



Dr. Ramezani describes clinical tools to aid assessment

HEAD PAIN AND PSYCHIATRIC ILLNESS: Applying the biopsychosocial model to care

Integrating neurologic, cognitive, and psychological aspects of this condition enhances clinical assessment

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More than 45% of people worldwide suffer from headache at some point in their life.¹ Head pain can lead to disability and functional decline, yet headache disorders often are underdiagnosed and poorly assessed. For example, 60% of migraine and tension-type headaches go undiagnosed and 50% of persons suffering from migraine have severe functional disability or require bed rest.²⁻⁴

Because head pain can be associated with secondary medical and psychiatric conditions, diagnosis can be challenging. This article reviews the medical and psychological aspects of major headaches and assists with clinical assessment. We present clinical interviewing tools and a diagram to enhance focused, efficient assessment and inform treatment plans.

Classification of headache

Headache is a common complaint, yet it is often underdiagnosed and ineffectively treated. The World Health Organization estimates that, globally, 50% of people with headache self-treat their pain.⁵ The International Headache Society classifies headache as primary or secondary; approximately 90% of complaints are from primary headache.⁶

Assessment and diagnosis of headache can be complex because of overlapping, subjective symptoms. It is important to have a general understanding of primary and secondary causes of headache so that interrelated symptoms do not obscure the most accurate diagnosis and

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Head pain

Clinical Point

Although most headaches are benign, ruling out secondary causes helps gauge the likelihood of developing severe sequelae

Table 1

Common causes of primary and secondary headache

| |
|--------------------------|
| Primary |
| Cluster |
| Migraine |
| Tension-type |
| Secondary |
| Vascular |
| Stroke |
| Temporal arteritis |
| Venous thrombosis |
| Infection |
| Meningitis |
| Sinusitis |
| Intracranial pathology |
| Aneurysm |
| Cerebrospinal fluid leak |
| Tumor |
| Psychiatric |
| Other |
| Drug-induced |
| Metabolic |
| Neuralgia |
| Trauma |
| Withdrawal |

effective treatment course. Although most headache complaints are benign, ruling out secondary causes helps gauge the likelihood of developing severe sequelae from underlying pathology.

By definition, **primary headaches** are idiopathic and commonly include migraine, tension-type, cluster, and hemicrania continua headache. **Secondary headaches** have an underlying pathology, which could improve by targeting the disorder. Common secondary causes of headache include:

- trauma
- vascular abnormalities
- structural abnormalities
- chemical (including medications)
- inflammation or infection
- metabolic conditions
- diseases of the neck and pericranial and intracranial structures
- psychiatric conditions.

Table 1 illustrates common causes of head pain. More definitive criteria for symptoms and diagnosis can be found in the International Classification of Headache Disorders.⁷

Primary headache

Tension-type is the most common primary headache, accounting for more than one-half of all headaches.⁷ Patients usually describe a tight pain in a bilateral band-like distribution, which could be caused by sustained neck muscle contraction. Pain usually builds in intensity and can last 30 minutes to several days. There is a well-established association between emotional stress or depression and the development of tension-type headaches.⁸

Migraine typically causes pulsating pain in a localized area of the head that lasts as long as 72 hours and can be associated with nausea, vomiting, photophobia, phonophobia, and aura. Patients report varying precipitating factors but commonly cite certain foods, menstruation, and sleep deprivation. Although rare, migraine with aura has been linked to ischemic stroke; most cases have been reported in female smokers and oral contraceptive users age <45.⁹

Because migraines can be debilitating, some patients—typically those with ≥ 4 attacks a month—opt for prophylactic medication. Effective prophylactics include amitriptyline, propranolol, divalproex sodium, and topiramate, which should be monitored closely and given a trial for several months before switching to another drug. Commonly used abortive treatments include triptans and anti-emetics such as metoclopramide.

