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Effect of Inflammatory Bowel Disease (IBD) and IBD Medications on Dyslipidemia Risk

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ABSTRACT

Background: Inflammatory bowel diseases (IBDs), including Crohn's disease (CD) and ulcerative colitis (UC), involve chronic intestinal inflammation and are associated with increased risks of clotting abnormalities, venous thromboembolism (VTE), cardiovascular diseases, and vascular complications, necessitating regular monitoring of lipid profiles to manage treatment and prevent complications.

Objectives: This study aimed to evaluate lipid profiles in patients newly diagnosed with ulcerative colitis (UC) and crohn's disease (CD) and follow up their lipid profiles after 4 months and 8 months from the diagnosis and start of treatment.

Subjects and methods: This prospective study was performed on 80 Egyptian IBD patients who were newly diagnosed and followed up their lipid profiles in outpatient clinics gastroenterology department at Ain Shams University hospitals. The patients were selected from The Department of Gastroenterology and Gastroenterology Outpatient Clinic through the period from January 2024 till December 2024.

Result: There was high statistically significant difference between lipid profile in different periods in CD and UC cases. Among the studied cases with IBD, CD was the dominating disease in 50 (62.5%) of the cases, the most common diagnosis was Ileocolonic in 18 (22.5%) of cases.

Conclusion: IBD and its medications can significantly impact the risk of hyperlipidemia. Chronic inflammation in IBD can disrupt lipid metabolism, leading to changes in cholesterol and triglyceride levels, increasing the risk of cardiovascular diseases.

Keyword: IBDs, Hyperlipidemia, CD, Dyslipidemia, UC.

INTRODUCTION

Intestinal inflammation and chronic, repeated exacerbations are hallmarks of IBDs, which lead to changes in gut functioning. IBDs include UC and CD [(1)]. With the exception of pathogenic infections, the interaction of genetic and environmental variables is one of the basic causes of these autoimmune illnesses. Although infectious etiology is not a factor in the diagnosis of IBD, clinical, endoscopic, radiological, and histological characteristics are [(2)].

IBDs are associated with vascular-related comorbidities, including ischemic vascular disorders, portal vein thrombosis, and deep vein thrombosis, as well as coagulation abnormalities [(3)]. VTE risk is 1.7–5.9 times higher in IBD patients than in the overall population, according to reports. The total prevalence of VTE in IBD participants was predicted to be 1–8%, and it has been observed to impact 0.55–6.15% of patients with IBD [(4)]. Furthermore, compared to the general population, IBD patients had double the VTE-associated death rate [(5)]. Additionally, a meta-analysis showed that IBD is linked to an 18% increased risk of CVD [(6)], and that women are at a greater risk than men [adjusted odds ratio (aOR), 1.28] [(3)].

Furthermore, there is a 3.4-fold increase in the risk of mesenteric ischemia and a 1.4-fold increase in the risk of VTE [(7)]. Therefore, it is essential to have trustworthy instruments for regularly evaluating lipid profile parameters during follow-up in order to modify treatment and avoid hyperlipidemia consequences.

So, to follow up the effect of the medical treatment of IBD on risk of development of hyperlipidemia. Also, to evaluate lipid profiles in patients newly diagnosed with UC and CD and follow up their lipid profiles after 4 months and 8 months from the diagnosis and start of treatment.

PATIENTS AND METHODS

Study setting: This prospective study included 80 and was performed in Outpatient Clinics Gastroenterology Department at Ain Shams University Hospitals. The patients were selected from the Department of Gastroenterology and Gastroenterology Outpatient Clinic through the period from January 2024 till December 2024.

Study population: Patients diagnosed with IBD attending Ain Shams University. All patients have baseline ultrasound, full labs, colonoscopy, CT enterography or MRE.

Inclusion criteria: Patients who were newly diagnosed and received biological or medical treatment, both sexes and age >18 years.

Exclusion criteria: Patients who were critically unstable, have mental health requirements, suffer from alcohol or drug abuse, pregnant women, and patients who receive chemo-therapy for malignancy.

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All patients were subjected to the following:

Full history taking, full clinical examination and laboratory investigations including: **Lipid profile:** at the time of diagnosis and after 4 months and 8 months from the diagnosis and start of treatment. To evaluate hyperlipidemia which is defined as abnormal lipid levels with total cholesterol (TC) ≥ 200 mg/dL, triglycerides (TGs) ≥ 150 mg/dL, high-density lipoprotein cholesterol (HDL-C) ≤ 40 mg/dL, and low density lipoprotein cholesterol (LDL-C) ≥ 130 mg/dL, complete blood picture, erythrocyte sedimentation rate, C-reactive protein, fecal cal-protectin, viral markers, colonoscopy report, pathology report and imaging; CT enterography or magnetic resonance enterography esidence and intestinal ultrasound.

