Chapter 16: Headache

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INTRODUCTION

Headache is among the most common reasons patients seek medical attention and is responsible, on a global basis, for more disability than any other neurologic problem. Diagnosis and management are based on a careful clinical approach augmented by an understanding of the anatomy, physiology, and pharmacology of the nervous system pathways mediating the various headache syndromes. This chapter will focus on the general approach to a patient with headache; migraine and other primary headache disorders are discussed in **Chap. 430**.

GENERAL PRINCIPLES

A classification system developed by the International Headache Society (*www.ihs-headache.org/en/resources/guidelines/*) characterizes headache as primary or secondary **(Table 16-1)**. *Primary headaches* are those in which headache and its associated features are the disorder itself, whereas *secondary headaches* are those caused by exogenous disorders (Headache Classification Committee of the International Headache Society, 2018). Primary headache often results in considerable disability and a decrease in the patient's quality of life. Mild secondary headache, such as that seen in association with upper respiratory tract infections, is common but rarely worrisome. Life-threatening headache is relatively uncommon, but vigilance is required in order to recognize and appropriately treat such patients.

TABLE 16-1

Common Causes of Headache

PRIMARY HEADACHE		SECONDARY HEADACHE	
TYPE	**%**	**TYPE**	**%**
Tension-type	69	Systemic infection	63
Migraine	16	Head injury	4
Idiopathic stabbing	2	Vascular disorders	1
Exertional	1	Subarachnoid hemorrhage	<1
Cluster	0.1	Brain tumor	0.1

Source: After J Olesen et al: *The Headaches*. Philadelphia, Lippincott Williams & Wilkins, 2005.

ANATOMY AND PHYSIOLOGY OF HEADACHE

Pain usually occurs when peripheral nociceptors are stimulated in response to tissue injury, visceral distension, or other factors **(Chap. 13)**. In such situations, pain perception is a normal physiologic response mediated by a healthy nervous system. Pain can also result when pain-producing pathways of the peripheral or central nervous system (CNS) are damaged or activated inappropriately. Headache may originate from either or both

mechanisms. Relatively few cranial structures are pain producing; these include the scalp, meningeal arteries, dural sinuses, falx cerebri, and proximal segments of the large pial arteries. The ventricular ependyma, choroid plexus, pial veins, and much of the brain parenchyma are not pain producing.

The key structures involved in primary headache are the following:

- The large intracranial vessels and dura mater, and the peripheral terminals of the trigeminal nerve that innervate these structures
- The caudal portion of the trigeminal nucleus, which extends into the dorsal horns of the upper cervical spinal cord and receives input from the first and second cervical nerve roots (the trigeminocervical complex)
- Rostral pain-processing regions, such as the ventroposteromedial thalamus and the cortex
- The pain-modulatory systems in the brain that modulate input from the trigeminal nociceptors at all levels of the pain-processing pathways and influence vegetative functions, such as the hypothalamus and brainstem

The *trigeminovascular system* innervates the large intracranial vessels and dura mater via the trigeminal nerve. Cranial autonomic symptoms, such as lacrimation, conjunctival injection, nasal congestion, rhinorrhea, periorbital swelling, aural fullness, and ptosis, are prominent in the trigeminal autonomic cephalalgias (TACs), including cluster headache and paroxysmal hemicrania, and may also be seen in migraine, even in children. These autonomic symptoms reflect activation of cranial parasympathetic pathways, and functional imaging studies indicate that vascular changes in migraine and cluster headache, when present, are similarly driven by these cranial autonomic systems. Thus, they are secondary, and not causative, events in the headache cascade. Moreover, they can often be mistaken for symptoms or signs of cranial sinus inflammation, which is then overdiagnosed and inappropriately managed. Migraine and other primary headache types are not “vascular headaches”; these disorders do not reliably manifest vascular changes, and treatment outcomes cannot be predicted by vascular effects. Migraine is a brain disorder and is best understood and managed as such.

