Reviews/Mini-Reviews

CLINICAL and TRANSLATIONAL NEUROSCIENCE

Clinical & Translational Neuroscience
January–June 2019: 1–7

DOI: 10.1177/2514183X19834768
journals.sagepub.com/home/ctn
SAGE

Neck pain and migraine: Association or cause?—A narrative review of the literature

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Abstract

This literature review deals with the question whether neck pain (NP) constitutes a symptom or a trigger of migraine. Firstly, a short survey about some techniques for measuring NP in association and relation with migraine is presented. Secondly, the arguments about NP as a symptom or a trigger of migraine are being reviewed and compared. The main questions are the following: Which tests can be used to distinguish NP as a trigger or a symptom of migraine? Is a therapy for NP an adequate method to treat migraine? Finally, the pros and cons of NP as a symptom or as a trigger will be reviewed, and possible treatment options will be suggested. This review found that no reliable and standardized tests exist to classify NP in relation to migraine. However, there is a comparability among these studies due to the common use of migraine definition in the "International Classification of Headache Disorders." Regarding the quality and methods, different types of studies were analyzed, for example, retrospective, prospective, and cross-sectional studies. Nevertheless, none of these types are specifically suited to show a causality between NP and migraine. In order to do this, the authors would suggest using a randomized controlled study. Another adequate study design might be a population-based case–control crossover study and calculating the population attributable risk. Furthermore, the pathophysiology of NP in migraine patients should be investigated in more detail. Besides their questionable suitability for showing a connection between NP and migraine, some studies were additionally subject to a population and selection bias. To sum up, part of the authors in the reviewed literature generate the hypothesis that NP is more often a symptom than a trigger of migraine. However, due to methodological flaws, more studies are necessary to confirm this hypothesis.

Keywords

Migraine, headache, neck pain, diagnosis, trigger, treatment

Introduction

Worldwide 10–15% of all adult people are affected by migraine headache.[1] Neck pain (NP) is a common phenomenon with a point prevalence between 5.9% and 38.5% with a mean prevalence of 7.6%.[2] It is known that there exists a temporal correlation between migraine and NP.[3] However, it is not clear yet whether NP should be rated as a symptom or a trigger (i.e., causal) of migraine. Even though migraine has been known as an illness for a long time,[4] the optimal treatment for many patients is still unclear.[5] Interestingly, NP was found to be associated with migraine more often than nausea, which is a characteristic symptom of migraine.[3,4,6,7] In the past, NP has frequently been neglected and not seen in correlation with migraine. Instead, NP was interpreted as a result of stress, tension, or physical burden.[6] According to other authors, NP is mostly ignored or only noticed when a distinct migraine attack is already present.[8] As a result, a causality between migraine and NP was considered less likely, which might have led to a less effective treatment. Therefore, it is

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important to find out whether NP may represent a possible trigger for migraine in order to further explore treatment options.[9]

In other parts of evidence-based medicine, including manual therapies, a great deal of attention is paid to the relation between NP and migraine.[10] Many patients are being treated based on the hypothesis that NP is a possible reason for migraine. However, since no causality has been proven yet, there is a risk of unnecessary therapies, and vice versa, patients may not be receiving the most efficient treatment.[11]

Methodology

By using a structured search strategy with inclusion and exclusion criteria, a search in the "PubMed" database was conducted in July 2017. The inclusion criteria were as follows: migraine associated with NP, a possible trigger, combined with manual treatment options, manual therapeutic strategies for migraine and NP, and self-reported and NP in correlation with the frequency of migraine attacks. On the other hand, the following exclusion criteria were applied: any comparison of costs, all not in relation to migraine and NP, published before 1995, case studies with less than 10 people, treatment methods for migraine without the influence of NP, other chronic illnesses, lifestyle, sociodemographic factors, pain management, articles not in English, patients on average younger than 18 years of age, and no full-text available. As search terms, migraine + synonyms and NP + synonyms were used. Additionally, search items such as tests and validity + synonyms were used. Please refer to the supplementary material for more detail.

