Original Article

Serum Lipids among Drug Naïve or Drug-Free Patients with Obsessive Compulsive Disorder and their Association with Impulsivity: A Comparative Study

Purvi Vats, Basudeb Das, Sourav Khanra

ABSTRACT

Background: The derangement of serum lipids is well documented in psychiatric disorders like schizophrenia, mania, and depression but not in obsessive compulsive disorder (OCD), where it has been inadequately examined. Also, serum lipid abnormalities are increasingly found in "impulsivity," an important sub-construct of OCD. Our study aimed to examine serum lipid profile among patients with OCD and its association with clinical profile and impulsivity among them. **Methods:** Forty drug naïve or drug-free (four weeks for oral and eight weeks for any depot psychotropics) patients with OCD according to International Classification of Disease -10th version (ICD-10): Diagnostic Criteria for Research (DCR) by the World Health Organization (WHO), from outpatient and inpatient departments of a tertiary care psychiatric hospital were recruited. Measures like Yale–Brown Obsessive Compulsive Scale (Y-BOCS), Hamliton Rating Scale for Depression (HAM-D), Barratt's Impulsivity Scale (BIS-11), and Hamilton Rating Scale for Anxiety (HAM-A) were administered. Forty age and sex-matched healthy controls (HC) were recruited after screening with General Health Questionnaire 12 (GHQ-12). Serum lipids were assessed in both the groups. **Results:** Serum high density lipoproteins (HDL) ($P < 0.001$; partial $\eta^2 = 0.176$) and apolipoprotein B ($P < 0.001$; partial $\eta^2 = 0.531$) were significantly higher in OCD group than age- and sex-matched HC. A trend toward lower serum HDL ($P = 0.06$; partial $\eta^2 = 0.060$) was observed among patients of OCD with high impulsivity. Serum HDL was negatively correlated with BIS attention ($r_s = -0.32$; $p = 0.03$), BIS motor ($r_s = 0.40$; $P = 0.01$), BIS non-planning ($r_s = -0.36$; $P = 0.02$), and BIS total ($r_s = -0.36$; $P = 0.01$) scores. Serum triglycerides (TG) ($r_s = 0.34$; $P = 0.03$) and apolipoprotein B ($r_s = -0.32$; $P = 0.04$) were negatively correlated with Y-BOCS compulsion score. Serum TG ($r_s = -0.45$, $P < 0.01$) and serum very low density lipoprotein (VLDL) was negatively ($r_s = -0.39$; $P = 0.01$) correlated with Y-BOCS total scores. Serum VLDL was positively ($r_s = 0.34$; $P = 0.03$) correlated with BIS motor scores. **Conclusions:** Serum lipid fractions are deranged among patients with OCD. Different lipid fractions have different associations with clinical profiles of OCD. Impulsivity among patients with OCD may have a specific association with

Access this article online	
Website: www.ijpm.info	**Quick Response Code**
DOI: 10.4103/IJPSYM.IJPSYM_299_19	

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Vats P, Das B, Khanra S. Serum lipids among drug naïve or drug-free patients with obsessive compulsive disorder and their association with impulsivity: A comparative study. Indian J Psychol Med 2020;42:281-9.

Department of Psychiatry, Central Institute of Psychiatry, Ranchi, Jharkhand, India

Address for correspondence: Dr. Sourav Khanra
Central Institute of Psychiatry, Ranchi, Jharkhand - 834 006, India.
E-mail: psyksk.cip@gmail.com

Submitted: 18-Jul-2019, **Revised:** 27-Aug-2019, **Accepted:** 16-Mar-2020, **Published:** 25-Apr-2020

serum lipids. A small sample size, use of self-report measure without adaptation for impulsivity, a lack of metabolic profile assessment among participants, and a lack of assessment of impulsivity among HC were the limitations of our tudy.

Key words: *Impulsivity, lipids, obsessive-compulsive disorder, OCD*
Key Messages: *Serum lipids are deranged in OCD. Serum lipids are also varyingly correlated with impulsivity and severity of OCD.*

Impulsivity is defined as a predisposition for reacting to stimuli in a rapid and unplanned fashion with a reduced concern of the potential consequences.[1,2] The construct of impulsivity is multifaceted,[3,4] impulsivity manifests as "actions which are poorly conceived, prematurely expressed, unduly risky or inappropriate to the situation and that often result in undesirable consequences."[5] Patton *et al*.[6] described three main impulsivity sub-traits, namely motor impulsiveness, defined as acting without thinking, cognitive impulsiveness, characterized by making quick cognitive decisions, and non-planning impulsiveness, characterized by present orientations or a lack of "futuring." Higher attentional/cognitive impulsivity sub-construct is a demonstrated risk factor for several psychological consequences.[7]A growing body of evidence highlights the potential implication of impulsivity in obsessive-compulsive disorder (OCD), which affects between 0.3 and 3.1% of the general population and places enormous personal, social, and economic burden on society. For example, a few studies examining trait impulsivity in OCD have found that OCD patients show higher attentional, motor, and non-planning impulsivity than healthy controls (HC) .[8] Individuals with OCD, compared to HC, tend to make risky decisions, favoring options that provide large initial rewards but ultimately lead to a disadvantageous outcome.[9] This consistent pattern of irrational responding might reflect an exacerbated anticipation for a reward.[10]

