

A Study on Changes in Plasma Cholinesterase in Patients with Acute Primary Angle Closure

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Research Article

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Abstract

Purpose

To analyze the differences in plasma cholinesterase (pChE) among patients with acute primary angle closure (APAC), patients with chronic primary angle closure glaucoma (CPACG) and normal people scheduled for cataract surgery and to analyze the relationship between intraocular pressure (IOP) and pChE in order to explore the significance of pChE in the pathogenesis of glaucoma.

Methods

Retrospective case series. Ninety-four patients with APAC, 72 patients with CPACG and 95 normal controls were enrolled in this study. All patients excluding those with diseases that may affect pChE underwent routine blood biochemical examination. Pearson correlation analysis was used to further analyze the correlation of IOP with pChE.

Results

There was no significant difference in age or sex among the three groups. The difference in IOP among the APAC (43.8 ± 12.2 mmHg), CPACG (25.6 ± 7.4 mmHg) and normal groups (13.6 ± 1.8 mmHg) was significant ($P = 0.000$). There was a significant difference in pChE between patients with APAC (7450.89 ± 1748.49 IU/L) and normal subjects (7994.68 ± 1321.90 IU/L) ($P = 0.000$) and between patients with APAC and those with CPACG (7969.44 ± 1572.14 IU/L) ($P = 0.000$). There was no significant difference in pChE between CPACG patients and normal subjects ($P = 0.932$). There was a moderate negative correlation between IOP and pChE in APAC patients ($r = -0.410$, $P = 0.000$), while there was no significant correlation in CPACG patients ($P = 0.228$) or normal subjects ($P = 0.341$).

Conclusion

APAC patients with higher IOP had lower pChE, which may have been related to IOP-induced neuroinflammation. It may provide a new strategy for optic nerve protection in glaucoma patients.

Background

Glaucoma is a major disease that leads to blindness. The incidence of glaucoma is increasing, and the disease has become a heavy health burden worldwide[1]. Primary angle closure glaucoma (PACG) is highly prevalent in Asia, especially in East Asia[2]. Acute attack of acute primary angle closure (APAC) is typically an ophthalmic emergency that can lead to extreme vision loss.

A large number of studies have shown that neuroinflammation plays a vital role in the pathogenesis of glaucoma, but its exact role is by no means clear. In human and animal models, numerous molecular pathways have been identified that are critical regulators of neuroinflammation, which may be associated with the etiology of glaucoma. The Tumor Necrosis Factor α pathway, complement cascade, and Toll-like receptors have received attention from researchers. In addition, interferons, interleukins, and various other pro- and anti-inflammatory cytokines may be related to glaucoma, at least in animal models of glaucoma with retinal ganglion cell (RGC) injury[3]. Investigations into the locations supposed to be most commonly affected in glaucoma have revealed the occurrence of essential events in the optic nerve head (ONH), functional abnormalities in RGCs, and potential neuroinflammation that may affect RGC somas, dendrites and synapses; the involvement of the peripheral immune system in the retina has also been discussed[3, 4].

Studies have attempted to link cholinesterase (ChE) enzymes to glaucoma in recent decades. In these studies, researchers have applied various methods to measure ChE activity and have obtained diverse results[5]. One previous investigation revealed lower levels of red blood cell acetylcholinesterase (AChE) in glaucoma patients than in normal individuals[6]. However, some subjects had organic disease, and the participants used of a variety of drugs that affected the plasma ChE content; in addition, differences among individuals in the results were obvious. The limited number of patients also had somewhat of an impact on the results.

AChE is a specific esterase that is concentrated in the brain, nerves and red blood cells and hydrolyzes predominantly choline esters. Another enzyme called butyrylcholinesterase (BChE) is a nonspecific choline esterase (also known as a pseudocholinesterase) that mainly exists in the plasma, pancreas, liver and central nervous system and can hydrolyze esters, including choline esters[7, 8]. AChE plays a key role in catalyzing hydrolysis of acetylcholine (ACh) in cholinergic synapses of the cerebrum and autonomic nervous system[9]. Although BChE also has some of these functions, its main mechanism in the brain remains unclear. The cholinergic anti-inflammatory pathway mediated by the neurotransmitter ACh exerts a direct inhibitory effect on proinflammatory cytokine production[10]. It can also enhance destruction of hydrolysis and decrease the concentration of ACh, thus triggering and maintaining a systemic inflammatory response[11–13].

