Headache
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ISSN 0017-8748
doi: 10.1111/head.12568
Published by Wiley Periodicals, Inc.

Brief Communications

Recurrent Spontaneous Paresthesia in the Upper Limb Could Be Due to Migraine: A Case Series

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Transient neurologic dysfunction is a characteristic feature of migraine. About 20% of migraineurs may experience various symptoms in the absence of any headache at one time or another. Visual auras are the most common auras of migraine, and migraine is considered as the most common cause of transient vision loss in young patients. Sensory auras are the second most common migrainous auras. However, the literature is silent for isolated sensory aura as a migraine equivalent. Herein we report 14 patients with recurrent episodic paresthesia in the limbs and other body parts. All patients fulfilled the diagnostic criteria of "typical aura without headache" of ICHD-3β. All patients were subjected to various investigations to rule out secondary causes. Ten patients received antimigraine drugs and all showed a positive response to therapy.

Recurrent spontaneous paresthesia is quite common in the general population and many patients remain undiagnosed. We speculate that a subset of patients might be related to migrainous sensory auras.

Key words: paresthesia, migraine, aura

(*Headache* 2015;55:1143-1147)

Paresthesia is an abnormal skin sensation with no apparent physical cause. It could be present anywhere in the body. However, it mostly involves upper and lower extremities.[1] It could start spontaneously or may be induced by prolonged compression of the nerves. The most common cause of transient paresthesia is prolonged pressure on nerves due to abnormal posture of the limbs or body.[2] Sensory aura is the second most common aura in migraine. International Classification of Headache Disorders (ICHD) recognizes typical aura without headache as a separate group (ICHD-3β code: 1.2.1.2).[3] Visual aura is the most common aura of migraine and it is frequently described in isolation (ie, visual auras without migraine headaches).[4,5] Migraine is considered as a most common cause of recurrent transient visual disturbances.[6] However, the literature is silent about the presence of isolated sensory aura. Herein we describe 14 patients fulfilling the criteria of typical aura (sensory) without headache.

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Accepted for publication March 1, 2015.

Conflict of Interest: None.

Financial Support: None.

Previous Presentation: This manuscript was not presented at any meeting or published, or is being considered for publication elsewhere.

Consent: Written informed consent was taken from the patient to publish the report.

CASE SERIES

Case 1 is the index case. This prompted us to find similar cases. We observed 14 cases over 3 years (January 2012 to December 2014). All cases fulfilled the criteria of typical aura (sensory) without headache (ICHD-3β code: 1.2.1.2).[3] Clinical features and other characteristics are summarized in the Table. Two cases are described in details. All patients gave consent for the publication. The study does not require approval by the local ethics committee as per the local regulations for the case reports.

Case 1: A 26-year-old woman had episodic spontaneous paresthesia in the upper limb and the face for about 4 years. Most attacks were on the right side. The paresthesia was perceived as unpleasant tingling and burning sensation. The patient noted marching of the sensory symptoms. Typically, paresthesia used to start in one of the hands and afterward extending to involve the whole limb, and at times even in the face. On asking, the patient confirmed the presence of paresthesia and numbness of the tongue, too. The marching of the symptoms was slow, and it took about 5–20 minutes to reach the arm/face from the hand. The overall duration of symptoms persisted for about 20–90 minutes. Initially, she got just 2–3 attacks in a year. But over the last 6–8 months it had exceeded 10 attacks per month. Most attacks started spontaneously. She never had nocturnal attacks. She did not recognize any precipitating or aggravating factors for paresthesia.

The patient had a history of migraine since her early childhood. The patient could not remember visual and any other auras with headache attacks. The frequency of headache attacks was less than one attack per month and she never received any preventive therapy for headaches. Initially, she denied the presence of headache and any migrainous symptoms (nausea, vomiting, photophobia, phonophobia, etc) during the attacks of paresthesia. However, in the follow up, when we asked to maintain a headache diary, she confirmed the presence of mild headache, nausea, and phonophobia in a few attacks of paresthesia.

