Current Pain and Headache Reports (2022) 26:79–84
https://doi.org/10.1007/s11916-022-01005-1

CHRONIC DAILY HEADACHE (SJ WANG AND SP CHEN, SECTION EDITORS)

Update of New Daily Persistent Headache

Kuan-Po Peng[1,2] · **Shuu-Jiun Wang**[2,3,4]

Accepted: 12 November 2021 / Published online: 25 January 2022

Abstract

Purpose of Review The diagnostic criteria of new daily persistent headache (NDPH) have been revised since 2013. The current review focused on the progress of NDPH research over the last few years.

Recent Findings Various new triggers and different NDPH mimics have been reported. The association with both cephalic and extracephalic pathologies suggests that NDPH is rather a syndrome with more than one disease mechanism. Recent clinical studies confirmed that migrainous headache remained the most prominent phenotype of NDPH, echoing the change of the diagnostic criteria in 2013. Diagnostic workup, including imaging studies, was unremarkable, except serving to exclude secondary etiologies. Studies on treatment options have yet shown promising targets, and randomized clinical trials are still lacking.

Summary Multiple mechanisms, both cranial and systemic, may be involved synergically in the generation of NDPH-like headaches. The search for effective treatment options should base on better understanding of disease mechanisms.

Keywords NDPH · RCVS · COVID-19 · Infection · Post-infection · Migrainous

Introduction

History and evolution of New Daily Persistent Headache

The diagnostic entity of new daily persistent headache (NDPH) has been introduced more than 30 years ago [1], featuring its pathognomonic presentation of persistent headache since the acute onset on a specific day. Over the years, the diagnostic criteria have been gradually evolved along with the cumulating clinical evidence which better depicts the clinical presentation and prognosis of this specific disease entity. According to the latest International Classification of Headache Disorders, 3rd version (ICHD-3), NDPH is featured by its daily and persistent characteristics, and the headache features are no longer restricted [2]—headaches in NDPH can be either tension-type headache (TTH)-like [3] or migrainous [4, 5], with and without other associating symptoms, such as photophobia or nausea.

Early clinical series of NDPH did not differ between idiopathic and secondary etiologies. Common secondary etiologies include viruses or other systemic infections, such as Epstein–Barr virus, Salmonella, or E. Coli [6]. Some studies also showed seasonal peaks of NDPH onset and suggested potential seasonal infectious or environmental link [4, 7]. NDPH was first introduced as a diagnostic entity into the ICHD in the 2nd version (ICHD-2), which was published in 2004 [8]. According to ICHD-2, and its successor, ICHD-3, NDPH was categorized under Sect. 4—other primary headache disorders. That means a secondary cause must be excluded before making the diagnosis of NDPH. The distinction between primary and secondary etiologies may sometimes be blurred, especially concerning an infection as a trigger. Besides, there are no specific biomarkers for NDPH. The diagnosis is exclusively based on the phenotype of "daily" and "persistent" headaches with a new-onset, both of which are, unfortunately, not specific. Such phenotype

This article is part of the Topical Collection on *Chronic Daily Headache*

✉ Kuan-Po Peng
k.peng@uke.de

1 Department of Systems Neuroscience, University Medical Center Hamburg-Eppendorf, Hamburg, Germany

2 Brain Research Center, National Yang Ming Chiao Tung University, Taipei, Taiwan

3 Department of Neurology, Neurological Institute, Taipei Veterans General Hospital, Taipei, Taiwan

4 College of Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan

of headache (daily and persistent) is not rare and has been reported as early as in 1890 after the Russian/Asiatic flu [9•], and recently after the COVID-19 pandemic [10•]. Post-infectious development of NDPH or persistent headache that developed during acute infection that persisted after the resolution of the infection remains relatively common and suggests the infection as a trigger to the development of NDPH. Should the patients with infection-associated/triggered NDPH be regarded as a primary or secondary headache? These patients may be coded under *§9.2—Headache attributed to systemic infection*, with their headache features mimicking those of NDPH.

