

Incidence of Metabolic Syndrome in Anti-Hepatitis C Virus Positive Patients

Mehmet Emirhan Işık,¹ Arzu Cennet Işık,² Semra Özgümüş,³
Ramazan Korkusuz,⁴ Sevtap Şenoğlu,⁴ Hayriye Esra Ataoglu⁵

¹Department of Infectious Diseases and Clinical Microbiology, University of Health Sciences, Koşuyolu High Specialty Training and Research Hospital, Istanbul, Turkey

²Department of Internal Medicine, University of Health Sciences, Kartal Dr. Lütfi Kırdar Training and Research Hospital, İstanbul, Turkey

³Department of Infectious Diseases and Clinical Microbiology, Izmit Seka State Hospital, Kocaeli, Turkey

⁴Department of Infectious Diseases and Clinical Microbiology, University of Health Sciences, Dr. Sadi Konuk Training and Research Hospital, İstanbul, Turkey

⁵Department of Internal Medicine, University of Health Sciences, Haseki Training and Research Hospital, İstanbul, Turkey

Submitted: 02.05.2020
Accepted: 01.07.2020

Correspondence:
Mehmet Emirhan Işık,
SBÜ Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Enfeksiyon Hastalıkları ve Klinik Mikrobiyoloji, Bölümü, İstanbul, Turkey
E-mail: emirhan82@gmail.com



Keywords: ATP and IDF criterias; hepatitis C; metabolic syndrome.



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

INTRODUCTION

Hepatitis C virus (HCV) infection is a common and serious health problem affecting people across the world. The global prevalence of HCV infection is 3%.^[1] This infection is one of the main causes of serious liver diseases, such as chronic liver disease, cirrhosis, and hepatocellular carcinoma. In contrast, metabolic syndrome (MS) is a fatal condition characterized by systemic disorders, such as coronary

artery disease (CAD), hypertension, dyslipidemia, diabetes mellitus or glucose intolerance, and abdominal obesity beginning with insulin resistance.^[2]

Dyslipidemia, a component of MS affects liver functions and causes fatty liver syndrome, fibrosis, and cellular destruction. Thus, this disorder is believed to cause more problems for people with HCV positivity and influence treatment response in patients who are scheduled to undergo treatment.^[3] Various trials have shown that advanced liver injury

ABSTRACT

Objective: Research has shown that hepatitis C infection, metabolic syndrome (MS), and non-alcoholic fatty liver syndrome are associated. This study was designed to assess the parameters of MS in people with hepatitis C antibodies.

Methods: We enrolled 104 patients, including 52 men and 52 women who visited our hospital and had anti-hepatitis C virus (HCV) positivity. In the patient group, different examinations were performed as per the MS criteria in the Adult Treatment Panel (ATP) III and International Diabetes Federation (IDF) guidelines.

Results: As per the ATP III criteria, the prevalence of MS was 36.5% (n=38) in the total population, 55.8% (n=29) in women, and 17.3% (n=9) in men in our study. When the IDF criteria were applied, the MS prevalence was 48.1% (n=50) in the total population, 65.4% (n=34) in women, and 30.8% (n=16) in men.

Conclusion: In our study, the prevalence of MS was significantly higher in female patients with HCV antibodies than in male patients.

may occur more easily in patients with non-alcoholic fatty liver disease (NAFLD) and other comorbidities.^[4] Central obesity, characterized by excess fat accumulation around the waist, is the most influential independent risk factor in the pathogenesis of NAFLD.^[5,6] In some non-obese patients with NAFLD, based on the body mass index calculations, central obesity, that shows a strong relationship with insulin resistance, is considered the most important finding of MS.^[6] In our study, MS parameters have been examined in people with HCV antibodies.

