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Chronic Pain: Evaluation and Treatment in the Emergency Department

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It's been a busy shift. The rack is full. There's a chart that keeps getting moved back further and further. Of course, everyone knows who that is. She's been here three times already this week. Sometimes she gets drugs and sometimes she doesn't. Of course the times she doesn't get them, she just comes back again later after shift change. She has been waiting now for well over 2 hours despite her complaint of 10 out of 10 pain. Reluctantly you take the chart and listen to her complaints with half an ear. It's not her back this time, but higher up in her chest. The pain sounds a little different as well. There is also that cough she has had for the past 2-3 weeks. And after you ask her, she admits she has coughed up blood. You order a chest x-ray, although the other staff scoff at you. After all, she is always here. "Just give her the drugs and she will leave." The x-ray shows a large tumor eroding several ribs. True story.

Patients with chronic pain can be some of the most difficult patients to deal with. They are often miserable and demanding, and their quest for pain relief can lead to addiction. While no physician wants to give drugs to someone who is just drug-seeking, it is often impossible to tell the drug-seekers from those who are truly in pain. As my mentor taught me, it is better to give pain medicine to someone who didn't need it than to withhold it from someone in pain.

In addition, as the short case above illustrates, we can sometimes ignore serious symptoms in patients who are "regulars" in our departments. Nearly all seasoned emergency physicians can relate a similar case they almost sent out. Because these patients can become bothersome to care for, we must be hypervigilant to avoid making errors.

As discussed in this article, the best way to address these patient, is to have a protocol in place. Once a patient is deter-

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mined to have a chronic pain syndrome, the primary care physician should discuss options for handling breakthrough pain and the use of the emergency department (ED). The ED should receive and use the protocol when the patient presents. Having and meeting expectations can decrease some of the frustration of an ED visit for both the patient and the physician. If a patient make several visits to the ED, the ED should initiate a protocol with the primary care physician or even on their own.

—Sandra M. Schneider, MD, FACEP, Editor

Introduction

Patients with chronic pain frequently present to emergency physicians for care. It is estimated that patients with an exacerbation of chronic pain represent 11.1% to 13.7% of all non-critical ED patients.[1,2] Compared to patients with acute pain, they are more likely to report their pain as severe and more likely to be frequent visitors to the ED.[2,3] Only 25% have seen a pain specialist.[2] Indeed, the emergency department is the default source of care for chronic pain patients with breakthrough pain who have failed their management regimen, or have failed to access a care provider who has met their desired expectation for pain relief.[4] The evaluation and treatment of patients with chronic pain is distinctly different from the management of patients with acute pain; however, emergency physicians may care for patients at any time from the first presentation of pain to an established chronic diagnosis. Chronic pain is generally defined as pain lasting for three months or longer. Patients who sustain pain of that duration may have seen several physicians and have had several diagnostic evaluations. Chronic pain is frustrating to patients who search for an organic cause and effective treatment.[5] Physicians are frustrated with what they perceive as unrealistic patient expectations for pain relief, persistent requests for opioids, concern about addiction, and patient refusal to accept a psychiatric explanation for symptoms.[5]

This review discusses the most common forms of chronic non-malignant pain presenting to the emergency department including back and neck pain, myofascial pain syndromes, transformed migraine, fibromyalgia, and several forms of neuropathic pain: cervical or lumbar radiculopathy, diabetic neuropathy, postherpetic neuralgia, HIV-related neuropathy, spinal cord injury pain, trigeminal neuralgia, post-stroke pain, and complex regional pain syndromes 1 (reflex sympathetic dystrophy) and 2 (causalgia). This review addresses opioid use in patients with chronic pain and discusses the randomized controlled trials that have assessed the efficacy of treatments for chronic pain.

Epidemiology

Chronic non-malignant pain is a common problem affecting 11-24% of the general population.[6-8] Back pain is the most common site for chronic pain, followed by the neck, extremities, and head.[7] The prevalence of neuropathic pain is 8% of the population.[9] Seventy-nine percent of patients who began with pain of three months duration will still have pain 4 years later.[10] Risk factors for chronic pain include increasing age, female gender, higher body mass, lesser education, and chronic illness.[6,8] Compared to those without pain, patients with chronic pain report higher usage of the health care services, yet have lower satisfaction with the care provided.[7] The cost of common pain conditions is estimated to be $61.2 billion per year in lost work productivity.[11] Prescription opioid use is four times more common in patients with chronic pain (12%) than in the general population (3%).[3] Assessing non-critical emergency department patients, as many as 40.5% have chronic pain.[2] Chronic pain patients are less likely to be satisfied with their ED visit,[3] yet are more likely to have 4 or more ED visits in a year.[2] Those seen in the ED are more likely to be unemployed and disabled, compared to patients in acute pain.[2]

Pathophysiology

Acute pain, or nociceptive pain, serves an important biologic function to warn the individual to stop the potentially injurious activity; in contrast, chronic pain serves no known protective function. Nociceptive pain is mediated by receptors on A delta and C-fibers, which respond to mechanical stretching, compression, thermal changes, and to chemical substances, such as prostaglandins, bradykinins, and other mediators of inflammation. For many chronic pain syndromes such as fibromyalgia, the pathophysiology is not understood. There is some evidence for increased sensitization of peripheral nociceptors in chronic

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Table 1. Signs and Symptoms of Non-Neuropathic Pain Syndromes

DISORDER	PAIN SYMPTOMS	SIGNS
Myofascial headache	Constant dull pain, occasionally shooting pain	Trigger points on scalp, muscle tenderness, and tension
Chronic tension headache	Constant dull pain	Diffuse tenderness of the scalp and associated tension
Transformed migraine	Initially migraine-like, becomes constant, dull; nausea, vomiting	Muscle tenderness and tension, normal neurologic exam
Myofascial neck pain	Constant dull pain, occasionally shooting pain, pain does not typically follow nerve distribution	Trigger points in area of pain, usually no muscle atrophy, poor ROM in involved muscle
Chronic neck pain	Constant dull pain, occasionally shooting pain, pain does not follow nerve distribution	No trigger points, poor ROM in involved muscle
Fibromyalgia	Diffuse muscular pain, stiffness, fatigue, sleep disturbance	Diffuse muscle tenderness, > 11 trigger points
Chronic back pain	Constant dull pain, occasionally shooting pain, pain does not follow nerve distribution	No trigger points, poor ROM in involved muscle
Myofascial back pain syndrome	Constant dull pain, occasionally shooting pain, pain does not typically follow nerve distribution	Trigger points in area of pain, usually no muscle atrophy, poor ROM in involved muscle