The neurobiological basis of impulsivity has received considerable attention in recent years, in terms of both the anatomical as well as neurochemical foundations.[11-14] Deficient central serotonergic transmission has been proposed as a biological substrate for impulsivity,[15] and a few studies have suggested serum cholesterol to be a surrogate marker for the same and demonstrated a correlation.[16] A few studies have looked into the biochemical foundations of impulsivity in psychiatric disorders. Buydens–Branchey *et al*.[17]suggest that the most important lipid fraction is high-density lipoprotein (HDL), whereas others[18] propose total cholesterol (TC) or the low-density lipoprotein (LDL) fraction to be the important one. The proposal that even the sub-constructs of impulsivity might have different biological underpinnings[19] led to studies exploring the relationship between the former and various cholesterol fractions, and between serum cholesterol and various measures of impulsivity across psychiatric diagnoses. The association was found in patients with schizophrenia,[20] major depressive disorder,[17] bipolar disorder,[21] and substance use.[22] Although derangements of lipid profile in psychiatric illnesses have been studied, as per our best of knowledge, research on the same among patients with OCD and its relationship with impulsivity have not been examined. Hence, this study was conducted with aims and objectives 1) to examine serum lipids among patients with OCD and to compare with age- and sex-matched HC, 2) to examine and compare serum lipids between OCD with high impulsivity and OCD with low impulsivity, and 3) to examine the correlation among clinical profiles of OCD, impulsivity, and serum lipids.

METHODS

Participants

The study was a hospital-based cross-sectional study conducted at the outpatient(OPD) and inpatient department (IPD) of a tertiary care psychiatric hospital in India. Participants were selected with the purposive sampling method. Patients fulfilling inclusion and exclusion criteria during the study period, from May 2018 to January 2019, were recruited. Thus, the study group (cases) comprised of 40 patients of both sexes, aged 18–60 years, who had given informed consent, having primary diagnosis of OCD according to International Classification of Disease 10th Version (ICD-10), Diagnostic Criteria for Research (DCR) by World Health Organization (WHO),[23] and who were drug naïve or not on psychotropics for last four weeks (eight weeks in case of depot anti psychotics). Patients with comorbid physical illness like diabetes mellitus, liver disease, renal disease, hypertension, thyroid dysfunction, etc.; those who were on lipid-lowering agents, oral contraceptives or beta blockers; and those with comorbid psychiatric illness fulfilling ICD-10 DCR[23] or with psychosis were excluded. The control group comprised 40 age-and sex-matched healthy individuals who had scored less than 3 on the General Health Questionnaire (GHQ-12)[24] and had given informed consent.

Tools

Socio-demographic datasheet

Socio-demographic information was collected in a pre designed, semi-structured socio-demographic proforma.

Yale-brown obsessive-compulsive scale (Y-BOCS)[25]

This rating scale is designed to rate the severity and type of symptoms in patients with OCD. In general, the ratings depend upon the patient's report; however, the final rating is based on the clinical judgment of the interviewer.

Barratt's impulsiveness scale-11 (BIS-11)[6]

It is the most commonly used self-report measure for assessing impulsivity in both clinical and research settings. Original English version of the scale without any adaptation was used for the study. The subscales were introduced into the scale in its version 10, in the recognition of the multi dimensional nature of impulsivity, which became evident after factor analytic studies. BIS 11 is a further improvement on that, with the labeling of the "Attentional Impulsiveness" subscale, defined as an inability to focus attention or concentrate. In the BIS 11, there are 30 personal statements designed to assess general impulsiveness,[6] considering the multi-factorial nature of the construct. Items are rated from 1 (absent) to 4 (most extreme), and scores range from 30 to 120. The BIS 11 identifies three components of impulsivity, namely attentional/cognitive impulsivity, motor impulsivity, and non-planning impulsivity.

Hamilton rating scale for depression[26]

The HDRS is the most widely used clinician-administered depression assessment scale. The original version contains 17 items (HDRS17) pertaining to the symptoms of depression experienced over the past week. The HDRS was originally developed for hospital inpatients, thus the emphasis on melancholic and physical symptoms of depression. A later 21-item version (HDRS21) includes four items intended to subtype the depression.

Hamilton anxiety rating scale (HAM-A)[27]

It was one of the first rating scales developed to measure the severity of anxiety symptoms and is still widely used today in both clinical and research settings. The scale consists of 14 items, each defined by a series of symptoms, and measures both psychic anxiety (mental agitation and psychological distress) and somatic anxiety (physical complaints related to anxiety).

General health questionnaire 12[24]

General Health Questionnaire (GHQ) is the most common assessment tool of mental well-being. Developed as a screening tool to detect those likely to have or be at a risk of developing psychiatric disorders, it is a measure of the common mental health problems/domains of depression, anxiety, somatic symptoms, and social withdrawal. GHQ 12 has been used most widely in other working populations, allowing for more valid comparisons.

