CLINICAL PRACTICE

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Nonspecific Low Back Pain

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This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the authors' clinical recommendations.

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A 37-year-old man reports he has had pain in his lower back for the past month. The pain is worse when he gets up in the morning, when it is associated with stiffness in the lower back. The patient has a history of episodes of low back pain that have typically occurred after vigorous sports activities. His medical history is otherwise unremarkable; he has not previously sought medical care for the pain. Physical examination shows that the patient's range of motion is limited on lumbar forward bending, and there is tenderness on palpation of the lower back. There are no neurologic deficits. How would you evaluate and treat this patient?

THE CLINICAL PROBLEM

Low back pain, typically defined as pain below the costal margin and above the inferior gluteal folds, with or without leg pain,[1] is worldwide the most prevalent and most disabling of the conditions that are considered to benefit from rehabilitation.[2] In a systematic review that included 165 studies from 54 countries, the mean point prevalence of low back pain in the general adult population was approximately 12%, with a higher prevalence among persons 40 years of age or older and among women; the lifetime prevalence was approximately 40%.[3]

Low back pain is classified as specific (pain and other symptoms that are caused by specific pathophysiological mechanisms of nonspinal or spinal origin) or nonspecific (back pain, with or without leg pain, without a clear nociceptive-specific cause).[4] Nonspinal causes of specific low back pain include hip conditions, diseases of the pelvic organs (e.g., prostatitis and endometriosis), and vascular (e.g., aortic aneurysm) or systemic disorders; spinal causes include herniated disk, spinal stenosis, fracture, tumor, infection, and axial spondyloarthritis. Lumbar disorders with radicular pain due to nerve-root involvement have a higher prevalence (5 to 10%) than other spinal causes; the two most frequent causes of such back pain are herniated disk and spinal stenosis.[5] The overall prevalence of the other spinal disorders is low among patients with acute low back pain. For example, among 1172 patients who presented to primary care clinicians in Australia with acute low back pain, only 11 (0.9%) were found to have serious spinal conditions (mostly fractures) during 1 year of follow-up.[6] The authors of a Dutch study that involved primary care patients reported axial spondyloarthritis in almost one quarter of adults 20 to 45 years of age who presented with chronic low back pain,[7] although these findings have not been replicated.

In contrast to low back pain caused by specific identifiable causes, nonspecific

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KEY CLINICAL POINTS

NONSPECIFIC LOW BACK PAIN

- Nonspecific low back pain is diagnosed on the basis of the exclusion of specific causes, usually by means of history taking and physical examination.
- Imaging is not routinely indicated in patients with nonspecific low back pain.
- Most patients with an acute episode of nonspecific low back pain will recover in a short period of time.
- Education and advice to remain active are recommended for patients with acute or chronic low back pain.
- For chronic low back pain, exercise therapy and behavioral therapy represent first-line options, with medications considered to be second-line options.

low back pain probably develops from the interaction of biologic, psychological, and social factors,[4] and it accounts for approximately 80 to 90% of all cases of low back pain.[1] Low back pain is usually classified according to pain duration as acute (<6 weeks), subacute (6 to 12 weeks), or chronic (>12 weeks).[8]

RISK FACTORS

Risk factors for an episode of nonspecific low back pain include physical risk factors (e.g., prolonged standing or walking and lifting heavy weights), an unhealthy lifestyle (e.g., obesity), psychological factors (e.g., depression and job dissatisfaction), and previous episodes of low back pain.[9] In a case–crossover study that included 999 patients with sudden-onset acute low back pain, performance of manual tasks (e.g., those involving heavy loads or awkward postures) and distraction during an activity or task were identified as triggers of a new episode of pain.[10]

