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The Effect of Antishemin on Some Respiratory Tissue Enzymes in Cerebral Ischemia Model

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Abstract

Today, biology, biochemistry, and molecular biological sciences are highly developed that glycolysis, Kreb's cycle, pentose monophosphate path have discoverd and to study all diseases, mainly biochemistry of ischemic heart disease "The membrane-redox potentials three-state line system dependent-full 9 stepped cycle of proton conductance" by M.Ambaga is necessary to learn. **Conclusion**: Lactate dehydrogenase (LDH ng / L) was increased by 39.8% -66.9% in brain tissue homogeneity, which is dominated by oxygen-rich oxidation or anaerobic oxidation, and brain energy may be possible to use the glycolysis line to full the deficit. In contrast, the levels of lactate dehydrogenase were decreased by 14.5-33.9% compared to the control group with Antischemin group100 mg / kg dose, and when brain is lack of oxygen, ATP is increased by endogenic antioxidant is elevation to supply the energy. The control group that did not use succinate dehydrogenase enzyme in the cerebral homogenate was reduced to 23.8% -39.75% compared to the healthy group in cerebral ischemia, which inhibited the loss of electrons and protons in the cellular respiratory chain, it seems to be lacking. On the basis of the 100mg / kg dosage of Antischemin, the activity of the cyclic dehydrogenase activity increased by 10.23% -22.5% in theday 1-21 of the cerebral ischemia. The biologically active ingredient in the naturally occurring extracts Astragalus membranaceus, Scutellaria baicalensis georgi, and Ginkgo biloba, the compounds are strongly antioxidant, with this action preventing electrons and proton transmit in mitochondrial membranes during acute and chronic deficiency of cerebral ischemic oxygen, thereby energizing the organs and the improvement of the supply.

Keywords: Cerebral ischemia; lactate dehydrogenase; succinate dehydrogenase; "The membrane-redox potentials three-state line system dependent-full 9 stepped cycle of proton conductance".

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1. Introduction

The living cells are the result of the evolutionary model that produces ATP and heat as energy resource by creating proton gradient with membrane compounds and free the electron and protons from protonized compounds from last 4 or 5 billion years [1]. Today, biology, biochemistry, and molecular biological sciences are highly developed that glycolysis, Kreb's cycle, pentose monophosphate path have discoverd and [1] to study all diseases, mainly biochemistry of ischemic heart disease "The membrane-redox potentials three-state line system dependent-full 9 stepped cycle of proton conductance" by M.Ambaga is necessary to learn [4-6]. This closed circuit consists of two parts, and A or the first one to seven is regulated by the internal membrane of mitochondria [2] and the respiratory chain or electronic transport chain reaction in the mitochondrial membrane is carried out and the result is ATP, the heat generated by human and animal life conditions is created. Lactate dehydrogenase is an enzyme located on the internal mitochondrial membrane, which is oxidized by lactic acid into pyruvate [7-9], initiating the metabolic rate of the living cell and the cell respiratory chain. Also, it shows the energy of the organs requiring oxygen, such as heart and brain, exhibits the intensity of oxygenation and phosphorylation. Succinate dehydrogenase is a II complex protein, which is located on the internal membrane of all prokaryotic and eukaryotic mitochondrial membranes [7-9]. Thus, in the model of the cerebral ischemia in experimental animals, these variations of the two parameters are one of the main areas of study.

Purpose: In cerebral ischemic created rat, we study the effect of Antischemin on lactate dehydrogenase (LDL ng/L) and succinate dehydrogenase (μmol/L) activity.

2. Material and methods

The cerebral ischemic models were performed by Farkes E and his colleagues 2007 method [3]. After anesthesized with ketamine of 220-280 grams and immobilized on the operating table, a special laryngoscope was inserted into the trachea by inserting a polyethylene tube and combine into a small animal ventilator R407 RWD Life Science with an average breathing rate of 94-104/min and 8-2.0 cc volume. The procedure is performed with head light in aseptic conditions. Neck side hair was cut and after sterilizing with 5% of iodine solution the operation area, left carotid artery was pulled out and wrapped with special suture. In control group water is given, in comparing group 40 mg/kg Bilobil is given and in the test group Antischemin (1;1;1) with dose of 100 mg/kg is given orally for a 28-day. During the day 1, 3, 7, 14, 21 and 28, the lactate dehydrogenase (LDL ng/L) and succinate dehydrogenase (μmol/L) were measured by ELISA in cardiac tissue homogenate.

3. Result

In our study, comparing LDH enzyme activation in untreated cerebral ischemic white rat with healthy group, it was increased by 46.37% on day 1 (control group 10.22±0.20 ng/L, healthy group 5.48±0.14 ng/L, $P<0.001$), 39.8% on day 3 (control group 10.23±0.10 ng/L, healthy group 6.15±0.14 ng/L, $P<0.001$), 3.02 times or 66.9% on day 7 (control group 12.91±0.12 ng/L, healthy group 4.27±0.02 ng/L, $P<0.001$), 47.3% on day 14 (control group 11.54±0.14 ng/L, healthy group 6.08±0.16 ng/L, $P<0.001$), and 1.97 times of 49.2% on day 21 respectively.

Table 1: The level of lactate dehydrogenase (LDL ng/L) in cerebral tissue of cerebral ischemia created experimental white rat and the effect of Antischemin.

