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Treating co-occurring chronic low back pain & generalized anxiety disorder

Abstract: *The complex, bidirectional correlation between chronic low back pain (CLBP) and generalized anxiety disorder (GAD), common ailments in primary care, can increase the risk of inadequate treatment. This article will review the relationship between CLBP and GAD and provide optimal management strategies for NPs caring for individuals with this dyad.*

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Chronic pain is a complex phenomenon involving biological, psychological, social, and cultural aspects. Assessing the various interrelated dimensions of chronic pain continues to present a clinical challenge. Anxiety disorders are a common comorbidity in patients with chronic pain and can greatly influence pain perception and patient function; chronic pain can also exacerbate anxiety symptoms. Most current guidelines for chronic low back pain (CLBP) emphasize the importance of assessing for symptoms of anxiety disorders. However, effective management of this co-occurrence remains unclear. This article will review the relationship between CLBP and generalized anxiety disorder (GAD) as well as provide optimal management strategies for individuals with co-occurring GAD and CLBP.

■ Significance of the issue

Individuals with either CLBP or GAD tend to have higher rates of impaired function, disability, and other comorbid mental health disorders.^{1,2} Twenty percent of individuals with CLBP have co-occurring GAD.³ The complex, bidirectional relationship between chronic pain and GAD is clearly documented;⁴⁻⁷ however, there is minimal literature that specifically examines assessment and treatment of this dyad.

The impact of anxiety on pain may be over- or underestimated when GAD coexists with CLBP. In some cases, clinicians mistakenly attribute pain symptoms to the presence of GAD when pain is actually caused by serious underlying pathology.⁸ Conversely, clinicians may overlook the diagnosis of GAD in pursuit of organic evidence. Failure to understand the intricate somatic and psychosocial relationship between CLBP and GAD can lead to misdiagnosis, unnecessary diagnostic testing, inadequate treatment, and further disability.

A comprehensive understanding of this relationship is essential for NPs to make appropriate clinical judgments. This article will suggest appropriate assessment and management approaches for CLBP with comorbid GAD; consider the patient-NP relationship role in diagnosis and treatment; and present indications for referral to a pain specialist or psychiatrist.

■ Chronic low back pain

Pain is defined as an “unpleasant sensory and emotional experience associated with actual and potential tissue damage” and becomes chronic when pain persists in spite of usual treatment and beyond the expected course for acute illness or injury (generally 3 to 6 months).⁹⁻¹¹ Chronic pain can be recurrent or

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ongoing.^{10,11} Although acute pain tends to be more nociceptive in nature, chronic pain is often both nociceptive and neuropathic in origin and is usually the result of myriad factors, including emotions, behaviors, pain beliefs, genetics, and sociodemographic factors.¹²⁻¹⁴

Low back pain (LBP) describes tension or pain over the lumbar region of the back.^{15,16} Potential etiologies of LBP are numerous and can include psychological distress, tumor, intra-abdominal disease, infection, ankylosing spondylitis, and spinal stenosis.¹⁵⁻¹⁷ Degenerative changes in facet joints, vertebral disks, and connective tissues may contribute to the development of CLBP.¹⁵ However, most cases do not have a clear etiology and are referred to as nonspecific due to the lack of association between symptoms, degenerative changes, and imaging.¹⁵

CLBP may be perpetuated by the fear-avoidance model, in which a patient's perceived fear of pain is greater than the pain itself.¹⁸ Movement is often the stimulus of pain in these cases, so patients restrict movement and develop further disability and pain.¹⁸

■ Generalized anxiety disorder

Similar to the fear-avoidance model for CLBP, patients with GAD often perceive an innocuous situation as threatening and will anticipate the worst possible outcome.¹⁹ Maladaptive coping techniques and threat avoidance behaviors, which can lead to cognitive, social, and physical disabilities, are often employed.²⁰ GAD is characterized by excessive and persistent worry that extends beyond 6 months and often has a fluctuating course, which may be further complicated by other major depressive and mental health disorders.²¹⁻²³ Symptoms of GAD may manifest somatically as muscle tension, restlessness, fatigue, and pain (including back, shoulder, and headache).^{21,23} Undiagnosed or untreated, GAD can contribute to lower productivity, higher morbidity and mortality, and higher prevalence of alcohol and drug abuse.¹⁹

■ CLBP and GAD

The complex relationship between chronic pain and GAD is clearly demonstrated and thought to be mutually exacerbating, yet pathophysiologic mechanisms are not clearly understood.^{4-7,20,24,25} It is hypothesized that both conditions share common elements of anxiety sensitivity, whereby individuals may interpret anxiety sensations as catastrophic and respond with heightened hypervigilance to a threat and a lowered alarm threshold.⁷

