



Primary Stabbing Headache

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Abstract

Purpose of Review To provide a comprehensive and updated review of the literature on primary stabbing headache.

Recent Findings Changes to the *ICHD-3* criteria have resulted in increased sensitivity to capture primary stabbing headache (PSH). According to the *ICHD-3*, the sharp stabbing pain is no longer restricted to the first division of the trigeminal nerve. Age, gender, and co-morbidities such as migraine seem to influence the prevalence of PSH. Subclassification into monophasic, intermittent, and chronic forms have been proposed in a recent prospective study and may be helpful from a prognostication perspective; however, further studies are required.

Summary Secondary etiologies for stabbing headaches are part of the differential diagnosis of primary stabbing headache; therefore, it is reasonable to perform neuroimaging. For severe frequent attacks, indomethacin continues to be considered first line. Other treatment options include COX2 inhibitors and melatonin.

Keywords Primary stabbing headache · Idiopathic stabbing headache · Ice pick headache · Jabs and jolts headache · Sharp short-lived head pain

Introduction

Primary stabbing headache (PSH) is a primary headache disorder that was first described by Lansche in 1964 as “ophthalmodynia periodica” [1]. PSH has been known as ice pick pains, jabs and jolts, needle-in-the-eye syndrome, and sharp short-lived head pain. The latest *International Classification of Headache Disorders, Third Edition (ICHD-3)* criteria was published in 2018 and describes PSH as “transient and localized stabs of pain in the head that occur spontaneously in the absence of organic disease of underlying structures or of the cranial nerves.”

Epidemiological of Primary Stabbing Headache

The epidemiological data from current studies is variable. Age, gender, referral bias, definition of PSH, and co-

morbidity with other headache disorders appear to affect this data.

The reported prevalence of PSH ranges from 0.2 to 35% [2–5]. Isolated PSH (exclusion of other primary headache disorders) had a prevalence of 1.5% in a study of patients presenting to a tertiary neurology clinic in China [6], while the prevalence of PSH at a Turkish headache clinic was reportedly 12.6% [7]. This difference may be due to referral bias and reporting bias since PSH is commonly associated with other headache disorders. Those who suffer with migraine are more likely to report PSH (42%) compared with controls (3%) [8].

Epidemiological studies have consistently shown that in the adult population, PSH occurs more commonly in females with a female to male ratio of 1.49–6.6:1 [4, 9]. The mean age of onset in the adult population has varied significantly in different studies and has ranged from age 28 in the largest epidemiological study of 627 patients (Vågå) in Norway [4, 10] to age 47 in a smaller study of 38 patients in Spain [5] and to age 53 in a veterans hospital of 80 patients in Taiwan [11].

Pediatric cases of PSH have also been reported [12, 13]. PSH occurs less frequently in children in comparison with adults [4]. A review of PSH in children found the mean age of onset is ages 4.5–9 and unlike the female predominance in adults, there appears to be no gender predominance in children [13].

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Diagnostic Criteria of Primary Stabbing Headache

The *ICHD-3* diagnostic criteria for Primary Stabbing Headache require all of A to E: data from [14•] *Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, 3rd edition. Cephalalgia, Vol.38(1)1–211*. Reprinted with permission from SAGE Publications, Ltd. [14•]

- A. Head pain occurring spontaneously as a single stab or series of stabs and fulfilling criteria B and C
- B. Each stab lasts for up to a few seconds
- C. Stabs recur with irregular frequency, from one to many per day
- D. No cranial autonomic symptoms
- E. Not better accounted for by another *ICHD-3* diagnosis

The *ICHD-3* diagnostic criteria for Probable PSH: data from [14•] *Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, 3rd edition. Cephalalgia, Vol.38(1)1–211*. Reprinted with permission from SAGE Publications, Ltd. [14•]

- A. Head pain occurring spontaneously as a single stab or series of stabs
- B. Two only of the following:
 1. each stab lasts for up to a few seconds
 2. stabs recur with irregular frequency, from one to many per day
 3. no cranial autonomic symptoms
- C. Not fulfilling *ICHD-3* criteria for any other headache disorder
- D. Not better accounted for by another *ICHD-3* diagnosis

Due to changes in the diagnostic criteria and our understanding of primary stabbing headache over the years, previously labeled PSH or its equivalents may be excluded or other variations of headache may be included in the current *ICHD-3* criteria for PSH. The enduring features of PSH within the *ICHD* have been the absence of any structural or secondary etiology for the head pain and/or the absence of another *ICHD* diagnosis.

