Clinical Utility of Lipid Profile in Antiphospholipid Syndrome

Hassan Abdelaty M Elsonbaty[1], Ali Ahmed Abou El-Maaty[2], Mohammed Abdel-Hassib[3] and Amr M Hawwash[4]

[1]Department of Rheumatology and Rehabilitation, [2]Department of Neurology, Faculty of Medicine, Helwan University Cairo, Egypt

[3]Department of Internal Medicine, Faculty of Medicine, Al-Azhar University, Egypt

[4]Department of Internal Medicine and Rheumatology, Faculty of Medicine, Ain Shams University, Egypt

Original Article

ABSTRACT

Background: Antiphospholipid syndrome is a chronic, multi-system autoimmune disorder characterized by thromboembolic events and/or obstetric complications. Exploring associated risk factors is essential in management.

Aim of the Work: To study the lipid profile in antiphospholipid syndrome, and the effect of used medications on the lipid profile.

Patients and methods: A cross-sectional study was conducted on 70 patients with antiphospholipid syndrome recruited from Rheumatology-Rehabilitation and Neurology outpatient clinics and inpatient wards in four university hospitals. They underwent thorough history taking with complete physical examination. Laboratory investigations included: antiphospholipid markers, complete blood count, erythrocyte sedimentation rate, complement 3, complement 4, and fasting lipid profile. Dedicated imaging modality for diagnosis of arterial or venous thrombotic events

Results: Neurological manifestations (specifically migraine, sinus thrombosis and TIAs) showed a significant association with higher cholesterol levels. Sinus thrombosis was associated with lower HDL, higher TG, higher TG/HDL ratio and higher Cholesterol/HDL ratio. Lipid profile showed no statistically significant relation with received medications (Hydroxychloroquine, systemic steroids, and Azathioprine).

Conclusion: Dyslipidemia are common in APS especially in patients with TIAs, sinus thrombosis and migraine. No significant relation between medications used in treatment of APS and lipid profile levels.

Key Words: Antiphospholipid syndrome, lipid profile, systemic lupus erythematosus.

Received: 26 November 2024, **Accepted:** 27 January 2025.

Corresponding Author: Amr M Hawwash, Lecturer of Internal Medicine and Rheumatology, Faculty of Medicine, Ain Shams University, Egypt, **Tel.:** 01123902218, **E-mail**: amrhawwash@yahoo.com

ISSN: 2735-3540, Vol. 76, No. 1, March 2025.

INTRODUCTION

Antiphospholipid syndrome (APS) is a chronic, multi-system autoimmune disorder characterized by thromboembolic events and/or obstetric complications that can be classified as a primary (without identified etiology) or secondary to other autoimmune diseases, usually systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), or Dermatomyositis[1-3].

The thrombotic event is a vascular form of the disease that characterized by venous, arterial or capillary thrombosis associated with persistently positive antiphospholipid antibodies (aPL), the antibodies are directed toward proteins that bind to cell membrane phospholipids which are responsible for the pathological mechanisms in APS[4], as they produce pro-coagulant and pro-inflammatory state[5].

Apart from the presence of aPL, additional prothrombotic mechanisms are required to trigger thrombosis, such as hypertension, smoking, obesity, diabetes, physical inactivity and dyslipidemia[6].

Many risk factors are proved while other factors such as hypertension and dyslipidemia have been associated with the risk of thrombosis in patients with APS. The Global Antiphospholipid Syndrome Score (GAPSS),

DOI: 10.21608/ASMJ.2025.339414.1342

which is a clinically validated tool to predict the risk of thrombosis in APS patients, includes both hypertension and dyslipidemia, along with the presence of aPL, as independent determinants of thrombosis risk in APS[7].

Dyslipidemia is a condition caused by dysregulation of the lipid metabolism and is associated with an increased risk of atherosclerosis and cardiovascular events[8, 9], such as coronary artery disease and stroke[10].

In patients with APS, dyslipidemia may further aggravate the underlying risk of thrombosis conferred by the presence of aPL[7]. Statin is the treatment of choice to control lipid levels and reduce cardiovascular morbidity. However, statin use may be challenging in APS patients with thrombosis since statins interact with vitamin K antagonists, the main anticoagulant drug used by these patients[11], increasing the risk of bleeding[12].

OBJECTIVES

This work aimed to study the lipid profile in antiphospholipid syndrome, and the effect of hydroxychloroquine, steroids and azathioprine on the lipid profile.

PATIENTS AND METHODS

A cross-sectional study was conducted at Rheumatology-Rehabilitation and Neurology outpatient clinics, and inpatient wards (Badr University Hospital, Helwan University, Ain Shams University Hospital and Alazhar University Hospital); from November 2022 to October 2023. The study enrolled 70 patients (13 males and 57 females) with APS diagnosed according to GAPSS score[7].

Patients had an age range from 30 to 70 years. Alcoholics, and patients who received lipid lowering drugs, estrogen, progesterone containing agents or thyroid medications were excluded from this study.

All patients underwent thorough history taking with complete general, rheumatological and neurological examination by three consultants of rheumatology and neurology who had at least 15 years' experience.

History and examination included age, sex, weight, height, history of hypertension, diabetes mellitus, type of APS either primary or secondary (SLE, RA, dermatomyositis or others), disease duration, history of abortion, DVT, stroke, TIAs, cerebral sinus thrombosis, migraine. Drug history included the current medication with focusing on doses (hydroxychloroquine, systemic steroid, azathioprine).

Laboratory investigation included: antiphospholipid markers, Complete blood count (CBC), erythrocyte sedimentation rate (ESR), complement 3 (C3), complement 4 (C4). Liver and renal functions were obtained.

