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A case–control study of wicket spikes using video-EEG monitoring

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ABSTRACT

Purpose: To investigate clinical characteristics associated with wicket spikes in patients undergoing long-term video-EEG monitoring.
Methods: A case–control study was performed in 479 patients undergoing video-EEG monitoring, with 3 age- (±3 years) and gender-matched controls per patient with wicket spikes. Logistic regression was utilized to investigate the association between wicket spikes and other factors, including conditions that have been previously associated with wicket spikes.
Results: Wicket spikes were recorded in 48 patients. There was a significantly higher prevalence of dizziness/vertigo ($p = 0.002$), headaches ($p = 0.005$), migraine ($p = 0.015$), and seizures ($p = 0.016$) in patients with wickets. The majority of patients with wicket spikes did not exhibit epileptiform activity on EEG; however, patients with history of seizures were more likely to have wickets ($p = 0.017$). There was no significant difference in the prevalence of psychogenic non-epileptic seizures between the groups. Wickets were more common on the left, during sleep, and more likely to be first recorded on day 1–2 of monitoring.
Conclusions: Patients with wicket spikes are more likely to have dizziness/vertigo, headaches, migraine, and seizures. Patients with history of seizures are more likely to have wickets. The prevalence of psychogenic non-epileptic seizures is not significantly higher in patients with wickets.

1. Introduction

Wicket spikes are monophasic arciform discharges that appear as single events or as brief runs at 6–11 Hz (wicket rhythms) recorded over the temporal head regions. They are further characterized as unilateral or independently bilateral surface negative signals of 60–210 μV amplitude. These discharges are typically recorded in adults, occur more on the left, and occur more often during light sleep; however, they have been reported to persist during rapid eye movement (REM) sleep. Wicket spikes can be mistaken for anterior or mid-temporal spikes given their shared electrographic features.[1–4] The incidence of wicket spikes has been reported at 0.8–3.25% based on regular EEG records.[1,5,6]

Wicket spikes were originally described by Reiher and Lebel as benign temporal discharges resembling mu rhythm or epileptic spikes recorded during both wakefulness and sleep in 39 out of 4458 EEGs. The authors concluded that wicket spikes should not be considered epileptiform or abnormal, given the absence of clinical association with epilepsy or any particular symptom complex. Additionally, symptoms such as syncope, headache, or vertigo were reported in 80% of the patients with wicket spikes.[1]

However, subsequent studies associated wicket spikes with posterior circulation cerebrovascular disease (CVD), head injury, and headaches.[2,4,7,8] while a recent report did not find such correlation.[5]

Previous studies have also ventured to delineate characteristics of patients with wicket spikes versus those with epilepsy. Data indicate that wickets are more commonly seen in middle-age populations than patients in their late teens and early adult years. Several authors have noted that the most frequent clinical diagnoses seen in patients with wickets were headache, migraine, CVD, seizures, near syncope, psychogenic non-epileptic seizures, anxiety, hyperventilation, or post-concussion syndrome. It is worth noting that while the association with CVD has been found to be age-related, headache, seizures, and psychiatric problems were significantly associated with wicket spikes independent of age.[5,9]

Wicket spikes often present a diagnostic challenge to non-epileptologists. There is evidence that misinterpretation of benign EEG discharges, such as wicket spikes, can result in misdiagnosis of epilepsy.[10,11] Krauss et al. found that as many as 54% of wicket rhythms were incorrectly interpreted as epileptiform activity.[9] In order to differentiate specific transient EEG findings from ictal activity, and therefore to avoid such diagnostic errors, these

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authors suggested obtaining additional EEG recordings. Crespel et al., reported a case of primary generalized epilepsy (PGE) misdiagnosed as partial epilepsy, for which the patient was referred for presurgical evaluation. Upon video-EEG monitoring, the seizures were reclassified as PGE, and focal discharges were identified as wicket rhythms.[12]

The available literature regarding wicket spikes and rhythms is limited to data obtained through regular EEGs. Given the limitations of routine EEG such as lower chances of capturing ictal or interictal activity, all stages of sleep, and inability to taper AEDs, we chose to examine patients who underwent inpatient long-term video-EEG monitoring. In order to better characterize the association between wicket spikes and ictal or interictal epileptiform discharges and other clinical characteristics, we compared patients with wicket spikes versus age- and gender-matched controls.

