Headaches in Older Individuals: Diagnosis and Management in Primary Care

Case Studies and Commentary, Jeffrey S. Royce, MD

ABSTRACT

- **Objective:** To review the diagnosis and management of headaches in older persons.
- **Methods:** Review of the literature and illustration of concepts via case presentations.
- **Results:** Headache disorders are very common in the elderly. Serious etiologies for new-onset headache abound in this population, which necessitates brain imaging as an initial step in the workup. Primary headache disorders such as migraine change in phenotype with aging. In addition, hypnic headache, a novel primary headache, occurs in patients over 60 years of age. Medical treatment of the older individual must take into account the specific diagnosis as well as the specific characteristics of the older patient. These may include reduced medication tolerance, contraindications to medications due to comorbid conditions, and aggravation of headaches by the medications themselves. Finally, medications that relieve headaches can, with chronic and regular use, cause medication overuse headaches in patients with primary headache disorders.
- **Conclusion:** The diagnosis and management of headache disorders in the elderly is challenging. Primary care physicians should become familiar with diagnostic criteria and appropriate management for common causes of headache in elderly persons.

Headache disorders are common in the elderly, although the prevalence of headache disorders decreases with increasing age. Importantly, in older patients the etiology of the headache shifts from benign conditions to more serious causes. The prevalence of primary headache disorders such as migraine, cluster, and tension-type headache (TTH) decreases with age, while secondary headache disorders such as giant cell arteritis (GCA) and intracranial mass lesions become more prevalent. The primary headache disorder hypnic headache is unique to the population of patients older than 60 years [1].

Aging also brings special considerations in the management of headaches. Primary headache disorders such as migraine and TTH may have atypical presentations. Aura may dissipate with age or may begin to occur as an isolated phenomenon as in late-life migraine accompaniments. There is an increase in comorbid medical conditions that may limit or contraindicate treatment options. Moreover, older patients tend to have a decreased tolerance to the side effects of medication. In addition, a more extensive workup, including metabolic as well as imaging studies, is required to exclude more serious etiologies of headache [1,2].

**Epidemiology**

Recent population surveys of people over age 65 reveal a headache prevalence rate of 36.6% to 40.6% for men and 49.7% to 62.1% for women. A cross-sectional general population survey of an English district in the United Kingdom in 1999 demonstrated a 3-month headache prevalence rate of 49.7% for women and a rate of 40.6% for men. The overall 3-month prevalence of headache for the entire study population aged 18 years and older was 70.3% [3].

The 1-year prevalence of headache in the 65-and-older population residing in 3 rural villages of Italy was surveyed by Prencipe in 1993 [4]. The overall headache prevalence in the study population was 51%. Women again had a higher prevalence at 62.1% as compared with the 36.6% rate for men. The prevalence rates for individual primary headache disorders were 44.5% for TTH, 11% for migraine, and 4.4% for chronic daily headache. The 1-year prevalence of

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symptomatic headache or secondary headaches due to organic disease—either systemic or intracranial—was 2.2%. Importantly, the prevalence of symptomatic headache due to neoplasia, subdural hematomas, or stroke increased to 15.3% for individuals with new-onset headache. This observation underscores the important role of contrast-enhanced central nervous system (CNS) imaging and laboratory testing in the initial evaluation of new-onset headache in the elderly population. In this subset of the population, TTH was the most prevalent new headache at 80.6%. Migraine accounted only for 4.2% of new-onset disorders [4].

**CASE STUDY 1**

**Initial Presentation**

An 87-year-old man presents with the new onset of headaches for 6 months following peripheral revascularization surgery of the lower extremities. His wife had died 3 months earlier. The patient complained of a daily dull aching in his left temple and in his neck, which he rates a 5 on a pain scale of 1 to 10. The pain is not worsened with activity and there is no associated nausea, photophobia, or sonophobia. There are no obvious triggers of the headache. He remembers having similar headaches when he first started his career as a judge. Aspirin and hydrocodone/acetaminophen analgesics relieve the headache. The patient’s other medications include clopidogrel, esomeprazole, gemfibrozil, and cholestyramine, none of which were newly started in the past 6 months. The patient has a past medical history of gastroesophageal reflux disease, hyperlipidemia, peripheral vascular disease, coronary artery disease treated with a 3-vessel bypass, and prostate cancer. There is no family history of headaches. The review of systems is negative except for recent depression and grief. Complete physical examination including fundoscopy is normal.