Key: ROM = range of motion

myofascial syndromes,[12] but increased central sensitivity has also been proposed.[13] Increased central sensitization may play a role in other chronic pain disorders such as transformed migraine (*defined in Table 1*).[14] There are several underlying mechanisms known for neuropathic pain: direct stimulation, deafferentation, automatic firing of damaged nerves, and sympathetically mediated pain.[15] For any individual neuropathic pain syndrome, more than one mechanism may produce symptoms. Lumbar disk herniation produces direct stimulation of nerves by mechanical compression and chemical irritation to the adjacent nerve roots causing sciatic nerve pain.

Deafferentation occurs when the normal path of sensation transmitted from peripheral nerves to the spinal cord, the brain stem, to the brain, is interrupted by injured or dysfunctional nerves. Interruption of peripheral or spinal cord progression can lead to increased irritability of neurons and firing of nerves further up the chain. This explains how phantom limb pain occurs. In a similar manner, damaged nerves from diabetic neuropathy or post-herpetic neuropathy may generate spontaneous firing of higher order neurons initiating pain. A stroke may interrupt the pain pathway with a lesion in the brain stem or brain itself leading to neuropathic pain. Nerve fibers that are damaged by disease or mechanical injury can fire spontaneously at the site of injury, while at the same time normal sensation is decreased in the area innervated by that nerve. This explains why many patients can have numbness in the same area of spontaneous pain. Any painful stimulus can trigger autonomic activity in the spinal cord at the same dermatomal level of the spinal cord resulting in changes in circulation and temperature. The sympathetic nerves release norepinephrine which can stimulate the primary sensory nerve, causing pain and provoking further sympathetic activity. This is the proposed mechanism for complex regional pain syndrome.

Psychological factors such as depression and anxiety predict chronic pain[16] and are common both before and after diagnosis.[17] Depression is diagnosed in 58% of chronic pain patients. Patients with chronic pain may exhibit behaviors such as drug use (opioid and other drugs), doctor shopping, disability (inability to work), dependence (loss of self-reliance), dramatization (exaggeration of symptoms in hopes of substantiating a physical diagnosis), and depression (despair and negative attitudes).[18] Fear of pain has been shown to affect behavior and promote disability after one year of ongoing pain.[19]

Clinical Features

Classically defined, chronic pain lasts longer than three months, but can be defined as pain that persists beyond the reasonable time for an injury to heal, or pain that persists a month beyond the usual course of an acute disease.

Table 2. Signs and Symptoms of Neuropathic Pain Syndromes

DISORDER	PAIN SYMPTOMS	SIGNS
Painful diabetic neuropathy	Symmetrical numbness and burning or stabbing pain in lower extremities, allodynia may occur	Sensory loss in lower extremities
Phantom limb pain	Variable: Aching, cramping, burning, squeezing, or tearing sensation	May have peri-incisional sensory loss
Trigeminal neuralgia	Paroxysmal, short bursts of sharp, electric like pain, in nerve distribution	Tearing or red eye may be present
HIV-related neuropathy	Symmetrical pain and paresthesias, most prominent in toes and feet	Sensory loss in areas of greatest pain symptoms
Postherpetic neuralgia	Allodynia, shooting, lancinating pain	Sensory changes in the involved dermatome
Post-stroke pain	Same side as weakness, throbbing, shooting pain, allodynia	Loss of hot and cold differentiation
Sciatica (neurogenic back pain)	Constant or intermittent, burning or aching, shooting or electric shock-like pain, may follow dermatome; leg pain > back pain	Possible muscle atrophy in area of pain, possible reflex changes
Complex regional pain type I (RSD)	Burning persistent pain, allodynia, associated with immobilization or disuse	Early: Edema, warmth, local sweating Late: Above alternates with cold; pale, cyanosis, eventually atrophic changes
Complex regional pain type II (causalgia)	Burning persistent pain, allodynia, associated with peripheral nerve injury	Early: Edema, warmth, local sweating Late: Above alternates with cold, pale, cyanosis, eventually atrophic changes

Key: ROM = range of motion; RSD = reflex sympathetic dystrophy.

The signs and symptoms of chronic pain syndromes are listed in Tables 1 (non-neuropathic syndromes) and 2 (neuropathic pain syndromes). The non-neuropathic syndromes share certain characteristics, the most common feature being muscle-related pain.

Patients should be asked to describe the nature of the current pain, including initiating, exacerbating, or relieving factors. Patients may not describe their pain as chronic, but further questioning will reveal several episodes of recurrent pain or an exacerbation of chronic pain. Similar prior episodes should be quantified and compared to the current episode. Sources and modes of treatment, including medications and dosages for physician-prescribed, over-the-counter, or alternative medications should be determined. Outcomes of previous therapeutic efforts and the effects on the patient's functional status are also important. As prior addiction to drugs is a relative contraindication to opioid treatment of chronic pain, the patient should be asked directly about alcohol and drug addiction. Experience with detoxification programs should also be noted. Current substance abuse is a frequent problem in chronic pain patients. Drug detoxification is often the first step of the therapeutic plan for new patients referred to a pain clinic. Finally, a review of systems should be done to rule out any other conditions with an emphasis on potential life- or limb-threatening conditions.

