THE EFFECT OF HEPATITIS C VIRUS INFECTION ON INSULIN RESISTANCE IN CHRONIC HEMODIALYSIS PATIENTS

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ABSTRACT

Background: Hepatitis C virus infection has strong relationship with insulin resistance in general population. Insulin resistance increases risk of cardiovascular events in chronic kidney disease patients.This work is intended to study the interrelation between Hepatitis C virus infection and insulin resistance among end stage renal disease patients on regular hemodialysis.
Methods: It included a total number of 90 subjects. They were divided into two groups: Group 1 (45 subjects Hepatitis C viruspositivepatientson regular hemodialysis), Group 2 (45 subjects Hepatitis C virusnegative patients on regular hemodialysis).All patients included in this study were subjected to the following: Full clinical assessment,Complete blood picture, alanine aminotransferase, aspartate aminotransferase, creatinine, blood urea, fasting plasma glucose, Coagulation profile, serum calcium, serum phosphorus, serum parathyroid hormone, Hepatitis C virusantibodies, human immunodeficiency virus antibodies, hepatitis surface antigen, fasting serum insulin and fasting serum C-peptide.
Results: Our study reported that no statically significant difference in Homeostatic model assessment of insulin resistance between the two studied groups. Homeostatic model assessment of insulin resistance has significant correlation to age, weight, body mass index,serum parathyroid hormone, serum creatinine, urea reduction ratio and serum ferritin.
Conclusion: We couldn't detect any strong correlation between Hepatitis C virus seropositivity and insulin resistance in hemodialysis patients, but we detected strong relationship of insulin resistance to age, weight, body mass index, serum parathyroid hormone, serum creatinine, urea reduction ratio and serum ferritin in hemodialysis patients.

Keywords: Insulin resistance, Hepatitis C virus, Chronic kidney disease, Hemodialysis

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Received: 1 September 2015
Accepted: 29 October 2015

INTRODUCTION

HCV infection causes several extra-hepatic manifestations. These manifestations are hypothesized to be due to activation of immune system and direct effect of virus itself[1].Type 2 DM is considered to be the most important extra-hepatic manifestation of infection by HCV virus [2].

Patients on regular dialysis are exposed to high risk for HCV infection. That is attributed to prolonged use of AV fistula and possible contact with contaminated instruments. The incidence of HCV infection in hemodialysis population differs widely among countries from only less than 5% in some countries up to approximately 60% in other countries [3].

The relationship between HCV and type 2 DM has been suggested by the high frequency of type 2 DM among HCV patients and high prevalence of HCV infection among type 2 diabetic patients [4].

Type 2 DM develops as a result of resistance to insulin and decreased beta cells production of insulin. In HCV core-gene transgenic mice the injection of insulin decreases blood sugar level but less potent effect than in control group and also basal

insulin was higher in HCV infected mice indicting insulin resistance in HCV infected mice[5].

Diabetic nephropathy is the commonest etiology of end-stage renal disease (ESRD) worldwide. The number of patients suffering from DM and associated CKD is increasing and is said to be epidemic in distribution with poor prognosis [6].

So CKD is commonly associated with abnormal metabolism of carbohydrate, resistance to insulin and infection by HCV [7]. The presence of insulin resistance predicts development of cardiovascular diseases which is the leading cause of death from CKD. Also progression of microalbuminurea and gross proteinuria in patients with renal impairment is strongly associated with insulin resistance [8].

The aim of our work was intended to study the interrelation between Hepatitis C virus infection and insulin resistance among end stage renal disease patients on regular hemodialysis.

SUBJECTS AND METHODS

This study was carried out in Internal Medicine Department, Zagazig University Hospitals, Faculty of Medicine, ZagazigUniversity.

A) Subjects

The study included 90 cases with end stage renal disease on regular hemodialysis three times per week. These patients were selected from hemodialysis unit of the internal medicine department, Faculty of Medicine, Zagazig University. They were classified into two main groups according to presence or absence of HCV antibodies.

1) Group 1 included 45 patients who were HCV positive and negative for HBsAg and anti-HIV Ab. They were 29 males and 16 females with age ranging from 21-73 years old with mean 46.22 ±13.38 years old.

2) Group2 included 45 patients who were HCV negative and negative for HBsAg and anti-HIV Ab.They were 25 males and 20 females with age ranging from 17-75 years old with mean 44.20 ±14.71 years old.

