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Predictors of Subclinical Atherosclerotic Cardiovascular Disease in Inflammatory Bowel Disease Patients

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ABSTRACT

Background: Inflammatory bowel disease (IBD) is a chronic intestinal disease that can be caused by a variety of environmental and genetic factors. Several processes that are chronically stimulated in IBD patients have been involved in the pathophysiology of atherosclerotic cardiovascular disease (ASCVD).

Objective: We aimed to estimate 10 years of risk ASCVD in patients with IBD, and to clarify the importance of laboratory and non-invasive imaging modalities in early detection and estimation of ASCVD risk in those populations.

Patients and methods: This was a cross-sectional observational study that was conducted on 80 candidates between October 2019 and October 2021, forty of them were IBD patients and forty were healthy volunteers. Patients were diagnosed as IBD by clinical picture, multi-slice spiral computed tomography (MSCT) abdomen, and colonoscopy followed by histopathological study of the biopsies according to ECCO consensus criteria. For the control group, 40 healthy volunteers were matched by gender and age without other known risk factors for atherosclerosis.

Results: 25 patients were diagnosed as ulcerative colitis, while 15 were diagnosed as Crohn's Disease. IBD patients showed significant dyslipidemia. The median and range of estimated ASCVD risk % among the patients were 27.0 (0.3-46.0), while for the control group, 7.0 (0.3-46.0), and P-value was 0.001. IBD patients had significantly higher pericardial fat in comparison with healthy controls (2.15 (0.04-4.80) vs 1.43 (0.04-3.20), P-value was 0.027*). Multivariate regression analysis showed that increased both pericardial fat; carotid intima-media thickness (CIMT); hyperlipidemia and disease activity were predictors for subclinical ASCVD in patients with IBD.

Conclusions: The risk of ASCVD is risen in IBD patients, particularly during active disease, with increased carotid intimal thickness and wall stiffness. Estimating the thickness of epicardial adipose tissue (EAT) and CIMT were significant predictors.

Keywords: IBD, ASCVD, Carotid duplex, Pericardial fat.

INTRODUCTION

Heart disease remains the top cause of mortality globally, despite enormous breakthroughs in illness detection and treatment. Obesity and type 2 diabetes, as well as genetic, environmental, nutritional, and lifestyle variables, can all contribute to cardiovascular disease (CVD). Aside from that, there's a lot of evidence indicating inflammation plays a function in heart disease and atherosclerosis etiology [(1, 2)].

Inflammatory bowel disease (IBD), which includes ulcerative colitis and Crohn's disease, is the most prevalent systemic inflammatory illness. IBD is a chronic intestinal illness that can be caused by several reasons, including hereditary predisposition and environmental influences [(3, 4)].

Several processes that are chronically stimulated in IBD patients have been involved in the pathophysiology of ASCVD. These include local and systemic inflammation; gut microbiome irregularities, endothelial dysfunction, thrombosis, lipid dysfunction, and the negative effects of IBD therapies, particularly corticosteroids. [(5)]

Our research attempted to assess the 10-year risk of ASCVD in IBD patients and specify the relevance of laboratory and non-invasive imaging modalities in early diagnosis and valuation of ASCVD risk in those IBD populations compared to the healthy population. It also elucidates the detailed ASCVD predictors of IBD patients among an Egyptian cohort, as well as the association between the types of ASCVD and the clinical manifestations of Crohn's disease (CD) and ulcerative colitis (UC) patients followed at the IBD clinic.

PATIENTS AND METHODS

This was a cross-sectional single-center observational study that was conducted on 80 candidates between October 2019 and October 2021, forty of whom were patients and forty of whom were healthy volunteers. The inclusion criteria were 40 patients who were diagnosed as IBD (Crohn's Disease and Ulcerative Colitis) by clinical picture, MSCT abdomen with contrast, and colonoscopy followed by histopathological study of the biopsies according to The European Crohn's and Colitis Organisation [ECCO] consensus criteria[(6)], who were attending to gastrointestinal unit however inpatients and/or outpatients of Assiut University Hospital either in activity or in remission. For the control group, 40 healthy volunteers were matched by gender and age.