Objective findings of acute pain include tachycardia, hypertension, diaphoresis, and muscle spasms on stimulation. Objective evidence of chronic pain includes muscle atrophy in the distribution of pain due to disuse, or skin temperature changes due to the effects of the sympathetic nervous system after disuse or secondary to nerve injury. Trigger points, another objective sign, are focal points of muscle tenderness and tension that when stimulated with pressure provoke referred pain, typically in the distribution of the involved muscle. Trigger points should be differentiated from simple focal tender portions of the muscle, as palpation of the later does not provoke referred pain. Objective evidence of pain does not have to be present for the pain to be factual. Recall that many patients with sickle cell crisis fail to mount a tachycardia despite severe pain.

Myofascial Headache and Chronic Tension Headache. Myofascial headache is a variant of tension headache and is characterized by the presence of trigger points on the scalp; constant,

squeezing pain; and occasionally shooting pain. Nausea, vomiting, neck pain, and neck tenderness may be present. It is important to differentiate this disorder from common tension headache because myofascial headache may benefit from local anesthetic injection of trigger points, whether that is accomplished in the ED or at referral.

Transformed Migraine. "Transformed migraine" is a syndrome in which classic or common migraine headaches change over time and develop into a chronic pain syndrome. In fact, chronic migraine (15 or more migraine days per month) is a precursor to transformed migraine.[20] In this regard, patients who initially have "vascular symptoms" eventually have predominantly muscular symptoms: non-throbbing, squeezing, bandlike pain associated with muscle tenderness and tension. One cause of this change is frequent treatment with opioids. Nausea and vomiting or failure of an oral antimigraine medication often prompts an ED visit. Some patients may retain some of their typical migraine features, but the headache becomes continuous rather than episodic.

Fibromyalgia. Fibromyalgia affects 2% of the U.S. population (3.4% of women, 0.5% of men).[21] The disorder is classified by the presence of 11 of 18 specific tender points, nonrestorative sleep, muscle stiffness, and generalized aching pain, with symptom duration of more than 3 months. Marked inter-patient variability is common in fibromyalgia for both clinical symptoms and response to medications. Seventy-five percent of patients with fibromyalgia remain in pain years after their diagnosis.[22]

Myofascial Back Pain and Chronic Back Pain. Risk factors for chronic back pain following an acute episode include male gender, advanced age, evidence of nonorganic disease, leg pain, prolonged initial episode, and significant disability at onset.[23] Passive coping strategies (dependence on medication and others for daily tasks) predicts significant disability with chronic back pain.[24] Myofascial back pain is characterized by constant dull and occasional shooting pain that does not follow a classic nerve distribution and the presence of trigger points. Pain may or may not be exacerbated by movement. Usually trigger points can be found at the site of greatest pain, and muscle atrophy is not found. Range of motion of the involved muscle is reduced, but there is no actual muscle weakness. Simple chronic back pain is differentiated from myofascial back pain by the absence of trigger points in the former.

Sciatica. Sciatica or neurogenic back pain is classically characterized by constant or intermittent pain that is burning, shooting, or aching. The pain is usually more severe in the leg than in the back and follows a dermatome. Muscle atrophy as well as reflex changes can be seen over time. Sciatica should be differentiated from myofascial or simple chronic back pain because it does not respond to the same medications as the former conditions and, in general, is more resistant to therapy.

Myofascial Neck Pain, Chronic Neck Pain, Radicular Neck Pain. Neck pain frequently follows an injury or motor vehicle crash, but may arise without an identifiable cause. The etiology, course, and treatment of neck pain has not been studied as frequently as that of back pain, but the guidelines for treatment are similar. Myofascial neck pain is distinguished by the presence of trigger points, while radicular neck pain is distinguished by the presence of a radicular distribution of symptoms that may include numbness and reflex changes. Like the case of sciatica, radicular neck pain is not expected to have the same response rate to drugs (i.e., nonsteroidal anti-inflammatory drugs and opioids) as with non-neurogenic neck pain.

Painful Diabetic Neuropathy. The symptoms of painful diabetic neuropathy are symmetrical numbness associated with burning, electrical, or stabbing pain in lower extremities. Patients may have hyperesthesia, dysesthesias, and/or deep aching pain. Allodynia (pain provoked with gentle touch of the skin) may occur in the areas of abnormal sensation. Stability of glycemic control is an important goal in these patients to slow the progression of symptoms.

Postherpetic Neuralgia. The classic pain of postherpetic neuralgia may follow the course of an acute episode of herpes zoster in 8-70% of cases; increased incidence is seen with advancing age. Pain is characterized by allodynia and shooting, lancinating (tearing or sharply cutting) pain. Often, patients have hyperesthesia in the involved dermatome. Occasionally there are pigmentation changes in the distribution of the involved dermatome, but this is not unique to postherpetic neuralgia.

Trigeminal Neuralgia. Typical symptoms of trigeminal neuralgia include paroxysmal, short bursts of sharp, electric-like pain in the nerve distribution of the trigeminal nerve. Pain is often provoked by muscle movements of the facial muscles as triggered by chewing, speaking, washing, brushing teeth, or something touching the face. Tearing of the eyes or red eye may be present.

Phantom Limb Pain. Phantom limb pain is quite variable in presentation but is more frequent in patients who had pain in the extremity before amputation. Pain may be aching, cramping, burning, tearing, or squeezing. Phantom limb pain occurs in 30-81% of amputations.[25]

Complex Regional Pain. Patients with complex regional pain syndrome (CRPS) type I, also known as reflex sympathetic dystrophy, and CRPS type II, also known as causalgia, may be seen in the ED as early as the second week after treatment of an acute injury.[26] These disorders cannot be differentiated from one another on the basis of signs and symptoms. CRPS type I occurs because of prolonged immobilization or disuse, and CRPS type II occurs because of a peripheral nerve injury. These disorders should be suspected when a patient presents with classic symptoms: allodynia and a persistent burning or shooting pain. Associated signs early in the course of the disease include edema, warmth, and localized abnormal sweating. Therefore, it may be difficult to distinguish this disorder from an underlying wound infection or osteomyelitis. Later signs include periods of edema and warmth that alternate with cold, pale, cyanotic skin and eventually atrophic changes. CRPS is an important diagnosis to make, since early steroid treatment may reduce ongoing symptoms. Furthermore, when complex regional pain is associated with a cast, pin, or external fixation apparatus, the device may need to be removed.[26]