2. Methods

2.1. Patient population and study design

We reviewed long term video-EEG recordings of patients admitted to the epilepsy monitoring unit, either for diagnostic purposes or for presurgical evaluation, from 2005 to 2008. The study was approved by the institutional review board. The inpatient monitorings were done using either 10–20 standard electrode placement system or a partial 10–10 electrode system for extra coverage of basal temporal head regions. A total of 479 patients were screened. All video-EEG recordings were read by one epileptologist (GKM) to avoid inter-interpreter variability. For the purposes of this study, the term "wickets" refers to both single wicket spikes and wicket rhythms (Figs. 1–3). Wicket spikes were found in 48 patients (79.2% female, mean age 46.6 ± 16.7), monitored for 1–6 days (mean 3.35 ± 1.63).

Clinical data were collected retrospectively through chart review and included the site of wicket spikes (right, left, bilateral), stage of sleep versus wakefulness, day of monitoring when the wickets were first recorded, presence or absence of epileptiform discharges, final EEG evaluation, and clinical diagnoses including conditions that have been associated with wicket spikes.

2.2. Statistical methods

In order to identify the clinical characteristics associated with wickets, a case–control design was implemented. Three controls per wicket case were individually matched for age (±3 years) and gender. The control group included patients who underwent video-EEG monitoring during the same time period as the wicket group, but did not have wicket spikes on EEG. Wicket and control groups were compared in terms of clinical characteristics by using a Student's unpaired *t*-test assuming unequal variance for continuous variables, and a chi-squared test for categorical variables. Logistic regression was utilized to investigate the association between wickets and age, gender, headache, migraine, dizziness/vertigo, autism, brain metastases, CVD, coronary artery disease, head trauma, hypertension, diabetes mellitus, seizures, depression, current use of antiepileptic drugs (AEDs), whether AEDs were reduced or stopped during monitoring, and whether activation procedures (hyperventilation and photic stimulation) were utilized during monitoring. Logistic regression analyses were adjusted for age and gender to control for potential residual confounding. All statistical analyses were performed using Stata 9 (StataCorp: Stata Statistical Software: Release 9.0, Stata Corporation, College Station, TX, 2005).

3. Results

3.1. Patient characteristics

Forty-eight patients were found to have wicket spikes and 144 controls were identified via individually matching for age (±3 years) and gender. Patient demographics and clinical characteristics for both wicket and control groups are presented in Table 1.