Ethical considerations: Following an explanation of the research's objective, study participants gave their written informed permission. Subject anonymity was guaranteed, and the study complied with Ain Shams University's Ethical Review Committee's guidelines (FMASU M S 709 2020/2021). The study adhered to the Helsinki Declaration throughout its execution.

Statistical analysis

The computer was given data, and IBM SPSS software package version 21.0 was used for analysis. In order to describe qualitative data, percentages and numbers were used. To confirm that the distribution was normal, the Kolmogorov-Smirnov test was employed. IQR, Mean ± SD, median, and range (minimum and maximum) were used to characterize quantitative data. The 5% level was used to determine the significance of the results. For quantitative variables that are regularly distributed, the student t-test (t) was used to compare two groups under study. Consistent actions for quantitative variables that are regularly distributed, ANOVA (F) was used to compare more than two repeated measures. The p-value was deemed significant if it was ≤ 0.05 or less.

RESULTS

This study was a prospective study that was performed in Outpatient Clinics at Ain Shams University Hospital over 80 Egyptian IBD patients who were newly diagnosed and followed up their lipid profiles. 87 patients were enrolled in our study, 7 of them were excluded (3 declined to participate and 4 lost to follow-up), so we were left with 80 cases included in the study.

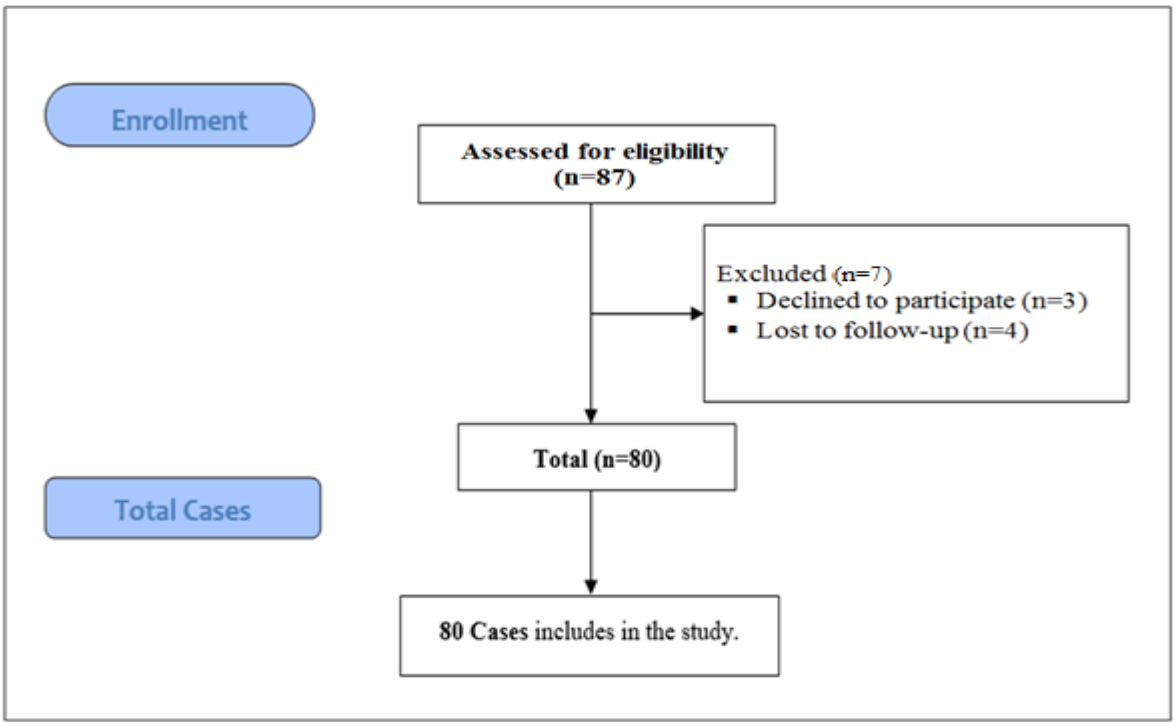

Figure (1): Flow chart of the studied cases.