The focus of this review lies on different theories about NP in correlation with migraine, either as a symptom or as a trigger. To provide some background about the possible identification criteria and measurement methods for migraine and NP, the following sections provide a short overview of techniques being used in the mentioned studies. It should however not to be interpreted as a complete analysis.

Criteria in migraine patients

In order to identify migraine patients, the criteria of the "International Classification of Headache Disorders" (ICHD) were cited the most (with a total of 20 times).[4,7] However, since the included articles were published in different years, the edition of the classification is not the same throughout, which might lead to different results. Nevertheless, the criteria provide a good basis for a cross-comparison of the patients, as a common baseline was used. It should be noted that the ICHD criteria cannot be used for measuring a correlation between NP and migraine but are merely a method to distinguish migraine from other kinds of headache.

Measurement of NP in migraine patients

Different studies used different quantification methods to obtain their results. This section will provide a short outline for the most commonly mentioned ones. The "pressure pain threshold" method was used seven times and thus the most frequent. It is a method for quantifying the pain threshold.[11–18] All tests have been conducted using an electronic pressure algometer, although the devices differ in terms of the producer and the surface size (0.5 vs. 1.0 cm^2). Even though the tested anatomic areas depended on the questionnaire, six of seven studies took at least one measurement at the back of the head.[12] In detail, four measured in the upper trapezius muscle,[11,14,16,17] two in the trigeminal area,[12,18] and one in the areas over the C2 nerve root, the greater occipital nerve, and the transverse process of C4.[13] All mentioned studies defined the threshold as changeover to pain and not absolute pain tolerance. This means that the patients were asked to report as soon as they feel a painful sensation.[11–14,16–18] According to the authors, the pain threshold is lower in migraine patients compared with the healthy control group.[14,17] A temporary or causal link between NP and migraine was however not explicitly examined in the aforementioned studies.

"Cervical range of motion" (CROM) was used in three studies, all of which used the same measuring instrument. The procedure of measuring the range of motion was similar in these three studies. The patients had to sit in a chair and make movements like flexion, extension, lateral flexion, and rotation, while the CROM device was measuring the movement.[11,13,19] This method was examined for NP as neither a symptom nor a trigger of migraine but solely for the purpose of determining the cervical spine range of motion.[20]

In addition, "diaries and interviews" seem to be a helpful tool, for example, to inform patients about medication intake as well as to find associated symptoms or potential triggers.[3,6,8,21] Some studies used this as the method of choice to draw conclusions regarding the relation between NP and migraine.[3,6,8,22] Diaries and interviews are subjective methods that are frequently used in association with migraine patients, allowing an adequate insight into their well-being.[23] Nonetheless, a disadvantage seems to be the lack of a standardized system, which makes comparing studies difficult.[3,6,8,22] The electronic diaries may be the most valuable because they are completed after every migraine attack or even daily.[24,25] While a high compliance is necessary, it may help authors to generate hypotheses about the association of NP and migraine.[3,6,8,22] Further, electronic diaries are more complete than paper diaries.[25] Still, the conclusions should be compared with caution, owing to divergent methodology and questions.

Results

In summary, the question whether NP constitutes a symptom or a trigger of a migraine has been approached with

various methods. Although the ICHD criteria have become the most commonly applied diagnostic method for migraine patients,[26,27] we still lack a valid method to identify NP as a symptom or as a trigger of migraine correctly. A further problem is the overlapping of different headache types in some patients: for example, migraine, cervicogenic headache, and tension headache.[4,7] Because some patients may suffer from more than one headache, which may have a time of overlap, the diagnosis can be challenging in certain cases. This outlines the importance of strictly applying the ICHD criteria as a common baseline method to create comparable patients, so that the best therapy method can be elaborated.