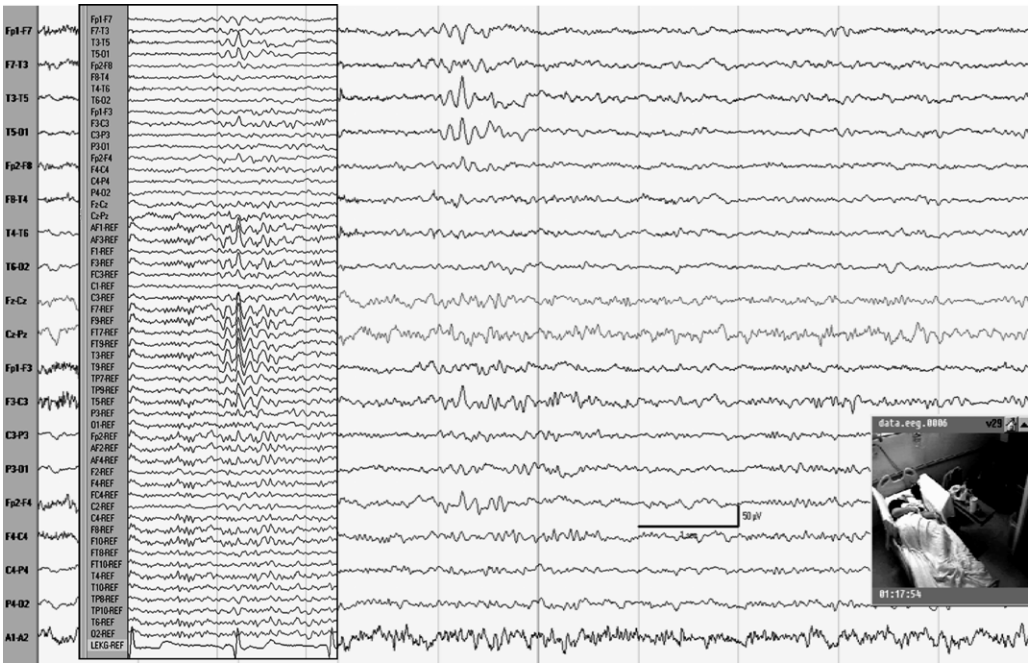

Fig. 1. A left temporal wicket spike and associated theta slow bursts during light sleep; background EEG remains symmetric. The inset shows the same wicket spike recorded with partial 10–10 electrode system revealing the extent of the field involving anterior-mid temporal (F7-T9) and frontal (AF3-AF1) areas; longitudinal montage.

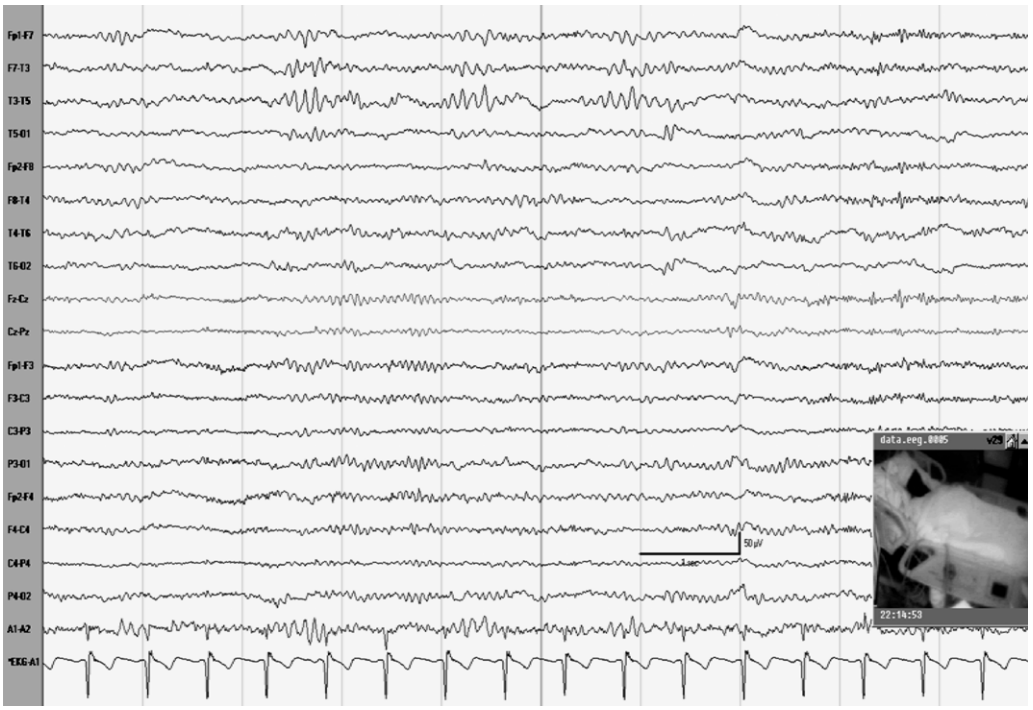

Fig. 2. Brief left temporal wicket rhythm during light sleep; background EEG remains symmetric; longitudinal montage.

