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HEADACHE (RB HALKER, SECTION EDITOR)

Headaches of the Elderly

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Abstract The prevalence of headache decreases in elderly age groups; however, headache remains a significant issue with unique diagnostic and therapeutic considerations in this population. While primary headache disorders such as migraine and tension-type headache still occur in the majority of cases, secondary headaches are more common with advancing age. Additionally, several rare primary headache disorders, such as hypnic headache and primary cough headache, occur more frequently in an elderly population and have distinct treatments. In this review, we provide an updated overview of the common, concerning, and unique headache disorders affecting the elderly.

Keywords Headache · Elderly · Geriatric · Migraine · Aura without headache · Hypnic headache · Primary cough headache · Sleep apnea headache · Giant cell arteritis · Cardiac cephalalgia · Subacute glaucoma · Medication overuse headache · Trigeminal neuralgia · Post-herpetic trigeminal neuropathy

Introduction

The overall prevalence of headache decreases in the elderly; however, despite this decrease, headache remains a substantial issue for those over 60 years old [1]. The prevalence of frequent headache, defined as more than two headaches per month, affects approximately 17 % of the elderly [2, 3]. Half of elderly patients who presented to a tertiary headache center were referred for an evaluation of a new headache [4•]. In one study, a daily headache was present in 3.9 % of a cohort of elderly individuals [5]. The majority of the headaches in the elderly are primary disorders (66 %) [6] (Fig. 1). However, headaches from serious causes are more common in those over age 65 and can comprise up to 15 % of new-onset headaches, significantly increased from an estimated 1.6 % for those under age 65 [7]. The headache types, presentation, and treatment options also change with advancing age. The term elderly is defined differently depending on region or culture; however, for the purpose of this review, the World Health Organization definition of age ≥60 years will be used [8]. This review provides an updated overview of the prevalent or concerning headaches types, including primary headache disorders, secondary headache disorders, and neuralgias facing the elderly.

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Primary Headaches

Migraine

Migraine remains the second most common primary headache diagnosis, behind tension-type headache, even among the elderly. A quarter of elderly patients presenting for tertiary headache referral are seen for an International Classification of Headache Disorders (ICHD) 2 primary diagnosis of migraine [4•]. While migraine may begin to remit in the late fifth to sixth decade [9, 10], the annual prevalence of migraine over 65 years of age is still approximately 10 % [3, 11]. New-onset migraine at age over 60 is said to be rare and should prompt an evaluation for a secondary cause [7, 11]. However, new-onset migraine in the fifth decade is not rare; in one study, 19 % of

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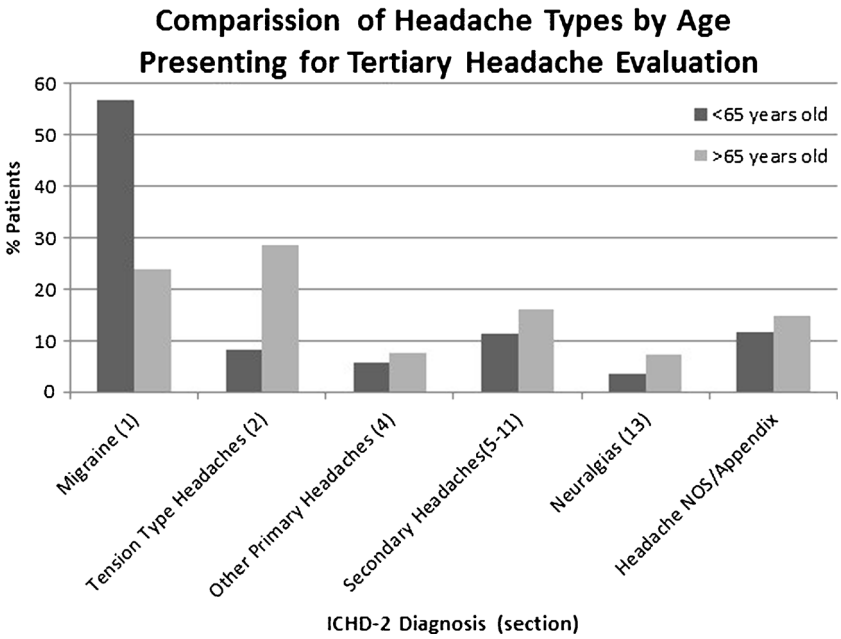

Fig. 1 Comparison of headache types by age presenting for tertiary headache evaluation. Adapted from Ruiz et al. [4•]

women were noted to have onset of migraine without aura after age 50 [12]. Migraine phenotype does change with time and is more likely to be described as bilateral or global with decreasing associated features including photophobia, phonophobia, nausea, and vomiting [13]. Elderly patients may note increase in associated neck pain, bilateral rhinorrhea, and lacrimation, but will often have better response to medication, including more mild headache days and greater ability to function during the attack [14]. This change in phenotype may be in part responsible for the increase in diagnosis as tension-type headache as compared to migraine seen in a recent study [4•] (Fig. 1).

