SCIE DOI: 10.14744/scie.2023.22599
South. Clin. Ist. Euras. 2023;34(4):335-341

Original Article

Assessment of Cardiovascular Risk Parameters in Unipolar and Bipolar Depression

Burcu Kök Kendirlioğlu,[1] Ayse Ece Buyuksandalyaci Tunc,[1]
Hidayet Ece Arat Celik,[1] Esma Corekli Kaymakcı,[1] Elif Sude Erturan,[2]
Şevin Demir,[3] Suat Kucukgoncu[1]

[1]Department of Psychiatry, Maltepe University Faculty of Medicine, İstanbul, Türkiye
[2]Maltepe University Faculty of Medicine, İstanbul, Türkiye
[3]Department of Family Medicine, Maltepe University Faculty of Medicine, İstanbul, Türkiye

Submitted: 16.05.2023
Revised: 15.08.2023
Accepted: 16.08.2023

Correspondence: Burcu Kök Kendirlioğlu, Maltepe University Faculty of Medicine Department of Psychiatry, İstanbul, Türkiye

E-mail: kokburcu@gmail.com

Keywords: Atherogenic coefficient; atherogenic index of plasma; Castelli risk index; coronary artery disease; depression; lipid ratios.

This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

ABSTRACT

Objective: There is strong evidence that excess and early cardiovascular disease (CVD) occurs in patients with unipolar depression (UD) and bipolar disorder - depressive episode (BD-d). The aim of this study is to evaluate plasma atherogenic index (AIP), atherogenic coefficient (AC), Castelli risk index I-II (CRI-I and II) and high-density lipoprotein (HDL) related ratios in UD, BD-d and HC.

Methods: The present study was designed as a retrospective and observational study. This study included 128 patients with UD, 184 individuals as a healthy control group and 34 patients with BD-d. AIP [log (TG/HDL)], AC [(TC-HDL)/HDL], CRI-I (TC/HDL) and II (LDL/HDL), MHR (monocyte/HDL), NHR (neutrophil/HDL), PHR (platelet/HDL) and LHR (lymphocyte/HDL) were calculated in these three groups. ANCOVA was used for age- and sex-adjusted means.

Results: There were significant differences in fasting glucose (FG) (p=0.016), LDL (p=0.004), HDL (p<0.001), TG (p<0.001), TC (p=0.01), AIP (p<0.001), NHR (p<0.001), MHR (p=0.007), LHR (p<0.001), PHR (p<0.001) between three groups. All these metabolic parameters were correlated with duration of disorder.

Conclusion: Along with the care of a depressive episode, the management of abnormal metabolic parameters should be planned during diagnosis, follow-up, and treatment. Assessment of AIP and NHR, especially in chronic and unresponsive processes, may be useful in both disorders. In future studies, it is recommended to conduct large-scale studies that are prospectively designed, that monitor the severity of the disease, and that evaluate the CVD risk parameters and treatment of the participants.

INTRODUCTION

Depression is one of the top ten leading causes of years lived with disability in the world. Major depressive disorder (MDD) ranked first, schizophrenia ranked fifth, and bipolar disorder (BD) ranked sixth in terms of disability.[1] MDD also contributed to the burden associated with ischemic heart disease and suicide.[2] There is strong evidence that adults with MDD and BD experience excess and premature cardiovascular disease (CVD). Before the use of psychotropic medications, there was an increased and early onset of CVD mortality in patients with MDD and BD, showing that the disease itself raises the risk of CVD.[3]

The frequency of metabolic syndrome (MetS) is observed at a high rate in patients with mood disorders, up to 44% of people with MDD and 37.3% of people with BD.[4] There is a high likelihood that a variety of factors are involved. These include the iatrogenic side effects of psychotropic medications, an unhealthy lifestyle, inadequate medical care for the psychiatric patient, and genetic and pathophysiological susceptibility.[5] Many lipid ratios (also known as atherogenic indices) have been shown in numerous studies to be diagnostic alternatives that can accurately predict the likelihood of CVD events and the effectiveness of treatment when conventional lipid profiles

are normal. Atherogenic index of plasma (AIP), Castelli's Risk Index I and II (CRI-I and II), atherogenic coefficient (AC), and high-density lipoprotein (HDL)-related ratios are some of them. It was shown that these ratios can help predict various CVD events.[6]