Data collection

After obtaining ethical permission from the institutional ethical committee, drug naïve patients from OPD and IPD of this institute, fulfilling inclusion and exclusion criteria, were selected. Those who gave informed consent were recruited for the study. The socio-demographic information was recorded.Y-BOCS, BIS -11, HAM-D, and HAM-A were administered on each recruited patient. A sample of 5 ml of venous blood was collected between 8 am and 9 am, after ensuring that the subjects have fasted for 12 hours. The blood sample was analyzed on the same day, within two hours of collection, during which the sample was stored at room temperature. Serum TC, HDL, LDL, very low-density lipoprotein (VLDL) and triglyceride (TG) estimation was done by an enzymatic method (using cholesterol esterase, cholesterol oxidase, and peroxidase). Apolipoproteins (Apo) A1 and B fractions were measured using immunoturbidometry.

Age and sex-matched healthy controls were selected through local advertisement, from the hospital staff. GHQ 12 was administered to check the eligibility for inclusion in the study. Serum lipids were measured following the protocol mentioned above.

Statistical analysis

Data were analyzed with Statistical Package of Social Science Version 25 (SPSS) for Windows. The frequency counts of the categorical variables were done and compared using the Chi-square test. Multivariate analysis, considering age in years as a covariate was performed to compare means between the groups. Partial $\eta^2$was calculated for effect size. Bonferroni correction was applied for multiple comparisons. Spearman correlation was used to examine the correlation among serum lipid fractions and clinical profile of the study group.

RESULTS

Sample characteristics

Table 1 shows the comparison of socio-demographic variables between the patients of OCD and HC. There is no significant difference between the two groups in their socio-demographic characteristics. Table 2 shows the descriptive statistics of the clinical profile of OCD patients.

Comparison of serum lipids between OCD HC

There was statistically significant difference in serum lipids between groups, $F(7, 71) = 14.75$, $P < 0.001$;

Table 1: Comparison of Sociodemographic characteristics between OCD cases and healthy controls (HC)

	Variables	OCD (*n*=40) *n* (%)/Mean±SD	HC (*n*=40) *n* (%)/Mean±SD	*t*/*U*/χ^2	df/*Z*	*P*
Age (in years)		32.78±8.45	31.58±8.53	0.63	78	0.52
Education (years)		12.90±2.59	12.90±2.59	800.00^U	0.00^Z	1.00
Sex	Male	17 (21.3%)	24 (30.0%)	2.45	1	0.17
	Female	23 (28.7%)	16 (20.0%)			
Religion	Hindu	35 (43.8%)	33 (41.3%)	0.39	1	0.75
	Others	5 (6.3%)	7 (8.8%)			
Occupation	Employed	12 (15.0%)	12 (15.0%)	0.00	1	1.00
	Unemployed	28 (35.0%)	28 (35.0%)			
Marital Status	Married	33 (41.25%)	24 (30%)	3.94	1	0.06
	Unmarried	7 (8.75%)	16 (20%)			
Family Type	Nuclear	15 (18.75%)	17 (21.25%)	0.20	1	0.82
	Joint/Extended	25 (31.25%)	23 (28.75%)			
Habitat	Rural	17 (21.25%)	15 (18.75%)	0.20	1	0.82
	Urban	23 (28.75%)	25 (31.25%)			
Family History of Psychiatric Illness	Yes	8 (10%)	11 (13.75%)	0.62	1	0.60
	No	32 (40%)	29 (36.25%)			
Family History of Medical Illness	Yes	8 (10%)	6 (7.5%)	0.34	1	0.77
	No	32 (40%)	34 (42.5%)			

OCD=obsessive compulsive disorder, HC=healthy control, U=Mann-Whitney U, χ^2=Chi square, df=degree of freedom, SD=standard deviation

Table 2: Clinical characteristics of OCD Cases

Variables	OCD (*n*=40) Mean±SD	Minimum	Maximum	Range
Age of onset (years)	28.63±7.29	14.00	49.00	35.00
Duration of illness (years)	4.45±4.84	0.10	25.00	24.90
HDRS	14.23±3.93	6.00	22.00	16.00
HAM A	11.80±5.33	3.00	24.00	21.00
Y-BOCS Obsession	14.60±1.98	12.00	18.00	6.00
Y-BOCS Compulsion	14.68±3.42	5.00	19.00	14.00
Y BOCS TOTAL	29.03±3.61	12.00	35.00	23.00
BIS Attention	12.38±3.09	8.00	23.00	15.00
BIS Motor	21.20±7.47	10.00	38.00	28.00
BIS Non-Planning	21.05±7.50	10.00	39.00	29.00
BISTotal	54.73±17.06	28.00	92.00	64.00

HDRS=Hamilton depression rating scale, HAM-A=Hamilton anxiety rating scale, Y BOCS=Yale brown obsessive-compulsive scale, BIS=Barratt's Impulsiveness scale-11

Wilk's Δ=0.40, partial η^2 = 0.59. Table 3 shows significant difference for serum HDL and serum apolipoprotein B between the groups. Table 4 shows statistically significant difference between the groups for serum HDL (P < 0.001, partial η^2 = 0.176) and apolipoprotein B (P < 0.001, partial η^2=0.53).