While the treatment approaches to migraine in the elderly are similar to those in the general population, care must be taken to select acute and preventative medications considering comorbidities. Unfortunately, most studies of migraine treatment exclude patients over 65, contributing to a deficit of published data in this population [15]. Vasoactive substances, such as triptans or dihydroergotamine, should not be prescribed to patients with known cardiovascular disease including stroke, or multiple vascular risk factors. However, in individuals that are at low cardiovascular risk, despite age, triptans should still be considered for acute treatment [16, 17•]. Extra caution must be taken regarding side effect profile, particularly cognitive and autonomic considerations, when selecting migraine preventative medications, but this should not preclude their general use [11].

Aura Without Headache

ICHD-3beta defines typical aura without headache as an aura consisting of visual, sensory, and/or speech language symptoms that are fully reversible meeting at least two of four typical migraine aura characteristics [18••]. A large collection of cases of transient spells closely resembling migraine auras, often without headache, occurring in the first time in patients over the age of 45 was extensively described by the landmark paper by C Miller Fisher [19]. Originally described as "late-life migraine accompaniments" of the 205 cases, 36 % were >60 years of age [20]. Various auras were described, but among the most common among an elderly population were visual aura followed by paresthesias, aphasia, dysarthria, and hemiplegia [21, 22]. While often described as a benign TIA mimic, recent reports have suggested that hemosiderin deposition may precipitate cortical spreading depression as a potential cause for these attacks. In two different series, four patients were found to have acute convexity subarachnoid hemorrhage associated with these aura-like symptoms [23, 24]. New-onset aura-like symptoms in an elderly patient should prompt evaluation for secondary causes including TIA, seizure, and intracranial hemorrhage [25•] (Table 1).

Hypnic Headache

Hypnic headache is a rare primary headache that occurs primarily on average at age 60 [26•, 27]. It is estimated that, while rare, it may comprise 1.7 % of elderly patients presenting for tertiary headache referral [4•]. The ICHD-3beta describes hypnic headache (HH) as a frequently occurring headache developing only during sleep, causing awakening and lasting up to 4 h without other characteristics [18••]. Previously described as "alarm clock headache," it is classically a dull, minor to moderate, bilateral headache lasting several hours that awakens the patient from sleep with the

Table 1 Typical clinical features of late-life migraine accompaniment

Visual symptoms (e.g., scintillating scotomas) over 15 to 60 min
Gradual migration of scintillation with involvement of both visual fields
March of paresthesias, often hand and face over 20 to 30 min
Progression of one accompaniment to another or from one body part to another
Occurrence of ≥2 similar spells
Headache in association with spells
Average duration of spell 15 to 30 min
Occurrence in 5th to 6th decade with identical spells over 5 to 10 years
Benign course with exclusion of secondary causes

Adapted from Vongvaivanich et al. [25•]

absence of migrainous and cranial autonomic features. A recent review of the literature examined the characteristics of published cases and found that HH may differ in several significant ways as compared to original description and current proposed criteria. The review noted that the HH does occasionally occur with less prominent cranial autonomic features (2–7 %), is most often sharp/stabbing/burning (68 %) as compared to dull (26 %), and is of moderate to severe intensity (94 %). Additionally, the review noted that while overt restlessness was not present, which would suggest a trigeminal autonomic cephalalgia, patients did often report initiating a motor activity following an attack (97 %) [26•].

HH-like presentation has been reported secondary to tumors of the posterior fossa and pituitary tumors. In addition, nocturnal arterial hypertension, angiotensin-converting enzyme inhibitor medications, and one insolated case of obstructive sleep apnea have also been reported [26•]. An MRI of the brain, an evaluation for nocturnal hypertension, and a review of medications should be considered as part of the evaluation for HH. In addition, other primary headache disorders that have a nocturnal predominance, such as cluster headache, should be considered. Treatment recommendations are based on limited evidence but most prominently recommend nocturnal caffeine, 60 mg or approximately one cup of coffee, as an acute treatment. For prevention, regular caffeine before bed, lithium, and indomethacin have all been described as being effective [27].

Primary Cough Headache

Primary cough or Valsalva maneuver headache is another rare primary headache disorder that occurs with a mean age above 60 [28, 29]. It is defined as a headache that is precipitated by coughing or other Valsalva maneuver, but not exercise, in the absence of a secondary cause. The headache is described as moderate to severe, explosive, and brief (<30 s) with a dull pain that can last up to 2 h [18••]. Often, symptoms of an upper respiratory tract infection precede the onset. The presence of a cough-induced headache should compel evaluation for a potential serious secondary cause. Symptomatic Chiari type 1 malformations are the most common secondary cause; however, in an older population, an intracranial tumor, particularly of the posterior fossa, should be considered. Other potential secondary causes in the literature include unruptured intracranial aneurysms, dissection, carotid/vertebrobasilar stenosis, acute sphenoid sinusitis, CSF volume depletion, and subdural hematoma [29, 30]. No consistent headache profile, including response to indomethacin, can differentiate between primary and secondary cough headache, and brain and vascular imaging is highly recommended. Despite the potential for a serious secondary cause, most cases of cough headache in an older population are primary in nature, with secondary causes being more common at younger ages [29].