The study reported that patients with MDD had higher serum total cholesterol (TC) levels than healthy controls (HCs). When patients with MDD were compared with HCs, dysregulated lipid levels were detected in patients with MDD. In terms of lipids, patients with MDD had significantly lower TC and very low-density lipoprotein (VLDL) levels despite higher triglyceride (TG) levels. However, it was not found significant differences between HDL and LDL values in studies when patients with MDD were compared with HCs.[7] It was shown that people with BD-depressive episode (BD-d) have lower levels of TG levels than people with unipolar depression (UD).[8] Compared to patients with MDD and BD-d, HC had lower LDL and TC levels.[9] According to the baseline results of the Rehabilitation According to an Initial Schizophrenia Episode study, total duration of psychiatric illness was strongly associated with increased body mass index (BMI), and waist circumference, but not with increased metabolic parameters except for the TG to HDL ratio in the first episode schizophrenia (FES). In contrast, duration of psychotropic medication was strongly associated with lower HDL levels and systolic blood pressure, and higher non-HDL cholesterol, TG, and TG to HDL cholesterol ratio.[10] Cardiometabolic risk factors and abnormalities are seen early in the course of FES and are probably related to the underlying disease, unhealthy lifestyle, and concomitant antipsychotic use.

AIP values were the highest in patients with BD-d compared with manic, euthymic, and HCs groups. AIP levels were inversely associated with the HDL and positively associated with the MetS, waist circumference, TC, LDL, and TG levels. Patients with BD showed an increase in cardiovascular risk during a depressed episode.[11] When compared with BD-manic episode (BD-m) patients and HCs, there is no significant difference between CRI-I, CRI-II, and AC values. However, AIP levels were higher in HCs in patients with BD-m.[12] The anti-inflammatory, antioxidant, and antithrombotic effects of HDL are significant, and it has the ability to block cytokine-induced production of endothelial cell adhesion protein.[13] In patients who had coronary artery disease, monocyte to HDL ratio (MHR) was found to be independently associated with coronary atherosclerosis.[14] Lymphocyte-to-HDL ratio (LHR) and neutrophil-to-HDL ratio (NHR) may become important markers with strong predictive potential for MetS, especially in women.[15]

MetS is more common in patients with psychiatric problems. This increased risk is caused by a complex network of mechanisms that work together to negatively influence the progression of psychiatric illness. MetS needs to be identified early and prevented. As mentioned above, lipid and hematological indicators can be potential inflammatory predictors of MetS, and these parameters are easily assessed from peripheral blood. Therefore, the aim of this study is to evaluate the association between AIP, AC, CRI-I, CRI-II, and HDL-related ratios (MHR, LHR, NHR, and PHR) in UD, BD-d, and HC.

MATERIALS AND METHODS

This study has a retrospective, observational, and descriptive design. This study included 128 patients with UD who were hospitalized at Maltepe University, Faculty of Medicine, Department of Psychiatry between 2016 and 2022. To compare the patient groups, 184 HCs were selected from people with no psychiatric diseases who presented for health screening at Maltepe University, Faculty of Medicine, Department of Family Medicine between 2020 and 2022. Data from 34 patients with Bipolar Depressive episode (BD-d) were included in the current study from a previously approved study by the Ethics Committee (Ethics Committee of Sisli Etfal Training and Research Hospital dated January 24, 2017, and decision number 745). Patients with BD-d who received a Hamilton Depression Score of 20 or above were included in the study. The diagnosis of UD and BD-d was made by an experienced psychiatrist according to the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). The study received ethical committee approval by the Clinical Research Ethics Committee of Maltepe University Faculty of Medicine (number: 2022/900/02 date: January 19, 2022).