Comparisons between OCD patients with high and low impulsivity (Median = 49.50)

With median split analysis, patients with OCD were classified as OCD with high impulsivity and OCD with low impulsivity. No significant difference was observed between these groups, F (7, 31)=0.96, P = 0.47; Wilk's Δ=0.82, partial η^2 = 0.17). Further analysis did not reveal any significant difference for serum lipid fractions between these groups. Tables 5 and 6 show between-subject effects and the comparisons.

Correlations among variables in patients of OCD

The findings suggest a significant negative correlation of Y-BOCS compulsion score with serum TG (r_s =-0.34, P = 0.03) and apolipoprotein B (r_s = -0.32, P = 0.04) levels. Y-BOCS total score negatively correlated with serum TG (r_s = -0.45, P < 0.01) and serum VLDL (r_s = -0.39, P = 0.01). Serum HDL levels negatively correlated with BI Sattention (r_s = -0.32, P = 0.03), BIS motor (r_s = -0.40, P = 0.01), and BIS non-planning (r_s = -0.36, P = 0.02), and BIS total (r_s = -0.36, P = 0.01). Additionally, BIS motor positively correlated with serum VLDL levels (r_s = 0.34, P = 0.03). Table 7 shows correlations among serum lipid fractions and clinical profile of OCD cases.

DISCUSSION

Socio-demographic characteristics

In the present study, the mean age of patients with OCD and control groups was 32.78 ± 8.45 years and 31.58 ± 8.53 years, respectively. Both cases and control groups are comparable in terms of age, with no statistically significant difference (P = 0.52). The mean duration of the education of patients with OCD was 12.90 ± 2.59 years. These are consistent with the findings of a recent multicentric study on gender differences in OCD.[28]

Our OCD group had more females than males. This was unlike a few studies where OCD has an approximately equal male and female gender[29,30] or the males had more representation.[28] Awareness about mental illnesses has increased, which probably brings more females to treatment than before. There

Table 3: Test of between subject effects (*n*=80)

	Type III Sum of Squares	df	Mean Square	*F*	*P*	Partial η^2
Serum Cholesterol	3686.86	1	3686.86	3.57	0.06	0.044
Serum HDL	1682.37	1	1682.37	16.44	<0.001***	0.176
Serum LDL	940.14	1	940.14	1.82	0.18	0.023
Serum TG	310.22	1	310.22	0.15	0.69	0.002
Serum VLDL	139.92	1	139.92	0.42	0.51	0.006
Serum Apolipoprotein A1	48.40	1	48.40	0.86	0.35	0.011
Serum Apolipoprotein B	2952.26	1	2952.26	87.34	<0.001***	0.531

HDL=High density lipoproteins, LDL=Low density lipoproteins, TG=Triglycerides, VLDL=Very low-density lipoproteins, ***$P<0.001$

Table 4: Comparison of Serum Lipids between OCD Cases and Healthy Controls (*n*=80)

Serum Lipids	Group (*n*=40)	Mean±SD	95% Confidence Interval		*P*[a]	Partial η^2
			Lower Bound	Upper Bound		
Serum Cholesterol	OCD	154.98±30.95	144.18	164.42	0.06	0.044
	HC	167.52±33.72	157.80	178.03		
Serum HDL	OCD	45.65±11.59	42.52	48.89	<0.001***	0.176
	HC	36.57±8.30	33.32	39.70		
Serum LDL	OCD	80.10±25.04	72.90	87.22	0.18	0.023
	HC	86.90±19.81	79.77	94.09		
Serum TG	OCD	120.47±52.29	106.18	134.21	0.69	0.002
	HC	123.87±34.58	110.13	138.16		
Serum VLDL	OCD	27.58±19.36	21.81	33.21	0.51	0.006
	HC	30.10±16.51	24.46	35.86		
Serum Apolipoprotein A1	OCD	132.81±8.44	130.49	135.22	0.35	0.011
	HC	131.30±6.30	128.930	133.661		
Serum Apolipoprotein B	OCD	51.72±7.23	49.983	53.648	<0.001***	0.531
	HC	39.72±4.20	37.802	41.467		

HC=Healthy Control, HDL=High density lipoproteins, LDL=Low density lipoproteins, TG=Triglycerides, VLDL=Very low-density lipoproteins, ***$P<0.001$, [a]Adjusted for multiple comparison: Bonferroni

Table 5: Tests of Between-Subjects Effects (*n*=40)

	Type III sum of squares	df	Mean square	*F*	*P*	Partial η^2
Serum Cholesterol	0.60	1	0.60	0.00	0.98	0.000
Serum HDL	482.47	1	482.47	3.75	0.06	0.092
Serum LDL	490.93	1	490.93	0.78	0.38	0.021
Serum TG	8297.18	1	8297.18	3.17	0.08	0.079
Serum VLDL	848.78	1	848.78	2.30	0.13	0.059
Serum Apolipoprotein A1	103.66	1	103.66	1.43	0.23	0.037
Serum Apolipoprotein B	66.83	1	66.83	1.30	0.26	0.034

HDL=High density lipoproteins, LDL=Low density lipoproteins, TG=Triglycerides, VLDL=Very low-density lipoproteins, $P<0.05$

was no statistically significant difference between the two groups (P = 0.179), which suggests homogeneity between the cases and HC in the terms of gender.