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**What is the differential diagnosis in an elderly patient with new-onset daily headaches?**

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**Table 1.** Drugs That May Induce Headache or Worsen Headaches

<table>
<thead>
<tr>
<th>Drug</th>
<th>Induce Headache or Worsen Headaches</th>
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<tbody>
<tr>
<td>Acetazolamide</td>
<td>Didanosine</td>
</tr>
<tr>
<td>Amantadine</td>
<td>Dihydramole</td>
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<tr>
<td>Antihistamines</td>
<td>Dipyridamole</td>
</tr>
<tr>
<td>Barbiturates</td>
<td>Dihydroergotamine</td>
</tr>
<tr>
<td>Beta-interferon</td>
<td>Disulfiram</td>
</tr>
<tr>
<td>Bromocriptine</td>
<td>Ergotamine</td>
</tr>
<tr>
<td>Caffeine</td>
<td>Estrogens</td>
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<tr>
<td>Ca antagonists</td>
<td>Glycosides</td>
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<tr>
<td>Chloroquine</td>
<td>Griseofulvin</td>
</tr>
<tr>
<td>Cimetidine</td>
<td>Guanethidine</td>
</tr>
<tr>
<td>Clofibrate</td>
<td>Immunoglobulins</td>
</tr>
<tr>
<td>Codeine</td>
<td>Interferons</td>
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Adapted from reference 5.
such as hypercalcemia and hypothyroidism are in the differential diagnosis.

**Serious Causes of Headache in Older Patients**

**Giant cell arteritis.** The first considered diagnosis is GCA, which is a chronic vasculitis that has a predilection for the cranial branches of the arteries of the aortic arch such as the temporal and ophthalmic arteries. The condition occurs in individuals over age 50, with the mean age being 72 years. The female-to-male ratio is 3:1. The patient’s overall presentation is highly variable and the headache is not specific to GCA [7]. The headache of GCA is bitemporal half of the time and may radiate to the neck, jaws, face, and tongue. A severe throbbing character is more typical, though there are no consistent characteristics defining the headache. The onset of the headache tends to be acute; however, the duration of the headache before presenting for medical care is 2 to 3 months [7,8].

In addition to headache, systemic symptoms of fever, anemia, fatigue, anorexia, weight loss, depressed mood, and generalized muscle aches are often noted. Polymyalgia rheumatica may accompany GCA 50% of the time and is characterized by abrupt onset of morning stiffness and pain involving the neck, shoulders, and hips. Jaw claudication, which is pain in the temporomandibular joint while chewing (especially firm foods), is reported in 30% to 40% of cases and is very specific for GCA [7,8]. Visual symptoms of diplopia, visual field loss and amaurosis fugax, and transient monocular blindness (seen in 10% of individuals) herald the involvement of the arteries supplying the optic nerve, which in turn may lead to ischemia and the dreaded complication of blindness due to optic nerve neuropathy. Diplopia caused by either ischemia of the extraocular muscles or oculomotor nerve involvement is another specific sign of GCA and occurs in 2% to 14% of individuals with GCA [7,8].

On examination classically the temporal artery is tender with nodules (beading), and a decreased pulse may be appreciated. The scalp overlying the temporal and parietal regions may be tender to the touch. On fundoscopic examination, pale, swollen optic disks indicate ischemic optic neuropathy [8].

Laboratory data may reveal anemia (seen in 44% of patients). An elevated erythrocyte sedimentation rate (ESR) of 50 mm/hr or greater is one of the American College of Rheumatology’s criteria for the classification of GCA (Table 2) [9]. In addition, the C-reactive protein level is elevated. Temporal artery biopsy is mandatory to establish the diagnosis and has a sensitivity of 85%.

When GCA is a suspected and is a highly probable diagnosis, corticosteroid treatment should be started immediately in order to prevent serious ophthalmologic complications, especially irreversible visual loss. The initiation of treatment of GCA with high-dose corticosteroids should not be delayed while awaiting the results of the biopsy as the resolution of the inflammatory infiltrate occurs slowly [10]. GCA is initially treated with prednisone 60 to 80 mg daily. The dose may be tapered when symptoms remit and the ESR normalizes, generally in 2 to 4 weeks. The course of therapy with corticosteroids is usually several months [8].

**CNS neoplasms.** The prevalence of secondary headache disorders such as brain tumors and subdural hematomas rises with increasing age, as demonstrated in the Prencipe study [4]. The most common primary CNS neoplasms are of glial cell origin, such as astrocytomas and oligodendrogliomas. The next most common is meningiomas followed by pituitary adenomas. Most commonly, the origins of metastatic brain tumors are the most prevalent primary neoplasms in the population: lung and breast cancers followed by melanomas and then kidney and gastrointestinal tract cancers [8,11].