B) Methods of Study

All subjects of the study were subjected to the following:

Thorough history and full clinical examination:

Special emphasis on duration of CKD, etiology of CKD (If known), duration of dialysis, drugs taken by the patients and presence of jaundice or ascites.Also pasthistory of DM, hypertension or smoking.

Laboratory investigations:

- Complete blood picture.
- Serum ALTand AST.
- Serum creatinine and blood urea.
- Fasting plasma glucose.
- PT, PTT and INR.
- Serum calcium and serum phosphorus.
- Serum iPTH.
- HCV Antibodies, HIV Antibodies, HBsAg.
- Fasting serum insulin and fasting serum C-peptide.

Kits **(Human insulin and C-peptide)** were provided by Chemux Bioscience, South San Francisco, USA. Fasting insulin was done to calculate HOMA-IR to identify presence of insulin resistance and fasting C-peptide was done to evaluate its correlation to insulin resistance in CKD especially in HCV infected patients.

- HOMA-IR which equals ([fasting insulin (mU/L) x fasting glucose (mmol/L)]/22.5). Results of HOMA-IR equal or more than 2.5 were considered positive for insulin resistance.

RESULTS

Table 1: Demographic data of group 1and group 2

Variable	Group 1	Group 2	Test	P
Age (years)				
Mean ± SD	46.22 ± 13.38	44.20 ± 14.71	0.682*	0.497
Median (Range)	51 (21 – 73)	46 (17 – 75)		(NS)
Age ≤48.5 years	46.7%	53.3%	0.400§	0.527
Age >48.5 years	53.5%	46.7%		(NS)
Sex				
Male	64.4%	55.6%	0.741§	0.389
Female	35.6%	44.4%		(NS)

(*): Independent Student t-test, **(§)**: Chi-square test and **(SD):** Standard deviation.

Table (1) shows no statistical significant differences between group 1 and group 2 regarding age and sex distribution.

Table 2: Clinical data of group 1 and group 2

Clinical data	Group 1	Group 2	Test	P
Weight(kg)				
Mean ± SD	63.60 ± 14.47	73.12 ± 19.07	-2.57•	0.010
Range	35 – 95	31 – 110		(S)
Height (m)				
Mean ± SD	1.66 ± 0.06	1.68 ± 0.04	-1.86•	0.062
Range	1.48 – 1.74	1.55 – 1.75		(NS)
BMI (kg/m^2)				
Mean ± SD	22.82 ± 4.55	25.50 ± 6.18	-2.56•	0.010
Range	14.57 - 32.87	12.26 - 37.62		(S)
SBP (mmHg)				
Mean ± SD	126.44 ± 16.53	129.55 ± 20.33	-0.64•	0.520
Range	90 - 160	90 - 180		(NS)
DBP(mmHg)				
Mean ± SD	80.22 ± 10.33	81.77 ± 12.48	-0.56•	0.577
Range	60 – 100	60 – 110		(NS)
Dialysis (years)				
Mean ± SD	7.93 ± 5.25	5.64 ± 4.40	-2.18•	0.029
Range	1 – 17	1 – 22		(S)

(•): Mann Whitney U test**, (§):**Chi-square test**, (SD):** Standard deviation, **(SBP):** Systolic blood pressure and (**DBP**): Diastolic blood pressure.

Table (2) shows no statistical significant differences between the two groups regarding height, systolic blood pressure and diastolic blood pressure. Weight was higher significantly in group 2 with mean value 73.12 kg and dialysis duration was longer significantly in group 1 with mean value7.93 years.