NP as a symptom in relation to migraine (temporal association)

In all of the included articles, no appropriate test or method was found to show a causal connection between NP and migraine. Apart from that, no pathophysiological mechanism that would explain such a causality is mentioned in the included articles. Due to the above problems, it is difficult to determine effective therapy options for NP and compare them with conventional medications such as non-steroidal anti-inflammatory drugs (NSAIDs) and/or triptans. Nevertheless, some authors have conducted comparative studies on that subject.[8,11] Most of the results discussed in this review are based on frequent diary entries from the patients.[3,6,8,22] To sum up the results for NP as a symptom, two studies concluded that NP may be a symptom of a migraine attack.[24,28] As such, Giffin et al.[24] observed the premonitory symptom of a stiff neck, which is considered as NP in this review. The authors therefore generated the hypothesis that NP may be a migraine symptom, due to its frequent temporal association with headache.[3,28,29] Similarly, Lampl et al. hypothesized that NP occurs as a symptom and part of the migraine attack in at least a subgroup of migraine patients.[28] Other studies support this finding, since they found NP to be more often associated with migraine than nausea, which is, after all, a characteristic symptom of migraine.[3,28] A possible counterargument is the general lifetime prevalence of NP, which is about 14–71%[2] or even 76.4%.[12] Approximately 30–50% of the general population suffers from NP at least once a year.[30] Another cross-sectional study shows a 1-year prevalence of 68.4%.[12] Furthermore, women are more often affected by NP and migraine than men.[12] A higher prevalence of a disease in an examined population generally results in an increased positive predictive value, even though the specificity and sensitivity did not change. The question arises whether NP is measured accidentally.[31,32] To our knowledge, this was not taken into consideration in the examined studies. More epidemiological research is needed to answer the question of causality adequately.

NP as a trigger in correlation with migraine (causation)

Diverse trigger factors for migraine attacks have been identified, one of the most common being stress named psychological distress.[5,33,34] In one of the studies, Kelman explored triggers for migraine in over 1200 patients and found that 75.9% of all migraineurs might be able to identify specific trigger factors.[5] The number of patients naming at least one trigger increased to 94.6%, as soon as they had a specific list with causative factors. Each patient named 6.7 triggers in average.[5] Another study by Baldacci et al., investigating 120 migraineurs, found even more triggers per person.[33] Spontaneously, 72.5% of the patients identified at least one possible cause for migraine, while when given a specific list, every patient could name at least one trigger.[33] On average, 7.2 triggers were mentioned, depending on the patients' allodynia. In detail, an average of 5.6 in nonallodynic patients and 9.0 in allodynic patients was reported.[33] Similar findings were confirmed by Chakravarty who stated that trigger factors are present in every migraine attack.[34] All that being said, trigger identification is individual and highly depends on patient perception. In other words, if a symptom is temporarily correlating with migraine, patients might point it out as a trigger, even though it could be just a symptom or premonitory symptom.

Discussion

This review tried to answer the question of NP being a symptom or a trigger of migraine, hence with NP being an associated factor or a causal factor in migraine patients. Or in other words, it is possible that NP is a symptom and a trigger in migraine.

NP was found to have a connection with migraine, occurring shortly (2 h) before and during the attack.[3,28,29] If NP was present only by chance during the migraine attack, logic dictates that it should also have been found independent of the attacks. However, two studies show a temporal correlation between the prevalence of migraine and NP.[3,6] Therefore, there is a probability of NP being associated with migraine. However, a closing causality has not been proven yet.