Post-stroke Pain. The symptoms of post stroke pain include a throbbing or shooting pain on the same side of the body affected

Table 3. Management of Non-Neuropathic Chronic Pain Syndromes

DISORDER	PRIMARY TREATMENT	SECONDARY TREATMENT	POSSIBLE REFERRAL OUTCOME
Myofascial headache	NSAIDs, Phenothiazines IV (acute only)	Tricyclic antidepressants	Trigger-point injections, optimization of medical therapy
Chronic tension headache	NSAIDs, Phenothiazines IV (acute only)	Tricyclic antidepressants	Optimization of medical therapy
Transformed migraine	Tricyclic antidepressants	Stop prior medications	Optimization of medical therapy, narcotic withdrawal
Myofascial neck pain	NSAIDs	Tricyclic antidepressants	Trigger-point injections, optimization of medical therapy
Chronic neck pain	Tricyclic antidepressants*	NSAIDs, Opioids	Optimization of medical therapy
Chronic back pain (no sciatica)	Tricyclic antidepressants*	NSAIDs, Opioids	Optimization of medical therapy
Myofascial back pain syndrome	NSAIDs	Tricyclic antidepressants	Trigger-point injections, optimization of medical therapy
Fibromyalgia	Cyclobenzaprine Tramadol	Amitriptyline, Pregabalin	Optimization of medical therapy, dedicated exercise program

Key: NSAIDs = nonsteroidal anti-inflammatory drugs

*Preferred tricyclic antidepressants are nortriptyline, start 25 mg/day, or maprotiline, start 50 mg/day.

by the stroke. Associated allodynia is common as is loss of hot and cold differentiation. Symptoms usually do not appear until several months after the inciting stroke, when recovery has reached a stable state.

HIV-related Neuropathy. Approximately 33% of patients with AIDS develop distal sensory polyneuropathy characterized by shooting pain, numbness, and burning sensations primarily on the soles, dorsum of the feet, and toes.[27] Sensory loss is common in the areas of greatest pain symptoms. Walking becomes difficult and quality of life is diminished.[28]

Diagnosis and Differential

The most important task of the emergency physician is to distinguish an exacerbation of chronic pain from a presentation that heralds a life- or limb-threatening condition. The history and physical examination should either confirm the chronic condition or point to the need for further evaluation when unexpected signs or symptoms are elicited. Because patients with chronic pain may be frequent visitors to the ED, the entire staff may prejudge their complaint as simply chronic or even factitious. Physicians should insist that routine procedures be followed, including a full triage assessment and a complete set of vital signs and timely assessment.

Rarely is a provisional diagnosis of a chronic pain condition made for the first time in the ED. One exception may be complex regional pain. The sharp pain from acute injuries, including fractures, should be significantly improved at 2 weeks post injury. Pain in an injured body part beyond this period should alert the clinician to the possibility of nerve injury, or CRPS secondary to immobilization. CRPS has the following diagnostic criteria: 1) presence of an initiating noxious event, or cause of immobilization; 2) continuing pain, allodynia, or hyperalgesia with which the pain is disproportionate to any inciting event; 3) evidence at some time of edema, changes in skin blood flow, or abnormal sweating in the region of pain; and 4) exclusion of other diagnoses that would explain the symptoms and dysfunction.

Definitive diagnostic testing of chronic pain conditions is difficult, requires expert opinion, and often involves expensive procedures such as magnetic resonance imaging (MRI), computed tomography (CT), and thermography. Furthermore, abnormal results do not confirm with certainty the cause of the pain. For example, abnormalities on MRI of the spine and extremities are common in asymptomatic patients.[29,30] Therefore, referral back to the primary source of care and eventual specialist referral are warranted to confirm the diagnosis.

Treatment of Non-Neuropathic Chronic Pain Syndromes

Evidence-based treatments for specific pain syndromes are

Table 4. Typical Features of Non-Compliance with Opioid Therapy

- Unexpected results on toxicologic screening
- Frequent requests for dose increases
- Concurrent use of non-prescribed psychoactive substances
- Failure to follow dose schedule
- Failure to adhere to dose schedule
- Failures to adhere to concurrently recommended treatments
- Frequently reported loss of prescriptions or medications
- Frequent visits to the emergency department for opioid therapy
- Missed follow-up visits
- Prescriptions obtained from a secondary provider
- Tampering with prescriptions

listed in Table 3. Treatment in the emergency department should never be regarded as definitive, and follow-up care is essential. Many emergency physicians will elect to prescribe short-acting nonspecific pain medications (which may or may not be helpful) for patients with chronic pain, deferring initiation of more specific therapy for the primary care physician or pain specialist. However, definitive therapy may be safely started in the ED provided follow-up is within a reasonable period (one to three weeks). In fact, several of the recommended medications will require two or more weeks to see maximal benefit. Specialty referral may provide optimization of therapy, trigger point injections, and novel treatments, such as botulinum injections.[31]

Opioids for Chronic Pain. The issue of opioid use for chronic pain was reviewed in detail by known experts in the field who recommended their cautious use with caveats.[32] Risk of adverse effects should be assessed prior to the use of opioids, including potential for addiction, which may be impossible in the context of an emergency department visit. Patients with a lack of motivation to improve, those with prior history of impaired control over drug use, compulsive use or continued use despite harm, or craving, can be expected to have adverse effects with opioid use.[32,33] Table 4 lists typical features of non-compliance with opioid therapy.[32] Patients who exhibit these behaviors may warrant discontinuation of opioid therapy if behaviors persist.[32] Drug diversion (selling prescribed opioids or passing them on to others) is illegal and should not be tolerated. In the absence of these behaviors, when function is enhanced, opioids have a legitimate place in the treatment of chronic pain.[32] Ideally, all chronic pain patients should sign a pain contract with their primary provider detailing goals of therapy and limitations of opioid use and behaviors. It should be acknowledged that complete relief of pain is an unrealistic goal in chronic pain conditions.