The natural history in primary cough headache is still not well understood. Those that have initial response to treatment (81 %) were pain free at 6 months without recurrence. Those that were treatment resistant were more likely to have persistent pain; however, ~42 % of these patients spontaneously remitted from 6 months to 2 years [29]. The treatment of choice is indomethacin. Therapeutic lumbar puncture and cerebrospinal fluid drainage has also been demonstrated to be effective [28, 29].

Secondary Headaches

Sleep Apnea Headache

Sleep apnea headache is defined as a morning headache, usually bilateral, lasting less than 4 h, occurring with the diagnosis and improving with the treatment of sleep apnea. Sleep apnea is defined as having an apnea/hypopnea index (AHI) of ≥5 [18••]. While sleep apnea is considered to have a peak incidence in middle-aged adulthood, the prevalence continues to increase with age [31]. In a general Norwegian population, an AHI of ≥5 (indicating sleep apnea) was most prevalent in the sixth decade of life (36.1 %), the oldest age group included in that study [32•]. Sleep apnea headache occurs in approximately 11.8 % of those with obstructive sleep apnea (OSA), with earlier studies suggesting a higher prevalence ranging from 15.2 to 18 % [33]. Although controversial, some recent studies suggest the severity of sleep apnea does not correlate with an increased prevalence of sleep apnea headache. Additionally, approximately 5 % of patients in a Norwegian general population had morning headache but did not have sleep apnea [32•].

The pathophysiology of sleep apnea headache remains unclear but is not solely related to oxygen desaturation and likely has additional factors [33]. A prior history of a primary headache disorder is more common in those with sleep apnea headache compared to those with OSA who do not develop headache. Treatment includes adequately treating the sleep

apnea. In one observational study, treatment with nasal CPAP provided headache resolution in 90 % of patients [34]. If there is no response to treatment, other early-morning primary headache disorders, such as hypnic headache, cluster headache, and caffeine withdrawal headache, should be considered. Other secondary causes of headache should also be excluded including conditions that may cause increased intracranial pressure which can be worsened by periods in the supine position.

Headache Attributed to Giant Cell Arteritis

Headache is the most common symptom experienced by patients with giant cell arteritis (GCA) reported in 73 %, and in 35 %, it is the presenting symptom [35]. GCA, previously referred to as temporal arteritis, should always be considered as a possible secondary cause of headache in those presenting with new or changed headache over age 50 [36]. The incidence of GCA increases progressively with age. Beginning in the age group 50–59, the incidence is 1.4 per 100,000, increasing to 10.7 per 100,00 in ages 60–69 and may be as high as 1100 per 100,00 in patients 85 years of age and above [37]. GCA is a medium- and large-vessel vasculitis that predominates, but is not isolated to, the cranial arteries. Unrecognized GCA can cause blindness from posterior ciliary artery involvement leading to anterior ischemic optic neuropathy. Additionally, GCA can cause a variety of other complications including other cranial neuropathies, large artery stenosis, aortic dissection/aneurysm, and rarely stroke [36]. GCA is also associated with polymyalgia rheumatica in approximately half of cases [38].

The diagnosis of GCA is guided by the American College of Rheumatology criteria which require three of five primary features to be present. This includes age 50 years or older, a new headache, a clinical temporal artery abnormality, an elevated ESR of 50 mm/h (may be age dependent), and/or an abnormal temporal artery biopsy [39]. The gold standard for diagnosis is the temporary artery biopsy which should be considered in all suspected cases. In addition to a thorough history, including the presence of jaw claudication and a temporal and extra-temporal vascular examination [40], various imaging modalities may also be helpful in the diagnosis. A recent review of color Doppler ultrasonography demonstrated a high specificity for GCA (91 %) when perivascular hypoechoic abnormalities (halo sign) were present in the temporal artery and large vessels. However, this was balanced with a low sensitivity (68 %) [41]. Contrast-enhanced T1-weighted MR images may also be helpful; in a recent prospective study, MR imaging had a diagnostic sensitivity of 78.4 % and specificity of 90.4 % with yield decreasing after 5 days of corticosteroids [42].

If GCA is suspected, patients should be treated promptly. Confirmatory testing, such as biopsy, should be arranged early (ideally within the first 7 days) into treatment when possible to increase diagnostic yield; however, a biopsy should not delay treatment. The mainstay of treatment remains corticosteroids. Recent consensus guidelines on management have been recommended by the European League Against Rheumatism and emphasize the immediate need for corticosteroid treatment [43].