To avoid the confounding effect of other known risk factors for atherosclerosis, the exclusion criteria were as follows:

- Previous history of HTN, dyslipidemia with lipid-lowering medications, or diabetes mellitus.

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- Previous history of cardiovascular or cerebrovascular events: congenital or valvular heart disease; any signs indicating cardiac involvement; non-sinus cardiac rhythms; any prior myocardial infarctions
- Neoplastic diseases.
- Thromboembolic and inflammatory rheumatic diseases, including rheumatoid arthritis, psoriatic arthritis, and ankylosing spondylitis; and other systemic diseases such as collagen diseases and hepatic, hemolytic, and renal diseases.

All research participants were subjected to a comprehensive medical history, with special attention paid to the frequency and diarrhea type, abdominal pain, oral and anal ulcers, fever, other extra-intestinal manifestations of IBD, and disease duration; treatment. The Partial Mayo score was used to assess disease activity in ulcerative colitis patients, while the Harvey-Bradshaw index has been used in Crohn's disease patients [(7)].

Methods:

Each participant fasted for at least 8 hours and refrained from smoking and caffeine consumption for at least 4 hours. Blood samples of 10 mL of venous blood were collected from all participants via peripheral vessel. Serum was separated immediately by centrifugation, and serum aliquots were frozen at 80°C until assayed for the following tests:

- CBC (HB level decreased during active disease) done on ABX Pentra XL80.
- Serum glucose liver function test, lipid profile, and CRP were done on Cobas Integra-400 plus analyzer
- Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP): For assessment of disease activity and patient follow-up.
- Liver Function Tests: as serum albumin decreases during disease flare.
- Lipogram serum levels of total cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides were determined using Cobas Integra-400 plus analyzer; low-density lipoprotein (LDL) cholesterol was calculated using the Fried Ewald equation.

Imaging interventions include:

- **Resting ECG**: for detection signs of ischemic heart disease (IHD) (St Segment depression; raising); Left ventricular hypertrophy (LVH) (by voltage criteria using CritiCare machine.
- **Resting Detailed transthoracic echocardiography TTE:**

Philips En Visor C heart disease has been used for transthoracic echocardiography. M-mode, two-dimensional echo was used to evaluate the study population in parasternal and apical views, with patients lying laterally in the decubitus position. The eyeball was used to assess left ventricular systolic function, and M-mode at the papillary muscle level was used to estimate EF (Techoliz method). Left ventricular diastolic function was assessed utilizing pulsed Doppler over mitral inflow for peak E and A wave velocity (cm/s) and E/A ratio, isovolumic relaxation time (IVRT), and pulsed Doppler tissue imaging for E' velocity (cm/sec) over lateral mitral annulus, with E/E ratio calculated. [(8)]**.** During four different cardiac cycles, epicardial fat thickness (EFT) was measured from the parasternal long-axis view on the free wall of the right ventricle at end-diastole. The hypoechoic space in front of the right ventricular free wall was defined as EFT. The maximum perpendicular distance to the aortic annulus was achieved and approximated [(9)].

Carotid U/S Duplex:

Used to measure carotid intima-media thickness and detect atheroma. The carotid arteries were examined using high-resolution B-mode ultrasonography with a LOGIQ P5 (GE Healthcare) linear probe at 7MHz. Patients were examined supine, with the neck turned 45° in the opposite direction of the site being inspected. IMT was measured at 10, and 15 mm adjacent to the carotid bifurcation on the far wall of both the right and left common carotid arteries. The IMT was defined as the distance between the first echogenic line's leading edge and the second echogenic line's leading edge.