Individuals with co-occurring CLBP and GAD tend to experience greater pain intensity due to physiologic changes within the neurologic system, negative emotions, hypervigilance to pain, and interpretation of pain as a dangerous stimulus.¹³ Anxiety can promote the release of proinflammatory mediators, which can damage tissues and heighten neural

processing.¹³ When strong emotions are the result of pain or a perceived threat, the body heightens neural processing and primes neural circuits, increasing the likelihood that bodily sensations will continue to be interpreted as painful.¹³ Over time, signals that normally represent anxiety are misinterpreted as pain, leading to the development or exacerbation of CLBP.²⁰

Although many psychological factors affect the pain experience, anxiety has independent and additive adverse reactions on individuals' functional status and response to pain treatment.² Patients with undiagnosed GAD are more likely to progress from subacute to chronic pain.²⁰ Failure to recognize GAD in patients with concurrent CLBP may lead to inadequate treatment and further disability.

■ Assessment

GAD and CLBP share common symptoms and cannot be fully disentangled. GAD and CLBP should be assessed separately in order to discern the relative contribution of each disorder to the overall clinical picture.

CLBP assessment. The critical role of the initial assessment of back pain is to identify significant pathology, such as: cauda equina syndrome, inflammatory arthritis, malignancy, trauma, infection, kidney disease, disease of the pelvic organs, dissecting aorta, or gastrointestinal disease.^{15,26-28} The focus here will be on nonspecific CLBP where significant pathology has been ruled out.

When a patient reports pain, it is often given less attention and credibility in cases when medical evidence is ambiguous.²⁹ A patient's pain experience may be discounted if the NP believes the cause of the pain is either psychological or physiologic or that the reported pain intensity seems out of proportion to the actual tissue damage.²⁹ NPs may feel uncertain when dealing with patients who report high pain intensity in the absence of obvious pathology and may minimize pain reports to ease their own uncertainty.⁸ When pain reports are deemed less credible, other patient complaints may also be minimized, and NPs are more likely to miss a critical diagnosis. However, pain remains a subjective experience, so self-report using common tools remains essential. Visual analogue scales offer a rapid assessment of pain intensity, while pain diaries and longer instruments (such as the Brief Pain Inventory or the Short Form McGill Pain Questionnaire) may be an option for patients to complete at home and bring to their next appointment.

Anxiety assessment. Screening for anxiety in patients with CLBP is advised given the high frequency of comorbidity. Numerous scales available to screen for and assess anxiety include the Hospital Anxiety and Depression Scale (HADS) and Generalized Anxiety Disorder-7-item (GAD-7) scale.³⁰ Alternatively, clinicians may use the simple question, "Have

you been feeling worried or anxious?" However, the effectiveness of this question is dependent on the patient's level of insight, as the majority of patients present with nonspecific somatic complaints rather than complaints of worry or anxiety.³¹ Geriatric-specific providers and tools for anxiety assessment, such as the Geriatric Anxiety Inventory and Geriatric Anxiety Scale, should be utilized.³² If appropriate adjustments are made, tools not specifically designed to assess anxiety in older adults can also be considered. For example, the GAD-7 is appropriate for older adults if the cut point is lowered from 10 to 5.³³

Medication adverse reactions or other pathophysiologic processes can manifest similarly to GAD and must be ruled out. Patients with GAD, CLBP, or a combination of the two are at increased risk for suicide, depression, co-occurring anxiety disorders, and insomnia, as well as alcohol and substance use; therefore, the NP must also consider these variables.^{30,34-37}

■ Approaches to management

Data for evidence-based treatment of co-occurring CLBP and GAD are limited, and treatment response is often diminished when compared with singular presentations.²⁴ This suggests that current evidence-based guidelines for the individual disorders are suboptimal in cases of comorbidity. It is presumed that appropriate GAD treatment will attenuate CLBP symptoms, and vice versa, based on the proposed interrelationship of mechanisms.²⁴ Treatment options are limited to those found effective for the individual disorders in lieu of effective treatment evidence regarding comorbid GAD and CLBP.