Physical examinations and laboratory tests were normal. Intracranial and cervical structural

Table.—Summary of Patients With Migrainous Sensory Auras

	Parameter (n-14)
Age (years) (mean ± SD) range	36.1 ± 13.0 (19-58)
M:F	1:1.8 (5:9)
Duration of illness (paresthesia) (months) (mean ±SD) Range	15.3 ± 12.4(3-48)
Characteristics of paresthesia	
Sites	
Upper limbs (UL)	14(100%)
Only UL	8 (57%)
Side fixed UL	1 (7%)
Face	5 (36%)
Lower limbs	5 (36%)
Hemi-sensory	4 (29%)
Whole body	4 (29%)
Tongue	4 (29%)
Marching of the symptoms	14 (100%)
Onset/progression of symptoms (duration of marching)	
<5 minutes	4 (29%)
>5 minutes	14 (100%)
Duration of paresthesia episode	
<5 minutes	5 (36%)
5–60 minutes	14 (100%)
>60 minutes	3 (21%)
Nocturnal attacks	0 (0%)
History of migraine	14 (100%)
Migrainous features during paresthesia	7 (50%)
Headache	6 (43%)
Nausea ± vomiting	3 (21%)
Photophobia/phonophobia	1 (7%)
History of visual disturbances	5 (36%)
Seeing flashes/flickering of light	5 (26%)
Blurry or loss of vision	3 (21%)
History of vertigo/dizziness	5 (36%)
Precipitating factors	
Fasting	3 (21%)
Lack of sleep	2 (14%)
Stress	2 (14%)
Physical signs	
Allodynia	4 (29%)
Physical tests for CTS	Negative in all
Neck examinations	Normal in all
Investigations	
MRI cervical/brain	Normal in all
Nerve conduction study	Normal in all
EEG (8 patients)	Normal in all
Biochemical parameters	Normal in all
Previous diagnosis (by previous physicians)	
Carpal tunnel syndrome	11 (76%)
Cervical radiculopathy/cervical spondylosis	7 (50%)
Plexopathy/other neuropathy	6 (43%)
Thoracic outlet syndrome	3 (21%)
Epilepsy	2 (14%)
Psychogenic	4 (29%)
Treatment response	
Antimigrainous therapy (10 patients)	Positive response in all
Reassurance (for 4 patients)	–

CTS = carpal tunnel syndrome.

pathologies were ruled out by a magnetic resonance imaging (MRI). Electroencephalogram (EEG) was normal. Nerve conduction study was normal.

The clinical impression was periodic syndrome linked to migraine. The patient fulfilled the ICHD-3β criteria of "typical aura without headache." We decided to give amitriptyline (25 mg daily) and sodium valproate (200 mg twice daily). In the next 9 months, she had only 6 attacks of paresthesia. Hence, drugs were gradually withdrawn (over 8 weeks). In the next 6 months, she had one such attack per month. We reassured the patient.

Case 2: A 37-year-old woman had suffered from migraine for more than 15 years. The patient visited the neurology outpatient clinic for episodic paresthesia in the limbs occurring for 2–3 years. Paresthesia was mostly described as pins and needle sensations. At times, she felt a burning sensation and numbness, too.

The paresthesia typically used to start in one of the hands and then spreading to involve the whole upper limb. Occasionally, it involved face, tongue, and lower limb on the same side (hemi-sensory pattern). Less frequently, it involved the entire body.

The duration of the paresthesia varied from 5 to 120 minutes. She did not recognize any precipitating factor for paresthesia. The frequency of the paresthesia was 1–2 attacks per month during the first year of the onset. Afterward, the frequency gradually increased to about 2–3 attacks per week.

She noted an increase in frequency of headache attacks over the past few months. It was 2–4 attacks per month. The patient did not find any correlation between paresthesia and migraine headaches. However, prospectively when we asked to maintain a headache diary, she confirmed the presence of mild-to-moderate headache, nausea, and phonophobia in a few attacks of paresthesia. The patient admitted having visual auras in the form of seeing flashes of light and blurring of vision. However, she did not recognize any relation of visual symptoms to headaches and paresthesia.