The average age of onset of the illness of our patient group was 28.63 ± 7.29 years, which is in somewhat agreement with other studies.[28,31,32] The mean duration of illness in the study was 4.45 ± 4.84 years, which was shorter than that found in a recent multicentric study.[28] We found that the majority of the OCD patients were married. Most studies show that patients are reluctant to get married, perhaps due to their involvement in OC rituals, and between the genders, females with OCD are more likely to get married.[33,34] Tripathi *et al*.[28] had found that the majority of their sample and the majority of females in their sample were married. Thus, the higher female representation may explain our finding on marital status. Most of the patients with OCD were unemployed (35%). There are several lines of evidence suggesting that unemployment and decreased economic productivity may be associated with OCD.[35-38]The majority of the patients were Hindus (43.8%), which is consistent with a recent study[28] and is presumably due to the larger representation of Hindus in the general population.

Clinical profile of OCD cases

The symptom severity of OCD was high, as evidenced by the total Y-BOCS score being 29.03 ± 3.61, which suggests that the cases had severe disorder on an average, with an almost equal severity of obsessive and compulsive symptoms. For total impulsivity scores, higher values were seen in motor and non-planning impulsivity than in attentional.

Comparison of serum lipid profile between OCD cases and HC

We found that OCD patients have significantly higher HDL and serum apolipoprotein B levels than

Table 6: Comparison of Serum Lipids between high impulsivity OCD and low impulsivity OCD cases (*n*=40)

	Groups	Mean±SD	95% Confidence Interval		P^a	Partial η^2
			Lower Bound	Upper Bound		
Serum Cholesterol	High impulsivity OCD	153.25±31.82	140.93	168.71	0.98	0.000
	Low impulsivity OCD	156.15±30.82	140.68	168.46		
Serum lipids-HDL	High impulsivity OCD	42.20±10.38	36.94	47.30	0.06	0.060
	Low impulsivity OCD	49.10±11.95	44.00	54.35		
Serum LDL	High impulsivity OCD	82.75±23.61	72.23	95.08	0.38	0.021
	Low impulsivity OCD	77.45±26.73	65.11	87.96		
Serum TG	High impulsivity OCD	133.55±52.78	111.74	158.46	0.08	0.079
	Low impulsivity OCD	107.40±49.66	82.48	129.20		
Serum VLDL	High impulsivity OCD	31.74±21.70	23.50	41.01	0.13	0.137
	Low impulsivity OCD	23.42±16.19	14.144	31.656		
Serum Apolipoprotein A1	High impulsivity OCD	131.3500±8.00181	127.334	135.094	0.23	0.037
	Low impulsivity OCD	134.3500±8.81551	130.606	138.366		
Serum Apolipoprotein B	High impulsivity OCD	50.7000±7.44877	47.142	53.682	0.26	0.034
	Low impulsivity OCD	52.7500±7.05523	49.768	56.308		

HDL=High density lipoproteins, LDL=Low density lipoproteins, TG=Triglycerides, VLDL=Very low-density lipoproteins, $P<0.05$, [a]Adjusted for multiple comparison: Bonferroni

Table 7: Spearman Correlations among OCD cases (*n*=40)

Variables		Serum TG	Serum Cholesterol	Serum HDL	Serum LDL	Serum VLDL	Serum Apolipoprtein A1	Serum Apolipoprotein B
Y-BOCS Obsession	r_s	-0.11	-0.22	-0.19	-0.05	-0.10	-0.09	0.08
	p	0.47	0.16	0.23	0.73	0.54	0.57	0.59
Y-BOCS Compulsion	r_s	-0.34*	0.02	0.17	0.01	-0.27	0.11	-0.32*
	p	0.03*	0.86	0.27	0.92	0.08	0.49	0.04*
Y-BOCS Total	r_s	-0.45**	-0.03	0.20	0.04	-0.39*	0.00	-0.26
	p	<0.01**	0.84	0.21	0.78	0.01*	0.98	0.09
HDRS	r_s	0.24	0.18	-0.04	0.07	0.18	-0.01	0.10
	p	0.12	0.26	0.78	0.63	0.25	0.93	0.52
HAM A	r_s	0.11	-0.02	-0.03	-0.11	0.10	-0.05	0.01
	p	0.47	0.88	0.80	0.47	0.52	0.75	0.90
BIS Attention	r_s	0.24	0.06	-0.32*	0.02	0.26	-0.04	-0.07
	p	0.12	0.70	0.03*	0.87	0.10	0.77	0.63
BIS Motor	r_s	0.29	0.02	-0.40**	0.14	0.34*	-0.23	-0.25
	p	0.06	0.90	0.01*	0.36	0.03*	0.13	0.10
BIS Non planning	r_s	0.25	-0.15	-0.36*	0.08	0.31	-0.15	-0.19
	p	0.11	0.33	0.02*	0.61	0.05	0.35	0.21
BIS Total	r_s	0.28	-0.07	-0.36*	0.11	0.33*	-0.20	-0.17
	p	0.07	0.65	0.01*	0.48	0.03*	0.21	0.27

