproduction of pain > 6/10 in intensity and at a pressure < 15–20 pounds per square inch above opening pressure at a volume of less than 3.0 mL of contrast.[75] The false-positive rate for low pressure (< 20 psi) discography has been reported to be as low as 6%.[76,77] Provocative discography can also be combined with computed tomography (ie, CT discogram) to identify the location and extent of annular disruption.[78] Discography is safe if conducted under specified standards and guidelines.[77] Additionally, the test can be conducted using a very small dose of a local anesthetic agent to relieve the pain (eg, functional anesthetic discogram).[79] One of the consequences of non coverage for diagnostic discography was a lack of not only doing the procedure in many parts of the United States, but training the future physicians about its pros and cons. Now it has been all but lost as a useful diagnostic tool. Given that many younger pain physicians received little if any formal training in discography and many centers no longer offer the test, the panel elected not to include discography as a mandatory algorithm requirement. We do, however, strongly encourage physicians who are unfamiliar with the procedure to seek out legacy interventionalists for hands on didactic instruction.

A promising imaging technology to assist physicians in identifying chemically sensitive, painful discs utilizes MRS of the lumbar intervertebral disc.[80] This approach employs proprietary signal processing software that transforms raw spectral MRS data into metabolic biomarkers, such as alanine, proteoglycans, lactic acid and propionate, to differentiate between painful and nonpainful discs (Aclarion, Broomfield, CO, USA).[81] This allows for the evaluation of early disc degeneration as well as the assessment of disease progression and the efficacy of intradiscal therapies.[82]

As first line interventions in the treatment of lumbar discogenic pain, we included three therapies currently available to the interventional pain physician, MSCs, PRP and NP allograft. There has been tremendous medical interest in the utilization of MSCs in the treatment of a number of musculoskeletal disorders due to their multilineage differentiation potential and immunomodulatory properties. However, MSC compliance with section 361 of the PHS Act as a tissue product has been mixed.[83] While most products adhere to FDA's characterization of the 361 homologous use requirement of "forming and replenishing the lymphohematopoietic system",[35] many MSC therapies likely skirt the corresponding minimal manipulation requirement. For example, culture expansion of MSCs is described as more than minimal manipulation by FDA.[35] Use of autologous cells without further manipulation and employing the same day surgical exception, on the other hand, is compliant under Section 361 of the PHS Act. Allogeneic MSC preparations have also be marketed as 361 compliant products although exact specifications for differentiating these preparations from regulated 351 products are not well defined.[83] This has created confusion in the marketplace.[84,85] There has also been a proliferation of questionable stem cell clinics and off shore medical tourism destinations promoting unproven autologous stem cell treatments.[86] In fact, it has been estimated that there are over 700 clinics offering direct-to-consumer marketing of stem cell treatments.[87] While our panel was generally supportive of MSC therapy for lumbar discogenic pain, we recommend any treatment utilizing MSCs ensure product consistency as well as optimize improvements in cell therapy outcomes for both patients and practitioners by adhering to the parameters developed by an international consensus committee based on the acronym DOSES: D—Donor, O—Origin tissue, S—Separation Method, E—Exhibited Characteristics, S—Site of Delivery.[87]

We elected not to include intradiscal steroid injections as a treatment option for lumbar discogenic pain. There is some evidence that steroids may have limited influence in ameliorating endplate inflammatory abnormalities.[88–90]

However, in line with previous society guidelines and meta-analyses, we are not currently recommending intradiscal steroid injections as an effective and durable treatment for discogenic low back pain.[90]

There are several encouraging first line intradiscal treatments under development and clinical evaluation as FDA regulated products indicated specifically for discogenic pain.[14,91,92] These include, but are not limited to, allogeneic precursor MSCs in a hyaluronic acid carrier (rexlemestrocel-L, Mesoblast, Melbourne, AU),[93] injectable allogeneic disc progenitor cells (Rebonuputemcel, DiscGenics, Salt Lake City, UT, USA),[94] a polymer composite hydrogel (PVA/PEG/PVP/barium sulfate; Hydrafil, ReGelTec, Baltimore, MD, USA),[95] a synthetic 7-amino acid peptide (SB-01, Spine BioPharma, New York, NY, USA) that binds to and induces down regulation of Transforming Growth Factor Beta 1 (TGFβ1),[96] spinal cord stimulation,[97] and a novel electroceutical procedure that employs a multielectrode catheter (Discure, Petach-Tikva, Israel).[98]