Participants

Participants aged 18–69 years were included. All patient records were reviewed retrospectively. Blood samples from patients admitted to the psychiatric ward for UD and BD-d were taken within 24 h of admission. Individuals with a history of alcohol and substance use disorder, hypertension, diabetes mellitus, heart disease, autoimmune or inflammatory disease, cancer, active infection, and use of medications likely to affect the immune system were excluded from the study. In the HCs group, in addition to these criteria, individuals were excluded if they were suspected of having an acute or lifetime psychiatric disorder or alcohol or substance use disorder on the basis of a routine consultation with their general practitioner. The logarithm of the molar ratio of TG to HDL is represented by the AIP, a novel lipid ratio. The two lipid ratios known as Castelli's risk indices or cardiac risk indices are CRI-I and CRI-II, which measure the ratio of TC to HDL and LDL to HDL, respectively. The ratio of non-HDL cholesterol to HDL is known as the AC.[7] NHR, LHR, MHR, and PHR were computed by dividing neutrophils, lymphocytes, monocytes, and platelets by HDL.[16]

Statistical Analyses

SPSS for Windows statistical software, version 26 (SPSS Inc., Chicago, IL, USA), was used for all statistical analyses. To describe numerical data, means and standard de-

viations were employed, and to convey categorical data, frequencies and percentages were utilized. Categorical variables were compared using the Chi-square test. The Kolmogorov–Smirnov test was used to judge the distribution's normalcy. To compare continuous variables between groups, the Kruskal–Wallis test or analysis of variance with Bonferroni post hoc analysis was utilized. Non-parametric data were compared pairwise using the Mann–Whitney U-test. The Spearman's correlation test was used to assess linear connections between variables. For age- and sex-adjusted means, analysis of covariance (ANCOVA) was utilized.

RESULTS

Data from 128 patients with UD (85 women and 43 men, 34 patients with BD-d (34 men), and 184 HCs (105 women and 79 men) participated in the study. The mean age was 42.9±10.7 years in the HC, 40.3±14.9 years in the UD, and 41.2±10.2 years in the BD-d group. It was not found any significant differences in age between the three groups (p=0.178).

It was determined significant differences in fasting glucose (FG) (p=0.016), LDL (p=0.004), HDL (p<0.001), TG (p<0.001), TC (p=0.01), AIP (p<0.001), CRI-I (p=0.038), AC (p=0.038), NHR (p<0.001), MHR (p=0.007), LHR (p<0.001), and PHR (p<0.001) between three groups. After Bonferroni correction, differences disappeared in CRI-I and AC. While evaluating the metabolic parameters of participants, age- and sex-adjusted means are shown in Table 1. Pairwise comparisons after Bonferroni correction between the groups are written in Table 2. There is a significant correlation between the total number of depressive episodes and MHR, p=0.023 (cc: −.184, n=153).

Duration of disorder in UD was 10.6±8.7 years (1–40 years) and in BD-d was 17.4±9.6 years (3–40 years). The total number of depressive episodes in UD was 5.2±1.9 (1–10). Total number of mood episodes in BD-d was 9.8±6.5 and total number of depressive episodes was 4.9±3.7 (1–15). The correlation between duration of disorder and metabolic parameters is shown in Table 3. No relationship was found with other parameters.

The patients in BD-d treated with lithium carbonate (62.9%), valproic acid (28.6%), carbamazepine (2.9%), lamotrigine (17.1%), antipsychotics (94%), and antidepressant (37.1%). We do not have any data about the treatment of patients with UD before hospitalization.

DISCUSSION

The primary result of this research is that there were significant differences in AIP, LHR, NHR, PHR, LDL, HDL, and TC when comparing HC group with UD and BD-d patients. Significant differences were found in MHR values between the HC and UD groups. No differences in CRI-I, CRI-II, and AC were found among the three groups. All these metabolic parameters were correlated with the duration of the disorder.

Previous studies have reported mixed results on metabolic parameters between HC, UD, and BD-d. Some researchers reported no differences in TC, HDL, LDL, and TG levels between those groups.[17,18] According to the other studies, TG was higher, and HDL was lower in pa-