Meperidine and ketorolac are popular second-line agents for migraine. Botulinum toxin A also has been used in severe cases to reduce the number of headache days in chronic migraine patients.⁶

Cluster headache is rare, but typically exhibits repeated burning and intense unilateral periorbital or retro-orbital pain that lasts 15 minutes to 3 hours over several weeks. Men are predominantly affected. Cluster headaches typically improve with oxygen treatment.

Biopsychosocial model of head pain

The biomedical model has helped iden-



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tify pathophysiological pain mechanisms and pharmacotherapeutic agents for headache. However, during assessment, limiting one's attention to the linear relationship between pathology, mechanism of action, and pain oversimplifies common questions clinicians face when assessing chronic head pain.

Advancements in the last 3 decades have expanded the conceptualization of head pain to integrate sociocultural, environmental, behavioral, affective, cognitive, and biological variables—otherwise known as the biopsychosocial model.^{10,11} The biopsychosocial model is a multidimensional theory that helps answer difficult clinical assessment questions and complex patient presentations (Table 2).¹⁰⁻¹³ Many unusual responses to pain treatment, questionable validity of pain behavior, and disproportionate pain perception and functional decline are explained by non-pathophysiological and non-biomechanical models.

Psychiatric comorbidity and head pain

Psychiatric conditions are highly prevalent among persons with primary headache. Verri et al¹⁴ found that 90% of chronic daily headache patients had ≥ 1 psychiatric condition; depression and anxiety were most common. Of concern, 1 study found that headache is associated with increased frequency of suicidal ideation among patients with chronic pain.¹⁵ It is critical for clinicians to screen for psychiatric comorbidities in patients with chronic headache. Conversely, clinicians might want to screen for headache in their patients with psychiatric illness.

Migraine. Mood disorders are common among patients who suffer from migraine. The rate of depression is 2 to 4 times higher in those with migraine compared with healthy controls.^{16,17} In a large-scale study, patients with migraine had a 1.9-fold higher risk (compared with controls) of having a comorbid depressive episode; a 2-fold higher risk of manic episodes; and a 3-fold higher risk of both

Table 2
Complicated patient presentations that can be clarified using the biopsychosocial model

| |
|--|
| In the absence of pathology, pain continues |
| In the presence of pathology, there is little pain |
| Response to pain intervention or normally effective medication is lacking |
| Response to effective pain intervention is rapid or short-lived |
| In the presence of pain, there is disproportionate functional decline |
| When pain is reduced or controlled, disproportionate functional decline remains |
| In the presence of severe pain, there is little or no distress despite the strong relationship between pain and emotions |
| Source: Adapted from references 10-13 |

mania and depression.¹⁸ In a study of 62 inpatients, Fasmer¹⁹ reported that 46% of patients with unipolar depression and 44% of patients with bipolar disorder experienced migraine (77% of the bipolar disorder patients with migraine had bipolar II disorder). Patients with migraine are at increased risk of suicide attempts (odds ratio 4.3; 95% CI, 1.2-15.7).²⁰

Tension-type headache. The relationship between psychiatric comorbidity in tension-type headache is well established. In contrast to what is seen with migraines, Puca et al²¹ found a higher prevalence of anxiety disorders (52.5%) than depressive disorders (36.4%) in patients with tension-type headache. Generalized anxiety disorder was one of the most prevalent anxiety conditions (83.3%), and dysthymia was the most prevalent mood disorder (45.6%). In the same study, 21.7% of patients were found to have a comorbid somatoform disorder.²¹

Emotional and cognitive factors can occur in patients with tension-type headache and a comorbid psychiatric condition. For example, difficulty identifying or recognizing emotions—commonly referred to as alexithymia—has been linked to tension-type headache.²² Additionally, maladaptive cognitive appraisal of stress is more

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A study found that headache is associated with increased frequency of suicidal ideation among patients with chronic pain



Head pain

Clinical Point

Head pain can be more intense when combined with untreated anxiety, depression, psychosis, or a substance use disorder

