RESEARCH PAPER

Thyroid function abnormalities in hepatitis C positive patients in a single center in Basrah

Amar A. Hussein[1], Alaa K. Mousa[2]

1. MBCHB, College of Medicine, University of Basrah, Iraq
2. Department of Medicine, College of Medicine, University of Basrah, Iraq

Received: 21.11.2021 **Accepted: 4.4.2022**

Abstract

Background: Hepatitis c virus (HCV) is global health problem it has many extrahepatic manifestations including hematologic, lymphoproliferative, endocrine, and renal diseases. The most common and important endocrine disorder are thyroid abnormalities.

The aim of this study: Is to investigate the correlation between HCV infection and thyroid function abnormalities and the factors that may affect it.

Patients and methods: This were a case-control study with 51 patients (27 males) were HCV positive who were attending Faiha Gastroenterology and Hepatology center and 49 HCV negative persons (34 males) from health workers and patients' relatives represent control group. They were sent for thyroid function test in form of Thyroid stimulating hormone (TSH), free tetraiodothyronine (FT4), total triiodothyronine (T3), anti-thyroid peroxidase antibody, thyroglobulin, and anti-thyroglobulin.

Results: thyroid function abnormalities were found in eighteen patients (35.3%) of HCV positive group ,and nine (18.4%)in control group but the difference was not significant ($p = 0.057$), also there was no difference in thyroid antibodies prevalence between the two groups ($p > 0.05$).The distribution of thyroid abnormalities in HCV patients was as follows: Four patients (7.8%) have hypothyroidism, Four (7.8%) have subclinical hypothyroidism, and Ten (19.7%) have weird thyroid function, while in control group: Four (8.2%) have subclinical hypothyroidism, Five(10.2%) have weird thyroid function and none have hyperthyroidism in both groups. Abnormal thyroid function was more in women with age group 30-40 years and was statistically significant.

Conclusion: Thyroid abnormalities are more in HCV positive patients where hypothyroidism is the most frequent, they are more in women, and more in 30-40 years old.

With the majority of hypothyroidism has autoimmune origin.

Keywords: Hepatitis c virus, thyroid abnormalities, thyroid antibodies.

Correspondence to: Amar A. Hussein, College of Medicine, University of Basrah, Iraq

✉ **E-mail: amaralaa@gmail.com**

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

Introduction

Hepatitis C virus (HCV),a single-strand RNA virus belong to Flaviviridae, is recognized as the causative agent of Hepatitis C.[1] According to the World Health Organization (WHO) 3 to 4 million people are newly infected with HCV every year and there are about 180 million patients infected with HCV in the world.[2] In Iraq, the prevalence of HCV is about 2.1%, 3.7%, 5.7% in Basrah, Karbala and Duhok respectively.[3-5] Approximately 70% of HCV patients will develop chronic hepatitis C (CHC) and around 20-35% of chronically infected patients progress to cirrhosis and have a higher risk of developing hepatocellular carcinoma.[6,7] Numerous extrahepatic manifestations have been reported in up to 74% of HCV patients[8-11] which include hematologic, lymphoproliferative disorders, endocrine disease, and renal diseases. The most common and important endocrine disorder are autoimmune thyroid disorders[12] which occur in about 7 to 20% of HCV patients before and after interferon alpha(IFN-α) treatment.[1316] It seems that INF-α triggers thyroiditis by both thyroid toxic mechanism and immune modulatory mechanism[12] A significant number of patients with CHC have been reported to develop biochemical thyroid dysfunction even without IFN-α treatment, the potential explanation proposed for this is either direct effect of HCV on thyroid cells, triggering thyroid autoimmunity by altering immune responsiveness, or both.[17] Recently reported, HCV can infect human thyroid cell in vitro[18] and has been detected in thyroid tissue from patients with CHC infection.[19] Liver plays an important role in the metabolism of thyroid hormones, as it is the most important organ in the peripheral conversion of tetraiodothyronine (T4) to triiodothyronine (T3) by type I deiodinase resulting to 5' deiodination of T4.[20] And also, it is involved in conjugation and circulation of thyroid hormones by synthesis of thyroid binding proteins.[21] A 17% increase in thyroid glandular volume in cirrhotic patients is reported when compared with controls[22] And, it is found that levels of thyroid hormones and their binding proteins are also altered in patients with hepatic disorders, especially in cirrhosis;[23] though, almost all are clinically euthyroid.[24] The most frequently observed change is decreased total T3 and free T3 concentration which is reported to be associated with the severity of hepatic dysfunction.[20,24,25]

