



Nummular Headache

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Abstract

Purpose of Review This article reviews nummular headache (NH), including the latest literature on the epidemiological and clinical features, the most recent proposed pathophysiology, and novel management.

Recent Findings NH is characterized by continuous or intermittent head pain confined to a focal circumscribed area (1–6 cm in diameter). It is usually of mild to moderate intensity, although some patients have severe continuous pain or disabling exacerbations. NH is a primary headache, though many secondary cases have been described. Evaluation requires exclusion of systemic and structural diseases. Gabapentin can be recommended as a first-line preventive treatment. If poorly tolerated or in refractory cases, botulinum toxin is a reasonable alternative.

Summary NH is a fairly common disorder in patients presenting to a headache clinic. Increased understanding of NH will lead to improved outcomes. Clinical trials would lead to enhanced knowledge of NH.

Keywords Nummular headache · Coin-shaped headache · Circumscribed cephalalgia · Epicrania · Primary headache

Introduction

Derived from the Latin word ‘nummus’ meaning coin, the term nummular headache (NH) was introduced in 2002 when Pareja and colleagues described 13 patients with well-circumscribed pain confined to an oval or elliptical-shaped region in the head [1••]. Two years later, the same group reported a second case series of 14 patients with NH [2]. Based on these reports, NH was included in the research diagnostic criteria in the second edition of the International Classification of Headache Disorders (ICHD-2) [3]. Since then, there have been over 280 cases reported in the literature including several large case series [4•, 5•, 6, 7•, 8–11]. The diagnostic criteria were further refined in the ICHD-3 [12].

We begin with a review of the diagnostic criteria outlined in the ICHD-3 as well as the epidemiologic and clinical features. We present the most current pathophysiologic mechanisms as well as the recommended evaluation and management of this disorder.

Diagnostic Criteria

The ICHD-3 criteria for NH require a continuous or intermittent sharply contoured head pain felt exclusively in a fixed, rounded, or elliptical-shaped area, typically 1 to 6 cm in diameter (Table 1). The description notes that the pain is highly variable in duration, but often continuous even in the absence of an underlying structural lesion [12]. It is specified that the painful area may be localized to any part of the scalp, but it is most common in the parietal region [12]. Rarely, the headache is bi- or multifocal, with each symptomatic area possessing all characteristics of nummular pain [12]. Spontaneous or triggered exacerbations may occur, and superimposed background pain may exist [12]. The affected area often shows variable combinations of hypoesthesia, hyperesthesia, dysesthesia, paresthesia, allodynia, and tenderness [12]. Other causes, including neural and dermatological lesions, must be excluded [12]. Table 2 denotes the criteria for probable NH.

The criteria for probable NH are similar to that of NH but only require three of the four characteristics.

Epidemiology

Nummular headache is thought to be uncommon, but its true prevalence and incidence are unknown [13]. In one hospital

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Table 1 2013 International Headache Society criteria for the diagnosis of nummular headache

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- A. Continuous or intermittent head pain fulfilling criterion B
 - B. Felt exclusively in an area of the scalp, with all of the following four characteristics:
 1. Sharply contoured
 2. Fixed in size and shape
 3. Round or elliptical
 4. 1–6 cm in diameter
 - C. Not better accounted for by another ICHD-3 diagnosis.
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Data from (2013) Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, 3rd edition (beta version). *Cephalalgia* 33:629–808. Reprinted with permission from SAGE Publications, Ltd. [12]

series, the incidence of NH was 6.4/100,000/year [2]. In a general neurology outpatient office, it represented 0.25% of all patients and 1.25% of all those presenting with a chief complaint of headache [7•]. There is, however, an absence of large-scale epidemiologic studies, and it is likely that the true incidence of NH is larger in the general population. Schwartz et al. reviewed over 250 published cases and found a female predominance with a ratio of 1.8:1 [11, 14]. The age of onset ranges from 4 to 82 years, being most common in the fifth decade, with a mean age of 45.4 years [11]. The duration of symptoms before diagnosis is 4.4 years with a range of < 1 month to 50 years [11, 14].