Intracranial mass lesions present with insidious and gradually progressive headaches. Headache as a presenting symptom of a CNS mass lesion occurs 20% to 50% of the time. Headache tends to be more common

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**Table 2. American College of Rheumatology Criteria for the Classification of Giant Cell Arteritis**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Description</th>
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<tbody>
<tr>
<td>Age of disease onset</td>
<td>&gt; 50 years</td>
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<tr>
<td>New headache</td>
<td></td>
</tr>
<tr>
<td>Temporal artery abnormality</td>
<td>Tenderness to palpation or decreased pulsation</td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate</td>
<td>≥ 50 mm/hr</td>
</tr>
<tr>
<td>Abnormal temporal artery biopsy</td>
<td></td>
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To establish the diagnosis, 3 of 5 criteria are needed.

Adapted from reference 9.
in infratentorial tumors, and the pain associated with the tumors tends to be occipital in location. Supratentorial tumors tend to have a more frontal distribution of the pain [8,11]. In a study by Forsyth and Posner [11] that evaluated 111 patients with brain tumors, the median age of the individuals was 56 years and headache was present in 53 of the patients (43%). TTH features were present in 77% of those with headache, and 9% had migraine-type headaches. Another 14% had mixed headache characteristics that could not be readily classified as a certain headache type. The classic brain tumor headache—severe, worse in the morning on rising, and associated with vomiting—occurred 17% of the time and mostly in individuals with increased intracranial pressure (ICP). Furthermore, headaches with increased ICP were distinctive in that they were resistant to treatment with common analgesics. In this study, patients with prior history of headaches were more likely to have a headache associated with their brain tumor. The headache was more severe and frequent than the prior headaches and associated with additional symptoms: seizures, mental status changes, hemiparesis, and other abnormal signs [11].

Subdural hematoma. Subdural hematomas occur more frequently with aging due to brain atrophy, which exposes the bridging veins of the dura to potential tearing. In addition, decreased hemostasis and an increased tendency for falls increases the subdural hematoma risk. A mild to moderate nonspecific headache is present in 80% of patients with chronic subdural hematomas. In addition patients may suffer confusion or fluctuating sensorium. The subdural space is extraparenchymal so that individuals are less likely to present with focal deficits and focal head pain [1,8].

### What laboratory investigations are required for the diagnostic workup in this patient?

The patient requires a complete blood count, chemistry screen, and measurement of ESR and thyroid-stimulating hormone (TSH) to look for metabolic as well as inflammatory causes of the headache. The patient requires an MRI with gadolinium or contrast-enhanced CT of his brain because he is over age 55 and has new-onset headache. In addition, the patient is taking anticoagulant medications and thus is at risk for intracranial bleeding.

### Further Assessment

The patient undergoes brain MRI with gadolinium, which is unremarkable. Likewise, TSH, CBC, and ESR are all normal. Furthermore, the patient notes that the headache has been abating over the past 2 weeks such that it is intermittent and at a decreased intensity of 2 to 3/10.

### What is the narrowed differential diagnosis?

**Tension-Type Headache and Cervicogenic Headache**

The differential diagnosis is reduced to TTH and cervicogenic headache (CHA). TTH is the most common headache in the elderly and increases in prevalence with increasing age. A TTH is a mild to moderate headache that is bilateral in location and dull or tightening in character and lasts for 30 minutes to 7 days. The headache is not worsened by routine activity and may in fact improve with exercise. Nausea, photophobia, and sonophobia are usually not associated features. The International Headache Society (IHS) ICDH-II diagnostic criteria for TTH are shown in Table 3.

Frequently disorders of the neck or cervical spine are confused with TTH. The pain of CHA is referred from a neck source and is perceived in the head or face.
Imaging studies display findings in the neck that may be a plausible source of the pain. In order to establish the diagnosis of CHA, there must be demonstrable evidence that the neck disorder is the causative factor [5]. Herein lies the challenge—degenerative changes are endemic in individuals over 40 years of age such that clinical features of the neck disorder such as neck pain, focal tenderness, and worsening of pain with cervical motion cannot establish the neck disorder as the cause of the headache. Diagnostic criteria for CHA most reliably then would be termination of the headache following an anesthetic block of the implicated cervical structure or its innervating nerves or surgical correction of the neck disorder [5].

**Migraine**

Although not included in this patient’s differential diagnosis, migraine is the second most prevalent primary headache in the older population after TTH [4]. A migraine is a moderately severe to severe unilateral headache that is pulsating in character and aggravated by routine activity and lasts 4 to 72 hours. In about 40% of patients, the pain may be bilateral. The headache pain is associated with nausea or vomiting and both photophobia and sonophobia. With advancing age, there is a reduction in severity, frequency, and duration of the attacks [1,8]. The IHS ICDH-II diagnostic criteria for migraine without aura are shown in Table 4.

