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RESEARCH ARTICLE

Serum Parameters of Primary Open Angle Glaucoma Patients in a Tertiary Hospital - A Pilot Study

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Abstract

Objective: To evaluate serum parameters in treatment-naive and treated Primary Open Angle Glaucoma (POAG), and no glaucoma eye patients.

Methods: Fasting blood samples of the participants were analyzed for electrolytes, proteins, urea, Blood Urea Nitrogen (BUN), creatinine, lipids, and liver enzymes.

Results: Electrolytes, including sodium, potassium, chloride, and total carbon dioxide showed no significant differences ($p > 0.05$). Total protein was slightly higher in treated POAG patients (7.8 ± 0.4 g/dL; $p = 0.048$), with elevated gamma-globulin (1.7 ± 0.2 g/dL). Urea and BUN were significantly increased in treated POAG ($p = 0.011$ and 0.014), while creatinine was no significantly elevated. Triglycerides were significantly higher in treated POAG (1.5 ± 0.7 mmol/L, $p < 0.001$); cholesterol, HDL, and LDL showed no differences. Liver enzymes were similar, though ALT was highest in treated POAG but remained within normal range.

Conclusion: These results indicate minor elevations in serum proteins, urea, BUN, and triglycerides in treated POAG patients, potentially reflecting metabolic effects of treatment. Most other parameters were comparable across groups. Findings suggest serum urea, total protein, and globulin hold promise in translational study for POAG, but larger studies are needed to confirm clinical relevance and their specificity for POAG diagnosis.

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Introduction

Unmanaged glaucoma remains a cause of irreversible blindness [1]. Primary Open Angle Glaucoma (POAG) is a common type

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of glaucoma [2] with very difficult indices including relentlessness, progressiveness, painlessness and visual loss. These features have grave implications for individuals living with POAG, their families and the public as visual impairment or blindness is abruptly discovered when not much help to preserve vision can be rendered [3,4]. Notably, early detection and appropriate management remain a cornerstone in preventing glaucoma blindness. There are efforts towards changing this POAG blinding narrative and its burdensome implications [3]. Some serum parameters may have translational potential in effective management of POAG.

This pilot study is necessarily a continuation of work that investigated urine and blood parameters of Individuals with Glaucoma (IWG) and its translational potential for managing POAG [5-8]. The initial study compared urine [6], serum [7], and hematological [8] parameters among individuals with POAG with those without glaucoma. It is hypothesized that ant glaucoma medication might have caused dehydration among others in individuals using them leading to differential in body fluid values between the two groups.

Methods

This study took place at two sites including a tertiary hospital and a private laboratory. The hospital has many medical specialties including ophthalmology. The department of ophthalmology provides medical, surgical and optical care for eye patients. Between 600 and 900 patients are present with different eye conditions at the hospital every month. Some of the eye condition include cataract, glaucoma, refractive error, presbyopia and conjunctivitis. The department has a complement of 7 ophthalmologists, 14 trainee ophthalmologists, 24 nurses, 5 optometrists, and 4 opticians among other supporting personnel. The private medical laboratory has capacity to process all the serum parameters investigated in this study.

The participants in this study were recruited from the eye clinics. They were in two main groups: those with and without primary open angle glaucoma. The participants with glaucoma were either on antiglaucoma drugs or not (newly diagnosed - antiglaucoma drug naive). Each of the participants underwent routine clerkship, examinations, investigations and diagnosis in the eye clinic. The diagnosis of POAG was based on gonioscopic open anterior chamber angle, intraocular pressure of at least 20 mmHg, and Cup Disc (CD) ratio of $\geq$ 0.5 and with or without any of family history of glaucoma, constricted visual field and optical coherence retinal nerve fibre layer thinning. Each participant was assigned a number and requested to present at the designated private laboratory before 10am after an overnight fasting for drawing of 10 milliliters of venous blood by the phlebotomist. The laboratory was blinded from the grouping of the participants. Any participant who failed to present at the laboratory was followed up on the phone. Meanwhile, any participant who expressed reservations including distant living address, tight work schedule, uncooperative in presenting at the laboratory for submission of blood sample was excluded from the study. The serum parameters investigated were sodium, potassium, chloride, protein total, albumin, alpha-1-globulin, alpha-2-globulin, beta-1-globulin, beta-2-globulin, gamma-globulin, total Carbon Dioxide (tCO_2), urea, creatinine, Blood Urea Nitrogen (BUN), creatinine, triglycerides, total cholesterol and high-density lipoprotein (HDL).

