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### **ABSTRACT**

The hypocampus, a temporal lobe structure that is most well known for its central role in emotional behavior, also plays a key role in epileptogenesis. Understanding the mechanisms of recurrent seizures in patients with idiopatic generalised epilepsy and the effect on hypocampus is crucial for the development of therapeutic interventions. Our aim in this study to investigate the existence of a possible effect of seizure recurrence on hippocampus measurements in patients with idiopathic generalized epilepsy.

### **METHODS**

24 patients with juvenil absence epilepsy (JAE) and 28 juvenil myoclonic epilepsy (JME) were included in this study. We selected 35 healthy children as the control group. Demographic and clinical features of patients (frequency and number of seizures) and also antiseizure drugs were recorded. Height and width of hypocampus were measured by two pediatric radiologists on T2 multiecho images obtained via a 1.5-T brain MRI. Patients were divided into two subgroups: group 1:patients with seizure control and group 2:patients with no seizure control within 24 weeks. The ratio of H/W of hypocampus were compared between control group and subgroups.

There were 24 patients with JAE and 28 patients with JME in the study. Children with JAE were significantly younger than the children with JME (p<0.001) and those in the control group (p<0.001). Sex distribution revealed no significant difference between the groups (p=0.969). Height of the right hippocampus was significantly shorter in JME patients than JAE patients and healthy children (p=0.028; p<0.001 respectively). The right and left hippocampus heights were significantly shorter in the JME group (p<0.001 and p=0.020). The ratio of the height and width on the right side was significantly higher in children with JME (p=0.004). Although the width of the left hippocampus was significantly thinner in the JME group (p=0.012), the ratio was similar in the groups (p=0.106) (Table 1). In children with JAE, the width for the left-sided measurements was significantly lower than healthy children (p=0.006). There were no significant differences in hippocampal metrics of the children with JAE and JME based on the presence of controlled and uncontrolled seizures (p>0.05).

Table 1. Demographic specifities and comparison of hippocampal metrics of children with JME and control group.

Sez	ς <sup>‡</sup>
	Male
	Female
Hip me	pocampal trics-right
Hir	pocampal metric

JME (n=28) Control (n=35) 149.0 [123.0 - 193.0] 0.575\* 161.0 [120.0 - 196.0] 12 (42.9) 0.999\*\* 15 (42.9) 16 (57.1) 20 (57.1) <0.001 Height (mm) § 89.0 [66.0 - 101.0] 76.0 [56.0 - 91.0] \* Width (mm) § 173.0 [123.0 - 205.0] 161.0 [113.0 - 205.0] 0.140\* Ratio § 479.0 [406.0 - 573.0] 531.0 [443.0 - 619.0] 0.004\* s-left Height (mm) § 78.5 [63.0 - 97.0] 88.0 [71.0 - 96.0] 0.020\* ppe Width (mm) § 167.5 [132.0 - 183.0] 182.0 [132.0 - 187.0] 0.012\* Ratio § 484.0 [402.0 - 598.0] 513.0 [402.0 - 598.0] 0.106\* <sup>‡</sup>: n (%), <sup>§</sup>: median [min-max] JME: juvenile myoclonic epilepsy \*. Mann-Whitney U test. \*\*. Pearson Chi-Square test.

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# **RESULTS**

1.Caciagli L, Wandschneider B, Xiao F, Vollmar C, Centeno M, Vos SB, Trimmel K, Sidhu MK, Thompson PJ, Winston GP, Duncan JS, Koepp MJ. Abnormal hippocampal structure and function in juvenile myoclonic epilepsy and unaffected siblings. Brain. 2019 Sep 1;142(9):2670-2687.

2.Kim JH, Kim JB, Suh SI, Kim DW. Subcortical grey matter changes in juvenile myoclonic epilepsy. Neuroimage Clin. 2017 Nov 3;17:397-404.

Our study highlights notable right/left differences in H/W ratio in JME patients. It is not clear yet whether these changes are the cause or the outcome of epilepsy, however, that increased in right H/W ratio of hippocampus in JME patients than healthy controls may be the disease progression. Our findings support the pathophysiological hypothesis hippocampal of network abnormality underlying JME which is associated with reorganization of function underlying imply and an neurodevelopmental mechanism in other studies. Future studies might help elucidate the pathophysiology behind the etiology of idiopatic generalised epilepsies.

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## CONCLUSIONS

## **REFERENCES**