*$P<0.05$, **$P<0.01$, rs=Spearman's correlation coefficient, HDRS=Hamilton depression rating scale, HAM A=Hamilton anxiety rating scale, Y-BOCS=Yale-Brown obsessive compulsive scale, BIS=Barratt's Impulsiveness scale-11, HDL=High density lipoproteins, LDL=Low density lipoproteins, TG=Triglycerides, VLDL=Very low density lipoproteins

healthy controls. Agargun *et al*.[39] revealed higher LDL, VLDL, and triglyceride levels, but lower HDL levels, than normal controls. Peter *et al*. noted similar findings.[40,41] Freedman *et al*.[42] found normal cholesterol levels in OCD. However, these findings are not consistent with the present study, which found higher serum HDL levels in the OCD group than in normal controls, along with other lipid fractions being similar between the two groups. A possible explanation for the contradictory findings in the present study might be the small size and other characteristics of our sample.

Regarding the role of serum apolipoproteins and human behaviors, much data do not exist. However, low apo B levels are seen in Indian males with a history of violent crimes compared to controls.[43] Kavoor *et al*.[21] showed a significant negative correlation between serum apo B levels and impulsivity in bipolar patients. But no study investigating the serum apolipoproteins in OCD patients has been done so far. The present study found statistically significant higher values of serum apolipoprotein B in the OCD group than HC. Whether this is related to the disease process of OCD or its specific characteristics needs to be further examined.

Comparison between high impulsivity and low impulsivity OCD patients

Our study did not find any difference in serum lipids between OCD with high or low impulsivity. A trend toward lower serum HDL ($P = 0.06$, partial $\eta^2 = 0.06$) was observed in OCD with high impulsivity group. Similar findings were associated with higher suicidal behavior, which can be attributed to high impulsivity in drug naïve patients of OCD.[44] Aguglia *et al*.[45] also found low serum HDL along with low serum triglycerides in patients of OCD having high impulsivity as manifested by their self-harming behaviors than normal controls. Several researchers have studied the role of low serum HDL cholesterol in impulsive behaviors.[17,46,47] They believe that HDL is the most important lipid fraction when considering impulsivity as a whole. Our study also has indicated this trend. Moreover, several studies have shown that OCD patients may have increased oxidative stress and that this correlates with disease severity; we may speculate that lipid peroxidation might be one of the causes of reduced serum HDL-C and altered lipid profile in these patients.[48,49] A trend towards higher serum VLDL was observed among OCD cases than HC,which is similar to an earlier study.[39]

Correlations between OCD clinical profile and serum lipid profile

We found significant negative correlations among serum TG, apolipoprotein B, and Y-BOCS compulsion scores. Y-BOCS total score was significantly negatively correlated with serum TG and serum VLDL. Serum HDL levels of OCD patients were negatively correlated with BIS total, BIS attention, BIS motor, and BIS non-planning. Additionally, BIS motor was positively correlated with serum VLDL. A previous study[39] showed lower serum HDL in a sample of OCD patients thanHC. These findings are in line with a few other studies.[40,41] But in none of these studies, the correlation among illness severity, impulsivity, and serum lipids were examined. The present results are not in concurrence with these studies, and as enough data between the correlation of individual scores on Y-BOCS and serum lipid fractions do not exist, this requires further research.

We found a statistically significant negative correlation between serum HDL levels and BIS total, BIS attention, BIS motor, and BIS non-planning. De Berardis *et al*.[44] and Aguglia *et al*.[45] had demonstrated low HDL levels associated with higher suicidal ideation, which is a measure of higher attentional impulsivity in drug naïve patients of OCD, but a correlation was not established between impulsivity and serum HDL levels. Kavoor *et al*.[21] revealed a statistically significant negative correlation between impulsivity and serum lipid fractions, namely total cholesterol and triglycerides, but not serum HDL in bipolar patients. Our findings are in contrast to the results of another recent study among the patients of schizophrenia where total cholesterol and LDL levels showed significant negative correlations with scores on impulsivity ($P < 0.01$) and serum TG level showed a negative correlation with impulsivity ($P < 0.05$).[20]

Limitations and future directions

Our study adds to the growing literature on the complex relationship among lipid fractions, impulsivity, and psychiatric disorders. But it suffers from some limitations. The sample size was small. Physical factors influencing serum lipids were not assessed. Tool to measure impulsivity was self-report measure without adaptation. Impulsivity was not assessed in the HC group. Future research including a larger sample size and metabolic profile should be conducted.