Nonpharmacologic. Decisions regarding nonpharmacologic therapies are dependent upon availability, cost, and patient preference. Nonpharmacologic therapies for CLBP, such as physiotherapy, physical activity, cognitive behavioral therapy (CBT), spinal manipulation, massage, and acupuncture have moderate evidence for effectiveness with minimal evidence to support one modality over the other.^{26,28} Walking, running, yoga, and tai chi reduce pain and anxiety symptoms.^{38,39} Despite this, patients with both GAD and CLBP may limit physical activity due to fear of worsening their pain and causing reinjury.²⁰

Because CBT is effective for GAD and CLBP individuals, there may be a synergistic benefit to both disorders by addressing aspects of catastrophic thinking and avoidance behaviors that limit participation in physical treatment modalities as well as addressing anxiety and pain-coping strategies.^{5,40-43} Loving-kindness meditation may also benefit patients with comorbid GAD and CLBP.⁴⁴ Research specific to GAD is unavailable, but preliminary research shows support for improved CLBP and psychological distress, including anxiety.⁴⁴

Pharmacologic treatment. Treatment of both CLBP and GAD often requires pharmacologic management. Nociceptive pain may play an important role in the symptoms of CLBP but is often either ignored or given too much attention, leading to poor pain control or opioid overprescription.^{2,45} Although acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) may be effective, adverse gastrointestinal, renal, hepatocellular, and cardiac effects and interactions limit long-term utility.^{26,28}

Opioids may be indicated for patients who continue to experience functional impairment despite an adequate trial of GAD treatment and optimized pharmacologic and non-pharmacologic methods.³⁹ However, inconsistent evidence supports the short-term effectiveness of opioids and evidence does not support long-term (beyond 16 weeks) use of opioids for CLBP.^{46,47}

Despite this inconsistent evidence, patients with concomitant pain and anxiety disorders are more frequently prescribed opioids than those without anxiety disorders.^{2,45} This may be due to or result in chemical coping, where individuals interpret emotional pain or distress as physical pain and attempt to treat both their emotional and physical pain with opioids.⁴⁸ If the underlying issues are not addressed, this can ultimately lead to reduced function, substance misuse, or substance use disorder (SUD). At the same time, opioid use

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in patients with chronic pain and GAD reduces response to anxiolytics.⁴⁹ If the NP decides to initiate a trial of opioids, there should be a clear treatment contract, with specific functional goals, a plan for reassessment, and the clear expectation that opioids will be discontinued if no functional improvements are seen within 3 to 6 months.^{42,50} The risks of substance misuse and SUD must be considered and monitored with appropriate interventions should misuse develop.

Antidepressant and antiepileptic medications can affect the psychiatric and somatic symptoms of GAD, the pain symptoms of CLBP, and the overall functioning of individuals with either condition.⁵¹⁻⁵⁶ FDA approved, first-line treatment for GAD includes selective serotonin reuptake inhibitors (SSRIs), including escitalopram oxalate (Lexapro), paroxetine hydrochloride (Paxil), and paroxetine mesylate (Pexeva), or serotonin and norepinephrine reuptake inhibitors (SNRIs), including duloxetine hydrochloride (Cymbalta).⁵⁴ Off-label medications for GAD include quetiapine

and bupropion.⁵⁴ Given that SNRIs and tricyclic antidepressants (TCAs) provide both analgesic and anxiolytic effects, these may be an appropriate first-line choice in individuals with comorbid CLBP and GAD.⁵¹⁻⁵⁶

In some cases, however, it may be more beneficial to use multiple agents to treat pain and anxiety rather than a single medication. For instance, while TCAs (used FDA off-label for CLBP and GAD) show some clinical efficacy for CLBP and GAD, they are also associated with multiple adverse reactions, have a risk of lethal overdose, and are inconsistent in analgesic effects for CLBP.^{52,53} As a result, it may be preferable to prescribe two alternative, more effective medications rather than a single TCA.

Patients receiving more than one medication to treat pain and anxiety must be monitored for potential drug interactions. For instance, the combination of NSAIDs and SSRIs has an additive risk for ulcers and gastrointestinal bleeding unless a proton-pump inhibitor is used.⁵⁷ SSRIs are not currently recommended for CLBP, apart from treatment for mood; however, since systematic reviews tend to broadly categorize antidepressant agents, Williamson and colleagues suggest that analgesic effects of SSRIs may be underestimated in the treatment of CLBP.⁵⁶

Short-term benzodiazepines are recommended as second-line therapy for the acute phase of GAD while awaiting response to SSRIs, SNRIs, or in severe GAD.^{39,53} Benzodiazepines may be less appropriate in this population, as patients with CLBP and GAD are at increased risk for chemical coping, withdrawal, addiction, and sedation, so alternatives may be required.⁵⁵ For instance, pregabalin (an off-label use for GAD) has a short onset of action and may be utilized so that benzodiazepines are not required while awaiting onset of an SSRI or SNRI.⁵⁸ Benzodiazepines are contraindicated in patients with a history of benzodiazepine or psychoactive substance abuse. NPs must closely monitor patients and avoid long-term treatment if benzodiazepines are prescribed.⁵³ Further research is required to determine the most efficacious pharmacologic agent for comorbid CLBP and GAD.