Table 1. Descriptive and metabolic parameters of participants

	Unipolar Depression (n=128) mean±SD	Bipolar Depression (n=34) mean±SD	Healthy Control (n=184) mean±SD	p	Test Statistic (F)
Sex (n-%)					
(male)	43- 33.6%	34 – 100%	79 – 42.9%		
Age (years)	40.3±14.9	41.2±10.2	42.9±10.7	0.178	1.734
FG (mg/dL)	102.06±1.62	92.68±3.1	97.6±1.24	0.016**	4.203
LDL (mg/dL)	123.107±3.78	110.31±7.24	133.36±2.91	0.004**	5.647
HDL (mg/dL)	46.85±1.23	47.25±2.36	54.79±0.95	<0.001*	15.043
TG (mg/dL)	149.42±8.71	181.69±16.68	118.49±6.7	<0.001*	8.539
TC (mg/dL)	201.03±4.11	189.89±7.8	211.87±3.16	0.010**	4.657
AIP	0.44±0.02	0.51±0.05	0.28±0.02	<0.001*	14.49
CRI- I	4.58±0.14	4.70±0.27	4.18±0.11	0.038**	3.292
CRI- II	2.82±0.11	2.77±0.22	2.66±0.08	0.570	0.563
AC	3.58±0.14	3.70±0.27	3.18±0.11	0.038**	3.292
NHR	99.72±4.26	110.02±8.20	82.30±3.29	<0.001*	8.505
MHR	13.91±0.56	14.27±1.07	11.93±0.43	0.007*	5.036
LHR	57.39±2.24	65.02±4.31	49.17±1.73	<0.001*	8.439
PHR	5628±190.04	6007.03±365.27	4833.57±146.69	<0.001*	8.340

FG: fasting glucose; LDL: low-density lipoprotein; HDL: high density lipoprotein; TG: triglyceride; TC: total cholesterol; NHR: neutrophil/HDL; MHR: monocytes/HDL; LHR: lymphocyte/HDL; PHR: platalet/HDL; AIP: Aterogenic Index Plasma; CRI-I: Castelli Index I; CRI-II: Castelli Index II; AC: Aterogenic Coeffient. Age- and sex-adjusted means were written. *: p≤0.001, **: <0.05.

Table 2. Pairwase comparisons between the groups

	Healthy Control vs. Unipolar Depression	Healthy Control vs. Bipolar Depression	Unipolar Depression vs. Bipolar Depression	N
AIP	<0.001*	<0.001*	0.820	333
CRI-I	0.091	0.245	1.000	335
CRI-II	0.906	1.000	1.000	335
AC	0.091	0.245	1.000	335
MHR	0.016**	0.139	1.000	337
LHR	0.012**	0.002*	0.380	337
NHR	0.004**	0.006*	0.834*	337
PHR	0.003**	0.010*	1.000	337
FG	0.375	0.014**	0.219	341
LDL	0.012**	<0.001*	0.078	338
HDL	0.005**	<0.001*	<0.001*	337
TG	0.136	<0.001*	0.002**	335
TC	0.016**	0.001*	0.213	337

FG: fasting glucose; LDL: low-density lipoprotein; HDL: high density lipoprotein; TG: triglyceride; TC: total cholesterol; NHR: neutrophil/HDL; MHR: monocytes/HDL; LHR: lymphocyte/HDL; PHR: platalet/HDL; AIP: Aterogenic Index Plasma; CRI-I: Castelli Index I; CRI-II: Castelli Index II; AC: Aterogenic Coeffient. ANCOVA was used and significant values were adjusted by the Bonferroni correction for multiple tests. *: p≤0.001, **: <0.05

tients with BD-d than in patients with UD.[19] In comparison to the general population, both BD and UD patients with a current depressive episode had higher levels of FG, TC, and LDL and lower levels of HDL.[20] In the present study, TG may serve as the discriminator between the three groups. Compared to HCs, HDL was decreased in both illness groups. This may be related to the lack of physical activity that occurs in depression. In addition, the fact that all patients are in a chronic course and use mood stabilizers, antidepressants, or antipsychotics for a long time may cause a deterioration of lipid parameters when assessing the duration of the illness.

The MHR ratio was predicted as a differential descriptive value for BD-d patients compared to HCs.[16] The negative correlation of MHR with the number of depressive episodes is a remarkable finding of the present study. This finding needs re-evaluation in future studies. While comparing BD-m, BD-d, and HC, there were differences in NHR, MHR, LHR, and PHR.[16] In the present study, NHR may be said to be a state marker for BD-d and UD. Considering that LHR and NHR have been suggested as strong predictors of MetS.[15] We can suggest that PHR may be important as a metabolic parameter in addition to these two parameters in this study.

Compared to BD-d and HC, UD had significantly higher Castelli's risk scores I and II. There were no significant differences in TC, LDL, TG, and FG between UD, BD-d, and HC.[21] In another study, it is not recommended to use CRI I and CRI II as indicators of mortality in patients with myocardial infarction. In addition, these indices do not predict the severity of coronary artery disease.[22] Although there was a significant difference in LDL, HDL, and TC between the healthy group and the patients, there was no difference between the CRI-I and CRI-II values; this may be related to the insufficient number of patients or CRI-I and CRI-II may not reflect metabolic abnormality in UD and BD-d.