Table 3: Laboratory data of group 1 and group 2

Variable	Group 1	Group 2	Test	P
WBC's (x103/mm3)				
Mean ± SD	5.67 ± 1.83	6.71 ± 2.04	-2.511•	0.012
Median (Range)	5.4 (3 – 10.3)	6 (3.30 – 11.40)		(S)
Hemoglobin (g/dl)				
Mean ± SD	10.82 ± 1.60	10.07 ± 1.66	-1.970•	0.049
Median (Range)	10.9 (7.6 –14.5)	10 (7 – 14.2)		(S)
Platelet (x103/mm3)				
Mean ± SD	187.51 ± 56.22	241.20 ± 83.83	-3.370•	0.001
Median (Range)	177 (96 – 371)	249 (115 – 508)		(HS)
Creatinine (mg/dl)				
Mean ± SD	10.66 ± 2.83	11.55 ±2.17	-1.529•	0.126
Median (Range)	10.59 (5.35 – 18.84)	11.19 (7.16 –1 5.69)		(NS)
URR%				
Mean ± SD	69.73 ± 8.10	67.34±7.94	-1.622•	0.105
Median (Range)	69.90 (55 – 95.60)	66.70 (55 – 92)		(NS)
URR% ≤68.5	42.2%	57.8%	2.178§	0.140
URR%>68.5	57.8%	42.2%		(NS)
Calcium (mg/dl)				
Mean ± SD	9.00 ± 0.82	9.11 ± 0.88	-0.484•	0.628
Median (Range)	9.08 (6.60 – 10.62)	8.83 (7.10 – 10.91)		(NS)
Phosphorus (mg/dl) Mean ± SD	4.41 ± 1.52	4.95 ± 1.63	-1.578•	0.115
Median (Range)	4.27 (2.21 – 7.45)	4.83 (2.29 – 9.69)		(NS)
iPTH (pg/ml)				
Mean ± SD	646.22 ± 486.87	396.03 ± 358.39	2.679•	0.007
Median (Range)	556 (12.40 – 1900)	190 (17.89 – 1100)		(S)
Ferritin (ng/ml)				
Mean ± SD	1289.78 ± 1291.67	905.16 ± 1035.49	-1.933•	0.043
Median (Range)	880 (155 – 6596)	485 (43.4 – 9465)		(S)
Transferrin saturation				
Mean ± SD	38.96 ± 23.40	32.88 ± 16.23	-0.924•	0.035
Median (Range)	30.7 (11.9 –98.39)	26.14 (12.01 – 68.95)		(S)

(•): Mann Whitney U test**, (§):**Chi-square test**, (SD):** Standard deviation**, (WBC's)**: White blood cells, (**URR**): Urea reduction ratio and (**iPTH**): Intact parathyroid hormone.

Table (3) shows no significant differences between group 1 and group 2 regarding serum creatinine, URR, serum calcium and serum phosphorous. WBC's count is significantly higher in group 2 with median 6 $*10^3$ cells /mm^3. Hemoglobin is significantly higher in group 1 with median 10.9 gm/dl. iPTH is significantly higher in group 1 with median 556 pg/ml. Platelets count is significantly higher in group 2 with median 249 $x10^3/mm^3$. Serum ferritin and transferrin saturation are significantly higher in group 1 with median values880ng/ml and30.7% respectively.

Table 4: Special investigation of group 1 and group 2

Variable	Group 1	Group 2	Test	P
Insulin (µIU/ml)				
Mean ± SD	11.21 ± 10.45	10.21 ± 10.16	-0.101•	0.920
Median (Range)	7.02 (1.55 – 40.77)	5.80 (1.32 – 52.72)		(NS)
C-peptide(ng/ml)				
Mean ± SD	8.67 ± 3.32	8.50 ± 2.77	-0.408•	0.683
Median (Range)	9.37 (3.42 – 12.70)	8.91 (3.52 – 12.70)		(NS)
Fasting Glucose (mg/dl)				
Mean ± SD	82.11 ± 50.66	83.84 ± 27.71	-1.618•	0.106
Median (Range)	72 (48.80 – 387.9)	77.80 (53.50 – 185.3)		(NS)
HOMA-IR				
Mean ± SD	2.115± 2.059	2.241 ± 2.671	-0.085•	0.932
Median	1.181	1.241		(NS)
Range	0.207 – 8.137	0.189 – 15.489		
Insulin resistance Negative				
Positive	75.6%	71.1%	0.227§	0.634
	24.4%	28.9%		(NS)

(•): Mann Whitney U test**, (§):**Chi-square test, **(SD):** Standard deviation and **(HOMA-IR):** Homeostatic model assessment of insulin resistance.

Table (4) shows no significant differences between group 1 and group 2 in fasting insulin, fasting C peptide, fasting glucose, HOMA-IR and insulin resistance..