While attempting to explore NP in migraineurs, an association between NP and a bad treatment outcome was found.[8,24] In other words, the presence of NP in patients during the migraine episode might be discussed as a possible predictor for poor outcome.[8,24] Among other things, the prevalence of NP during migraine attacks is correlated with a higher "neck disability score," a questionnaire that measures the influence of NP on daily activities based on 10 items.[15] Consequently, the disability in daily life (e.g., problems with concentration, work, and sleep) is higher.[15] Moreover, the study found NP to be more present in chronic migraineurs and so stated the hypothesis of a

correlation between the frequency of NP and disability.[15] Similarly, Ford et al. found that NP is an important predictor for disability.[35] On the contrary, Ashina et al. found a stronger correlation between episodic migraine and NP, rather than chronic migraine. These differences in frequency can likely be explained by the different study models used. Ashina et al. recruited people with migraine for a cross-sectional study from the general population,[12] while Ford et al. only included migraine patients from a headache and pain center for their retrospective study.[35] Other distinctive discrepancies were found in the way of diagnosing migraine. Other apparent methodological differences in the above studies were the use of questionnaires and interviews in one study,[12] while, in the other, diagnosis was made by a neurologist.[35] As a result, a valid comparison is not possible. However, it is important to note that the methodology and design of aforementioned studies, while being suited for some analyses, are not adequate to draw conclusions on the question of causality.[3,24,28,29,31,36] Additionally, a substantial amount of studies were subject to a potential selection and population bias. For example, an important bias in these studies was introduced by exclusively including people from a headache clinic.[3,5,6,8,11,12,15,24,28,29,33,35,37] As a result, the findings cannot be directly generalized for all migraine patients. Another factor is the different understanding of trigger, premonitory symptom, and symptom. There exists no clear definition and differentiation yet, and so the comparison in this review is to be handled with care.

The increased disability level in migraineurs associated with the prevalence of NP may be interpreted as NP representing allodynia in migraine patients.[3,6,8] Based on the assumption that allodynia has an influence on the chronicity of migraine, one may explain why NP is observed more often in patients with higher migraine attack frequencies.[3,6,8,33] Allodynia is described by the authors as a possible risk factor for changeover from episodic to chronic migraines.[38] Because the studies are not randomized controlled trials nor of cross-sectional design, a causality cannot be proven conclusively.[3,6,31] The underlying mechanism for NP in migraineurs might be around the trigeminocervical complex (TCC), which is discussed in more detail in a review by Bartsch et al.[39] and an article by Busch and Goadsby.[40] This pathophysiological process is based on two mechanisms: Firstly, there is a convergence from nociceptive afferences, both functional and anatomical. Secondly, there is a sensitization of the trigeminocervical neurons.[39,40] It has been shown that nociceptive afferences from the meninges and the cervical structures demonstrate an overlap, with the neurons of the TCC being the main switches.[39,40] This helps to explain how a central pain such as migraine can interconnect through TCC and partially be presented as the symptom of NP.[39–41] Moreover, the respective neurons are sensitized during a migraine attack that can potentially lead to the development of allodynia and hypersensitivity, a phenomenon often observed in migraineurs.[39,40] Another question that needs to be reviewed is whether NP has a common pathophysiology with migraine. The discovery that NP represents allodynia,[3,6,8] as well as the evidence that an acute triptan therapy is helpful against NP in migraineurs would lead to this conclusion.[8] This would also explain why NP is more often present when the frequency of the migraine attacks increases.

To summarize, all abovementioned studies demonstrate NP being a symptom in some migraine patients

Baldacci et al. found that, with an increasing number of triggers, the severity level of allodynia increases.[33] This leads to the assumption that triggers and allodynia share a common way in pathophysiology as mentioned earlier in the discussion on the TCC. Should triggers and allodynia indeed reinforce one another, then this should be taken into consideration for therapeutic purposes to decrease the risk of changeover into chronic migraines.[33]

Some studies dealt intensively with the question whether NP is an influenceable factor, a premonitory symptom or a consequence of migraine.[5,12,28,29] In contrast to the studies above, Kelman noticed that there is not always a trigger factor present in an attack.[5] Just 8.8% of the examined patients reported to often be able to define triggers.[5] NP, defined as "NP that worsened headache or is the reason of the headache," was mentioned in 38.4% of the cases as an occasional trigger and in 10.6% as a frequent one.[5] This puts NP compared with other factors, for example, stress, in the midfield of all triggers,[5] which leads to the assumption that NP is experienced as a trigger for some part of the migraineurs.

