Postictal Headache with a Single Neurocysticercal Lesion: A Comparative Observational Study

Sir,

Seizure and headache are both paroxysmal hyperexcitability disorders and probably have shared underlying pathogenic mechanisms.[1] They are also the two most common presentations of neurocysticercosis (NCC).[2,3] Postictal headaches (PIH) are defined by the International Headache Society ICHD3 classification under nonvascular intracranial disorders (7.6.2) as any headache that has developed within 3 hours of seizure onset and resolves spontaneously after seizure termination (within 72 hours), and is not better accounted for by any other diagnosis.[4] Although, the impact of PIH on epilepsy is likely to be significant, it has not been adequately studied and questions regarding its occurrence are missing from the quality of life questionnaires. Studies have found that less than 10% seizure patients were asked about and prescribed anti-headache medications by their treating physicians.[5,6]

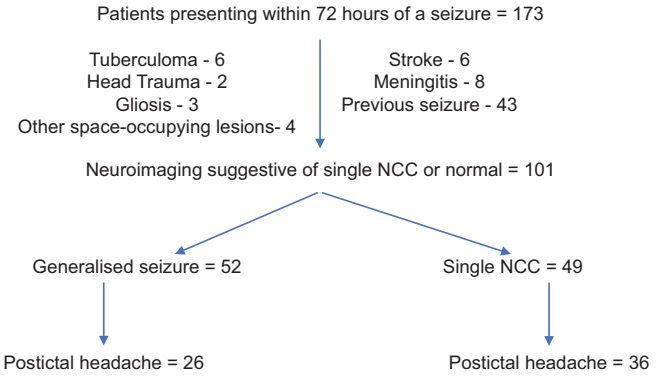
Studies have shown patients with NCC to have more frequent headaches when compared to other structural brain lesions,[2] but no study has looked at the occurrence of postictal headache in patients with NCC. We conducted an observational study and screened all adult patients presenting to our hospital between January 2017 and December 2017, who presented to us within 72 hours of the first seizure. They were evaluated for their seizure type and its cause by neuroimaging (Computed Tomography [CT]/ Magnetic Resonance Imaging [MRI] brain) and those found to have a single NCC or normal scans were included (flowchart). Written informed consent was taken from the patient and institutional ethics clearance was taken for the study. These patients were separated into two groups:

Table 1: Patient characteristics

Demographics	Patients with Generalised Seizures (*n*=52)	Patients with Neurocysticercosis (*n*=49)	*P*
Mean age (in years)	28±11	29±10	
Sex (Male)	24 (46.2%)	27 (55.1%)	
Seizure onset	Generalised tonic clonic seizures-52	Focal onset-49	
		Focal only- 31	
		Secondary generalisation-18	
Neuroimaging	Normal	NCC lesion- 42	
		Calcific lesion with oedema- 7	
Perilesional oedema	-	Present-40	
		Absent-9	
Electroencephalogram	Normal- 38	Normal-20	
	Generalised spike and wave discharges- 14	Lateralised discharges-5	
		Not done-24	
Past headache	Total-15 (28.8%)	Total-12 (24.5%)	0.829
	Migraine- 9	Migraine- 8	
	Tension-type- 6	Tension-type- 4	
Postictal headache	26 (50%)	36 (73.5%)	0.261

Table 2: Characteristics of postictal headache

Postictal Headache	Patients with Generalised Seizures (*n*=26)	Patients with Neurocysticercosis (*n*=36)	*P*
Duration			0.3913
<6 h	10	10	
6 h-24 h	12	22	
24 h-72 h	4	4	
Headache onset			0.3735
<1 h	24	35	
1-2 h	2	1	
2-3 h	0	0	
Migrainous headache	16	15	0.2635
Mild	8	4	
Moderate	7	10	
Severe	1	1	
Tension-type headache	10	21	0.1315
Mild	6	14	
Moderate	4	7	
Severe	0	0	
Laterality			0.0016
Unilateral	2	16	
Ipsilateral		12	
Contralateral		4	
Bilateral	24	20	
Preictal headache	0	13	0.0029



generalised seizure (GS) and seizure associated with NCC and evaluated for the presence of PIH. "Mild" headache was defined as minimal unpleasantness without procrastination and need for analgesic. "Moderate" grade was defined as discomfort with procrastination and/or the need for analgesic.[7] "Severe" grade was the one that significantly impaired the individual's activities of daily living. Migraine and tension-type headache (TTH) were defined according to the ICHD3 classification.[4]