CONCLUSION

There is a significant statistical difference in serum HDL and serum apolipoprotein B between drug naïve patients of OCD and age- and sex-matched HC. Serum HDL was negatively correlated with all scores of impulsivities. Serum TG and apolipoprotein B were negatively correlated with the Y-BOCS compulsion score. Serum TG and serum VLDL were negatively corelated with Y-BOCS total score, whereas only serum VLDL was positively correlated BIS motor scores.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Fineberg NA, Potenza MN, Chamberlain SR, Berlin HA, Menzies L, Bechara A, *et al*. Probing compulsive and impulsive behaviors, from animal models to endophenotypes: A narrative review. Neurospychophramacol 2010;35:591-604.
2. Chamberlain SR, Leppink EW, Redden SA, Grant JE. Are obsessive-compulsive symptoms impulsive, compulsive or both?Compr Psychiatry 2016;68:111-8.
3. Weinstein K.Impulsivity in an epidemiological catchment area sample of the general population: A confirmatory factor analysis study of the Barratt Impulsiveness Scale[thesis on the internet]. Montreal, Quebec: McGill University; 2012. Available from: https://www. semanticscholar.org / paper/ Impulsivity-in-an-epidemiological-catchment-area-of-Weinstein/d9f2f9e76915c4253da0f63908c2e8bdd3b11e9a. [Last accessed on 2019 Mar 01].
4. Dickman SJ. Impulsivity and information processing. **In:** McCown WG, Johnson JL, Shure MB, editors.The Impulsive client: Theory, Research, and Treatment. Washington, DC,

US: American Psychological Association; 1993. p. 151-84.
5. Daruna JH, Barnes PA.A neurodevelopmental view of impulsivity. In: McCown WG, Johnson JL, Shure MB, editors. The Impulsive Client: Theory, Research, and Treatment. Washington, DC, US: American Psychological Association; 1993. p. 23-37.
6. Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt impulsiveness scale.J Clin Psychol 1995;51:768-74.
7. Corruble E, Benyamina A, Bayle F, Falissard B, Hardy P. Understanding impulsivity in severe depression? A psychometrical contribution.Prog NeuropsychopharmacolBiol Psychiatry 2003;27:829-33.
8. Ettelt S, Ruhrmann S, Barnow S, Buthz F, Hochrein A, Meyer K, *et al*.Impulsiveness in obsessive-compulsive disorder: Results from a family study.Acta PsychiatrScand 2007;115:41-7.
9. Prochazkova L, Parkes L, Dawson A, Youssef G, Ferreira GM, Lorenzetti V, *et al*. Unpacking the role of self-reported compulsivity and impulsivity in obsessive-compulsive disorder.CNS Spectr 2018;23:51-8.
10. Fontenelle LF, Oostermeijer MS, Ferreira GM, Lorenzetti V, Luigjes MJ, Yücel M. Anticipated reward in obsessive-compulsive disorder. EurNeuropsychopharmacol2016;26:888-9.
11. Figee M, van de Nmunckhof P, Schuurman R, Denys D. Neuroimaging deep brain stimulation in psychiatric disorders. In: Denys D, Feenstra M, Schuurman R, editors. Deep Brain Stimulation: ANew Frontier in Psychiatry. New York, USA: Springer; 2012. p. 225-39.
12. Knutson B, Adams CM, Fong GW, Hommer D. Anticipation of increasing monetary reward selectively recruits nucleus accumbens.J Neurosci 2001;21:RC159.
13. Basar K, Sesia T, Groenewegen H, Steinbusch HW, Visser-Vandewalle V, Temel Y. Nucleus accumbens and impulsivity.Prog Neurobiol 2010;92:533-57.
14. Pattij T, Vanderschuren LJ. The neuropharmacology of impulsive behaviour.TrendsPharmacol Sci 2008;29:192-9.
15. Troisi A. Cholesterol in coronary heart disease and psychiatric disorders: Same or opposite effects on morbidity risk?Neurosci Biobehav Rev 2009;33:125-32.
16. Vevera J, Fišar Z, Kvasnička T, Zdeněk H, Stárková L, Češka R, *et al*. Cholesterol-lowering therapy evokes time-limited changes in serotonergic transmission.Psychiatr Res 2005;133:197-203.
17. Buydens-Branchey L, Branchey M, Hudson J, Fergeson P. Low HDL cholesterol, aggression and altered central serotonergic activity.Psychiatr Res 2000;93:93-102.
18. Troisi A. Low cholesterol is a risk factor for attentional impulsivity in patients with mood symptoms.Psychiatr Res 2011;188:83-7.
19. Barratt ES, Stanford MS, Kent TA, Alan F. Neuropsychological and cognitive psychophysiological substrates of impulsive aggression.Biol Psychiatr 1997;41:1045-61.
20. Kavoor AR, Mitra S, Kumar S, Sisodia AK, Jain R. Lipids, aggression, suicidality and impulsivity in drug-naïve/drug-free patients of schizophrenia.Asian J Psychiatr2017;27:129-36.
21. Kavoor AR, Ram D, Mitra S. Lipid correlates of attentional impulsivity in first episode mania: Results from an Indian population.Indian J Psychol Med 2014;36:378-84.
22. Conklin SM, Stanford MS. Premeditated aggression is associated with serum cholesterol in abstinent drug and alcohol dependent men.Psychiatr Res 2008;157:283-7.
23. World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research. Geneva: World Health Oranizatrion; 1993.
24. Goldberg D. Manual of the General Health Questionnaire. NFER Nelson; 1978.
25. Goodman WK, Price LH, Rasmussen SA, Mazure C, Fleischmann RL, Hill CL, *et al*. The Yale–Brown Obsessive-Compulsive Scale. I. Development, use, and reliability. Arch Gen Psychiatry 1989;46:1006-11.
26. Hamilton M. Diagnosis and rating scale for depression.Br J Psychiatry 1960;3:76-8.
27. Hamilton M. The assessment of anxiety states by rating.BrJ Med Psychol1959;32:50-5.
28. Tripathi A, Avasthi A, Grover S, Sharma E, Lakdawala BM, Thirunavukarasu M, *et al*. Gender differences in obsessive-compulsive disorder: Findings from a multicentric study from India. Asian J Psychiatr 2018;37:3-9.
29. Lochner C, Stein DJ. Gender in obsessive-compulsive disorder and obsessive-compulsive spectrum disorders. Arch Womens Ment Health 2001;4:19-26.
30. Raines AM, Oglesby ME, Allan NP, Mathes BM, Sutton CA, Schmidt NB. Examining the role of sex differences in obsessive-compulsive symptom dimensions.Psychiatr Res 2018;259:265-9.
31. Rasmussen SA, Eisen JL. The epidemiology and clinical features of obsessive-compulsive disorder.Psychiatr Clin North Am 1992;15:743-58.
32. Anholt GE, Aderka IM, van Balkom AJ, Smit JH, Schruers K, van der Wee NJ, *et al*. Age of onset in obsessive-compulsive disorder: Admixture analysis with a large sample. Psychol Med 2014; 44:185-94.
33. Desilva P, Rachman S, Seligman MEP. Prepared phobias and obsessions: Clinical outcome. Behav Res Ther 1977;15:65-77.
34. Neziroglu F, Grunes MS, McKay D. Family involvement in the behavioral treatment of obsessive-compulsive disorder: A preliminary investigation. Behav Ther 2001;32:803-20.
35. Fontenelle LF, Mendlowicz MV, Marques C, Versiani M. Trans-cultural aspects of obsessive-compulsive disorder: Adescription of a Brazilian sample and a systematic review of international clinical studies. J Psychiatr Res 2004;38:403-11.
36. Mohammadi MR, Ghanizadeh A, Rahgozar M, Noorbala AA, Davidian H, Afzali HM, *et al*. Prevalence of obsessive-compulsive disorder in Iran.BMC Psychiatr 2004;4:2.
37. Cilliçilli AS, Telcioğlu M, Aşkın R, Kaya N, Bodur S, Kucur R. Twelve-month prevalence of obsessive-compulsive disorder in Konya, Turkey.Compr Psychiatr 2004;45:367-74.
38. Crino R, Slade T, Andrews G. The changing prevalence and severity of obsessive-compulsive disorder criteria from DSM-III to DSM-IV.Am J Psychiatr 2005;162:876-82.
39. Agargun MY, Dulger H, Inci R, Kara H, Ozer OA, Sekeroglu MR, *et al*. Serum lipid concentrations in obsessive-compulsive disorder patients with and without panic attacks.Can J Psychiatr 2004;49:776-8.
40. Peter H, Hand I, Hohagen F, Koenig A, Mindermann O, Oeder F, *et al*. Serum cholesterol level comparison: Control subjects, anxiety disorder patients, and obsessive-compulsive disorder patients.Can J Psychiatr 2002;47:557-61.
41. Peter H, Tabrizian S, Hand I. Serum cholesterol in patients with obsessive compulsive disorder during treatment with behavior therapy and SSRI or placebo. IntJ Psychiatr Med 2000;30:27-39.
42. Freedman DS, Byers T, Barrett DH, Stroup NE, Eaker E, Monroe-Blum H. Plasma lipid levels and psychologic characteristics in men. Am J Epidemiol 1995;141:507-17.
43. Chakrabarti N, Sinha VK, Sinha BN. A study of lipid profile and apolipoproteins A1 and B: Their relationship to aggression and psychopathology in male patients with psychosis.J Forens Psychiatry Psychol 2004;15:314-24.