NATURAL HISTORY AND PROGNOSIS

Low back pain is increasingly understood to be a long-lasting condition with a variable course rather than isolated, unrelated episodes. According to a systematic review of prospective inception cohort studies (33 cohorts and 11,166 patients) that were conducted mainly in primary care and involved a variety of approaches to treatment,[11] new-onset episodes of low back pain generally abated substantially within 6 weeks, and by 12 months the average reported pain levels were low (6 [95% confidence interval {CI}, 3 to 10] on a scale of 0 to 100, with lower scores indicating less pain). Research on the course of nonspecific low back pain has identified three main pain-trajectory subgroups that emerge in the year after presentation and apply to both acute and chronic low back pain: a recovery trajectory in which the patient's condition improves rapidly or gradually toward a state of no or little pain, an ongoing trajectory in which the patient has moderate or fluctuating pain, and a persistent trajectory in which the patient perceives constant and severe pain (Fig. 1).[12] The majority of patients with acute low back pain (approximately 70%) have a pain trajectory that is prognostic for recovery, whereas this trajectory is less frequent in patients with chronic low back pain (approximately 30%), a population in which many patients (40 to 50%) have an ongoing pain trajectory.

According to a review of observational studies, factors that are consistently associated with poor outcomes (i.e., ongoing pain, disability, or both) in patients with low back pain included the presence of widespread pain, poor physical functioning, somatization, high pain intensity, long pain duration, high levels of depression or anxiety (or both), previous episodes of low back pain, and poor coping strategies.[13] An observational study with 5 years of follow-up that involved 281 patients with nonspecific low back pain showed higher risks of a persistent pain trajectory among patients with high pain intensity (relative risk ratio per unit increase, 1.87; 95% CI, 1.33 to 2.64), low socioeconomic status (relative risk ratio, 5.39; 95% CI, 1.80 to 16.2), negative illness perceptions (negative cognitive and emotional responses to low back pain) (range of relative risk ratio per unit increase, 0.83 [95% CI, 0.71 to 0.97] to 1.19 [95% CI, 1.06 to 1.34]), and passive coping behaviors (helplessness and reliance on others with regard to coping with low back pain) (relative risk ratio per unit increase, 1.90; 95% CI, 1.17 to 3.08).[14]

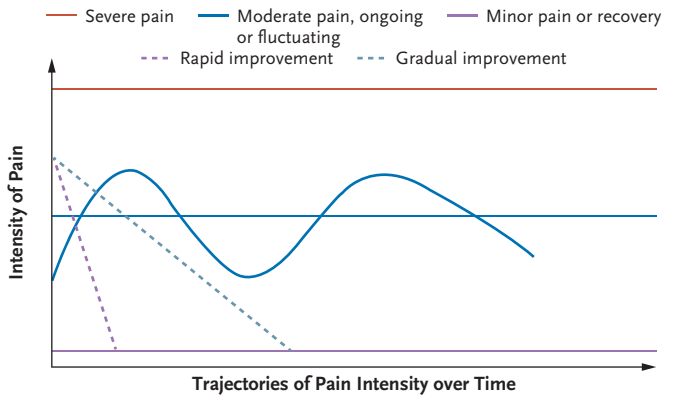

Figure 1. Simplified Principal Trajectories of Pain Intensity among Patients with Low Back Pain.

Adapted from Kongsted et al.[12]

STRATEGIES AND EVIDENCE

DIAGNOSIS AND EVALUATION

Diagnosis of nonspecific low back pain is made after specific disorders of spinal and nonspinal origin are ruled out. A detailed history taking and physical examination can point to spinal conditions or nonspinal conditions that may lead to specific intervention. The history should include attention to red flags (e.g., history of cancer or trauma, parenteral drug use, long-term glucocorticoid use, immunocompromise, fever, and unexplained weight loss), since their presence warrants consideration of an occult serious diagnosis (e.g., cancer, infection, or inflammatory disease) and close follow-up, although only some of these historical features have been shown to be useful predictors of such serious diagnoses. For example, in systematic reviews, a strong clinical suspicion for cancer[15] or a history of cancer[15,16] has been associated with an increased likelihood of a malignant condition, whereas other classic red flags (e.g., unexplained weight loss or fever) did not substantially affect the post-test probability of cancer.[15] Older age (>70 years), trauma, and the prolonged use of glucocorticoids have been associated with a high specificity for and considerable increased probability of spinal fracture, with the highest probability of fracture seen when multiple features are present.[16] History taking should also elicit whether pain is limited to the lower back or is more widespread; the latter may point to other conditions, such as fibromyalgia.