Perhaps NDPH is a heterogeneous disease entity, consisting of both primary and secondary etiologies. More specifically, as some have suggested, that NDPH is rather a syndrome than a distinct disease [11]. In the current review, we will focus on the progress of our understanding of NDPH over the last few years. Besides, we will revisit the issue, whether NDPH should be regarded as an exclusively primary headache disorder.

Epidemiology

NDPH is rare, and epidemiological studies of NDPH are hence scarce. NDPH belongs to the group of chronic daily headache (CDH), defined as having more than 15 days of headache in a month. The prevalence of CDH is approximately 4% in the general population [12, 13]. In tertiary center-based studies, NDPH accounts for 0.9–35% of CDH in pediatric population [14, 15] and 2.5–10.8% in the adult population [5, 16]. It has been hence suspected that NDPH may be more common in pediatric CDH patients compared to adult CDH patients. In community-based settings, two studies from Spain and Norway reported a one-year prevalence of NDPH of 0.1% [13] and 0.03% [17], respectively, confirming that NDPH is very rare in the general population. However, both studies used the more restrictive ICHD-2 criteria, which only allowed patients with TTH phenotypes to be diagnosed as NDPH. Therefore, the actual number may be slightly underestimated. Of note, since the introduction of ICHD-3 in 2018 [2], or its predecessor, ICHD-3-beta in 2013 [18], there have been no new epidemiological studies on NDPH.

Triggers of NDPH

Infectious Episodes

Miscellaneous triggers associated with NDPH have been reported, most commonly recent infectious episodes or stressful life event [4, 5]. Besides the early reported triggers of Epstein–Barr virus, and Salmonella/E. Coli [6, 19], a recent study investigated 450 patients with Dengue fever and identified three possible cases of NDPH [20]. After the pandemic of COVID-19, persistent headaches have been reported after the acute episode of COVID-19 [10•, 21], some of which also fulfilled the diagnostic criteria of NDPH [10•]. Therefore, the development of NDPH is not restricted to specific pathogens: both virus and bacterial infection are probable triggers. Moreover, long before the introduction of the disease entity of NDPH in 1986 [1], similar presentation of daily persistent (NDPH-like) headaches have been described as early as 1890 after the Russian/Asiatic flu [9•], suggesting the history of NDPH should be much longer than 30 years. Even though a potential infectious trigger is not restricted to specific pathogens, in most of the reported infectious triggers, the pathogen usually caused a systemic infection, rather than a local infection. These patients may also fulfill the diagnosis of *§9.2—Headache attributed to systemic infection* according to the ICHD-3 [2].

Cervicogenic Triggers

Not all patients had had an infection as a trigger to NDPH. Rozen reported in a large NDPH series (n = 97) that 53% of the patients reported no specific triggers [22]. In this study, besides the commonly reported triggers, 9% of the patients had the onset of NDPH-like headache after various surgical procedures that require intubation, suggesting a possible cervicogenic etiology in a subgroup of NDPH patients [22].

Other Triggers

In another case series, seven NDPH patients with an initial trigger of a single Valsalva event have been reported. Of note, none of these patients had papilledema, but cerebrospinal fluid pressure/volume reducing medication such as acetazolamide achieved more than 90% reduction in headache frequency in five out of seven patients [23•]. This subtype of patients suggests that a potential role of abnormal reset of the CSF pressure/distribution may also contribute to typical NDPH phenotypes. The various triggers of NDPH further reinforced the idea that NDPH is not a homogenous disease, but a syndrome with various etiologies.

Clinical Characteristics

A recent study in Italy looked into 46 pediatric patients with NDPH following ICHD-3 criteria [24]. The headache features are mostly migrainous (62%), and 75% of them had an onset in the winter months. Surprisingly, up to 80% of patients had an initially good clinical response to common migraine prophylactic treatment in the first year, but at follow-up one year later, 54% of them returned to a remitting form of headache.

Two unfavorable outcome predictors were identified, including the lack of obvious trigger and no pharmacological treatment [24], the latter of which echoes the earlier study that patients with early (within 6 months) pharmacological treatment after the onset of NDPH were more likely to have a favorable outcome [5]. NDPH may be easier to treat before the chronification has been well-established. Another American study enrolled 245 pediatric patients in one single tertiary referral center between 2016 and 2018. The patients were predominantly female and with typical migrainous features. Medication overuse headache has been identified in 36% of the study cohort, and most of the patients had unsatisfactory responses to treatment [25•].

