ABSTRACT - Wicket spikes (WS) are a benign eletroencephalogram (EEG) variant, seen mainly in adults, during somnolence, in the temporal regions, in many clinical situations. WS can appear in trains or isolatedly, sometimes being difficult to differentiate from epileptiform activity. We reviewed 2,000 EEG's, found 65 with WS (3.25%) and compared them with 65 normal EEG without WS. There was statistically significant (SS) association between WS and age over 33; adolescent age was correlated to absence of WS and age over 65, to the presence of WS; there was an inverse correlation between WS and epilepsy, related to differences in age; a SS association with cerebrovascular disorders disappeared after controlling for age; a SS correlation with headache was also related to age; female predominance was not SS. There was a great variety of clinical situation associated with WS. We conclude that WS are a inespecific normal variant of the EEG that is age-related.

KEY WORDS: Wicket spikes, benign variants, eletroencephalogram.

Wicket spikes (WS) are a relatively uncommon eletroencephalografic (EEG) pattern of uncertain significance. They were described for the first time by Lebel and Reither in 1977. In spite of being frequently cited, it has been infrequently investigated. This pattern is seen during wakefulness, somnolence and synchronized sleep, particularly in the last two. It can be found through all ages, but mainly in adults, in both hemispheres. Its preferential localization is on the left hemisphere. WS are characterized by trains of arciform waves, with a frequency of 6 to 11 Hz, that looks like the mu rhythm. Generally, they present a smooth increase of amplitude, ranging from 60 to 200 microvolts, followed by an abrupt decrease of amplitude, to finally reach the basal line. However, they may also
occur as isolated waves, with “phase-reversal”, sometimes being difficult to differentiate them from epileptiform activity in the temporal lobe\textsuperscript{1-7}. Figure 1 shows typical WS.

Other patterns, such as fourteen and six per second positive spikes, rhythmic temporal teta bursts of drowsiness (psychomotor variant), subclinical rhythmic eletrographic (teta) discharge of adults (SREDA), six-Hertz spike and wave (phantom), among others, are also benign variants of the normal EEG. All have a characteristic morphology but can pose some difficulty to be differentiated of epileptiform abnormalities\textsuperscript{1,5-7}.

The objective of this paper is to review the EEG characteristics of WS and to correlate it with some clinical data in a case-control study.

METHOD

We reviewed 2,000 EEG’s, performed between 1992 and 1995, that were at least 30 minutes long, registered in wake as well as in somnolence and that had the intermittent fotoestimulation and eyes open/eyes closed activation procedures. The hyperventilation was not performed if there was any contraindication. Sleep records were always spontaneous. We could find 65 EEG’s with a WS pattern. Then, we retrospectively collected the clinical (age, sex and diagnostic hypothesis [DH]) and computed tomography scan data, from hospital file-cards. We also obtained 65 normal EEG, that did not show WS, performed during 1995 and backwards, noting age, sex and DH for comparison to the WS group.

The EEG studies were all performed in an eight channel EEG machine and 21 electrodes were carefully placed, obeying the international 10-20 system and using monopolar and bipolar montages.

RESULTS

We found 65 WS out of 2,000 EEG’s, resulting in a frequency of 3.2%. Seventy per cent of our WS patients and 60 of non-WS patients were women. This difference was not statistically

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig1.png}
\caption{Fig 1. Fragments of EEG showing runs of Wicket Spikes (WS) at F7 and T4 (left) and an isolate WS at T4 (midle). For comparison, see an anterior temporal spike (right).}
\end{figure}
significant (SS). The mean age of WS patients was 47.7 (standard deviation [SD]= 16.9) and of non-WS patients was 33.7 (SD= 18.8) years. Among all 130 patients, there were 23 younger than 20, 51 between 20 and 45, 38 between 46 and 65 and 18 over 65. We found more old patients in the WS group (13 out of 18) and more teenagers in the non-WS group (19 out of 23). Adolescent age was associated with the non-WS group (Odds Ratio [OR]=0.16; 95% confidence interval [CI]=0.04-0.55, p=0.00). The young adult and adult groups didn’t show SS associations; the old group (over 65) had an OR of 3.00 (95% CI= 0.9-10.53) for WS. Patients older then the mean age of both WS and non-WS group showed an elevated OR for WS (4.05 [95% CI=1.79-9.27] and 3.36 [95% CI=1.49-7.66] respectively).