If a herniated disk is suspected, a positive ipsilateral straight-leg-raising test (in which pain results when the leg on the side of the back or leg pain is raised) is highly sensitive (in 92% of patients), and a positive contralateral straight-leg-raising test (in which pain is produced when the leg opposite the side of the back or leg pain is raised) is highly specific (in 90% of patients).[5] In the case of radiculopathy, a neurologic evaluation can rule out weakness, loss of sensation, or decreased reflexes; if any of these features are present, referral to a specialist may be indicated. Other maneuvers on physical examination have generally low diagnostic accuracy for the identification of other sources of low back pain (i.e., facet joints, sacroiliac joints, and disks).[17,18]

Screening tools can be used to estimate the risk that acute nonspecific low back pain will become chronic. The Predicting the Inception of Chronic Pain (PICKUP) tool is a validated prediction model that estimates the risk of chronic low back pain on the basis of five measures (i.e., disability compensation, presence of leg pain, pain intensity, depressive symptoms, and perceived risk of persistent pain) among patients who have an initial episode of low back pain.[19] A meta-analysis of studies that assessed other screening questionnaires showed that the Subgroups for Targeted Treatment (STarT) Back screening tool and the Örebro Musculoskeletal Pain Questionnaire, although not informative predictors of chronic pain, are predictive of subsequent disability; the latter was also highly predictive of work absenteeism.[20] The use of these screening tools in practice allows for the early identification of patients who are at risk for persistent low back pain–related disorders and may guide treatment.[21]

IMAGING

Routine imaging is not recommended in patients with nonspecific low back pain. Systematic reviews of observational studies have shown inconsistent findings with regard to the association between abnormal imaging findings and low back pain (Table S1 in the Supplementary Appendix, available with the full text of this article at NEJM.org).[22,23] In a study that included patients 65 years of age or older who presented

with acute low back pain without radiculopathy, the use of early imaging (e.g., radiography, magnetic resonance imaging, or computed tomography) was not associated with improved patient outcomes at 1 year.[24] Nevertheless, imaging may be performed when informative red flags are present, when there is a neurologic deficit, or when persistent low back pain with or without nerve-root involvement does not abate with conservative care.

TREATMENT

Numerous randomized, controlled trials and systematic reviews have assessed the effectiveness of interventions for nonspecific low back pain. Tables 1 and 2 summarize the pooled effects on acute and chronic low back pain, respectively, and the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) approach to the certainty of evidence obtained from systematic reviews of randomized trials that assessed the interventions that have most frequently been evaluated by practice guidelines.[27-45] Overall, first-line treatments are currently represented by nonpharmacologic interventions, which should be prioritized before pharmacologic treatment is prescribed.[46]

Acute Low Back Pain

Patient education and advice to remain active should represent routine care for patients with acute low back pain.[46] Education may address the benign, nonspecific nature and favorable course of low back pain, and patients should be encouraged to continue with regular activities. A meta-analysis of randomized trials showed (with moderate-certainty evidence) that individual patient education, as compared with usual care or other control, although not effective for pain, was effective in reassuring patients and reduced primary care visits due to low back pain at 1 year.[47] Meta-analyses of randomized trials support the use of a few sessions of spinal manipulative therapy or acupuncture for the reduction of pain, although the certainty of evidence for spinal manipulative therapy is moderate and that for acupuncture is low.[30,33] Heat and massage therapy are without risks and are reasonable to try, although the benefit of these therapies is supported only by limited data.[31,32] Exercise therapy that is prescribed or planned by a health professional has not been shown to be effective in patients with acute low back pain (Table 1),[29] but may be considered in patients at risk for poor recovery, given evidence from randomized trials of the effectiveness of exercise therapy in alleviating chronic low back pain[46] (Table 2) and in reducing the risk of episodes of low back pain.[48]