CLINICAL EVALUATION OF ACUTE, NEW-ONSET HEADACHE

The patient who presents with a new, severe headache has a differential diagnosis that is quite different from the patient with recurrent headaches over many years. In new-onset and severe headache, the probability of finding a potentially serious cause is considerably greater than in recurrent headache. Patients with recent onset of pain require prompt evaluation and appropriate treatment. Serious causes to be considered include meningitis, subarachnoid hemorrhage, epidural or subdural hematoma, glaucoma, tumor, and purulent sinusitis. When worrisome symptoms and signs are present **(Table 16-2)**, rapid diagnosis and management are critical.

TABLE 16-2

Headache Symptoms That Suggest a Serious Underlying Disorder

Sudden-onset headache
First severe headache
“Worst” headache ever
Vomiting that precedes headache
Subacute worsening over days or weeks
Pain induced by bending, lifting, coughing
Pain that disturbs sleep or presents immediately upon awakening
Known systemic illness
Onset after age 55
Fever or unexplained systemic signs
Abnormal neurologic examination
Pain associated with local tenderness, e.g., region of temporal artery

A careful neurologic examination is an essential first step in the evaluation. In most cases, patients with an abnormal examination or a history of recent-onset headache should be evaluated by a computed tomography (CT) or magnetic resonance imaging (MRI) study of the brain. As an initial screening procedure for intracranial pathology in this setting, CT and MRI methods appear to be equally sensitive. In some circumstances, a lumbar puncture (LP) is also required, unless a benign etiology can be otherwise established. A general evaluation of acute headache might include cranial arteries by palpation; cervical spine by the effect of passive movement of the head and by imaging; the investigation of cardiovascular and renal status by blood pressure monitoring and urine examination; and eyes by funduscopy, intraocular pressure measurement, and refraction.

The patient’s psychological state should also be evaluated because a relationship exists between head pain, depression, and anxiety. This is intended to identify comorbidity rather than provide an explanation for the headache, because troublesome headache is seldom simply caused by mood change. Although it is notable that medicines with antidepressant actions are also effective in the preventive treatment of both tension-type headache and migraine, each symptom must be treated optimally.

Underlying recurrent headache disorders may be activated by pain that follows otologic or endodontic surgical procedures. Thus, pain about the head as the result of diseased tissue or trauma may reawaken an otherwise quiescent migraine syndrome. Treatment of the headache is largely ineffective until the cause of the primary problem is addressed.

Serious underlying conditions that are associated with headache are described below. Brain tumor is a rare cause of headache and even less commonly a cause of severe pain. The vast majority of patients presenting with severe headache have a benign cause.

SECONDARY HEADACHE

The management of secondary headache focuses on diagnosis and treatment of the underlying condition.

MENINGITIS

Acute, severe headache with stiff neck and fever suggests meningitis. LP is mandatory. Often there is striking accentuation of pain with eye movement. Meningitis can be easily mistaken for migraine in that the cardinal symptoms of pounding headache, photophobia, nausea, and vomiting are frequently present, perhaps reflecting the underlying biology of some of the patients.

Meningitis is discussed in Chaps. 138 and 139.

INTRACRANIAL HEMORRHAGE

Acute, maximal in <5 min, severe headache lasting >5 min with stiff neck but without fever suggests subarachnoid hemorrhage. A ruptured aneurysm, arteriovenous malformation, or intraparenchymal hemorrhage may also present with headache alone. Rarely, if the hemorrhage is small or below the foramen magnum, the head CT scan can be normal. Therefore, LP may be required to diagnose definitively subarachnoid hemorrhage.

Subarachnoid hemorrhage is discussed in Chap. 429, and intracranial hemorrhage in Chap. 428.

BRAIN TUMOR

Approximately 30% of patients with brain tumors consider headache to be their chief complaint. The head pain is usually nondescript—an intermittent deep, dull aching of moderate intensity, which may worsen with exertion or change in position and may be associated with nausea and vomiting. This pattern of symptoms results from migraine far more often than from brain tumor. The headache of brain tumor disturbs sleep in about 10% of patients. Vomiting that precedes the appearance of headache by weeks is highly characteristic of posterior fossa brain tumors. A history of amenorrhea or galactorrhea should lead one to question whether a prolactin-secreting pituitary adenoma (or polycystic ovary syndrome) is the source of headache. Headache arising de novo in a patient with known malignancy suggests either cerebral metastases or carcinomatous meningitis. Head pain appearing abruptly after bending, lifting, or coughing can be due to a posterior fossa mass, a Chiari malformation, or low cerebrospinal fluid (CSF) volume.