The IHS diagnostic criteria for migraine are very specific and are most applicable to a study population. Briefer diagnostic criteria more suitable to clinical practice have been proposed. In a study that sought to establish the validity of a brief migraine screening tool (the ID Migraine screener), Lipton and colleagues found the 3 best predictors for migraine were nausea, disability (inability to function), and photophobia, whose acronym is PIN [12]. A yes answer to 2 of the 3 questions assessing for these symptoms gives an 81% probability of having migraine. A yes to all 3 portends a 93% probability of meeting IHS diagnostic criteria for migraine.

Another study that sought to abbreviate the migraine diagnostic criteria was conducted by Martin et al and involved 1529 patients from 4 practices [13]. The authors found that the criteria of nausea, photophobia, and worse with exertion had a sensitivity and specificity for the diagnosis of migraine of 82% and 86%, respectively. If nausea, photophobia, and pulsating were used, the sensitivity and specificity were 80% and 88%, respectively. Martin concludes that a patient would have a greater than 75% likelihood of having migraine if either abbreviated criteria were positive and the patient had both a normal neurologic examination and secondary headache disorders were ruled out.

Migraine is subclassified into migraine with or without aura. Aura is defined as fully reversible evolving focal neurologic symptoms that precede or accompany migraine. Between 23% and 43% of individuals who have migraine headaches have aura [14]. The most common aura symptoms are visual and consist of positive phenomena such as flashing lights and wavy lines. Negative visual phenomena of scotoma, blurring, and tunnel vision also occur and may follow the positive visual symptoms. In addition, sensory aura of hand and arm or mouth numbness or paresthesia may occur. Finally, aphasia, dysarthria, and slurred speech occur less commonly. Aura tends to lessen or disappear with age [1].

**Table 4. International Headache Society ICDH-II Diagnostic Criteria for Migraine Without Aura**

| A. At least 5 attacks fulfilling criteria B-D |
| B. Headache attacks lasting 4–72 hours (untreated or unsuccessfully treated) |
| C. Headache has at least 2 of the following characteristics: |
| 1. Unilateral location |
| 2. Pulsating quality |
| 3. Moderate or severe pain intensity |
| 4. Aggravation by or causing avoidance of routine physical activity (eg, walking or climbing stairs) |
| D. During the headache at least 1 of the following: |
| 1. Nausea and/or vomiting |
| 2. Photophobia and phonophobia |
| E. Not attributable to another disorder |

ICH = International Classification of Headache Disorders. (Adapted from reference 5.)

• **What is the preferred treatment of this patient with TTH?**

In a recent meta-analysis, tricyclic antidepressants (TCAs) were found to substantially reduce headache...
pain and decrease the headache burden in TTH [15]. The beneficial effect of treatment with the TCA increased over time. The dose of the TCA was on average 50% of maximum. The researchers also concluded that TCAs were more efficacious than selective serotonin reuptake inhibitors at preventing both TTH and migraine headache. Adverse effects were dry mouth, drowsiness, and weight gain. The adage of start low and go slow is most appropriate when using this class of medication in the elderly.

Case Resolution
The patient opted to treat the headaches with acetaminophen as needed as he felt that they were beginning to resolve. With the passage of time, the headaches abated.

CASE STUDY 2

Initial Presentation and Evaluation
A 60-year-old woman presents with an 8-month history of recurrent episodes of bilateral vision loss, numbness of the left hand, lips, and mouth but not the tongue, and a sensation of falling to the right. The episodes occur several times per month and last 15 minutes. She has a very slight pressure sensation in the left side of her head that she does not describe as painful. She has a past medical history of hypertension, hyperlipidemia, and anxiety. She denies any significant past history of headache. Her family history is positive for hypertension in her mother and her father had a stroke. Her review of systems is positive for mild bilateral hearing loss, cough, chest pain, diarrhea which follows an episode, and anxiety. Her current medications are topiramate, amitriptyline, pravastatin, lisinopril, and low-dose aspirin. Her complete physical examination including fundoscopy is entirely normal.

- **What is the differential diagnosis for the 60-year-old patient with minimal headache and neurologic symptoms?**

As in the previous case, serious conditions must be considered first. Therefore, transient ischemic attacks (TIA) due to cerebral thrombosis or embolism and cranial or carotid artery dissection are the pressing concerns. In addition, seizure disorder and hemato-

logic disorders such as polycythemia and thrombocythemia, lupus, and hyperviscosity syndrome also need to be considered. Therefore, the patient should undergo imaging of the CNS including contrast MRI of the brain and angiographic studies of her carotid arteries, vertebral arteries, and intracranial circulation. In addition, laboratory testing should include a CBC, chemistry profile, ESR, and TSH.