This study was approved by the Institution Review Board. Further, information on this study was explained to each of the participants and written informed consent was obtained. The study was conducted following the tenets of declaration of Helsinki on research conduct in human participants.

3. The data was collated, entered, cleaned and analyzed including simple proportion,

percentage, mean and ANOVA statistics. The p value was taken at $p < 0.05$. **Results**

Sociodemographic characteristics

The fasting serum of 38 participants including newly diagnosed POAG (4), normal (21), and POAG on treatment (13) mean age 44.0SD14.2 (Range 19-74) years were investigated. There were 20 (52.6%) male and 18 (47.4%) female. Their educational status includes non-formal 2 (5.3%), Quranic 1 (2.6%), primary 2 (5.3%), secondary 6 (15.8%), and tertiary 27 (71.0%). Their occupation includes skilled 27 (71.0%), semi-Skilled 6 (15.8), and unskilled 5 (13.2%). Marital status includes single 9 (23.7%), married 26 (68.4%), divorce 1 (2.6%) and widow 2 (5.3%). Their religion was Christianity 29 (76.3%) and Islam 9 (23.7%). Their ethnicity includes Yoruba 9 (23.7%), Igbo 8 (21.1%), Hausa 2 (5.3%) and others 19 (50.0%). Five (13.2%) participants had relatives with a history of glaucoma and 33 (86.8%) had no family history of glaucoma. Thirty (78.9%) had no comorbidities, but 7 (18.4) had hypertension and 1 (2.6%) diabetes mellitus.

Serum electrolytes of the participants

The mean values for serum electrolytes for the three groups of participants are displayed in table 1. Sodium: Newly diagnosed POAG 139.0 ± 1.8 mmol/L, normal participants 132.1 ± 29.5 mmol/L and POAG on treatment 137.4 ± 2.9 mmol/L, [$F(2,35) = 0.31$, $p = 0.735$)]. Potassium: Newly diagnosed POAG 4.3 ± 0.4 mmol/L, normal participants 10.6 ± 28.3 mmol/L and POAG on treatment 19.0 ± 36.2 mmol/L, [$F(2,35) = 0.494$, $p = 0.615$]. Chloride, newly diagnosed POAG 106.0 ± 2.5 mmol/L, normal participants

Table 1: Analysis of serum electrolytes of the participants.

Serum Parameters (mmol/L) & ANOVA	Group	N	Mean	SD	SE	95% CI for Mean		Min	Max
						LB	UB		
Sodium $F(2,35) = 0.31$, $p = 0.735$	New POAG	4	139.0000	1.82574	0.91287	136.0948	141.9052	137.00	141.00
	Normal	21	132.0967	29.46534	6.42986	118.6842	145.5091	4.03	146.00
	POAG on Rx	13	137.3846	2.90225	0.80494	135.6308	139.1384	131.00	140.00
	Total	38	134.6324	21.92432	3.55659	127.4260	141.8387	4.03	146.00
Potassium $F(2,35) = 0.494$, $p = 0.615$	New POAG	4	4.2825	0.44079	0.22039	3.5811	4.9839	3.70	4.68
	Normal	21	10.6229	28.27666	6.17047	-2.2485	23.4942	3.78	134.00
	POAG on Rx	13	19.0254	36.16145	10.02938	-2.8268	40.8775	3.90	101.00
	Total	38	12.8300	29.67267	4.81354	3.0768	22.5832	3.70	134.00
Chloride $F(2,35) = 0.714$, $p = 0.497$	New POAG	4	106.0000	2.44949	1.22474	102.1023	109.8977	103.00	109.00
	Normal	21	104.7857	2.45770	0.53631	103.6670	105.9044	100.00	111.00
	POAG on Rx	13	105.4615	1.61325	0.44743	104.4867	106.4364	103.00	108.00
	Total	38	105.1447	2.18703	0.35478	104.4259	105.8636	100.00	111.00
TCO2 $F(2,35) = 0.237$, $p = 0.790$	New POAG	4	26.2500	4.34933	2.17466	19.3292	33.1708	22.00	32.00
	Normal	21	26.8095	2.89170	0.63102	25.4932	28.1258	22.00	35.00
	POAG on Rx	13	26.1538	2.15430	0.59750	24.8520	27.4557	23.00	31.00
	Total	38	26.5263	2.76793	0.44902	25.6165	27.4361	22.00	35.00

Table 2: Analysis of serum protein of the participants.