Brain tumors are discussed in Chap. 90.

TEMPORAL ARTERITIS

Temporal (giant cell) arteritis is an inflammatory disorder of arteries that frequently involves the extracranial carotid circulation **(SEE ALSO Chaps. 32 AND 363)**. It is a common disorder of the elderly; its annual incidence is 77 per 100,000 individuals aged ≥50. The average age of onset is 70 years, and women account for 65% of cases. About half of patients with untreated temporal arteritis develop blindness due to involvement of the ophthalmic artery and its branches; indeed, the ischemic optic neuropathy induced by giant cell arteritis is the major cause of rapidly developing bilateral blindness in patients >60 years. Because treatment with glucocorticoids is effective in preventing this complication, prompt recognition of the disorder is important.

Typical presenting symptoms include headache, polymyalgia rheumatica **(Chap. 363)**, jaw claudication, fever, and weight loss. Headache is the dominant symptom and often appears in association with malaise and muscle aches. Head pain may be unilateral or bilateral and is located temporally in 50% of patients but may involve any and all aspects of the cranium. Pain usually appears gradually over a few hours before peak intensity is reached; occasionally, it is explosive in onset. The quality of pain is infrequently throbbing; it is almost invariably described as dull and boring, with superimposed episodic stabbing pains similar to the sharp pains that appear in migraine. Most patients can recognize that the origin of their head pain is superficial, external to the skull, rather than originating deep within the cranium (the pain site usually identified by migraineurs). Scalp tenderness is present, often to a marked degree; brushing the hair or resting the head on a pillow may be impossible because of pain. Headache is usually worse at night and often aggravated by exposure to cold. Additional findings may include reddened, tender nodules or red streaking of the skin overlying the temporal arteries, and tenderness of the temporal or, less commonly, the occipital arteries.

The erythrocyte sedimentation rate (ESR) is often, although not always, elevated; a normal ESR does not exclude giant cell arteritis. A temporal artery biopsy followed by immediate treatment with prednisone 80 mg daily for the first 4–6 weeks should be initiated when clinical suspicion is high; treatment should not be unreasonably delayed to obtain a biopsy. The prevalence of migraine among the elderly is substantial, considerably higher than that of giant cell arteritis. Migraineurs often report amelioration of their headache with prednisone; thus, caution must be used when interpreting

the therapeutic response.

GLAUCOMA

Glaucoma may present with a prostrating headache associated with nausea and vomiting. The headache often starts with severe eye pain. On physical examination, the eye is often red with a fixed, moderately dilated pupil.

Glaucoma is discussed in Chap. 32.

PRIMARY HEADACHE DISORDERS

Primary headaches are disorders in which headache and associated features occur in the absence of any exogenous cause. The most common are migraine, tension-type headache, and the TACs, notably cluster headache. These entities are discussed in detail in **Chap. 430**.

CHRONIC DAILY OR NEAR-DAILY HEADACHE

The broad description of chronic daily headache (CDH) can be applied when a patient experiences headache on 15 days or more per month. CDH is neither a single entity nor a diagnosis; it encompasses a number of different headache syndromes, both primary and secondary **(Table 16-3)**. In aggregate, this group presents considerable disability and is thus specially mentioned here. Population-based estimates suggest that about 4% of adults have daily or near-daily headache.