Cardiac Cephalalgia

Cardiac cephalalgia is defined as a “migraine-like” headache that usually, but not always, is precipitated by exertion, occurring during an episode of myocardial ischemia. In contrast to other headache disorders, it is relieved by nitroglycerine [18••]. It is a rare secondary headache disorder, with less than 50 cases reported in the literature, but up to a nearly third of those cases presented with headache as their sole manifestation of myocardial ischemia [44]. In one literature review, the mean age of presentation was 62.5(±12.7)years of age with headache being the singular presenting manifestation more often with increasing age [45]. The headache is often heterogenous and may occur in any area of the head and may be unilateral or bilateral. The pain is almost always severe and a thunderclap presentation has been described. Additional features such as photophobia, phonophobia, and nausea occur in approximately a third. Usually, the headache occurs immediately upon exertion, but 33 % of reported cases have occurred at rest. Baseline cardiac tests may potentially be normal and abnormalities may only be apparent upon stress testing [44]. The headache is usually responsive to nitroderivatives, a unique feature among headache disorders and which typically aggravates migraine. In all reported cases, the headache resolves after revascularization or treatment of myocardial ischemia but may recur upon coronary restenosis [45, 46]. Although rare, in older patients presenting with exertional headache, especially with additional cardiovascular risk factors, evaluation for myocardial ischemia should be considered.

Headache Attributed to Subacute Glaucoma

Acute angle closure glaucoma is an urgent sight-threatening condition due to an acute rise in intraocular pressure secondary to obstruction of the aqueous outflow at the trabecular meshwork. It classically presents with acute unilateral ocular pain, eye redness, visual change (often seeing “halos around lights”), and a mid-dilated unreactive pupil but can be described initially by a patient as a unilateral severe headache [47]. However, a more insidious presentation of elevated intraocular pressure is subacute angle closure glaucoma (SACG), an intermittent form of glaucoma with recurrent episodes of elevated intraocular pressure lasting minutes to hours. Unrecognized SACG can lead to gradual optic nerve

damage and visual loss. SACG will most often present as mild to moderate eye pain, with visual blurring often occurring in dim light [48]. However, the condition may instead present with headache as a main or sole symptom of these attacks of elevated intraocular pressure. A recent case review of cases of SACG with headache as a primary presenting symptom noted a mean age of presentation of 60 years. The headache can be described as frontal, unilateral, or rarely diffuse. It may occur with nausea. It is typically nonpulsatile and without photophobia. The length of the headache is minutes to several hours, but less than 4 h. It may occur several times a month or as frequent as daily. In one review, there was an average time of 2.6 years from presentation to diagnosis for which two thirds were misdiagnosed as migraine despite not meeting criteria. Visual field loss was present in those who had symptoms longer (average 3.6 years). All patients had improvement or resolution of their headache with laser peripheral iridotomy [49, 50]. In patients presenting with headaches of a few hours presenting in dim light conditions, especially with visual blurring or halos, referral for evaluation of elevated intraocular pressure including gonioscopy is warranted.

Medication Overuse Headache

Medication overuse headache (MOH) is currently defined as a headache occurring 15 days or more per month as a consequence of regular acute headache medication use for 10 to 15 days per month (depending on the medication) for at least 3 months. By definition, there should be improvement or potential resolution after stopping the medication [18••]. MOH is prevalent across the general population occurring from 0.5 to 2.6 % [51•]. Prevalence in an elderly population is no exception, with prevalence ranging from 1.0 to 1.7 % in those 65 years or older. Similar to the general population, medication overuse for headache in the elderly is a significant risk factor for developing a daily headache as well as transforming an episodic primary headache disorder, commonly episodic migraine, to a chronic pattern [52, 53]. Elderly patients with a chronic daily headache, such as chronic migraine, are at higher risk for severe functional impairment and depression. Despite this potential disability, in one Chinese study, only one quarter of patients with daily headache consulted a physician for evaluation [53]. When evaluating an elderly patient with frequent headaches, a thorough history of prescription and nonprescription medications should be obtained. Simply withdrawing the overused medication may result in significant headache improvement at 2 months in approx. 50 % of patients [54] and may result in improved response to other headache treatments as well.

Headache Attributed to Cranial or Cervical Vascular Disorders and Nonvascular Intracranial Disorders

Headache attributed to cranial or cervical vascular disorders and headache attributed to nonvascular intracranial disorders are both consecutive sections of the ICHD-3beta that comprise many of the worrisome secondary causes of headache including subarachnoid hemorrhage, intracranial hemorrhage, ischemic stroke, nontraumatic subdural hematoma, infection, and intracranial neoplasm [18••]. Given that the prevalence of all these conditions increases with age and many may present with headache as a prominent symptom, it is prudent that a worrisome intracranial disorder be considered in the elderly patient. In one review of patients presenting to a hospital for headache, the risk of a serious secondary cause increased tenfold in those age ≥65 [7]. Worrisome features concerning for a sinister secondary cause include the following "red flags": age over 50, "worst headache," systemic signs or symptoms (fevers, chills, weight loss, etc.), neurologic signs or symptoms, thunderclap onset (headache increasing to maximum intensity in less than 1 min), sudden headache pattern change or new headache, or headache precipitated by change in position or Valsalva [55] (Table 2). In a review of cases of sudden death that presented with a chief complaint of headache, consistent red flags included age >50 (55 % of cases), thunderclap headache, worst headache, and history of seizure/collapse/loss of consciousness. The majority of the all-age deaths (60 %) resulted from vascular events (aneurysm rupture, intracranial hemorrhage, arteriovenous malformation, GCA, and dissection) with the remainder from tumor (17 %), meningitis (6 %), and other (16 %) [56•]. If red flags are present, in addition to a full neurologic evaluation, urgent neuroimaging should be considered. Head CT may initially be appropriate in an emergent setting; however, if available and patient is otherwise stable, MRI is preferred. Vascular imaging and lumbar puncture should also be considered acutely, especially in cases of thunder clap onset. Although often benign, the incidence of a worrisome secondary cause for headache is increased in the elderly and should receive careful attention especially in the presence of the above red flags.