Headache Syndromes. The treatment of the listed headache syndromes is similar. (*See Table 3.*) A 1995 meta-analysis found few if any medications relieve chronic headaches better than placebo.[34] Nonsteroidal anti-inflammatory drugs (NSAIDs) are the most commonly used drugs for all forms of chronic pain, even when their efficacy is in question.[35] The choice of NSAID has not been shown to make a difference in pain relief in most instances. Use of Cox-2 inhibitors has been associated with increased cardiovascular complications, and several drugs have been pulled from the market. When selecting a standard NSAID, adding a proton-pump inhibitor reduces gastrointestinal complications, and the combination is cheaper than daily use of the Cox-2 inhibitors.[36] Chlorpromazine 10 mg intravenously with or without diphenhydramine to reduce dystonic reactions, and other phenothiazines, has been used with success in the treatment of non-specific headache in the ED.[37,38] Amitriptyline up to 100 mg per day (start at 25 mg HS) has been shown to be effective in chronic headaches and with transformed migraine headaches.[39] The selective serotonin re-uptake inhibitors (SSRIs) have not been shown to be effective in this group of patients.[40]

Back Pain and Neck Pain Syndromes (Non-neuropathic). Therapy of chronic back pain has been better studied than chronic neck pain, and many of the recommendations for back pain can be generalized for neck pain. Use of opioids for chronic back pain was systematically reviewed with meta-analysis, and no significant long-term benefit was found.[41] It should be noted that the majority of the studies entered into this meta-analysis individually concluded that opioids reduced chronic back pain (including acetaminophen-oxycodone combinations, acetaminophen-codeine combinations, long-acting oxycodone, long-acting oxymorphone, and transdermal-fentanyl). The meta-analysis also assessed nine studies where aberrant medication behaviors were reported and these ranged from 5% to 24% of those treated.[41] The final conclusion was that opioids are commonly prescribed for chronic back pain, their efficiency is limited to short-term control only, and there is some risk of associated addiction. While nonsteroidal anti-inflammatory agents have a well established place in the management of acute nonspecific back pain, their role in the management of chronic back pain is less clear due to fewer studies and their side effect profile with long-term use including abdominal pain, diarrhea, edema, dry mouth, rash, dizziness, headache, and tiredness.[42] The highest qualities studies showing efficacy of NSAIDs in chronic back pain studied etoricoxib, a drug that has been denied approval in the United States by the FDA. Diclofenac at a dose of 150 mg/day had equal efficacy compared to 60 mg/day of etoricoxib in a 4-week trial involving 446 patients with chronic back pain randomized to one of the two drugs.[43] In the 1997 systematic review comparing studies that varied between 2 and 6 weeks of therapy, the authors concluded piroxicam, indomethacin, ibuprofen, diclofenac, ketoprofen, naproxen, and diflunisal were equally as effective in the short-term treatment of chronic low back pain, moderate evidence.[44] Although studies of 3 months or more are lacking in patients with chronic low-back pain, it is reasonable to use NSAIDs in this population provided that side effects are anticipated when the decision is made to prescribe them. A 2003 meta-analysis assessed the efficacy of muscle relaxants in the treatment of chronic back pain.[45] Two high quality randomized controlled trials (RCT) involving 222 patients using tetrazepam found it effective in chronic back pain, however, this drug is not approved in the United States, and benefit was only 14 days.[45] Diazepam and cyclobenzaprine appear to offer little benefit compared to placebo.[45] A 2003 meta-analysis assessed the efficacy of

Table 5. Management of Neuropathic Chronic Pain

DISORDER	PRIMARY TREATMENT	SECONDARY TREATMENT	POSSIBLE REFERRAL OUTCOME
Trigeminal neuralgia	Carbamazepine	Opioids	Optimization of medical therapy
HIV neuropathy pain	Lamotrigine	Gabapentin	Optimization of medical therapy
Spinal cord pain	Pregabalin	Opioids	Optimization of medical therapy
Painful diabetic neuropathy	Tricyclic antidepressants* Gabapentin	Pregabalin Tramadol Duloxetine	Optimization of medical therapy
Postherpetic neuralgia	Tricyclic antidepressants* Gabapentin	Tramadol Opioids	Optimization of medical therapy Regional nerve blockade
Phantom limb pain	Gabapentin	Tramadol Opioids	Optimization of medical therapy Sympathectomy
Sciatica (Neurogenic back pain)	Prednisolone Diclofenac	Tricyclic antidepressants* Opioids	Epidural steroids, surgery
Post-stroke pain	Tricyclic antidepressants*	Gabapentin	Optimization of medical therapy
Complex regional pain types I and II (RSD and causalgia)	Acute: Prednisone	Chronic: Calcitonin (type I)	Alendronate or clodronate Sympathetic nerve blocks, spinal analgesia

Key: NSAIDs = nonsteroidal anti-inflammatory drugs; RSD = reflex sympathetic dystrophy,
*Preferred tricyclic antidepressants are amitriptyline, start 25 mg/day, or imipramine, start 25 mg/day

antidepressants in the treatment of chronic back pain.[46] Five randomized, placebo-controlled trials using tricyclic and tetracyclic antidepressants involving 440 patients found mild to moderate decreases in pain. The benefit was most pronounced in the two highest quality studies, using nortriptyline 25-100 mg per day or maprotiline 50-150 mg/day. The benefit of these cyclic antidepressants appears to be independent of their antidepressant effects as in both of these studies, patients with clinical depression were excluded.[46] Bedrest is not recommended as an adjunct.