Table (1) showed demographic data where the mean age of IBD patients was 28.05 ± 5.87 years with range (18-37) years, among the studied cases there were 39 (48.8%) females and 41 (51.2%) males.

In around 40–50% of cases, the disease only affected the small intestine. In another 30–40% of cases, it affected the colon as well. Only the colon was involved in the remaining situations.

Table (1): Descriptive for baseline data of the studied patients

Baseline data		Total no.= 80
Age (Years)	Mean ±SD	28.05 ± 5.87
	Range	18 – 37
Sex	Female	39 (48.8%)
	Male	41 (51.2%)
BMI (kg/m^2)	Mean ±SD	27.2 ± 5.73
	Range	17 – 35.6
Unintended weight loss (%)	<5	62 (77.5%)
	10-May	16 (20%)
	>10	2 (2.5%)
Disease	CD	50 (62.5%)
	UC	30 (37.5%)
Diagnosis	Perianal	6 (7.5%)
	Proctitis	4 (5%)
	Ileal	15 (18.8%)
	Ileocolonic	18 (22.5%)
	Colonic	10 (12.5%)
	Left-sided colitis	12 (15%)
	Extensive colitis	14 (17.5%)
	Upper gastrointestinal	1 (1.2%)

Table (2) Showed descriptive data for changes of mean range of total cholesterol, HDL, LDL and triglycerides at baseline, after 4 months and 8 months of the studied patients.

Table (2): Descriptive for lipid profile at baseline, after 4 months and 8 months of the studied patients

		Total no.= 80
Baseline lipid profile		
Total cholesterol (mg/dL)	Mean ± SD	160.86 ± 17.17
HDL (mg/dL)	Mean ± SD	55.34 ± 7.41
LDL (mg/dL)	Mean ± SD	99.48 ± 13.92
Triglycerides (mg/dL)	Mean ± SD	103.68 ± 26.76
Lipid profile after 4 months		
Total cholesterol l(mg/dL)	Mean ± SD	176.8 ± 19.44
HDL (mg/dL)	Mean ± SD	54.76 ± 7.5
LDL (mg/dL)	Mean ± SD	104.09 ± 15.15
Triglycerides (mg/dL)	Mean ± SD	108.55 ± 26.51
Lipid profile after 8 months		
Total cholesterol (mg/dL)	Mean ± SD	187.44 ± 21.26
Hyperlipidemia	No	56 (70%)
	Yes	24 (30%)
HDL (mg/dL)	Mean ± SD	54.33 ± 7.65
LDL (mg/dL)	Mean ± SD	107.2 ± 17.08
Triglycerides (mg/dL)	Mean ± SD	113.5 ± 22.92

Table (3) showed high significant difference value for total cholesterol changes after 4 months in the group whose baseline Mean was 182.29 ± 8.07. While, there was non-significant difference in LDL, HDL and Triglycerides values after 4 months compared to their baseline ranges.

Table (3): Comparison between cases with and without development of hyperlipidemia regarding lipid profile at baseline and after 4 months of the studied patients

Lipid profile		Hyperlipidemia		Test value	P-value	Sig.
		No	Yes			
		No.= 56	No.= 24			
Baseline						
Total cholesterol (mg/dL)	Mean ± SD	151.68 ± 10.49	182.29 ± 8.07	-12.749•	<0.001	HS
HDL (mg/dL)	Mean ± SD	54.87 ± 7.34	56.42 ± 7.62	-0.852•	0.397	NS
LDL (mg/dL)	Mean ± SD	98.55 ± 13.96	101.62 ± 13.87	-0.904•	0.369	NS
Triglycerides (mg/dL)	Mean ± SD	101.05 ± 25.10	109.79 ± 26.55	-1.345•	0.182	NS
After 4 months						
Total cholesterol (mg/dL)	Mean ± SD	166.41 ± 11.85	201.04 ± 9.29	-12.720•	<0.001	HS
HDL (mg/dL)	Mean ± SD	54.34 ± 7.52	55.75 ± 7.53	-0.769•	0.444	NS
LDL (mg/dL)	Mean ± SD	102.93 ± 14.64	106.79 ± 16.27	-1.046•	0.299	NS
Triglycerides (mg/dL)	Mean ± SD	105.27 ± 26.67	116.21 ± 25.02	-1.712•	0.091	NS

Table (4) showed that statistically, there was non-significant difference between UC and CD laboratory investigations (Hb, WBC, PLT, CRP & FCP).