There were eight patients with cerebrovascular disorders (5 with stroke, 2 with vertebrobasilar insufficiency and 1 with transient ischemic attacks), all in the WS group. This resulted an SS association, with an OR non-calculable (because one of the values was 0) and a p<0.00, in the two-tailed Fischer exact test (because one of the values was 5).

Twenty one of 65 (32%) of WS group and 8 of 65 (12%) of non-WS group had an EEG because of headache, a SS association, with and OR of 0.29 (95% CI=0.11-0.79; p<0.01).

Sixty three percent of non-WS patients had an EEG because of a DH of epilepsy, while only 14% of WS had an EEG because of a DH of epilepsy. This was SS with an OR of 0.09 (95% CI=0.04-0.24; p<0.00). There were 9 patients with an DH of epilepsy in the WS group; in all these, the WS were lateralized (p<0.05). This association, however, became inverse when we also considered the control group, with an OR of 0.27 (95% CI=0.1-0.67) of lateralized WS for the absence of seizures - confirming the inverse association between WS and the DH of epilepsy.

There was a greater variation of clinical diagnosis among the WS patients. Table 1 shows the clinical diagnosis in the two groups.

<table>
<thead>
<tr>
<th>Clinical diagnosis</th>
<th>WS</th>
<th>non-WS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>24/37</td>
<td>9/14</td>
</tr>
<tr>
<td>Cerebrovascular disorders</td>
<td>10/15</td>
<td>0/0</td>
</tr>
<tr>
<td>Seizures</td>
<td>7/10</td>
<td>41/63</td>
</tr>
<tr>
<td>Psychiatric</td>
<td>4/6</td>
<td>0/0</td>
</tr>
<tr>
<td>CNS neoplasm</td>
<td>3/4</td>
<td>0/0</td>
</tr>
<tr>
<td>Subdural hematoma</td>
<td>3/4</td>
<td>0/0</td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>2/3</td>
<td>0/0</td>
</tr>
<tr>
<td>Cardiopathy</td>
<td>2/3</td>
<td>0/0</td>
</tr>
<tr>
<td>Dementia</td>
<td>2/3</td>
<td>2/3</td>
</tr>
<tr>
<td>Myopathy</td>
<td>0/0</td>
<td>2/3</td>
</tr>
<tr>
<td>Post-neurosurgery</td>
<td>0/0</td>
<td>2/3</td>
</tr>
<tr>
<td>Learning difficulties</td>
<td>0/0</td>
<td>1/1</td>
</tr>
<tr>
<td>Undetermined</td>
<td>1/1</td>
<td>0/0</td>
</tr>
<tr>
<td>Others</td>
<td>7/10</td>
<td>8/12</td>
</tr>
<tr>
<td>Total</td>
<td>65</td>
<td>65</td>
</tr>
</tbody>
</table>

Others: vertigo, insomnia, syncope, anxiety, polyneuropathy. CNS, central nervous system.
The neurological examination was abnormal in seven WS patients (10.7%), with deficits such as aphasia and hemiparesis. There was no correlation between the diseased cerebral hemisphere and the side of predominance of the WS. Thirty two patients had a CT scan and 30 of these were normal.

The WS were observed in the wake state but mainly during somnolence and the initial stages of sleep. They appeared more frequently in short trains, around one second duration. The WS occurred always in the anterior and medio temporal regions. In only two occasions, there was spread to ipsilateral central areas. The WS were noted exclusively in left hemisphere in 34 patients (52.3%), in the right hemisphere in 11 (16.9%) and bilaterally in 20 (30.7%). All the patients presented a stable and symmetric cerebral electrical activity, without pathological or uncertain patterns. The activation procedures did not show significant alterations.