Carotid intima-media thickness (CIMT) is a straightforward, non-invasive, reliable, and ancillary vascular parameter for the detection of subclinical atherosclerosis in IBD patients. The normal ranges for people aged 30 to 40, 41 to 50, and over 50 are 0.44-0.57 mm, 0.42-0.5 mm, 0.44-0.57 mm, and 0.46-0.7 mm, respectively [(10)]**.**

Ethical approval:

The Faculty of Medicine Human Ethics Review Committee in Assuit University approved this study on 19/3/2019, with reference number 1700685. Every patient signed informed written consent for the acceptance of the operation. This work has been carried out following The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical analysis

Data entry and data analysis were done using SPSS version 22 (Statistical Package for Social Science). Data were presented as number, percentage, mean, standard deviation, median, and range. Chi-square test and Fisher Exact test were used to compare between qualitative variables. Independent samples t-test was used to compare quantitative variables between groups, Pearson correlation was used to measure the correlation between quantitative variables in the case of parametric data. Mann-Whitney test was used to compare quantitative variables between groups, Spearman correlation was done to measure the

correlation between quantitative variables for non-parametric data. P-value considered statistically significant when P < 0.05. Univariate and multivariate logistic regression models were used to evaluate associations between CRFs and IBD.

RESULTS

Forty IBD patients who attended the Gastroenterology outpatient clinic or were admitted to Assuit University Hospital within twelve months were enrolled in the study. 25 patients were diagnosed as ulcerative colitis, while 15 were diagnosed as Crohn's disease.

Both groups had insignificant differences regarding demographic data (age, gender, smoking, blood pressure). But, IBD patients showed significant anemia, elevated acute phase reactants, liver cell injury, and significant dyslipidemia as shown in **(Table 1)**.

It was noticed that Cohn's patients were 37.5% in comparison with UC were 62.5%, among those noticed 17 patients were in activity state while others were controlled on medical treatment as depicted in **(Table 2).**

Table (3) shows the estimated ASCVD risk percentage among studied cases. It was noticed that patients with IBD had a significantly higher estimated risk 10 years ASCVD in comparison with healthy controls, P= 0.001*.

Table (4) Shows Echocardiographic parameters of the studied population. IBD patients had significantly higher pericardial fat in comparison with healthy controls (2.15 (0.04-4.80) vs 1.43 (0.04-3.20), p=0.027*). Both groups had insignificantly different other Echocardiographic parameters apart from only 2 cases with IHD segmental wall motion abnormality, valvular heart disease (aortic stenosis & mitral regurgitation), Carotid intima-media thickness (IMT) by ultrasound in the studied patients. Patients with IBD had significantly higher bilateral carotid IMT.

Based on the current study the following were the predictors for subclinical ASCVD in patients with IBD; increased pericardial fat (p=0.005*), increased carotid ultrasound duplex IMT (P= 0.001*), hyperlipidemia (Cholesterol p= 0.016*, HDL p= 0.001*), disease activity C-reactive protein (CRP) (p= 0.002* as an activity marker as displaced in **(Table 5).**