It is important to draw the distinction between discogenic and vertebrogenic pain syndromes as they have different treatment algorithms. While there may be diagnostic overlap, we eschew the use of the term "discovertebral" that has entered the medical lexicon. Specific ICD-10-CM codes have been issued for discogenic (M51.360 – M51.379) and vertebrogenic (M54.51) pain underscoring the distinction between these pain generators.[1] A necessary feature of vertebrogenic pain is the presence of Type 1 or Type 2 Modic changes on MRI. These are commonly seen with other findings such as inflammation, edema, vertebral endplate changes, disruption and fissuring of the endplate, and vascularized fibrous tissues within the adjacent marrow. Edematous signal (Type 1 Modic change), and changes to the vertebral body marrow including replacement of normal bone marrow by fat (Type 2 Modic change) can also be associated with discogenic pain but are not necessary features in the differential diagnosis. Direct disc stimulation with provocative or anesthetic discography or the use of MRS may aid in pinpointing whether the disc or the endplate is the definitive source of low back pain, but an unspecified proportion of patients may exhibit characteristics of both pain syndromes.

Additionally, it is important to emphasize that interventionalists performing intradiscal procedures should ensure rigorous aseptic technique and consider preprocedural screening for *Cutibacterium acnes* colonization, especially in patients with chronic lumbar discogenic pain and recurrent infections,[99] to minimize infection risk and optimize outcomes.

The approach taken in developing the current discogenic pain algorithm was distinctly different than would be used in the formation of clinical practice guidelines, which are comprehensive dossiers and offer multiple options with graded levels of evidence.[15] In contrast, we provide unambiguous alternatives and clear stopping points. As such, the proposed algorithm should be considered general guidance, appreciating that the treatment trajectory for some patients may fall outside this definitive rule set. In reality, each clinical decision should result in a personalized treatment plan based on discussions between physician and patient about the risk/benefit profile.

The modified Delphi methodology we employed has several limitations including bias associated with participants altering their response to align with the majority view, limited open discussion, the dependence on the expertise of the responder to interpret the results, as well as the lack of guidelines and standards for selecting the expert panel members. We also note the distinction between clinical and mathematical algorithms in this context with the former utilized to guide clinical decision-making by providing a structured approach to diagnosis, treatment, and patient care and the latter used to perform calculations, analyze data, make predictions, and suggest treatments.

The operative treatment of spinal pathology in degenerative disc disease has traditionally been under the management purview of orthopedic spine surgeons and neurosurgeons. However, the recent commercial adoption of several minimally invasive treatment options has extended these intradiscal interventions to an expanded group of physician subspecialties. Interventional pain physicians play an integral role not only in the accurate diagnosis of lumbar discogenic pain but also in utilizing the burgeoning armamentarium of first line interventions to avert surgical intervention and restore and preserve the natural physiology, mechanics and structure of the intervertebral disc. Thus, we believe a team approach that integrates the knowledge base of imaging, pain management and orthopedic/neurosurgical specialists provides the most comprehensive method for managing patients with the full array of available intradiscal treatments and surgical options. This algorithm provides a straightforward clinical decision process for identifying, evaluating and treating patients with chronic lumbar discogenic pain.

Conclusion

The usefulness and clinical adoption of this algorithm is predicated on a grasp of several key takeaway points and the flexibility to incorporate rapidly emerging new technologies into the continuum of care. Many patients with chronic lumbar discogenic pain are in their 30–50s underscoring the reality that their spine care will span many forthcoming decades. In summary, the starting point for the physical examination assessment is in the identification of patients with midline low back pain with no neurologic deficits that is increased with forward flexion and sitting who have a positive sustained hip flexion test that narrows the diagnostic aperture to anterior column involvement as the primary pain source. The identification of painful intervertebral discs is done via MRI and MRS which represents a potentially novel breakthrough in the precise and personalized diagnosis of lumbar discogenic pain.[100] When these advancements in quantitative imaging are combined with effective first line intradiscal interventions, the potential exists to optimize spine care by restoring and preserving native disc structure and mechanics. There has been a renewed appreciation of the importance of the intervertebral disc as a catalyst in spine degeneration and this has hastened the development of new diagnostic and treatment technologies aimed at averting spinal surgery. A concerted and collaborative effort will be necessary from all stakeholders to amass the supporting compendium of data to fulfill regulatory and reimbursement requirements so that effective treatments can be made available to patients.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

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