Electroencephalographic paroxysmal abnormalites and cortical dysplasia are not always associated with epileptic seizures: A case of NREM parasomnia in adulthood

T. Baldini,[1] G. Loddo,[1] F. Mignani,[2] L. Licchetta,[1,], F. Bisulli,[1,2] P. Tinuper,[1,2] F. Provini[1,2]
[1]Department of Biomedical and Neuromotor Sciences, University of Bologna, Bologna, Italy
[2]IRCCS Institute of Neurological Sciences, Bologna, Italy

INTRODUCTION

The differential diagnosis of paroxysmal motor episodes occuring during sleep is challenging and includes arousal disorders (AD) and sleep-related hypermotor epilepsy (SHE). A correct diagnosis is essential to allow a correct management of the patient (especially avoiding inappropriate surgical procedures). We describe the case of a patient with an AD starting during adolescence and persisting in adulthood, with increasing frequency and intensity.

CASE PRESENTATION AND DISCUSSION

A 28-year-old-woman was admitted for sleepwalking episodes during which she reported injuries. Sleepwalking started when she was 13 and the episodes had progressively increased in frequency and intensity throughout the years. Sometimes, they were characterized by falls out of bed or running out of the room. The patient also reported episodes characterized by abrupt awakenings and screaming, occurring in the first part of the night and recurring every night. All the episodes were associated with fear and anguish, and increased in frequency during stressful circumstances.

The patient underwent EEG (also after sleep deprivation), 48-h nocturnal video-polysomnographic (VPSG) monitoring, and brain MRI. EEG tracings revealed interictal left fronto-temporal paroxysmal activities. Brain MRI showed a left temporal focal cortical dysplasia (FCD). During VPSG monitoring we recorded 13 non-stereotyped episodes arising from deep sleep of different intensity. The episodes were characterized by head flexion, head and trunk flexion, head flexion or extension and limb movement, or frightening expressions, often followed by talking or screaming. The episodes could be spontaneous or evoked by noises.

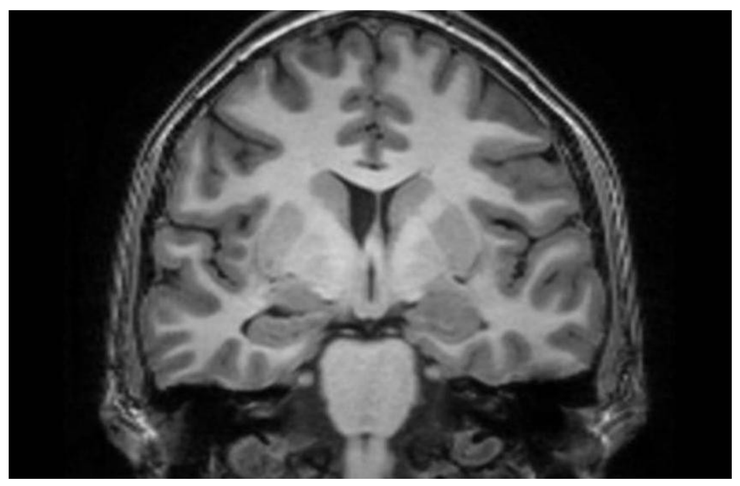

Figure 1. T1-w MRI, coronal plane, showing blurring of the left amigdala, suggestive of focal cortical dysplasia (FCD).

The temporal course (onset during the adolescence, persistence and intensification during adulthood), along with the presence of EEG abnormalities and the finding of FCD, was suggestive of an epileptic origin of these episodes. However, the semiological features of the episodes, especially those observed during the VPSG monitoring (absence of stereotipy, ballic or rythmic movements, dystonic postures or dyskinesia and presence of triggering factors), support the diagnosis of AD.

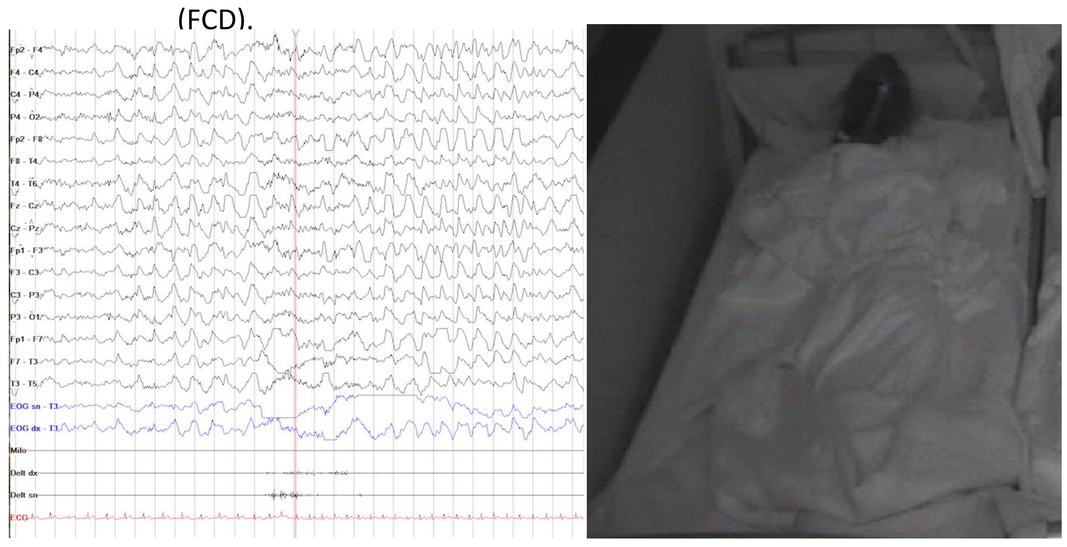

Figure 2. Video-polysomnographic (VPSG) monitoring showing a typical episode characterized by head extension and talking. The episode arose from deep sleep and was associated with a sleep-to-wake transition associated with tachycardia and tachipnoea.

A diagnosis of AD was made and the patient was treated with clonazepam 0.5 mg at bedtime, with resolution of the episodes at a one year of follow-up.

CONCLUSIONS

VPSG still represents the gold standard for the diagnosis of AD and is particularly useful in the differential from SHE, particularly in adulthood and in ambiguous cases.

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