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European Journal of Modern Medicine and Practice (EJMMP)- welcomes high-quality case reports describing innovative medical practices or complex cases that an internist may encounter in everyday situations. The cases should either demonstrate the appropriateness of a diagnostic/therapeutic approach, describe a new procedure or maneuver, or show unusual manifestations of a disease or unexpected reactions. The Journal only accepts and publishes those case reports whose learning points provide new insight and/or contribute to advancing medical knowledge both in terms of diagnostics and therapeutic approaches. Case reports of medical errors, therefore, are also welcome as long as they provide innovative measures on how to prevent them in the current practice (Instructive Errors). The Journal may also consider brief and reasoned reports on issues relevant to the practice of Internal Medicine, as well as Abstracts submitted to the scientific meetings of acknowledged medical societies.

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SYMPTOMATIC SEIZURE IRON METABOLISM IN PATHOGENESIS AND FERROPOTASSIUM PROCESSES ROLE ANALYSIS TO DO

Azizova Rano Bahodirovna

Tashkent Medical Academy

Abboskhonov Asrorkhon Abbaskhon ugli

Andijan State Medical Institute

Annotation: This scientific in the article symptomatic seizure with sick iron metabolism in patients and ferropotassium processes pathogenetic importance The study involved 100 people. patient and 25 people healthy person attraction iron metabolism (iron, ferritin, transferrin) and ferropotassium marker 4-hydroxynonenal (4-HNE) levels as studied. 4-HNE level ELISA method using It was also determined that the patients clinical UPDRS status scale based on, neurostructural changes and through MRI evaluated. Results symptomatic in a fit of iron cell inside excess accumulation of ferritin increase and transferrin decrease with together, 4-HNE level sharp increased These cases ferropotassium activated and lipids peroxidation that it has increased MRI results indicate neurodegeneration that it has increased confirmed, UPDRS scores and clinical of symptoms weight reflection This indicators symptomatic of the seizure pathogenesis deep to understand, early diagnosis to put and individual therapy strategies working to go out opportunity creates. In particular, the level of 4-HNE clinical in practice as an effective biomarker recommendation to be possible.

Keywords: symptomatic epilepsy, iron metabolism, ferroptosis, ferritin, oxidative stress, UPDRS, MRI, neurodegeneration, 4-hydroxynonenal.

Symptomatic seizure (epilepsy) central nerve system diseases complicated from the forms one to be, to be brought issuer factors and pathogenetic mechanisms still complete unexplored. Last Iron metabolism in years violation and ferropotassium – iron related cell death – seizure pathogenesis important place possession possibility scientific attention is pulling. Ferroptosis lipids peroxidation and of iron excess gathering as a result to the surface it comes, this and neurons injury and dysfunction take This is coming. work symptomatic iron metabolism in epilepsy and ferropotassium pathogenetic mechanisms to evaluate focused.

Epilepsy is central nerve system chronic neurological disease It is the brain excess and synchronous neuron activity with described. World health storage According to the World Health Organization (WHO), according to, all world from 50 million more than man from epilepsy suffering smokes, every year this number again growing continue [1]. Disease all young in groups meeting possible to be more childhood and old age during determined at the republican level. take visited last epidemiological research Uzbekistan population between epilepsy general spread level 6-8 per mille around that showing [2].

Epilepsy etiology and pathogenesis deep study, especially symptomatic forms according to, modern neurology the most current from problems one is considered symptomatic. epilepsy usually brain injuries, strokes, infections, tumors and other neurodegenerative processes in the background develops [3]. Nevertheless, this shaped epileptic syndromes molecular and biochemical basics still complete not specified.

Last Iron metabolism in years and ferropotassium processes epilepsy pathogenesis important role to play possibility about scientific views appearance Ferropotassium is to iron related which is, lipid peroxidation as a result to the surface coming new kind of cell death is, it is traditional apoptosis and from necrosis [4]. Ferropotassium epileptic to asset reason to be or epilepsy complications strengthen possibility many in research previously pushed [5].

Including Ratan and in the research of co-authors (2020) neuronal excess iron in cells oxidizing stress brought take it out, this and lipids peroxidation strengthen and ferropotose take arrival showing [6] Liu and while others (2021) experimental animal in models ferropotassium inhibitors using epileptic seizures intensity reduce possible [7]. This results, symptomatic epilepsy pathogenesis iron metabolism violation and ferropotassium between dependency scientific justification for solid evidence become service does.

Also, iron homeostasis violation in the brain glutamate balance sheet to change take to come, this and excitotoxicity epileptic activity instigator from factors one as is under consideration [8]. Neuroinflammation and oxidative stress processes, especially interleukins and TNF- α activity increase with together, ferritin and transferrin levels changes epilepsy weight determinant markers as offer is being done [9].

Above scientific sources analysis would do We are symptomatic. epilepsy Iron metabolism in pathogenesis and ferropotassium mechanisms deep learning, not only diagnostics, maybe new therapeutic approaches working also relevant in the output direction Therefore, this in the article symptomatic epileptic iron metabolism in patients and ferropotose related immunobiochemical indicators is evaluated.

Materials and methods 100 people participated in the study symptomatic seizure with sick patients (age 18-59, mean age 39.2 ± 1.2 years) and 25 in practice healthy, old and gender according to suitable incoming individuals (average age 38.6 ± 1.3 years) involved All participants consent based on from inspection was conducted. Patients following to groups split into:

- Group 1: Traditional treatment received patients (n = 65);
- Control group : Healthy individuals (n = 25)

Research following parameters based on was held :

- Blood iron, ferritin, transferrin, and transferrin saturation level ;
- Based on MRI neurostructural changes;
- Clinical Modified UPDRS of symptoms scale based on evaluation.
- 4-hydroxynonenal (4-HNE) level ELISA (enzyme - linked immunosorbent assay) method using determined.