Table 2 Headache "red flags" for secondary causes

Systemic signs or symptoms	Fevers, chills, myalgias, weight loss
Neurologic signs or symptoms	Focal or mental status changes
Onset—sudden	Sudden or thunderclap presentation
Onset—age	Onset over 50
Pattern change	If prior history
Progressive	
Precipitated by Valsalva or position change	
Papilledema	

Adapted from Dodick [55]

Cervicogenic Headache

Cervicogenic headache is a controversial secondary headache disorder defined as a headache caused by a disorder of any component of the cervical spine with or without accompanying neck pain [18••]. Despite the increase in cervical degenerative disease in the elderly, the overall incidence of cervicogenic headache in one population review is 27.8 years of age. However, in this study, subjects over 65 years of age were excluded [57]. It remains to be demonstrated that cervicogenic headache is indeed more prevalent in an elderly population. It is possible that the proposed secondary headache disorders *headache attributed to upper cervical radiculopathy* and *headache attributed to cervical myofascial pain* may show an increased incidence in an elderly population and is an area open for additional study. In selected cases with a headache with temporal relationship to a potential provoking disorder of the cervical spine, diagnostic and therapeutic high cervical blocks may be considered. Elderly patients with posterior predominant head pain, especially with paroxysmal attacks of sharp or throbbing pain and tenderness to palpation along the greater, lesser, and/or third occipital nerves, may have occipital neuralgia [18••]. Occipital neuralgia has been noted to occur in approximately 1.5 % of elderly patients presenting for tertiary headache evaluation [4]. For suspected occipital neuralgia, a peripheral occipital nerve block should be considered which can be both diagnostic and therapeutic.

Neuralgias

Trigeminal Neuralgia

Trigeminal neuralgia (TN) is a short-lasting, paroxysmal, severe shooting/stabbing/shock-like unilateral pain occurring primarily in the distribution along the second or third divisions of the trigeminal nerve. Attacks are often precipitated by innocuous tactile stimuli to the affected side of the face. TN is currently divided into two general classifications. The first is classical TN which is either idiopathic or due to neurovascular compression, most frequently by the superior cerebellar artery. The second is painful trigeminal neuropathy which is due to a structural lesion such as demyelination from multiple sclerosis, space-occupying mass, or herpes zoster (acute or post-herpetic neuralgia) [18••]. While the onset of TN is typically not thought of as a condition that is predominately in an elderly population, the peak age of incidence does fall between the fifth and seventh decades [58]. In a recent study, classical TN had an average age onset of 52.9 years; however, the average age of all patients studied was 62.3 years. The majority described the pain as significantly severe and many will have persistent facial pain in between attacks. Approximately 30 % of patients reported sensory abnormalities despite the absence of trigeminal neuropathy or surgical treatment [59•]. In a recent blinded prospective study using 3-T MRI, neurovascular contact of the trigeminal nerve was prevalent in patients with classical TN in both the symptomatic (89 %) and asymptomatic (78 %) sides, with 70 % of patients having bilateral neurovascular contact. Neurovascular contact graded as severe was more predictive of the symptomatic (53 %) versus asymptomatic (13 %) side when present (OR 11.6) [60]. The significance of this is unclear but may provide new insight in the potential etiology of classical TN.

Treatment of classical TN is primarily through medications with surgical interventions considered for more refractory causes, although there are no available studies addressing the time course into treatment when a surgical procedure should be offered. Carbamazepine or oxcarbazepine titrations are both considered first-line treatment for classical TN. There is some evidence to suggest lamotrigine or baclofen may be helpful as well [61]. Care should be taken for appropriate monitoring of these medications, especially in an elderly population. Recently, onabotulinumtoxinA injection has been shown to be effective in reducing pain in a small placebo-controlled trial [62]. Surgical techniques that are graded as possibly effective include percutaneous gasserian ganglion procedures, gamma knife radiosurgery of the trigeminal root, and neurovascular depression of the trigeminal root. However, guidelines as to when to recommend a procedural intervention, and which procedural intervention to recommend for which patient with classical TN, have not yet been well established [61].