Fibromyalgia. Tricyclic antidepressants (TCAs) and related drugs (cyclobenzaprine) have some efficacy when compared with placebo. In five clinical trials, Cyclobenzaprine (30-50 mg/day) showed a moderate response rate.[47] Several studies demonstrated that low-dose amitriptyline (25-50 mg/day) produces significant pain score reductions, as compared to placebo controls, in 25-45% of patients.[48-50] A placebo-controlled trial of tramadol (75-300 mg/day) plus acetaminophen (1300-4000 mg/day) found this combination provides better pain relief than placebo.[51] A multicenter placebo-controlled trial of pregabalin (150-450 mg/day) found significantly improved pain compared to placebo.[52]

Treatment Summary for Non-neuropathic Pain Conditions. For short-term use, NSAIDs are an acceptable treatment option for most of the non-neuropathic chronic pain conditions despite the potential for adverse reactions. NSAIDs are less effective in chronic pain conditions compared to acute pain conditions. Opioids require a careful assessment of the patient's potential for aberrant drug behaviors or side effects before being used for short-term therapy. Ultimately, the more specific treatments listed in Table 3 should be instituted, whether at the ED visit or at the follow-up primary care visit. Patients should be informed that complete relief of pain is an unrealistic goal for the ED visit, and a 50% reduction of pain plus improved function should be considered a "good" outcome for long-term management.[4]

Treatment of Neuropathic Pain

Evidence-based treatments are summarized in Table 5. In 2005, a meta-analysis of the use of opioids in neuropathic pain was completed including 14 trials classified as short term (less than 24 hours) and 8 trials classified as intermediate term (median of 28 days).[50] In the short-term trials, opioids provided no significant efficacy. However, for the intermediate trials, opioids provided a significant reduction in pain.[53] Long-acting morphine (30-90 mg three times daily) or long-acting oxycodone (10-30 mg three times daily) were the most frequently used drugs. The results of this meta-analysis imply that a patient who does not respond initially to an opioid dose (as in the short-term studies) may eventually improve as shown in the intermediate-term studies.

Tricyclic antidepressants (TCAs) have been shown to be the most consistently effective therapy in patients with neuropathic pain.[54,55] Drugs such as amitriptyline 75 mg/day, or imipramine 150 mg/day are effective for central post-stroke pain, painful diabetic neuropathy, post-herpetic neuralgia, sciatica, and non-diabetic polyneuropathy. Antidepressants have not been shown to be effective for spinal cord injury pain, phantom limb pain, and HIV-related neuropathy. Several selective serotonin reuptake inhibitors (fluoxetine, venlafaxine, paroxetine, citalopram) have been shown to be less effective than TCAs in neuropathic pain.[54,55] The exception is duloxetine, which recently has been shown to be effective in diabetic neuropathy in three RCTs at a dose of 60-120 mg/day.[56-58]

Gabapentin has been shown to be effective in painful diabetic neuropathy[59] and phantom limb pain (small trial).[60] Gabapentin has been found to be beneficial in a study of more than 300 patients with a wide range of neuropathic pain syndromes.[61] However, patients with complex regional pain syndrome randomized to gabapentin did not improve. The dosing of gabapentin is 300 mg at bedtime day one, 300 mg bid day two, and 300 mg tid day three; the drug should be titrated up to 1800 mg/day over 2 weeks. Pregabalin has found to be effective in painful diabetic neuropathy (dose 300 to 600 mg/day),[62-64] as well as in spinal cord injury related pain (dose 150 to 600 mg/day).[65] Carbamazepine has been the long-standing recommended treatment of trigeminal neuralgia.[54,55]

HIV-related neuropathy has been shown to be resistant to many therapies commonly used for neuropathic pain including TCAs. Lamotrigine at a target dose of 600 mg/day was found to be effective in an RCT, but only in patients receiving neurotoxic antiretroviral therapy (i.e., didanosine, zalcitabine, or stavudine).[66] Gabapentin was found to be effective in a small RCT (29 patients) at a dose of 2400 to 3600 mg/day. Patients in the gabapentin group had a high rate of increased somnolence (80%).[67] Results of this trial conflict with the study by Serpell et al, discussed above.[61]

Tramadol was shown to be effective in a trial of painful diabetic neuropathy , an extension of the study showed benefit out to 6 months.[67] A recent meta-analysis of six RCTs concluded that tramadol is effective in the treatment of neuropathic pain.[68] The typical dose is 200-400 mg/day divided four times daily, starting at 50 mg bid.

Sciatica or Radicular Back Pain. Although prior studies of NSAIDs have found little benefit in sciatica, two recent multicenter RCTs including 1021 patients compared two different doses of oral meloxicam (a selective COX-2 inhibitor, at 7.5 mg and 15 mg PO QD), to placebo; the second compared the same doses of meloxicam to diclofenac (50 mg PO TID).[69] The studies found significant pain improvement occurred with diclofenac and either meloxicam dosage at 6 hours, 3 days, and 1 week, compared to placebo. No studies of muscle relaxants have assessed their use in sciatica, and therefore cannot be recommended as evidence-based. Tricyclic antidepressants have been found to be effective in patients with sciatica.[54,55]

Steroids have been used for the treatment of acute sciatica for decades with the belief that steroids should reduce inflammation secondary to nerve root compression. However, the improvement associated with their use appears to be brief. Acute administration of prednisolone or prednisone significantly reduces pain scores but the benefit appears to last approximately 24 hours.[70] Epidural steroids have benefit, but a meta-analysis and subsequent trial found pain relief lasts 3 weeks, but at 6 to 52 weeks no benefit over placebo was found.[71,72] Bed rest is not recommended as an adjunct.