The first edition of *ICHD*, published in 1988, referred to PSH as a idiopathic stabbing headache [15]. The pain was described as stabbing and lasted for a fraction of a second. Indomethacin responsiveness and the association with migraine were noted in the comment section of the criteria [15].

In the *ICHD-2*, published in 2004, idiopathic stabbing headache was changed to primary stabbing headache [16]. Additionally, *ICHD-2* classification did not allow for

accompanying symptoms Both *ICHD-I* and *ICHD-II* noted that the pain was confined to the first division of the trigeminal nerve (orbital, temple, and parietal).

The new 2018 *ICHD-3* diagnostic criteria are the most sensitive to capture only primary headaches causing stabbing pain. Importantly, *ICHD-3* does not comment on response to indomethacin and does not limit the location of the stabbing pain to the first division of the trigeminal nerve [14•]. In fact, the latest criteria indicate that PSH can occur in various locations and may be extratrigeminal in up to 70% of patients ([11], [15]). In addition, the criteria for “no accompanying symptoms” in *ICHD-II* were changed to “no cranial autonomic symptoms” to account for the presence of photophobia, phonophobia, nausea, and dizziness in some cases ([17], [18]).

Clinical Features of Primary Stabbing Headache

The latest evidence for clinical features of PSH are based on a few prospective observational studies, retrospective case control studies, case series, case reports, non-systematic review articles, and expert opinion. The *ICHD-3* criteria were clinically tested in the field ([19•], [18]).

The clinical features of primary stabbing headache include the type of pain, duration, frequency, location, and lack of associated symptoms. PSH is characterized as paroxysmal attacks of moderate to severe sharp/stabbing pain.

The stabbing pain occurs spontaneously and reportedly occurs in irregular patterns. There is no circadian or circannual pattern, and patients may be awoken from their sleep with stabbing pain [11]. Based on epidemiological studies and expert opinion, 80% of stabs are consistently less than or equal to 3 s [9, 11, 20]; however, rarely they can range from 10 to 120 s in duration [14•]. The mean duration of the pain was 1.42 s in a group of 280 migraine patients with distinct comorbid primary stabbing headaches [21].

Most often, patients experience single short stabs, but they may also present with a series of stabs [9, 11]. Attacks of stabbing pain are typically infrequent, occurring one to a few times a day, although rarely the pain can occur repetitively for days to 1 week [14•].

The stabbing pain can occur unilaterally or bilaterally [22]. The location of the stabs was traditionally described in anterior cranial regions, and predominantly in the ophthalmic division of the trigeminal nerve; however, stabs can occur in any region, including maxillary and mandibular divisions of the trigeminal nerve as well as retroauricular, parietal, and occipital regions [11, 23, 24]. Some patients may have pain in the cervical dermatomes of the occipital and nuchal region [25]. In those with migraine and PSH, the location of the stabs tends

to localize to the hemisphere more affected by headaches from migraine ([11], 2018).

A prospective observational trial of 65 patients found that different demographic characteristics are linked to variable clinical courses and patterns [26, 27••]. Three distinct patterns were identified: monophasic, intermittent, and chronic. These patterns were determined based on frequency of the stabs (daily vs intermittent/non-daily), clinical course (remitted or not) and total disease duration (< 3 or > 3 months duration) [26, 27••]. Stabbing pain in the monophasic pattern was more severe, more frequent, daily, side-locked, and single stabs that responded well to treatments such as indomethacin, steroid, gabapentin, or tricyclic antidepressants [26, 27••]. In contrast, the characteristics of the chronic daily stabbing pain pattern was longer in duration, not fixed in location, multiple, less responsive to treatment, and more common in the female population [26, 27••].

Several previous studies of primary stabbing headache would no longer fit the current *ICHD-3* criteria. Many studies prior to *ICHD-2* criteria would have included patients with described cranial autonomic features such as miosis, ptosis, conjunctival injection, lacrimation, and rhinorrhea. Cranial autonomic symptoms distinguish PSH from short-lasting unilateral neuralgiform headache attacks with conjunctival injection (SUNCT) and short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA). In addition, previous studies on primary/idiopathic stabbing headache may have excluded patients who would now be included in the *ICHD-3* criteria such as those with stabbing pain outside of the first division of the trigeminal nerve.