Almost half (46.7%) of NH patients have a concurrent or prior headache diagnosis of migraine, tension-type headache (TTH), medication overuse headache, or primary stabbing headache (PSH) [11]. In the majority of cases, there is no identified precipitant, though 12.8% of persons with NH report a remote history of head trauma [11]. One study found that there was no significant difference between persons with NH and controls with respect to anxiety or depression, and no correlation between depression or anxiety with pain intensity, exacerbations, size, or frequency. The authors concluded that self-reported depression and anxiety were not related to the presence of NH [15].

Clinical Features

The primary characteristic of NH is that the pain and sensory dysfunction always occur within a small, fixed, well-circumscribed area. The symptomatic area is more often round (80.2%) than oval/elliptical (19.8%), and typically 1 to 6 cm in diameter (mean 3.5 cm) [11]. Some patients have described areas as small as 0.6 cm [16] or as large as 10 cm [9]. Patients can often delineate the outline of the affected region with their finger [17]. Shape and size typically are unchanged with time [14]. Most often the pain is strictly unilateral, with the right side (53.4%) being slightly more affected than the left (38.7%) or the midline (7.9%). The pain is most often located in the parietal region (55.7%), and less often involves the occipital (19.6%), temporal (13.0%), or frontal (11.7%) regions [11].

Pain is most commonly described as pressure-like, sharp, or stabbing. Pain intensity is usually mild to moderate, although some patients report severe pain. A mean baseline intensity of 5.7 was reported using the analogical visual scale (AVS) [7•]. Cases precipitated by sexual intercourse, coughing, Valsalva maneuver, and menstruation have been reported [18, 19]. Superimposed exacerbations of pain that last seconds to hours are present in 59.1% of patients [10, 11, 20]. On average, the exacerbations increase the base

Table 2 2013 International Headache Society criteria for the diagnosis of probable nummular headache

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- A. Continuous or intermittent head pain fulfilling criterion B
 - B. Felt exclusively in an area of the scalp, with three only of the following four characteristics:
 1. Sharply contoured
 2. Fixed in size and shape
 3. Round or elliptical
 4. 1–6 cm in diameter
 - C. Not fulfilling ICHD-3 criteria for any other headache disorder
 - D. Not better accounted by another ICHD-3 diagnosis
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Data from (2013) Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, 3rd edition (beta version). *Cephalalgia* 33:629–808. Reprinted with permission from SAGE Publications, Ltd. [12]

AVS by two points. Exacerbations are most commonly reported as a stabbing quality and can occur spontaneously or be triggered by tactile or other stimuli [14]. Clinical features and overall treatment response appear to be similar regardless of the presence of acute exacerbations [7, 14].

Abnormal sensation in the affected area is common and reported by up to 48.6% of those with NH [7]. Symptoms include numbness, allodynia, hypoesthesia, and hyperesthesia. The sensory abnormalities may be present even during remissions of pain [14].

Associated migrainous and autonomic features are uncommon but may occur [11]. Case reports have described bilateral lacrimation, rhinorrhea, and photophobia with pain exacerbations [13, 21]. There are also case reports of trophic changes within the affected area including cutaneous atrophy, alopecia, and erythema [14, 22–24]. Skin biopsies were performed on three patients with trophic changes and were found to be non-specific [22]. A case has been reported of a 4-year-old child who developed NH in a patch of lighter colored hair that had been present since birth [25].

The temporal pattern is episodic in one-third and chronic in two-thirds of patients [17]. Rarely, the episodic pattern evolves into the chronic course [2, 9, 17]. When symptomatic, the pain may be intermittent, fluctuating, or continuous [17, 26]. There is evidence that suggests a circadian pattern [14]. When not continuous, NH mainly occurs during the daytime and has only rarely been reported at night [2, 11, 17, 27].