Table 3

AMPS questionnaire

| | |
|--------------------------------|--|
| Anxiety | Does nervousness or anxiety cause a problem for you in your daily life? |
| Mood | Depression Have you felt depressed, sad, or hopeless lately? Have you engaged in pleasurable activities lately? Have you had thoughts of hurting or killing yourself? |
| | Mania or hypomania Have you ever felt overly happy to the point where family or friends are worried about you? Have you ever had too much energy, to the point where you did not need to sleep? |
| Psychosis | Do you feel that people are trying to hurt, spy on, or follow you? Have you seen visions or heard voices that other people cannot see or hear? |
| Substance use disorders | How much alcohol do you drink each day? |
| | Have you recently used cocaine, methamphetamine, marijuana, heroin, phencyclidine, LSD, ecstasy, or other drugs of abuse? |

common among patients with tension-type headache when compared with those without headaches.²³ Being mindful of and recognizing these co-occurring emotional and cognitive factors will help clinicians construct a more accurate assessment and effective behavioral treatment plan.

Clinical assessment with a useful mnemonic

Clinical assessment of psychiatric illness is essential when evaluating chronic pain patients. Using the acronym **AMPS** (Anxiety, Mood, Psychosis, and Substance use disorders) (Table 3) is an efficient way for the clinician to ask pertinent questions regarding common psychiatric conditions that could have a direct effect on chronic pain.²⁴ Head pain can be more intense when combined with untreated anxiety, depression, psychosis, or a substance use disorder. Untreated anxiety, for example, can amplify sympathetic response to pain and complicate treatment.

Investigating head pain patients for an underlying mood disorder is essential to providing successful treatment. Consider:

- starting psychotherapy modalities that address both pain and psychiatric illness, such as cognitive-behavioral therapy (CBT)
- reframing unhelpful pain beliefs
- managing activity-rest levels

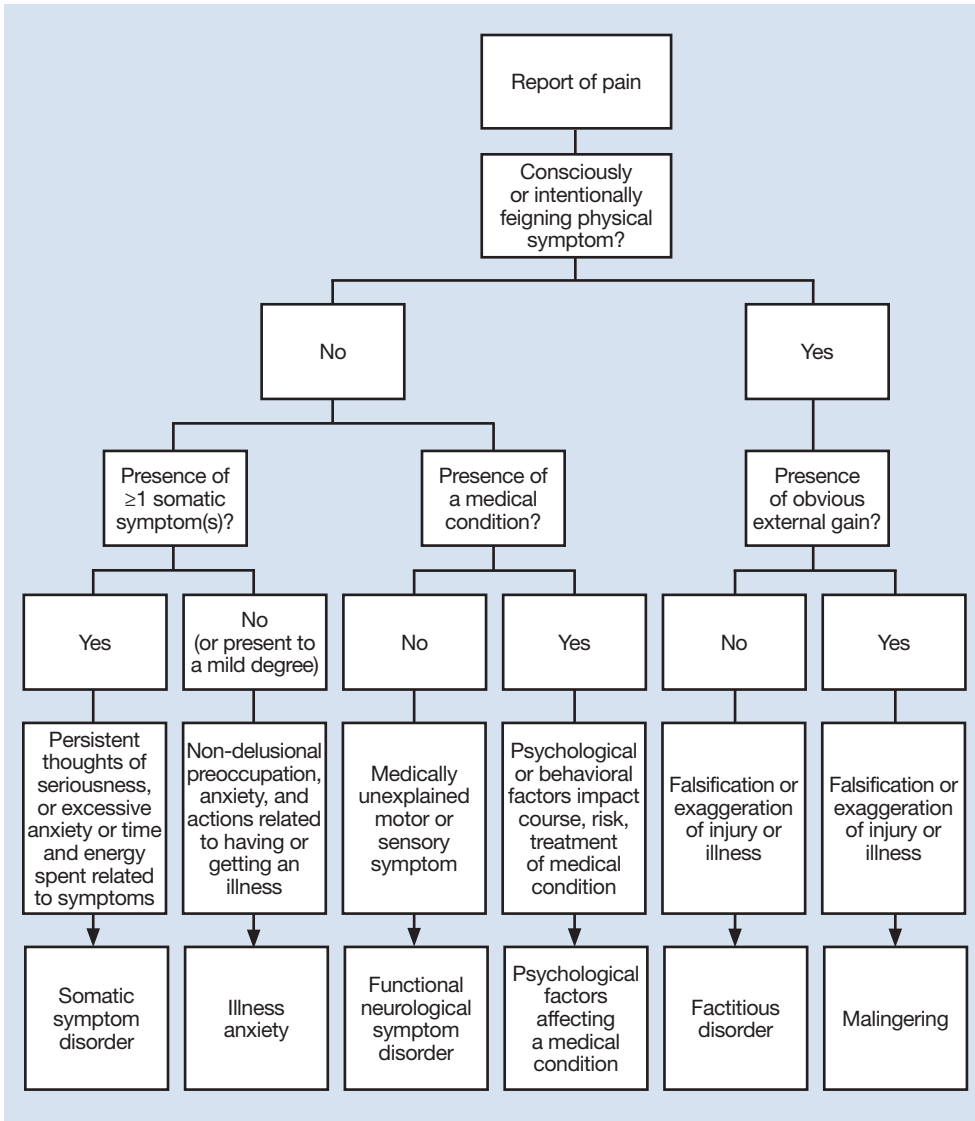
- biofeedback
- supportive group therapy
- reducing family members' reinforcement of the patient's pain behavior or sick role.²⁵