Triggers for PSH are not common, but a few case reports have described some potential triggers for paroxysmal stabbing pain particularly in the patients with co-morbid migraine. Head motion, rapid alterations in posture, physical exertion, and bright lights in patients during a migraine attack appear to trigger stabbing pain in the same location as the migraine [28]; however, these triggers are an unlikely culprit in true primary stabbing headache.

Pediatric PSH

A recent review of *ICHD-3* beta highlights that headache disorders in children have bio-psycho-social aspects that distinguish clinical presentation and management, although these specific aspects have not been elucidated for PSH [29]. A retrospective analysis of 83 youth showed that PSH can be associated with family history [17]. Pain from PSH in children can be located in many regions, including more occipital predominance in comparison with adults [30, 31]. Pediatric PSH is less often associated with other headache types, including migraine [13, 32], but may be associated with extracephalic symptoms such as abdominal pain [33].

Proposed Mechanism

The pathophysiology of PSH is unknown. Current theories include irritation of trigeminal and extratrigeminal nerves and/or intermittent impairment of central pain processing leading to hyper-excitability of neurons or spontaneous synchronous discharge of neurons. Ephaptic impulses are presumed to travel to the corresponding peripheral nerve distribution with the perception of stabbing pain [11, 34]. Increased incidence of PSH with migraine has suggested a potential vascular mechanism [35–38] as well as the proposed segmental disinhibition of central pain processing [28]. Other theories include dural sinus stenosis [39] and brainstem inflammation or focal demyelination [40, 41]; although these theories would suggest a secondary etiology for the stabbing headache.

Differential Diagnosis

The differential diagnosis for PSH includes short-lasting, stab-like primary and secondary headaches, and may provisionally include probable PSH. The diagnosis of all primary headaches must exclude secondary etiologies. Several articles report secondary aetiologies for the stabbing pain including infectious, autoimmune, vascular, and neoplastic aetiologies [20].

Small studies and case reports have identified various secondary vascular abnormalities in patients with stabbing head pain: ischemic stroke, non-traumatic subarachnoid hemorrhage, unruptured intracerebral aneurysm, thalamic hemorrhage, and dural sinus stenosis [20, 39, 42]. In a prospective study of more than 2000 patients with acute ischemic stroke or TIA, 20% of the patients who presented with headache at the onset of their ischemia described a stabbing headache [43].

Inflammatory, autoimmune, and infectious etiologies such as herpetic meningoencephalitis, herpes zoster, giant cell arteritis, Behcet's, Sjogren's disease, systemic lupus erythematosus, and multiple sclerosis have been associated with stabbing headache [20, 40, 41, 44–46].

Neoplastic etiologies such as pituitary adenomas and meningiomas have also been associated with stabbing headaches [47, 48].

After the secondary etiologies have been considered and ruled out, the differential diagnosis of stabbing headaches is limited to primary headaches disorders. The duration, frequency, location, presence or absence of cranial autonomic features, and triggers are used to determine the primary headache disorder.

Trigeminal autonomic cephalalgias such as SUNCT, SUNA, paroxysmal hemicrania, and hemicrania continua can be considered in the differential diagnosis, but these conditions can be distinguished from PSH given the accompanying cranial autonomic symptoms [49]. Although PSH, like paroxysmal hemicrania and hemicrania continua, is

responsive to indomethacin, these latter headache disorders have longer duration of pain and presence of cranial autonomic features, which differentiates them from PSH.

Other primary headaches that can mimic PSH include headaches associated with triggers such as primary cough headache, primary headache associated with sexual activity, primary exertional headache, and cold stimulus headache.

Trigeminal neuralgias consist of paroxysmal pain and are characterized by brief electric shock attacks of facial pain. The pain is triggered by mechanical stimuli such as brushing teeth, eating, and speaking, and the distribution of the pain is mostly in maxillary and mandibular trigeminal nerve branches [16].

Investigations

Recurrent stabbing headaches could be due to secondary aetiologies; therefore, neuroimaging is reasonable. CT or MRI of the brain could be performed to investigate for potential secondary causes including meningiomas and stroke [42, 45, 47]. Blood work including ESR is also reasonable in patients over the age of 50 who present with stabbing pain particularly if they have additional features of giant cell arteritis [45].