TABLE 16-3

Classification of Daily or Near-Daily Headache

Primary		
>4 H DAILY	**<4 H DAILY**	**SECONDARY**
Chronic migraine[a]	Chronic cluster headache[b]	Posttraumatic Head injury Iatrogenic Postinfectious
Chronic tension-type headache[a]	Chronic paroxysmal hemicrania	Inflammatory, such as Giant cell arteritis Sarcoidosis Behçet's syndrome
Hemicrania continua[a]	SUNCT/SUNA	Chronic CNS infection
New daily persistent headache[a]	Hypnic headache	Medication-overuse headache[a]

[a]May be complicated by medication overuse. [b]Some patients may have headache >4 h/d.

Abbreviations: CNS, central nervous system; SUNA, *s*hort-lasting *u*nilateral *n*euralgiform headache *a*ttacks with cranial autonomic symptoms; SUNCT, *s*hort-lasting *u*nilateral *n*euralgiform headache attacks with *c*onjunctival injection and *t*earing.

APPROACH TO THE PATIENT WITH CHRONIC DAILY HEADACHE

The first step in the management of patients with CDH is to diagnose any secondary headache and treat that problem **(Table 16-3)**. This can

sometimes be a challenge when the underlying cause triggers worsening of a primary headache. For patients with primary headaches, diagnosis of the headache type will guide therapy. Preventive treatments such as tricyclics, either amitriptyline or nortriptyline, at doses up to 1 mg/kg, are very useful in patients with CDH arising from migraine or tension-type headache or where the secondary cause has activated the underlying primary headache. Tricyclics are started in low doses (10–25 mg daily) and may be given 12 h before the expected time of awakening in order to avoid excessive morning sleepiness. Medicines including topiramate, valproate, propranolol, flunarizine (not available in the United States), candesartan, and the newer calcitonin gene-related peptide (CGRP) pathway monoclonal antibodies, or gepants-CGRP receptor antagonists (see **Chap. 430**) are also useful when the underlying issue is migraine.

MANAGEMENT OF MEDICALLY INTRACTABLE DISABLING PRIMARY HEADACHE

The management of medically intractable headache is difficult, although recent developments in therapy are at hand. Monoclonal antibodies to CGRP or its receptor have been reported to be effective and well tolerated in chronic migraine and are now licensed for use in clinical practice. Noninvasive neuromodulatory approaches, such as single-pulse transcranial magnetic stimulation and noninvasive vagal nerve stimulation, which appear to modulate thalamic processing or brainstem mechanisms, respectively, in migraine have been used in clinical practice with success. Noninvasive vagal nerve stimulation has also shown promise particularly in chronic cluster headache, chronic paroxysmal hemicrania, and hemicrania continua, and possibly in short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA) and short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT) **(Chap. 430)**. Other modalities are discussed in **Chap. 430**.

MEDICATION-RELATED AND MEDICATION-OVERUSE HEADACHE

Overuse of analgesic medication for headache can aggravate headache frequency, markedly impair the effect of preventive medicines, and induce a state of refractory daily or near-daily headache called *medication-overuse headache*. A proportion of patients who stop taking analgesics will experience substantial improvement in the severity and frequency of their headache. However, even after cessation of analgesic use, many patients continue to have headache, although they may feel clinically improved in some way, especially if they have been using opioids or barbiturates regularly. The residual symptoms probably represent the underlying primary headache disorder, and most commonly this issue occurs in patients prone to migraine.

Management of Medication Overuse: Outpatients

For patients who overuse analgesic medications, it is often helpful to reduce and eliminate the medications, although this approach is far from universally effective. One approach is to reduce the medication dose by 10% every 1–2 weeks. Immediate cessation of analgesic use is possible for some patients, provided there is no contraindication. Both approaches are facilitated by use of a medication diary maintained during the month or two before cessation; this helps to identify the scope of the problem. A small dose of a nonsteroidal anti-inflammatory drug (NSAID) such as naproxen, 500 mg bid, if tolerated, will help relieve residual pain as analgesic use is reduced. NSAID overuse is not usually a problem for patients with daily headache when an NSAID with a longer half-life is taken once or twice daily; however, overuse problems may develop with shorter-acting NSAIDS. Once the patient has substantially reduced analgesic use, a preventive medication should be introduced. Another widely used approach is to commence the preventive at the same time the analgesic reduction is started. It must be emphasized that *preventives may not work in the presence of analgesic overuse, particularly with opioids*. The most common cause of unresponsiveness to treatment is the use of a preventive when analgesics continue to be used regularly. For some patients, discontinuing analgesics is very difficult; often the best approach is to inform the patient that some degree of headache is inevitable during this initial period.