Post-herpetic Trigeminal Neuropathy

Herpes zoster, or shingles, is a common reactivation of varicella zoster virus (VZV) typically causing a painful vesicular eruption in a dermatomal distribution. While VZV reactivation is not an uncommon occurrence across multiple age groups, the incidence increases progressively in unvaccinated persons above the age of 50 [63]. VZV reactivation presents in the cranial nerves from 13 to 30 % of cases, for which 80 % are in the first division of the trigeminal nerve [64, 65]. Acutely, this may cause a painful trigeminal neuropathy which may present up to 7 days prior to the herpetic eruption [18••]. As a late consequence of reactivation, and a potential disabling complication, post-herpetic neuralgia is a persistent and often severe, debilitating pain that continues in the distribution of the rash for at least 3 months after the rash has resolved [63]. The probability of acute reactivation leading to post-herpetic neuralgia also increases with age. In one trial, the incidence of post-herpetic neuralgia occurred in 6.9 % of individuals 60–60 years of age and increased to 18.5 % in those 70 or older [66].

Treatment of post-herpetic trigeminal neuropathy is first focused at reducing the risk of occurrence by addressing the acute reinfection with prompt antiviral medication. Antiviral medication given within 72 h of clinical symptoms has been shown to reduce the severity and duration of the acute pain. This may, although the trials were not powered to demonstrate, reduce of the incidence of post-herpetic neuralgia [67]. Treatment of post-herpetic neuralgia includes topical medications such as lidocaine or capsaicin and systemic treatments including gabapentin, pregabalin, and tricyclic antidepressants [68].

Conclusion

The evaluation and treatment of headache in an elderly population require special consideration and care. While overall decreased in those ≥60 years old, headaches still occur with significant frequency and severity. The type, presentation, and treatment considerations of headache disorders change with advancing age. For a new or different headache, a thorough evaluation for a secondary cause should first be considered. Although secondary causes of headache are more common in this age group, primary headache disorders still constitute the majority of cases in a mature population. Initial treatment options are often similar to other adult age groups, keeping in mind a potential increased susceptibility to side effect profiles and complicating comorbidities.

Compliance with Ethics Guidelines

Conflict of Interest Thomas P. Bravo declares that he has no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1. Stovner LJ, Hagen K, Jensen R, et al. The global burden of headache: a documentation of headache prevalence and disability worldwide. Cephalalgia. 2007;27:193–210.
2. Cook N, Evans DA, Funkenstein H, et al. Correlates of headache in a population-based cohort of elderly. Archives of Neurology. 1989;46:1338–44.
3. Prencipe M, Casini AR, Ferretti C, Santini M, Pezzella F, Scaldaferri N, et al. Prevalence of headache in an elderly population: attack frequency, disability, and use of medication. J Neurol Neurosurg Psychiatry. 2001;70:377–81.
4. • Ruiz M, Pedraza MI, de la Cruz C, et al. Headache in the elderly: a series of 262 patients. Neurologia. 2014;29:321–6. **A large retrospective review of elderly patients presenting for tertiary headache evaluation. Ruiz et al. provide epidemiologic data for prevalence of different headache disorders at advanced ages; an area not well studied in the current literature**.
5. Fuh JL, Wang SL, Lu SR, et al. A 13 year long-term outcome study of elderly chronic daily headache. Cephalagia. 2008;28:1017–22.
6. Solomon G, Kunkel R, Frame J. Demographics of headache in elderly patients. Headache. 1990;30:273–6.
7. Pasqual J, Berciano J. Experience in the diagnosis of headaches that start in elderly people. Journal of Neurology, Neurosurgery and Psychiatry. 1994;57:1255–7.
8. "Definition of an older or elderly person." World Health Organization. Web. 15 Feb. 2015. <http://www.who.int/healthinfo/survey/ageingdefnolder/en>.
9. Rasmussen BK, Lipton RB. Prognosis of migraines. In: Olesen J, Goadsby PJ, Ramadan NM, Tfelt-Hansen P, Welch KMA, editors. The headaches. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 429–31.
10. Victor TW, Hu X, Campbell JC, et al. Migraine prevalence by age and sex in the United States: a life-span study. Cephalalgia. 2010;30:1065–72.
11. Haan J, Hollander J, Ferrari MD. Migraine in the elderly: a review. Cephalalgia. 2007;27:91–106.
12. Rasmussen BK, Olesen J. Migraine with aura and migraine without aura: an epidemiological study. Cephalalgia. 1992;12:221–8.
13. Martins KM, Bordini CA, Bigal ME, et al. Migraine in the elderly: a comparison with migraine in young adults. Headache. 2006;46:312–6.
14. Kelman L. Migraine changes with age: IMPACT on migraine classification. Headache. 2006;46:1161–71.
15. Landy SH, Lobo BL. Migraine treatment throughout the lifecycle. Expert Rev Neurotherapeutics. 2005;5:343–53.
16. Dodick D, Lipton RB, Martin V, et al. Consensus statement: cardiovascular safety profile of triptans (5-$HT_{1B/1D}$ agonists) in the acute treatment of migraine. Headache. 2004;44:414–25.
17. • Roberto G, Raschi E, Piccinni C, et al. Adverse cardiovascular events associated with triptans and ergotamines for treatment of migraine: systematic review of observational studies. Cephalgia. 2015;35:118–31. **A thorough systematic review of the literature regarding the cardiovascular safety of vasoactive substances for treatment of headache that provides strong, although not definitive, evidence that triptans are safe in patients without preexisting cardiovascular disease**.
18. •• Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013;33:629–808. **This is the current standard for the classification of headache disorder, currently in a beta version**.
19. Fisher CM. Late-life migraine accompaniments as a cause of unexplained transient ischemic attacks. Can J Neurol Sci. 1980;7:9–17.
20. Fisher CM. Late-life migraine accompaniments—further experience. Stroke. 1986;17:1033–42.
21. Bamford CC, Mays M, Stewart SJ. Unusual headaches in the elderly. Curr Pain Headache. 2011;15:295–301.
22. Donnet A, Daniel C, Milandre L, et al. Migraine with aura in patients over 50 years of age: the Marseille's registry. J Neurol. 2012;259:1868–73.
23. Kleinig TJ, Kiley M, Thompson PD. Acute convexity subarachnoid haemorrhage: a cause of aura-like symptoms in the elderly. Cephalalgia. 2008;28:658–63.

