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## Research Article

### THE EFFECT OF ANTISCHEMIN ON FAT METABOLISM IN CEREBRAL ACUTE AND CHRONIC ISCHEMIA

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#### ABSTRACT

In the cerebral ischemic disease, fat metabolism loss due to lack of oxygen supply in brain cell and deficiency of ATP-high energy enzyme, which in turn we aim to study the effects of Antischemin in fat metabolism. In the composition of Antischemin, herbs such as *Scutellaria baicalensis*, *Astragalus membranaceus* and *Ginkgo biloba*. Global brain ischemia model induction: Unilateral permanent occlusion-Common carotid artery. We have performed using Farkes E et al 2007 methodology. In cerebral ischemic white rat, Antishemin group animals' CH, TG, LDL reduced by 7-13%, 3-16% and 11-16% respectively and HDL increased by 2-19% from day 1-21. Antishemin preparations have been shown to have a positive effect on fat metabolism due to ATP's high energy composition deficiency during acute and chronic cerebral ischemic disease.

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#### INTRODUCTION

In our country, most common disease such as atherosclerosis and diabetes, the main cause of the vascular endothelial damage causing metabolic imbalance in vital organs such as brain and heart is the oxygen deficiency and proton-electron flow loss of "9-step circuits of proton-electron flow in 14 billion cells of human body" [1-6]. Thus, within pathophysiologic path multi-factor dependent levels, producing autoimmune reaction and hypersensitivity inhibiting medicine in hepatic, renal and autoimmune disease as prevention and management is one of the most pressing issues in the medical field of our country [7]. Acute ischemic and cerebral strokes lead to long-term disability and leading in mortality [8]. In the cerebral ischemic disease, fat metabolism loss due to lack of oxygen supply in brain cell and deficiency of ATP-high energy enzyme, which in turn we aim to study the effects of Antischemin in fat metabolism.

#### MATERIALS AND METHODS

Global brain ischemia model induction: Unilateral permanent occlusion – Common carotid artery

We have performed using Farkes E et al 2007 methodology [9]. On white rat with weight of 220-280 gram, we started sedation with ketamine, and have secured rat to immobile which after attached rat to ventilator. We have maintained experimental animal body temperature between 36.7-37°C and reinforced immobile rat to surgery table. With help of laryngoscopy, we have introduced polyethylene tube to trachea, and attached it to ventilator (small animal ventilator R407 RWD Life Science/ and have ventilated 112-114/min, 1.8-2.0 cc volume and performed surgical procedure in aseptic condition with help of surgical headlight.

After cutting hair of lateral neck we have cleaned surgical site using 5% iodine solution and have occluded artery by suture. Gave water to control group, Bilobil to comparison group, 100 mg/kg dose of Antischemin to experimental group for 21 days. On 1, 3, 7, 14, 21 days of experiment animals were sedated with ketamine hydrochloride and we have taken sample blood for heart and measured CH, TG, LDH and HDL concentration were measured by using Enzyme-linked immunosorbent assay kit.

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## RESULTS

In control group without treatment, CH increased up to 39.4-58.1% in days 3-14, indicating that severe fat metabolism loss occurs due to lack of oxygen supply in brain cell and deficiency of ATP-high energy enzyme. During this period, Antischemin group CS decreased by 7-13% immediately than control group ( $P \leq 0.001$ ).

**Table 1** The effect of Antischemin on cholesterol (CH) concentration in cerebral ischemic model of white rat.

No	Days of experiment	Plasma CH mmol/L			
		Healthy	Control	Antischemin 100 mg/kg	Bilobil 40 mg/kg
1	Day 1	76.84±1.24	91.29±1.16	88.62±1.85	87.57±0.67
2	Day 3	77.21±1.05	184.68±10.88*	161.58±14.95**	142.42±9.45**
3	Day 7	89.9±0.98	148.49±4.16*	137.95±5.22	136.87±5.51**
4	Day 14	100.2±0.75	178.33±1.58*	164.98±2.38	164.25±1.99
5	Day 21	71.8±1.23	98.62±1.79	88.27±0.80**	86.47±1.11**

\*- When compared control group measurements with healthy group  $P \leq 0.05$ ,  $P \leq 0.001$

\*\* - When compared treatment group measurements with control group  $P \leq 0.05$ ,  $P \leq 0.001$