3.2. Clinical conditions associated with wicket spikes versus controls

Comparison of the wicket and control groups indicate statistically significant differences ($p < 0.05$) in frequency of headaches, migraines, dizziness/vertigo, seizures, tapering of AEDs, and use of activation procedures during monitoring (Table 1). All of these correlations persisted in regression analysis. Additionally, adjustment for both age and gender did not alter the significance of these relationships (Table 2).

Specifically, patients with headache had 3.19 times increased odds of having wickets compared to patients without headache (95% CI 1.39, 7.34; $p = 0.006$), and this relationship persisted after adjustment for both age and gender (3.34× increased odds, 95% CI 1.43, 7.83; $p = 0.005$). Prevalence of migraine was higher in the

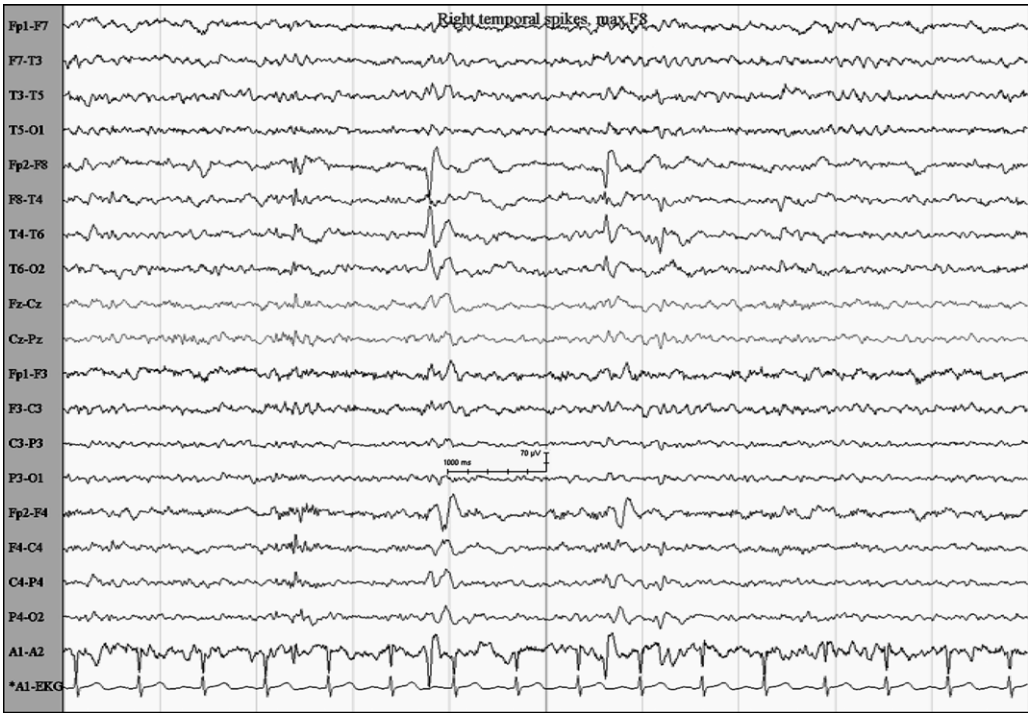

Fig. 3. Right anterior-mid temporal single spike discharges with a following slow wave during wakefulness in a patient with hippocampal sclerosis and refractory epilepsy. There is significant background theta/delta slowing in the right anterior head regions; longitudinal montage; video image not shown.

Table 1
Wicket group ($n = 48$) vs. control group ($n = 144$); descriptive statistics.