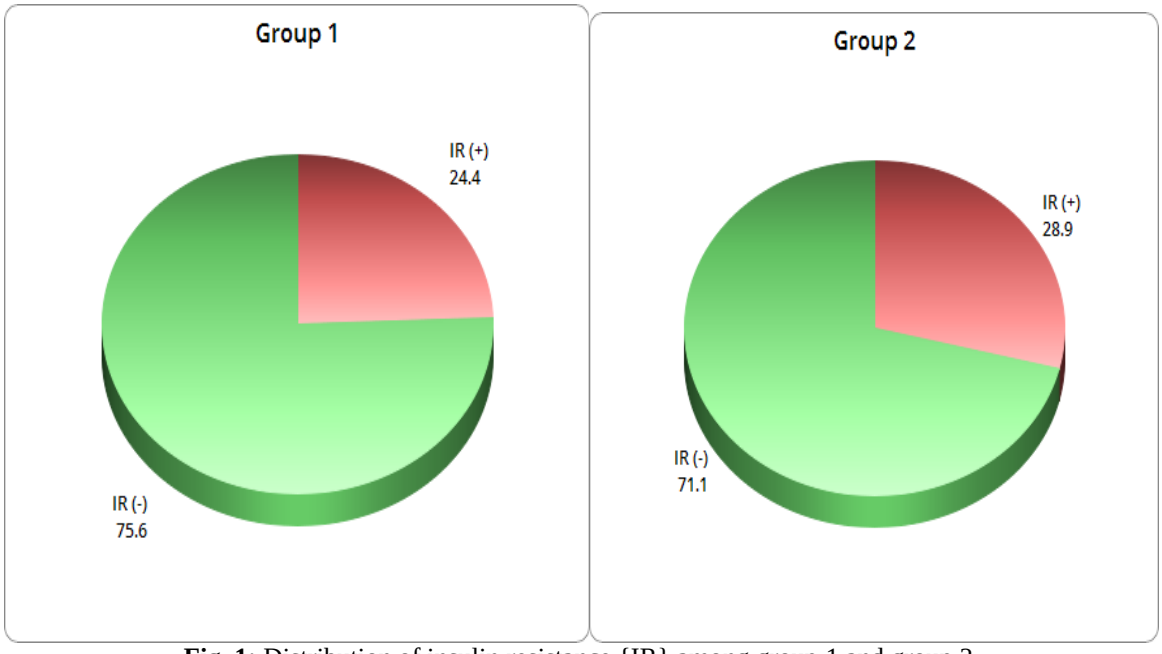

Fig. 1: Distribution of insulin resistance {IR} among group 1 and group 2.

Table 5: HOMA-IR among URR% subgroupsin group 1 and group 2.

HOMA-IR	Group 1	Group 2	Test	p-value
URR% ≤68.5	(n=19)	(n=26)	-0.506•	0.613
Mean ± SD	2.469 ± 1.664	2.280 ± 1.882		(NS)
Median	1.925	1.698		
Range	0.399 – 5.872	0.220 – 8.259		
URR% >68.5%	(n=26)	(n=19)	-0.069•	0.945
Mean ± SD	1.857 ± 2.303	2.188 ± 3.537		(NS)
Median	0.923	0.738		
Range	0.207 – 8.137	0.189 – 15.489		
Test	-2.253•	-1.885•		
p-value (Sig.)	0.024 (S)	0.059 (NS)		

(•) Mann Whitney U test, (**URR**): Urea reduction ratio, **(SD):** Standard deviationand **(HOMA-IR):** Homeostatic model assessment of insulin resistance.

Table (5) shows no significant differences of HOMA-IR between low URR subgroups in either group 1 or group 2 and also between high URR subgroups in either group 1 or group 2. HOMA-IR is significantly higher with low URR subgroup of group 1 versus high URR subgroup in group 1.