The aim of this study is to investigate the correlation between HCV infection and thyroid function abnormalities and the factors that may affect it.

Patients and methods

Study design and population

This case-control study was conducted in Faiha Gastroenterology and Hepatology Center, Basrah, Iraq from July to October 2019. A total number of 100 person were enrolled in this study of which, 51 patients (27 male) were HCV positive who were attending Gastroenterology and Hepatology Center, after taking information regarding age, gender, duration since HCV diagnosis and treatment, whereas 49 HCV negative persons (34 male) from health workers and patients' relatives who were healthy with no disease represent the control group.

Inclusion criteria

Patients with HCV infection confirmed by PCR, with an age 10 and above, asymptomatic and having no signs of acute illness, chronic liver or thyroid disease, with normal liver ultrasound,

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

taking no medications apart from direct antiviral agent (DAA) in the form of (sofosbuvir / ledipasvir). They were categorized as new cases (patient who were just diagnosed with HCV), and old cases (patients who have been previously diagnosed with HCV and were coming for follow up visits).

Exclusion criteria

1. Patients with a preexisting thyroid disease, were taking medication for thyroid disease or having goiter and other clinical signs of hyper or hypothyroid.
2. Any patient taking medication that affect thyroid function such as amiodarone, steroid, furosemide, carbamazepine.
3. Patients with cirrhosis, or having signs of chronic liver disease.
4. Patients with other forms of hepatitis (hepatitis B virus, alcoholic and non-alcoholic fatty liver disease).
5. Pregnant women or taking oral contraceptive pills.
6. HCV patients taking INF-α.
7. Patients with acute illness.

Thyroid function

After informed verbal consent with explanation of the study, A 5 milliliter of blood withdrawn from each person, centrifuged and sent for liver function test (LFT), thyroid function test in the form of TSH, FT4, total T3, anti-thyroid peroxidase antibody (Anti-TPO), thyroglobulin (TG) and anti-thyroglobulin (Anti-TG) in Faiha Specialized Diabetes Endocrine and Metabolism Center. Analysis of the serum was done in the same day by (COBAS® e 411, Roche, Germany), and a normal range for TSH (0.27-4.2µIU/ml), freeT4(0.93-1.7ng/dl), T3(0.8-2ng/ml), Anti-TPO(0-34IU/ml), TG (1.4-78ng/ml), Anti TG(0-115IU/ml).

The results were categorized as the following(1):

1. Hyperthyroid: patients with elevated T3, FT4 and low TSH.
2. Hypothyroid: patients with elevated TSH, low FT4.
3. Subclinical hypothyroid: patients with elevated TSH, normal T3 and T4.
4. Euthyroid: patients with normal TSH, T3, T4.

And patients that cannot be categorized according to the previous list will be considered as weird thyroid function.

Statistics

- The analysis of data was carried out using IBM SPSS® Statistics version 25.
- Chi square test was used for analysis of frequencies.
- T test and Mann-Whitney U test to compare between groups.
- A p-value of less than 0.05 was considered statistically significant.

Results

(Table-1) shows the general characteristics of both groups. The mean age of our study participants was 31.04 ± 15.90, 34.08 ± 15.27 years in HCV positive patients and control group respectively. In HCV positive patients' group, 34 patients (66.7%) were newly diagnosed and not receive DAA, and 17 patients (33.3%) have duration since HCV diagnosis range from 1 to 5 months had receive DAA treatment. Fourteen patients (27.5%) had elevated liver enzyme.