For both depression and BD, the AIP and AC indices were significantly elevated in patients with mood disorders compared to controls.[23] AIP could be recommended as a potential biomarker for predicting CVD events in developing countries.[24] In the present study, similar results were obtained for AIP. Physical activity lowers cardiovascular risk by several pathways, including weight loss, enhanced endothelium and immunological function, and reduced blood pressure. Those who have UD or depressed symptoms are more likely to be sedentary than those who do not have a mood disorder. The AIP score can be used to assess cardiovascular risk for both UD and BD-d.

The fronto-striatal-limbic circuit is altered in BD, UD,

Table 3. The corelation between duration of disorder and metabolic parametres

	HDL	TG	AIP	CRI-I	CRI-II	AC	LHR	MHR	NHR	PHR
Duration of Disorder (p)	0.003**	0.001*	0.001*	0.001*	0.003**	0.001*	0.006**	0.018**	0.029**	0.029**
Correlation Coeffient	-.249	.279	.282	.283	.255	.283	.232	.201	.186	.187

Sperman Correlation test was used. HDL: high density lipoprotein; TG: triglyceride; LHR: lymphocyte/HDL; MHR: monocytes/HDL; PHR: platalet/HDL; NHR: neutrophil/HDL; AIP: aterogenic index; CRI-I: castelli risk index I; CRI-II: castelli risk index II; AC: aterogenic coeffient; *: p≤0.001, **: <0.05.

and obesity, which are multisystemic disorders marked by abnormalities in the inflammatory, metabolic, and endocrine systems.[25] These syndromes are considered as two different sides of inflammation, mainly because they are affected by common inflammatory processes. In addition to adipokines (cytokines secreted by adipocytes) such as adinopectin, leptin, and resistin, changes in cytokines such as TNF-α, IL-6, and IL-1 have also been reported in MetS.[26] The satiety hormone leptin shares structural similarities with pro-inflammatory cytokines. While leptin increases Th1 cytokine secretion, it inhibits Th2 cytokine production. According to epidemiological data, those with a lifetime history of depression have elevated blood leptin levels, and those with high serum leptin levels are more likely to experience major depressive episodes over the course of 5 years.[27] Some recent studies showed that resistin concentrations in patients diagnosed with major depression are associated with BMI. In addition, it has been noted that antidepressants lower resistin levels in persons with remission.[28] They influence brain activity, inflammatory alterations, and insulin resistance, which have all been proposed as shared mechanisms for the development of MetS and mood disorders.[25] In a study suggesting that ischemia-modified albumin (IMR) value can predict metabolic risk in patients with BD, unipolar disorder, and schizophrenia, the relationship of IMR with oxidative stress was emphasized and it was suggested that disease process and MetS may have a common physiopathology, especially in BD.[29] We can interpret the AIP, NHR, LHR, and PHR values as an indication of increased metabolic and cardiovascular risk for both BD-d and UD. Factors such as drug use and sedentary lifestyle alone cannot explain why these values are higher in patients. The pathophysiology of UD and BD-d may be similar to that of MetS. The need to monitor both processes simultaneously is supported by the fact that there is a negative association between disease duration and HDL and a positive association with all other metabolic markers. Treatment of metabolic abnormalities and promotion of physical activity should be included in the management of depression.

When depressed patients were divided into obese and non-obese groups, it was found that the obese group had more depressive episodes and an increased risk of recurrence.[30] In the current study, only a significant relationship was found between the number of depressive episodes and MHR. However, metabolic parameters other than HDL were positively associated with the duration of the illness, suggesting that the chronicity of depression can be assessed by means of metabolic parameters.

The main limitation of the study is its retrospective design. The fact that the severity of depression in the patients could not be assessed using a scale is another important limitation. Although the fact that the patients with BD-d included only male participants made the evaluation difficult, ANCOVA was used to remove the gender effect in the analysis. We did not have sufficient data on the treatment of patients with UD before hospitalization is another limiting factor. The lack of assessment of participants' MetS parameters, such as height, weight, BMI, waist circumference, and arterial blood pressure, may have been insufficient to assess the validity of these new metabolic parameters.