Serum Parameters (g/dL) & ANOVA	Group	N	Mean	SD	SE	95% CI for Mean		Min	Max
						LB	UB		
Protein, total F(2,35) = 3.308, p = 0.048	New POAG	4	7.3500	0.26458	0.13229	6.9290	7.7710	7.10	7.70
	Normal	21	7.3905	0.47001	0.10256	7.1765	7.6044	6.40	8.40
	POAG on Rx	13	7.7615	0.39272	0.10892	7.5242	7.9989	7.30	8.70
	Total	38	7.5132	0.45629	0.07402	7.3632	7.6631	6.40	8.70
Albumin F(2,35) = 0.881, p = 0.423	New POAG	4	4.1525	0.20123	0.10061	3.8323	4.4727	3.89	4.32
	Normal	21	4.1786	0.31650	0.06907	4.0345	4.3226	3.66	4.73
	POAG on Rx	13	4.3100	0.29510	0.08185	4.1317	4.4883	3.61	4.82
	Total	38	4.2208	0.29998	0.04866	4.1222	4.3194	3.61	4.82
Alpha-1-Globulin F(2,35) = 0.261, p = 0.771	New POAG	4	0.2925	0.02363	0.01181	0.2549	0.3301	0.26	0.31
	Normal	21	0.2938	0.04260	0.00930	0.2744	0.3132	0.24	0.40
	POAG on Rx	13	0.2823	0.05434	0.01507	0.2495	0.3151	0.22	0.39
	Total	38	0.2897	0.04487	0.00728	0.2750	0.3045	0.22	0.40
Alpha-2-Globulin F(2,35) = 0.180, p = 0.836	New POAG	4	0.6675	0.02872	0.01436	0.6218	0.7132	0.65	0.71
	Normal	21	0.6595	0.10486	0.02288	0.6118	0.7073	0.50	0.93
	POAG on Rx	13	0.6877	0.18254	0.05063	0.5774	0.7980	0.45	1.04
	Total	38	0.6700	0.13034	0.02114	0.6272	0.7128	0.45	1.04
Beta-1-Globulin F(2,35) = 1.114, p = 0.339	New POAG	4	0.3800	0.02944	0.01472	0.3332	0.4268	0.34	0.41
	Normal	21	0.4138	0.05563	0.01214	0.3885	0.4391	0.28	0.52
	POAG on Rx	13	0.4292	0.06714	0.01862	0.3887	0.4698	0.35	0.54
	Total	38	3.308	0.05839	0.00947	0.3963	0.4347	0.28	0.54
Beta-2-Globulin F(2,35) = 2.164, p = 0.130	New POAG	4	0.3025	0.06898	0.03449	0.1927	0.4123	0.23	0.39
	Normal	21	0.3843	0.08109	0.01770	0.3474	0.4212	0.27	0.53
	POAG on Rx	13	0.3900	0.07095	0.01968	0.3471	0.4329	0.29	0.50
	Total	38	0.3776	0.07913	0.01284	0.3516	0.4036	0.23	0.53
Gamma-Globulin F(2,35) = 1.673, p = 0.202	New POAG	4	1.5600	0.19253	0.09626	1.2536	1.8664	1.32	1.76
	Normal	21	1.4605	0.36456	0.07955	1.2945	1.6264	0.94	2.66
	POAG on Rx	13	1.6592	0.21792	0.06044	1.5275	1.7909	1.31	2.15
	Total	38	1.5389	0.31444	0.05101	1.4356	1.6423	0.94	2.66

104.8 ± 2.5 mmol/L and POAG on treatment 105.5 ± 2.5 mmol/L, [F(2,35) = 0.714, p = 0.497]. Total carbon dioxide, newly diagnosed POAG 26.3±4.5mmol/L, normal participants 26.8±2.9 mmol/L and POAG on treatment 26.2 ± 2.2 mmol/L, [F(2,35) = 0.237, p = 0.790].