Management of Medication Overuse: Inpatients

Some patients will require hospitalization for detoxification. Such patients have typically failed efforts at outpatient withdrawal or have a significant medical condition, such as diabetes mellitus or epilepsy, which would complicate withdrawal as an outpatient. Following admission to the hospital, medications are withdrawn completely on the first day, in the absence of a contraindication. Antiemetics and fluids are administered as required; clonidine is used for opioid withdrawal symptoms. For acute intolerable pain during the waking hours, aspirin, 1 g IV (not approved in the United States), is useful. IM chlorpromazine can be helpful at night; patients must be adequately hydrated. Three to five days into the admission, as the effect of the withdrawn substance wears off, a course of IV dihydroergotamine (DHE) can be used. DHE, administered every 8 h for 5 consecutive days, a treatment that is not stopped short if headache settles, can induce a significant remission that allows a preventive treatment to be established. Serotonin 5-HT_3 receptor antagonists, such as ondansetron or granisetron, or the neurokinin receptor antagonist, aprepitant, may be required with

DHE to prevent significant nausea, and domperidone (not approved in the United States) orally or by suppository can be very helpful. Avoiding sedating or otherwise side effect–prone antiemetics is helpful.

NEW DAILY PERSISTENT HEADACHE

New daily persistent headache (NDPH) is a clinically distinct syndrome with important secondary causes; these are listed in **Table 16-4**.

TABLE 16-4

Differential Diagnosis of New Daily Persistent Headache

PRIMARY	SECONDARY
Migrainous-type	Subarachnoid hemorrhage
Featureless (tension-type)	Low cerebrospinal fluid (CSF) volume headache
	Raised CSF pressure headache
	Posttraumatic headache[a]
	Chronic meningitis

[a]Includes postinfectious forms.

Clinical Presentation

NDPH presents with headache on most if not all days, and the patient can clearly, and often vividly, recall the moment of onset. The headache usually begins abruptly, but onset may be more gradual; evolution over 3 days has been proposed as the upper limit for this syndrome. Patients typically recall the exact day and circumstances of the onset of headache; the new, persistent head pain does not remit. The first priority is to distinguish between a primary and a secondary cause of this syndrome. Subarachnoid hemorrhage is the most serious of the secondary causes and must be excluded either by history or appropriate investigation **(Chap. 429)**.

Secondary NDPH

Low CSF Volume Headache In these syndromes, head pain is positional: it begins when the patient sits or stands upright and resolves upon reclining. The pain, which is occipitofrontal, is usually a dull ache but may be throbbing. Patients with chronic low CSF volume headache typically present with a history of headache from one day to the next that is generally not present on waking but worsens during the day. Recumbency usually improves the headache within minutes, and it can take only minutes to an hour for the pain to return when the patient resumes an upright position.

The most common cause of headache due to persistent low CSF volume is CSF leak following LP **(Chap. S9)**. Post-LP headache usually begins within 48 h but may be delayed for up to 12 days. Its incidence is between 10% and 30%. Beverages with caffeine may provide temporary relief. Besides LP, index events may include epidural injection or a vigorous Valsalva maneuver, such as from lifting, straining, coughing, clearing the eustachian tubes in an airplane, or multiple orgasms. Spontaneous CSF leaks are well recognized, and the diagnosis should be considered whenever the headache history is typical, even when there is no obvious index event. As time passes from the index event, the postural nature may become less apparent; cases in which the index event occurred several years before the eventual diagnosis have been recognized. Symptoms appear to result from low volume rather than low pressure: although low CSF pressures, typically 0–50 mm CSF, are usually identified, a pressure as high as 140 mm CSF has been noted with a documented leak.

