International Research Journal in Global Engineering and Sciences. (IRJGES)
ISSN : 2456-172X | Vol. 2, No. 3, September - November, 2017 | Pages 41-45

A Comparative Review of Lipid-Bound Neuraminic Acid Contents in Membranes of Erythrocytes in Patients Suffering by Glaucoma

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Abstract: The purpose of this work used to be to study the content of total and lipid-bound neuraminic (sialic) acid in erythrocyte membranes in patients with glaucoma. The results of our studies confirmed that glaucoma is accompanied via an enlarge in the total content of neuraminic acid bought from glycoproteins and glycolipids. A find out about of the content of gangliosides made it possible to realize a reduce in all fractions of gangliosides, which is related with the hydrolytic breakdown of gangliosides and with the enlarge of free neuraminic acid. The records acquired are interpreted in connection with the destruction of membranes, leading to a disruption of cellular functions.

Keywords: Glaucoma; Erythrocyte membrane; Gangliosides; Neuraminic acid

Introduction

Glaucoma is one of the important reasons of blindness and vision impairment, in spite of the apparent successes in the prognosis and treatment of this disease. Risk elements affecting the incidence of glaucoma encompass older age, heredity (glaucoma in close relatives), diabetes, issues of glucocorticoid metabolism, arterial hypotension. Glaucoma is characterised by means of multiplied intraocular strain (IOP), main to a disruption in the outflow of intraocular fluid in the eye and optic nerve, which leads to the demise of the neurons of the eye. The etiology of glaucoma is associated with ischemia and the development of neurodegenerative processes. A giant range of research have proven that glaucoma has a shut relationship with systemic cardiovascular pathology on the one hand [1], on the other, some researchers indicate the presence of shut connections of primary glaucoma with such neurodegenerative ailments as Alzheimer's disorder and Parkinson's disorder [2,3]. Numerous researchers agree with that the foremost mechanism underlying the development of glaucoma, even when the level of IOP is stabilized, is apoptosis. The dying of retinal ganglion cells in glaucoma develops towards the historical past of activation of apoptosis. It is believed that by apoptosis, 5,000 ganglion cells per year die in the eye, with glaucoma this range may be doubled [4,5]. Apoptosis is characterized via the obligatory destruction of neuronal membranes, main to a disruption of cell functions.

One of the principal factors of the membranes is glycoproteins and gangliosides. These compounds in their shape include a carbohydrate issue and take part in the techniques of transmembrane transport, intercellular contact, signal transmission approaches are related with them (receptor and non-receptor tyrosine kinases, mobile antigens, adhesion molecules), participate in synaptic transmission, receptor reactions, formation and storage of memory. The essential function in these procedures is attributed to the presence in their shape of neuraminic acids.

Neuraminic (sialic) acids are polyfunctional mixes with solid corrosive properties. Generally speaking, they are not found in the free shape, they are incorporated into the sythesis of different starch containing substances, for example, glycoproteins, glycolipids (gangliosides), oligosaccharides. Involving the end position in the particles of these substances, neuraminic acids significantly affect their physicochemical properties and organic action.

A few creators show that the substance of N-acetylneuraminic corrosive can be utilized as a biochemical marker of apoptotic and necrotic harm [6,7]. In view of the prior, we directed a relative investigation of the aggregate and lipid-bound neuraminic corrosive substance in erythrocyte layers in glaucoma. Complex assessment of the substance and arrangement of aggregate and lipid-bound neuraminic corrosive in glaucoma in erythrocyte films can be helpful for comprehension the pathogenetic highlights of this infection.

Material and Methods

Analyses were done in blood erythrocyte layers of patients with glaucoma who are being treated in the eye center. All patients experienced a standard ophthalmologic examination.