Table (4): Comparison between CD and UC cases regarding laboratory investigations of the studied patients

		Disease		Test value	P-value	Sig.
		CD	UC			
		No.= 50	No.= 30			
Hb (g/dL)	Mean ± SD	11.87 ± 1.28	12.34 ± 1.16	-1.659•	0.101	NS
WBC (mcL)	Mean ± SD	6.01 ± 1.36	6.16 ± 1.34	-0.475•	0.636	NS
PLT (mcL)	Mean ± SD	232.54 ± 79.86	236.77 ± 9.5	-0.219•	0.827	NS
CRP (mg/L)	Median (IQR)	9.5 (3.3 - 16)	6.5 (2.1 - 12)	-1.531≠	0.126	NS
	Range	0.4 – 25	0.6 – 24			
FCP (µg/g)	Mean ± SD	1694.5 ± 63.87	1720.7 ± 53.9	-1.880•	0.064	NS

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant
•: Independent t-test; ≠: Mann-Whitney test.

Table (5) showed that there was non-significant value between changes occurring in CD and changes occurring in UC.

Table (5): Comparison between CD and UC cases regarding lipid profile at baseline, after 4 months and 8 months of the studied patients

Baseline lipid profile		Disease		Test value	P-value	Sig.
		CD	UC			
		No.= 50	No.= 30			
Total cholesterol (mg/dL)	Mean ± SD	159.88 ± 17.43	162.5 ± 16.91	-0.658•	0.512	NS
HDL (mg/dL)	Mean ± SD	55.06 ± 7.93	55.8 ± 6.54	-0.430•	0.668	NS
LDL (mg/dL)	Mean ± SD	97.5 ± 14.65	102.77 ± 12.13	-1.657•	0.102	NS
Triglycerides (mg/dL)	Mean ± SD	103.36 ± 25.61	104.2 ± 26.22	-0.135•	0.893	NS
Lipid profile after 4 months						
Total cholesterol (mg/dL)	Mean ± SD	176.48 ± 19.15	177.33 ± 20.24	-0.189•	0.851	NS
HDL (mg/dL)	Mean ± SD	54.6 ± 8.03	55.03 ± 6.65	-0.249•	0.804	NS
LDL (mg/dL)	Mean ± SD	102.04 ± 16.01	107.5 ± 13.14	-1.575•	0.119	NS
Triglycerides (mg/dL)	Mean ± SD	108.88 ± 22.11	108 ± 22.62	0.143•	0.887	NS
Lipid profile after 8 months				**Test value**	**P-value**	**Sig.**
Total cholesterol (mg/dL)	Mean ± SD	187.16 ± 20.33	187.9 ± 23.08	-0.150•	0.881	NS
Hyperlipidemia	No	36 (72%)	20 (66.7%)	0.254*	0.614	NS
	Yes	14 (28%)	10 (33.3%)			
HDL (mg/dL)	Mean ± SD	54.22 ± 8.13	54.5 ± 6.9	-0.158•	0.875	NS
LDL (mg/dL)	Mean ± SD	105.06 ± 17.99	110.77 ± 15.07	-1.457•	0.149	NS
Triglycerides (mg/dL)	Mean ± SD	114.3 ± 28.38	112.17 ± 21.05	0.329•	0.743	NS

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant, *: Chi-square test; •: Independent t-test.

Table (6) showed non-sig. diff. in percentage of change in Lipid profile of CD and UC after 4 months and 8 months.

Table (6): Comparison between CD and UC cases regarding percentage of change in lipid profile after 4 and 8 months of the studied patients

		CD No. = 56	UC No. = 24	Test value	P-value	Sig.
% of change after 4 months						
Total cholesterol	Median (IQR)	10.68(8.62 – 12.99)	9.47(8.09 – 10.87)	-1.963≠	0.050	NS
	Range	-1.45 – 18.06	0 – 17.52			
HDL	Median (IQR)	0(-2.04 – 0)	-0.76(-3.45 – 0)	-0.831≠	0.406	NS
	Range	-5.77 – 3.08	-6.25 – 2			
LDL	Median (IQR)	5.21(-0.81 – 8.2)	4.4(0 – 10.2)	-0.075≠	0.941	NS
	Range	-5.19 – 15.85	-5.33 – 16.67			
Triglycerides	Median (IQR)	6.59(0.99 – 9.57)	3.21(-1.8 – 9.76)	-0.666≠	0.506	NS
	Range	-8.2 – 19.32	-4.12 – 25.86			
% of change after 8 months						
Total cholesterol	Median (IQR)	17.78(14.94 – 21.12)	16.8(13.9 – 19.05)	-1.391≠	0.164	NS
	Range	-1.45 – 25.69	0.71 – 26.28			
HDL	Median (IQR)	0(-4.08 – 1.49)	-2.25(-6.45 – 1.52)	-0.888≠	0.375	NS
	Range	-10.42 – 4.55	-8.33 – 3.92			
LDL	Median (IQR)	8.13(-1.63 – 15.57)	7.79(0 – 17.98)	-0.065≠	0.948	NS
	Range	-7.79 – 25	-9.33 – 24.44			
Triglycerides	Median (IQR)	12.36(1.98 – 18.81)	6.36(-2.78 – 17.83)	-0.527≠	0.598	NS
	Range	-16.39 – 34.09	-7.22 – 50			