Bifocal presentations have been described in several cases [5, 7, 8, 10, 28, 29]. Some present in a sequential manner moving from one painful area to another, while others report the addition of a second painful region or even a simultaneous appearance of two painful regions [14]. NH has also been reported with multiple foci [23, 30].

Pathogenesis

The pathophysiological mechanisms of NH are uncertain. Unlike migraine and TTH, there does not appear to be a diffuse hypersensitivity in NH but rather a non-generalized or focal neural process [14, 31]. In a study examining pericranial tenderness among subjects with NH, TTH, or serving as controls, only those with TTH had it [31]. Another study found that increased mechanical pain sensitivity with low pressure pain threshold is restricted to the symptomatic area [32]. Because symptoms and signs are confined to a small area, it strongly suggests that NH is caused by a peripheral, local process rather than a central mechanism, but this remains unproven [14].

The theory of a peripheral pathogenesis is reinforced by multifocal NH in that each symptomatic area maintains all characteristics of NH [17]. In contrast, a central disorder generates symptoms in more diffuse areas due to central pathways receiving convergent inputs from separate afferent sources

[33]. Furthermore, the topography of central pain has less distinct boundaries and tends to spread over time [17]. Trophic changes accompanying NH also suggest a dysfunction of the peripheral nervous system, as it is well known that alterations of the skin and subcutaneous tissue are common in peripheral neuropathies [34].

Some authors believe that the size and shape of the symptomatic area as well as the local sensory dysfunction suggest neuralgia of a terminal branch of a peripheral nerve. However there are two features which challenge this hypothesis: (1) the lack of response to local anesthetic injections and (2) the existence of painful areas spanning the midline [14, 17]. It has been suggested that dysfunction of the transdiploic and intradiploic nerves (which interconnect the dura and all the layers of the scalp) may give rise to precisely localized head pain [17]. This would also help explain the lack of response to local anesthetics, as these nerves are unapproachable by conventional anesthetic blocks.

When it was first described, some authors believed that NH was psychogenic in origin [35], but studies suggest that persons with NH do not differ significantly from a control population with regard to anxiety and depression [14, 15, 20]. Additionally, there is no correlation between emotional states and pain intensity, frequency, or size [15].

The rates of coexisting primary headache disorders such as migraine are fairly high (46.7%) in the NH population [11]. It is unclear if NH is pathogenetically related to the primary headache disorder or is a distinct entity [11]. Since the source of NH is unclear, many believe that it should be considered as an epicrania or headache probably stemming from the epicranial tissues (internal and external layers of the skull, all layers of the scalp, and epicranial nerves and arteries) [17, 36].

Differential Diagnosis and Evaluation

NH is considered a primary headache, and the ICHD-3 criteria require the absence of any underlying disease or structural lesion. Nonetheless, “secondary” or “symptomatic” forms have been reported, most of which suggest a crucial role of the epicranial tissue [37]. There have been several cases associated with vascular anomalies. One 24-year-old patient with Marfan syndrome with severe focal, frontal pain was found to have an underlying aneurysm and experienced complete resolution of the pain through surgical resection [38]. Lopez-Ruiz et al. describe two patients in whom superficial fusiform aneurysms were identified in the painful area with Doppler ultrasound or angiographic techniques [37]. Complete remission was obtained after excision in one of the cases [37]. Other cases of NH have been reported where the pain corresponded with the location of a calcified hematoma [39], biopsy confirmed linear scleroderma [40], or varicella-zoster virus [41]. One patient reported onset of symptoms after an insect bite in

the affected region [22]. In other cases, bony anomalies have been implicated, such as in Paget's disease [11], fibrous dysplasia [6], craniosynostosis [42], and Langerhan histiocytosis [43]. Of note, two patients with Langerhans had complete remission after surgical excision [43]. Intracranial lesions such as meningioma [44••] and arachnoid cysts [45] have also been found underlying NH. Another patient reportedly developed NH in the contralateral hemicranium after surgical resection of a pituitary adenoma [6]. Future clinical criteria may help to distinguish between primary and secondary NH [14].