Serum protein of the participants

The mean values for serum protein for the three groups of participants are displayed in table 2. Protein, total: Newly diagnosed POAG 7.4 ± 0.3 g/dL, normal participants 7.4 ± 0.5 g/dL and POAG on treatment 7.8 ± 0.4 g/dL, [F(2,35) = 3.308, p = 0.048]. Albumin: Newly diagnosed POAG 4.2 ± 0.2 g/dL, normal participants 4.2 ± 0.3 g/dL and POAG on treatment 4.3 ± 0.3 g/dL, F(2,35) = 0.881, p = 0.423]. Alpha-1-Globulin: newly diagnosed POAG 0.3 ± 0.0 g/dL, normal participants 0.3 ± 0.0 g/dL and POAG on treatment 0.3 ± 0.1 g/dL, [F(2,35) = 0.261, p = 0.771]. Alpha-2-Globulin: newly POAG 0.7 ± 0.0 g/dL, normal participants 0.7 ± 0.1 g/dL and POAG on treatment 0.7 ± 0.2 g/dL, [F(2,35) = 0.180, p = 0.836]. Beta-1-Globulin: newly diagnosed POAG 0.4±0.1 g/dL, normal participants 0.4 ± 0.1 g/dL and POAG on treatment 0.4 ± 0.1 g/dL, [F(2,35) = 1.114, p = 0.339]. Beta-2-Globulin: Newly diagnosed POAG 0.3 ± 0.1 g/dL, normal participants 0.4 ± 0.1 g/dL and POAG on treatment 0.4 ± 0.1 g/dL, [F(2,35) = 2.164, p =0.130]. Gamma-Globulin: newly diagnosed POAG 1.6 ± 0.2 g/dL, normal participants 1.5 ± 0.4 g/dL and POAG on treatment 1.7 ± 0.2 g/dL, [F(2,35) = 1.673, p = 0.202].

Serum urea, blood urea nitrogen and creatinine of the participants

The mean values for serum urea, blood urea nitrogen and creatinine for the three groups of

participants are displayed in table 3. Urea: Newly POAG 2.7 ± 0.9 mmol/L, normal participants 3.5 ± 1.0 mmol/L and POAG on treatment 4.4 ± 1.1 mmol/L, [$F(2,35) = 5.118$, $p = 0.011$]. Blood urea nitrogen: Newly diagnosed POAG 1.3 ± 0.4 mmol/L, normal participants 1.7 ± 0.5 mmol/L and POAG on treatment 2.0 ± 0.5 mmol/L, [$F(2,35) = 4.842$, $p = 0.014$]. Creatinine: newly diagnosed POAG 73.5 ± 13.6 µmol/L, normal participants 73.1 ± 15.9 µmol/L and POAG on treatment 85.7 ± 16.4 µmol/L, [$F(2,35) = 2.642$, $p = 0.085$].

Serum lipids of the participants

The mean values for serum lipids for the three groups of participants are displayed in table 4. Total Cholesterol: newly POAG 24.0 ± 39.4 mmol/L, normal participants 39.3 ± 51.7 mmol/L and POAG on treatment 9.5 ± 13.7 mmol/L, [$F(2,35) = 2.081$, $p = 0.140$]. Cholesterol HDL: Newly diagnosed POAG 57.0 ± 111.3 mmol/L, normal participants 63.0 ± 83.7 mmol/L and POAG on treatment 45.9 ± 73.8 mmol/L, [$F(2,35) = 0.168$, $p = 0.846$]. Cholesterol LDL: Newly

Table 3: Analysis of serum urea, blood urea nitrogen and creatinine of the participants.