Evans reported seven patients with a daily non-persistent headache from the onset on one specific day with a daily duration of ≥ 4 h. The majority (71.4%) had migraine-like features, and the headache remains refractory to acute, preventive, or other treatment including nerve blocks [26]. These patients meet the ICHD-3 criteria for NDPH, except the headache being non-continuous. The author proposed that these patients may belong to a variant of NDPH, a daily but non-persistent variant. Notably, before being officially listed in the ICHD-2 in 2004 [8], NDPH was already included in the Silberstein–Lipton (S–H) criteria for CDH published in 1994 [27]. According to the S–H criteria, CDH was defined as ≥ 4 h/day and ≥ 15 days/month [27]. Therefore, NDPH, as a subtype of CDH, does not need to be persistent according to S–H criteria, similar to the cases reported by Evans. Nonetheless, whether these patients should be regarded as a variant of NDPH awaits further investigation. Without the persistent headache (and without any disease markers), not much is left with NDPH entity, except for an acute onset and refractoriness to most treatment options, the latter of which is, strictly speaking, not a criterion for NDPH.

Psychiatric Comorbidity and Disease-associated Disability

One study in India recruited 55 patients with NDPH and used established batteries to evaluate depressive symptoms, anxiety, somatoform disorders, and pain catastrophizing. The NDPH cohort was compared with age-/sex-matched healthy controls, and patients with another chronic pain disorder—low back pain [28]. In this study, psychiatric comorbidity was very frequent (32.7–85.5%) in NDPH patients, and significantly more frequent than those with low back pain or healthy controls. Moreover, NDPH patients with typical migrainous features had even higher depression and pain catastrophizing behavior than their counterparts with TTH-like headaches [28]. This study echoes the earlier studies [4, 5] and suggests the high disease burden of NDPH not only to the headache but also the psychiatric comorbidities. A recent descriptive study on disease-associated disability was conducted in Spain. Eighteen patients with NDPH were interviewed, and the disease-associated disability and impact were evaluated [29]. The "disease burden" was not quantified, but the disease-associated disability and its impact on everyday life were high [29]. This is in line with an earlier study that NDPH patients had high disability accessed using Short Form 36 (SF-36) Health Survey or Migraine Disability Assessment (MIDAS) [5]. A recent large-sized study compared the symptoms and disability of 1,170 adolescent patients with daily persistent headaches, 84.3% of whom with chronic migraine (CM) and 115 of whom (13.2%) with a diagnosis of NDPH. There were no clinically meaningful differences in headache features and associated disability, suggesting the disease burden of NDPH may be as high as CM in adolescent patients [30].

Imaging Diagnostic Workup

One earlier study looked into the brain imaging studies of 82 NDPH patients, 9 of whom received CT scans, the rest 73 MRI scans [5]. In this study, all CT scans were normal. Abnormal MRI findings were detected in 15 (20.5%) out of 73 patients, most of which being non-specific findings including white matter spots or single old lacunar infarction [5]. Recently, one study retrospectively reviewed 97 patients with primary NDPH and found that the majority of them (84/97) had no white matter abnormalities, which are sometimes seen in migraine patients. In those with white matter abnormalities (13/97), patients have either comorbid cardio-/cerebrovascular diseases or migraine [31]. Both studies combined suggest that the brain MRI in patients with NDPH should be normal.

NDPH-mimics with Secondary Causes

Sousa et al. reported a case with a typical presentation of NDPH, but the MRI showed evidence of radiologically isolated syndrome. Follow-up MRI three months later showed evidence of new contrast-enhanced lesions, and multiple sclerosis was subsequently diagnosed [32]. Rozen and Beams reported a case of NDPH with thunderclap headache onset, and nimodipine, standard treatment for idiopathic thunderclap headache, rapidly alleviated the symptoms completely [33]. Subsequent studies also reported several cases of NDPH with a typical thunderclap headache onset [34, 35]. Two cases of NDPH-like headaches after the acute bouts of reversible cerebral vasoconstriction syndrome (RCVS) have been reported [36]. These cases suggest that either they belong to a subtype of NDPH, which shares a similar etiology with the RCVS-spectrum disorders; or vasoconstriction in RCVS may be regarded as a trigger for NDPH-like headache. Lee et al. reported two unrelated pediatric patients with sphenoid sinusitis without nasal symptoms but only typical NDPH presentations [37]. Evens and Timm reported