Films of red platelets were confined from the erythrocyte mass, which was washed with an isotonic NaCl arrangement. The washed erythrocyte mass was suspended in a cushion arrangement (0.01 M NaHCO3, 0.003 M NaCl, EDTA) trailed by centrifugation at 12000 g on a K-24 axis [8]. To decide the aggregate neuraminic corrosive, the without protein filtrate of erythrocyte films experienced hydrolysis, bringing about the arrival of neuraminic acids from the sialoglycoproteins, which, when responded with acidic and sulfuric acids, lead to the arrangement of a shaded compound at lifted temperatures [9]. Lipid-bound neuraminic corrosive is principally found in gangliosides, so we decided its substance in gangliosides. To decide lipid-bound sialic corrosive in erythrocyte films, extraction was completed with a methanol-chloroform blend (1: 2). The supernatant with the lipids removed in that was gathered and the pellet was reextracted multiple times. To the joined lipid extricate, cool refined water (in the measure of 1/5 of the aggregate concentrate) was included, the blend was altogether blended and centrifuged (at 3000 rpm for 10 minutes). The lower chloroform layer comprising of phospholipids and impartial glycosphingolipids was isolated from the upper fluid methanol layer containing the glycolipids by centrifugation. The upper watery methanol layer was dialyzed against refined water for 2 days at 4°C, with a continuous difference in water in the dialyzer. The dialysis arrangement got after dialysis was dissipated on a turning evaporator and connected to a segment with DEAESephadex A-25. Gangliosides were controlled by thin-layer chromatography in a dissolvable framework chloroform: methanol: 2.5 M smelling salts (60: 35: 8). The substance of GB was made a decision by the measure of N-acetylneuraminic

corrosive, which was controlled by the resorcinol technique [10]. The information acquired were prepared factually utilizing Student's test.

Results and Discussion

As the results of the review shown (Figure 1), an expansion in the aggregate neuraminic corrosive substance is seen in films of blood erythrocytes in patients with glaucoma. At present, it has been built up that the erythrocyte layer in its structure and capacities is indistinguishable to the films of the cells of the body in general and, in this association, can fill in as an advantageous and open question for the investigation of unsettling influences in layers. Typically, generally speaking, in free frame, neuraminic acids are found in a little sum. The aggregate dimension of neuraminic acids unreservedly flowing in the circulatory system is the entirety of the discharged neuraminic acids because of the breakdown of glycoproteins and glycolipids. Because of hydrolytic decay, its substance rises, which is related with the procedure of sialing and desialization of proteins and glycolipids in the body? Cleavage of N-acetylneuraminic corrosive from glycolipids and glycoproteins happens by the compound neuraminidase (sialidase). Taking out neuraminic corrosive from the parts of cell films, this catalyst changes the structure and generally speaking charge of the layer. Concentrates as of late demonstrate that neuraminidase, related with the plasma film of the cell, has an imperative physiological criticalness for the cells of the human sensory system. Neuraminidase changing the subjective and quantitative arrangement of glycoproteins and gangliosides in cell layers adjusts the action of protein kinases, and, therefore, the response of the phone to specific flags that lead to the phosphorylation of various proteins, which animates or lessens the translation of specific qualities. A few creators propose that neuraminidase limited in the plasma film can straightforwardly initiate the receptor for tyrosine kinase A, severing off the sialic corrosive buildup [11,12].

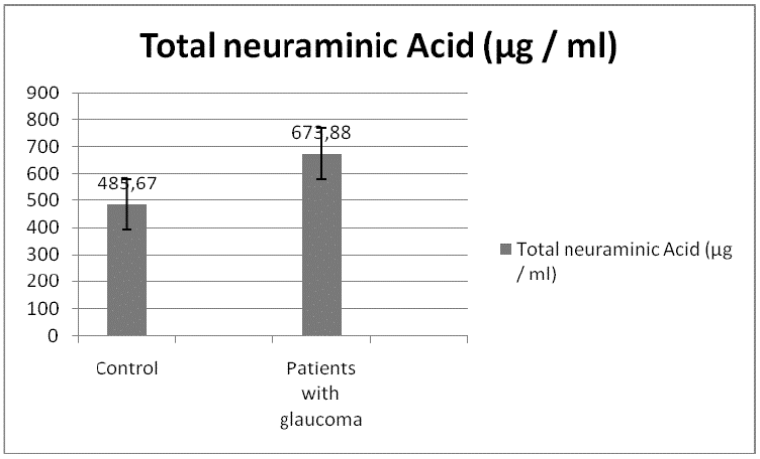

Figure 1 The content of total neuraminic acid in erythrocyte membranes in practically healthy people and patients with glaucoma. (N = 25), P<0.01.