Serum Parameters & ANOVA	Group	N	Mean	SD	SE	95% CI for Mean		Min	Max
						LB	UB		
Urea (mmol/L) $F(2,35) = 5.118$, $p = 0.011$	New POAG	4	2.6750	0.88835	0.44418	1.2614	4.0886	2.10	4.00
	Normal	21	3.5000	0.98995	0.21602	3.0494	3.9506	1.90	6.20
	POAG on Rx	13	4.3462	1.07364	0.29777	3.6974	4.9949	2.30	6.40
	Total	38	3.7026	1.11827	0.18141	3.3351	4.0702	1.90	6.40
BUN (mmol/L) $F(2,35) = 4.842$, $p = 0.014$	New POAG	4	1.2750	0.41932	0.20966	0.6078	1.9422	1.00	1.90
	Normal	21	1.6524	0.46435	0.10133	1.4410	1.8637	0.90	2.90
	POAG on Rx	13	2.0385	0.50421	0.13984	1.7338	2.3432	1.10	3.00
	Total	38	1.7447	0.52179	0.08465	1.5732	1.9162	0.90	3.00
Creatinine (µmol/L) $F(2,35) = 2.642$, $p = 0.085$	New POAG	4	73.5000	13.57694	6.78847	51.8961	95.1039	58.00	91.00
	Normal	21	73.1429	15.91944	3.47391	65.8964	80.3893	26.00	103.00
	POAG on Rx	13	85.6923	16.38793	4.54519	75.7892	95.5954	53.00	112.00
	Total	38	77.4737	16.58677	2.69073	72.0217	82.9256	26.00	112.00

Table 4: Analysis of serum lipids of the participants.

Serum Parameters (mmol/L) & ANOVA	Group	N	Mean	SD	SE	95% CI for Mean		Min	Max
						LB	UB		
Total Cholesterol $F(2,35) = 2.081$, $p = 0.140$	New POAG	4	23.9775	39.36753	19.68376	-38.6650	86.6200	2.82	83.00
	Normal	21	39.3119	51.68733	11.27910	15.7841	62.8397	2.74	147.00
	POAG on Rx	13	9.5354	13.70789	3.80189	1.2518	17.8190	3.02	55.00
	Total	38	27.5111	42.71556	6.92938	13.4708	41.5513	2.74	147.00
Cholesterol HDL $F(2,35) = 0.168$, $p = 0.846$	New POAG	4	57.0425	111.30528	55.65264	-120.0690	234.1540	1.04	224.00
	Normal	21	62.9457	83.66249	18.25665	24.8630	101.0284	0.78	253.00
	POAG on Rx	13	45.9146	73.75139	20.45496	1.3471	90.4821	1.16	252.00
	Total	38	56.4979	81.33288	13.19394	29.7644	83.2313	0.78	253.00
Cholesterol LDL $F(2,35) = 1.946$, $p = 0.158$	New POAG	4	2.8375	0.96824	0.48412	1.2968	4.3782	1.64	3.85
	Normal	21	14.7562	38.02950	8.29872	-2.5546	32.0670	1.60	141.00
	POAG on Rx	13	43.3631	62.56283	17.35181	5.5567	81.1694	1.51	156.00
	Total	38	23.2882	47.74300	7.74494	7.5954	38.9809	1.51	156.00
Triglyceride $F(2,35) = 9.598$, $p = 0.000$	New POAG	4	0.6350	0.29126	0.14563	0.1715	1.0985	0.30	0.93
	Normal	21	0.7852	0.37052	0.08085	0.6166	0.9539	0.40	2.17
	POAG on Rx	13	1.4785	0.65578	0.18188	1.0822	1.8747	0.54	2.67
	Total	38	1.0066	0.58441	0.09480	0.8145	1.1987	0.30	2.67

Table 5: Analysis of serum alkaline phosphatase, alanine transaminase, and aspartate transaminase of the participants.

Serum Parameters (U/L) & ANOVA	Group	N	Mean	SD	SE	95% CI for Mean		Min	Max
						LB	UB		
ALP F(2,35) = 0.353, p = 0.705	New POAG	4	64.2500	28.39454	14.19727	19.0679	109.4321	39.00	105.00
	Normal	21	75.2381	30.99339	6.76331	61.1301	89.3461	42.00	170.00
	POAG on Rx	13	69.8462	18.37048	5.09505	58.7450	80.9473	51.00	99.00
	Total	38	72.2368	26.60973	4.31667	63.4904	80.9832	39.00	170.00
ALT (SGPT) F(2,35) = 0.726, p = 0.491	New POAG	4	16.5750	3.47215	1.73608	11.0500	22.1000	12.20	19.70
	Normal	21	18.8619	10.82804	2.36287	13.9330	23.7908	4.00	45.80
	POAG on Rx	13	22.0615	7.42737	2.05998	17.5732	26.5499	11.60	35.90
	Total	38	19.7158	9.25523	1.50140	16.6737	22.7579	4.00	45.80
AST (SGOT) F(2,35) = 0.104, p = 0.901	New POAG	4	24.0750	2.47167	1.23584	20.1420	28.0080	22.50	27.70
	Normal	21	25.3476	6.98853	1.52502	22.1665	28.5288	16.80	40.80
	POAG on Rx	13	24.8385	2.07145	.57452	23.5867	26.0902	21.90	28.30
	Total	38	25.0395	5.33436	.86535	23.2861	26.7928	16.80	40.80