To date, there have been no definite conclusions about the relationship between glaucoma and pChE. Therefore, the aim of our study was to investigate the association of pChE activity with inflammation in glaucoma patients and normal controls. In addition, the courses of inflammation caused by acute and chronic intraocular pressure (IOP) elevation are different, which may lead to various effects in different types of glaucoma. We thus also analyzed the differences in pChE between different subtypes of glaucoma: acute glaucoma and chronic glaucoma.

Methods

This was a retrospective case series. All participants in this study received a detailed explanation of the study and gave written informed consent in accordance with the principles embodied in the Declaration of Helsinki. This study was approved by the Ethical Review Committee of Peking University First Hospital. The participants in this study were recruited retrospectively and consecutively between January 2018 and December 2019. Ninety-four patients with APAC, 72 patients with chronic primary angle closure glaucoma (CPACG) and 95 normal controls were enrolled in this study.

Each participant underwent a comprehensive ophthalmic examination that included best-corrected visual acuity (BCVA) measurement, slit-lamp examination, stereoscopic examination of the fundus, Goldmann applanation tonometry, and B-scanning.

All patients (excluding those with diabetes, Alzheimer's disease, liver disease or other diseases that may affect pChE) underwent routine blood biochemical examination. Venous blood was drawn within one week after an acute attack in patients with APAC. There was no history of acute attack in the previous three months in both patients with APAC and patients with CPACG.

APAC

Patients with typical cases were chosen as participants in the Department of Ophthalmology, Peking University First Hospital. An eye with recent acute attack was selected for each patient. All APAC candidates were required to meet the following inclusion criteria[14]: (1) exhibition at least two of the following manifestations: nausea and/or vomiting, ocular or peri-ocular pain, and a history of intermittent blurred vision with halos; (2) IOP as measured by Goldmann applanation tonometer higher than 21 mmHg; (3) emergence of at least three of the following manifestations: conjunctival hyperemia, corneal epithelial hyperemia, pupil medium size, and shallow anterior chamber; and (4) completion of ultrasound biomicroscope (UBM) examination to determine if there was a narrow angle caused by pupillary block[15].

CPACG

The diagnosis of CPACG mainly depended on the angle (extent of iridotrabecular meshwork closure $\geq 180^\circ$), and the International Society of Geographic and Experimental Ophthalmology standards were also followed[16]. The CPACG group included patients with IOP higher than 21 mmHg and intermittent or chronic angle closure glaucoma with optic disc damage or retinal nerve fiber layer defects and corresponding visual field damage without other fundus diseases. If both eyes met the inclusion criteria, the eye that was more severe was included in the group.

Control Group

The control group was composed of cataract patients without glaucoma optic neuropathy and no history of IOP higher than 21 mmHg. The cataract patients were scheduled to undergo phacoemulsification and intraocular lens implantation surgery. One eye was randomly enrolled.

Participants in the three groups with any of the following conditions were excluded: secondary acute angle closure caused by uveitis, lens subluxation, trauma, iris neovascularization, tumors, and lens dilation caused by cataract. Patients who had previously been diagnosed with other eye diseases (age-related macular degeneration [AMD], diabetic retinopathy, retinal vein occlusion, or retinal artery occlusion) or had a history of eye surgery. And those patients who were diagnosed with immunosuppressive diseases, autoimmune diseases, or systemic inflammation were also excluded.

Statistical Analysis

Continuous data are presented as means \pm standard deviations, and categorical data are presented as numbers. One-way ANOVA was used to analyze continuous variables. Chi-square test was used to determine the significance of differences in categorical variables. Pearson correlation analysis was used to further analyze the correlation between IOP and pChE. Values of $P < 0.05$ were considered to indicate statistical significance. The Statistical Package for the Social Sciences for Windows (v.12.0.0, SPSS Inc., Chicago, IL) was used for all statistical analyses.

