http://dx.doi.org/10.35630/2199-885X/2020/10/33

URIC ACID AS A RISK FACTOR FOR CATARACTS IN PATIENTS WITH GOUT

Kibriyo Odinaeva[™] ₪, Mikhail Frolov ₪, Nashaat Sultan Afif Al Khateeb ₪

¹ Peoples Friendship University of Russia, Moscow, Russia

🖂 kima.med-91@mail.ru

ABSTRACT — AIM. To determine the concentration of uric acid in body fluids and its relationship with the development of cataract.

METHODS. The study included 170 patients with male cataracts, aged 30 to 75 years, who were divided into two groups: the first group (control) — patients without gout 60 patients (60 eyes), the second group (study) — patients with cataract complicated by gout 110 patients (110 eyes). All patients signed an informed consent to participate in the study and the processing of personal data. In addition to standard ophthalmic research methods, all patients were also determined: the level of uric acid in the blood serum, in the tear and in the moisture of the anterior chamber. The diagnosis of gout is established by signs of ACR/EULAR (2015). Microsoft Excel 2019 and Statistic 20. These parameters allow you to analyze deviations from the normal, a comparison of two independent groups using the nonparametric Mann-Whitney test. Data were median (25%; 75% percentile). Differences at which p <0.05 are considered statistically significant.

RESULTS. The concentration of uric acid in biological fluids in patients of the study group was significantly increased compared with patients in the control group (p < 0.0001) CONCLUSIONS. In patients with cataracts and with concomitant gout disease, an increase in the concentration of uric acid is observed not only in the blood, but also in the tear and moisture of the anterior chamber, which may be a risk factor for the development of cataracts. Measuring the concentration of uric acid in body fluids can be a useful tool for monitoring the health status and condition of eye tissues.

KEYWORDS — cataract, uric acid, gout.

INTRODUCTION

Uric acid is the end product of the exchange of purine bases. In normal concentrations, one of its functions is to provide effective antioxidant ability. Elevated plasma uric acid levels can lead to gout and are associated with coronary heart disease, diabetes, and renal failure (Boscia et al., 2000). One reason for this may be that uric acid may act as a prooxidant. It is known that antioxidants under certain conditions are able to act as prooxidants — substances with opposite properties that are detrimental to cells. The eye is vulnerable to oxidative stress. According to some authors (Bunin et al., 1973), changes in the composition of the aqueous humor of the anterior chamber with the formation of abnormal metabolites leads to destructive changes in eye tissue, including cataracts.

MATERIALS AND METHODS

The study included 170 patients with male cataracts, aged 30 to 75 years, who were divided into two groups: the first group (control) — patients without gout 60 patients (60 eyes), the second group (study) — patients with cataract complicated by gout 110 patients (110 eyes). All patients signed an informed consent to participate in the study and the processing of personal data. In addition to standard ophthalmic research methods, all patients were also determined: the level of uric acid in the blood serum, in the tear and in the moisture of the anterior chamber. Gout was diagnosed using ACR/EULAR (2015) criteria. The material was processed statistically using computer programs Microsoft Excel 2019 and Statistic 20. Since the distribution of the parameters of the studied sample deviates from normal, the comparison of two independent groups was carried out using the nonparametric Mann-Whitney test. Data were presented as median (25%; 75% percentile). Differences at which p <0.05 were considered statistically significant.

Samples of tear fluid, aqueous humor and blood serum were taken from patients undergoing cataract phacoemulsification. A 30 µl tear fluid sample was collected in the morning before surgery atraumatically from the lower lateral lacrimal meniscus with a capillary tube (5–25 μl; Roche Diagnostics GmbH, Vienna, Austria). After centrifugation, the samples were transferred to plastic microtubes and stored at -70° C until measurements were made by high performance liquid chromatography (HPLC). Moisture of the anterior chamber was obtained by transcortineal paracentesis of the anterior chamber with a 28 gauge needle connected to an insulin syringe. 100 ml of a moisture sample of the anterior chamber were taken from each patient. Samples were immediately transferred to the laboratory, centrifuged at 1500 g for 10 minutes, and stored at -70° C until use. Uric acid in the tear and in the moisture of the anterior chamber was determined by chromatographic method with electrochemical (EC) detection. The chromatograph (model HP 1100; USA) was equipped with a binary pump, an automatic

109

sample injection system, and an ESA Coulochem Model 5200A with a model 5011 analytical cell. Data was introduced in Chem Station version 8.04. All injections were performed in duplicate.

RESULTS

In all patients of the control and study groups, the level of uric acid in the blood serum, in daily urine, in the tear and in the moisture of the anterior chamber (MIC) was determined (Table 1).

The concentration of uric acid in various media in patients of the study group was significantly increased compared with patients in the control group (p < 0.0001) (Tabl. 1).