MATERIALS AND METHODS

One hundred and four patients who visited the polyclinics of our hospital in the period from January 2012 to August 2012 and had anti-HCV positivity were enrolled. The anti-HCV levels of the patients were examined using the ELISA (Triturus, Spain). Total 52 patients were men, and 52 were women. The waist circumference, height, and weight of all the patients were measured and recorded. Moreover, data regarding their age, sex, previous laboratory tests conducted at our hospital, preprandial blood glucose level, total cholesterol level, low-density lipoprotein (LDL) cholesterol level, and high-density lipoprotein lipase (HDL) cholesterol level, and triglyceride level were recorded. Information regarding the patients' medical histories was obtained; in addition, the health-related information of the patients and their families were recorded. Data on the presence of CAD, hyperlipidemia, hypertension, obesity, and diabetes mellitus were collected. During the physical examination, waist circumference, pulse rate, and arterial tension values were measured. Body weight was measured using a weighing scale that had a sensitivity of 0.1 kg on which the subjects stood wearing light clothing and no shoes. Further, waist circumference was measured with mild expiration on the plane passing through the spina iliaca anterior superior and the lower costa with the patient in the standing position.

The IDF and ATP criteria were used for establishing a diagnosis of MS. The ATP III criteria use abdominal obesity (waist circumference >102 cm for men and >88 cm for women), hypertriglyceridemia (triglyceride level \geq 150 mg/dL), reduced HDL (<40 mg/dL for men and <50 mg/dL for women), hypertension (blood pressure \geq 130/85 mmHg), and hyperglycemia (fasting blood glucose \geq 110 mg/dL) for MS diagnosis. As per the IDF criteria, at least two of the following factors must be present to establish a diagnosis of MS: abdominal obesity (waist circumference >94 cm for men and >80 cm for women), hypertriglyceridemia (triglyceride level \geq 150 mg/dL), reduced HDL cholesterol level (<40 mg/dL for men and <50 mg/dL for women), high blood glucose (fasting plasma glucose level \geq 100 mg/dL), and high blood pressure (\geq 135/80 mmHg).

We used the SPSS (Statistical Package for Social Sciences for Windows) 16.0 package program for statistical analyses; for numerical data, the average \pm standard deviation values were calculated, while for categorical data, percentages and frequencies were determined. Homogeneity of the distribution of the values in each group was examined using the Kolmogorov-Smirnov Z test. The Student T test was used for comparing normally distributed numerical data. The Mann-Whitney U test was used for paired comparisons of non-normal numerical data. Chi-Square test was used for evaluating the categorical variables. $p < 0.05$ or 95% confidence interval was considered to indicate statistical significance.

RESULTS

Hundred and four patients who visited the polyclinics at Haseki Research and Training Hospital and had anti-HCV positivity were enrolled. Fifty-two of the patients were men, and 52 of them were women. The average age of the patients was 55.79 ± 18.24 y. The prevalence of HbsAg was 2.9% (n=3) and that of HIV was 1% (n=1).

Table I. Clinical and laboratory levels of the patients with anti-HCV positivity (average [AVG] \pm Standard deviation [SD])

	Female	Male	General
Age	56.88 \pm 17.70	54.69 \pm 18.87	55.79 \pm 18.24
Waist circumference	84.98 \pm 8.71	88.87 \pm 8.32	86.92 \pm 8.70
Glucose	117.40 \pm 49.18	117.52 \pm 69.17	117.46 \pm 59.72
Triglyceride	130.12 \pm 59.90	118.90 \pm 60.01	119.52 \pm 60.61
HDL cholesterol	45.00 \pm 13.29	32.75 \pm 12.69	41.88 \pm 13.31
LDL cholesterol	104.75 \pm 33.43	92.60 \pm 41.48	98.67 \pm 37.98
ALT	44.15 \pm 44.46	50.81 \pm 58.46	47.48 \pm 51.79
AST	48.27 \pm 49.07	54.10 \pm 58.24	51.18 \pm 53.67
ALP	99.50 \pm 82.06	91.21 \pm 38.26	95.36 \pm 63.84
GGT	55.06 \pm 55.73	52.88 \pm 62.27	53.97 \pm 58.81
HbA1c	5.89 \pm 1.58	6.01 \pm 1.95	5.95 \pm 1.77
Systolic blood pressure	122.88 \pm 12.57	121.15 \pm 11.65	122.02 \pm 12.09
Diastolic blood pressure	76.35 \pm 12.52	75.38 \pm 11.28	75.87 \pm 11.87

HDL: High-density lipoprotein; LDL: Low-density lipoprotein; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; ALP: Alkaline phosphatase; GGT: Gamma-glutamyl transferase; HbA1c: Hemoglobin A1c.