Results

1. General characteristics and biochemical indexes of the APAC, CPACG and normal subjects (Table 1)

Table 1
General characteristics and biochemical indexes of the APAC, CPACG and normal subjects

	APAC (I)	CPACG (II)	Normal Subjects (III)	<i>P</i> Value	<i>P</i> _{I-II} Value	<i>P</i> _{I-III} Value	<i>P</i> _{II-III} Value
n (males/females)	94 (42/52)	72 (32/40)	95 (45/50)	0.909			
Age (years)	68.51 \pm 5.42	69.14 \pm 5.91	71.62 \pm 5.84	0.720			
IOP (mmHg)	43.8 \pm 12.2	25.6 \pm 7.4	13.6 \pm 1.8	< 0.01	< 0.01	< 0.01	< 0.01
pChE (U/L)	7450.89 \pm 1748.49	7969.44 \pm 1572.14	7994.68 \pm 1321.90	< 0.01	< 0.01	< 0.01	0.932
ALT (U/L)	17.29 \pm 14.36	19.22 \pm 14.09	16.64 \pm 6.53	0.481			
AST (U/L)	17.47 \pm 5.52	19.19 \pm 12.00	19.30 \pm 4.17	0.378			
Reference ranges: pChE 4300–13200 U/L, ALT 7–40 U/L, AST 13–35 U/L							

There was no significant difference in age or sex ratio among the three groups ($P= 0.720$ and 0.909 , respectively). There was a significant difference in IOP among the APAC group, the CPACG group and the normal group ($P < 0.001$; APAC group [43.8 ± 12.2 mmHg] > CPACG group [25.6 ± 7.4 mmHg] > control group [13.6 ± 1.8 mmHg]).

With regard to the biochemical indexes, there was a significant difference in pChE among the APAC group, the CPACG group and the normal control group ($P < 0.001$; APAC group [7450.89 ± 1748.49 U/L] < CPACG group [7969.44 ± 1572.14 U/L], APAC group [7450.89 ± 1748.49 U/L] < normal group [7994.68 ± 1321.90]). There was a significant difference between the APAC group and the normal group ($P < 0.01$) and between the APAC group and the CPACG group ($P < 0.001$), but there was no significant difference between the CPACG and normal control group ($P = 0.932$). There were also no significant differences in ALT and AST among the three groups ($P = 0.481$ and 0.378 , respectively). These results were shown in Table 1. The average number of antiglaucoma drugs in the CPACG group was 2.8.

2. Correlation analysis of pChE and IOP in APAC and CPACG patients

2.1 Correlation analysis of pChE and IOP in APAC patients (Fig. 1)

2.2 Correlation analysis of pChE and IOP in CPACG patients (Fig. 2)

Discussion

Inflammation is a key factor in multiple diseases. It has been demonstrated that inflammation plays significant roles in the pathological processes of AMD, diabetic retinopathy and other ophthalmic diseases[17, 18]. Howel et al. found that in the early stage of glaucoma, microglial cells, glial cells, and immune cells from other blood sources can trigger a neuroinflammatory response. Other relevant studies have also suggested that pathological changes occur in synapses, dendrites and somatic cells in the early stage of glaucoma[19]. The investigators in the previous studies have suggested that these changes probably result from a retinal inflammatory response in the early stage of glaucoma. The role of inflammation in the neurodegeneration caused by glaucoma is a popular research topic, but unclear yet.

In this study, we found that the pChE level in the APAC group (7450.89 ± 1748.49 IU/L) was significantly lower than that in the normal group (7994.68 ± 1321.90 IU/L). In addition, the pChE level in the APAC group was significantly lower than that in the CPACG group (7969.44 ± 1572.14 IU/L), whose IOP was much lower than that of the APAC group. There was a significant difference in pChE level between the APAC and CPACG groups ($P < 0.001$). There was no significant difference in sex among the three groups ($P = 0.932$). The average ages of the patients in the APAC, CPACG groups and normal subjects were 68.51 ± 5.42 years, 69.14 ± 5.91 years, 71.62 ± 5.84 years, respectively, with no significant difference among the three groups ($P = 0.720$). The previous results showed that BChE activity was negatively correlated with age, while it was positively correlated with triglyceride, cholesterol, and albumin concentrations[20, 21]. Therefore, in addition to other systemic factors that may cause changes in pChE, such as age, sex, and systemic diseases, we speculate that the acute inflammation of due to the rapid rise of IOP may be one of the main reasons for the significant difference in pChE. Narendra et al. also found that AChE level was

lower in glaucoma subjects than in normal individuals[5]. However, the number of 15 glaucoma patients selected in the previous study was limited, and individual differences were obvious. In addition, a study observing changes in AChE and pChE levels in 19 glaucoma patients with chronic glaucoma (without clear delineation of the type of glaucoma) within hours to two weeks revealed that the AChE level in the glaucoma group was lower than that in the normal control group[6].