Conclusion

Our study is one of the first to evaluate all the new metabolic parameters in both UD and BD-d. Our findings suggest that AIP, NHR, LHR, and PHR should be monitored in these disorders. Along with the care of a depressive episode, the management of abnormal metabolic parameters should be planned during diagnosis, follow-up, and treatment. Assessment of AIP, especially in chronic and unresponsive processes, may be useful in both disorders. In future studies, it is recommended to conduct large-scale studies that are prospectively designed, monitor disease severity, and evaluate participants' MetS parameters and treatments.

Ethics Committee Approval

This study approved by the Maltepe University Faculty of Medicine Clinical Research Ethics Committee (Date: 19.01.2022, Decision No: 2022/900/02).

Informed Consent

Retrospective study.

Peer-review

Externally peer-reviewed.

Authorship Contributions

Concept: B.K.K., S.K., A.E.B.T., H.E.A.Ç.; Design: B.K.K., A.E.B.T., H.E.A.Ç.; Supervision: Ş.D., S.K., B.K.K.; Fundings: B.K.K., A.E.T., H.E.A.Ç.; Materials: E.Ç.K., E.S.E.; Data: E.Ç.K., E.S.E.; Analysis: B.K.K., H.E.A.Ç., A.E.B.T.; Literature search: F.Ç.K., E.S.E., Ş.D., S.K.; Writing: B.K.K., A.E.B.T., H.E.A.Ç.; Critical revision: S.K., Ş.D.

Conflict of Interest

None declared.

REFERENCES

1. Murray CJ, Vos T, Lozano R, Naghavi M, Flaxman AD, Michaud C, et al. Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990-2010: A systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380:2197–223. [CrossRef]
2. Ferrari AJ, Charlson FJ, Norman RE, Patten SB, Freedman G, Murray CJ, et al. Burden of depressive disorders by country, sex, age, and year: Findings from the global burden of disease study 2010. PLoS Medicine 2013;10:e1001547. [CrossRef]
3. Goldstein BI, Carnethon MR, Matthews KA, McIntyre RS, Miller GE, Raghuveer G, et al. Major depressive disorder and bipolar disorder predispose youth to accelerated atherosclerosis and early cardiovascular disease: A scientific statement from the American Heart Association. Circulation 2015;132:965–86. [CrossRef]
4. Vancampfort D, Stubbs B, Mitchell AJ, De Hert M, Wampers M, Ward PB, et al. Risk of metabolic syndrome and its components in people with schizophrenia and related psychotic disorders, bipolar disorder and major depressive disorder: A systematic review and