Table 2. Comparison of the patients with metabolic syndrome and the patients without metabolic syndrome according to the ATP criteria

	ATP	N	Average	SD	p
Age	None	66	53.33	19.951	0.048
	Available	38	60.05	14.060	
ALT	None	66	45.41	53.063	0.593
	Available	38	51.08	50.008	
AST	None	66	47.89	53.366	0.413
	Available	38	56.89	54.443	
ALP	None	66	94.65	64.938	0.883
	Available	38	96.58	62.752	
GGT	None	66	48.06	61.207	0.178
	Available	38	64.24	53.656	
Cholesterol	None	65	160.77	46.705	0.545
	Available	38	166.58	47.017	
LDL	None	66	96.21	37.474	0.386
	Available	38	102.95	38.984	
LDH	None	66	210.17	98.594	0.091
	Available	38	247.18	119.033	
HbA1c	None	66	5.564	1.3779	0.008
	Available	38	6.637	2.1535	

HDL: High-density lipoprotein; LDL: Low-density lipoprotein; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; ALP: Alkaline phosphatase; GGT: Gamma-glutamyl transferase; HbA1c: Hemoglobin A1c; SD: Standard deviation.

Waist circumference was 84.98 ± 8.71 cm in women and 88.87 ± 8.32 cm in men. The average waist circumference of the patients was 86.92 ± 8.70 cm. The mean fasting plasma glucose level was 117.46 ± 59.72 mg/dL for the total population, 117.40 ± 49.18 mg/dL for the female subjects and 117.52 ± 69.17 mg/dL for the male subjects. The average triglyceride level of the patients was 119.52 ± 60.61 mg/dL; the average HDL cholesterol level was 41.88 ± 13.31 mg/dL for the total population, 45.00 ± 13.29 mg/dL for the female population and 32.75 ± 12.69 mg/dL for the male population. The mean triglyceride level of the female subjects was 130.12 ± 59.90 mg/dL, while that of the male subjects was 18.90 ± 60.01 mg/dL. The average LDL cholesterol level was 98.67 ± 37.98 mg/dL for the total study population, 104.75 ± 33.43 mg/dL for the female subjects and 92.60 ± 41.48 mg/dL for the male subjects. The average systolic and diastolic blood pressure values for the study population were 122.02 ± 12.09 mmHg and 75.87 ± 11.87 mmHg, respectively.

Furthermore, in all patient groups, the ALT level was 47.48 ± 51.79 U/L IU/L, the AST level was 51.18 ± 53.67 U/L, the ALP level was 95.36 ± 63.84 U/L, the GGT level was 53.97 ± 58.81 U/L, and the HbA1C level was $5.95 \pm 1.77\%$. The prevalence of primary hypertension was 40.4% (n=42), while that of diabetes mellitus was 25% (n=26).

There was no significant difference in the age, ALT level, AST level, GGT level, ALP level, total cholesterol level, LDL cholesterol level, HDL cholesterol level, LDH level, and glycosylated hemoglobin (HbA1C) levels of patients who were classified as having or not having MS as per the

Table 3. Comparison of the patients with metabolic syndrome and the patients without metabolic syndrome according to the IDF criteria

	IDF	N	Average	SD	p
Age	None	54	51.74	20.967	0.016
	Available	50	60.16	13.654	
ALT	None	54	46.04	57.543	0.769
	Available	50	49.04	45.309	
AST	None	54	50.22	57.691	0.851
	Available	50	52.22	49.536	
ALP	None	54	87.00	57.120	0.167
	Available	50	104.38	69.858	
GGT	None	54	48.30	63.685	0.309
	Available	50	60.10	53.024	
Cholesterol	None	53	156.28	36.435	0.139
	Available	50	169.94	55.028	
LDL	None	54	93.39	27.768	0.141
	Available	50	104.38	46.204	
LDH	None	54	216.37	106.689	0.473
	Available	50	231.60	108.783	
HbA1c	None	54	5.300	0.7682	<0.001
	Available	50	6.664	2.2278	

HDL: High-density lipoprotein; LDL: Low-density lipoprotein; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; ALP: Alkaline phosphatase; GGT: Gamma-glutamyl transferase; HbA1c: Hemoglobin A1c; IDF: International Diabetes Federation.