Headache occurs in 20% of ischemic strokes (range across 11 studies, 8%–34%). Half of the headaches associated with stroke are unilateral and focal. The severity of the headache is mild to moderate. Two-fifths of patients have nausea with the headache and about one-fourth have vomiting. Photophobia and phonophobia are seen a fourth of the time as well. The headache may be worsened by valsalva and nitroglycerine. The mean duration is 3.8 days. Interestingly, headache associated with stroke is more common in normotensive individuals, females, younger individuals with a history of migraine, and posterior circulation infarction[17,18]. Posterior circulation infarcts tend to be associated with more severe bilateral headaches, and the location of the headaches may be occipital, frontal, or both. There is no correlation between headache and stroke severity.

Headache is also associated with TIA, occurring at a frequency of 17% to 54% of the time. The mean duration of headache in TIA is 17 hours. Most TIAs associated with internal carotid, middle cerebral, or basilar artery disease last less than 15 minutes. Headache is uncommon in lacunar infarcts [16,17].

Late-life migraine accompaniments (LLMA) are recurrent episodes of visual scintillation buildup, marching paresthesias, aphasia, dysarthria, and paresis that occur without typical migraine headache pain. Individuals over the age of 40 may present with TIA features. Half of these individuals have no headache. Approximately 50% of individuals have a past history of headache. The neurologic symptoms occur as a flurry in middle life and generally run a benign course [16].

Further Evaluation
The patient underwent MRI and magnetic resonance angiography (MRA) of the brain and ultrasound of her carotid and vertebral arteries, all of which were normal. Her laboratory test results were unremarkable. She was diagnosed with LLMA after excluding...
all other serious vascular and medical disorders that cause stroke-like symptoms.

• How does one differentiate a TIA from a migraine accompaniment or from an aura?

This case demonstrates an increasing pattern of aura symptoms seen in patients over the age of 40, which is contrary to the trend seen in older patients who have migraine with aura in which aura symptoms tend to lessen. The aura characteristic that differentiates it from a TIA is its slow progression over at least 5 minutes. The aura of migraine evolves gradually and has a bimodal progression; both positive symptoms (scintillations, tingling) are followed by negative symptoms (scotoma, numbness). Scintillations do not occur in ischemia in the distribution of the middle cerebral and anterior cerebral arteries. In LLMA visual symptoms build up over 5 to 30 minutes. The march of paresthesias in LLMA occurs over 15 to 30 minutes. The only stroke to resemble this pattern is a pure sensory stroke due to a thalamic infarct [16].

• What are the therapeutic options for this patient?

The case patient has hypertension and anxiety comorbid with her LLMA. As she has a cough which may be related to the ACE inhibitor, her antihypertensive agent can be changed to a β blocker or a calcium channel blocker. Magnesium oxide 400 mg twice daily is a treatment for aura as well as migraine with aura.

Treatment in This Patient

The patient was prescribed verapamil extended-release once daily. Her topiramate was discontinued due to lack of efficacy and the potential for memory and speech impairment. Amitriptyline, low-dose aspirin, and pravastatin all were continued unchanged.

Two months later at her follow-up visit, the patient reported substantial improvement and was no longer experiencing neurologic episodes, her cough had resolved, and her blood pressure was controlled.

CASE STUDY 3

Initial Presentation and Evaluation

A 61-year-old woman presents with severe nightly frontal headaches that awaken her from sleep around 12:00 AM and continue until late morning, up to 12 hours. Initially, the headaches occurred every other night for 2 weeks and thereafter became nightly. She notes that while dreaming, the headache attack is incorporated into her dream and she thus awakens with a headache. The headache is a frontal headache that is throbbing in character, and there is neither associated photophobia nor phonophobia. She denies any cranial autonomic symptoms. She will, however, develop nausea at the end of the attack. The headaches are partially relieved by sitting up in a recliner. The patient has no antecedent history of headaches. Her past history is significant for hyperlipidemia and sleep apnea. Her family history is positive for migraine in her mother, daughter, and maternal grandfather. The patient’s physical examination is entirely normal including fundoscopy.

• What is the differential diagnosis of this older individual’s nocturnal headache?

Intracranial mass lesions are always in the differential diagnosis as well as GCA/temporal arteritis, both of which may occur at night. Cluster headache may present as isolated nocturnal attacks. (See “Cluster Headache” below.) In addition, sleep apnea may cause early morning headaches on awakening due to hypoxemia.

Hypnic Headache

Hypnic headaches are a rare primary headache disorder of patients over the age of 60. The prevalence of this disorder is unknown. In fact, patients have had the headaches for 5 ± 8 years before diagnosis is made [19,20]. The mean age of onset is 60 ± 10 years. The female-to-male ratio is 3:1. The headache character tends to be dull 55% to 57% of the time and throbbing 40% of the time. The headache is located bilaterally 60% of the time. The intensity of the pain varies from moderate (56%–60%) to severe (37%–39%). The average pain attack duration is about 1 to 2 hours and may range from 15 to 500 minutes. As for attack frequency, 70% of patients experience 4 or
more attacks per week and 50% experience daily attacks [19,20].