meta-analysis. World Psychiatry 2015;14:339–47. [CrossRef]
5. Ozpercin PU, Kendirlioglu BK, Sozen S, Yuksel O, Cihnioğlu R, Kalelioglu T, et al. Decreased circulating urokinase plasminogen activator receptor (uPAR) concentration in acute episodes of bipolar disorder; could it be a reflection of axonal injury? Psychoneuroendocrinology 2018;90:122–6. [CrossRef]
6. Abid H, Abid Z, Abid S. Atherogenic indices in clinical practice and biomedical research: A short review. Baghdad J Biochem Appl Biol Sci 2021;2:60–70. [CrossRef]
7. Bharti V, Bhardwaj A, Hood K, Elias DA, Metcalfe AW, Kim JS. A systematic review and meta-analysis of lipid metabolomic signatures of major depressive disorder. J Psychiatr Res 2021;139:197–205. [CrossRef]
8. Liu Y, Zhang X, Wang P, Yang M, Li N. Comparison of clinical features, serum lipid, and uric acid levels in patients with unipolar depression and bipolar depression. 2022;32:313–9. [CrossRef]
9. Erzin G, Yüksel RN, Usta MB, Topçuoğlu C, Örsel S. Assessment of irisin levels in patients with bipolar depression and unipolar depression. Ann Clin Anal Med 2020;11:583–6. [CrossRef]
10. Correll CU, Robinson DG, Schooler NR, Brunette MF, Mueser KT, Rosenheck RA, et al. Cardiometabolic risk in patients with first-episode schizophrenia spectrum disorders: Baseline results from the RAISE-ETP study. JAMA Psychiatry 2014;71:1350–63. [CrossRef]
11. Kalelioğlu T, Ünalan P, Kök B, Sözen Ş, Yüksel Ö, Akkuş M, et al. Atherogenic index of plasma as a cardiovascular risk marker in manic, depressive, and euthymic stages of bipolar disorder. Turk Kardiyol Dern Ars 2018;46:32–8. [CrossRef]
12. Kalelioglu T, Genc A, Karamustafalioglu N, Emul M. Assessment of cardiovascular risk via atherogenic indices in patients with bipolar disorder manic episode and alterations with treatment. Diabetes Metab Syndr 2017;11:473–5. [CrossRef]
13. Cockerill GW, Rye K-A, Gamble JR, Vadas MA, Barter PJ. High-density lipoproteins inhibit cytokine-induced expression of endothelial cell adhesion molecules. Arterioscler Thromb Vasc Biol 1995;15:1987–94. [CrossRef]
14. Akboga MK, Balci KG, Maden O, Ertem AG, Kirbas O, Yayla C, et al. Usefulness of monocyte to HDL-cholesterol ratio to predict high SYNTAX score in patients with stable coronary artery disease. Biomark Med 2016;10:375–83. [CrossRef]
15. Chen T, Chen H, Xiao H, Tang H, Xiang Z, Wang X, et al. Comparison of the value of neutrophil to high-density lipoprotein cholesterol ratio and lymphocyte to high-density lipoprotein cholesterol ratio for predicting metabolic syndrome among a population in the southern coast of China. Diabetes Metab Syndr Obes 2020;13:597–605. [CrossRef]
16. Wei Y, Wang T, Li G, Feng J, Deng L, Xu H, et al. Investigation of systemic immune-inflammation index, neutrophil/high-density lipoprotein ratio, lymphocyte/high-density lipoprotein ratio, and monocyte/high-density lipoprotein ratio as indicators of inflammation in patients with schizophrenia and bipolar disorder. Front Psychiatry 2022;13:941728. [CrossRef]
17. Zhang SF, Chen HM, Xiong JN, Liu J, Xiong J, Xie JZ, et al. Comparison of cognitive impairments with lipid profiles and inflammatory biomarkers in unipolar and bipolar depression. J Psychiatr Res 2022;150:300–6. [CrossRef]
18. Shapiro LR, Kennedy KG, Dimick MK, Goldstein BI. Elevated atherogenic lipid profile in youth with bipolar disorder during euthymia and hypomanic/mixed but not depressive states. J Psychosom Res 2022;156:110763. [CrossRef]
19. Wysokiński A, Strzelecki D, Kłoszewska I. Levels of triglycerides, cholesterol, LDL, HDL and glucose in patients with schizophrenia, unipolar depression and bipolar disorder. Diabetes Metab Syndr 2015;9:168–76. [CrossRef]
20. Moreira FP, Jansen K, de Azevedo Cardoso T, Mondin TC, da Silva Magalhaes PV, Kapczinski F, et al. Metabolic syndrome in subjects with bipolar disorder and major depressive disorder in a current depressive episode: Population-based study: Metabolic syndrome in current depressive episode. J Psychiatr Res 2017;92:119–23. [CrossRef]
21. Vargas HO, Nunes SOV, Barbosa DS, Vargas MM, Cestari A, Dodd S, et al. Castelli risk indexes 1 and 2 are higher in major depression but other characteristics of the metabolic syndrome are not specific to mood disorders. Life Sci 2014;102:65–71. [CrossRef]
22. Drwiła D, Rostoff P, Nessler J, Konduracka E. Prognostic value of non-traditional lipid parameters: Castelli Risk Index I, Castelli Risk Index II, and triglycerides to high-density lipoprotein cholesterol ratio among patients with non-ST-segment elevation myocardial infarction during 1-year follow-up. Kardiologiia 2022;62:60–6. [CrossRef]
23. Nunes SOV, de Melo LGP, de Castro MRP, Barbosa DS, Vargas HO, Berk M, et al. Atherogenic index of plasma and atherogenic coefficient are increased in major depression and bipolar disorder, especially when comorbid with tobacco use disorder. J Affect Disord 2015;172:55–62. [CrossRef]
24. Fernández-Macías JC, Ochoa-Martínez AC, Varela-Silva JA, Pérez-Maldonado IN. Atherogenic index of plasma: Novel predictive biomarker for cardiovascular illnesses. Arch Med Res 2019;50:285–94. [CrossRef]
25. Soczynska JK, Kennedy SH, Woldeyohannes HO, Liauw SS, Alsuwaidan M, Yim CY, et al. Mood disorders and obesity: Understanding inflammation as a pathophysiological nexus. Neuromolecular Med 2011;13:93–116. [CrossRef]
26. Bornstein S, Schuppenies A, Wong M, Licinio J. Approaching the shared biology of obesity and depression: The stress axis as the locus of gene-environment interactions. Molecular Psychiatr 2006;11:892–902. [CrossRef]
27. Pasco JA, Jacka FN, Williams LJ, Henry MJ, Nicholson GC, Kotowicz MA, et al. Leptin in depressed women: Cross-sectional and longitudinal data from an epidemiologic study. J Affect Disord 2008;107:221–5. [CrossRef]
28. Weber-Hamann B, Kratzsch J, Kopf D, Lederbogen F, Gilles M, Heuser I, et al. Resistin and adiponectin in major depression: The association with free cortisol and effects of antidepressant treatment. J Psychiatr Res 2007;41:344–50. [CrossRef]
29. Tunç S, Atagün Mİ, Neşelioğlu S, Bilgin YY, Başbuğ HS, Erel Ö. Ischemia-modified albumin: A unique marker of global metabolic risk in schizophrenia and mood disorders. Psychiatr Clin psychopharmacol 2019;29:123–9. [CrossRef]
30. Fagiolini A, Chengappa KR, Soreca I, Chang J. Bipolar disorder and the metabolic syndrome: Causal factors, psychiatric outcomes and economic burden. CNS Drugs 2008;22:655–69. [CrossRef]

