SV2A PROTEIN LEVELS IN THE KAINIC ACID EPILEPSY RAT MODEL DURING THE ACUTE PHASE

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Introduction

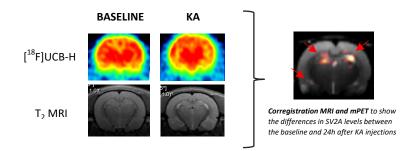
The Kaïnic Acid model (KA) is one of the most validated models of temporal lobe epilepsy (TLE) (Lévesque *et al.*,2016). Its administration induces status epilepticus (SE), characterized by an extensive neuronal damage in limbic structures (Sperk *et al.*,1983). Post-mortem studies, such as the epilepsy model presented in (Wang *et al.*, 2014), show a reduction of SV2A protein levels during the chronic phase, however, no data have been reported during the acute phase (0-48h after KA injection).. The present pilot study is undertaken to evaluate *in vivo*, with the specific radiotracer [¹⁸F]UCB-H (Bretin *et al.*, 2015; Warnock *et al.*, 2014), the SV2A expression 24h after a SE produced by KA administration.

Methods

Two Sprague-Dawley rats were scanned at two different times: baseline, and 24h after three systemic injections of 5mg/kg KA. The scanning process consisted of a first scan with microPET (Focus 120), during 1 hour, using [¹⁸F]UCB-H (41 \pm 5 MBq IV tail vein). This is followed by MRI (9.4T Agilent, anatomical T2). A coregistration was performed with PMOD 3.6 software. Data were expressed as SUV and AUC were calculated for the different brain regions.

Results

[¹⁸F]UCB-H microPET images exhibited a small reduction (around 10%) in SV2A brain levels after KA injections compared to the baseline, marked in thalamus, hippocampus and amygdale. MRI images obtained 24h after KA injections are in accordance with previous histological studies, revealing inflammatory edema, tissue necrosis and increased ventricle volume (Sperk *et al.*, 1983).



Conclusions

These preliminary results obtained in KA treated rats show that [¹⁸F]UCB-H is able to detect alterations in SV2A levels in relevant regions for epilepsy. This radiotracer emerges as a valuable tool to follow in vivo SV2A through longitudinal studies. KA model in rats deserves as a tool for the study of epilepsy, exhibiting the same features than the human disease.

References

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