Treatment

The main therapy for PSH is indomethacin [50]. Responsiveness to indomethacin is not specific to PSH. Paroxysmal hemicrania, hemicrania continua, primary cough headache, and hypnic headaches also respond to indomethacin [51, 52].

Response to indomethacin is now known to vary, and some experts estimate up to 60% of patients with PSH may respond to indomethacin treatment [50]. Indomethacin is a potent non-steroidal anti-inflammatory drug (NSAID) that reversibly inhibits cyclooxygenase (COX)-1 and -2, inhibits polymorphonuclear leukocyte motility, decreases mucopolysaccharide biosynthesis, and may have a vasoconstrictor effect [53]. Indomethacin can inhibit nitric oxide release and decrease cerebral blood flow as well as lower cerebral spinal fluid pressure [54]. The mechanism of indomethacin for PSH may be anti-inflammatory. Response to indomethacin is usually quick. Dose ranges from 75 to 250 mg/day in divided doses. Dose-related side effects include dyspepsia, gastrointestinal bleed, and renal toxicity [52]; therefore, long-term indomethacin use is often limited. Depending on the clinical burden, most indomethacin treatment is given prophylactically. Indomethacin is not FDA approved for children < 15 years old, although Myers et al. recommended a trial of indomethacin in preadolescents with severe paroxysmal pain without autonomic symptoms in whom secondary aetiologies have been ruled out [30].

For patients with inadequate response, contraindications or intolerance to indomethacin, alternative treatment options suggested from small observational studies include other NSAIDs, such as selective COX-2 inhibitors, etoricoxib [55] and celecoxib [56], melatonin [57–61], onabotulinumtoxin A (BoNTA) [62], gabapentin [63], topiramate [39], acetazolamide [64], and nifedipine [65]. Similar to indomethacin, the mechanism of benefit from acetazolamide and topiramate may be from their effect to lower of intracranial pressure, although this remains to be studied in PSH [66, 67].

Melatonin was recently studied in a systematic review for use in primary headache disorders, but not specifically PSH, which showed the evidence is lacking for melatonin as first-line therapy but it may be a treatment option in difficult headache cases or pediatric primary headaches in the short term [58, 68, 69]. Melatonin is a pineal hormone that is structurally similar to indomethacin but with fewer adverse effects [70]. The most common side effects are dizziness, nausea, drowsiness, and headache [70]. The mechanism of melatonin in PSH is not known, although it may influence the hypothalamus, improve sleep, and have some anti-inflammatory effects [58].

In a prospective observational trial of 24 patients with PSH, BoNTA was injected into the primary stabbing zone(s) [62]. Patients received 5 units of BoNTA into each area where they had the stabs. The mean dose was 11.81 ± 7.17 units. Three subjects had complete remission, 19 subjects had decrease in stabbing pain that was sustained for 63 days, and 2 patients had no improvement [62]. No adverse effects were reported and therefore BoNTA may be a logical treatment option for PSH.

In children, there is little data on therapeutic options and pharmacological treatment is as conservative as possible. Indomethacin is not often used in children less than age 15, but there have been some case reports of effective indomethacin treatment for two patients ages 2.5 and 5 [30]. Other successful treatment options for suspected PSH in children have been described in small studies, including amitriptyline [31] and paracetamol, although in this study, headaches lasted greater than 3 min and, therefore, were not likely PSH [12]. Melatonin has also been reported as a possible option in the PSH population with co-morbid migraine [68, 69].

Prognosis

In general, PSH is considered a benign condition with very short duration of pain and infrequent attacks that may remit with time.

Conclusion

Modification of the diagnostic criteria of primary stabbing headache to include stabbing pain outside of the first division

of the trigeminal nerve appears to have resulted in increased diagnostic sensitivity. Epidemiological data on PSH will need to update to reflect this change. Although indomethacin continues to be the main therapy for PSH, other treatment options including selective COX-2 inhibitors, melatonin, and onabotulinumtoxin A may be considered.

Compliance with Ethical Standards

Conflict of Interest Esma Dilli reports honoraria from Allergen and royalties from Lippincott. Danielle Murray reports no potential conflicts of interest.

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

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- Of major importance

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