Among pharmacologic interventions, acetaminophen was not shown to be effective in a large clinical trial (Table 1),[34] whereas nonsteroidal antiinflammatory drugs (NSAIDs) have shown benefit.[35] However, caution is advised in the use of NSAIDs in older adults and in patients with coexisting conditions such as renal disease. Topical NSAIDs (e.g., topical diclofenac) have been shown to have fewer adverse events than oral NSAIDs, but their efficacy has not been rigorously studied in patients with low back pain. Results of a meta-analysis of the effects of muscle relaxants suggested that the use of nonbenzodiazepine antispasmodics begun within the first 2 weeks of the onset of pain had positive effects, but the analysis was based on very-low-certainty evidence.[36] These and other muscle relaxant agents had no significant effect on pain or disability during longer follow-up and were associated with a higher risk of adverse events.[36] Given the lack of data and the associated risk of addiction, the use of opioids should be minimized; weak opioids (e.g., tramadol) may be considered for use in carefully selected patients.[46]

Chronic Low Back Pain

In patients with chronic low back pain, education should play a key role, with supervised exercise and behavioral therapy as other first-line therapeutic options. Head-to-head randomized, controlled trials that compared these approaches have shown similar beneficial effects on pain in the short term (with low-to-moderate-certainty evidence),[39] although the effects of exercise and behavioral interventions over longer follow-up are unclear as compared with the effects of usual care or other conservative interventions.[38,39] A recent systematic review with network meta-analysis that included more than 200 randomized trials of 11 different types of exercise showed that most types of exercise had beneficial effects on alleviating pain and improving functioning, as compared with minimal treat-

Table 1. Effectiveness of Interventions on Pain Intensity and Physical Functioning Outcomes at Immediate-Term Follow-up (0–4 Weeks) in Patients with Acute Nonspecific Low Back Pain.*

Intervention vs. Control	Pain Intensity			Physical Functioning		
	No. of Studies	Pooled Effect (95% CI)†	Evidence Certainty	No. of Studies	Pooled Effect (95% CI)†	Evidence Certainty
Conservative intervention						
Advice to stay active vs. bed rest[27]	3	−0.4 (−4.0 to 3.2)	Low	2	−3.5 (−1.1 to −5.9)	Moderate
Individual patient education vs. no intervention[28]	3	NS	Moderate‡	3	NS	Very low‡
Individual patient education vs. non-educational interventions[28]	6	NS	Low‡	6	NS	Very low‡
Exercise therapy vs. no intervention or sham[29]	3	0.6 (−11.5 to 12.7)	Low‡	3	−2.8 (−15.3 to 9.7)	Low‡
Exercise therapy vs. other conservative interventions[29]	7	−0.3 (−0.7 to 0.1)	Low‡	6	−1.3 (−5.5 to 2.8)	Low‡
Spinal manipulative therapy vs. sham or other interventions[30]	NK	−9.8 (−17.0 to −2.5)	Moderate	NK	−2.9 (−6.6 to 1.0)	Moderate
Superficial heat vs. sham or non-heated wrap[31]	1	−32.2 (−38.7 to −25.7)	Very low‡	2	−8.8 (−12.8 to −1.2)	Very low‡
Massage therapy vs. inactive interventions[32]	1	−24.8 (−37.0 to −12.8)	Very low	1	−6.0 (−12.7 to 0.7)	Very low
Acupuncture vs. sham[33]	2	−9.4 (−17.0 to −1.8)	Low‡	3	NS	Moderate‡
Pharmacologic intervention						
Acetaminophen vs. placebo[34]	1	1.5 (−1.3 to 4.3)	High	1	−1.9 (−4.8 to 1.0)	High
NSAID vs. placebo[35]	4	−7.3 (−11.0 to −3.6)	Moderate	2	−8.4 (−12.1 to −4.8)	High
Muscle relaxant vs. placebo						
Nonbenzodiazepine antispasmodic agent[36]	16	−7.7 (−12.1 to −3.3)	Very low	7	−3.3 (−7.3 to 0.7)	Very low
Antispastic agent[36]	1	−1.6 (−15.3 to 12.1)	Low	1	2.0 (−15.6 to 19.6)	Low
Benzodiazepine[36]	1	2.0 (−9.8 to 13.8)	Moderate	1	0 (−13.2 to 13.2)	Low
Opioid vs. placebo[37]§	0	—	Very low‡	0	—	Very low‡