a patient with typical NDPH presentations and benign non-toxic multinodular goiter which compressed carotid and vertebral artery, and the headache was completely resolved after thyroidectomy [38]. Another case of NDPH secondary to skull base metastasis has been reported. Following radiosurgery, the patient had remarkable pain improvement (NRS score reduced from 10 to 2), but the headache remained daily persistent [39]. A recent case of NDPH secondary to low-pressure headaches due to CSF-venous fistula at the T7-8 paraspinal region has been reported. The headache is typical for NDPH and did not respond to medical treatment or epidural blood patch. However, after surgical ligation of the right T7/T8 nerve root, the headache was completely resolved [40]. Another case of Nutcracker syndrome (aortomesenteric compression) mimicking NDPH has also been reported, and the patient had nearly complete resolution of headache symptoms after aortomesenteric decompression [41].

Secondary headache with a typical NDPH presentation is rather common, and the causes are miscellaneous. Rozen proposed three T's to help to identify possible secondary causes [42•]. The first T stands for specific triggers, including viral illness, cervical spine positioning, Valsalva event, or drug exposure; the second T for Trendelenburg position, headache worsening, and relieving factor associated CSF pressure/volume changes; and the third T for Thunderclap headache. The proper identification of a secondary cause provides the possibility to treat the NDPH-like headache by treating the underlying secondary conditions. Additionally, the miscellaneous secondary causes suggest there may be more than one mechanism in switching the trigemino-nociception from the "no pain" to "persistent pain" state. This mechanism is not restricted to the cranial region; nor is it necessarily in the CNS including the spinal cord. Therefore, the exclusion of a secondary cause of NDPH may sometimes be difficult.

Treatment Options

In a retrospective non-controlled study, greater occipital nerve block relieved headaches in 33% of pediatric NDPH patients with a favorable side effects profile [43]. Nerve blocks, not restricted to the occipital nerve, were commonly applied to pediatric/adolescent patients with NDPH, up to 67%, based on the acceptable side effects profiles; however, its efficacy has not been well-studied or established [44]. One adult study even tried multiple cranial blocks simultaneously, and out of ten NDPH patients, nine had no response, and one patient had only partial response [45]. A recent study followed up the long-term outcomes of NDPH patients with migrainous features who received occipital nerve stimulation. After a median of eight years, only one out of nine patients still showed positive responses to occipital nerve stimulation. The accumulating evidence suggests occipital nerve block/stimulation is probably not effective in most patients. However, occipital nerve block/stimulation still works against some other CDH than NDPH, suggesting different underlying pathophysiology of NDPH compared to the other CDH diagnoses [46]. In sum, the evidence to support nerve block in patients with NDPH is until today insufficient and needs further investigation.

Another study explored the role of subanesthetic dosage of ketamine infusion in refractory headache patients, and 8 out of 14 (57.1%) NDPH patients had an average of ≥ 1.5 (0–10 scale) reduction in pain intensity [47]. Ketamine works as an NDMA receptor antagonist, and this study suggests a potential role of NDMA in the pathogenesis of NDPH. Another study investigated the role of OnabotulinumtoxinA following the PREEMPT protocol [48] in NDPH patients. Of the 12 patients, after two sessions of OnabotulinumtoxinA therapy, i.e., in 6 months, six (50%) had a reduction in headache frequency, and five out of ten (50%) had a reduction in headache severity. Even slightly more patients responded to the treatment after receiving four sessions of treatment [49]. However, in this study, the reduction in headache frequency and intensity was not clearly defined. These results await further replication studies.