Table 1. The concentration of uric acid in various environments in the control and study groups

The concentration of uric acid in various environments	Control group	Study group	Р
In the blood, µmol/l	277.0 (251.0-304.3) ²	697.55 (426.7-801.65) ²	< 0.00011
In a tear, mcg/ml	10.9 (10.6-11.3) ²	23,65 (17.2-27.6) ²	< 0.0001 ¹
In the MIC, mcg/ml	8.0 (7.7-8.3) ²	19.15 (11.925-23.025) ²	< 0.00011

Note: 1 — Mann-Whitney criteria are used for statistical analysis; 2 — median (25% quartile, 75% quartile)

DISCUSSION

Uric acid is the end product of the exchange of purine bases. In normal concentrations, one of its functions is to provide effective antioxidant ability (Cutler, 1984). It was shown that in vivo urates are components of the main extracellular and intracellular antioxidant mechanisms (Yu, 1994). Uric acid is present not only in serum or plasma, but also in sweat, nasal and bronchial fluids. (Housley et al., 1995; Choy et al., 2000; Huang et al., 2002). Elevated plasma uric acid levels can lead to gout and are associated with coronary heart disease, diabetes, and renal failure (Lehto et al 1998). One reason for this may be that uric acid can act as a prooxidant and participate in oxidative stress. It is known that antioxidants under certain conditions are able to act as prooxidants substances with opposite properties that are detrimental to cells.

The eye is vulnerable to oxidative stress. It has been argued that oxidative mechanisms play an important role in the pathogenesis of cataracts (Boscia et al., 2000; Aksoy & Keles, 2001). An increased risk of cataract was found with an increase in serum uric acid levels (Durant et al., 2006).

Our study showed that with gout, elevated uric acid levels not only in blood serum, but also in the tear and moisture of the anterior chamber, which can be a risk factor for cataracts.

The composition of the tear fluid is of interest because tears are the first barrier protecting the cornea from oxidative damage by photodynamic reactions and toxic chemicals (Gogia et al., 1998; Rose et al., 1998). Water humor also protects the eye tissue from changes caused by light, to which the lens and retina are especially sensitive.

Measuring the concentration of uric acid in body fluids can be a useful tool for monitoring the health status and condition of eye tissues. Further research is needed to confirm our findings.

REFERENCES.

- AKSOY, H., & KELES, S. (2001). Diabetic cataract and the total antioxidant status in aqueous humour. Clin Chem Lab Med, 39, 143–145.
- BOSCIA, F., GRATTAGLIANO, I., VENDEMI-ALE, G., MICELLI-FERRARI, T., & ALTOMARE, E. (2000). Protein oxidation and lens opacity in humans. Invest Ophthalmol Vis Sci, 41, 2461–2465.
- BUNIN, A. YA., & YAKOVLEV, A. A. (1973). Abnormal metabolites. Bulletin of Ophthalmology, 5, 5–8.
- CHOY, C. K. M., BENZIE, I. F. F., & CHO, P. (2000). Ascorbic acid concentration and total antioxidant activity of human tear fluid measured using the FRASC assay. Invest Ophthalmol Vis Sci, 41, 3293–3298.
- CUTLER, R. G. (1984). Urate and ascorbate: their possible roles as antioxidants in determining longevity of species. Arch Gerontol Geriatr, 3, 321–348.
- DURANT, J. S., FROST, N. A., TRIVELLA, M., & SPARROW, J. M. (2006). Risk factors for cataract subtypes waterclefts and retrodots: two case-control studies. Eye, 20, 1254–1267.
- GOGIA, R., RICHER, S. P., & ROSE, R. C. (1998). Tear fluid content of electrochemically active components including water-soluble antioxidants. Curr Eye Res, 17, 257–263.
- Housley, D. G., MUDWAY, I., KELLY, F. J., ECCLES, R., & RICHARDS, R. J. (1995). Depletion of urate in human nasal lavage following in vitro ozone exposure. Int J Biochem Cell Biol, 27, 1153–1159.
- HUANG, C. T., CHEN, M. L., HUANG, L. L., & MAO, I. F. (2002). Uric acid and urea in human sweat. Chin J Physiol, 45, 109–115.
- LEHTO, S., NISKANEN, L., RONNEMAA, T., & LAAKSO, M. (1998). Serum uric acid is a strong predictor of stroke in patients with non-insulin dependent diabetes mellitus. Stroke, 29, 653–639.
- Rose, R. C., RICHER, S. P., & BODE, A. M. (1998). Ocular oxidants and antioxidant protection. Proc Soc Exp Biol Med, 217, 397–407.
- Yu, B. P. (1994). Cellular defenses against damage from reactive oxygen species. Physiological Reviews, 74(1), 139–162. doi:10.1152/physrev.1994.74.1.139