№	Trial days	Lactate dehydrogenase (LDH ng/L)			
		Healthy n=6	Control n=6	Antischemin-100 mg/kg n=6	Bilobil-40 мг/кг n=6
1	Day 1	5.48±0.16	10.22±0.20*	8.70±0.26**	8.78±0.16**
2	Day 3	6.15±0.14	10.23±0.10*	8.74±0.27**	8.67±0.19**
3	Day 7	4.27±0.02	12.91±0.12*	8.53±0.25**	5.76±0.27**
4	Day 14	6.08±0.16	11.54±0.14*	8.91±0.10**	7.08±0.12**
5	Day 21	6.29±0.11	12.40±0.19*	9.16±0.17**	9.22±0.27**

*- When control group is compared to healthy group P≤ 0.05, P≤ 0.001

**- When treated group is compared to control group P≤ 0.05, P≤ 0.001

Comparing LDH enzyme activation in Antischemin used group with control group, it was decreased by 14.87% on day 1 (control group 10.22±0.20 ng/L, treated group 8.70±0.26 ng/L, P<0.05), 14.56% on day 3 (control group 10.23±0.10 ng/L, treated group 6.15±0.14 ng/L, P<0.05), 33.9% on day 7 (control group 12.91±0.12 ng/L, treated group 8.53±0.25 ng/L, P<0.001), 22.79% on day 14 (control group 11.54±0.14 ng/L, treated group 8.91±0.10 ng/L, P<0.05), and 26.12% on day 21 respectively. Also in Bilobil used group, it was decreased by 14.09% on day 1 (control group 10.22±0.20 ng/L, Bilobil group 8.78±0.16 ng/L, P<0.05), 9.7% on day 3, 55.38% on day 7 (control group 12.91±0.129 ng/L, Bilobil group 5.76±0.27 ng/L, P<0.001), 38.65% on day 14 (control group 11.54±0.14 ng/L, Bilobil group 7.08±0.12 ng/L, P<0.001), and 25.64% on day 21 respectively (control group 12.4±0.14 ng/L, Bilobil group 9.22±0.27 ng/L, P<0.001).

Table 2: The level of succinate dehydrogenase (μmol/L) in cerebral tissue of cerebral ischemia created experimental white rat and the effect of Antischemin.

№	Trial days	Succinate dehyrogenase (SDH ng/L)			
		Healthy n=6	Control n=6	Antischemin-100 mg/kg n=6	Bilobil-40 mg/kg n=6
1	Day 1	26.06±0.4	15.70±0.17*	17.49±0.24**	17.19±0.33**
2	Day 3	25.05±0.28	17.13±0.48*	20.33±0.44**	19.88±0.36**
3	Day 7	27.11±0.27	19.06±0.19*	24.11±0.25**	23.83±0.13**
4	Day 14	26.35±0.09	19.56±0.19*	25.24±0.25**	24.13±0.28**
5	Day 21	25.46±0.13	19.38±0.23*	22.70±0.39**	20.49±0.26**

*- When control group is compared to healthy group P≤ 0.05, P≤ 0.001

**- When treated group is compared to control group P≤ 0.05, P≤ 0.001

Comparing succinate dehydrogenase enzyme activation in cerebral ischemic white rat with healthy group, it was decreased by 39.75% on day 1 (healthy group 26.06±0.39 μmol/L, control group 15.70±0.173 μmol/L,

P<0.001), 31.6% on day 3 (healthy group 25.05±0.28 μmol/L, control group 17.13±0.48 μmol/L, P<0.001), 29.6% on day 7 (healthy group 27.11±0.27 μmol/L, control group 19.06±0.43 μmol/L, P<0.001), 25.7% on day 14 (healthy group 26.35±0.09 μmol/L, control group 19.56±0.19 μmol/L, P<0.001) and 23.8% on day 21 (healthy group 25.46±0.13 μmol/L, control group 19.38±0.23 μmol/L, P<0.001), respectively. Comparing succinate dehydrogenase enzyme activation in Antischemin 100 mg/kg used group with control group, it was increased by 10.23% or 17.49±0.24 μmol/L on day 1, (control group 15.70±0.173, Antischemin group 17.49±0.24 μmol/L), 15.74% on day 3 (control group 17.13±0.48, Antischemin group 20.33±0.44 μmol/L), 20.9% on day 7 (control group 19.06±0.43, Antischemin group 24.11±0.25 μmol/L), 22.5% on day 14 (control group 19.56±0.19, Antischemin group 25.24±0.25 μmol/L), and 14.6% on day 21 (control group 19.38±0.23, Antischemin group 22.70±0.39 μmol/L) respectively. Also in group of Bilobil with dose of 40 mg/kg, it was increased by 8.66% on day 1, 13.8% on day 3 (control group 17.13±0.48, Bilobil group 19.88±0.369 μmol/L, P<0.05), 20% on day 7 (control group 19.06±0, Bilobil group 23.83±0.13μmol/L), 18.94% on day 14 (control group 19.56±0.19, Antischemin group 24.13±0.28 μmol/L), and 5.42% on day 21, respectively.

Abbreviation: Succinate dehyrogenase=SDH, Lactate dehydrogenase=LDH

4. Discussion

The positive effect of Antischemin preparation with compositions of Astragalus membranaceus, Scutellaria baicalensis georgi, and Ginkgo biloba plants on tissue respiratory chain by change of lactate dehydrogenase and succinate dehydrogenase. Even if it's different from our study methods, some of the researchers demonstrate the same mechanism for results, including Russia, Center for Scientific Research of Pavlov it is defined that intra-cellular succinate dehydrogenase and lactate dehydrogenase were analyzed by histochemistry in cerebral ischemic reperfusion it was activated in 2, 3 and 5 layers of neurons and CA1, CA2, CA3 and CA4 of hypocamp and refers to the completion of the electronic transmission of the inner layer of mitochondria [3]. As a result, when vital organs such as brain and heart in hypoxi, electron transmitting succinate dehydrogenase activity decreases and lactate dehydrogenase activity increased due to in the lack of oxygen and ATP, electron-proton flow slows down and glycolysis increases in metabolism.

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