* If data for more than one follow-up between 0 and 4 weeks were present, the follow-up closer to 1 week was used. NK denotes number of studies not known, NS no significant between-group differences reported in the individual trials and no pooled effect, and NSAID nonsteroidal antiinflammatory drug.

† A negative value indicates a pooled effect in favor of the intervention. When the pooled effect was calculated as a standardized mean difference, it was converted to a scale of 0 to 100 by multiplying the standardized mean difference by 20 for pain intensity and by 12 for physical functioning. These values approximately represent the standard deviations of the most-used instruments (i.e., numerical rating scale for pain intensity and Oswestry Disability Index[25] for physical functioning) for these constructs. When the pooled effect was calculated on a scale of 0 to 24 (Roland–Morris Disability Questionnaire[26]), it was converted to a scale of 0 to 100 by multiplying the effect by 4.17.

‡ Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) scores were calculated by the authors, not reported by pooled-study investigators.

§ No trials of opioids for the treatment of acute nonspecific low back pain are available.

ment.[49] As compared with other exercises, Pilates therapy (which is focused on isometric contractions of the core muscles, attention to body movement, and improved posture) and McKenzie therapy (which involves repeated movement directional exercises, postural training, and education about patients' self-management of pain) resulted in reduced pain and improved functioning.

Behavioral therapies include respondent therapy (which involves relaxation techniques to reduce the physiologic response to pain), operant therapy (which is aimed at ceasing positive reinforcement of pain behaviors and promoting

Table 2. Effectiveness of Interventions on Pain Intensity and Physical Functioning Outcomes at Short-Term Follow-up (1–4 Months) in Patients with Chronic Nonspecific Low Back Pain.*