Median (IQR) and range: non-parametric test; ≠: Mann-Whitney test.
P-value > 0.05: Non-significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

Table (7) showed that there was high significant difference in changes occurring in lipid profile regarding total cholesterol, HDL, LDL and triglycerides.

Table (7): Comparison of lipid profile at baseline, after 4 months and 8 months among all the studied patients

		Baseline	After 4 months	After 8 months	Test value	P-value	Sig.
Total cholesterol (mg/dL)	Mean ±SD	160.86 ± 17.17	176.8 ± 19.44	187.44 ± 21.26	665.687•	<0.001	HS
HDL (mg/dL)	Mean ±SD	55.34 ± 7.41	54.76 ± 7.5	54.33 ± 7.65	18.034•	<0.001	HS
LDL (mg/dL)	Mean ±SD	99.48 ± 13.92	104.09 ± 15.15	107.2 ± 17.08	58.340•	<0.001	HS
Triglycerides (mg/dL)	Mean ±SD	103.68 ± 25.75	108.55 ± 26.51	113.5 ± 27.92	48.974•	<0.001	HS

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant
•: Repeated Measures ANOVA test.

Table (8) showed non-significant difference in the group on biological treatment. While, there was high significant difference in the group on CCS for induction of remission and 5-ASA. And the same for the group on CCS for induction of remission and immuran.

Table (8): Comparison of lipid profile at baseline, after 4 months and 8 months among different types of drug

		Baseline	After 4 months	After 8 months	Test value	P-value	Sig.
Biological							
Total Cholesterol (mg/dL)	Mean ± SD	162.59 ± 19.59	163.41 ± 21.66	165.3 ± 24.91	2.935•	0.075	NS
HDL (mg/dL)	Mean ± SD	55.96 ± 7.88	55.96 ± 7.87	55.63 ± 8.03	1.114•	0.308	NS
LDL (mg/dL)	Mean ± SD	96.59 ± 13.09	97.07 ± 13.49	97.3 ± 14.25	0.372•	0.601	NS
Triglycerides (mg/dL)	Mean ± SD	100.07 ± 24.46	101.96 ± 23.96	103.93 ± 23.53	3.093•	0.090	NS
CCS+induction+5-ASA							
Total (mg/dL) Cholesterol (mg/dL)	Mean ± SD	161.26 ± 15.97	177.07 ± 15.92	187.78 ± 16.81	242.892•	0.000	HS
HDL (mg/dL)	Mean ± SD	55.59 ± 6.74	54.74 ± 6.98	54.19 ± 7.26	11.343•	0.002	HS
LDL (mg/dL)	Mean ± SD	103.11 ± 15.03	107.85 ± 15.81	111.22 ± 17.26	20.306•	0.000	HS
Triglycerides (mg/dL)	Mean ± SD	102.37 ± 25.05	105.89 ± 26.23	109.59 ± 27.32	8.371•	0.007	HS
Immuran + CCS induction							
Total Cholesterol (mg/dL)	Mean ± SD	160.58 ± 19.13	174.73 ± 23.28	184.58 ± 26.45	115.495•	0.000	HS
HDL (mg/dL)	Mean ± SD	54.42 ± 7.76	54.08 ± 7.77	53.85 ± 7.68	2.124•	0.155	NS
LDL (mg/dL)	Mean ± SD	98.69 ± 13.22	103.35 ± 14.37	106.42 ± 16.42	20.254•	0.000	HS
Triglycerides (mg/dL)	Mean ± SD	108.77 ± 22.97	113.96 ± 27.87	119.12 ± 28.74	14.513•	0.001	HS

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant
•: Repeated Measures ANOVA test.