Table (1): Demographic and laboratory of the studied patients with IBD

Demographic data	Patients (n= 40) Mean ± SD	Controls (n= 40) Mean ± SD	P-value
Age: (years)	36.50 ± 7.08	37.48 ± 6.62	0.527
Smoker (No., %)	11 (27.5%)	12 (30.0%)	0.805
SBP (mmHg)	119.25 ± 7.97	120.75 ± 7.64	0.393
DBP (mmHg)	79.75 ± 8.62	80.75 ± 8.59	0.605
Hemoglobin (g/dl)	11.80 ± 1.47	13.75 ± 1.10	0.000*
WBCs (10*9/L)	8.47 ± 2.18	8.60 ± 2.04	0.780
PLTs (10*9 /L)	250.70 ± 54.20	258.72 ± 55.29	0.514
ESR (mm/h)	26.0 ± 5.62	7.5 ± 1.51	0.000*
CRP (mg/dl)	21.0 ± 4.56	5.7 ± 1.02	0.000*
Total bilirubin (umol/L)	6.15 ± 1.23	5.80 ± 1.11	0.180
Direct bilirubin	2.88 ± 0.10	3.14 ± 0.38	0.277
Total protein (g/L)	65.63 ± 5.48	69.57 ± 8.27	0.014*
Albumin (g/L)	33.18 ± 4.65	41.63 ± 4.73	0.014*
ALT (U/L)	16.69 ± 3.95	33.35 ± 4.71	0.000*
AST (U/L)	28.60 ± 5.74	33.53 ± 6.83	0.001*
ALP (U/L)	68.90 ± 7.76	65.22 ± 5.94	0.333
RBS (mmol/L)	4.99 ± 1.1	5.54 ± 1.4	0.112
Triglycerides (mg/dl)	139.5 ± 30.35	120.0 ± 25.64	0.014*
Cholesterol (mg/dL)	170.5 ± 39.81	141.5 ± 33.65	0.002*
HDL (mg/dL)	51.0 ± 11.51	60.0 ± 13.45	0.018*
LDL (mg/dL)	81.0 ± 18.83	68.0 ± 15.32	0.038*

Table (2): Clinical data of the studied IBD patient

	No. (n= 40)	%
Types of IBD:		
Crohn's disease.	15	37.5%
Ulcerative colitis.	25	62.5%
Activity state:		
Controlled	17	37.5%
Activity	23	62.5 %
Gastrointestinal manifestations:		
Bloody diarrhea and bleeding	23	62.5%
Abdominal pain	10	25%
Vomiting	5	12 %
Abdominal distention	13	32.5%
Severe colitis	23	62.5%
Duration of disease by years (Mean ± SD)	5.48 ± 1.55	
Treatment:		
Conventional	27	67.5%
Biological	13	32.5%
Dyslipidemia signs:		
Present	16	40.0%
Absent	24	60.0%

Table (3): Estimation ASCVD risk % among the studied population

ASCVD risk estimator %	Patients (n= 40)	Controls (n= 40)	P-value
	Median (Range)	Median (Range)	
Calculated risk 10 years	27.0 (0.3-46.0)	7.0 (0.3-46.0))	0.001*
Risk with optimal risk factors	11.0 (0.3-18.0)	5.0 (0.3-8.0)	0.004*

Table (4): Echocardiographic and carotid doppler parameters of the studied population.

	Patients (n= 40)	Controls (n= 40)	P-value
Pericardial fat: (mm)			
Median (Range)	2.15 (0.04-4.80)	1.43 (0.04-3.20)	0.027*
Diastolic function: No. (%)			
Normal	39 (97.5%)	40 (100.0%)	1.000
Dysfunction	1 (2.5%)	0 (0.0%)	
Ejection fraction:			
Mean ± SD	66.67 ± 6.51	65.34 ± 5.70	0.164
LVED: (cm)			
Mean ± SD	2.95 ± 0.88	2.71 ± 0.73	0.246
LVES:			
Mean ± SD	4.02 ± 0.87	3.88 ± 0.76	0.209
Segmental wall motion abnormality	2(5.0%)	0 (0.0%)	0.494
Valvular heart disease	2 (5.0%)	0 (0.0%)	0.494
Carotid US (IMT) right			
Median (Range)	0.08 (0.04-1.80)	0.05 (0.02-1.40)	0.032*
Carotid US (IMT) left			
Median (Range)	0.07 (0.06-1.70)	0.04 (0.03-1.30)	0.011*
Carotid Duplex: No. (%)			
Normal	33 (82.5%)	40 (100.0%)	0.012*
Abnormal	7 (17.5%)	0 (0.0%)	

IMT: intima-media thickness.