Dedicated imaging modality for diagnosis of arterial or venous thrombotic events (e.g., arterial and venous duplex, brain MRI and MRV).

After fasting ten hours, lipid profile including: cholesterol (Chol), triglycerides (TG), high density lipoprotein (HDL), low density lipoprotein (LDL), were measured. Abnormalities in lipid profile were defined as cutoffs of cholesterol level > 200 mg/dl, LDL levels of >150 mg/dL, TG levels of >150 mg/ dL, and HDL levels of <40 mg/dL. The nature of the study was explained to all patients and their caregivers.

- **Ethics approval and consent to participate:**

We obtained agreement to achieve our study from Research Ethics Committee (REC) for Human Subject and animal Research at the Faculty of Medicine, Helwan University, Cairo, Egypt (Serial: 124:2022). All patients gave written informed consent.

Statistical analysis

Statistical analysis was conducted using SPSS 27th edition. Categorical variables were presented in number and percent. Quantitative variables were presented in mean, standard deviation, minimum and maximum. Comparison between groups was conducted using Mann Whitney U test after normality testing by Shapiro Wilk test. Correlation between quantitative variables was conducted using Spearman correlation test. Any *p value* below 0.05 was considered significant.

RESULTS

We enrolled 70 patients diagnosed with APS; they had a mean age of 41.2±7.7 years, and mean BMI 26.6±1.7 kg/m^2. Most of the included patients were females accounting for 81.4% compared to 18.6% males. Regarding chronic diseases, diabetes accounted for 28.6%, followed by

hypertension in 20%, and smoking in 10% of the included patients.

The largest proportion of patients were diagnosed with primary APS accounting for 57.1%, followed by secondary APS accounting for 42.9%, which was subdivided in to 24 (80%) patients with SLE, 4 (13.3%) patients with rheumatoid arthritis and two patients with dermatomyositis. The mean duration of disease was 10.3±5 months. The clinical picture and laboratory characteristics are shown in (Table 1) and (Figure 1).

Table 1: Demographic, clinical characteristics and laboratory results of the included patients.

		Mean ±SD	Min-Max
Age (years)		41.2±7.7	30-65
BMI (kg/m^2)		26.6±1.7	24-65
		N	%
Sex	Male	13	18.6%
	Female	57	81.4%
Diabetes	No	50	71.4%
	Yes	8	11.4%
	First diagnosed	12	17.1%
Hypertension	No	56	80.0%
	Yes	14	20.0%
APS type	Primary	40	57.1%
	Secondary	30	42.9%
Secondary APS (N=30)	SLE	24	80.0%
	RA	4	13.3%
	Dermatomyositis	2	6.7%
		Mean ±SD	Min-Max
Disease duration		10.3±5	1-25
		N	%
Abortion in female patients (N=57)	No	37	64.9%
	Yes	20	35.1%
DVT	No	53	75.7%
	Yes	17	24.3%
Stroke	No	54	77.1%
	Yes	16	22.9%
TIAs	No	59	84.3%
	Yes	11	15.7%
Sinus thrombosis	No	60	85.7%
	Yes	10	14.3%
Migraine	No	57	81.4%
	Yes	13	18.6%
Anemia	No	15	21.4%
	Yes	55	78.6%
Leucopenia	No	58	82.9%
	Yes	12	17.1%
Thrombocytopenia	No	60	85.7%
	Yes	10	14.3%

		Mean ±SD	Min-Max
Hemoglobin	gm/dL	10.6±1.2	8-12.5
Total leukocyte count	10/cc	6±1.9	3.2-9.8
Platelet count	10/cc	200±45.3	129-290
Erythrocyte sedimentation rate	First hour	74.7±12.5	50-100
Urea	mg/dL	45.6±11.9	18-70
Creatinine	mg/dL	1±0.2	0.7-1.2
C3	mg/dL	96.5±23.3	65-145
C4	mg/dL	16.6±4.4	10.5-30
Cholesterol	mg/dL	236.7±43	146-311
TG	mg/dL	253.3±99.2	90-461
HDL	mg/dL	41.1±5.6	29-55
LDL	mg/dL	145.1±33.3	64.4-188.8
TG/HDL		6.5±3.2	1.6-13.9
Cholesterol/ HDL	6±1.7	3.1-10.1	
		N	%
Hydroxychloroquine	No	26	37.1%
	Yes	44	62.9%
Dose of Hydroxychloroquine	No	26	37.1%
	200 mg/d	19	27.1%
	400 mg/d	25	35.7%
Systemic steroids	No	19	27.1%
	Yes	51	72.9%
Dose of systemic steroids	No	19	27.1%
	10 mg/d	42	60.0%
	More than 10 mg/d	9	12.9%
Azathioprine	No	37	52.9%
	Yes	33	47.1%

APS= antiphospholipid syndrome, BMI= Body mass index, DVT= deep vein thrombosis, TIAs= transient ischemic attacks, C3= complement 3, C4= complement 4, HDL= high density lipoprotein, LDL= low density lipoprotein, TG= triglycerides.