DISCUSSION

Intestinal inflammation and chronic, repeated exacerbations are hallmarks of IBDs, which have no known treatment and cause changes in gut functioning [(8)]. IBDs are associated with thrombophilia, thrombosis in the deep veins, thrombosis in the portal vein, and ischemic vascular disorders [(9)]. IBD patients are reported to have a 1.7–5.9 times greater risk of VTE than the general population. This was shown to afflict 0.55–6.15% of IBD patients, although the overall prevalence of VTE in IBD patients was predicted to be 1-8% [(10)]. Furthermore, compared to the general population, individuals with IBD had a twofold higher risk of VTE-associated death [(11)]. Furthermore, a study showed that having IBD is linked to an 18% increased risk of cardiovascular disease, with the risk being larger in women than in men. Furthermore, the risk of mesenteric ischemia increases 3.4-folds, whereas the risk of VTE increases 1.4-fold [(12)].

This study was conducted on 80 IBD patients. They were 50 CD and 30 UC. In our study as regarding demographic data, we found that the mean age of IBD patients studied cases was 28.05 ± 5.87 with range of 18-37 years. Among the studied cases there were 39 (48.8%) females and 41 (51.2%) males. Our findings align with the findings of **Shivashankar *et al.*** [(13)] research, which comprised 893 IBD patients, 410 CD patients, and 483 UC patients. When CD was diagnosed, the median age was 29.5 years (range: 3.8-93.1years), while UC was diagnosed at 34.9 years (range: 1.2-94.1years). Of the 272 UC patients, 299 were females (51%), while the remaining 272 were males (56%). The most prevalent age group for CD and UC diagnoses was 20–29 years old.

In Our study, there were 39 (48.8%) females and 41 (51.2%) males. which showed greater incidence in males than females. **This goes with Jiang *et al.*** [(14)] who showed that males predominate in IBD. Male to female patient ratios in UC and CD are 1.53:1 and 2.32:1, respectively. While, our study did not go with the study of **Shah *et al.*** [(15)] which was made on 95,605 CD sufferers (52,774 women and 42,831 men) 112,004 individuals with UC (61,672 men and 50,332 women), demonstrated that there was no gender difference in the frequency of UC until the age of 45, at which point males had a noticeably greater incidence of UC than women, with the exception of the 5–9 age group.

In our study the mean BMI was 27.2 ± 5.73 with range of 17-35.6 kg/m^2 and according to unintended weight loss there were 62 (77.5%) < 5%, 16 (20%) 5-10% and 2 (2.5%) >10%. This is similar to **Chan *et al.*** [(16)] that looked at the possibility of a link between incident IBD and obesity. It was created on men and women with CD or UC who were between the ages of 20 and 80. The study showed that BMI was in CD = 25.1 ± 3.8 & in UC = 25.4 ± 3.7.

In our study cases with CD was the dominating disease in 50 (62.5%) of the cases, the most common diagnosis was Ileocolonic in 18 (22.5%) of cases then Ileal 18.8% and 17.7% extensive colitis. This goes with **Silverberg *et al.*** [17] who found that the cecum, ileocecal valve, and terminal ileum are the most frequently affected locations upon presentation in CD. In around 40–50% of cases, the disease only affects the small intestine, in another 30–40% of cases, it affects the colon as well. Only the colon is involved in the remaining situations. Anemia in IBD can arise from a variety of causes, including dietary variables, inflammatory changes during active illness, and blood loss from mucosal injury. It is well established that inflammatory cytokines, particularly TNF-α, disrupt erythropoiesis through their systemic effects on bone marrow stem cells and negatively impact duodenal iron absorption through hepcidin [18].

In our study, the mean hemoglobin was 12.04 ± 1.25 gm/dl. Mean of fecal calprotectin (FCP) was 1704 ± 61.32 µg/g at the time of diagnosis. This was comparable to a study that was carried out at a tertiary care teaching hospital in Eastern India, which included both newly diagnosed UC patients and older UC patients who were receiving routine follow-up. The study found that FCP levels rose as the degree of endoscopic inflammation, as measured by the Mayo score, increased. The median (IQR) FCP levels were 3000µg/g (1342–3000) during active disease and 88µg/g (58–167) following remission (P<0.0001). FCP and Mayo score had connection values of r=0.527 (P<0.0001) during active disease and r=0.663 (P<0.0001) following remission [19]. As the severity of the illness worsened, FCP levels rose as well (r=0.503, P<0.0001). Research has shown inconsistent findings about the relationship between FCP and illness severity. FCP levels and the severity of the illness in UC patients have been found to significantly correlate in several studies. However, another research, found no connection between the degree of illness and FCP concentration [20].