Table (5): Multivariate linear regression analysis for prediction of ASCVD in patients with IBD

	Unstandardized coefficients		Standardized coefficients	t	P-value	95.0% C.I. for B	
	B	SE	Beta			Lower	Upper
Hemoglobin (g/dl)	0.719	0.013	0.012	0.288	0.609	0.128	6.864
CRP (mg/L)	0.122	0.001	0.851	**11.510**	**0.002***	0.015	0.639
Cholesterol (mg/dL)	3.112	0.543	0.143	**2.369**	**0.016***	0.461	6.964
HDL (mg/dL)	0.508	0.011	0.921	**21.227**	**0.001***	0.281	0.927
Carotid US	3.721	0.802	0.658	**4.626**	**0.001***	2.078	5.347
Pericardial fat	2.514	0.620	0.572	**2.760**	**0.005***	1.006	6.321

P-value was significant if < 0.05

DISCUSSION

One-third of IBD patients experience symptoms in their eyes, skin, musculoskeletal system, and cardiovascular system. Recent findings suggest that systemic inflammation plays a role in the development of atherosclerosis in IBD patients, including ischemic heart disease, cerebrovascular accidents, mesenteric thrombosis, and venous thromboembolic events, as well as associated risk factors and mortality rates in IBD patients, and to further evaluate prospects and preventive factors. This result makes me want to investigate how atherosclerosis develops in persons with systemic inflammatory illness. However, research on IBD, which is characterized by persistent inflammation, has been limited [(11)]. In addition, several IBD-related treatments, such as corticosteroids, which elevate blood pressure and change glucose metabolism, as well as the avoidance of aspirin-containing medications (due to the danger of aggravating IBD), may increase the risk of ASCVD in IBD patients [(12)].

According to the current findings, 72% of the sample group was at risk for ASCVD within the next ten years. In our study, the estimated 10-year ASCVD risk percent among studied cases revealed that patients with IBD had a significantly higher estimated risk than healthy controls utilizing the Framingham risk score, which is a 10-year risk of ASCVD score based on age, hypertension, diabetes mellitus, tobacco use, and dyslipidemia [(13)].

This percentage is higher than in another study, with the first large study on the risk of IHD in IBD Canadian patients, conducted by **Bernstein *et al.*** [(14)], documenting a 26% increase in IHD risk in IBD patients compared to non-IBD individuals. This could be explained as we diagnosed patients later than these studies. Also, because we didn't have such expensive drugs, our patients received more corticosteroids with higher flare rates than patients receiving biological therapy, resulting in more ASCVD, as confirmed by **Singh *et al.*** [(15)], who demonstrated that corticosteroids, particularly when taken in high doses, increase the risk of ASCVD in IBD patients. The use of any oral steroids (current or prior) was associated with a 25% increased risk of heart failure and ASCVD but not stroke**.** Corticosteroids also have a significant effect on traditional ASCVD risk factors including hyperlipidemia, hyperglycemia, and hypertension [(16)].

This percentage is consistent with many studies in the region and other countries, such as the study by **Panhwar *et al.*** [(17)] which found that IBD patients had a significantly higher risk of developing a myocardial infarction than non-IBD patients. Further to that, a study conducted by **Yarur *et al.*** [(18)] revealed an increased risk of IHD in IBD patients, nearly threefold higher than the control. Similarly, **Rungoe *et al.* (2013)** reported a significantly increased risk of IHD in IBD patients compared to non-IBD individuals in a nationwide Danish population-based cohort study.

However, this was in contrast to a research conducted in the United States by **Ha *et al.*** [(19)], which encountered no overall increased risk of IHD in IBD patients. Also, a small Indian study published by **Biyyani *et al.*** [(20)] reported that the Framingham risk score was significantly lower in patients with IBD and coronary artery disease when compared to controls. One likely interpretation for these widely divergent results is that the aforementioned studies included study populations of varying ages, racial/ethnic distribution patterns, treatment categories, disease duration, disease activity, and dietary habits. Methodological issues in research project sample selection and inclusion criteria could also account for some of these differences.