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

Table 1. General characteristics of the patients with HCV and control. (N = 100)

	HCV = 51	Control = 49	*P* value
	M ± SD or No. (%)	M ± SD or N0. (%)	
Age (years)	31.04 ± 15.90	34.08 ± 15.27	0.332
Gender			
Male	27(52.9)	34(69.4)	0.092
Female	24(47.1)	15(30.6)	
Duration since diagnosis of HCV (months) New cases	34(66.7)	-----	
Old cases	17(33.3) 2.11 ± 1.31	-----	
Sofosbuvir / ledipasvir Yes	17(33.3)	-----	
No	34(66.7))	-----	
Liver enzymes Elevated	14(27.5)	-----	
Normal	37(72.5)	-----	
TSH*	1.70	1.90	0.845
FT4	1.1 ± 0.28	1.23 ± 0.69	0.191
T3	1.44 ± 1.12	1.55 ± 1.40	0.668

*** Median**

(Table-2), shows a numeric but not statistically significant excess of thyroid function abnormalities was seen in HCV patients 18 (35.3%) in comparison to nine (18.4%) in the control group, P = 0.057. It also shows that there is no significant difference in thyroid antibodies between the two groups (p > 0.05)

Table 2. Thyroid status in patients with HCV and controls. (N=100)

Thyroid status	HCV patients N = 51(%)	Control N = 49(%)	P value
Thyroid function test			
Normal	33(64.7)	40(81.6)	0.057
Abnormal	18(35.3)	9(18.4)	
Thyroid antibody			
Anti TPO Positive Negative	6(11.8) 45(88.2)	6(12.2) 43(87.8)	0.941
TG Positive Negative	3(5.9) 48(94.9)	0(0) 49(100)	0.243
Anti TG Positive Negative	6(11.8) 45(88.2)	5(10.2) 44(89.8)	0.803

The distribution of the abnormalities was as following: Four patients (14.8%) were hypothyroid and all of them were HCV positive patients, eight patients (29.6%) were subclinical hypothyroid half of those were HCV positive, and fifteen (55.6%) had weird thyroid function two third of them were HCV positive as showed in (Figure-1).

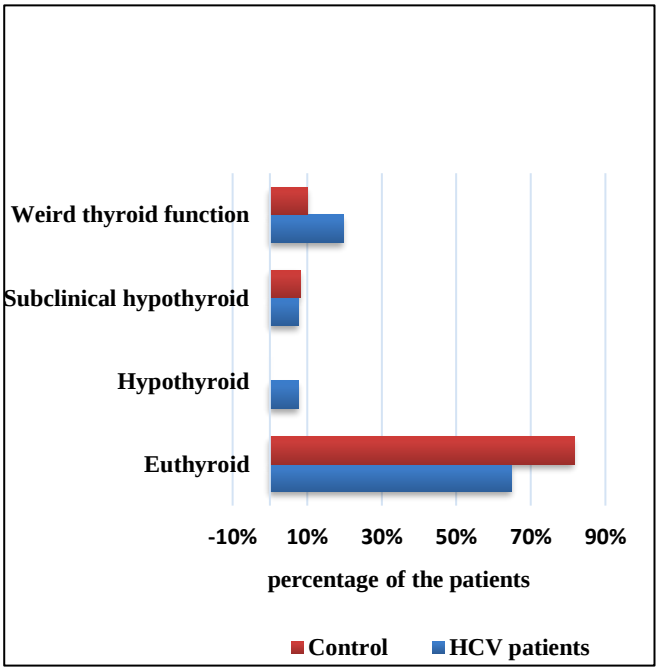

Fig 1. Thyroid function in HCV patients and control

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

(Table-3), showed that thyroid abnormalities significantly affected by age groups (P = 0.007) and were more in patients aged 30-40 years (55.6% of abnormal thyroid function), gender (p = 0.038) and were more in female (66.7% of abnormal thyroid function), while they were not significantly affected by duration since diagnosis, receiving treatment or whether liver enzymes were elevated or normal.