ATP III criteria. As per the IDF criteria, only the HbA1C level of those with and without MS differed significantly ($p < 0.001$).

As per the ATP III criteria, 36.5% (n=38) of the patients, including 55.8% (n=29) women and 17.3% (n=9) men were diagnosed with MS. In contrast, according to the IDF criteria, 48.1% (n=50) of the total population, 65.4% (n=34) of the female population, and 30.8% (n=16) of the male population had MS.

DISCUSSION

Paul D. Berk, a famous hepatologist who has worked as the editor of the journal titled "Hepatology" for several years defines hepatitis C as "the virus that created hepatology".^[7] It differs from other hepatitis viruses in that hepatitis C is generally progresses asymptotically and causes cirrhosis and hepatocellular carcinoma; no immunoglobulin or vaccine that protects against this disease has been found, and the actual prevalence of the disease remains unknown owing to its asymptomatic disease course.^[8]

In our cohort of HCV infection patients, those with MS were significantly older. In various studies, the prevalence of MS has been shown to increase with age, with an on-going increase in the prevalence rates among middle-aged and elderly populations.^[3,9]

There was no significant difference in the prevalence of MS in our patients with HCV infection as compared to the prevalence in the METSAR data.^[10] However, the prev-

alence of MS in our female HCV infection patients was significantly higher when diagnosed as per the IDF criteria than that as per the ATP III criteria (65.4%–51.1% vs. 55.8%–39.6%, respectively).

In our study, the prevalence of MS was 36.5% as per the ATP criteria and 48.1% as per the IDF criteria. Moreover, the prevalence of MS in the female population was 55.8% (n=29), while that in the male population was 17.3% (n=9) as per the ATP III criteria. In contrast, the MS prevalence was 65.4% (n=34) in female subjects and 30.8% (n=16) in male subjects as per the IDF criteria. Based on the results of the METSAR (Metabolic Syndrome Prevalence Survey in Turkey) (2004), the MS prevalence in adults aged ≥20 y was 35%.^[10] In our study, the prevalence of MS was 41.1% in women and 28.8% in men.

The main effects of HCV on the liver are inflammation, carcinoma, and slow-progressing liver fibrosis that causes cirrhosis. HCV infection is defined as a systemic infection that influences lipid metabolism, oxidative stress, mitochondrial function, gene expression, and stimulation pathways. In an average 38% of HCV infection patients, at least one extrahepatic symptom is observed during the course of the disease.^[11]

The mortality and morbidity related to coronary heart disease have increased in patients in MS that includes various disorders, such as abdominal obesity, lipid metabolism disorders, hypertension, diabetes mellitus, insulin resistance with or without glucose intolerance, microalbuminuria, thrombosis, and inflammation tendency.^[12] Insulin resistance is associated with a tendency to develop characteristics of MS, such as dyslipidemia and hypertension. Moreover, it is a central metabolic disorder that leads to the development of type 2 diabetes. Hepatitis C infection can induce metabolic disorders even without causing liver fibrosis.^[13,14]

MS has reached epidemic proportions in our country. Moreover, it has been estimated that MS is present in 3 out of every 8 adults and is present in 53% of coronary disease patients. As per the TEKHARF study, the prevalence of MS has increased at the rate of 38% in the previous 10 y; this prevalence rate is 38% in men aged >30 y and 43% in women aged >30 y. Therefore, MS is present in about 9.2 million adults aged >30 y, and 5.3 million of these adults are women. In addition, MS, which is more prevalent in women, is believed to be responsible for 50% of all coronary heart diseases in Turkey.^[15] Insulin resistance plays a central role in MS and plays an important role in the fine line of treatment between thrombosis and fibrinolytic system.

As per the TURDEP-II results, the prevalence of DM was 13.7%. In our study, the rate of DM was 25% in both, women and men. The prevalence of diabetes was slightly lower in men than in women, and no significant difference was found between women and men.^[16]

In our study, the rate of HT was 40.4%. Three important studies have been conducted on this subject in our coun-

try TEKHARF study is the oldest one among these studies and includes long-term follow-up. In the TEKHARF study, the rate of HT was 33.7%.^[2] According to the study on the prevalence of hypertension in Turkey (PatenT), the rate of HT revised based on age and gender was 31.8%.^[17] In contrast, the rate of HT was 41.7% in the METSAR study that analyzed 4261 subjects to determine the prevalence of MS in Turkey.^[10] Our results are not significantly different from those reported previously.