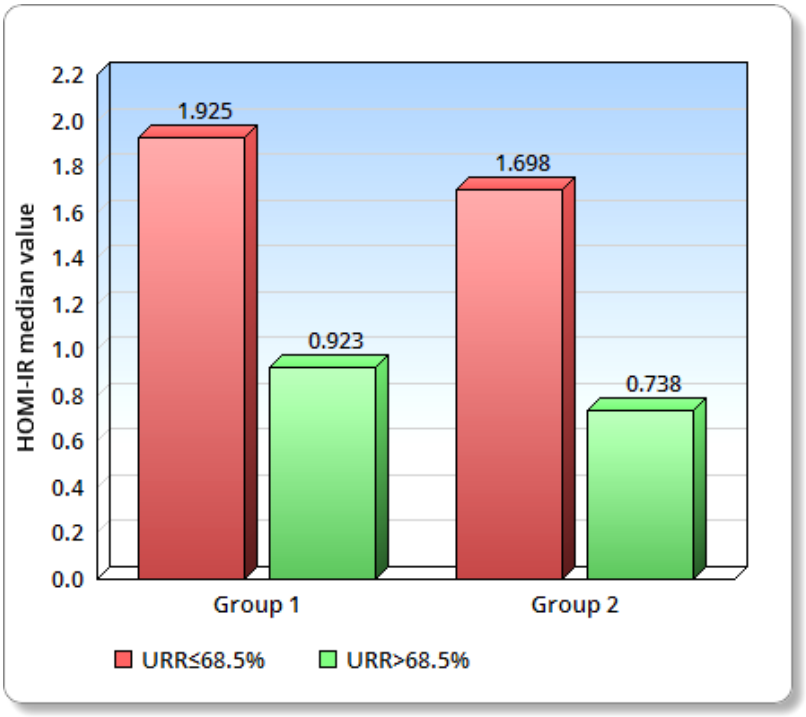

Fig. 2: HOMA-IR median value among URR subgroups of group 1 and group 2

Table 6: Correlation betweenHOMA-IR and selected study parameters

Variables	Group 1		Group 2	
	r	p-value	r	p-value
Age (years)	+0.205	0.176 (NS)	+0.347	0.019 (S)
Weight (kg)	+0.284	0.059 (NS)	+0.411	0.005 (HS)
Height (m)	+0.019	0.903 (NS)	+0.084	0.583 (NS)
BMI (kg/m^2)	+0.278	0.064 (NS)	+0.439	0.003 (HS)
Systolicblood pressure (mmHg)	+0.096	0.532 (NS)	-0.017	0.913 (NS)
Diastolicblood pressure (mmHg)	+0.006	0.970 (NS)	+0.151	0.323 (NS)
Hemodialysis duration (years)	+0.061	0.691 (NS)	-0.115	0.450 (NS)
WBC's (x10^3/mm^3)	-0.063	0.679 (NS)	+0.173	0.255 (NS)
Hemoglobin (g/dl)	+0.010	0.950 (NS)	+0.080	0.601 (NS)
Platelet count (x10^3/mm^3)	-0.077	0.616 (NS)	+0.035	0.822 (NS)
Creatinine (mg/dl)	+0.270	0.073 (NS)	-0.230	0.129 (NS)
URR%	-0.378	0.010 (S)	-0.269	0.074 (NS)
Calcium (mg/dl)	-0.319	0.033 (S)	+0.112	0.465 (NS)
Phosphorus (mg/dl)	+0.471	0.001 (HS)	+0.188	0.216 (NS)
iPTH (pg/ml)	+0.375	0.011 (S)	-0.257	0.089 (NS)
Ferritin (ng/ml)	-0.227	0.133 (NS)	-0.132	0.387 (NS)
Transferrin saturation (%)	-0.313	0.036 (S)	-0.277	0.066 (NS)
Fasting Insulin (µIU/ml)	+0.904	<0.001 (HS)	+0.952	<0.001 (HS)
Fasting C peptide (ng/ml)	+0.652	<0.001 (HS)	+0.508	<0.001 (HS)
Glucose (mg/dl)	+0.194	0.202 (NS)	+0.558	<0.001 (HS)

(**r**): Spearman's rank correlation coefficient, (**BMI**): Body mass index, **(WBC's)**: White blood cells, (**URR**): Urea reduction ratio and (**iPTH**): Intact parathyroid hormone.

This table shows that HOMA-IR in group 1 is strongly correlated to URR, serum calcium, serum phosphorus, iPTH, transferrin saturation, fasting insulin and fasting C peptide. HOMA-IR in group2 is strongly correlated to age, weight, BMI, fasting insulin, fasting C peptide and fasting blood glucose.