	Wicket % (n)	Control % (n)	p-Value*
Age (years, mean ± SD)	46.6 ± 16.7	46.6 ± 16.4	0.990
Female gender	79.2 (38)	79.2 (114)	1.000
Headache	27.1 (13)	10.4 (15)	0.005
Migraine	14.6 (7)	4.3 (6)	0.015
Dizziness/vertigo	16.7 (8)	0.69 (1)	<0.001
Autism	0 (0)	0 (0)	1.000
Brain tumor	0 (0)	2.1 (3)	0.313
CVD	10.4 (5)	14.6 (21)	0.465
CAD	6.3 (3)	4.17 (6)	0.554
Head trauma	10.4 (5)	7.6 (11)	0.546
Hypertension	25.0 (12)	16.0 (23)	0.161
Diabetes mellitus	8.3 (4)	10.4 (15)	0.675
Seizures	66.7 (32)	46.5 (67)	0.016
Depression	10.4 (5)	9.7 (14)	0.889
Currently on AED	66.7 (32)	58.7 (84)	0.331
AEDs tapered	65.6 (21)	40.7 (33)	0.017
Activation	29.8 (14)	16.2 (21)	0.044

* p-Value calculated from unpaired t-test assuming unequal variance for continuous variables, chi-squared test for categorical variables. CAD, coronary artery disease; CVD, cerebrovascular disease; AED taper, antiepileptic drugs tapered during monitoring; Activation, hyperventilation/photic stimulation.

wicket group than in the control group ($p = 0.015$). Patients with a reported history of seizures had 2.30 times increased odds of having wickets when compared to patients without seizures (95% CI 1.16, 4.55; $p = 0.017$), and this relationship persisted after adjustment for both age and gender (2.35× increased odds, 95% CI 1.17, 4.69; $p = 0.016$). In patients with reported history of dizziness/vertigo, there was a 28.60 times increased odds of having wickets (95% CI 3.5, 235.4; $p = 0.002$), and this relationship persisted upon adjustment for age and gender (28.6× increased odds; 95% CI 3.5, 235.7; $p = 0.002$).

Diagnoses based on the results of video-EEG monitoring were grouped into the following categories: normal EEG with no clinical events, normal EEG with psychogenic non-epileptic seizures (PNES), generalized epileptiform discharges, focal epileptiform discharges, diffuse slow activity, and focal slow activity (Table 3). Overall, 19.8% of patients with wickets had evidence of partial or generalized epileptiform discharges (vs. 31.5% in controls; $p = 0.007$ for focal epileptiform discharges). Patients with wicket spikes were more likely to have a normal EEG compared to patients without wickets (56.3% vs. 32.6%, $p = 0.004$), and were less likely to have focal epileptiform discharges than controls (27.3% vs. 8.3%, $p = 0.007$).

Additionally, patients with wickets had 2.78 times increased odds of AED cessation or reduction during monitoring (95% CI 1.18, 6.52, $p = 0.019$), and this relationship persisted after adjustment for age and gender (2.76× increased odds, 95% CI 1.17, 6.50, $p = 0.020$). The use of activation procedures was also associated with wickets, as patients with wickets had 2.20 times increased odds of having activation procedures when compared to controls (95% CI 1.01, 4.81; $p = 0.047$), and this relationship persisted after adjustment for age and gender (2.24× increased odds; 95% CI 1.02, 4.89; $p = 0.044$). The role of sleep deprivation was not studied.

3.3. Characteristics of wicket group

Wicket spikes were usually observed within 1–2 days of monitoring. The majority of wickets (39/84, 81%) occurred during sleep, and most of them (62.5%) were left-sided. There was also a trend towards wickets being associated with wakefulness (25.0%) versus drowsiness (18.8%), but the absolute difference was minimal (Table 4).

4. Discussion

Wicket spikes are benign EEG signals that are frequently misinterpreted as epileptiform discharges. Using long-term video-EEG monitoring, we were able to document associations between wicket spikes and clinical events. This study found that most patients with wicket spikes did not show epileptiform discharges on video-EEG monitoring. These results are consistent with previous findings that suggest a negative correlation between epileptiform discharges and the presence of wicket spikes in routine EEGs.[5]

Furthermore, wicket spikes were positively correlated with prior diagnosis of seizures. In this study, 14.6% of the patients in the wicket group received the diagnosis of PNES. Previous studies have not specifically documented PNES, but they have noted a significant association between wicket spikes and psychiatric problems[9]; this relationship may be reflected in the PNES documented in this study. However, the proportions of PNES in the wicket and control group were not statistically significantly different ($p = 0.433$).