Elevated CSF tumor necrosis factor-alpha (TNF-α) levels have been found in 19 of 20 NDPH patients, as well as 16 of 16 CM patients; however, the serum TNF-alpha levels were normal, suggesting a specific role of CNS inflammation, not systemic inflammation, in chronic daily headache disorders, including NDPH [50]. Venlafaxine, a serotonin–norepinephrine reuptake inhibitor (SNRI), commonly used to treat depression, may inhibit the upregulation of TNF-α [51]. Tariq et al. reported a patient with a 6-year history of NDPH, which had not responded to more than 20 different medical treatments. The patient had dramatic headache reduction in headache intensity (from 9 to 3, 0–10 scale) after three months of venlafaxine treatment up to a dosage of 300 mg daily. Headache recurred after 3 weeks of venlafaxine wash out but remitted again after reinstation of venlafaxine treatment [52]. This is only one single case, but the role of venlafaxine and/or other TNF-alpha antagonist may be worthy of further investigation.

Conclusion

NDPH as a clinical syndrome is featured by the pathognomonic daily persistent headache with an acute onset. Miscellaneous secondary causes suggest the heterogeneous nature of NDPH-like headaches: both primary and secondary causes etiologies may lead to the same clinical presentation. Still, under the current diagnostic criteria, NDPH was listed as a primary headache disorder; secondary causes should be excluded, which is not easy and may sometimes

be underrecognized. To better understand whether the arbitrary differentiation of primary and secondary NDPH is reasonable, we need head-to-head comparison studies to see whether the clinical course of primary and secondary NDPH differs and to investigate whether it is meaningful to define a headache syndrome when the pathophysiology remains largely unknown [53]. Perhaps the more critical question to ask in NDPH, regardless of a primary or secondary etiology, is the biological switch from "no headache" to "persistent headache." Primary or secondary causes may be simply the internal and external force that turned the switch on.

Compliance with Ethical Standards

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.

Conflict of Interest The authors have no financial conflict of interest with any content of this manuscript.