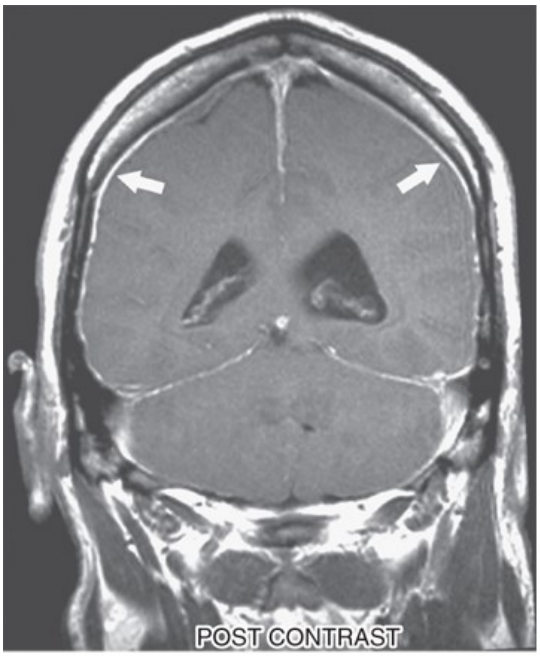
Postural orthostatic tachycardia syndrome (POTS; **Chap. 440**) can present with orthostatic headache similar to low CSF volume headache and is a

diagnosis that needs consideration in this setting.

When imaging is indicated to identify the source of a presumed leak, an MRI with gadolinium is the initial study of choice **(Fig. 16-1)**. A striking pattern of diffuse meningeal enhancement is so typical that in the appropriate clinical context the diagnosis is established. Chiari malformations may sometimes be noted on MRI; in such cases, surgery to decompress the posterior fossa is *not* indicated and usually worsens the headache. Spinal MRI with T2 weighting may reveal a leak, and spinal MRI may demonstrate spinal meningeal cysts whose role in these syndromes is yet to be elucidated. The source of CSF leakage may be identified by spinal MRI with appropriate sequences, or by CT, preferably digital subtraction, myelography. In the absence of a directly identified site of leakage, 111In-DTPA CSF studies may demonstrate early emptying of the tracer into the bladder or slow progress of tracer across the brain suggesting a CSF leak; this procedure is now only rarely employed.

FIGURE 16-1

Magnetic resonance image showing diffuse meningeal enhancement after gadolinium administration in a patient with low cerebrospinal fluid (CSF) volume headache. (*Source: JL Jameson, AS Fauci, DL Kasper, SL Hauser, DL Longo, J Loscalzo: Harrison's Principles of Internal Medicine, 20th Edition Copyright © McGraw Hill Education. All rights reserved.*)



Initial treatment for low CSF volume headache is bed rest. For patients with persistent pain, IV caffeine (500 mg in 500 mL of saline administered over 2 h) can be very effective. An electrocardiogram (ECG) to screen for arrhythmia should be performed before administration. It is reasonable to administer at least two infusions of caffeine before embarking on additional tests to identify the source of the CSF leak. Because IV caffeine is safe and can be curative, it spares many patients the need for further investigations. If unsuccessful, an abdominal binder may be helpful. If a leak can be identified, an autologous blood patch is usually curative. A blood patch is also effective for post-LP headache; in this setting, the location is empirically determined to be the site of the LP. In patients with intractable headache, oral theophylline is a useful alternative that can take some months to be effective.

Raised CSF Pressure Headache

Raised CSF pressure is well recognized as a cause of headache. Brain imaging can often reveal the cause, such as a space-occupying lesion.

Idiopathic intracranial hypertension (pseudotumor cerebri) NDPH due to raised CSF pressure can be the presenting symptom for patients with idiopathic intracranial hypertension, a disorder associated with obesity, female gender, and, on occasion, pregnancy. The syndrome can also occur without visual problems, particularly when the fundi are normal. These patients typically present with a history of generalized headache that is

present on waking and improves as the day goes on. It is generally present on awakening in the morning and is worse with recumbency. Transient visual obscurations are frequent and may occur when the headaches are most severe. The diagnosis is relatively straightforward when papilledema is present, but the possibility must be considered even in patients without funduscopic changes. Formal visual field testing should be performed even in the absence of overt ophthalmic involvement. Partial obstructions of the cerebral venous sinuses are found in a small number of cases. In addition, persistently raised intracranial pressure can trigger a syndrome of chronic migraine. Other conditions that characteristically produce headache on rising in the morning or nocturnal headache are obstructive sleep apnea or poorly controlled hypertension.