According to an article published in the Journal of Viral Hepatitis in May 2012, although HCV infection was previously considered as a risk for diabetes, it was thought that the rapid increase in obesity prevented this risk in the development of diabetes.^[18]

Insulin resistance may result from steatosis caused by HCV infection. Intracellular lipid accumulation induces insulin resistance and initiates diabetes. Reduction in the amount of intracellular triglycerides improves insulin sensitivity.^[19]

Oliveira et al.^[20] assessed 125 patients with HCV genotype I and found that 21.6% of the patients had MS. Thus, MS was very prevalent in the group without obesity and type 2 diabetes and was related to hypertension, insulin resistance, abdominal obesity, and overweight. In addition, high GGT and increased plasma glucose were associated with female sex in patients with MS. In our study, female sex was considered to be a risk factor for the development of MS.

In the comprehensive National Health and Nutrition Examination Survey [NHANES III (1988–1994)] conducted by the United States of America, the revised odds ratio was 3.8 for diabetes mellitus in patients aged ≥40 y with HCV positivity.^[21] In the multivariable analysis of patients who had hepatitis C infection but not cirrhosis, the odds ratio was 4.3. Further, it was thought that the relationship between HCV infection and diabetes mellitus is independent of cirrhosis development.^[22] In a recent prospective, sectional study, the prevalence of diabetes mellitus in the controls was 14.5%, while that in patients with HCV positivity was 7.3%.^[23,24]

Oncül et al.^[25] showed that the serum insulin levels in patients with chronic HCV infection were higher than those in controls. In the general population, hepatitis C seropositivity and carotid artery plaque development are independently interrelated. Moreover, insulin resistance is a known risk factor for atherosclerosis.

As per the data of the United States Renal Data System (USRDS), HCV infection is defined as a risk factor in the development of diabetes mellitus after transplantation.^[23,24] The relationship between increased risk of diabetes mellitus after transplantation and the HCV infection is especially noted in patients treated with tacrolimus. In a retrospective study performed by Bloom et al.^[26] (2002), in patients without diabetes mellitus before transplantation, the prevalence of diabetes mellitus after renal transplantation was 39.4% in those with HCV positivity before transplantation and 9.8% in those with HCV negativity.

As per various studies that have shown that obesity negatively impacts the treatment response in chronic hepatitis C, there are many possible underlying mechanisms for the role of obesity in a compromised therapeutic response. Obesity may influence drug distribution, biotransformation, and drug excretion. It remains unclear whether liver damage or increase in viral replication caused by obesity is attributable to compromised immunity or another mechanism.^[27,28]

CONCLUSION

In our study, we compared patients who were diagnosed with MS and those who were not diagnosed with MS as per the IDF and ATP III criteria; we also assessed the prevalence of MS in patients with HCV infection. We could not find any difference between the frequency of metabolic syndrome and liver enzymes. The reason for this may be that we cannot access file records regarding HCV burden and stage of our patient group. There was no significant different in the prevalence of MS in our patients with HCV infection and that reported in the METSAR data.

Ethics Committee Approval

Nil.

Informed Consent

Retrospective study.

Peer-review

Internally peer-reviewed.

Authorship Contributions

Concept: M.E.I., H.E.A.; **Design:** M.E.I., A.C.I.; **Supervision:** M.E.I., H.E.A.; **Fundings:** M.E.I., S.Ö., R.K., S.Ş.; **Materials:** M.E.I., A.C.I., S.Ö., R.K., S.Ş.; **Data:** M.E.I., A.C.I., S.Ö., R.K., S.Ş.; **Analysis:** M.E.I., A.C.I., H.E.A.; **Literature search:** M.E.I., A.C.I., S.Ö., R.K., S.Ş.; **Writing:** M.E.I.; **Critical revision:** H.E.A.

Conflict of Interest

None declared.