Furthermore, Kelman found an overlap between the trigger "NP" and the localization of migraine in the area of the neck.[29] A statistically significant difference regarding the neck area was spotted by comparing chronic and episodic migraine.[29] This supports the report that NP occurs more often in chronic migraineurs.[15] Since patients were recruited exclusively in a headache center, confounders as well as a population bias may have had an influence on the presented results.[5] This and the large number of patients ($n > 1200$) may result in finding a significant correlation, which in reality is potentially small.[5]

In a prospective cohort study, Lampl et al. also tried to answer the question whether NP is part of the migraine attack or a premonitory symptom.[28] Just 7.4% of the included patients noticed NP 2–48 h before the attack, and nearly half of them noted additional symptoms that can already be associated with migraine.[28] Eventually, the study concluded that NP is rather a part than a premonitory symptom of migraine.[28] Due to the different study designs, it must remain open whether, in the 7.4%, NP is a trigger or a symptom. As a matter of fact, this is true for all of the above studies looking at NP as a trigger.[5,28,33,34] On the one hand,

these articles[33,34] showed that every migraineur has at least one trigger. On the other hand, the theory is mentioned that trigger factors are present in attacks anyways, whether you find them or not.[33,34] As triggers are manifold and dependent on individual perception, this might explain why the studies have slightly different outcomes.

This leads to the conclusion that, while some patients interpret their NP as a trigger, one cannot rule it out as a premonitory symptom and vice versa. To answer these questions, a randomized controlled trial with migraine patients should be conducted. Here, migraine patients could be divided into two groups, where one group would get a manual neck treatment and exercise for the neck musculature, while the other group would treat migraine using the usual medication only. If a decrease of migraine attacks were to be found in the first group, this would suggest that NP is also a trigger in migraine. Another possibility is to find the pathophysiological way of NP in migraine patients and try to answer the question whether NP is a symptom or/and a trigger in this way.

Interestingly, some studies have shown manual therapeutic interventions to be effective in the treatment of migraineurs.[9,11,42] Different explanations are possible. One study compared two patient groups with 25 people each:[11] One group got medication only, and the other one an extra 8 h of passive physiotherapy. Both groups showed a significant improvement of the migraine. However, as no additional effect was shown in the physiotherapy group after 4 weeks of intervention in terms of headache frequency and intensity, this effect can be explained with the medication only. That being said, there still was evidence in the physiotherapy group with respect to an increased satisfaction with the therapy.[11] It is just a comparative study with only physiotherapy as treatment or just usual treatment of migraine, since both groups received the same, new medication. Moreover, $n = 25$ subjects per group is not sufficient to get significant results with only two interventions at the same time. To sum up, the effectiveness of passive physiotherapy in migraineurs cannot be judged. This example will help to understand the other results.[9,42] As such, therapy by qualified care professionals like chiropractors or physiotherapists may have an influence on migraineurs and may help in combination with other factors to improve the satisfaction and lower the nociceptive afferences.[11] None of the included studies prove that treating NP exclusively with manual therapies can reduce migraine attacks. This correlation supports the theory that NP is a trigger in at least some migraineurs.

Conclusions

The main goal of all therapeutic approaches is the patients' welfare. While therapeutic strategies for migraine have significantly improved, that is, using new medications among other options, not all patients are treated optimally.[5] That is why NP treatment is an important and promising topic, in the management of migraine patients. For optimal therapy, the underlying pathophysiology must be well understood. However, this basic question has not yet been answered adequately with respect to the relation between NP and migraine.[3,6]