Sixty-two patients had a history of PIH. Their clinical characteristics have been detailed in Tables 1 and 2. Both

groups had similar baseline characteristics except that there were more males in the NCC group. All the patients in the GS group had a generalised tonic clonic seizure and normal neuroimaging with electroencephalogram (EEG) either being normal or showing generalised spike and wave discharges. In the NCC group, a viable parenchymal lesion was seen in 85.7% patients whereas a calcific lesion with perilesional oedema was seen in the remainder. Past history of interictal headache was similarly distributed in both the groups (25-30%). A majority of our patients developed PIH within 1 hour of seizure. Studies report that PIH usually starts within 5 minutes after seizure onset and only rarely delayed beyond 1 hour.[8]

We found that PIH was more common in the NCC group (73.4%) compared to the GS group (50%). This is in contrast to most studies,[4] which report PIH to be more common with GSs group. Our study looked at NCC patients presenting with first ever seizure and still found headache prevalence to be higher (73.4%) than that described in most studies (12-52%), which have enrolled patients with epilepsy and found that younger age at onset, long duration of epilepsy and drug refractoriness are risk factors for developing PIH.[1]

Patients with past headache had a similar PIH, reiterating a probable shared pathophysiologic mechanism between the two processes. However, 13 headache naïve patients had a moderate intensity preictal headache up to 4- 48 hours before seizure onset which continued postictally as the same subtype. All these patients belonged to the NCC subgroup and could have a diagnostic value for patients likely having an imaging substrate for the seizures (P - 0.0029). Most cases of migrainous PIH had moderate-severe headaches (50% in the GS group and 73.3% in the NCC group) while even amongst the Tension-type Headache (TTH) subgroup, a significant proportion fell in this category (30-40%). Most previous studies on PIH have found similar results.[1]

The PIH was unilateral in 16 (ipsilateral to the lesion-12; contralateral-4) and bilateral in 20 cases associated with NCC. In contrast, only two patients had unilateral headache in the generalised seizure group (P - 0.0016). All patients in the NCC group with PIH had perilesional oedema. Four patients had perilesional oedema but no PIH and nine patients had no perilesional oedema. Thus, the perilesional oedema associated with NCC might be responsible for the increased PIH rates found in our study when compared to other structural brain lesions.

PIH is thought to arise due to the changes in the regional blood flow and cerebral diffusion[9] following a seizure. Similar changes have been described following the activation of the trigeminovascular system in migraine patients. A common biological link between the two has also been found through mutations in the Na/K ATPase and calcium channels leading to both seizures and migraines.

Our study has a few limitations. Headache severity was qualitatively assessed which could introduce bias as the same headache severity can elicit different responses amongst different individuals and we would have been better served by using a quantitative tool like Visual Analogue Scale (VAS) for the same. We could not comment on whether the lobar location and laterality of the NCC impacted PIH occurrence due to the small sample size.

Through our study, we want to highlight the occurrence of the commonly ignored, yet disabling postictal headache, as a common manifestation following seizures with special emphasis on its occurrence in patients with NCC. Not only might it have a structural localising value but may also have a diagnostic value in distinguishing seizures from non-epileptic events.[10] Only by actively inquiring, and thereby, treating the same can we truly alleviate the patients' symptomatic burden and help them lead a better quality of life.

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Conflicts of interest

There are no conflicts of interest.

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