References

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

1. Vanast W. New daily persistent headaches: definition of a benign syndrome. Headache. 1986;26:317.
2. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38:1–211.
3. Takase Y, Nakano M, Tatsumi C, Matsuyama T. Clinical Features, Effectiveness Of Drug-Based Treatment, and Prognosis of New Daily Persistent Headache (NDPH): 30 Cases in Japan. Cephalalgia. 2004;24:955–9.
4. Robbins MS, Grosberg BM, Napchan U, Crystal SC, Lipton RB. Clinical and prognostic subforms of new daily-persistent headache. Neurology. 2010;74:1358–64.
5. Peng K-P, Fuh J-L, Yuan H-K, Shia B-C, Wang S-J. New daily persistent headache: Should migrainous features be incorporated? Cephalalgia. 2011;31:1561–9.
6. Santoni JR, Santoni-Williams CJ. Headache and Painful Lymphadenopathy in Extracranial or Systemic Infection: Etiology of New Daily Persistant Headaches. Intern Med. 1993;32:530–2.
7. Li D, Rozen TD. The clinical characteristics of new daily persistent headache. Cephalalgia. 2002;22:66–9.
8. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. Cephalalgia 2004;24 Suppl 1:9–160.
9.• Rozen TD. Daily persistent headache after a viral illness during a worldwide pandemic may not be a new occurrence: Lessons from the 1890 Russian/Asiatic flu Cephalalgia. 2020;40:1406–1409. **Narrative review of NDPH-like headaches in the history.**
10.• Caronna E, Ballvé A, Llauradó A, et al. Headache: A striking prodromal and persistent symptom, predictive of COVID-19 clinical evolution Cephalalgia. 2020;40:1410–1421. **First description of NDPH-like headaches after aute COVID-19 infection.**
11. Prakash S. New Daily Persistent Headache: Disease or Syndrome? Headache. 2013;53:678–9.
12. Scher AI, Stewart WF, Liberman J, Lipton RB. Prevalence of frequent headache in a population sample. Headache. 1998;38:497–506.
13. Castillo J, Muñoz P, Guitera V, Pascual J. Kaplan Award 1998. Epidemiology of chronic daily headache in the general population. Headache. 1999;39:190–6.
14. Baron EP, Rothner AD. New daily persistent headache in children and adolescents. Curr Neurol Neurosci Rep. 2010;10:127–32.
15. Koenig MA, Gladstein J, McCarter RJ, Hershey AD, Wasiewski W, Pediatric Committee of the American Headache Society. Chronic daily headache in children and adolescents presenting to tertiary headache clinics. Headache. 2002;42:491–500.
16. Bigal ME, Sheftell FD, Rapoport AM, Lipton RB, Tepper SJ. Chronic daily headache in a tertiary care population: correlation between the International Headache Society diagnostic criteria and proposed revisions of criteria for chronic daily headache. Cephalalgia. 2002;22:432–8.
17. Grande RB, Aaseth K, Lundqvist C, Russell MB. Prevalence of new daily persistent headache in the general population. The Akershus study of chronic headache. Cephalalgia. 2009;29:1149–55.
18. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013;33:629–808.
19. Diaz-Mitoma F, WalterJ V, DavidLJ T. Increased frequency of Epstein-Barr virus excretion in patients with new daily persistent headaches. The Lancet. 1987;329:411–5.
20. Bordini CA, Valença MM. Post-Dengue New Daily Persistent Headache. Headache. 2017;57:1449–50.
21. Sampaio Rocha-Filho PA, Voss L. Persistent Headache and Persistent Anosmia Associated With COVID-19. Headache. 2020;60:1797–9.
22. Rozen TD. Triggering Events and New Daily Persistent Headache: Age and Gender Differences and Insights on Pathogenesis-A Clinic-Based Study. Headache. 2016;56:164–73.
23.• Rozen TD. New daily persistent headache (NDPH) triggered by a single Valsalva event: A case series Cephalalgia. 2019;39:785–791. **Specific trigger of NDPH suggestive of potential pathophysiological connection.**
24. Papetti L, Sforza G, Tarantino S, Moavero R, Ruscitto C, Ursitti F, Ferilli MAN, Vigevano F, Valeriani M. Features and Management of New Daily Persistent Headache in Developmental-Age Patients. Diagnostics. 2021;11:385.
25.• Strong E, Pierce EL, Langdon R, Strelzik J, McClintock W, Cameron M, Furda M, DiSabella M. New Daily Persistent Headache in a Pediatric Population J Child Neurol. 2021;36:888–893. **Large cohort of pediatric NDPH patients.**
26. Evans RW. Primary non-continuous new daily persistent headache: Seven cases and proposed diagnostic criteria. Cephalalgia Rep. 2021;4:251581632199834.
27. Silberstein SD, Lipton RB, Solomon S, Mathew NT. Classification of Daily and Near-Daily Headaches: Proposed Revisions to the IHS Criteria. Headache. 1994;34:1–7.
28. Paliwal VK, Uniyal R, Aneez A, Singh LS. Do paroxysmal hemicrania and hemicrania continua represent different headaches? A retrospective study. Neurol Sci. 2019;40:2371–6.
29. Palacios-Ceña D, Talavera B, Gómez-Mayordomo V, García-Azorín D, Gallego-Gallego M, Guerrero ÁL, Cuadrado M. The Day My Life Changed: A Qualitative Study of the Experiences of Patients With New Daily Persistent Headache. Headache. 2020;60:124–40.
30. Reidy BL, Riddle EJ, Powers SW, Slater SK, Kacperski J, Kabbouche MA, Hershey AD. Clinic-based characterization of continuous headache in children and adolescents: Comparing youth with chronic