diagnosed POAG 2.8±1.0 mmol/L, normal participants 14.8 ± 38.0 mmol/L and POAG on treatment 43.4 ± 62.6 mmol/L, [F(2,35) = 1.946, p = 0.158]. Triglyceride: Newly POAG 0.6 ± 0.3 mmol/L, normal participants 0.8 ± 0.4 mmol/L and POAG on treatment 1.5 ± 0.7, [F(2,35) = 9.598, p = 0.000].

Serum alkaline phosphatase, alanine transaminase, and aspartate transaminase of the participants

The mean values for serum alkaline phosphatase, alanine transaminase, and aspartate transaminase in U/L for the three groups of participants are displayed in table 5. Alkaline phosphatase: newly POAG 64.3±28.4 U/L, normal participants 75.2 ± 31.0 U/L and POAG on treatment 69.8 ± .18.4 U/L, [F(2,35) = 0.353, p = 0.705]. Alanine transaminase: Newly diagnosed POAG 16.6 ± 2.5 Ul/L, normal participants 18.9 ± 10.8 U/L and POAG on treatment 22.1 ± 7.4 U/L, [F(2,35) = 0.726, p = 0.491]. Aspartate transaminase: newly diagnosed POAG 24.1±2.5 U/L, normal participants 25.4±7.0 U/L and POAG on treatment 24.8 ± 2.1 U/L, [F(2,35) = 0.104, p = 0.901].

Discussion

This was a pilot study on serum parameters of Primary Open Angle Glaucoma (POAG) patients in a tertiary hospital. The fasting serum parameters, including electrolytes (Na, K, Cl, HCO_3), proteins (Total, albumin, globulin), urea, Blood Urea Nitrogen (BUN), creatinine, lipids (Cholesterol, triglyceride), and liver enzymes (ALP, ALT, AST), were analysed for three distinct groups of participants (38): Newly diagnosed POAG patients (antiglaucoma treatment naive), normal patients (non-glaucoma, not on antiglaucoma treatment), and POAG patients on antiglaucoma treatment. The study attempted to find out whether anti-glaucoma medication interferes with the serum parameters' values. Should antiglaucoma medications interfere insignificantly with serum parameters and should this study affirmed previous report [7] of significant differences among selected serum parameters then such might be investigated further for potentials as POAG biomarkers.

Baseline Characteristics

The baseline characteristics of the study participants included age, sex, education, occupation, marital status, religion, ethnicity, family history of glaucoma, and comorbidities. The cohort consisted of 38 participants, with a mean age of 44.0 ± 14.2 years, and a nearly equal gender distribution. Most participants

had tertiary education (71%) and were skilled workers (71%). Hypertension was the most common comorbidity (18.4%). A small proportion (13.2%) had a family history of glaucoma, which is a known risk factor for POAG [1,9]. Ethnic diversity was noted, with Yoruba and Igbo being the predominant groups. These findings align with previous studies highlighting demographic variations in glaucoma prevalence [5]. However, the small sample size limits generalizability [10]. The cohort had a relatively young mean age (44.0 ± 14.2 years), implying the working age group should be targeted for glaucoma screening towards early detection and appropriate management. The high proportion of tertiary-educated participants (71%) may reflect selection bias, as educated individuals are more likely to seek eye care. However, the study site is a tertiary public hospital located within a national capital space which is populated by many educated individuals who might be gainfully employed and able to afford glaucoma treatment. The predominance of Yoruba and Igbo participants aligns with regional demographics, but Hausa (5.3%) were underrepresented. Genetic and lifestyle differences among ethnic groups may affect glaucoma susceptibility [11].