The headache pain awakens the patient from sleep and is often incorporated into the patient’s dreams. Some patients with hypnic headache report that sitting up relieves or prevents the headache. Nausea is the most common associated feature, occurring in 21% of patients; photophobia is the second most common associated feature, occurring only 5% to 15% of the time. Ptosis and lacrimation are seen in 2% and 6% of case, respectively [20].

Further Evaluation

Laboratory testing reveals a normal ESR, CBC, chemistry profile, and TSH level. The patient also undergoes brain MRI, which demonstrates an incidental 7- to 8-mm focal hyperintensity within the left occipital lobe consistent with a cavernous angioma. A brain MRA scan is entirely normal.

• What are the treatment options for this patient?

Hypnic headaches are most often responsive to lithium carbonate at a dose of 300 to 600 mg at bedtime. Side effects include diarrhea, tremor, polydipsia, and polyuria. Thyroid and renal function should be evaluated at initiation of therapy and periodically thereafter. Secondary treatment options are caffeine 40 to 60 mg at bedtime and indomethacin 25 to 75 mg at bedtime. These 2 treatments are associated with frequent side effects, especially at bedtime and especially in the elderly. Antidepressant medication and β blocker therapy are not efficacious [19,21]. The case patient was started on lithium carbonate 300 mg at bedtime and was headache free at the follow-up visit.

Cluster Headache

Another often nocturnal primary headache that may be seen in older individuals is cluster headache. This headache disorder may occur for the first time in 10% of cluster patients after the age of 60 years or recur after many years of remission. The mean age of onset, however, is 28 years [22]. Cluster headaches belong to the class of short-lasting autonomic cephalgias. The attacks are severe in intensity, unilateral, and periorbital, orbital, or temporal in location and last 15 minutes to 3 hours, though most attacks last 45 to 90 minutes. They are associated with cranial autonomic features of ipsilateral lacrimation, conjunctival injection, nasal congestion, or rhinorrhea. In addition many individuals become restless or agitated unlike migraine patients, who prefer to lie still and quiet so as not to aggravate their headache. The cluster attacks occur with clock-like periodicity, with 75% of the attacks occurring between 9 PM and 10 AM. Cluster bouts generally run for 2 to 12 weeks, with a seasonal occurrence during the spring and the fall [22].

As with all headaches occurring de novo after age 55, the new-onset cluster headache patient must undergo CNS imaging, as pituitary tumors or posterior fossa lesions may present as a cluster headache attack. In addition the differential includes acute angle-closure glaucoma, GCA, and carotid dissection.

Acute cluster headache is treated with high-concentration oxygen (100%) administered at a high flow rate of 7 to 15 L/min through a non-rebreather mask for 15 to 20 minutes until the headache resolves. It is important to deliver the oxygen by mask as nasal cannula is entirely ineffective. Sumatriptan is also effective, but only as the intranasal or subcutaneous preparations. Triptans must be used cautiously in the older patient and are contraindicated for the patient with coronary artery disease, peripheral vascular disease, and cerebrovascular disease as they may cause stroke or heart attack. Prophylaxis is accomplished most effectively with verapamil; lithium carbonate and topiramate are secondary options. Again caution must be exercised when using these medications in older patients due to cognitive side effects. Prednisone may be used initially as transition therapy to begin control of the cluster bouts while the verapamil is being initiated [23].

Trigeminal Neuralgia

Trigeminal neuralgia (TN) may be confused with cluster headache as the attacks are of short duration and unilateral. Unlike in cluster headache, the average age of onset is later in life, in the sixth and seventh decades. Approximately 90% of the cases occur after 40 years of age, and the female-to-male ratio is 3:2. TN is a cranial neuralgia that causes unilateral face pain in the distribution of the maxillary and mandibular divisions of the the trigeminal nerve, unlike
the ophthalmic branch distribution of the pain of the cluster headache. The pain is neuropathic and as such its character is intermittent and electrical sharp or stabbing. The pain may be precipitated by stimulation of trigger areas such as the nasolabial fold or the chin. It occurs in very brief paroxysms, lasting seconds to 2 minutes followed by a period of refractoriness to triggering [5,8].

In most cases the etiology of the neuralgia is idiopathic and is denoted as classical TN. Younger individuals with bilateral face pain may have multiple sclerosis. Individuals with sensory defects in the affected trigeminal nerve division or lack of a refractory period may have a vascular loop or neoplasm such as an acoustic neuroma compressing the nerve and are denoted as having symptomatic TN. Therefore, contrast MRI of the brain with special attention to the internal auditory canal and cerebellopontine angle is in order.