Continuing from the way that the gangliosides contain lipidbound neuraminic corrosive, we explored the substance of gangliosides in erythrocyte films. As the aftereffects of the examination, appeared in Figure 2. Gangliosides of erythrocyte films are spoken to by 4 parts of gangliosides: monosyalans, gangliosides, trisialgs, and tetrasial gangliosides, varying in the

substance of neuraminic acids in them. As the aftereffects of the investigation appeared, glaucoma reflected in Figure 2 is portrayed by a decline in the substance of all divisions of gangliosides. It is trusted that mono-and disial G3 have a high conversion standard, while tri- and tetrasialgs are portrayed by a lower swapping scale. All the basic changes in GB because of desialization, principally influence the charge and influence the electrogenic idea of the layers, which influences the conduction of nerve driving forces in neurons and the control of this procedure. In patients with glaucoma, abatement in the substance of all portions of gangliosides in erythrocyte layers is watched.

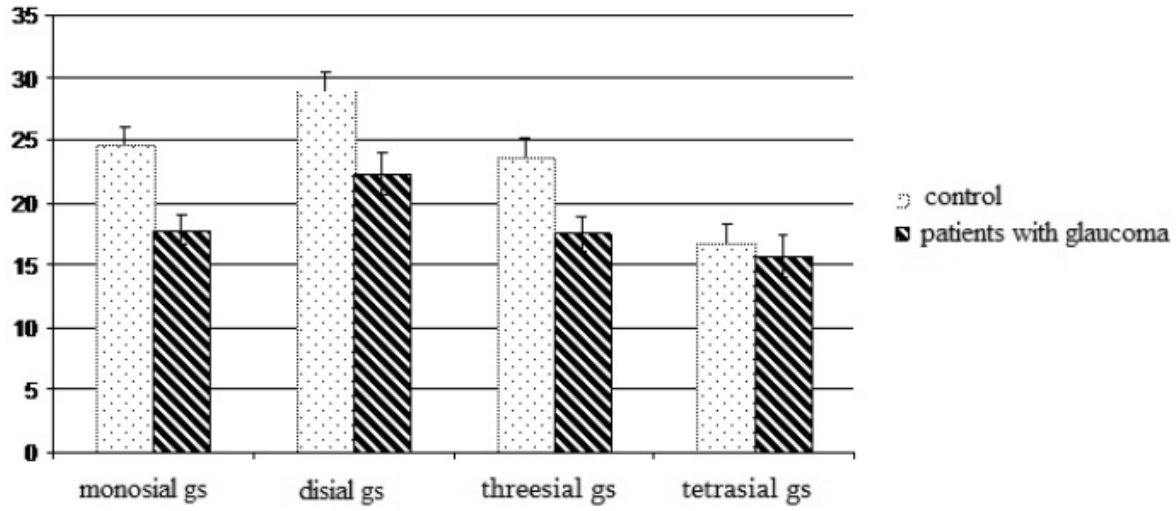

Figure 2 The fractional composition of gangliosides in erythrocyte membranes in practically Healthy people and patients with glaucoma (% difference from control) (n=20), p <0.01, **p<0.05 (n=20).

As noticed, the aggregate centralization of neuraminic acids in the films of erythrocytes in glaucoma is expanded. As officially noticed, an expansion in the dimension of neuraminic acids in natural liquids mirrors the procedures of the demolition of film structures of the mind tissue. A few scientists trust that the substance of N-acetylneuraminic corrosive can be utilized as a biochemical marker of apoptosis and necrotic harm [6].

An expansion in the substance of N-acetylneuraminic corrosive in the cisospinal liquid of patients with strokes has appeared, the high substance of N-acetylneuraminic corrosive on the main day corresponds with the seriousness of the patients' condition and the immeasurability of the ischemic zone of the mind [7] and mirrors the power of the procedures of decimation of neuronal films going with necrotic harm. Furthermore, it is realized that in layers gangliosides can be cut to ceramide and sphingosine, which actuate apoptosis and hinder cell expansion. We can't help suspecting that an expansion in the substance of free sialic acids in the blood plasma and erythrocyte films might be the explanation behind the cleavage of neuraminic acids from glycoproteins and gangliosides.It is worth mentioning the fact that the protective effect of GM1 on neurons of the retina in optic nerve trimming is studied. These authors found that the introduction of GM1 protects retinal neurons from death and leads to an increase in the level of pERK1/2 and pCREB (transcription factor). Thus, the results of our study revealed a role for N-acetylneuraminic acids in the pathogenesis of glaucoma development.

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