In our investigation, we found that there were changes in lipid profiles of IBD patients during follow up. These changes were increasing in T. cholesterol, LDL, triglycerides while, HDL decreases. This goes against **Romanato *et al.*** [21] whose study showed that T cholesterol. and LDL-C values were considerably lower in 94 individuals with active IBD (34 with UC and 60 with CD) than in healthy participants. However, there were no significant variations in HDL-C or TG levels between IBD and control participants. In disagreement with our study, **Agouridis *et al.*** [22] compared to healthy persons, IBD patients had lower levels of LDL-C and T. cholesterol. This observation is more pronounced in CD patients than in UC patients. Furthermore, no noteworthy changes in HDL-C and TG levels have been documented in these individuals. Our study does not go with meta-analysis, which was made by **Chen *et al.*** [23] whose study showed that serum lipid levels were evaluated between CD and UC, active and inactive, mild and non-mild, and IBD patients and healthy persons respectively. TC, HDL-C, and LDL-C values were considerably lower in IBD patients than in healthy controls. The TC level was much lower in CD groups than in UC groups. The TC and LDL-C values of the active IBD and non-mild UC groups were considerably lower than those of the inactive IBD and mild UC groups respectively.

According to the study's findings, IBD patients had lower total serum lipid levels than healthy people, and these levels were inversely correlated. In agreement with our results partially, **Koutroumpakis *et al.*** [9] made a study on 701 IBD patients (54% CD & 46% UC). IBD patients were more likely to have low HDL and high triglycerides (24 vs. 17 and 33 vs. 25%) and less likely to have high total cholesterol and high LDL cholesterol (6 vs. 13 and 5 vs. 10%) than the general population (all $p < 0.001$). In CD, median triglycerides were greater and median total cholesterol was lower than in UC (171 vs. 184 & 123 vs. 100 mg/dL, both $p < 0.001$). The multiple regression analysis revealed that lipid profile was independently linked with hospitalizations (low cholesterol) and IBD procedures (low cholesterol and high triglycerides). The study concluded that low total cholesterol and high triglyceride levels are more common in IBD patients (particularly CD) than in healthy controls and are independently linked with more severe illness. This goes against **Bigeh *et al.*** [24] who showed that in contrast to non-IBD patients, lipid levels in IBD patients are much lower, according to the vast majority of research requiring lipid examination. Additionally, this discovery appears to be more noticeable in CD patients than in UC patients. All lipid components, including total cholesterol, HDL, LDL, and triglycerides, have also been shown to be at reduced levels. According to one research, 94 IBD patients had reduced levels of total cholesterol and LDL when compared to healthy people. However, despite having decreased cholesterol levels, a small research showed that IBD patients still had elevated carotid artery thickness, homocysteine, and hs-CRP levels, which are early indicators of atherosclerotic cardiovascular disease (ASCVD). In disagreement with our study, **Soh *et al.*** [25] made a study to ascertain if serum lipid profiles and IBD are related. The Korean National Healthcare Insurance service's claims data was used in a population-based research conducted across the country. Enrollment and follow-up were conducted with 9 706 026 participants who were getting medical examinations in 2009. During follow-up, those who acquired UC or CD were identified. The effect of serum lipid profiles on the development of IBD was defined by calculating the adjusted hazard ratio (aHR) by age, sex, body mass index, cigarette smoking, alcohol consumption, exercise, income, and underlying

comorbidities. This research revealed during a median follow-up of 7.3 years that 7,058 people (0.07%) had IBD. Lower blood total cholesterol (TC) levels were linked to a greater prevalence of CD but not UC when compared to the highest quartile of TC values. CD, but not UC, was more common when blood LDL-C levels were lower. Furthermore, but not UC, a greater prevalence of CD was linked to lower blood HDL-C levels. Conversely, a higher prevalence of UC but not CD was linked to lower blood triglyceride levels. According to the study's findings, CD was linked to reduced blood TC, LDL-C, and HDL-C values. UC was associated with low serum triglyceride levels.

In our study, it was found that females have higher risk to develop hyperlipidemia than males. This goes with **Tien *et al.*** [8] who showed that after adjusting for all variables, the risk of hyperlipidemia in IBD was 2.10 times greater in females.