Additionally, patients with wicket spikes were significantly more likely to have a history of headaches than gender- and age-matched controls ($p = 0.006$). This finding is consistent with previous

Table 2
Clinical variables associated with wickets vs. control group.

	Unadjusted		Adjusted for age, gender	
	OR (95% CI)	p-Value	OR (95% CI)	p-Value
Age (years)	1.00 (0.98, 1.02)	0.990	1.00 (0.98, 1.02)	0.990
Gender (female)	1.00 (0.44, 2.24)	1.000	1.00 (0.45, 2.24)	0.999
Headache	3.19 (1.39, 7.34)	0.006	3.34 (1.43, 7.83)	0.005
Migraine	3.84 (1.22, 12.07)	0.021	4.08 (1.26, 13.15)	0.019
Dizziness/vertigo	28.60 (3.47, 235.44)	0.002	28.62 (3.48, 235.66)	0.002
Autism	NA	NA	NA	NA
Brain tumor	NA	NA	NA	NA
CVD	0.68 (0.24, 1.92)	0.467	0.65 (0.22, 1.93)	0.440
CAD	1.53 (0.37, 6.38)	0.557	1.57 (0.36, 6.82)	0.549
Head trauma	1.41 (0.46, 4.27)	0.548	1.42 (0.46, 4.36)	0.543
Hypertension	1.75 (0.80, 3.87)	0.164	2.02 (0.82, 4.98)	0.125
Diabetes mellitus	0.78 (0.25, 2.48)	0.676	0.78 (0.24, 2.50)	0.670
Seizures	2.30 (1.16, 4.55)	0.017	2.35 (1.17, 4.69)	0.016
Depression	1.08 (0.37, 3.17)	0.889	1.08 (0.36, 3.23)	0.887
Currently on AED	1.40 (0.71, 2.79)	0.332	1.41 (0.71, 2.81)	0.330
AEDs tapered	2.78 (1.18, 6.52)	0.019	2.76 (1.17, 6.50)	0.020
Activation	2.20 (1.01, 4.81)	0.047	2.24 (1.02, 4.89)	0.044

CAD, coronary artery disease; CVD, cerebrovascular disease; AED, antiepileptic drugs; Activation, hyperventilation/photic stimulation.

Table 3
EEG diagnoses, reported as % (*n*).

EEG diagnosis	Wickets (*n* = 48)	Controls (*n* = 144)	*p*-Value*
Normal EEG (no clinical events)	56.3 (27)	32.6 (47)	0.004
Normal EEG (psychogenic non-epileptic seizures)	14.6 (7)	10.4 (15)	0.433
Generalized epileptiform discharges	10.4 (5)	4.2 (6)	0.107
Focal epileptiform discharges	8.3 (4)	27.3 (39)	0.007
Diffuse slow activity	8.3 (4)	18.1 (26)	0.108
Focal slow activity	2.1 (1)	7.6 (11)	0.168

* *p*-Value calculated using chi-squared test.

reports.[5,9] Our study purposefully distinguished between a history of headaches and a more specific diagnosis of migraines. Previous studies have shown a significant association between concurrent migraine and epilepsy.[13] While nonspecific headaches can be associated with seizures, they are usually described in the context of pre- and post-ictal events as opposed to a separate comorbid diagnosis. In our patients, prevalence of migraine was higher in the wicket group than in the control group ($p = 0.015$). However, it is difficult to distinguish the two with certainty in this retrospective study, as it could not be confirmed whether examiners had reliably distinguished history of headaches from migraines. Patients with dizziness or headaches are rarely referred for EEG except in cases of questionable seizures. In such complicated cases the findings of statistically significant associations between those symptoms and wicket spikes may help with diagnosis. In case of migraine, there is evidence of involvement of similar cellular mechanisms as in epilepsy,[14] which may explain the development of the "epileptiform-like" wicket spikes, possibly representing increased synchrony in a critical mass of neurons.