Assessing for somatic symptom disorders

In addition to using the AMPS approach for psychiatric assessment, clinicians should evaluate for somatization, which can present as head pain. Somatic symptom disorders (SSD) are a class of conditions that are impacted by affective, cognitive, and reinforcing factors that might or might not be consciously or intentionally produced. Patients with an SSD have somatic symptoms that are distressing or cause significant disruption of daily life because of excessive thoughts, feelings, or behaviors related to the somatic symptoms, for ≥ 6 months. The Figure outlines SSD, related conditions, and their respective prominent symptoms to assist in the differential diagnosis.²⁶

Note that some headache conditions present with severe distress because of their abrupt onset and severity of symptoms (eg, cluster headaches). Therefore, the expectation and likelihood of psychological disturbance should be factored into a diagnosis of SSD and related conditions as seen in the Figure.

Is the patient's head pain related to a somatic symptom condition?



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Specific questions about cultural beliefs and rituals as they relate to exacerbations of head pain are of value

Secondary factors of unusual pain behavior or treatment response. The role of thoughts, affect, and behaviors is clinically meaningful in understanding SSD and similar conditions. Specific questions about cultural beliefs and rituals as they relate to exacerbations of head pain are of value. *Table 4*^{13,27} (page 24) lists behavioral, cognitive, and affective dimensions of head pain using the biopsychosocial model, and further clarifies common questions that arise with unusual pain response and complex patient presentations, which were outlined in the beginning of the article.

Because depression and anxiety can be comorbid with head pain, it is important to recognize psychological factors that contribute to pain perception. Indifference or denial of emotional stress as a result of severe pain and disability can imply a somatization process, which could suggest emotional disconnection or dissociation from somatic functioning.²⁸ This finding can be a component of alexithymia, in which a person is disconnected from emotions and how emotions impact the body. Therefore, recognizing alexithymia assists in identifying psychological factors when patients deny mood symptoms, particularly in tension-type headache.