DISCUSSION

The 3.2% of WS that we found was a little higher than what is reported in the literature. It may be explained by the fact that we actively searched for WS in our material. Nevertheless, it may be considered that WS are not a very rare pattern.

Although we found a predominance of women, it was not SS when compared with the control group, in accordance with the literature. We found a clear association between WS an age: patients older then the mean age of WS and non-WS groups had a greater chance of presenting WS; of 18 patients older then 65, 13 were in the WS group, with an OR of 3.0 for WS; the adolescent ones were associated to absence of WS (p<0.01). This association was also noted by other authors.

Of the patients with cerebrovascular disorders (CVD), eight were in the WS group, with a SS association. We found no association between CVD and ages over 45 or 65, but the mean age of CVD patients was 50.5 (SD=12.1), very close to the WS group (47.7 [SD=16.9]) and much bigger than the non-WS group (33.7 [SD=18.8]). Moreover, when we examined only adult or older patients, the association between WS and CVD disappeared. The mean age of the patients in the study of Asokan et al., that concluded for a relation between WS and CVD, was 70 year old. We, contrary to these authors, believe that WS are associated primarily to age and age is associated to CVD. However, in a retrospective study, the presence of minor indicatives of CVD might have been missed.

The most interesting associations was between WS and epilepsy. Examining the 130 patients, we found an inverse correlation between the DH of epilepsy and the presence of WS. However, we also found that when the WS is lateralized, there is an association with a DH of epilepsy. Our center is a tertiary one, where the majority of patients are referenced. When a lateralized WS is seen in a less specialized center, it is more frequently confused with an epileptiform abnormality. If these patients repeat the EEG in our center, the DH will be epilepsy. When the correct pattern is identified, an artificial association between WS and a DH of epilepsy appears.

The reported low frequency of epilepsy in the WS patients argues against any strong association between them; in the extensive study of Hughes and Olson, 38% of their WS had epilepsy, while Reiher and Lebel found only 21%. We found only 10.7% and an inverse correlation between WS and epilepsy: when the DH was epilepsy, the chance of seeing WS was lower than with other DH. It is possible that, when there is a clear pathological pattern, the registration of a considered benign one, such as WS, be lower. Or that when epileptiform abnormalities are also present, one may assume that all the patterns are epileptiform, because they may be very similar. These explanations, however, are improbable, because we actively searched for WS. We think that this inverse correlation is caused by a confounder variable: age. The mean age of the WS was 47.7 (SD=16.9), while the mean age of our epilepsy patients was 33.8 (SD=17.0), very close to the non-WS group (33.7 [SD=18.8]). WS are very rare in the young, as our data also clearly showed.

We found a SS between headache and WS. The mean age of patients with headache was 48.4 (SD=17.5), very close to the mean age of the WS group. Again the association of headache to WS
may be an indirect effect of age. However, other authors have found an increased incidence of headache among WS patients\(^5\). The greater variety of clinical diagnosis in the WS group shows its clinical inespecificity and is in agree with what is reported. The most frequent clinical diagnosis were headache, CVD, seizures and psychiatric problems, similar to what was found by other authors\(^3,5\).

Similarly, only 7 out of 65 patients (10.7\%) had abnormal neurological examination, with no correlation between the diseased hemisphere and the side of the WS.

In conclusion WS are a not very rare EEG pattern. It may look like epileptiform potentials and needs to be carefully differentiated from them. In our series, it was correlated to headache and increasing age. We found an inverse correlation of WS to epilepsy, that we explain based on differences of mean age between the groups. Its correlation to clinically evident CVD disappeared when we controlled for age. However, its relation to subclinical CVD can not be ruled out. It must be considered a age-related nonspecific EEG pattern, not rare in adult and elderly patients, seen in a wide range of clinical complaints.

REFERENCES