The combined category of dizziness and vertigo, which was present in 8 out of 48 (16.7%) wicket patients compared to one patient (0.69%) in the control group, showed a strong association with wicket spikes ($p < 0.001$). Despite few case reports of epileptic vertigo, there is no strong evidence to correlate dizziness/vertigo with seizures.[15] Therefore, presence of dizziness/vertigo might help differentiate wicket patients from those with epilepsy. Prior studies have not specifically evaluated an association between wickets and dizziness or vertigo. However, Krauss et al.[9] reported that wicket patients are more likely to have experienced episodes of syncope, light-headedness, and fall related to their clinical events, which could represent a subjective description of dizziness/vertigo.

Patients with wicket spikes were less likely to have epileptiform discharges, and were more likely to have a normal EEG when compared to patients without wickets. While they had slightly higher prevalence of PNES, this difference was not significant compared to controls. We did not study the timing of wickets to the occurrence of epileptic or non-epileptic seizures.

Our study found that wicket spikes were most often lateralized to the left hemisphere and were present more during N1 and N2 sleep (stage 1 and 2 non-REM sleep, respectively) than wakefulness, which is consistent with previous data.[5,9] Previous studies have reported a higher incidence of wickets in somnolence compared to wakefulness, but the current study indicates the opposite. This difference could reflect the higher ratio of wakefulness to drowsiness during long term monitoring than a routine EEG, which has been the source of available data on wicket spikes.

We also documented that the median day for the first wicket spike to be observed was day 1–2 of video-EEG recording; this finding suggests a higher likelihood of detecting wicket spikes if a long-term, rather than routine, EEG is obtained. However, the high incidence of wickets in our patients (10.0%) may not reflect the true incidence in the general population, as this study was performed in a select group of patients at a tertiary care center.

The observed increase in likelihood of AED tapering and activation procedures in patients with wickets seems to reflect the standard procedures used in epilepsy monitoring units to induce seizures. Therefore, this procedure is more likely to have been used in patients without clinical seizures or true epileptiform activity throughout the monitoring. However, the association between tapering the AEDs and clinical events was not studied.

Table 4
Characteristics of wicket spikes on long term video-EEG monitoring (*n* = 48).

Clinical condition		% (*n*) or mean ± SD
Laterality	Right	10.4 (5)
	Left	62.5 (30)
	Bilateral	25.0 (12)
Day of monitoring when wicket spikes first recorded	Median (IQR)	1 (1)
	Mean (SD)	1.42 (0.65)
	Day 1	66.7 (32)
	Day 2	25.0 (12)
	Day 3	8.3 (4)
Length of EMU stay (days)		3.35 ± 1.63
Stage of sleep/wakefulness	Awake	25.0 (12)
	Drowsy	18.8 (9)
	Asleep	81.3 (39)
	N1	43.8 (21)
	N2	41.7 (20)
	Awake and drowsy	6.3 (3)
	Awake and asleep	14.6 (7)
	Drowsy and asleep	10.4 (5)
Activation procedure used		29.8 (14)

EMU, epilepsy monitoring unit; N1 & N2, stage 1 & 2 non-REM sleep, respectively.

5. Conclusion

To our knowledge, this is the first study of wicket spikes using long-term video-EEG monitoring. The findings of this case–control study provide further data to better delineate wicket spikes and their clinical implications including non-epileptic seizures, and to differentiate them from true epileptiform activity thereby preventing misdiagnosis of epilepsy. The fact that the EEGs were reviewed by a single interpreter, while avoiding the inter-interpreter variability, might be a possible weakness of this study. Larger studies with more patients might be needed to confirm these findings.

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