In this study, we found that pChE level decreased when IOP increased in acute angle closure patients, consistent with results of Lampón et al. Lampón found a significant negative correlation between BChE and high-sensitivity C-reactive protein (hs-CRP)[22]. When the hs-CRP concentration was higher than 3 mg/L, there was a significant negative correlation between hs-CRP and BChE ($P < 0.001$). When patients had systemic acute inflammation (hs-CRP > 10 mg/L), the relationship between the two variables also supported this conclusion. C-reactive protein (CRP) is a ring-shaped pentameric protein synthesized by the liver. During inflammatory reactions, CRP is secreted into the blood after secretion of interleukin-6 by T-cells and macrophages. Various previous studies have shown that CRP is a biomarker of systemic inflammation[23]. The findings indicate that a higher CRP level is associated with greater systemic inflammatory activity and lower BChE activity. In addition, studies have shown that the activity of BChE and other esterases is decreased in frail elderly individuals and that the decreased activity is associated with increased levels of inflammatory markers, suggesting that inflammation may mediate the effects of frailty on metabolism[24]. All these results are similar to the acute inflammation caused by the acute increased IOP associated with glaucoma in this study.

Our results showed that in APAC patients, pChE was negatively correlated with IOP ($r = -0.410$, $P < 0.001$). However, there was no correlation in CPACG patients ($P = 0.228$). Some researchers have linked chronic low-grade inflammation (defined by CRP levels[25, 26] and BChE activity[27–29]) to metabolic syndrome, insulin resistance, obesity, and cardiovascular risk. I-te Lee et al suggested that the level of systemic inflammation can be reflected by the level of serum CRP and is related to high IOP in patients with and without metabolic syndrome[30]. Although the pathological mechanism of the association of systemic inflammation and IOP is not clear, the results of their research showed a positive correlation between IOP and CRP levels. Higher systemic inflammatory activity is associated with lower BChE activity. Therefore, in this study, pChE decreased with increasing IOP. The negative correlation between BChE activity and inflammatory markers may be causal. Previous studies have revealed relationships between lower BChE activity and higher risks of mortality by acute stroke[31], cardiovascular disease[32] and long-term dialysis[33], which may be caused by the negative correlation between BChE activity and inflammation grade. The significance of pChE and inflammation in glaucoma needs to be further explored.

There are several limitations of our present study. First, as it was a retrospective study, it focused on the changes in ChE levels in plasma. In the future, pChE and other inflammatory indexes could be further analyzed in the aqueous humor, and CRP could be detected in plasma. This will help to identify patients in different stages of glaucoma. In addition, investigation of the relationships between different levels of CRP and indexes such as pChE should be performed. Further studies to investigate the correlation between systemic inflammation and glaucoma are warranted.

Conclusions

In conclusion, we found that pChE level in APAC patients was significantly lower than that in CPACG patients and normal subjects, which may have been related to changes in the systemic inflammatory response induced by acute IOP elevation in the context of glaucoma. Such research on pChE in glaucoma patients may provide a basis for anti-inflammatory treatment of glaucoma and suggest a new strategy for future optic nerve protection in patients with glaucoma.

Abbreviations

ACh

acetylcholine; **BChE** = butyrylcholinesterase; **pChE** = plasma cholinesterase; **APAC** = acute primary angle closure; **CPACG** = chronic primary angle closure glaucoma; **IOP** = intraocular pressure; **PACG** = primary angle closure glaucoma; **ONH** = optical nerve head; **AChE** = red blood cell cholinesterases; **BCVA** = best-corrected visual acuity; **UBM** = ultrasound biomicroscope; **RGC** = retinal ganglion cell; **hsCRP** = high-sensitive C reactive protein; **AMD** = age-related macular degeneration

Declarations

Ethics approval and consent to participate

This study is a retrospective investigation. The research is based entirely on the clinical routine and is obtained from the patient's medical record system. Therefore, no informed consent is required, and it has been approved by the ethics committee of our hospital.