Table 7: Univariate logistic regression of potential predictors of insulin resistance in all studied patients (N=90)

Variables	β	SE	OR	95% CI	p-value
HCV (+ve)	-0.228	0.478	0.796	(0.312 – 2.032)	0.643 (NS)
Age (years)	0.035	0.019	1.036	(0.999 – 1.075)	0.049 (S)
Male	-0.328	0.482	0.721	(0.280 – 1.853)	0.497 (NS)
Weight (kg)	0.035	0.014	1.035	(1.007 – 1.065)	0.016 (S)
Height (m)	0.741	4.199	2.097	(0.00 – 7863.2)	0.860 (NS)
BMI (kg/m^2)	0.123	0.045	1.131	(1.035 – 1.235)	0.007 (HS)
SBP (mmHg)	0.003	0.013	1.003	(0.978 – 1.029)	0.816 (NS)
DBP (mmHg)	0.011	0.021	1.011	(0.971 – 1.054)	0.586 (NS)
Hemodialysis duration (years)	0.023	0.047	1.023	(0.933 – 1.122)	0.027 (NS)
WBC's (x10^3/mm^3)	-0.078	0.123	0.925	(0.726 – 1.178)	0.528 (NS)
Hemoglobin (g/dl)	-0.141	0.147	0.686	(0.651 – 1.158)	0.337 (NS)
Platelet (x10^3/mm^3)	-0.004	0.004	0.996	(0.989 – 1.003)	0.253 (NS)
Creatinine (mg/dl)	-0.025	0.094	0.975	(0.811 – 1.172)	0.037 (S)
URR (%)	-0.022	0.031	0.979	(0.921 – 1.040)	0.025 (S)
Calcium (mg/dl)	0.028	0.281	1.028	(0.592 – 1.784)	0.921 (NS)
Phosphorus (mg/dl)	0.247	0.150	1.281	(0.954 – 1.719)	0.100 (NS)
iPTH (pg/ml)	0.000	0.001	1.000	(0.999 – 1.002)	0.042 (S)
Ferritin	0.000	0.000	1.000	(1.000 – 1.001)	0.049 (S)
Transferrin saturation	-0.002	0.012	0.998	(0.975 – 1.022)	0.881 (NS)

(**β**): regression Coefficient, (**SE**): standard error, (**OR**): Odds Ratio, (**95%CI**): 95% confidence interval, (**BMI**): Body mass index, **(SBP):** Systolic blood pressure, (**DBP**): Diastolic blood pressure**, (WBC's)**: White blood cells, (**URR**): Urea reduction ratio and (**iPTH**): Intact parathyroid hormone.

Table (**6**) shows that there are significant correlation between insulin resistance and each of the variables age, weight, BMI, creatinine, URR, iPTH and ferritin. Also showed no significant correlation between insulin resistance and each of the variables HCV seropositivity, sex, height, blood pressure, hemodialysis duration, WBC's, Hemoglobin, platelet count, serum calcium, serum phosphorus and transferrin saturation.

DISCUSSION

Infection by Hepatitis C virus (HCV) is considered to be one of most important health problems in Egypt. Prevalence of HCV infection in Egypt, especially at ages 15–59 years old, is the highest worldwide. This high prevalence was explained by the wide use of parenteral anti-bilharzial drugs during the period between 1960 to end of 1970 [9].

HCV is commonly associated with insulin resistance in general population and occurrence of many complications of HCV infection is proved to be strongly related to insulin resistance. These complications include fatty liver, liver fibrosis, hepatoma and lack of response to antiviral drugs [10].

CKD is commonly associated with insulin resistance and presence of insulin resistance predicts the development of cardiovascular disease which is the leading cause of death from CKD. Death due to acute cardiovascular events is ten times more in hemodialysis patients than in general population. Also progression of microalbuminuria and gross proteinuria in patients with renal impairment is strongly associated with insulin resistance [8].

Aim of our study is to investigate the correlationof HCVseropositivity to insulin resistance in chronic hemodialysis patients and to detect the risk factors for insulin resistance in hemodialysis patients.

As regard age and sex distribution in our study there were no significant differences among group 1 and group 2, these results were in agreement with **Tsai et al.,**[11]and **Ozdemir et al.,**[12].

In comparison of clinical data of group 1 and group 2, therewere no statistical significant differences between the two groups regarding height, systolic blood pressure and diastolic blood pressure. Dialysis duration was longer significantly in group 1 with mean duration 7.93 ± 5.25 years and these results were in agreement with **Tsai et al.,**[11] and **Ozdemir et al.,**[12]. This can be explained by the longer the dialysis duration the higher the chance of nosocomial transmission of HCV due to sharing items between patients, contamination of dialysis machines, contaminated gloves used by working nurses and lack of proper sterilization of machines after each dialysis session[13].

