Original article

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HMGB-1, TLR4, IL-1R1, TNF-α, and IL-1β: novel epilepsy markers?

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Epileptic Disorders

Inflammation and epilepsy

- Some specific inflammatory molecules and their receptors may mediate neuronal cell loss and contribute to synaptic plasticity in this process.
- Various factors, such as trauma, stroke, febrile seizure, status epilepticus, infection, and genetic mutations can lead to activation in microglia, astrocytes and neurons.
- The best known markers for revealing this effect, in addition to interleukin-1β (IL-1β), is high mobility group box 1 protein (HMGB-1), which is involved in IL-1/TLR (toll-like receptor) signal activation.

Inflammation and epilepsy

- The release of these inflammation molecules occurs with the effects described, and together with an increase in Calcium, this results in NMDA receptor activation, in turn, leading to seizures.
- In addition to these markers, there are studies showing that tumour necrosis factor-α (TNFα) and interleukin-1β (IL-1β) are elevated during ischaemia and seizure.

Our aim and what we found…

- There are not many clinical studies on this subject. Therefore, this study was to compare HMGB-1, TLR4, IL-1β, IL-1R1, and TNF-α levels in patients with mild and severe epilepsy.

- In our study; HMGB-1, TLR4, TNF-α, and IL-1β levels in the severe epilepsy group were higher than in the control group and the mild epilepsy group, and were also higher in the mild epilepsy group than in the control group.
- IL-1R1 was also higher in the severe epilepsy group than in the control group.

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