Intervention vs. Control	Pain Intensity			Physical Functioning		
	No. of Studies	Pooled Effect (95% CI)†	Evidence Certainty	No. of Studies	Pooled Effect (95% CI)†	Evidence Certainty
Conservative intervention						
Individual patient education vs. non-educational interventions[28]	1	NS	Moderate‡	2	NS	Moderate‡
Exercise therapy vs. no intervention or usual care[38]	26	–16.4 (–20.3 to –12.4)	Moderate	30	–7.4 (–9.2 to –5.6)	Moderate
Exercise therapy vs. other conservative interventions[38]	47	–8.6 (–13.1 to –4.1)	Moderate	44	–4.0 (–6.0 to –1.9)	Moderate
Behavioral therapy vs. waiting list[39]	5	–12.0 (–19.4 to –4.4)	Low	4	–4.4 (–10.4 to 1.6)	Low
Behavioral therapy vs. usual care[39]	2	–5.2 (–9.8 to –0.6)	Moderate	2	–2.4 (–4.9 to 0.2)	Moderate
Spinal manipulative therapy vs. sham[40]	8	–7.5 (–19.9 to 4.8)	Low	6	–8.8 (–16.2 to –1.3)	Low
Spinal manipulative therapy vs. guideline-recommended interventions[40]	17	–3.2 (–7.8 to 1.5)	Moderate	16	–3.0 (–4.9 to –1.1)	Moderate
Massage therapy vs. inactive interventions[32]	7	–15.0 (–18.0 to –12.0)	Low	6	–8.6 (–12.6 to –4.7)	Low
Massage therapy vs. active interventions[32]	12	–7.4 (–12.4 to –2.6)	Very low	6	–2.9 (–7.4 to 1.6)	Very low
Acupuncture vs. sham[41]	5	–10.0 (–17.2 to –2.8)	Low	3	–4.6 (–8.3 to –0.8)	Moderate
Acupuncture vs. no intervention[41]	3	–10.1 (–16.8 to –3.4)	Moderate	3	–4.7 (–8.6 to –0.7)	Moderate
Yoga vs. nonexercise intervention[42]	5	–4.5 (–7.0 to –2.1)	Moderate	7	–9.1 (–15.0 to –3.2)	Low
Yoga vs. exercise intervention[42]	1	–15.0 (–19.9 to –10.1)	Very low	2	–4.1 (–12.0 to 3.8)	Very low
Multidisciplinary rehabilitation vs. usual care[43]	9	–11.0 (–16.6 to –5.6)	Low	9	–4.9 (–7.4 to –3.8)	Moderate
Multidisciplinary rehabilitation vs. physical intervention[43]	12	–6.0 (–10.8 to –1.2)	Low	13	–4.7 (–8.2 to –1.2)	Low
Pharmacologic intervention						
Acetaminophen vs. placebo[34]§	0	—	Very low‡	0	—	Very low‡
NSAID vs. placebo[44]	6	–7.0 (–10.7 to –3.2)	Low	4	–3.5 (–5.4 to –1.7)	Low
Muscle relaxant vs. placebo						
Antispastic agent[36]	1	–5.4 (–13.7 to 2.9)	Very low	1	–3.2 (–8.3 to 1.8)	Very low
Other[36]	1	–19.9 (–31.5 to –8.3)	Moderate	1	–5.6 (–20.6 to 9.4)	Low
Antidepressant vs. placebo						
Serotonin–noradrenaline reuptake inhibitor[45]	4	–5.3 (–7.3 to –3.3)	Moderate	4	–3.5 (–5.2 to –1.9)	Moderate
Selective serotonin-reuptake inhibitor[45]	3	1.5 (–5.4 to 8.4)	Low	1	–2.2 (–8.1 to 3.7)	Low
Tricyclic antidepressant[45]	7	–10.0 (–21.5 to 1.6)	Very low	4	–12.9 (–26.5 to 0.6)	Very low
Opioid vs. placebo[37]¶	13	–9.0 (–11.7 to –6.2)	Very low	0	NA	NA

* If data for more than one follow-up between 1 and 4 months were present, the follow-up closer to 3 months was used.
† A negative value indicates a pooled effect in favor of the intervention. When the pooled effect was calculated as standardized mean difference, it was converted to a scale of 0 to 100 by multiplying the standardized mean difference by 20 for pain intensity and by 12 for physical functioning. These values represent approximately the standard deviations of the most-used instruments (i.e., numerical rating scale for pain intensity and Oswestry Disability Index[25] for physical functioning) for these constructs. When the pooled effect was calculated on a scale of 0 to 24 (Roland–Morris Disability Questionnaire[26]), it was converted to a scale of 0 to 100 by multiplying the effect by 4.17.
‡ GRADE scores were calculated by the authors, not reported by pooled-study investigators.
§ No trials of acetaminophen for the treatment of chronic nonspecific low back pain are available.
¶ In this study cited for opioid versus placebo, physical functioning was not a specified outcome, so pooled effect and evidence certainty for that outcome are not applicable (NA).

healthy behaviors, including exercise), and cognitive therapy (which focuses on identifying and modifying negative thoughts with regard to pain and disability); randomized, controlled trials comparing these therapies have shown they have similar effects on pain and functioning.[39] The choice of therapy from among conservative interventions should take into consideration the patient's preferences and other factors, such as out-of-pocket costs.