A study on the risk of IHD in IBD by **Singh *et al.*** [(15)] observed a 19% increase in the risk of IHD in IBD patients, with the vulnerability being higher in the female gender; however, in our study, the incidence of ASCVD was lower in female patients, which may be attributed to the fact that they were receiving combined oral contraceptive (COC) pills, which leads to a lower risk of ASCVD, which mediates its cardioprotective actions by increasing angiogenesis and vasodilatation and decreasing ROS, estradiol E2 limits cardiac remodeling and reduces heart hypertrophy through these mechanisms.

Osterman *et al.* [(21)] observed an increased prevalence of ASCVD in UC patients, which was consistent with our findings that the prevalence of the different estimated 10-year ASCVD risk was more common in ulcerative colitis patients than in Crohn's disease patients. However, a study by **Tsai *et al.*** [(22)] concluded that young Crohn's disease patients have

more aggressive disease and a higher disease burden early in life. We believe that such discrepancies in the prevalence of comorbid conditions are due to differences in the samples analyzed and surveyed areas.

Carotid intima-media thickness is commonly cited as a good predictor of early atherosclerotic disease. **Wu *et al.*** [23] reported that patients with IBDs had significantly higher CIMT, similar to **Papa *et al.*** [24] who discovered increased CIMT in IBD patients, and **Dagli *et al.*** [25] who proposed that IBD is a risk factor for atherosclerosis. These previous studies are consistent with our results, which show a significantly higher prevalence of carotid intima-media thickness (CIMT) in the studied IBD population compared to the control population, which is evidenced by a significant positive correlation between carotid Duplex Doppler parameters and ASCVD risk estimator (p= 0.014*) in that population. This might be due to chronic low-grade inflammation during remission periods and bouts of increased inflammatory activity during the flare [16]**.** However, this is contradicted by a study by **Theocharidou *et al.*** [26], which encountered that CIMT did not vary among IBD patients or between disease activity and the treated group. This could be attributed to the fact that the disease duration of the patients enrolled was too short to affect CIMT, as **Maharshak *et al.*** [27] found that there was a debatable benefit to performing CIMT due to milder disease activity in the study population may explain the contradictory results.

The measurement of epicardial adipose tissue thickness may be a reliable indicator of atherosclerotic cardiovascular disease [17]. Subsequent studies have found a link between epicardial adipose tissue thickness and metabolic syndrome, as well as atherosclerosis. Increased epicardial fat quantity is accompanied by the occurrence of coronary heart disease and serious adverse cardiovascular outcomes, according to **Iacobellis *et al.*** [28]**.** In our study, pericardial fat was significantly higher compared to healthy controls, which is consistent with many other research findings, including one by **Bachar *et al.*** [29]**,** who discovered that epicardial fat thickness (EFT) correlated positively and strongly with coronary atherosclerosis. In addition, a recent study found a link between arterial stiffness and EFT, implying that echocardiographic EFT evaluation could be a simple tool for detecting subclinical atherosclerosis early on.

Biyyani *et al.* [30] demonstrated that total cholesterol and HDL-C levels were considerably lower, while low-density lipoprotein cholesterol (LDL-C) and triglyceride levels were substantially higher in male patients with IBD, which is supported by our findings, as is the significant association that we discovered between lipid profile changes and disease surrogate markers.