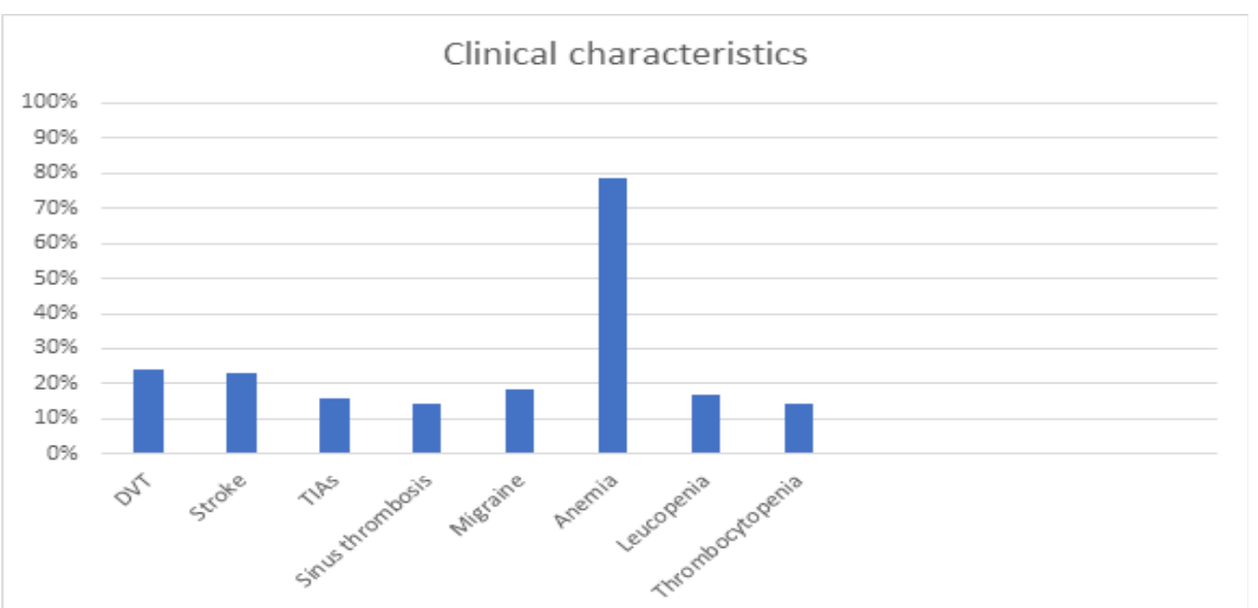

Fig. 1: Percentages of patients with positive clinical and laboratory characteristics

Most of patients received hydroxychloroquine (62.9%) with a dose of 200 mg in 19 (27.1%) patients, and 400 mg in 25 (35.7%) patients. Fifty one (72.9%) received systemic steroids, with dose 10 mg/day in 42 (60%) patients and more than 10 mg/day in 9 (12.9%) patients. Thirty three (47.1%) patients received azathioprine with a dose of 100-150 mg/day.

There was negative correlation between ESR, and HDL levels (*p value* 0.049), and positive correlation between TG, TG/HDL ratio and ESR level (*p value*s 0.019, 0.017 respectively). Otherwise, there was no statistically significant correlation between disease duration, C3, C4 levels and lipid profile. This is presented in (Table 2) and (Figure 2).

Table 2: Correlation between lipid profile, disease duration, ESR, C3 and C4.

		Chol	TG	HDL	LDL	TG/HDL	Chol/ HDL
Disease duration	Rho	0.055	0.092	-0.085	0.036	0.12	0.087
	P value	0.649	0.451	0.485	0.766	0.323	0.476
ESR	Rho	0.1	0.280	-0.236	-0.004	0.284	0.183
	P value	0.412	0.019	0.049	0.977	0.017	0.13
C3	Rho	0.053	-0.212	-0.06	0.192	-0.154	0.061
	P value	0.662	0.079	0.623	0.112	0.203	0.615
C4	Rho	-0.039	0.121	-0.08	-0.093	0.111	0.01
	P value	0.751	0.318	0.513	0.444	0.362	0.934

ESR= Erythrocyte sedimentation rate, C3=complement 3, C4= complement 4, Chol= cholesterol, TG= triglyceride, HDL= high density lipoprotein, LDL= low density lipoprotein.