NH should be differentiated from epicrania fugax, which is a paroxysmal, unilateral, shooting pain that starts in a focal area and spreads forward rapidly within the ipsilateral side along a linear or zigzag trajectory lasting 1–10 s [46]. The originating area may remain tender between attacks, and this can pose some challenges in its differentiation from NH [17]. Nonetheless, NH, by definition, remains in a circumscribed small area. Paroxysms do exist in NH, but they always remain in the same location [17].

PSH is also perceived in a specific point on the scalp. It can be differentiated from NH by its spatial and temporal characteristics [14]. In PSH, there are short pain paroxysms that last 1–3 s and are usually multifocal [47], while the pain from NH is longer lasting, if not continuous, and confined to a specific area. One can differentiate multi-focal NH and PSH by the patient's ability to demarcate precisely the area of pain in NH [14].

NH should be differentiated easily from cranial neuralgias such as supraorbital, auriculotemporal, and occipital neuralgias. The neuralgias produce pain that is perceived within the territory supplied by a given nerve, while NH pain is in a well-defined round or elliptical boundary [17]. In neuralgias, the nerve is hypersensitive to palpation [14]. The neuralgias are also responsive to anesthetic blockade, while NH is not [14].

When there are cutaneous lesions, the differential should include certain dermatologic lesions such as alopecia areata (a painless process) or nummular dermatitis (classified as a contact dermatitis with eczematous pruriginous lesions) [48]. Trichodynia, which is a painful condition of the scalp, has features that overlap with NH [49].

A careful history coupled with a complete physical and neurologic examination is crucial to accurate diagnosis. It is important to inspect and palpate the scalp as well as the pericranial muscles, nerves, and arteries fully. Skin biopsy can be beneficial when there are cutaneous abnormalities [14]. Neuroimaging with magnetic resonance imaging and/or computed tomography is necessary to rule out underlying structural abnormalities. Laboratory studies should include a complete blood count, basic metabolic panel, liver function tests, thyroid function tests, erythrocyte sedimentation rate, c-reactive protein, alkaline phosphatase, antinuclear antibodies, rheumatoid factor, angiotensin-converting enzyme, and urine analysis [13]. One case series found a high prevalence of

autoimmune disease. Out of the 23 patients with NH, 16 had laboratory abnormalities, and 15 of those were eventually diagnosed with Sjogren syndrome, rheumatoid arthritis, or antiphospholipid syndrome [8].

Management

Some patients with NH do not require treatment, simply reassurance. In patients with moderate to severe pain or concern for secondary mechanism, treatment is warranted [11, 14, 27]. There are no clinical trials to date, and the only support comes from anecdotal evidence, case reports, or small case series.

For patients with mild continuous pain, intermittent pain, or as an add-on for symptomatic treatment, nonsteroidal anti-inflammatory drugs (NSAIDs) have been beneficial in over 60% of cases [11, 14]. Reported responses vary among non-effective [1••, 6, 7•, 10, 21, 50], partial [5, 35, 51], and excellent [2, 7•, 21, 52, 53].

In one patient with migrainous features, triptans were effective as an acute therapy [26]. Oral triptans were effective in two patients with symptomatic NH due to superficial arterial aneurysm [37]. As previously mentioned, subcutaneous anesthetic injections typically do not provide benefit [1•, 21, 24, 44, 50, 51, 54]. In cases with persistent, moderate to intense pain, and lack of response to analgesics, daily preventive therapy may be indicated. Preventive therapy was deemed necessary in 58.3% of patients in the largest case series [7•].

Gabapentin has been used in more than 60 published cases and provided benefit in over 60% of patients. Gabapentin effects neuromodulation by interacting with voltage-gated calcium channels [14, 55]. Gabapentin 600–1200 mg total daily medication is recommended for the management of NH (recommendation class C) [14].