24. Izenberg A, Aviv RI, Demaerschalk BM, et al. Crescendo transient aura attacks: a transient ischemic attack mimic caused by focal subarachnoid hemorrhage. Stroke. 2009;40:3725–9.
25.• Vongvaivanich K, Lertakyamanee P, Silberstein SD, et al. Late-life migraine accompaniments: a narrative review. Cephalalgia. 2014; [Epub ahead of print]. **A current comprehensive literature review of late-life migraine accompaniments and potential secondary causes**.
26.• Holle D, Naegel S, Obermann M. Hypnic headache. Cephalalgia. 2013;33:1349–57. **A comprehensive review of hypnic headache that includes a review of prior studies on clinical characteristics that contributed to a change to the diagnostic criteria**.
27. Liang JF, Wang SJ. Hypnic headache: a review of clinical features, therapeutic options and outcomes. Cephalalgia. 2014;34:795–805.
28. Boes CJ, Matharu MS, Goadsby PJ. Benign cough headache. Cephalalgia. 2002;22:772–9.
29. Chen PK, Fuh JL, Wang SJ. Cough headache: a study of 83 consecutive patients. Cephalalgia. 2009;29:1079–85.
30. Cordenier A, De Hertogh W, De Keyser J, et al. Headache associated with cough: a review. J Headache Pain. 2013;14:42.
31. Duran J, Esnaola S, Rubio R, et al. Obstructive sleep apnea–hypopnea and related clinical features in a population-based sample of subjects aged 30 to 70 yr. Am J Respir Crit Care Med. 2001;163: 685–9.
32.• Kristiansen HA, Kværner KJ, Akre H, et al. Sleep apnoea headache in the general population. Cephalalgia. 2012;32:451–8. **A large population study on sleep apnea headache that provides evidence that there may not be a direct relationship between hypoxia and occurrence of headache**.
33. Russell MB, Kristiansen HA, Kværner KJ. Headache in sleep apnea syndrome: epidemiology and pathophysiology. Cephalalgia. 2014;43:752–5.
34. Goksan B, Gunduz A, Karadeniz D, et al. Morning headache in sleep apnoea: clinical and polysomnographic evaluation and response to nasal continuous positive airway pressure. Cephalalgia. 2009;29:635–41.
35. Caselli RJ, Hunder GG, Whisnant JP. Neurologic disease in biopsy-proven giant cell (temporal) arteritis. Neurology. 1988;38:352–9.
36. Smith JH, Swanson JW. Giant cell arteritis. Headache. 2014;54: 1273–89.
37. Huston KA, Hunder GG, Lie JT. Temporal arteritis: a 25-year epidemiologic, clinical, and pathologic study. Annals of Internal Medicine. 1978;88:162–7.
38. Myklebust G, Gran JT. A prospective study of 287 patients with polymyalgia rheumatica and temporal arteritis: clinical and laboratory manifestations at onset of disease and at the time of diagnosis. Br J Rheumatol. 1996;35:1161–8.
39. Hunder GG, Bloch DA, Michel BA, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. Arthritis Rheum. 1990;33:1122–8.
40. Grayson PC, Tomasson G, Cuthbertson D, et al. Association of vascular physical examination findings and arteriographic lesions in large vessel vasculitis. J Rheumatol. 2012;39:303–9.
41. Arida A, Kyprianou M, Kanakis M, Sfikakis PP. The diagnostic value of ultrasonography-derived edema of the temporal artery wall in giant cell arteritis: a second meta-analysis. BMC Musculoskelet Disord. 2010;11:44.
42. Klink T, Geiger J, Both M, et al. Giant cell arteritis: diagnostic accuracy of MR imaging of superficial cranial arteries in initial diagnosis-results from a multicenter trial. Radiology. 2014;273: 844–52.
43. Mukhtyar C, Guillevin L, Cid MC, et al. EULAR recommendations for the management of large vessel vasculitis. Ann Rheum Dis. 2009;68:318–23.
44. Bini A, Evangelista A, Castellini P, et al. Cardiac cephalgia. J Headache Pain. 2009;10:3–9.
45. Wei JH, Wang HF. Cardiac cephalalgia: case reports and review. Cephalalgia. 2008;28:892–6.
46. Grace A, Horgan J, Breathnach K, et al. Anginal headache and its basis. Cephalalgia. 1997;17:195–6.
47. Ohringer G, Burke T, Shaikh A. Unilateral headache and loss of vision. BMJ. 2014;348:1188.