Unipolar ve Bipolar Depresyonda Kardiyovasküler Risk Parametrelerinin Değerlendirilmesi

Amaç: Unipolar depresyon (UD) ve bipolar bozukluk - depresif dönem (BB-d) hastalarında erken kardiyovasküler hastalık (KVH) oluştuğuna dair güçlü kanıtlar vardır. Bu çalışmanın amacı, UD ve BD-d'de plazma aterojenik indeks (AIP), aterojenik katsayı (AC), Castelli risk indeksi I-II (CRI-I ve II) ve yüksek yoğunluklu lipoprotein (HDL) ile ilişkili oranları değerlendirmektir.

Gereç ve Yöntem: Retrospektif ve gözlemsel olarak tasarlanan bu çalışmaya 128 UD hastası, 184 sağlıklı kontrol grubu ve 34 BB-d hastası dahil edildi. AIP [log (TG/HDL)], AC [(TC-HDL)/HDL], CRI-I (TC/HDL) ve II (LDL/HDL), MHR (monosit/HDL), NHR (nötrofil/HDL), PHR (trombosit/HDL) ve LHR (lenfosit/HDL) değerleri hesaplandı. Yaş ve cinsiyete göre düzenlenmiş ortalamalar için ANCOVA kullanıldı.

Bulgular: Açlık glukozu (FG) ($p=0.016$), LDL ($p=0.004$), HDL ($p<0.001$), TG ($p<0.001$), TC ($p=0.01$), AIP ($p<0.001$), CRI-I ($p=0.038$), AC ($p=0.038$), NHR ($p<0.001$), MHR ($p=0.007$), LHR ($p<0.001$), PHR ($p<0.001$) değerleri arasında üç grup arasında anlamlı farklılık bulunmuştur. Bonferroni düzeltmesinden sonra, CRI-I ve AC'deki farklılıklar kaybolmuştur. Tüm bu metabolik parametreler hastalığın süresi ile korele idi.

Sonuç: Depresif epizodun tanısı, takibi ve tedavisi sırasında anormal metabolik parametrelerin yönetimi planlanmalıdır. AIP'nin ve NHR'nin özellikle kronik ve yanıtsız süreçlerde değerlendirilmesi her iki bozuklukta da yararlı olabilir. Gelecekteki çalışmalarda, prospektif olarak tasarlanmış, hastalığın ciddiyetini izleyen, katılımcıların KVH risk parametrelerini ve tedavilerini değerlendiren geniş ölçekli çalışmaların yapılması önerilmektedir.

Anahtar Sözcükler: Aterojenik katsayı; aterojenik plazma indeksi; Castelli risk indeksi; koroner arter hastalığı; depresyon; lipid oranları.