**Table 2** The effect of Antischemin on triglyceride (TG) concentration in cerebral ischemic model of white rat.

No	Days of experiment	Plasma TG mmol/L			
		Healthy	Control	Antischemin 100 mg/kg	Bilobil 40 mg/kg
1	Day 1	14.31±0.24	17.54±0.14*	16.98±0.07	16.28±0.20
2	Day 3	15.85±0.21	19.28±0.40*	17.09±0.25**	16.32±0.24**
3	Day 7	16.85±0.49	19.96±0.25*	17.29±0.46**	19.11±0.30
4	Day 14	14.68±0.24	20.51±0.35**	17.34±0.19**	16.85±0.12
5	Day 21	16.0±0.19	23.44±0.09**	19.68±0.41**	19.91±0.32**

\*- When compared control group measurements with healthy group  $P \leq 0.05$ ,  $P \leq 0.001$

\*\* - When compared treatment group measurements with control group  $P \leq 0.05$ ,  $P \leq 0.001$

In control group without treatment, TG in plasma increased up to 16-32% in days 1-21. During this period, Antishemin group TG concentration decreased by 3-16% immediately than control group ( $P \leq 0.001$ ). (Table 2)

**Table 3** The effect of Antishemin on low density lipoprotein (LDL) in cerebral ischemic model of white rat.

No	Days of experiment	Plasma LDL mmol/L			
		Healthy	Control	Antischemin 100 mg/kg	Bilobil 40 mg/kg
1	Day 1	5.03±0.26	6.99±0.12*	6.19±0.10	6.03±0.10**
2	Day 3	4.33±0.2	6.87±0.11*	6.37±0.17**	6.63±0.17**
3	Day 7	5.65±0.61	9.53±0.09*	7.94±0.14	8.05±0.15
4	Day 14	5.02±0.13	8.86±0.19*	6.79±0.33**	6.93±0.13**
5	Day 21	5.53±0.14	7.88±0.14*	6.85±0.11**	7.11±0.11

\*- When compared control group measurements with healthy group  $P \leq 0.05$ ,  $P \leq 0.001$

\*\* - When compared treatment group measurements with control group  $P \leq 0.05$ ,  $P \leq 0.001$

In control group without treatment, LDL in plasma increased up to 28-43% in days 1-21. During this period, Antishemin group TG concentration decreased by 11-16% immediately than control group ( $P \leq 0.001$ ). (Table 3)

**Table 4** The effect of Antishemin on high density lipoprotein (HDL) in cerebral ischemic model of white rat.

No	Days of experiment	Plasma HDL mmol/L			
		Healthy	Control	Antischemin 100 mg/kg	Bilobil 40 mg/kg
1	Day 1	10.2±0.23	7.70±0.08*	8.25±0.08**	8.19±0.14
2	Day 3	11.4±0.27	8.77±0.18*	8.94±0.20	9.24±0.19**
3	Day 7	10.8±0.25	7.39±0.44*	7.70±0.42	7.60±0.39
4	Day 14	10.9±0.21	7.36±0.12*	8.52±0.10**	8.70±0.17
5	Day 21	11.0±0.16	8.72±0.17*	10.7±0.15**	10.4±0.1

\*- When compared control group measurements with healthy group  $P \leq 0.05$ ,  $P \leq 0.001$

\*\* - When compared treatment group measurements with control group  $P \leq 0.05$ ,  $P \leq 0.001$

In control group without treatment, HDL decreased by 21-32% in days 1-21, indicating that in organs such as brain and heart metabolism slows down leading to micro-circulation complications due to fat metabolism imbalance, high density lipoprotein loss and increase in cholesterol, triglyceride and low density lipoprotein. Due to the lack of normal electron and proton flow in the brain cells, that use high amount of oxygen, leads to ATP production reduced in mitochondrial internal membrane and fat metabolism turns into the most inefficient form. During this period, Antishemin group HDL increased by 2-19% immediately than control group ( $P \leq 0.001$ ).

## DISCUSSION

In cerebral ischemic white rat, Antishemin group animals' CH, TG, LDL reduced by 7-13%, 3-16% and 11-16% respectively and HDL increased by 2-19% from day 1-21. Comparing to studies of Zayed AE and Saleh A, the use of Gingko biloba in fat metabolism, the result was not strong enough in cerebral ischemia but comparing to control group, it was statistically significant in some days. Also, as a comparing group. Bilobil in fat metabolism was similar to Antishemin. HDL increasing in Antishemin may result in prostacyclin-thromboxan balance by improving micro-circulation and blood supply due to increase in prostacyclin production in hypoxic lesion in organs such as brain and heart, which have high necessity of ATP and oxygen.

## CONCLUSION

Antishemin preparations have been shown to have a positive effect on CH, TG, LDL reduced by respectively and HDL increased during acute and chronic cerebral ischemic disease.

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