Table 3. Correlation between HCV patient's parameters and thyroid disorder N=51

Parameter	Thyroid function No. (%) or M ± SD		
	Normal N = 33(100)	Abnormal N = 18 (100)	P value
Gender			0.038
Male	21(63.6)	6(33.3)	
Female	12(36.4)	12(66.7)	
Age (years)	29.39 ± 18.33	34.06 ± 9.86	0.322
10-20	14(42.4)	3(16.7)	0.007
20-30	7(21.2)	1(5.5)	
30-40	4(12.1)	10(55.6)	
> 40	8(24.3)	4(22.2)	
Liver enzymes			0.537
Normal	23(69.7)	14(77.8)	
Elevated	10(30.3)	4(22.2)	
Sofosbuvir/ledipasvir			1.00
Yes	11(33.3)	6(33.3)	
No	22(66.7)	12(66.7)	
Duration since diagnosis of HCV			
New cases	22(66.7)	12(66.7)	1.00
Old cases	11(33.3)	6(33.3)	
Within old cases	2.36 ± 1.36	1.66 ± 1.21	0.312

(Table-4,5), represent the correlation between thyroid antibodies and different parameters and they show that there is strong association between anti TPO, anti TG and hypothyroidism (p = 0.005, p < 0.001 respectively), while they are not affected by other parameters.

Table 4. Correlation between patients' parameters and anti TG

Parameters	Anti TG M ± SD or N (%)		P value
	Positive N = 6(100)	Negative N = 45(100)	
Gender			0.088
Male	1(16.7)	26(57.8)	
Female	5(83.3)	19(42.2)	
Age(years)	36.17 ±7.46	30.36 ± 16.65	0.406
10-20	0(0)	17(37.8)	0.102
20-30	1(16.7)	7(15.6)	
30-40	4(66.7)	10(22.2)	
> 40	1(16.7)	11(24.4)	
Duration since diagnosis of HCV (months)			
New cases	4(66.7)	30(66.7)	1.00
Old cases	2(33.3)	15(33.3)	
Within old cases	1.00 ± 00	2.26 ± 1.33	0.212
Liver enzymes			0.109
Normal	6(100)	31(68.9)	
Elevated	0(0)	14(31.1)	
Sofosbuvir/ledipasvir			1.00
Yes	2(33.3)	15(33.3)	
No	4(66.7)	30(66.7)	
Thyroid function			
Euthyroid	1(16.7)	32(71.1)	<0.001
Hypothyroid	4(66.7)	0(0)	
Subclinical hypothyroid	0(0)	4(8.9)	
Weird thyroid function	1(16.7)	9(20.0)	

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

Table 5. Correlation between patients' parameters and anti TPO

Parameters	Anti TPO M ± SD or N (%)		P value
	Positive N = 6(100)	Negative N = 45(100)	
Gender			
Male	1(16.7)	26(57.8)	0.097
Female	5(83.3)	19(42.2)	
Age(years)	37.17 ± 16.29	30.22 ± 15.78	0.320
10-20	1(16.7)	16(35.6)	
20-30	1(16.7)	7(15.6)	0.817
30-40	2(33.3)	12(26.7)	
> 40	2(33.3)	10(22.2)	
Duration since diagnosis of HCV (months)			
New cases	4(66.7)	30(66.7)	
Old cases	2(33.3)	15(33.3)	1.00
Within old cases	2.00 ± 1.41	2.13 ± 1.35	0.898
Liver enzymes			
Normal	5(83.3)	32(71.1)	0.529
Elevated	1(16.7)	13(28.9)	
Sofosbuvir/ledipasvir			
Yes	2(33.3)	15(33.3)	1.00
No	4(66.7)	30(66.7)	
Thyroid function			
Euthyroid	2(33.3)	31(68.9)	
Hypothyroid	3(50.0)	1(2.2)	
Subclinical hypothyroid	1(16.7)	3(6.7)	0.005
Weird thyroid function	0(0)	10(22.2)	