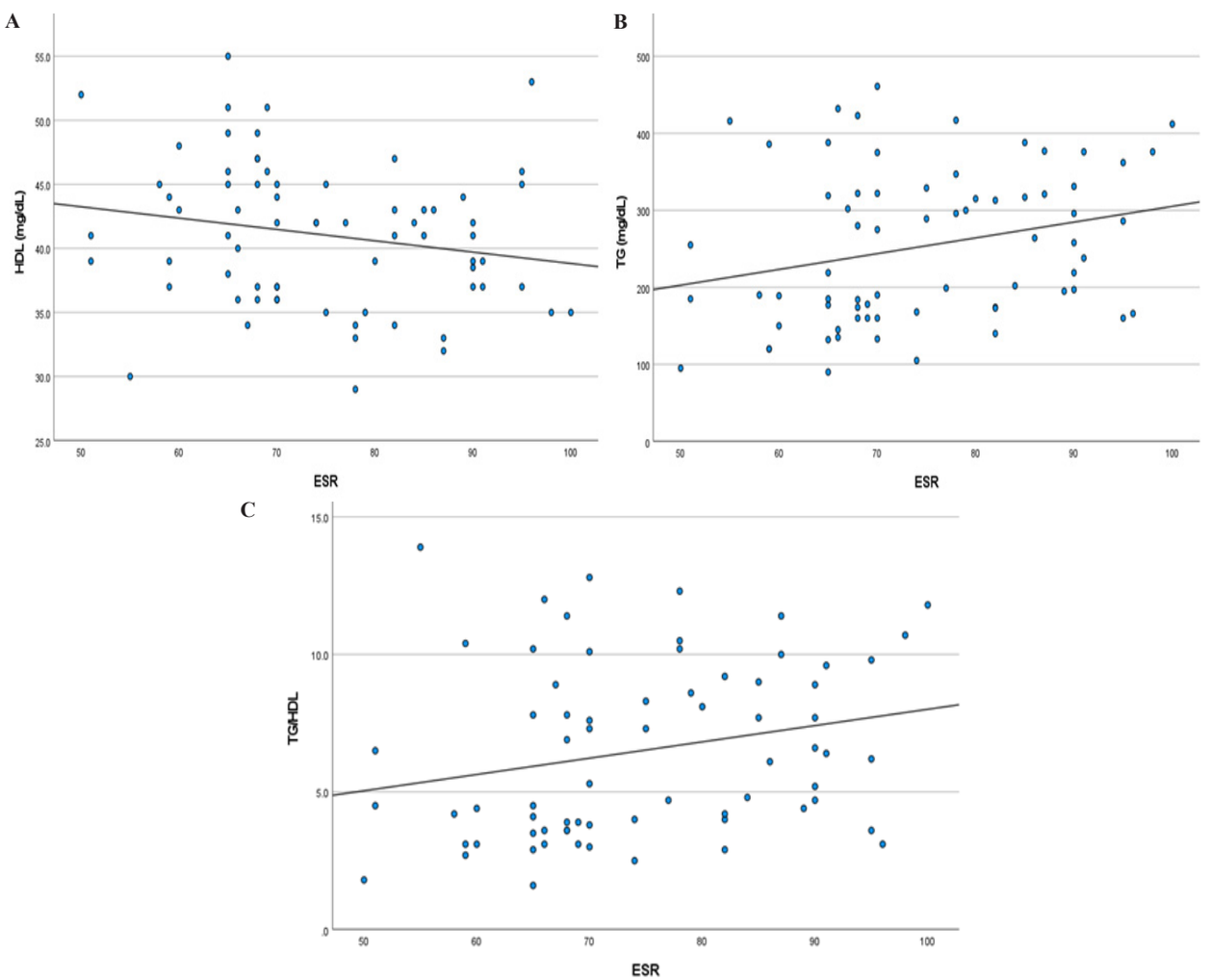

Fig. 2: Scatter plot showing the correlation between ESR and (A) HDL level, (B) Triglycerides level, (C) Triglycerides/HDL levels

Lipid profile showed no statistically significant differences according to received treatment including hydroxychloroquine, systemic steroids, and azathioprine as shown in (Table 3).

Table 3: Comparison of lipid profile according to received treatment.

	Hydroxychloroquine				
	No		Yes		
	Mean	SD	Mean	SD	*P value*
Cholesterol (mg/dL)	233.2	44.7	238.8	42.4	0.798
TG (mg/dL)	252.4	98.9	253.9	100.6	0.985
HDL (mg/dL)	41.4	5.6	40.9	5.7	0.644
LDL (mg/dL)	141.3	34	147.4	33.1	0.422
TG/HDL ratio	6.4	3.2	6.6	3.2	0.894
Cholesterol/HDL ratio	5.8	1.7	6.1	1.7	0.601
			Systemic steroids		
	No		Yes		
	Mean	SD	Mean	SD	*P value*
Cholesterol (mg/dL)	238.1	45.6	236.2	42.4	0.968
TG (mg/dL)	251.7	115	253.9	94	0.702
HDL (mg/dL)	40.6	5.4	41.2	5.7	0.620
LDL (mg/dL)	147.7	31.7	144.2	34.1	0.751
TG/HDL ratio	6.6	3.6	6.5	3	0.911
Cholesterol/HDL ratio	6.1	1.8	5.9	1.7	0.895
			Azathioprine		
	No		Yes		
	Mean	SD	Mean	SD	*P value*
Cholesterol (mg/dL)	235.2	41.1	238.4	45.6	0.651
TG (mg/dL)	244.9	86.7	262.8	112.2	0.642
HDL (mg/dL)	41.3	5.8	40.8	5.5	0.676
LDL (mg/dL)	145	34.3	145.3	32.7	0.906
TG/HDL ratio	6.2	2.8	6.8	3.6	0.580
Cholesterol/HDL ratio	5.9	1.6	6	1.8	0.751

TG= triglyceride, HDL= high density lipoprotein, LDL= low density lipoprotein.

We found that neurological manifestations specifically migraine, sinus thrombosis and TIAs were significantly associated with higher cholesterol levels compared to patients without those symptoms with *p-values* 0.01, 0.014, and 0.046 respectively (Table 4).

Migraine patients had significantly lower HDL, higher LDL, TG/HDL ratio and Cholesterol/ HDL ratio compared to those without migraine with *p values* <0.05. As well as, sinus thrombosis was associated with lower HDL, higher TG, higher TG/HDL ratio and higher Cholesterol/ HDL ratio compared to those without sinus thrombosis with *p values* <0.05. On the other hand, no significant difference in the lipid profile was found between APS patients with and without DVT or stroke (Table 4).

Table 4: Comparison of lipid profile according to disease characteristics and complications.