Head pain

Clinical Point

Indifference or denial of emotional stress as a result of severe pain and disability can imply a somatization process

Table 4

Biopsychosocial theories of pain in the clinical assessment

| Biopsychosocial domain | Evidence-based theory or principle | Specific dimension to assess |
|--|--|--|
| Behavioral-learning | Operant conditioning | <i>Positive reinforcement:</i> Acute pain behavior is reinforced by attention received from a significant other or provider, which develops into chronic pain behavior, eg, solicitous spouse elicits more pain behavior (eg, guarding, bracing, moaning) |
| | | <i>Lack of positive reinforcement:</i> Normal level of activity and self-pacing are not reinforced. Pain leads to social and occupational isolation, which increases disability behavior |
| <i>Negative reinforcement:</i> Avoiding pain, unpleasant emotion, or activity by taking pain medications or reducing physical activity. For example, the patient responds to marital conflict with onset of pain behavior to avoid unpleasantness, which further increases pain behavior | | |
| <i>Sensitization:</i> Heightened sensitivity to pain sensation, which is an intensified response to continuous pain stimuli. Unexpectedly, there is a lack of habituation in intensity of a pain response | | |
| | Classical conditioning | <i>Tension-pain cycle:</i> Acute pain (unconditioned stimulus) leads to sympathetic activity (unconditioned response). The pairing of body position, thoughts, people, or environmental stimuli (neutral stimulus) and fear of pain (unconditioned stimulus) with sympathetic activity (conditioned response) are linked. This cycle leads to an anticipatory fear of pain response and increased sympathetic activity in the absence of nociception |
| Cognitive-affective-sociocultural | Directionality and dimensionality | <i>Emotion regulation:</i> Emotions act as precipitating, exacerbating, consequential, or reciprocal factors of pain |
| | Neuroscience theories (eg, neuromatrix theory, localization theory, and cortical reorganization/constructivism theory) | <i>Neurocognition:</i> Pain interrupts attention, executive function, learning, and memory retrieval |
| | | <i>Attention bias:</i> Hyperattentiveness to physical sensation can lead to over-response to pain stimuli. Increase of attention to pain stimuli leads to brain activity in central pain centers as seen on fMRI study. Decrease of attention or distraction away from pain stimuli leads to brain deactivation of central pain centers as seen on fMRI |
| | <i>Emotions, pain interpretation, and progression of pain experience:</i> When acute pain becomes chronic, localized nociceptive brain activity changes to emotional brain circuits' activity. This suggests that affective-cognitive aspects are involved in the interpretation of pain and the constructive meaning the brain assigns to the pain based on experience as acute pain progress to chronic pain | |
| Cognitive theory | <i>Dysfunctional thoughts:</i> Catastrophizing thoughts (ie, magnification of a single event into a never-ending harmful outcome) further increases pain and distress. Beliefs or expectations and the coping response modify pain perception | |
| Appraisal of stress | Appraisal of stress | <i>Cultural beliefs and self-efficacy:</i> Sociocultural beliefs and self-efficacy of level of control over physical health determine pain tolerance |
| | | <i>Positive attribution:</i> Positive appraisal correlates with lower pain intensity; negative appraisal correlates with higher pain intensity |

fMRI: functional magnetic resonance imaging

Source: Adapted from references 13,27

Functional assessment to rule out the disproportional impact of pain on daily activities is helpful in understanding the somatization process. Neurocognitive functioning should be assessed, particularly because frontal and subcortical dysregulation has been observed in head pain sufferers.^{29,30} Patients with cognitive changes as a result of a medical illness (eg, stroke, head concussion, brain tumor, or seizures) are especially at risk for neurocognitive dysfunction.

Neuropsychological assessment can be useful, not only to assess neurocognitive functioning (eg, Repeated Battery for the Assessment of Neuropsychological Status) but to identify objective test profiles associated with altered motivation (eg, Rey 15-Item Test, Minnesota Multiphasic Personality Inventory-2-Restructured Form F Scale, Personality Assessment Inventory [PAI] Negative Impression Management) and somatization processes (eg, PAI Somatization Scale). These instruments help to identify the severity of psychiatric and neurocognitive symptoms by comparing scores to normative (eg, healthy control group), clinical (eg, somatization, traumatic brain injury, mild cognitive impairment), and altered motivation (eg, persons instructed to exaggerate symptoms) databases.