Electrolyte Parameters

There were no observed significant differences in sodium, potassium, chloride, or total carbon dioxide levels between newly diagnosed POAG, normal participants, and POAG on treatment ($p > 0.05$). This suggests that electrolyte imbalances may not play a major role in POAG pathogenesis or treatment response. However, some studies have linked altered potassium levels to Intraocular Pressure (IOP) fluctuations [12]. The wide standard deviations, particularly in potassium levels (Normal participants: 10.6 ± 28.3 mmol/L), indicate high variability. The extreme potassium value is physiologically implausible (normal range: 3.5-5.0 mmol/L). This suggests haemolysis during sample processing or lab error, warranting exclusion or retesting [13]. Chloride levels were consistent across groups, supporting prior findings that chloride channels may not play a major role in POAG [14]. Though nonsignificant intergroup differences, sodium was the highest in newly diagnosed POAG (139.0 mmol/L). Sodium-potassium pumps (Na^+/K^+-ATPase) in the ciliary epithelium regulate aqueous humor production. Dysregulation may contribute to IOP elevation [15]. Also of note, no significant differences in chloride and total CO_2 across groups, but chloride levels were marginally higher in POAG (106.0 *vs.* 104.8 mmol/L in normal). Physiologically, chloride channels (CFTR - Cystic Fibrosis Transmembrane Conductance Regulator) modulate trabecular meshwork outflow. Polymorphisms in CFTR are linked to glaucoma risk [16]. Moreover, different glaucoma drugs may uniquely affect electrolyte balance. For instance, oral Carbonic Anhydrase Inhibitors (CAIs) are more likely to cause metabolic acidosis, lowering serum bicarbonate [17].

Protein Parameters

This work observed total protein levels were significantly different among groups ($p = 0.048$), with POAG on treatment showing slightly higher levels (7.8 ± 0.4 g/dL). Albumin and globulin fractions did not differ significantly. The higher total protein in treated POAG (7.8 g/dL) could reflect chronic inflammation from long-term medication use, such as prostaglandin analogs which upregulate cytokines like IL-6 [18,19]. Elevated gamma-globulin in treated POAG (1.7 ± 0.2 g/dL) may suggest an immune or inflammatory response to therapy [20], further supported by the immunological implications of elevated gamma-globulins, as autoantibodies against retinal proteins are reported in POAG [21]. The clinical relevance of these minor variations requires further investigation. Elevated levels of gamma-globulin in treated POAG (1.7 g/dL) may indicate immune activation, as some glaucoma drugs (e.g., prostaglandin analogs) modulate

cytokines [22]. Alpha-2-macroglobulin, though not measured here, is implicated in glaucoma *via* TGF-β signalling, suggesting further studies should include this marker [23]. Meanwhile, measuring specific inflammatory markers such as C-Reactive Protein (CRP) can confirm systemic inflammation in treated POAG as well as IL-6/TNF-α, the Cytokines linked to trabecular meshwork degeneration [24].

Renal Function Parameters (Urea, BUN, Creatinine)

Significant differences were observed in urea (p = 0.011) and BUN (p = 0.014), with POAG on treatment showing higher levels than newly diagnosed and normal participants. Drugs such as oral CAIs (Acetazolamide) inhibit renal carbonic anhydrase, reducing bicarbonate reabsorption and increasing urea excretion [25]. Furthermore, hypertension, present in 18.4% of the cohort, independently elevates BUN [26]. Creatinine was also elevated in treated POAG (85.7 ± 16.4 µmol/L) but not significantly (p = 0.085). These findings suggest potential renal function alterations in glaucoma patients on long-term medication, possibly due to drug metabolism [27], as carbonic anhydrase inhibitors are known to affect renal parameters [28]. Topical CAIs rarely affect renal labs, but oral CAIs can elevate urea/BUN [29]. The higher mean creatinine in treated POAG (85.7 µmol/L) may reflect age-related decline in Glomerular Filtration Rate (GFR), not solely drug effects [30]. Medication records are needed to clarify this finding, and the creatinine trend toward higher levels in treated POAG warrants continued monitoring.