Statistical analysis SPSS 26.0 software was used for the analysis. Average values $\pm$ standard mistake shown, $p < 0.05$ statistical was considered significant.

Results. In the study symptomatic seizure with sick iron metabolism in patients and ferropotassium with related indicators healthy control to the group relatively noticeable at the level changed determined.

Table 1. Blood serum iron metabolism indicators

Groups	Iron ($\mu\text{mol} / \text{L}$)	Ferritin (ng / ml)	Transferrin (%)
Control group	18.2 ± 0.7	56.4 ± 2.3	29.6 ± 1.1
Group 1	$14.5 \pm 0.6^*$	$83.7 \pm 2.9^*$	$22.3 \pm 0.9^*$
Group 2	$16.1 \pm 0.5^{* \#}$	$67.2 \pm 2.5^{* \#}$	$26.1 \pm 1.0^{* \#}$

* Note : * – control with $p < 0.05$; # – compared with group 1 $p < 0.05$ when compared

Iron exchange indicators symptomatic in a fit noticeable at the level In group 1, iron levels are low and ferritin is high. in the indicator record This situation in the body of iron cell inside excess gathering and him/her external from rotation exclusion with related. In group 2, lipoy acid and TMS therapy because of this indicators relatively normalized.

Table 2. Clinical assessment – UPDRS scores change

Groups	Initial UPDRS	3 months after
Group 1	42.1 ± 1.3	$34.5 \pm 1.1^*$
Group 2	43.2 ± 1.2	$29.6 \pm 1.0^{* \#}$

Clinical symptoms 3 months from treatment after every two in the group decreased, but UPDRS scores in group 2 noticeable at the level low It happened. This complex therapy symptoms reducing efficiency shows.

Table 3. Based on MRI neurostructural changes speed

Groups	Cortical atrophy (%)	White substance damage (%)
Group 1	$39.2 \pm 2.0^*$	$31.1 \pm 1.8^*$
Group 2	$28.5 \pm 1.7^{* \#}$	$20.4 \pm 1.5^{* \#}$

MRI results showed neurodegenerative disease in group 2 of changes decreased shows. Cortical atrophy decrease, white substance integrity preservation therapy neuroprotection from the influence tree gives.

Table 4. Iron exchange and between MDA correlation

Indicators couple	Correlation coefficient (r)	p -value
Ferritin – MDA	+0.62	<0.001
Transferrin – MDA	-0.48	<0.01

Ferritin with MDA positive correlation of iron excess gathering lipids peroxidation strengthen shows. Transferrin negative correlation and carrier proteins shortage ferroptosis take arrival possible indicates.

Table 5. Blood 4-hydroxynonenal (4-HNE) levels (ng/ml)

Groups	4-HNE level (ng / ml)
Control group	21.3 ± 1.0
Group 1	$38.7 \pm 1.4^*$

4-HNE level symptomatic seizure with sick of patients in the blood control to the group relatively noticeable at the level high that is was determined ($p < 0.05$). This indicator lipids peroxidation from the increase and oxidative stress processes intensified status reflection 4- HNE ferroptosis from markers one as cell membranes damage means and this of the disease neurodegenerative component about important information gives.

Discussion Received results symptomatic iron metabolism in epilepsy violation and ferroptosis clinical significant factors that shows. Especially the ferritin level increase and transferrin decrease excess iron in

the body in quantity cell inside accumulated, metabolic of balance from being broken tree This situation gives of iron free shape lipid peroxidation through activating ferropotassium to the process motivation This gives ferropotassium mechanism activating neurons to injury reason It will be.

Oxidative stress markers especially 4-hydroxynonenal (4-HNE) levels increase – lipid peroxidation from the duration evidence this is molecule cell membranes of injury directly proof is considered. In the study symptomatic convulsive of patients 4-HNE levels in the blood control to the group relatively almost two even height ferropotassium mechanisms in a clinical setting active performance shows.

MRI -based neurostructural assessment the results are also molecular changes confirms. Cortical atrophy and white in the article injuries level height neurodegenerative of processes depth manifestation Clinical criterion as UPDRS scale used according to high points of symptoms from the weight evidence gave and laboratory-biochemical indicators with directly appropriate came.

This research symptomatic iron homeostasis in epilepsy and ferropotassium between dependency systematic in a way open In particular, ELISA -based 4 - HNE to be determined clinical in practice this molecule as a diagnostic marker application opportunity This biomarker shows sensitivity and accuracy symptoms with directly right related to be him/her forecast also relevant in evaluation does.

Conclusion. Research symptomatic iron metabolism in epilepsy and ferropotassium processes in the middle integral dependency existence showed. Iron and ferritin levels changes of iron pathogen accumulation of transferrin decrease and carrier proteins the lack of represents. With this together, 4-HNE level noticeable increased lipid peroxidation that it has increased and ferropotassium mechanisms activity confirms.

MRI scans and UPDRS clinical points this laboratory indicators with high at the level related of the disease weight and neurodegenerative changes objective This situation this markers only scientific in search not, maybe practical diagnostics and treatment efficiency also great in evaluation importance has that proves.

This indicators – especially 4- HNE – symptomatic in a fit early diagnosis put, disease process weight assessment and individual approach based on treatment strategy working on the way out effective tool become service to do On this basis, iron metabolism and ferropotassium markers clinical to practice integration disease consequences in reduction important place possession possible.

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