Complex Regional Pain Syndromes (CRPS). Complex regional pain syndromes are the most resistant to pharmacotherapy of all neuropathic pain conditions.[54,55] Two trials of intravenous infusion of bisphosphonate therapy reduced pain, one trial with 7.5 mg alendronate[73] and another with 300 mg/day of clodronate.[74] These therapies require daily infusions. A meta-analysis of 21 RCTs of therapies for CRPS type 1 concluded that intranasal calcitonin is beneficial at a dose of 200 IU. No other therapy was recommended by this meta-analysis for CRPS type 1.[75] In the past, corticosteroids (prednisone 10 mg daily) have been recommended on the basis of two small trials (59 patients total).[76,77]

Treatment Summary for Patients with Neuropathic Pain. Patients should be informed that opioids are less commonly effective in neuropathic pain than with other forms of pain. Furthermore, patients who do not respond acutely may respond to the same dose of an opioid several weeks into therapy;[53] therefore, dose escalation may not be warranted. In general, tricyclic antidepressants are the most effective therapy for patients with neuropathic pain, with the exceptions being spinal cord injury pain, phantom limb pain, HIV-related neuropathy, and CRPS. Exceptions to these guidelines are common, especially when depression accompanies chronic pain. Gabapentin has shown great promise in several neuropathic conditions, including painful diabetic neuropathy, post-herpetic neuropathy, phantom limb pain, and post-stroke pain. Pregabalin has been found to be effective in painful diabetic neuropathy and post spinal cord injury pain. Duloxetine has been shown to be effective in painful diabetic neuropathy. NSAIDs generally are in-effective in neuropathic pain conditions. As described above, complete relief of pain is an unrealistic goal for patients with chronic pain, both for the ED visit and for follow-up care.

Patient Disposition

Admission is rarely needed for pain alone in patients with chronic pain. One of the most productive components to the management of chronic pain in the emergency department is successful referral of the patient back to the patient's source of primary care or to a pain specialist of optimization of medication and consideration for additional testing or procedures to treat pain, such as trigger point injections, botulinum injections, or surgical procedures. Furthermore, a patient-provider endorsed pain contract is the ideal way that opioids are judiciously administered.

Summary

The evaluation and care of patients with chronic pain in the emergency department is different from the care of patients with

acute pain. Patients with chronic pain can be expected to exhibit different symptoms and to respond to different medications than patients with acute pain.

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Physician CME Questions

1. Which of the following statements is true concerning chronic pain?
   A. Patients with chronic pain respond to the same medications as patients with acute pain.
   B. The pathophysiology of chronic pain is identical to acute pain.
   C. Symptoms and signs of chronic pain are identical to symptoms of acute pain.
   D. The emergency physician who is evaluating a patient with chronic pain should differentiate an exacerbation of chronic pain from a life- or limb-threatening condition.

2. Compared to the general population, which of the following statements concerning patients with chronic pain is true?
   A. Patients with chronic pain are four times more likely to be taking prescribed opioids.
   B. Patients with chronic pain are more likely to be satisfied with their medical care.
   C. Patients with chronic pain are more likely to enjoy good health.
   D. Patients with chronic pain are more likely to hold a professional degree.

3. Which of the following statements is true regarding the pathophysiology of chronic pain?
   A. The pathophysiology of chronic pain is well established for all known syndromes.
   B. The occurrence of chronic pain serves no known protective biologic function.
   C. The sensations of chronic pain are transmitted exclusively on A delta fibers to the brain from the periphery.
   D. The pathophysiology of neuropathic pain does not appear to be affected by deafferentation.

4. Which of the following statements is true concerning the psychology of patients with chronic pain?
   A. Depression and anxiety rarely occur in chronic pain patients before the onset of pain.
   B. Depression and anxiety rarely occur in chronic pain patients after the onset of pain.
   C. Addiction to narcotics rarely occurs in chronic pain patients.
   D. Fear of pain has been shown to affect behavior and promote disability one year after the onset of ongoing pain.

5. Myofascial pain syndromes of the head, neck, and back can be differentiated from simple headache, neck, and back pain by the presence of:
   A. narcotic addition in the patients diagnosed.
   B. sleeping disturbances in the patients diagnosed.
   C. tender areas of the muscles involved.
   D. trigger points.

6. Which of the following statements is true regarding transformed migraine headache?
   A. Transformed migraine is another name for common migraine.
   B. Transformed migraine should be treated identically to classic migraine.
   C. Transformed migraine may be preceded by chronic migraine and resemble chronic tension headache in its latter stages.
   D. Transformed migraine has no association with opioid use by patients.

7. Which of the following statements is true regarding fibromyalgia?
   A. Fibromyalgia is more common in men than women.
   B. Fibromyalgia is most effectively treated with long-acting opioids.
   C. Fibromyalgia responds quickly and almost completely when appropriate medication is prescribed.
   D. Cyclobenzaprine and amitriptyline have demonstrated efficiency in patients with fibromyalgia.

8. Which of the following statements is true regarding gabapentin?
   A. Gabapentin has demonstrated efficacy in patients with both painful diabetic neuropathy and post-herpetic neuralgia.
   B. Gabapentin is the first drug of choice for trigeminal neuralgia.
   C. Gabapentin is the drug of choice in complex regional pain syndrome.
   D. Gabapentin should not be used in patients with phantom limb pain.

9. Which of the following statements is true regarding the use of opioids in patients with chronic pain?
   A. Opioids should never be prescribed in patients with chronic pain.
   B. Drug diversion is noted to occur in 51% of patients with chronic pain.
   C. A patient-provider endorsed opioid contract can be used to set goals and limits for opioid use by patients with chronic pain.
   D. Opioids have never been shown to benefit patients with chronic pain.

10. Which of the following statements regarding chronic back pain is true?
   A. NSAIDs are more effective in acute back pain than in chronic back pain, but diclofenac specifically has been shown to provide some benefit in patients with chronic back pain.
   B. Bed rest is a mandatory component to the care of patients with back pain.
   C. Opioid use in chronic back pain patients appears to pose no risk for addiction.
   D. Diazepam and cyclobenzaprine have been shown to have clear benefit in randomized controlled trials of chronic back pain patients.