	DVT		
	No	Yes	
	Mean ±SD	Mean ±SD	*P value*
Cholesterol (mg/dl)	261.1±61.4	242.7±39.8	0.296
TG (mg/dl)	275.5±119.4	277.6±106.2	0.907
HDL (mg/dl)	37.2±8.1	39.6±4.5	0.150
LDL (mg/dl)	161±45.5	148.2±28.3	0.215
TG/HDL	7.1±3.8	7.3±3.3	0.753
Cholesterol/HDL	6.6±2.2	6.3±1.6	0.848
	Stroke		
	No	Yes	
Cholesterol (mg/dl)	252.9±57.5	268.8±56	0.320
TG (mg/dl)	275.9±119.8	276.4±118.9	0.889
HDL (mg/dl)	38.6±7.3	35.1±7.2	0.132
LDL (mg/dl)	154±41.2	171.1±44	0.142
TG/HDL	7±3.7	7.5±3.8	0.524
Cholesterol/HDL	6.3±2	7.1±2.2	0.291
	TIAs		
	No	Yes	
Cholesterol (mg/dl)	251.3±55.6	287.6±59.2	0.046
TG (mg/dl)	271.8±114.3	298.5±125.5	0.478
HDL (mg/dl)	38.6±7.6	33.3±4.3	0.023
LDL (mg/dl)	153.8±40.2	179.9±47.6	0.043
TG/HDL	7±3.7	7.5±3.9	0.611
Cholesterol/HDL	6.4±2.1	7.2±2.1	0.239
	Sinus thrombosis		
	No	Yes	
Cholesterol (mg/dl)	249.4±54.3	298.9±57.5	0.014
TG (mg/dl)	261.5±107.7	363.1±128.7	0.020
HDL (mg/dl)	38.9±7.3	31.1±3.3	<0.001
LDL (mg/dl)	154.3±42	179.6±37.9	0.081
TG/HDL	6.7±3.5	9.6±4	0.026
Cholesterol/HDL	6.3±2	7.8±2	0.019
	Migraine		
	No	Yes	
Cholesterol (mg/dl)	248.3±55	295.6±52.6	0.010
TG (mg/dl)	265.4±115.2	322.7±109.5	0.086
HDL (mg/dl)	39.4±7	30.8±4.5	<0.001
LDL (mg/dl)	151.5±39.2	185.9±44.7	0.005
TG/HDL	6.7±3.5	8.9±4.1	0.048
Cholesterol/HDL	6.2±1.9	8.8±2.2	0.016

DVT= deep vein thrombosis, TIAs= transient ischemic attacks, TG= triglycerides, HDL= high density lipoprotein, LDL= low density lipoprotein.

As regard abortion in this study, interestingly, it was associated with normal cholesterol levels accounting for 61.5% with *p value* 0.023. Also, it was associated with normal HDL and normal LDL levels accounting for 41.3%, 53.6% respectively with the same *p values* 0.044 (Table 5,7,8).

Moreover, in the current study, Anemia was associated with elevated TG levels accounting for 83.6% of the patients with *p value* 0.008, (Table 6). On the other hand, thrombocytopenia and leucopenia showed no association with lipid profile abnormalities (Table 5-8).

Table 5: Comparison of cholesterol level according to type of APS and clinical characteristics.

		Cholesterol (>200 mg/dL)				
		Normal (N=15)		Elevated (N=55)		
		N	%	N	%	*P value*
APS type	Primary	8	53.3%	32	58.2%	0.737
	Secondary	7	46.7%	23	41.8%	
Abortion in female patients		8	61.5%	12	27.3%	0.023
DVT		3	20.0%	14	25.5%	0.662
Stroke		3	20.0%	13	23.6%	0.766
TIAs		1	6.7%	10	18.2%	0.277
Sinus thrombosis		0	0.0%	10	18.2%	0.074
Migraine		1	6.7%	12	21.8%	0.181
Anemia		12	80.0%	43	78.2%	0.879
Leucopenia		5	33.3%	7	12.7%	0.061
Thrombocytopenia		0	0.0%	10	18.2%	0.074

APS= antiphospholipid syndrome, DVT= deep vein thrombosis, TIAs= transient ischemic attacks.

Table 6: Comparison of triglycerides levels according to type of APS and clinical characteristics.

		Triglycerides (>150 mg/dL)				
		Normal (N=9)		Elevated (N=61)		
		N	%	N	%	*P value*
APS type	Primary	6	66.7%	34	55.7%	0.536
	Secondary	3	33.3%	27	44.3%	
Abortion in female patients		4	57.1%	16	32.0%	0.192
DVT		3	33.3%	14	23.0%	0.498
Stroke		2	22.2%	14	23.0%	0.961
TIAs		0	0.0%	11	18.0%	0.165
Sinus thrombosis		1	11.1%	9	14.8%	0.771
Migraine		0	0.0%	13	21.3%	0.125
Anemia		4	44.4%	51	83.6%	0.008
Leucopenia		2	22.2%	10	16.4%	0.665
Thrombocytopenia		0	0.0%	10	16.4%	0.19

APS= antiphospholipid syndrome, DVT= deep vein thrombosis, TIAs= transient ischemic attacks.

Table 7: Comparison of HDL level according to type of APS and clinical characteristics.

		HDL (<40 mg/dL)				
		Normal (N=56)		Low level (N=14)		
		N	%	N	%	*P value*
APS type	Primary	34	60.7%	6	42.9%	0.227
	Secondary	22	39.3%	8	57.1%	
Abortion in female patients		19	41.3%	1	9.1%	0.044
DVT		14	25.0%	3	21.4%	0.780
Stroke		14	25.0%	2	14.3%	0.393
TIAs		7	12.5%	4	28.6%	0.139
Sinus thrombosis		5	8.9%	5	35.7%	0.010
Migraine		9	16.1%	4	28.6%	0.282
Anemia		44	78.6%	11	78.6%	1.000
Leucopenia		10	17.9%	2	14.3%	0.751
Thrombocytopenia		8	14.3%	2	14.3%	1.000

HDL= high density lipoprotein, APS= antiphospholipid syndrome, DVT= deep vein thrombosis, TIAs= transient ischemic attacks.

Table 8: Comparison of LDL level according type of APS and clinical characteristics.

		LDL (>150 mg/dL)				
		Normal (N=31)		Elevated (N=39)		
		N	%	N	%	*P value*
APS type	Primary	15	48.4%	25	64.1%	0.187
	Secondary	16	51.6%	14	35.9%	
Abortion in female patients		15	53.6%	5	17.2%	0.004
DVT		7	22.6%	10	25.6%	0.767
Stroke		5	16.1%	11	28.2%	0.232
TIAs		3	9.7%	8	20.5%	0.216
Sinus thrombosis		3	9.7%	7	17.9%	0.326
Migraine		3	9.7%	10	25.6%	0.088
Anemia		24	77.4%	31	79.5%	0.834
Leucopenia		8	25.8%	4	10.3%	0.086
Thrombocytopenia		4	12.9%	6	15.4%	0.768

LDL= low density lipoprotein, APS= antiphospholipid syndrome, DVT= deep vein thrombosis, TIAs= transient ischemic attacks.