Tricyclic antidepressants have been used for the treatment of NH in over 40 published cases and provided at least partial benefit in 45% of patients [11]. Beneficial responses have been reported with amitriptyline [6, 16, 56], nortriptyline [27], and clomipramine [14]. Other drugs that have been reported to be effective include indomethacin [5•, 14, 16, 35, 53, 57], carbamazepine [56, 58], topiramate [10, 26], pregabalin [18, 40, 56], cyclobenzaprine [19], neurotropin [29, 59], and palmitoylethanolamide [60]. Valproate, lamotrigine, and flunarizine are reported as not effective [56].

Botulinum toxin type A (BoNTA) has been used in numerous cases with at least partial benefit in the majority of patients [10, 11, 50, 61, 62]. One case report described a patient with NH in an area of alopecia who had improvement of both the pain and the alopecia following treatment with BoNTA [24]. A recent case series of 19 patients with refractory NH treated with BoNTA in doses ranging from 12.5–50 units found that 78.9% had at least partial response to the procedure [14, 63]. Based on these reports, BoNTA is a reasonable choice for patients for whom oral treatments such as those discussed

above have been ineffective or not well tolerated [14]. Injections should be placed at several points distributed over the painful area and its perimeter with 2.5–5 units per point.

NH related to varicella-zoster infection is reported to respond well to anti-viral therapy [41]. Scleroderma-associated NH has been treated successfully with local steroids [40]. In the secondary cases mentioned previously, good results have been achieved by addressing the underlying cause. The patient with the meningioma went into full remission after surgery [44••]. There were also positive responses with surgical excision of the superficial aneurysm [37] and the lesions in two patients with underlying Langerhans histiocytosis [43].

Even in cases where no clear underlying lesion is found, interventional procedures might lead to improvement. One patient reported pain relief with a TENS unit [64]. A recent study with 49 NH patients showed significant benefit with surgical arterectomy with local anesthesia [65]. On the other hand, another patient initially had pain relief following focal scalp excision of the affected area, but eventually had reappearance of NH in an area that overlapped with the former one [66]. Three patients were treated with surgical interventions of the trigeminal nerve without significance response [9].

Prognosis

Because NH is a relatively newly described entity, there is a paucity of literature on its natural history and long-term prognosis. Evidence suggests that it is a benign condition that may spontaneously remit after a variable duration of symptoms [17]. Spontaneous remission can occur in time with or without recurrence [1•, 5•, 6, 9, 14, 17, 23]. In the largest case series, 38.7% of patients reported spontaneous remission [7•]. Persistent remissions after treatment withdrawal have also been reported [53, 67]. In some cases, however, the disorder may last for years or even decades. Cases in which NH was attributed to a cranial structural lesion were found to have favorable outcomes after surgical treatment [17].

Conclusion

NH is a well-defined clinical disorder that presents fairly frequently to headache clinics, even though the true incidence and prevalence of NH in the general population are unknown. It is characterized by focal head pain that occurs in a well-circumscribed round or elliptical area in the absence of an underlying lesion. It is a primary headache and requires the exclusion of systemic and structural disease by history, physical examination, laboratory testing, and neuroimaging. “Secondary” or “symptomatic” cases have been reported from intracranial processes or scalp, bony, or cutaneous lesions. The pathophysiology of NH is likely from the peripheral nervous system. Many patients, especially those with moderate to

severe pain or those who have frequent exacerbations, require treatment. No clinical trials have been conducted for the treatment of NH, so the strength of evidence-based recommendations is low. Analgesics are often effective for mild cases. Gabapentin can be recommended as a first-line preventive treatment. If poorly tolerated or in refractory cases, BoNTA is a reasonable alternative.

Compliance with Ethical Standards

Conflict of Interest Danielle Wilhour and Claire E.J. Ceriani each declare no potential conflicts of interest. Stephanie J. Nahas reports personal fees from Allergan, Amgen, Avanir, electroCore, Eli Lilly, Supernus, Teva, and Biohaven, outside the submitted work.

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.

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