CME Answer Key

1. D; 2. A; 3. B; 4. D; 5. D; 6. C; 7. D; 8. A; 9. C; 10. A

Emergency Medicine Reports
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Chronic Pain

Signs and Symptoms of Non-Neuropathic Pain Syndromes

DISORDER	PAIN SYMPTOMS	SIGNS
Myofascial headache	Constant dull pain, occasionally shooting pain	Trigger points on scalp, muscle tenderness, and tension
Chronic tension headache	Constant dull pain	Diffuse tenderness of the scalp and associated tension
Transformed migraine	Initially migraine-like, becomes constant, dull; nausea, vomiting	Muscle tenderness and tension, normal neurologic exam
Myofascial neck pain	Constant dull pain, occasionally shooting pain, pain does not typically follow nerve distribution	Trigger points in area of pain, usually no muscle atrophy, poor ROM in involved muscle
Chronic neck pain	Constant dull pain, occasionally shooting pain, pain does not follow nerve distribution	No trigger points, poor ROM in involved muscle
Fibromyalgia	Diffuse muscular pain, stiffness, fatigue, sleep disturbance	Diffuse muscle tenderness, > 11 trigger points
Chronic back pain	Constant dull pain, occasionally shooting pain, pain does not follow nerve distribution	No trigger points, poor ROM in involved muscle
Myofascial back pain syndrome	Constant dull pain, occasionally shooting pain, pain does not typically follow nerve distribution	Trigger points in area of pain, usually no muscle atrophy, poor ROM in involved muscle

Key: ROM = range of motion

Signs and Symptoms of Neuropathic Pain Syndromes

DISORDER	PAIN SYMPTOMS	SIGNS
Painful diabetic neuropathy	Symmetrical numbness and burning or stabbing pain in lower extremities, allodynia may occur	Sensory loss in lower extremities
Phantom limb pain	Variable: Aching, cramping, burning, squeezing, or tearing sensation	May have peri-incisional sensory loss
Trigeminal neuralgia	Paroxysmal, short bursts of sharp, electric like pain, in nerve distribution	Tearing or red eye may be present
HIV-related neuropathy	Symmetrical pain and paresthesias, most prominent in toes and feet	Sensory loss in areas of greatest pain symptoms
Postherpetic neuralgia	Allodynia, shooting, lancinating pain	Sensory changes in the involved dermatome
Post-stroke pain	Same side as weakness, throbbing, shooting pain, allodynia	Loss of hot and cold differentiation
Sciatica (neurogenic back pain)	Constant or intermittent, burning or aching, shooting or electric shock-like pain, may follow dermatome; leg pain > back pain	Possible muscle atrophy in area of pain, possible reflex changes
Complex regional pain type I (RSD)	Burning persistent pain, allodynia, associated with immobilization or disuse	Early: Edema, warmth, local sweating Late: Above alternates with cold; pale, cyanosis, eventually atrophic changes
Complex regional pain type II (causalgia)	Burning persistent pain, allodynia, associated with peripheral nerve injury	Early: Edema, warmth, local sweating Late: Above alternates with cold, pale, cyanosis, eventually atrophic changes

Key: ROM = range of motion; RSD = reflex sympathetic dystrophy.

Management of Non-Neuropathic Chronic Pain Syndromes

DISORDER	PRIMARY TREATMENT	SECONDARY TREATMENT	POSSIBLE REFERRAL OUTCOME
Myofascial headache	NSAIDs, Phenothiazines IV (acute only)	Tricyclic antidepressants	Trigger-point injections, optimization of medical therapy
Chronic tension headache	NSAIDs, Phenothiazines IV (acute only)	Tricyclic antidepressants	Optimization of medical therapy
Transformed migraine	Tricyclic antidepressants	Stop prior medications	Optimization of medical therapy, narcotic withdrawal
Myofascial neck pain	NSAIDs	Tricyclic antidepressants	Trigger-point injections, optimization of medical therapy
Chronic neck pain	Tricyclic antidepressants*	NSAIDs, Opioids	Optimization of medical therapy
Chronic back pain (no sciatica)	Tricyclic antidepressants*	NSAIDs, Opioids	Optimization of medical therapy
Myofascial back pain syndrome	NSAIDs,	Tricyclic antidepressants	Trigger-point injections, optimization of medical therapy
Fibromyalgia	Cyclobenzaprine Tramadol	Amitriptyline, Pregabalin	Optimization of medical therapy, dedicated exercise program

Key: NSAIDs = nonsteroidal anti-inflammatory drugs

*Preferred tricyclic antidepressants are nortriptyline, start 25 mg/day, or maprotiline, start 50 mg/day.

Management of Neuropathic Chronic Pain

DISORDER	PRIMARY TREATMENT	SECONDARY TREATMENT	POSSIBLE REFERRAL OUTCOME
Trigeminal neuralgia	Carbamazepine	Opioids	Optimization of medical therapy
HIV neuropathy pain	Lamotrigine	Gabapentin	Optimization of medical therapy
Spinal cord pain	Pregabalin	Opioids	Optimization of medical therapy
Painful diabetic neuropathy	Tricyclic antidepressants* Gabapentin	Pregabalin Tramadol Duloxetine	Optimization of medical therapy
Postherpetic neuralgia	Tricyclic antidepressants* Gabapentin	Tramadol Opioids	Optimization of medical therapy Regional nerve blockade
Phantom limb pain	Gabapentin	Tramadol Opioids	Optimization of medical therapy Sympathectomy
Sciatica (Neurogenic back pain)	Prednisolone Diclofenac	Tricyclic antidepressants* Opioids	Epidural steroids, surgery
Post-stroke pain	Tricyclic antidepressants*	Gabapentin	Optimization of medical therapy
Complex regional pain types I and II (RSD and causalgia)	Acute: Prednisone	Chronic: Calcitonin (type I)	Alendronate or clodronate Sympathetic nerve blocks, spinal analgesia

Key: NSAIDs = nonsteroidal anti-inflammatory drugs; RSD = reflex sympathetic dystrophy,
*Preferred tricyclic antidepressants are amitriptyline, start 25 mg/day, or imipramine, start 25 mg/day

Typical Features of Non-Compliance with Opioid Therapy

- Unexpected results on toxicologic screening
- Frequent requests for dose increases
- Concurrent use of non-prescribed psychoactive substances
- Failure to follow dose schedule
- Failure to adhere to dose schedule
- Failures to adhere to concurrently recommended treatments
- Frequently reported loss of prescriptions or medications
- Frequent visits to the emergency department for opioid therapy
- Missed follow-up visits
- Prescriptions obtained from a secondary provider
- Tampering with prescriptions

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