DISCUSSION

APS is chronic autoimmune systemic disorder composed of either thrombotic and/or obstetric events existing in patients with permanent antiphospholipid antibodies[13]. The thrombotic events characterized by arterial, venous, or micro-vascular thrombosis with common predominant location especially in cerebral arterial vessels and lower limbs deep veins, however, cerebral venous circulation, hepatic venous system and visceral veins are possibly affected[14].

In our study that enrolled 70 patients with APS, there are 57.1% primary APS while secondary APS accounted for 42.9% (SLE=80%, RA=13.3% and dermatomyositis=6.7%). No significant difference was found between primary and secondary APS as regard lipid profile. Díaz-Coronado and colleagues, in their study that included 352 patients, found that 67.6% had primary APS while 32.3% were diagnosed as secondary APS[1].

Our study revealed that abortion was presented in 35.1% from 57 female suffered from APS; this was in agreement with other several studies that revealed that the prevalence of abortion between patients with APS was approximately 22-75%[15,16]. Interestingly, in this study, abortion was significantly associated with normal cholesterol, LDL, and HDL levels.

Many hypotheses were postulated to explain the high prevalence of fetal loss among females with APS including fetal arterial embolism, placental infarction and villus microtubule embolism as results of antibodies causing pathological changes[17,18]. Lin and Qiu 2010, documented several factors responsible for abortion in patients with APS range from genetic, metabolic, hormonal factors to autoimmune disorder, infection, uterine anatomy and hypercoagulable state[18].

In this study, acute ischemic strokes and TIAs were common nervous system clinical manifestations (22.9% and 15.7%, respectively) while Cervera and his colleagues reported prevalence of ischemic stroke and TIAs of 19.8 and 11.1%, respectively[19]. When patients presented with TIAs and those without TIAs manifestations compared together, we found statistically significant high cholesterol level, LDL level (P= 0.046 and 0.043 respectively) as well as statistically significant low level of HDL (P= 0.023) in patients with TIAs, while we did not found any statistically significant correlation with TG, TG/HDL and cholesterol/HDL ratio. This was in concordance with data reported by Ribeiro and Carvalho, who found that ischemic presentations in APS were attributed to development of atherosclerosis due to many factors including high LDL, hypercholesterolemia, hypertriglyceridemia and low level of HDL[20].

Thrombotic condition in APS patients can be attributed to several mechanisms for examples presence of in situ thrombosis, cardiac embolisms that can be originated from cardiac valvular apparatus involvement with deposition of immune complexes and subsequent thickening of valve leaflets (Liebman-Sacks endocarditis)[21], and also the role of oxidative stress that cause damage to blood vessels through the direct effect of antibodies deposition[22], in addition to coagulation cascade changes, protein C molecules inhibition, annexin and antithrombin, platelet activation, and marked increased endothelial adhesion molecules expression[23].

Deep venous thrombosis in our study was accounted for 24.3% and sinus thrombosis was in 14.3% of the patients. This is close to the result revealed by Díaz-Coronado and colleagues, who reported that, DVT was presented in 17.3% of studied APS patients[1].

On comparing patients with and without sinus thrombosis, we found statistically significant high level of cholesterol, TG, TG/HDL ratio and cholesterol/HDL (P=0.014, 0.020, 0.026 and 0.019 respectively) and with low HDL (P=<0.001) in patients with sinus thrombosis versus patients without sinus thrombosis. However, we did not found any significant difference between both groups as regards LDL levels.

Many risk factors, including APS itself, can contribute in thrombotic manifestation in addition to many other risks for atherosclerosis like smoking, hypertension, obesity and dyslipidemia, however, a few studies on lipid abnormalities were performed in patients with APS that documented marked dyslipidemia in those patients when were compared with general population[24,25].

In this research, when the clinical presentations were compared with the high risk level of lipid profile, type of APS (primary and secondary), and other clinical presentations (e.g., TIAs, DVT, migraine and sinus thrombosis) did not exhibit any significant relation.

As regards patients presented with migraine in our study, we found statistically significant high level of cholesterol, LDL, TG/HDL ratio and cholesterol/HDL ratio in patients with migraine versus non migraine patients (*P*=0.010, 0.005, 0.048 and 0.016 respectively) and negative correlation with HDL in migraine group (*P*=<0.001). Meanwhile, We did not found any significant difference between both groups as regard TG levels.

Moreover, in the current study, a statistically significant positive correlation was found between ESR and TG as well as TG/HDL (*P* = 0.019, and 0.017 respectively), while ESR and HDL exhibited statistically significant negative relation (*P* = 0.049). additionally, the high level of TG (> 150 mg/dl) was significantly associated with anemia (*P*=0.008). On the other hand, we did not found any statistically significant relation between serum cholesterol levels, TG, HDL, LDL, TG /HDL, and Cholesterol/HDL when correlated with disease duration, C3 and C4 levels.

In this issue, Zhou and his colleagues found that TG and LDL levels showed statistically significant increase in the patients with other autoimmune diseases like SLE while HDL was significantly decreased in those patients[25,26].

Finally, regarding medication commonly used in APS cases, we did not found any effect of therapy received (Hydroxychloroquine, systemic steroid or Azathioprine) on lipid profile.