If the clinician pursues neurocognitive assessment, direct referral to a neuropsychologist, referral to neurologist, or administration of a cognitive screening tool such as the Montreal Cognitive Assessment, Saint Louis University Mental Status, or Cognitive Log is recommended. If the cognitive screening is positive, next steps include: referring for full neuropsychological assessment, which includes complete cognitive and motor testing, personality testing, and integration of neuroimaging data (eg, MRI, CT scans, and/or EEG).

Assessing the patients' self-talk or thought patterns as they describe their head pain will help clinicians understand belief systems that may be distorting the reality of the medical condition. For example, a patient might report that "my pain feels like someone is hitting me with an axe"; this is a catastrophic thought that

can distort the clarity and perceptibility of pain. Encouraging patients to monitor and analyze their anxiety and associated negative thoughts is an important strategy for improving mood and decreasing somatization. Recording daily thoughts and CBT can help the patient identify and appropriately address his (her) cognitive distortions and futile thinking.

When implementing a treatment plan for somatization disorder, we propose the mnemonic device **CARE MD**:

- CBT
- Assess (by ruling out a medical cause for somatic complaints)
- Regular visits
- Empathy
- Med-psych interface (help the patient connect physical complaints and emotional stressors)
- Do no harm.

Clinical recommendations

Chronic head pain can be debilitating; psychodiagnostic assessment should therefore be considered an important part of the diagnosis and treatment plan. After ruling out common and emergent primary or secondary causes of head pain, consider psychiatric comorbidities. Depression and anxiety have a strong bidirectional relationship with chronic headache; therefore, we suggest evaluating patients with the intention of alleviating both psychiatric symptoms and head pain.

It is important to diligently assess for common psychiatric comorbidities; using the AMPS and CARE MD mnemonics, along with screening for somatization disorders, is an easy and effective way to evaluate for relevant psychiatric conditions associated with chronic head pain. Because many patients have unusual and complicated responses to head pain that can be explained by non-pathophysiological and non-biomechanical models, using the biopsychosocial model is essential for effective diagnosis, assessment, and treatment. Abortive and prophylactic medical interventions, as well as behavioral, sociocultural, and cognitive assessment, are vital to a comprehensive treatment approach.

continued

Clinical Point

Depression and anxiety have a strong bidirectional relationship with chronic headache



Head pain

Clinical Point

Abortive and prophylactic medical interventions, as well as behavioral, sociocultural, and cognitive assessment, are vital

Related Resources

- Pompili M, Di Cosimo D, Innamorati M, et al. Psychiatric comorbidity in patients with chronic daily headache and migraine: a selective overview including personality traits and suicide risk. *J Headache Pain*. 2009;10(4):283-290.
- Sinclair AJ, Sturrock A, Davies B, et al. Headache management: pharmacological approaches [published online July 3, 2015]. *Pract Neurol*. doi:10.1136/practneurol-2015-001167.

Drug Brand Names

| | |
|------------------------------|-------------------------|
| Amitriptyline • Elavil | Meperidine • Demerol |
| Botulinum toxin A • Botox | Metoclopramide • Reglan |
| Divalproex sodium • Depakote | Propranolol • Inderide |
| Ketorolac • Toradol | Topiramate • Topamax |

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Bottom Line

The psychodiagnostic assessment can help the astute clinician identify comorbid psychiatric conditions, psychological factors, and somatic symptoms to develop a comprehensive biopsychosocial treatment plan for patients with chronic head pain. Rule out primary and secondary causes of pain and screen for somatization disorders. Consider medication and psychotherapeutic treatment options.