The lipid alterations in CD and UC did not differ significantly in our investigation. This goes against **Biyyani *et al.*** [26] who demonstrated that in comparison to UC patients, CD patients had much lower levels of TC, while CD patients had significantly lower levels of TG than healthy controls. The fact that CD more frequently affects the small intestine might be one explanation for these findings. Bile acid absorption is mostly the responsibility of the terminal ileum. Large amounts of cholesterol and bile acids may be expelled with stools when small intestine absorption is impaired, which might result in a drop in lipid profiles. In line with our findings, **Romanato *et al.*** [21] shown that the biochemical and clinical disease activity characteristics of patients with active CD and those in remission are published. T. cholesterol ($p<0.01$), HDL ($p=0.01$), and LDL cholesterol ($p=0.01$) were significantly higher in remission individuals than in patients with recurrent active disease. This goes against **Biyyani *et al.*** [26] who reported that IBD patient population's lipoprotein profiles were characterized. A comparison was made between 393 IBD patients (241 females and 190 CD patients) and the population database from the National Health and Nutrition Examination (NHANES) Survey 2005-2006. Overall cholesterol (TCHOL) and HDL-C levels are lower in IBD patients, while LDL-C levels are higher in those having NHANES data, according to this study.

In our study, results showed that there was non-significant difference in lipid profile changes in the group of IBD patients on anti-TNFα (infliximab or adalimumab). While, there was high significant difference in lipid profile in the group on CCS (induction) and 5_ASA (maintenance). And the same for the group on CCS (induction) and Imuran (maintenance). In agreement with our study, **Miranda-Bautista *et al.*** [27] who studied cholesterol levels in IBD patients receiving anti-TNFα therapy. It involved a retrospective analysis of the clinical records of 128 consecutive IBD patients who had at least three infliximab doses or two adalimumab doses and a minimum one-year clinical follow-up. Lipid profiles were obtained before to therapy initiation and during one and three years of follow-up (total, HDL and LDL cholesterol, and triglycerides). They found that after one and three years of treatment, there were no appreciable changes in the lipid profiles of IBD patients receiving anti-TNFα medication. This goes partially with **Ferreiro *et al.*** [28] study which compared triglycerides and showed almost statistically significant alterations ($p=0.05$) in the lipid profile of IBD patients receiving anti-TNFα medication in months 0, 4, 8, and 12, while cholesterol did not ($p>0.5$). During the follow-up, there was no statistically significant change in the lipid profile, and it was determined after a year of follow-up, triglycerides in IBD patients receiving anti-TNFα. maintenance medication rose nearly substantially. Although it has little therapeutic significance, Anti-TNFα medication may contribute to lipid profile alterations. It is consistent with a research by **Sleutjes *et al.*** [29] that conducted a systematic literature search of observational cohort studies and randomized controlled trials that evaluated lipid levels both before and after the induction ($\leq$10 weeks) and maintenance (>10 weeks) of IBD medication. Random effects models were used to pool the data from 11 trials, totaling 1663 patients. In six investigations (1211 patients) for whom individual data were available, the impact of patient and illness factors on therapy effects on total cholesterol levels was examined using linear mixed models. The study concluded that corticosteroids and tofacitinib caused the largest increase in total cholesterol levels, but anti-TNFα medications did not. This supports the findings of **Sleutjes *et al.*'s** [29] investigation of the impact of several drug classes on lipid profiles. Serum lipid levels are markedly raised by prednisone and tofacitinib induction treatment, whereas other medication classes showed no change. Because adjusting for systemic inflammation did not change the results, the data appear to be drug-specific. Prednisone plus tofacitinib induction treatment is found to dramatically raise levels of total cholesterol, HDL-C, and LDL-C. Following induction therapy with thiopurines, methotrexate, infliximab, adalimumab, vedolizumab, and ustekinumab, these lipid alterations are not seen. The observed lipid alterations are drug-specific and happen independently of systemic inflammation management, despite the fact that there is an inverse relationship between lipid levels and inflammation. It is necessary to do research on the long-term effects of lipid alterations linked to tofacitinib exposure.

CONCLUSION

IBD and its medications can significantly impact the risk of hyperlipidemia. Chronic inflammation in IBD can disrupt lipid metabolism,

leading to changes in cholesterol and triglyceride levels, increasing the risk of cardiovascular diseases. Certain IBD medications, such as corticosteroids and biologics, can influence lipid levels, while tofacitinib, a JAK inhibitor used in UC, can restore lipid levels without significantly increasing cardiovascular risk. Regular monitoring of lipid levels is crucial, and adjusting treatment plans to balance disease control and lipid management can help mitigate cardiovascular risks.

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