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## Letter to the Editor: New Observation

# Nerve Blocks (Follow the Pain Pattern) for New Daily Persistent Headache

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A 25-year-old female with depression (on duloxetine 60 mg) presented to the headache clinic with a 1-year history of new onset continuous and unremitting headache. Her headache was localized to the bilateral frontotemporal and occipital regions. She described it as throbbing and pressure-like, associated with photophobia, phonophobia and nausea. It was not associated with positional change or Valsalva maneuvers. The onset of her headaches developed suddenly on a specific date and time. She denied a preceding history of headache, trauma, infection, surgery, travel or sick contacts. Over-the-counter analgesics (ibuprofen and acetaminophen) and rizatriptan provided no relief. She denied other neurological and systemic symptoms. She was diagnosed with migraine by her general neurologist.

Her neurological examination was unremarkable. Magnetic resonance imaging of her head with gadolinium did not reveal any structural lesions to explain her headache. She was diagnosed with suspected new daily persistent headache (NDPH) based on her clinical history and normal imaging. She was prescribed amitriptyline, titrated from 10 mg up to 30 mg and was on it for 3.5 years. Topiramate, titrated up to 100 mg, was added for 1 year. Both medications were discontinued due to intolerance and ineffectiveness. Later, divalproex acid, titrated up to 500 mg twice daily was prescribed and tried for 6 months, but it was discontinued due to brain fog and lack of efficacy. She did not have drug insurance coverage for onabotulinum toxin A or calcitonin gene related peptide monoclonal antibodies or receptor antagonists.

Occipital nerve blocks (NB) with local anesthetics were suggested based on failure to oral medications and lack of insurance coverage for advanced headache treatments such as onabotulinum toxin A or calcitonin gene related peptide-based treatment. Bupivacaine 0.5% (5 mg/ml) was injected to the greater (3 ml each side, 6 ml in total), lesser (2 ml each side, 4 ml in total). She had no relief after three sessions (1 month apart).

On further head and neck examination, she had tenderness on palpation in multiple nerve distributions including bilateral greater and lesser occipital, auriculotemporal, supraorbital and supratrochlear nerves. Extensive (Follow the pain pattern) NB were suggested as the next step of treatment based on her pain pattern and examination findings. Bupivacaine 0.5% (5 mg/ml) was injected to the greater (3 ml each side, 6 ml in total), lesser (2 ml each side, 4 ml in total), auriculotemporal nerves (1 ml each side, 2 ml in total), supraorbital and supratrochlear nerves (0.25 ml each side, 1 ml in total) bilaterally. Pain freedom was achieved within 15 minutes of administration and sustained for 4 weeks, with only mild reemergence of low-intensity headache 3 to 4 days before her next set of monthly injections. Subsequent injections provided a similar response for the next 3 months.

NDPH is a sudden onset continuous headache that lasts more than 3 months and is clearly remembered with pain becoming unremitting within 24 hours according to the International Classification of Headache Disorders 3rd edition.[1] The headache may be tension or migraine-like or both. It has two subtypes: a self-limiting subtype that typically resolves within several months without therapy, and a refractory subtype that is resistant to aggressive treatment regimens.[1]

Although the exact pathophysiology of NDPH remains unclear, previous studies have shown that it can be associated with recent history of infection, surgery, trauma, stressful events or it can be completely spontaneous.[2] Rozen and Swidan proposed that NDPH might be related to the release of proinflammatory cytokines resulting in central nervous system inflammation.[3] It may also be related to a hypersensitized state that was set off from an inciting event. The response to NB in patients with NDPH has been previously reported, however no guidance has been provided from previous literature on the details and who is appropriate and suitable to receive NB as a treatment for NDPH.

Robbin et al. identified 23 NDPH patients who received NB (bupivacaine) in painful areas that demonstrated improvement with headache severity. It provided a 60% acute response, consistent with at least one-day decrease in pain intensity.[4] However, no information was reported on how long the NB lasted and where the painful areas were. The painful areas may be reflective of the nerve distributions that are amenable to nerve blockade.

Puledda et al. found that among 22 children and adolescents with NDPH, 13 (59%) showed improvement after receiving greater occipital NB with a combination of 1% lidocaine and methylprednisolone. Additionally, 2 patients experienced sustained relief for over 3 months. Methylprednisolone and lidocaine were mixed, and no patients received local anesthetics alone.[5] It is unknown if

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the percentage response may be greater than 59% if other nerves were blocked.

In this case, bilateral NB to the greater, lesser, auriculotemporal, supraorbita, and supratrochlear nerves was suggested based on the pain pattern, and the response was dramatic. The positive response to NB was sustained, and the presence of wearing off before the next treatment suggests ongoing NB benefit. However, the duration of the analgesic effect from NB remains unpredictable. It could be hypothesized that following the pain pattern with NB injection can potentially be a treatment option for NDPH. By targeting the afferent transmission from the peripheral nerves (broader location and targeting more peripheral inputs if the exam is suggestive), it may contribute to a more adequate decrease in the afferent transmission to the dorsal root ganglion in the trigeminocervical complex (TCC) in the brainstem and upper cervical spine. Thus, it will decrease the projection from the TCC to the thalamus, from which thalamocortical neurons relay sensory information to multiple cortical areas, causing multiple features such as headache, photophobia, phonophobia and nausea. By modulating the afferent transmission from the peripheral nerves, it may contribute to decreasing trigeminal activation and reducing the risk of central sensitization and chronic pain. The inadequate response to the initial bilateral greater and lesser occipital NB may be related to inadequate peripheral afferent transmission blockade.

The advantage of NB lies in its tolerability and side effect profile. The use of NB can minimize the risk of developing medication overuse headache, which is often comorbid in NDPH patients. A clue to predict response to NB may be elicited during examination based on tenderness upon palpation in the nerve distribution (for example: greater and lesser occipital, auriculotemporal, supraorbital and supratrochlear nerves). Hence, local tenderness as a selection criterion should be considered in future trials. There are still many uncertainties beyond the location of peripheral NB, including the type of local anesthetics used (longer-acting vs. shorter-acting) and whether steroids are included. This is not a well-published area, and most studies are heterogeneous. Larger trials should be conducted to evaluate the efficacy of NB in NDPH patients.

**Data availability of material.** The material analyzed during the current case is available from the author on a reasonable request.

**Author contributions.** Dr Aqeel Almutairi & Dr Paul Cooper & Dr Tommy Lik Hang Chan assessed, examined the patient and wrote the manuscript.

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**Consent for publication.** Informed written consent was obtained from the patient.

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