Two predominant scenarios are existing where NP may represent an important factor of migraine treatment and could change a lot in the patients' treatment: Assuming that NP may represent a trigger for migraine, caused by peripheral mechanisms in genetically predisposed patients, one could assume that manual interventions such as chiropractic, physiotherapy, and massage therapy could help to treat NP, thereby reducing the intensity and/or frequency of migraine attacks; secondly, assuming that NP may be a premonitory symptom (like aura) of central origin due an impending migraine attack, hence before the headache has even fully developed. This is interesting, as one may argue that an early and acute treatment of the migraine attack might be effective during the premonitory phase, hence when the patient is experiencing NP before the migraine. As such, patients could be treated earlier by means of medication, relaxation techniques, and so on for improved quality of life. Specifically, one could start earlier with a specific medication as an example. Apart from that, NP as premonitory symptom but with a peripheral etiology would be difficult to act on. Since in this case NP starts only a few hours before the actual migraine attack, there is a close window of time for possible manual interventions, apart from self-mobilization, trigger point therapy, warm or cold application, or NSAIDs. We would like to point out that the frequency and period of drug intake need to be monitored carefully to avoid any risk of medication overuse headache.[43] Furthermore, we would like to stress the fact that the two aforementioned points are assumptions, as there are still limited data on the subject that does not allow for conclusions in either direction.

First of all, the question whether NP is a symptom or a trigger in correlation with migraine cannot be answered conclusively in this review. None of the included studies used a study design that would allow to prove a causality between NP and migraine. There is no clear support for NP as trigger. Hypothetically however, a solution including both NP as a (premonitory) symptom and sometimes as a trigger is possible. Two of the included articles clearly support the hypothesis of NP being a premonitory symptom and a part of the migraine attack itself.[24,28] Accordingly, the possibility of an early treatment might exist, meaning that an acute therapy could be started at the beginning of NP, in order to prevent a full migraine attack, or at least reduce its symptoms.[8]

At the moment, there are no valid clinical or diagnostic tests nor standardized approaches to clearly distinguish NP during migraine as a trigger or a symptom. Although different clinical tests have been used, the international classification criteria[4,7] often were mentioned in the covered literature. Even if there exists an overlap in the

different types of headache, these criteria are the current "gold standard" for a reliable diagnosis of migraineurs.[4,7] The "neck disability index" on the other hand could verify a correlation between the frequency and the level of disability.[15] Disability due to NP is more predominant and described in patients with frequent attacks.[15] Diaries represent another useful and informative outcome measure for the stage of pain at treatment, presence of headache in general, and associated symptoms and risk factors for migraine,[3,6,8,22] providing an insight into the subjective well-being of the patients. However, necessary conditions for these are both high patient compliance and reliability.[22]

Two studies suggest that NP may be a premonitory symptom and an expression of the impending migraine attack.[24,28] These findings are supported by the observed high prevalence of NP during migraine attacks.[3,28] Another study reports that NP can be used as a marker for a poor treatment outcome.[8] Nevertheless, causality between NP and migraine cannot be verified, due to unsuitable study design.

It has been well established that there exist different triggers for the development of migraine attacks.[5,33,34] Among those, some patients clearly identify NP as a trigger for their migraine.[5] This remains a subjective assessment however. Although pathophysiological mechanisms are known, there is a lack of clear evidence for adequate recommendations with respect to the cotreatment of NP.

Additionally, no statement about the efficacy of manual therapeutic interventions can be made. Although, a therapy might help, its improving effect is probably rather based on the weekly care and lowering of the nociceptive afferences than decreasing a trigger such as NP.[11] Finally, NP seems to be a symptom of the attack in most patients and a provoking trigger only in small parts of the patients. For this small group, manual therapy can be recommended, even though causality has not been proven.

Open questions and outlook

Following questions remain open: What is the neuropathophysiology of NP in migraine? How can one use the theory of TCC for all migraineurs? What are the consequences in therapy management? What kind of tests or questionnaires could help to classify NP correctly and treat the patient most efficiently? Will treatment of NP be able to prevent patients from chronification of the migraine?

Further research by the help of well-designed studies will be necessary to answer these open questions.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Supplemental material

Supplemental material for this article is available online.

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