Response to therapy. Treatment success for patients with GAD and CLBP is defined as improvement in patients' overall functioning and symptoms of pain and anxiety.^{17,19} Patients with either CLBP or GAD rarely experience total remission of pain or anxiety symptoms, instead experiencing periods of remissions and exacerbations.^{15,19,22} Because expected response to treatments are often unclear, it can be challenging to monitor treatment success. In such cases, therapy should be directed by patient goals, tolerance of therapy, and expected responses to interventions.

Referral. Diagnosis and treatment of CLBP and GAD is within the NP scope of practice, but psychiatric referral or consult may be indicated when diagnosis is uncertain; anxi-

ety symptoms have recurred; comorbid substance use, mood, or anxiety disorders are present; and if response to treatment is poor.^{22,37} Psychiatry services may not be readily accessible; therefore, NPs should be aware of treatment options for co-occurring mental health disorders. With appropriate training, NPs may utilize CBT methods in combination with other treatment modalities for patient management. Referral to a mental health specialist should be considered if a trial of patient self-management with an NP is unsuccessful.

Indications for a referral to a pain clinic are less clear and will depend on NP experience, available resources, and regional regulatory factors that impact NP prescribing. Patients whose GAD and CLBP symptoms are poorly controlled with conservative management should be referred to a pain clinic to receive assessment and treatment by mental health practitioners, nurses, and pain specialists. Because pain clinics may have long waiting lists and are often unavailable outside major centers, primary care NPs may be responsible for managing the complex need of patients with concurrent CLBP and GAD. Numerous community programs are available to provide nonpharmacologic therapies for patients with CLBP and GAD.

■ Implications for practice

While knowledge of available treatments is essential to effectively manage GAD and CLBP, a mutual, trusting, and reassuring patient-provider relationship is equally important. Patients with either CLBP or GAD often have a history of negative experiences within the medical system and discordant goals of care.^{59,60} NPs can help dispel misconceptions, rebuild patient trust, and provide nonjudgmental support.

Patients with CLBP and GAD may have detrimental beliefs regarding their illness. Organic evidence of disease is often absent in patients with nonspecific CLBP, but patients may search for an organic cause; have unreasonable expectations for symptom resolution; have unwarranted fears that physical activity will cause further damage and exacerbate pain; or believe that by endorsing symptoms of anxiety their complaints of pain will be discredited by the NP.^{18,61} As a result, they may have poor adherence to treatments, perceive stigmatization, and lose trust in medical practitioners.⁵⁹⁻⁶¹ Individuals with nonspecific CLBP often perceived that their reports of pain were not believed but attributed to drug-seeking; participants felt their pain experience was regarded as purely psychological and, therefore, less relevant.⁶⁰

NPs may inadvertently foster negative illness beliefs and contribute to stigmatization. Since patients with co-occurring CLBP and GAD are more likely to have an ambiguous diagnosis and more severe, chronic pain, care providers are more likely to discount these patients' reports of high intensity pain and judge the validity of patients' pain reports

on the basis of psychological characteristics and social demographics.^{8,59} This increases the risk of inaccurate diagnosis and/or dissolution of the patient-provider relationship. Conversely, NPs may order unnecessary diagnostic testing for patients with nonspecific CLBP in an attempt to identify an organic cause, potentially perpetuating patients' false beliefs that pain results solely from tissue damage.⁵⁹ When NPs identify their own underlying assumptions, they can become more aware of how their actions affect patient care.

■ Moving forward

The complex, bidirectional correlation between CLBP and GAD can increase patients' pain perceptions, level of disability, and risk for adverse outcomes and inadequate treatment. Pain and anxiety intensity may be substantial but not necessarily reflective of the severity or presence of organic pathology. Available evidence for optimal management of comorbid CLBP and GAD is limited. Individually effective treatments may have synergistic effects and should be employed in the context of a positive patient-provider partnership to achieve optimal pain and anxiety reduction and maximum function. NPs should consider conducting research on interventions to improve functional outcomes for patients with CLBP and GAD. 

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