Discussion

This is a pilot study in Basrah, Iraq that investigate thyroid function abnormalities in patients with HCV. Our study shows that 15.6% of HCV positive patients had hypothyroidism (half of them have subclinical hypothyroidism), 19.6% (N=10) of HCV patients has weird thyroid function (they have normal TSH but abnormal T3 and T4, six patients had elevated TT3, 1 had elevated FT4, and 3 had decreased FT4) and 64.7% (N=33) have normal thyroid function, and none has hyperthyroidism. These results are different from other studies where the most frequently observed change was decreased total T3 and free T3 concentration which is reported to be associated with the severity of hepatic dysfunction,[20,24,25] because most of our cases are newly diagnosed with HCV infection, having normal hepatic function and none of them in cirrhosis. While the control group shows only 18.4% of them have abnormal thyroid function, 8.2% have subclinical hypothyroidism and 10.2% have weird thyroid function. But there was no significant difference between the two groups in thyroid abnormalities or in thyroid auto antibodies, may be because no patient received INF-α treatment. These results were in accordance of studies done in other countries which show frequencies of thyroid abnormalities around 7% to 20%,[13-16] but were different from a study done in Thi-Qar, Iraq which shows that the frequency of thyroid abnormalities is around 27.5% and that hyperthyroidism was more than hypothyroidism[26] which may be explained by difference in patients sampling as they include all HCV-ELISA positive patients and not confirmed by PCR. According to our study results, hypothyroidism was more in HCV positive patient than the control group that mimic other studies, but differ from them in the point that

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

none in our study has hyperthyroidism, may be because most of our patients are newly diagnosed and none of them were in cirrhosis. It also shows thyroid abnormalities were significantly affected by gender and were more in female and more in patients aged from 30-40 years, that goes with other studies.[16,27] Also this study shows thyroid antibodies (Anti TPO, anti TG) were significantly more in patients with hypothyroidism as compared to others which indicate it has autoimmune origin, which goes with other studies[16,26] A meta-analysis of 12 studies shows that the prevalence of Anti TPO antibody is about 1.95 fold than controls and the prevalence of hypothyroidism is more with pooled risk of 3.10(95% CI: 2.19-4.40) in HCV positive patient than controls,[28] while our study shows no difference in the prevalence of anti TPO between HCV positive patients and controls, and the risk of hypothyroidism is more in HCV positive patient but not statistically significant and this may be because small sample size, type of study and different criteria for inclusion. This study also shows thyroid abnormalities were not significantly affected by liver enzymes either elevated or not which was differ from other studies[16,29] could be due to differences in sampling size and that no patient has signs of CHC. Also, whether the patients received DAA treatment or not which also differ from other study[30] may be because our patients not complete their treatment regime.

In Conclusion and recommendation, thyroid abnormalities are common in HCV positive patients where hypothyroidism is the most frequent. Women are more frequent than men in thyroid abnormalities, and more in the age group 30-40 years. In HCV patients, the majority of hypothyroidism has autoimmune origin while no relation between thyroid antibodies and subclinical hypothyroidism. Further studies with larger samples and long periods of follow up are needed to confirm our results and identify other causes of thyroid abnormalities in HCV positive patients, and thyroid function needs to be monitored in HCV positive patients.