REFERENCES

- Thomas DL, Ray SC, Lemon SM. Hepatitis C. In: Mandell GL, Bennett JE, Dolin R, editors. Principles and practice of infectious diseases. 6th Ed. Philadelphia: Elsevier Churchill Livingstone; 2005. p. 1950–81.
- Türkiye Endokrinoloji ve Metabolizma Derneği Metabolik Sendrom Çalışma Grubu. Metabolik Sendrom Kılavuzu 2009. Available at: http://temd.org.tr/admin/uploads/tbl_yayinlar/metabolik_sendrom.pdf. Accessed Dec 10, 2020.
- Cheng YL, Wang YC, Lan KH, Huo TI, Huang YH, Su CW, et al. Anti-hepatitis C virus seropositivity is not associated with metabolic syndrome irrespective of age, gender and fibrosis. Ann Hepatol 2015;14:181–9.
- Bang KB, Cho YK. Comorbidities and Metabolic Derangement of NAFLD. J Lifestyle Med 2015;5:7–13.
- Dassanayake AS, Kasturiratne A, Rajindrajith S, Kalubowila U, Chakravarthi S, De Silva AP, et al. Prevalence and risk factors for non-alcoholic fatty liver disease among adults in an urban Sri Lankan population. J Gastroenterol Hepatol 2009;24:1284–8.
- Scorletti E, Calder PC, Byrne CD. Non-alcoholic fatty liver disease and cardiovascular risk: metabolic aspects and novel treatments. Endocrine 2011;40:332–43.
- Berk PD. Introduction. Hepatitis C: The virus that created hepatology. Semin Liver Dis 2000;20:i–ii.
- Alexander CM, Landsman PB, Teutsch SM, Haffner SM; Third National Health and Nutrition Examination Survey (NHANES III); National Cholesterol Education Program (NCEP). NCEP-defined metabolic syndrome, diabetes, and prevalence of coronary heart disease among NHANES III participants age 50 years and older. Diabetes 2003;52:1210–4.
- Alter MJ, Kruszon-Moran D, Nainan OV, McQuillan GM, Gao F, Moyer LA, et al. The prevalence of hepatitis C virus infection in the United States, 1988 through 1994. N Engl J Med 1999;341:556–62.
- Kozan Ö, Oğuz A, Abacı A, Erol C, Öngen Z, Temizhan A, et al. Türkiye Metabolik Sendrom Prevalans Çalışması (METSAR) Sonuçları. II. Metabolik Sendrom Sempozyumu. İstanbul Mart 2005.
- Merle P, Trepo C. Treatment of extrahepatic diseases caused by hepatitis B and hepatitis C Viruses. In: Thomas H, Lemon S, Zuckerman A, editors. Viral Hepatitis. 3rd ed. New Jersey: Blackwell Publishing; 2005. p. 780–93.
- Chang ML. Metabolic alterations and hepatitis C: From bench to bedside. World J Gastroenterol 2016;22:1461–76.
- Bugianesi E, Salamone F, Negro F. The interaction of metabolic factors with HCV infection: does it matter? J Hepatol 2012;56:S56–S65.
- Hsu CS, Liu CH, Liu CJ, Hsu SJ, Chen CL, Hwang JJ, et al. Association of metabolic profiles with hepatic fibrosis in chronic hepatitis C patients with genotype 1 or 2 infection. J Gastroenterol Hepatol 2010;25:970–7.
- Onat A. TEKHARF çalışması 2009. Available at: <http://tekharf.org/>
- Satman I, Omer B, Tutuncu Y, Kalaca S, Gedik S, Dinccag N, et al; TURDEP-II Study Group. Twelve-year trends in the prevalence and risk factors of diabetes and prediabetes in Turkish adults. Eur J Epidemiol 2013;28:169–80.
- Altun B, Arıcı M, Nergizoglu G, Derici U, Karatan O, Turgan C, et al; Turkish Society of Hypertension and Renal Diseases. Prevalence, awareness, treatment and control of hypertension in Turkey (the PatenT study) in 2003. J Hypertens 2005;23:1817–23.
- Stepanova M, Lam B, Younossi Y, Srishord MK, Younossi ZM. Association of hepatitis C with insulin resistance and type 2 diabetes in US general population: the impact of the epidemic of obesity. J Viral Hepat 2012;19:341–5.
- Chou CJ, Haluzik M, Gregory C, Dietz KR, Vinson C, Gavrilova O, et al. WY14,643, a peroxisome proliferator-activated receptor alpha (PPAR α) agonist, improves hepatic and muscle steatosis and reverses insulin resistance in lipodystrophic A-ZIP/F-1 mice. J Biol Chem 2002;277:24484–9.
- Oliveira LP, Jesus RP, Boulhos RS, Mendes CM, Lyra AC, Lyra LG. Metabolic syndrome in patients with chronic hepatitis C virus genotype 1 infection who do not have obesity or type 2 diabetes. Clinics (Sao Paulo) 2012;67:219–23.
- Mehta SH, Brancati FL, Sulkowski MS, Strathdee SA, Szkoł M, Thomas DL. Prevalence of type 2 diabetes mellitus among persons with hepatitis C virus infection in the United States. Ann Intern Med 2000;133:592–9.
- Lecube A, Hernandez C, Genesca J, Esteban JI, Jardi R, Simo R. High prevalence of glucose abnormalities in patients with hepatitis C virus infection: A multivariate analysis considering the liver injury. Diabetes Care 2004;27:1171–5.