44. De Berardis D, Serroni N, Marini S, Rapini G, Carano A, Valchera A, *et al*. Alexithymia, suicidal ideation, and serum lipid levels among drug-naïve outpatients with obsessive-compulsive disorder.Braz J Psychiatry 2014;36:125-30.
45. Aguglia A, Albert U, Maina G. Serum lipids and lifetime suicide attempts in patients with Obsessive-Compulsive Disorder.J ObsessiveCompuls Relat Disord 2017;15:1-6.
46. Horsten M, Wamala SP, Vingerhoets A, Orth-Gomer K. Depressive symptoms, social support, and lipid profile in healthy middle-aged women. Psychosom Med 1997;59:521-8.
47. Maes M, Christophe A, Delanghe J, Altamura C, Neels H, Meltzer HY. Lowered ω3 polyunsaturated fatty acids in serum phospholipids and cholesteryl esters of depressed patients. Psychiatr Res 1999;85:275-91.
48. Behl A, Swami G, Sircar SS, Bhatia MS, Banerjee BD. Relationship of possible stress-related biochemical markers to oxidative/antioxidative status in obsessive-compulsive disorder. Neuropsychobiol 2010;61:210-4.
49. Kuloglu M, Ustundag B, Atmaca M, Canatan H, Tezcan AE, Cinkilinc N. Lipid peroxidation and antioxidant enzyme levels in patients with schizophrenia and bipolar disorder. Cell BiochemFunct2002;20:171-5.