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MEDICAL SCIENCES

THE ROLE OF IRON METABOLISM DISRUPTION AND FERROPTOSIS ACTIVATION IN THE PATHOGENESIS OF SYMPTOMATIC EPILEPSY

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Introduction. Symptomatic epilepsy is a subtype of epilepsy characterized by seizures resulting from identifiable structural or metabolic brain abnormalities. The condition is highly prevalent, with the World Health Organization estimating that over 50 million people globally suffer from epilepsy. Despite the availability of various antiepileptic drugs, the underlying molecular mechanisms, particularly in symptomatic forms, are not yet fully understood.

Recent attention has been drawn to the role of ferroptosis — a regulated form of cell death dependent on iron and lipid peroxidation — as a potential contributor to neurodegeneration and seizure activity. This study is aimed at examining the role of iron metabolism and ferroptosis in the pathogenesis of symptomatic epilepsy through the assessment of biochemical and neuroimaging markers and their correlation with clinical severity.

Materials and Methods. This observational study included 125 participants: 100 patients diagnosed with symptomatic epilepsy and 25 healthy volunteers matched by age and gender. The patient group was subdivided into:

- Group 1 (n=65): Received standard antiepileptic therapy.
- Group 2 (n=35): Received standard therapy supplemented with alpha-lipoic acid and transcranial magnetic stimulation (TMS).

The following parameters were evaluated:

- Serum iron, ferritin, and transferrin concentrations.
- 4-HNE levels using ELISA (enzyme-linked immunosorbent assay).
- Clinical symptoms quantified using the UPDRS (modified scale).
- MRI scans to assess cortical atrophy and white matter damage.

All participants gave informed consent. Statistical analysis was performed using SPSS 26.0, with significance set at $p<0.05$. Correlations between markers were analyzed using Pearson's coefficient.

Results. Biochemical analysis revealed a statistically significant reduction in serum iron and transferrin levels and an increase in ferritin and 4-HNE levels in epilepsy patients compared to controls.

- Ferritin increased to 83.7 ± 2.9 ng/ml (vs. 56.4 ± 2.3 in controls; $p<0.05$).
- Transferrin decreased to $22.3 \pm 0.9\%$ (vs. $29.6 \pm 1.1\%$; $p<0.05$).
- 4-HNE rose to 38.7 ± 1.4 ng/ml (vs. 21.3 ± 1.0; $p<0.05$), indicating oxidative stress and lipid peroxidation.
- MRI analysis revealed increased cortical atrophy (39.2%) and white matter damage (31.1%) in Group 1.

In contrast, Group 2 patients receiving combination therapy showed:

- Improved biochemical markers, with normalized ferritin and transferrin levels.
- Reduced UPDRS scores (29.6 ± 1.0 vs. 42.1 ± 1.3 in Group 1).
- Decreased neurodegenerative changes on MRI (cortical atrophy 28.5%; white matter damage 20.4%).

There was a strong positive correlation between ferritin and 4-HNE ($r = +0.62$, $p<0.001$) and a negative correlation between transferrin and 4-HNE ($r = -0.48$, $p<0.01$).

Conclusion. This study highlights the significant association between iron metabolism abnormalities and ferroptosis in symptomatic epilepsy. Elevated ferritin and 4-HNE levels, along with MRI and UPDRS findings, point to an ongoing neurodegenerative process driven by iron-induced oxidative stress. The 4-HNE biomarker, in particular, demonstrates strong potential for use in early diagnosis, severity assessment, and monitoring treatment effectiveness. Integration of ferroptosis markers into clinical protocols could improve outcomes for epilepsy patients and facilitate personalized treatment approaches.

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