Other therapies for chronic low back pain include spinal manipulative therapy, massage therapy, yoga, and multidisciplinary rehabilitation.[32,42,43] A systematic review with moderate-certainty evidence showed no clinically relevant differences in effects on pain and functioning with spinal manipulative therapy as compared with recommended first-line options (Table 2).[40] Multidisciplinary interventions that combine physical and psychological components may be especially suited for patients with low levels of functioning and with psychosocial risk factors for poor outcomes, although data showing superior effectiveness for this patient group are lacking.[43]

There is at best moderate-certainty evidence to support various pharmacologic options for the management of chronic low back pain (Table 2). NSAIDs can be considered in patients at low risk, although the effects appear to be modest and are supported by low-certainty evidence.[44] Muscle relaxants and antidepressants (e.g., serotonin and norepinephrine reuptake inhibitors) may be used as adjuvant therapy in some patients, although they have had limited effectiveness (with evidence of moderate to very low certainty) (Table 2) and have potential risks.[36,45] The use of opioids should be limited to very carefully selected patients and only for short periods of time with appropriate monitoring.[46] Invasive therapies, such as epidural glucocorticoid injections and surgery, are rarely indicated for nonspecific low back pain.[46]

AREAS OF UNCERTAINTY

There is some controversy regarding the term "nonspecific" low back pain, since structures such as muscles, joints, or disks (or a combination of these) may be causing the pain but are not readily identified by means of history taking and physical examination. Some patients with nonspecific low back pain may have symptomatic spinal osteoarthritis; in contrast to osteoarthritis of the peripheral joints, there are no diagnostic criteria for spinal osteoarthritis, and data are needed to guide its diagnosis and management.

High-quality randomized trials are needed to assess the effects on pain and function of several interventions, including heat, massage therapy, NSAIDs (oral and topical), muscle relaxants, and opioids for acute low back pain and NSAIDs, muscle relaxants, and antidepressants for chronic low back pain. Also, data are needed to inform whether the effects of these or other interventions vary according to patient characteristics. A meta-analysis of individual patient data from 27 trials did not show clinically relevant modifiers of the effect of exercise on chronic low back pain.[50] In a trial involving patients with low back pain that compared usual care with care stratified according to prognosis (estimated with the use of the STarT Back tool; patients at low risk received minimal intervention, those at medium risk received physical therapy, and those at high risk received "psychologically informed" physical therapy), patients in the stratified-care groups had greater reduction of disability and low back pain–related health care costs than those who received usual care.[21] However, these positive findings were not confirmed by subsequent trials conducted in primary care settings in the United States.[51,52]

GUIDELINES

A previously published overview summarized the recommendations of 15 clinical practice guidelines for the management of nonspecific low back pain in primary care.[53] More recent guidelines (e.g., those of the American College of Physicians) have moved away from pharmacotherapy (owing to limited efficacy and risk of adverse effects) in favor of initial nonpharmacologic care for both acute and chronic low back pain.[54] The recommendations presented here are generally consistent with those guidelines.

CONCLUSIONS AND RECOMMENDATIONS

The patient described in the vignette is experiencing an acute episode of recurrent low back

pain. In the absence of worrisome findings on the history and physical examination, imaging would not be recommended. The PICK-UP tool or the Örebro Musculoskeletal Pain Questionnaire may be used to evaluate the patient for risk of the episode becoming chronic. The patient should be reassured of the very high likelihood that there is no serious condition causing his low back pain and of the anticipated favorable prognosis of the current episode. He should be encouraged to continue his regular activities, even if he has some pain when engaging in them. We would suggest considering the use of a heating pad (although this recommendation is based on limited data[31]); short-term use of NSAIDs may be helpful in the absence of contraindications. If the low back pain does not abate within 2 months after the first visit, we would recommend referral to a specialist for supervised exercise or behavioral therapy. We would consider referral for exercise therapy earlier if there is concern about a risk of the condition becoming chronic, given the evidence of the benefit of exercise in alleviating chronic low back pain and minimizing the risk of recurrent low back pain. We would engage in shared decision making with the patient, with treatment decisions guided by his preferences and priorities.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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