23. Zein CO, Levy C, Basu A, Zein NN. Chronic hepatitis C and type II diabetes mellitus: a prospective cross-sectional study. *Am J Gastroenterol* 2005;100:48–55.
24. Konrad T, Zeuzem S, Vicini P, Toffolo G, Briem, D, Lormann J, et al. Evaluation of factors controlling glucose tolerance in patients with HCV infection before and after 4 months therapy with interferon-alpha. *European Journal of Clinical Investigation* 2003;30:111–21.
25. Oncül O, Top C, Cavuplu T. Correlation of serum leptin levels with insulin sensitivity in patients with chronic hepatitis-C infection. *Diabetes Care* 2002;25:937.
26. Bloom RD, Rao V, Weng F, Grossman RA, Cohen D, Mange KC. Association of hepatitis C with posttransplant diabetes in renal transplant patients on tacrolimus. *J Am Soc Nephrol* 2002;13:1374–80.
27. Woodward RS, Schnitzler MA, Baty J, Lowell JA, Lopez-Rocafort L, Haider S, et al. Incidence and cost of new onset diabetes mellitus among U.S. wait-listed and transplanted renal allograft recipients. *Am J Transplant* 2003;3:590–8.
28. Lam NP, Pittrak D, Speralakis R, Lau A. Effect of obesity on pharmacokinetics and biologic effect of interferon- α in hepatitis C. *Digestive Diseases and Sciences* 1997;42:178–85.

Anti-HCV Pozitif Hastalarda Metabolik Sendrom Sıklığı

Amaç: Literatürde hepatit C enfeksiyonu, metabolik sendrom ve non-alkolik karaciğer yağlanması arasında ilişki olduğu görülmüştür. Çalışmamızda Hepatit C antikoru saptanan kişilerde metabolik sendrom parametreleri incelendi.

Gereç ve Yöntem: Çalışmaya, hastanemize başvuran ve anti HCV pozitifliği saptanan 52'si erkek ve 52'si kadın olmak üzere 104 hasta alındı. Hasta grubunda ATP III ve IDF'nin belirlediği metabolik sendrom kriterlerine göre ayrı ayrı inceleme yapıldı.

Bulgular: ATP kriterlerine göre metabolik sendrom oranı %36.5 (n=38) saptandı. Kadınlarda ATP kriterlerine göre metabolik sendromlu oranı %55.8 (n=29), erkeklerde bu oran %17.3 (n=9) bulundu. IDF kriterlerine göre ise tüm hastalar içinde metabolik sendromlu oranı %48.1 (n=50) olarak saptandı. Kadınlarda IDF kriterlerine göre metabolik sendromlu hasta oranı %65.4 (n=34) iken; erkeklerde bu oran %30.8 (n=16) bulundu.

Sonuç: Çalışmamızda HCV antikoru saptanan kadın hastalarda metabolik sendrom oranı erkek hastalara göre belirgin olarak yüksek saptanmıştır.

Anahtar Sözcükler: ATP ve IDF kriterleri; Hepatit C; metabolik sendrom.