References

1. Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo J. Harrison's Principles of Internal Medicine, Twentieth Edition (Vol.1 & Vol.2): McGraw-Hill Education; 2018.
2. Waheed Y, Saeed U, Anjum S, Afzal MS, Ashraf M. Development of Global Consensus Sequence and Analysis of Highly Conserved Domains of the HCV NS5B Prote in. Hepat Mon. 2012; 12(9):e6142.
3. Hussein NR, Balatay AA, Sezgin AT, Jawad RS. The distribution of HCV in subjects attending hospitals in Duhok City, Iraq. Asian Pacific Journal of Tropical Biomedicine. 2017; 7(3): 262-264.
4. Kamaz Z. HCV Prevalence, Risk Factors and Precaution Practices among Patients in Karbala City. 2019; 4:117.
5. Zainab HA-M, Hawazin YT. The prevalence rate of hepatitis c virus in basrah city and the common causative risk factors. Iraqi National Journal of Medicine . 2019; 1(2):59-60.
6. Korean Association for the Study of the L. KASL clinical practice guidelines: management of hepatitis C. Clin Mol Hepatol. 2016; 22(1):76-139.
7. Zhou M, Li H, Ji Y, Ma Y, Hou F, Yuan P. Hepatitis C virus infection in the general population: A large community-based study in Mianyang, West China. Bioscience trends. 2015; 9(2):97-103.

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

8. Rosenthal E, Cacoub P. Extrahepatic manifestations in chronic hepatitis C virus carriers. Lupus. 2015; 24(4-5):469-482.
9. Matthews PC, Jeffery K, Klenerman P, Barnes E, Cooke G. Screening and treatment for hepatitis C: a balanced perspective. BMJ (Clinical research ed). 2015; 350:h644.
10. Ward CJ, Lee V. Hepatitis C screening of men who have sex with men. BMJ (Clinical research ed). 2015; 350: h642.
11. Ferrari SM, Fallahi P, Mancusi C, Colaci M, Manfredi A, Ferri C, et al. HCV-related autoimmune disorders in HCV chronic infection. La Clinica terapeutica. 2013; 164(4):e305-12.
12. Tomer Y. Hepatitis C and interferon induced thyroiditis. Journal of autoimmunity. 2010; 34(3): J322-6.
13. Ganne-Carrie N, Medini A, Coderc E, Seror O, Christidis C, Grimbert S, et al. Latent Autoimmune Thyroiditis in Untreated Patients with HCV Chronic Hepatitis: a Case-control Study. Journal of autoimmunity. 2000; 14:189-193.
14. Stefanova-Petrova D, Tzvetanska A, Naumova E, Mihailova A, E H, Dikova R, et al. Chronic hepatitis C virus infection: Prevalence of extrahepatic manifestations and association with cryoglobulinemia in Bulgarian patients. World journal of gastroenterology: WJG. 2007; 13: 6518-6528.
15. Shao C, Huo N, Zhao L, Gao Y, Fan X, Zheng Y, et al. The presence of thyroid peroxidase antibody of IgG2 subclass is a risk factor for thyroid dysfunction in chronic hepatitis C patients. European journal of endocrinology. 2013;168(5): 717-722.
16. Ashshi A, El-shemi A, AlZanbagi A, Refaat B. Prevalence of thyroid disorders and the correlation of thyroid profile with liver enzymes, serum activin-A and follistatin during the treatment of patients with chronic hepatitis C genotype 1 and 4. Journal of Clinical and Experimental Investigations. 2014; 5: 343-353.
17. Akeno N, Blackard JT, Tomer Y. HCV E2 protein binds directly to thyroid cells and induces IL-8 production: a new mechanism for HCV induced thyroid autoimmunity. Journal of autoimmunity. 2008; 31(4): 339-344.
18. Blackard JT, Kong L, Huber AK, Tomer Y. Hepatitis C virus infection of a thyroid cell line: implications for pathogenesis of hepatitis C virus and thyroiditis. Thyroid: official journal of the American Thyroid Association. 2013; 23(7): 863-870.
19. Bartolome J, Rodriguez-Inigo E, Quadros P, Vidal S, Pascual-Miguelanez I, Rodriguez-Montes JA, et al. Detection of hepatitis C virus in thyroid tissue from patients with chronic HCV infection. Journal of medical virology. 2008; 80(9): 1588-1594.
20. Malik R, Hodgson H. The relationship between the thyroid gland and the liver. QJM : monthly journal of the Association of Physicians. 2002; 95(9):559-569.
21. Glinoer D, Gershengorn MC, Robbins J. Thyroxine-binding globulin biosynthesis in isolated monkey hepatocytes. Biochimica et biophysica acta. 1976; 418(2): 232-244.
22. Bianchi GP, Zoli M, Marchesini G, Volta U, Vecchi F, Iervese T, et al. Thyroid gland size and function in patients with cirrhosis of the liver. Liver. 1991; 11(2): 71-77.
23. Yamanaka T, Ido K, Kimura K, Saito T. Serum levels of thyroid hormones in liver diseases. Clinica chimica acta; international journal of clinical chemistry. 1980; 101(1): 45-55.
24. Borzio M, Caldara R, Borzio F, Piepoli V, Rampini P, Ferrari C. Thyroid function tests in chronic liver disease: evidence for multiple