Lipid Profile

It was also observed that triglyceride levels were significantly higher in POAG on treatment (1.5 ± 0.7 mmol/L, $p < 0.001$), while total cholesterol, HDL, and LDL did not differ significantly. Elevated triglycerides may reflect metabolic changes due to glaucoma therapy or comorbid conditions [31]. Prostaglandin analogs (Latanoprost) may alter lipid metabolism through PPAR-γ modulation [32], though evidence is conflicting [33]. Some studies link dyslipidemias to glaucoma risk [34], and metabolic syndrome (Hypertension + dyslipidemia) is associated with POAG progression [35]. Future studies should correlate lipid profiles with specific glaucoma medications. Some studies suggest statins (A potential confounder) reduce IOP by improving trabecular outflow [36].

Liver Enzyme Parameters

No significant differences were found in ALP, ALT, or AST among groups ($p > 0.05$). This suggests that liver function is not markedly affected in POAG or its treatment. However, ALT levels were slightly higher in treated POAG (22.1 ± 7.4 U/L) - though within normal limits (< 40 U/L) - possibly indicating mild drug-induced liver stress [37]. This merits monitoring, as oral CAIs rarely cause hepatotoxicity, even in mild, long-term use [38]. Baseline liver tests are recommended before initiating oral Carbonic Anhydrase Inhibitors (CAIs) [38]. Larger studies with longitudinal monitoring are needed [39].

Study Limitations

It is important to acknowledge the limitations in this study for better understanding and interpretations of the findings. The study's small sample size of 38 participants restricts its statistical power and limits the ability to generalize the findings to a broader population [10]. The cross-sectional design only reveals associations between variables without establishing cause-and-effect relationships. The study did not adequately control for potential confounding variables such as diet, medication adherence, and existing comorbidities which may influence the study outcomes. Laboratory results showed variability, particularly the anomalous potassium value (10.6 mmol/L ±

28.3 mmol/L), far outside normal physiological ranges, suggesting measurement or procedural errors [13]. Furthermore, the analysis did not differentiate between glaucoma medication classes, making it difficult to attribute observed effects to any specific drug. Finally, the study did not report the duration of glaucoma treatment, a critical factor for assessing time-related effects of therapy. Moreover, Intraocular Pressure (IOP) was not part of the analysis, leaving an incomplete understanding of how the investigated variables relate to glaucoma severity or control.

Conclusion

The study did not reveal clear differences among the three groups that would support definitive conclusions about the evaluated serum parameters in POAG, particularly given the small sample size. Some minor variations were observed in serum proteins, urea, blood urea nitrogen (BUN), and triglycerides across treatment-naive POAG patients, normal controls, and treated POAG cases. Most electrolytes, liver enzymes, and lipid measures showed no significant differences. The slightly higher triglycerides and urea levels in treated POAG patients may suggest possible metabolic effects related to therapy, but this requires further investigation. Larger, longitudinal studies are necessary to validate these findings and clarify their clinical relevance. Despite its limitations, this study adds preliminary insight into systemic biochemical changes associated with glaucoma, although the clinical implications of the subtle metabolic differences observed remain uncertain.

Recommendations

Future (Fully funded) research should focus on larger cohorts with detailed medication histories and long-term follow-up (age-matched controls and longitudinal) to clarify the systemic effects of glaucoma and its treatment. The investigated biomarkers should be expanded to include alpha-2-macroglobulin, GGT, and eGFR. Research should prioritize multi-centre studies with IOP correlation and oxidative stress markers.

There should be standardized protocols to minimize variability, especially fasting blood samples, repeat assays, and medication logs. There should be medication-specific analyses to differentiate drug effects from disease pathology. There should be correlation analysis of biochemical parameters with IOP to assess if biochemical changes predict IOP fluctuations.

Clinicians should periodically (Annually) monitor renal and metabolic parameters (Lipids, blood sugar etc) in POAG patients on long-term therapy, especially those with comorbidities like hypertension and diabetes. The prostaglandin analogs should be preferred over Carbonic Anhydrase Inhibitors (CAIs) in patients with metabolic syndrome.

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Declaration of conflicting interest

None of the authors has any conflict of interest to disclose.

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Ethical Approval and Informed Consent Statements

This study was approved by the Institution

Review Board. Further, information on this study was explained to each of the participants and written informed consent was obtained. The study was conducted following the tenets of declaration of Helsinki on research conduct in human participants.

Data Availability Statement

The data is available with corresponding author and can be accessed on demand.

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