Consent for publication

All presentations of case reports included in this manuscript have consent for publication.

Availability of data and materials

The datasets used during the current study are available from the corresponding author upon reasonable request.

Competing interests

All authors declare that they have no competing interests.

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Authors' contributions

TT collected the data and drafted the manuscript. YC designed the study, performed the surgeries, and was involved in collecting the data and drafting the manuscript, and also critically revised the manuscript and has given final approval of the version to be published. ML participated in the design of the study and the collection of the data. YF and YZP participated in the design of the study. All authors read and approved the final manuscript.

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Figures

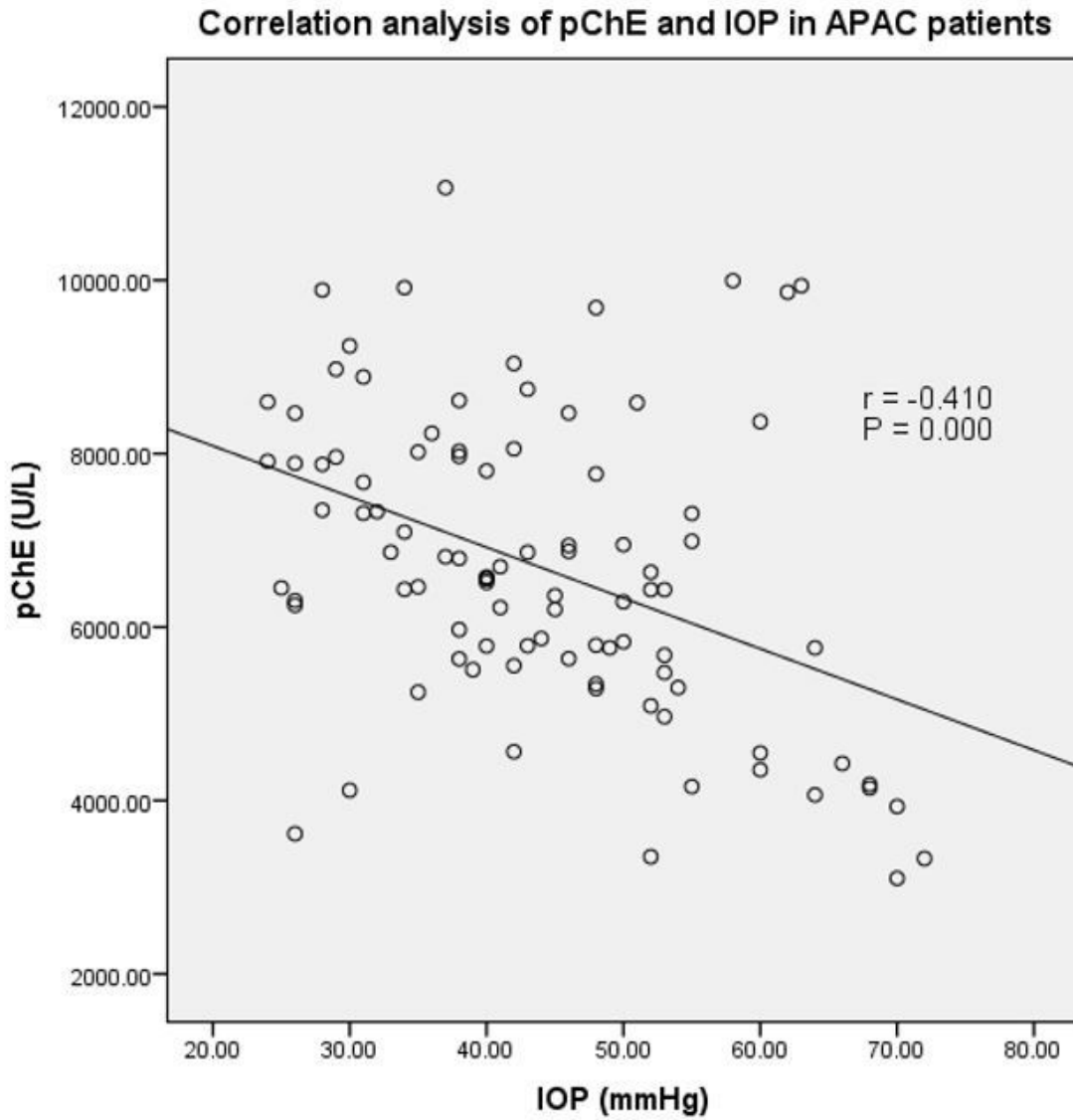


Figure 1

There was a moderate negative correlation between pChE and IOP in APAC patients ($r = -0.410$, $P < 0.001$).