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

abnormalities despite clinical euthyroidism. Gut. 1983; 24(7): 631-636.

25. Kayacetin E, Kisakol G, Kaya A. Low serum total thyroxine and free triiodothyronine in patients with hepatic encephalopathy due to non-alcoholic cirrhosis. Swiss medical weekly. 2003; 133(13-14): 210-213.
26. Hasan A. Investigation of Thyroid Function Disturbance in Patients Infected with Hepatitis C Virus. Journal of International Pharmaceutical Research. 2018; 45:82-88.
27. Batool N, Elahi S, Saleem N, Ashraf A. Thyroid Dysfunction in Non-Interferon Treated Hepatitis C Patients Residing in Hepatitis Endemic Area. BioMed research international. 2017; 2017:2390812.
28. Shen Y, Wang XL, Xie JP, Shao JG, Lu YH, Zhang S, et al. Thyroid Disturbance in Patients with Chronic Hepatitis C Infection: A Systematic Review and Meta-analysis. Journal of gastrointestinal and liver diseases: JGLD. 2016; 25(2):227-234.
29. Mao XR, Zhang LT, Chen H, Xiao P, Zhang YC. Possible factors affecting thyroid dysfunction in hepatitis C virus-infected untreated patients. Experimental and therapeutic medicine. 2014; 8(1):133-140.
30. Wahid B, Waqar M, Rasool N, Wasim M, Khalid I, Idrees M. Prevalence of thyroid stimulating hormone dysfunction among sofosbuvir-treated HCV-infected patients: A real-world clinical experience. Journal of medical virology. 2019; 91(3):514-517.

http://dx.doi.org/10.33762/mjbu.2022.132265.1092

اضطرابات الغدة الدرقية في المرضى المصابين بالتهاب الكبد الوبائي سي في البصرة

الخلفية: فيروس التهاب الكبد الوبائي سي له تأثير على الغدة الدرقية.

الهدف: هو دراسة العلاقة بين التهاب الكبد الوبائي سي واضطرابات الغدة الدرقية **الطريقة:** هذه الدراسة تشمل ٥١ مريضًا و ٤٩ شخصًا سليم. تم إرسالهم لاختبار وظائف الغدة الدرقية في شكل TSH، T4، T3، والأجسام المضادة.

النتائج: اضطراب الغدة الدرقية وجد في ٣٥,٣ ٪ من المرضى، حيث ان ٧,٨ ٪ قصور الغدة الدرقية، ٧,٨٪ قصور ودون السريري، و ١٩,٧٪ وظيفة الغدة غير المعتادة.

الخلاصة: ان اضطرابات الغدة الدرقية كانت أكثر انتشارا في مرضى التهاب الكبد الوبائي سي وان قصور الغدة الدرقية هو الأكثر تكرارا.

الكلمات المفتاحية: فيروس التهاب الكبد سي، تشوهات الغدة الدرقية، الأجسام المضادة للغدة الدرقية.

http://dx.doi.org/10.33762/mjbu.2022.132265.1092