The treatment of choice for classical TN is carbamazepine, which delivers pain relief in up to 90% of patients. Secondary choices are clonazepam, valproic acid, baclofen, gabapentin and topiramate [27]. Trigeminal nerve ablative procedures and neurostimulation may be used in medication-refractory cases. Surgical decompression is the treatment of choice for symptomatic TN caused by a vascular loop at the trigeminal nerve’s entry zone into the pons.

**CASE STUDY 4**

**Initial Presentation and Evaluation**

A 66-year-old woman presents with daily headaches for 6 months. The headaches are frontal and periorbital in location and are rated moderate in intensity but become severe twice per day, worsening with activity and eating. The pain radiates to her left arm. The worst headache pain is associated with nausea, photophobia, and sonophobia. She denies any recent head trauma. Recently, she was transported to the emergency department for a severe attack. En route to the hospital the patient was treated with oxygen and nitroglycerine, which quickly relieved her headache. She has smoked 1 to ½ packs of cigarettes per day for 43 years. Her review of systems is positive for sweats, postnasal drainage, cough productive of clear phlegm, constipation, lumbar back pain, hot flashes, insomnia, and anxiety. The patient’s past medical history is significant for hypertension, carotid stenosis, and myasthenia gravis. She recalls no family history of headaches. The patient’s medications are lisinopril, metoprolol, verapamil, azathioprine, amantadine, and low-dose aspirin. The patient’s physical examination is remarkable for a right carotid bruit.

**What is the differential diagnosis for this 66-year-old with 6 months of daily headaches?**

The differential includes intracranial mass lesions, chronic migraine, chronic subdural hematoma, GCA, hypothyroidism, medication-induced headache, particularly from amantadine, and cardiac cephalgia. Migraine first appearing after age 50 is uncommon, with a prevalence rate around 2% to 3%. The patient has multiple risk factors for coronary artery disease, including smoking, hypertension, and established atherosclerosis in the carotid arterial system. Notably, nitroglycerine and oxygen relieved the headache, which is the characteristic response of angina to these therapies. The only headache disorder that is oxygen responsive at very high concentrations and flow rate is cluster headache. However, nitroglycerine aggravates and triggers cluster attacks and migraine headache attacks. In addition chronic obstructive pulmonary disease with hypercapnia can cause headaches.

**What are the next steps in the workup?**

The workup here takes a 2-pronged approach. The first approach for headache in the patient over age 50 is to rule out intracranial mass lesions with contrast imaging studies, preferably MRI and MRA. The second approach is to assess the patient for coronary artery ischemia with radionuclide myocardial perfusion studies. The TSH level and the ESR are needed to rule out hypothyroidism and GCA, respectively.

**Further Evaluation and Treatment**

The patient has a normal TSH and ESR. Gadolinium-enhanced MRI of her brain shows no intracranial lesion and brain MRA is negative as well. However, her stress radionuclide myocardial perfusion imaging study is strongly positive for ischemia.
in the left anterior descending artery (LAD) distribution. At coronary angiography she is found to have a significant LAD lesion and undergoes angioplasty. Following the angioplasty her headaches resolve.

- **What is the cause of this patient’s headache?**

The patient’s diagnosis is cardiac cephalgia, which is an anginal headache. The headache of cardiac cephalgia may be severe and aggravated by exertion. The headache develops along with the myocardial ischemia and is associated with nausea in 30% of the cases reported in the literature. There is neither a standard character nor location of the pain. Chest pain, arm pain, and epigastric pain are present in half of the reported cases. The pathophysiology of this headache is yet to be elucidated [24].

In addition to cardiac cephalgia, this patient would fulfill the diagnosis of chronic daily headache, which had a prevalence of 4.4% in the over-65 group in Prencipe’s study [4]. Due to the regular and frequent use of medications, both prescribed and over the counter, in the older patient, medication overuse headaches and medication-induced headaches are common and present as chronic daily headaches. It is important to review with the older patient who has daily headaches all medicines prescribed and otherwise and discontinue all nonessential ones.

In addition it is common for existing migraines to worsen due to medications prescribed for other conditions. Vasodilators such as nitroglycerine and dipyridamole and antihypertensives such as nifedipine are classic examples. It is important to remember that elderly patients have reduced tolerance to medication and are more susceptible to their side effects. Again nonessential medications should be discontinued and substitution of a medication not likely to cause headache should be considered. Medications which cause headache are listed in Table 1 [5].