48. Ritch R, Lowe RF. Angle-closure glaucoma: clinical types. In: Ritch R, Shields MB, Krupin T, editors. The glaucomas. St. Louis: Mosby-Year Book; 1996. p. 821–40.
49. Nesher R, Epstein E, Stern Y, et al. Headaches as the main presenting symptom of subacute angle closure glaucoma. Headache. 2005;45:166–76.
50. Shindler KS, Sankar PS, Vlpe NJ, et al. Intermittent headaches as the presenting sign of subacute angle closure glaucoma. Neurology. 2005;65:757–8.
51.• Westergaard ML, Hansen EH, Glumer C, et al. Definitions of medication-overuse headache in population-based studies and their implications on prevalence estimates: a systematic review. Cephalalgia. 2014;34:409–25. **An extensive review of the varied prior definitions of medication overuse headache and the overall prevalence among different groups**.
52. Prencipe M, Casini AR, Feretti C, et al. Prevalence of headache in an elderly population: attack frequency, disability, and use of medication. J Neurol Neurosurg Psychiatry. 2001;70:377–81.
53. Wang SJ, Fuh JL, Lu SR, et al. Chronic daily headache in Chinese elderly prevalence, risk factors, and biannual follow-up. Neurology. 2000;54:314–9.
54. Zeeberg P, Olesen J, Jensen R. Probable medication-overuse headache: the effect of a 2-month drug-free period. Neurology. 2006;66: 1894–8.
55. Dodick DW. Pearls: headache. Seminars in Neurology. 2010;30: 74–81.
56.• Lynch KM, Brett F. Headaches that kill: a retrospective study of incidence, etiology and clinical features in cases of sudden death. Cephalalgia. 2012;32:972–8. **A retrospective study of fatal cases presenting with headache that demonstrate concerning 'red flags' for serious secondary causes**.
57. Sjaastad O, Bakketeig LS. Prevalence of cervicogenic headache: Vågå study of headache epidemiology. Acta Neurol Scand. 2008;117:173–80.
58. Rozen TD, Capobianco DJ, Dalessio DJ. Cranial neuralgias and atypical facial pain. In: Silberstein SD, Lipton RB, Dalessio DJ, editors. Wolff's headache and other head pain. 7th ed. New York: Oxford University Press; 2001. p. 509–24.
59.• Maarbjerg S, Gozalov A, Olesen J, et al. Trigeminal neuralgia—a prospective systematic study of clinical characteristics in 158 patients. Headache. 2014;54:1574–82. **A large prospective study of the clinical characteristics of TN which had the notable findings of a high percentage of persistent facial pain, cranial autonomic symptoms and bedside sensory abnormalities**.
60. Maarbjerg S, Wolfram F, Gozalov A, et al. Significance of neurovascular contact in classical trigeminal neuralgia. Brain. 2015;138:311–9.
61. Gronseth G, Cruccu G, Alksne J, et al. Practice parameter: the diagnostic evaluation and treatment of trigeminal neuralgia (an evidence-based review). Neurology. 2008;71:1183–90.
62. Wu CJ, Lian YJ, Zheng YK, et al. Botulinum toxin type A for the treatment of trigeminal neuralgia: results from a randomized, double-blind, placebo-controlled trial. Cephalalgia. 2012;32:443–60.
63. Harpaz R, Ortega-Sanchez IR, Seward JF, Advisory Committee on Immunization Practices (ACIP) Centers for Disease Control and Prevention (CDC). Prevention of herpes zoster: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep. 2008;57(RR-5):1.

64. Carbone V, Leonardi A, Pavese M, et al. Herpes zoster of the trigeminal nerve: a case report and review of the literature. Minerva Stomatol. 2004;53(1–2):49–59.
65. Moller L. Aspects of the natural history of herpes zoster. Acta Derm Venereol. 1969;49:569–83.
66. Oxman MN, Levin MJ, Johnson GR, et al. A vaccine to prevent herpes zoster and postherpetic neuralgia in older adults. N Engl J Med. 2005;352(22):2271.
67. Wood MJ, Kay R, Dworkin RH, et al. Oral acyclovir therapy accelerates pain resolution in patients with herpes zoster: a meta-analysis of placebo-controlled trials. Clin Infect Dis. 1996;22:341–7.
68. Johnson RW, Rice ASC. Postherpetic neuralgia. N Engl J Med. 2014;371:1526–33.