These study results, do not agree with Yusuf **Uysal *et al.*** [31], who published that there were no significant differences in serum levels of different cholesterol types, between IBD patients and controls. This could be attributed to the fact that our patients had more active disease for a longer time as chemokine production could affect lipoprotein metabolism and might in part be held responsible for lipid derangements in patients with IBD**.** Also, the presence of inflammatory cytokines such as tumor necrosis factor (TNF), interleukin-6 (IL-6), and interferon-Gama downregulate lipolytic enzyme activity, resulting in dyslipidemia [31]**.**

Furthermore, the distal ileum, which is responsible for bile acid absorption, is frequently affected, especially with CD. Lipoprotein changes in IBD can be explained by the loss of bile acids and cholesterol in the stool as a result of malabsorption [19]. So, lipid profiles in IBD are likely the result of a complicated combination of inflammatory cytokines, acute phase reactants, and intestinal integrity disruption (either surgical disruption or a functional disruption from severe inflammation). Given the low levels of HDL-C and elevated levels of LDL-C in individuals with IBD, a more aggressive strategy to profiling and treating dyslipidemia is necessary [31].

In our study, we discovered that there were significant direct correlations between epicardial fat thickness (EFT) and lipid profile, CIMT, and estimated ASCVD risk among IBD patients versus healthy controls. Furthermore, we discovered that EFT values in IBD patients were directly correlated with CRP levels and that there were independent associations between EFT, CRP, and IBD. A direct correlation, on the other hand, was discovered between EFT and CIMT values. Furthermore, patients with IBD who had carotid plaques had significantly higher EFT values than patients with IBD who did not have carotid plaques. These findings lend credence to the notion that dyslipidemia and atherosclerosis are linked and correlated with IBD activity.

There was a significant positive correlation between estimated ASCVD risk 10 years with clinical activity state and laboratory ESR and CRP among patients with IBD affection, which agreed with the study by **Kristensen *et al.*** [32] who reported a risk of myocardial infarction (MI) of IBD patients according to disease activity, whereas the risk was not increased during periods of remission, thereby supporting the hypothesis that chronic inflammation acts as a risk. However, **Uysal *et al.*** [31] found no significant difference in carotid intima-media and epicardial fat thickness values between active and inactive disease periods in both groups. This could be because long-term anti-TNF therapy reduces arterial and aortic stiffness in patients with IBD. This result corroborated our previous findings, **Zanoli *et al.*** [33]**,** as well as similar evidence reported in another chronic inflammatory disorder, **Vlachopoulos *et al.*** [34] It is unclear whether anti-TNF therapy improves functional (i.e., endothelial dysfunction) and/or structural arterial

stiffening (alterations in the arterial wall structure, intima-media thickness).

The findings of our study are noteworthy for a variety of reasons. To begin, we are conducting a national survey of people who have not yet developed ASCVD. Because of the higher prevalence of Cardiovascular Risk Factors (CRFs) in this population, it is a desirable admissible objective for cardiovascular disease primary prevention. Second, we encountered that the relation between IBD and CRFs was stronger in non-elderly participants than in the elderly. This observation may be clarified by the fact that the vast majority of IBD patients are diagnosed at a young age, and younger age is associated with a more progressive disease, indicating a greater inflammatory burden. Another reason is that the relative contribution of inflammation may lessen as the severity of symptoms declines and individuals who do not have IBD develop cardiovascular risk factors. As a result, early scanning and assertive risk factor management may benefit IBD patients. Third, the positive relationship between IBD and personal CRFs, as well as average/poor CRF profiles, suggests that cardiometabolic risk factors may be clustered in IBD patients. Further research is needed to investigate the complex interplay between IBD's chronic inflammatory state, the side effects of specific IBD therapies (e.g., corticosteroids), and CRFs. Chronic inflammatory conditions are considered a risk inducer by the European Society of Cardiology (ESC), prompting clinicians to seriously reconsider statin therapy in early-onset or intermediate-risk patients.

Our study limitations were mainly the small number of the study group. Also, long-term follow-up for IBD patients who have susceptibility to develop acute cardiac events.

CONCLUSION

The risk of ASCVD is raised in IBD patients, particularly during active disease. ASCVD in IBD patients favors non-traditional risk factors such as relatively young age and female gender. Estimating the thickness of epicardial adipose tissue (EAT) and carotid intima-media thickness (CIMT) could be used to screen for this risk in IBD patients.

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