migraine to those with new daily persistent headache. Cephalalgia. 2020;40:1063–9.
31. Rozen TD. New daily persistent headache: A lack of an association with white matter abnormalities on neuroimaging. Cephalalgia. 2016;36:987–92.
32. Aguiar de Sousa D, Geraldes R, Gil-Gouveia R, Sá JC. New daily persistent headache and radiologically isolated syndrome. J Neurol. 2013;260:2179–81.
33. Rozen TD, Beams JL. New daily persistent headache with a thunderclap headache onset and complete response to Nimodipine (A new distinct subtype of NDPH). J Headache Pain. 2013;14:100.
34. Jamali SA, Rozen TD. An RCVS Spectrum Disorder? New Daily Persistent Headache Starting as a Single Thunderclap Headache (3 New Cases). Headache. 2019;59:789–94.
35. Robbins MS, Evans RW. The heterogeneity of new daily persistent headache. Headache. 2012;52:1579–89.
36. Ling Y-H, Wang Y-F, Lirng J-F, Fuh J-L, Wang S-J, Chen S-P. Post-reversible cerebral vasoconstriction syndrome headache. J Headache Pain. 2021;22:14.
37. Lee J, Rhee M, Suh ES. New daily persistent headache with isolated sphenoiditis in children. Korean J Pediatr. 2015;58:73.
38. Evans RW, Timm JS. New Daily Persistent Headache Caused by a Multinodular Goiter and Headaches Associated With Thyroid Disease. Headache. 2017;57:285–9.
39. Ogunlaja O, Zhang N. New Daily Persistent Headache Syndrome Secondary to Clival Metastasis Within an Osseous Hemangioma. Headache. 2019;59:1609–10.
40. Duvall JR, Robertson CE, Whealy MA, Garza I. Clinical Reasoning: An underrecognized etiology of new daily persistent headache. Neurology. 2020;94:e114–20.
41. Stubberud A, Cheema S, Tronvik E, Matharu M. Nutcracker syndrome mimicking new daily persistent headache: A case report. Cephalalgia. 2020;40:1008–11.
42.• Rozen TD. The Three T's of NDPH (How Clinical Observations Have Led to Improved Treatment Outcomes). Headache. 2019;59:1401–1406. **Narrative review on potential secondary causes of NDPH.**
43. Gelfand AA, Reider AC, Goadsby PJ. Outcomes of Greater Occipital Nerve Injections in Pediatric Patients With Chronic Primary Headache Disorders. Pediatr Neurol. 2014;50:135–9.
44. Szperka CL, Gelfand AA, Hershey AD. Patterns of Use of Peripheral Nerve Blocks and Trigger Point Injections for Pediatric Headache: Results of a Survey of the American Headache Society Pediatric and Adolescent Section. Headache. 2016;56:1597–607.
45. Miller S, Lagrata S, Matharu M. Multiple cranial nerve blocks for the transitional treatment of chronic headaches. Cephalalgia. 2019;39:1488–99.
46. Lagrata S, Cheema S, Watkins L, Matharu M. Long-Term Outcomes of Occipital Nerve Stimulation for New Daily Persistent Headache With Migrainous Features. Neuromodulation Technol Neural Interface ner. 2020;13282.
47. Pomeroy JL, Marmura MJ, Nahas SJ, Viscusi ER. Ketamine Infusions for Treatment Refractory Headache. Headache. 2017;57:276–82.
48. Aurora S, Dodick D, Turkel C, DeGryse R, Silberstein S, Lipton R, Diener H, Brin M. OnabotulinumtoxinA for treatment of chronic migraine: Results from the double-blind, randomized, placebo-controlled phase of the PREEMPT 1 trial. Cephalalgia. 2010;30:793–803.
49. Ali A, Kriegler J, Tepper S, Vij B. New Daily Persistent Headache and OnabotulinumtoxinA Therapy. Clin Neuropharmacol. 2019;42:1–3.
50. Rozen T, Swidan SZ. Elevation of CSF Tumor Necrosis Factor ? Levels in New Daily Persistent Headache and Treatment Refractory Chronic Migraine. Headache. 2007;47:1050–5.
51. Li Z, Qi D, Chen J, et al. Venlafaxine inhibits the upregulation of plasma tumor necrosis factor-alpha (TNF-α) in the Chinese patients with major depressive disorder: A prospective longitudinal study. Psychoneuroendocrinology. 2013;38:107–14.
52. Tariq Z, Board N, Eftimiades A, Ibrahim R. Resolution of new daily persistent headache by a tumor necrosis factor alpha antagonist, Venlafaxine. SAGE Open Med Case Rep. 2019;7:2050313X1984780.
53. Lipton RB, Manack A, Ricci JA, Chee E, Turkel CC, Winner P. Prevalence and Burden of Chronic Migraine in Adolescents: Results of the Chronic Daily Headache in Adolescents Study (C-dAS): May 2011. Headache. 2011;51:693–706.