Physical examinations and biochemical tests were normal. MRI of the brain and cervical spine revealed no abnormality. EEG and nerve conduction studies were normal.

The patient received propranolol (40 mg daily) and topiramate (100 mg/day). Both migraine headache and paresthesia reduced in the frequency. In the first month of the treatment, she had 4 attacks of paresthesia. Thereafter, she had only 5 attacks in the next 8 months. The gradual withdrawal of both drugs (after 9 months of therapy) was successful. She had only 4 attacks in the next 6 months of follow up.

DISCUSSION

Herein we describe 14 patients with spontaneous recurrent paresthesia in the limbs, fulfilling the ICHD-3β criteria for typical aura without headache.[3] All patients had marching of sensory symptoms. Fifty percent of patients noted at least one migrainous symptom with paresthesias. All other possible causes were ruled out by detailed clinical examinations, investigations, and long-term follow up. Response to antimigraine drugs also favors a diagnosis of migrainous sensory auras.

About 20% of migraineurs experience various transient symptoms in the absence of any headache at one time or another.[7,8] These isolated symptoms are termed "acephalgic migraine or migraine equivalent or migraine aura without headache." It is said that 1–6% of population may have isolate aura without headache.[9,10] About 16% of persons of >55 years may have an isolated aura without headache.[11] So it could be speculated that a large number of patients might have paroxysmal neurological symptoms (with or without headache) as a migrainous aura.

Visual aura is the most common aura of migraine. Transient or episodic visual disturbance in patients younger than 45 years is most likely due to migraine.[6] Sensory aura is the second most common type of aura.[4,5] Therefore, just like transient vision loss, it could be hypothesized that transient or episodic sensory symptoms could be due to migraine.

Miller Fisher published 2 articles on late-life migrainous accompaniments (205 patients).[11,12] Visual symptoms were the most common features. However, paresthesia was noted in the large number of patients in that observation. Many of them had paresthesias in the absence of visual symptoms.[11,12]

Various observations suggest that sensory migrainous aura could be in 30–54% of patients with

migraine with auras.[4] So it could be speculated that a large number of patients might have paroxysmal isolated sensory symptoms (like an isolated visual aura). However, to the best of the literature search, we did not find any case report/series mentioning isolated sensory auras as a presenting feature. Probably these cases remain undiagnosed. The previous diagnoses in this case series were carpal tunnel syndrome, cervical spondylosis/cervical radiculopathy, brachial plexopathy, psychogenic, seizure, etc.

A diagnosis of transient neurological dysfunction as migraine is usually facilitated by the presence of current or past history of migraine. A presence of migrainous headaches or migrainous features during attacks also favors the diagnosis of migraine.[7,8] However, a possibility of recall bias always exists. In our cohort, only two patients admitted having any type of headache preceding or accompanying or following the paresthesia in their first visit. Four other patients reported headache at the time of paresthesia in the follow-up visits.

Upper limb is the most common site (>90%) of the sensory aura of migraine. Face is the next most common site. Rarely patients may have sensory symptoms in the legs (24%) and trunks (18%).[4,5] All our patients felt paresthesia in the upper limb. About 57% of patients also reported paresthesia in the other part of the body. Four patients (29%) reported episodic paresthesia in the tongue. Episodic paresthesia in the tongue is mostly due to migraine.[11,12] Therefore, our observation is in accordance with the prevalence of sensory auras with migraine in the general population. Moreover, a few observations have also demonstrated that recurrent pain in the limbs could be due to migraine.[7] Recurrent paresthesia is quite common in the general population and many remain undiagnosed.[13] We speculate that a subset of patients might be related to migrainous sensory auras.

LIMITATION

All cases fulfilled the diagnostic criteria of typical aura without headaches. Still, we cannot rule out a possibility of other secondary cause as full investigations were not done on each patient. It is a case series. Therefore, a possibility of unrecognized selection bias and recall bias exists.

In conclusion, our observations indicate that migraine could be an important cause of recurrent spontaneous paresthesia in the limbs (especially upper limb). As our observation is retrospective, it must be substituted by future prospective studies.

STATEMENT OF AUTHORSHIP

Category 1

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