Evaluation of patients suspected to have raised CSF pressure requires brain imaging. It is most efficient to obtain an MRI, including an MR venogram, as the initial study. If there are no contraindications, the CSF pressure should be measured by LP; this should be done when the patient is symptomatic so that both the pressure and the response to removal of 20–30 mL of CSF can be determined. An elevated opening pressure and improvement in headache following removal of CSF are diagnostic in the absence of fundal changes.

Initial treatment is with acetazolamide (250–500 mg bid); the headache may improve within weeks. If ineffective, topiramate is the next treatment of choice; it has many actions that may be useful in this setting, including carbonic anhydrase inhibition, weight loss, and neuronal membrane stabilization, likely mediated via effects on phosphorylation pathways. Severely disabled patients who do not respond to medical treatment require intracranial pressure monitoring and may require shunting. If appropriate, weight loss should be encouraged.

Posttraumatic Headache

A traumatic event can trigger a headache process that lasts for many months or years after the event. The term *trauma* is used here in a very broad sense: headache can develop following an injury to the head, but it can also develop after an infectious episode, typically viral meningitis; a flulike illness; or a parasitic infection. Complaints of dizziness, vertigo, and impaired memory can accompany the headache. Symptoms may remit after several weeks or persist for months and even years after the injury. Typically, the neurologic examination is normal and CT or MRI studies are unrevealing. Chronic subdural hematoma may on occasion mimic this disorder. Posttraumatic headache may also be seen after carotid dissection and subarachnoid hemorrhage and after intracranial surgery. The underlying theme appears to be that a traumatic event involving the pain-producing meninges can trigger a headache process that lasts for many years.

Other Causes

In one series, one-third of patients with NDPH reported headache beginning after a transient flulike illness characterized by fever, neck stiffness, photophobia, and marked malaise. Evaluation typically reveals no apparent cause for the headache. There is no convincing evidence that persistent Epstein-Barr virus infection plays a role in NDPH. A complicating factor is that many patients undergo LP during the acute illness; iatrogenic low CSF volume headache must be considered in these cases.

Treatment

Treatment is largely empirical and directed at the headache phenotype. Tricyclic antidepressants, notably amitriptyline, and anticonvulsants, such as topiramate, valproate, candesartan, and gabapentin, have been used with reported benefit. The monoamine oxidase inhibitor phenelzine may also be useful in carefully selected patients. The headache usually resolves within 3–5 years, but it can be quite disabling.

PRIMARY CARE AND HEADACHE MANAGEMENT

Most patients with headache will be seen first in a primary care setting. The challenging task of the primary care physician is to identify the very few worrisome secondary headaches from the very great majority of primary and less dangerous secondary headaches **(Table 16-2)**.

Absent any warning signs, a reasonable approach is to treat when a diagnosis is established. As a general rule, the investigation should focus on identifying worrisome causes of headache or on helping the patient to gain confidence if no primary headache diagnosis can be made.

After treatment has been initiated, follow-up care is essential to identify whether progress has been made against the headache complaint. Not all headaches will respond to treatment, but, in general, worrisome headaches will progress and will be easier to identify.

When a primary care physician feels the diagnosis is a primary headache disorder, it is worth noting that >90% of patients who present to primary care

with a complaint of headache will have migraine **(Chap. 430)**.

In general, patients who do not have a clear diagnosis, have a primary headache disorder other than migraine or tension-type headache, or are unresponsive to two or more standard therapies for the considered headache type, should be considered for referral to a specialist. In a practical sense, the threshold for referral is also determined by the experience of the primary care physician in headache medicine and the availability of secondary care options.

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FURTHER READING

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