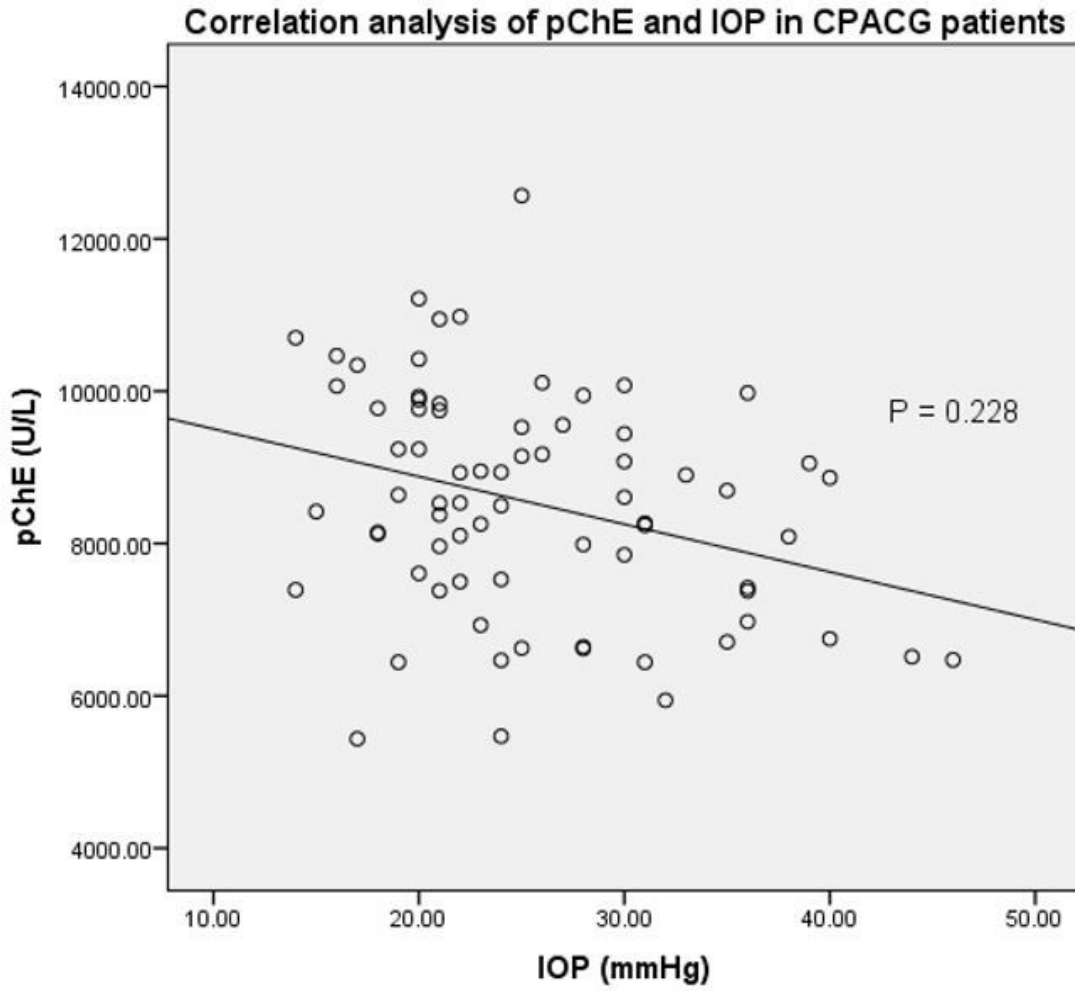


Figure 2

There was no correlation between pChE and IOP in CPACG patients ($P = 0.228$).