**CASE STUDY 5**

**Initial Presentation and Evaluation**

A 56-year-old female presents with 15 years of moderately severe daily headaches. The headaches are bitemporal and feel like a band around her head. Occasionally the headaches are severe and vise-like. The pain radiates to her shoulders and neck and worsens with activity. She has no associated photophobia, sonophobia, or nausea. Fifteen years ago she had a week of very severe headaches which required her to be at bed rest. Since that time she has taken hydrocodone/acetaminophen 5/500 four times daily to treat the headache. Her headaches have not changed in severity, frequency, or form for years. Her review of systems is positive for night sweats, sinus trouble, palpitations, fatigue, and insomnia. Her past medical history is significant for depression, hyperlipidemia, insomnia, and vasomotor symptoms of menopause and tension headaches. She has had normal thyroid studies in the past. Her family history is negative for headaches. The patient’s medications are fexofenadine, acrivastine/pseudoephedrine, azelastine nasal spray, escitalopram, zolpidem CR, atorvastatin, estradiol, medroxyprogesterone, hydrocodone/acetaminophen, and carisoprodol. The patient’s physical examination is entirely normal.

- **What is the differential diagnosis for this patient with daily headaches for 15 years?**

The differential diagnosis of long-standing moderately severe daily headaches includes chronic TTH, new daily persistent headache, medication-induced headache, and medication overuse headache. It is unlikely that serious intracranial lesions would continue unchanged for 15 years with a normal physical examination. Her headache started at approximately age 41 years, so GCA is unlikely.

The patient presented here has medication overuse headache and a past history of TTH. Medication overuse headaches are common in all age-groups of individuals who suffer headaches. All medications that relieve headache can cause medication overuse headaches. The medications used to treat migraines, ergotamine (classically), triptans, opioids, combination analgesics containing butalbital, aspirin, and caffeine as well as simple analgesics such as acetaminophen and NSAIDS can all cause medication overuse headache [5].

The character, location, severity and associated features of medication overuse headaches vary considerably from patient to patient. The headache disorder is defined by its persistence and frequency. Medication
overuse headaches in general are mild to moderate in severity and have a diffuse pressure/pressing quality and tend to be bilateral in location. The headaches are associated with scalp tenderness and suboccipital triangle tenderness. The headache is provoked by slight physical or mental activity. The headaches tend to be worse upon awakening as the previous dose of offending medication is at the end of its effective dosing interval [25]. This type of headache was formerly known as rebound headache. The headache either resolves or returns to its previous pattern within 2 months after cessation of the causative medication. Importantly, preventive medications are mostly ineffective during medication overuse [5].

**What is the treatment strategy for this patient?**

First and foremost the patient must have the hydrocodone compound withdrawn or tapered until stopped. Most headache specialists would begin preventive medications even though the efficacy is reduced due to the medication overuse. In addition acute headache abortive medications less likely to contribute to rebound are necessary for headache exacerbations (Table 5) [26].

**Treatment**

The patient is started on gabapentin 100 mg 3 times daily. Orphenadrine 100 mg twice daily combined with chlorpromazine 25 mg 3 times daily as needed is prescribed for headache exacerbations. Her hydrocodone/acetaminophen and carisoprodol are discontinued. At her follow up visit 2 months later, she is having only 3 mild TTH episodes per month.

**SUMMARY**

Headache disorders occur very commonly in older patients, with a prevalence rate averaging about 50% of woman and 30% of men over the age of 65 years. Serious etiologies for new-onset headache abound in older patients, which necessitates brain imaging as an initial step in the workup. Primary headache disorders such as migraine change in phenotype with aging. In addition hypnic headache, a novel primary headache, occurs in the older patient over 60 years of age. Medication treatment of the older individual must take into account the specific diagnosis as well as the specific characteristics of the older patient. These may include reduced medication tolerance, contraindications to medications due to comorbid conditions, and aggravation of headaches by the medications themselves. Finally, medications which relieve headaches can, with chronic and regular use, cause medication overuse headaches in patients with primary headache disorders.

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**REFERENCES**

2. Edmeads J. Headaches in older people, How are they different in this age-group? Postgrad Med 1997;101:91–100.

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**Table 5. Medicines to Use Acutely That Do Not Cause Medication Overuse Headache**

<table>
<thead>
<tr>
<th>Medicines to Use Acutely That Do Not Cause Medication Overuse Headache</th>
</tr>
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<tbody>
<tr>
<td>Hydroxyzine</td>
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<tr>
<td>Baclofen</td>
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<tr>
<td>Tizanidine</td>
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<tr>
<td>NSAIDs</td>
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<tr>
<td>Neuroleptics</td>
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<tr>
<td>Benzodiazepines</td>
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<tr>
<td>Various muscle relaxants, including metaxalone and methocarbamol (which have a central effect)</td>
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</tbody>
</table>

Adapted from reference 26.

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