CONCLUSION

Dyslipidemia are common in APS especially in patients with TIAs, sinus thrombosis and migraine. No significant relation between medications used in treatment of APS and lipid profile levels.

LIMITATION OF OUR STUDY

We included a relatively small sample size. Larger number of patients with diverse habits, cultural and ethnic groups is needed.

AUTHORS' CONTRIBUTIONS

HAME, AAM, MAA, and AMH carried out the work. HAME performed the protocol. HAME, AAM, MAA, and AMH were responsible for collecting the scientific data. AAM wrote the initial draft of the manuscript. All did revision of manuscript. All authors read and approved the final version to be published.

AVAILABILITY OF DATA AND MATERIALS

The data supporting the results of this article are included within the article.

FINANCIAL SPONSORSHIP AND SUPPORT

Self-funding by authors.

CONFLICT OF INTERESTS

There is no conflicts of interest.

ABBREVIATIONS

APS: Antiphospholipid syndrome,

aPL: Antiphospholipid Antibodies,

BMI: Body Mass Index,

C3: Complement 3,

C4: Complement 4,

Chol: cholesterol,

DVT: Deep Venous Thrombosis,

ESR: Erythrocyte Segmentation Rate,

GAPSS: Global Antiphospholipid Syndrome Score,

HDL: High Density Lipoprotein,

LDL: Low Density Lipoprotein,

MRI: Magnetic Resonance Imaging,

MRV: Magnetic Resonance Venography,

RA: Rheumatoid Arthritis,

SLE: Systemic Lupus erythematosus,

TIAs: Transient Ischemic Attacks,

TG: Triglyceride.

ACKNOWLEDGMENT

The authors are grateful to all patients for their participation in this study.

REFERENCES

1. **Díaz-Coronado JC, Herrera-Uribea S, Hernández-Parraa D, Betancur-Vásquezb L, Lacouture-Fierrob J, González-Hurtadob D, *et al.*** Antiphospholipid syndrome (APS): Clinical and immunoserological differences between primary and secondary APS in a Colombian cohort. REV Colomb Rheumatol. 2021; 28(3):191–196.

2. **Danowski A, Rego J, Kakehasi AM *et al.*** Diretrizes para o tratamento da síndrome do anticorpo antifosfolipídeo. Rev Bras Reumatol. 2013; 53:184–192.

3. **Santamaria JR, Badziak D. Barros MFd, Mandelli FL, Cavalin LC and Sato MS.** Síndrome antifosfolípide. Anais Brasileiros de Dermatologia. 2005; 80:225–239.

4. **Espinosa G, Cervera R.** Antiphospholipid syndrome: frequency, main causes and risk factors of mortality. Nat Rev Rheumatol. 2010; 6:296–300.

5. **Ruiz-Irastorza G, Crowther M, Branch W, Khamashta MA.** Antiphospholipid syndrome. Lancet (London, England). 2010; 376:1498–1509.

6. **Da Silva FF, Levy RA, de Carvalho JF.** Cardiovascular risk factors in the antiphospholipid syndrome. J Immunol Res. 2014; 2014:621270.

7. **Sciascia S, Sanna G, Murru V, Roccatello D, Khamashta MA, Bertolaccini ML.** GAPSS: the Global Anti-Phospholipid Syndrome Score. Rheumatology (Oxford, England). 2013; 52(8), 1397–1403.

8. **Pereira JA, Rondó PH, Lemos JO, de Oliveira E, Rocha C, Hipólito T.** Nutritional status and lipid profle of young children in Brazil. J Trop Pediatr. 2013; 59:54–58.

9. **Souza LJD, Souto Filho JTD, Souza TFD, Reis AF, Gicovate Neto, C, Bastos, DA, *et al.*** Prevalence of dyslipidemia and risk factors in Campos dos Goytacazes, in the Brazilian State of Rio de Janeiro. Arquivos Brasileiros de Cardiologia. 2003; 81:257–64.

10. **Ageno W, Becattini C, Brighton T, Selby R, Kamphuisen PW.** Cardiovascular risk factors and venous thromboembolism: a meta-analysis. Circulation. 2008; 117:93–102.

11. **Finazzi G, Marchioli R, Brancaccio V, Schinco P, Wisloff F, Musial J, *et al.*** A randomized clinical trial of high-intensity warfarin vs. conventional antithrombotic therapy for the prevention of recurrent thrombosis in patients with the antiphospholipid syndrome (WAPS). J Thrombos Haemost JTH. 2005; 3:848–53.

12. **Araújo ACO, Domingues RB, van Bellen B.** Comparison between the conventional method and a portable device for determination of INR. J Vasc Brasileiro. 2014; 13:88–93.

13. **Miyakis S, Lockshin MD, Atsumi T, Branch DW, Brey RL, Cervera R, *et al.*** International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS). J Thromb Haemost. 2006; 4:295-306.

14. **Cervera R, Piette JC, Font J, Khamashta MA, Shoenfeld Y, Camps MT, *et al.*** Antiphospholipid syndrome: clinical and immunologic manifestations and patterns of disease expression in a cohort of 1,000 patients. Arthritis Rheum 2002; 46:1019-27.

15. **Robertson B, Greaves M.** Antiphospholipid syndrome: an evolving story. Blood Rev. 2006; 20(4): 201-212.

16. **Singh NK, Behera DR, Agrawal A, Singh MN, Kumar V, Godhra M, *et al.*** Hospital based prospective longitudinal clinical and immunologic study of 179 patients of primary anti-phospholipid syndrome. Int. J Rheum Dis. 2013; 16(5): 547-555.

17. **Fujieda Y, Amengual O and Atsumi T:** Pathogenic role of antiphospholipid antibodies: An update. Lupus.2018; 27:2012–2013. 2018.

18. **Lin QD and Qiu LH:** Pathogenesis, diagnosis, and treatment of recurrent spontaneous abortion with immune type. Front Med China.2010; 4:275–279.

19. **Cervera R, Boffa MC, Khamashta MA, Hughes GR.** The Euro-Phospholipid project: epidemiology of the antiphospholipid syndrome in Europe. Lupus. 2009; 18:889–93.

20. **Ribeiro AR, Carvalho JF.** Fatores de risco tradicionais para doença cardiovascular na síndrome antifosfolípide 36 primária comparada à secundária: um estudo de 96 pacientes [Traditional risk factors for cardiovascular disease in primary antiphospholipid syndrome (APS) when compared with secondary APS: a study with 96 patients]. Acta Reumatol Port. 2010; 35(1):36-41.

21. **Ricarte IF, Dutra LA, Abrantes FF, Toso FF, Barsottini OGP, Silva GS, *et al.*** Neurologic manifestations of antiphospholipid syndrome. Lupus.2018; 961203318776110.

22. **Jara LJ, Medina G, Vera-Lastra O, Shoenfeld Y.** Atherosclerosis and antiphospholipid syndrome. Clin Rev Allergy Immunol. 2003; 25:79–88.

23. **Sangle NA, Smock KJ.** "Antiphospholipid antibody syndrome," Archives of Pathology & Laboratory Medicine. 2011; 135 (9): 1092–1096.

24. **Medina G, Gutiérrez-Moreno AL, Vera-Lastra O, Saavedra MA, Jara LJ.** Prevalence of metabolic syndrome in primary antiphospholipid syndrome patients. Autoimmun Rev. 2011; 10:214–7.

25. **Rodrigues C, Bonfá E, Caleiro M, Vendramini M, Bueno C, Lopes JB, *et al.*** Association of arterial events with the coexistence of metabolic syndrome and primary antiphospholipid syndrome. Arthritis Care Res. 2012; 64:1576–83.

26. **Zhou B, Xia Y, She J.** Dysregulated serum lipid profile and its correlation to disease activity in young female adults diagnosed with systemic lupus erythematosus: a cross-sectional study. Lipids Health Dis. 2020; 19(1):40.

الاستفادة الإكلينيكية من تحليل الدهون بالدم لمرضى متلازمة الأجسام المضادة للفوسفوليبيد

حسن عبدالعاطي السنباطي[١]، علي احمد ابوالمعاطي[٢]، محمد عبدالحسيب[٣]، عمرو محمد حواش[٤]

[١]قسم الروماتيزم و التاهيل، [٢]قسم المخ و الاعصاب، كلية الطب، جامعة حلوان

[٣]قسم الباطنة العامة، كلية الطب، جامعة الأزهر، مصر

[٤]قسم الباطنة العامة والروماتيزم والمناعة، كلية الطب، جامعة عين شمس، مصر

الخلفية: متلازمة الأجسام المضادة للفوسفوليبيد هي اضطراب مناعي ذاتي مزمن، يتميز بحدوث تجلط بالأوعية الدموية أو مضاعفات ولادية. يُعدُّ استكشاف عوامل الخطر المرتبطة بها أمرًا ضروريًا لإدارتها.

الهدف: دراسة مستوى الدهون في متلازمة الأجسام المضادة للفوسفوليبيد، وتأثير الأدوية المستخدمة على مستوى الدهون.

المرضى والمنهجية: أُجريت دراسة مقطعية على ٧٠ مريضًا ومريضة يعانون من متلازمة الأجسام المضادة للفوسفوليبيد، وتم جمعهم من عيادات الروماتيزم والتأهيل وعيادات الأعصاب الخارجية وقسم المرضى الداخلي في أربعة مستشفيات جامعية. خضع المرضى لتقييم شامل للتاريخ المرضي وفحص إكلينيكي كامل. تضمنت الفحوصات المعملية: صورة دم كاملة، معدل ترسيب كريات الدم الحمراء، المكمل الثالث (C٣)، المكمل الرابع (C٤)، وتحليل شامل للدهون بعد الصيام، ودلائل مضادات الفوسفوليبيد. والفحوصات المناسبة لتشخيص الجلطات الوريدية والشريانية المختلفة.

النتائج: أظهرت المضاعفات العصبية (وخاصة الصداع النصفي، تجلط الجيوب الوعائية، ونوبات نقص تدفق الدم المؤقتة) ارتباطًا كبيرًا بمستويات الكوليسترول المرتفعة. وكان تجلط الجيوب الوعائية مرتبطًا بانخفاض مستوى الكوليسترول عالي الكثافة، وزيادة الدهون الثلاثية، وارتفاع نسبة الدوهن الثلاثية إلى الكوليسترول عالي الكثافة ونسبة الكوليسترول الكلي إلى الكوليسترول عالي الكثافة. لم تُظهر أي علاقة ذات دلالة إحصائية بين مستويات الدهون والأدوية المستخدمة (هيدروكسي كلوروكوين، الكورتيكوستيرويدات، والأزاثيوبرين).

الخلاصة: اضطرابات الدهون شائعة في متلازمة الأجسام المضادة للفوسفوليبيد، خصوصًا لدى المرضى الذين يعانون من نوبات نقص تدفق الدم المؤقتة، تجلط الجيوب الوعائية، والصداع النصفي. وكان انخفاض مستوى الكوليسترول عالي الكثافة شائعًا لدى المرضى المصابين بتجلط الجيوب الوعائية. لم نجد أي علاقة ذات دلالة إحصائية بين الأدوية المستخدمة لعلاج متلازمة الأجسام المضادة للفوسفوليبيد ومستويات الدهون.