In our study WBC's and platelets were significantly lower in group 1 than group 2. These results are in agreement with **Tsai et al.,** [11] and can be due to replication of the HCV virus in bone marrow, splenic sequestration and immune mediated [14].

Hemoglobin was higher in group 1 and these results are in agreement with **Alsaran et al.,** [15] and can be explained by that hepatic inflammation induces erythropoietin production by liver cells and also due to altered iron metabolism [16].

As regard metabolic profile of group 1 and group 2, there were no significant differences regarding serum creatinine, URR, serum calcium and serum phosphorous. Serum ferritin, iPTH and transferrin saturation were significantly higher in group 1. These results are in the agreement with **Tsai et al.,**[11] and can be explained by that HCV induces

inflammation and increases circulating cytokinesthat stimulate precursors of osteoclasts, and increases RANKL formation by osteoblasts, and so increased bone loss leading to elevated PTH level [17]. Also liver inflammation increases release of ferritin in circulation as acute phase reactant and alters iron metabolism [18].

We found special laboratory tests of group 1 and group 2 showed that there were no significant differences between the two groups regarding fasting insulin, fasting C peptide, fasting glucose, HOMA-IR and insulin resistance. These results are in agreement with **Tsai et al.,** [11] and **Adam et al.,**[19], and against **Ozdemir et al.,**[12] and this can be explained by other factors played role in our study including efficacy of dialysis, relatively small number of patients and BMI was significantly higher in group 2.

As regard HOMA-IR in between low URR subgroups in group 1 versus group 2 and in between high URR subgroups in group 1 versus group 2 there were no significant differences but HOMA-IR was significantly higher with low URR subgroup of group 1 versus high URR subgroup of group 1. These results are showing the strong relationship between HOMA-IR and URR and these results are in agreement with **Chu et al.,** [20] and can be explained by that with low URR uremic toxins accumulate and affect insulin signaling pathway [20].

Our study showed that HOMA-IR in group 1 is strongly correlated to URR, serum calcium, serum phosphorus, iPTH, transferrin saturation, fasting insulin and fasting C peptide and these results are in agreement with **Tsai et al.,** [11] and **Adam et al.,**[19] and can be explained by that inflammation induces circulating cytokines that results in hyperparathyroidism [16] and elevated iron indices [18]. Low URR is also another important factor that increases prevalence of insulin resistance with HCV infection due to accumulated uremic toxins [20]. Each of elevated fasting blood sugar, fasting insulin and fasting C-peptide can be used as a marker of insulin resistance due to their strong relationship with HOMA-IR.

HOMA-IR in group 2 in our study was strongly correlated to age, weight, BMI, fasting insulin, fasting C peptide and fasting blood glucose. These results are in agreement with **Adam et al.,** [19] and **Gayoso-Diz et al.,** [21] and can be explained by that insulin resistance increases with age due to decreased physical activity, increased visceral obesity and imbalance of sex hormones in old age [22]. Alsoinsulin resistance increases with obesity due to higher free fatty acids and circulating adipokinesinducing systemic resistance to insulin [23].

In our study analysis of the risk of insulin resistance in patients with CKD on regular hemodialysis by univariate binary logistic regression based on demographics, clinical characteristics and laboratory profiles. The results indicate that the probability of insulin resistance was significantly greater with older age [odds ratio (OR): 1.036, 95% confidence interval (CI): 0.999 – 1.075, P = 0.049], higher body weight [odds ratio (OR): 1.035, 95% confidence interval (CI): 1.007 – 1.065, P = 0.016], higher BMI [odds ratio (OR): 1.131, 95% confidence interval (CI): 1.035 – 1.235, P = 0.007], higher serum Creatinine [odds ratio (OR): 0.975 , 95% confidence interval (CI): 0.811 – 1.172, P = 0.037], lower URR [odds ratio (OR): 0.979 , 95% confidence interval (CI): 0.921 – 1.040, P = 0.025], higher iPTH [odds ratio (OR): 1.000, 95% confidence interval (CI): 0.999 – 1.002, P = 0.042] and higher serum Ferritin [odds ratio (OR): 1.000, 95% confidence interval (CI): 1.000 – 1.001, P = 0.049].

CONCLUSION

In conclusion, we couldn't detect any strong correlation between HCV seropositivity and insulin resistance in hemodialysis patients, but we detected strong relationship of insulin resistance to age, weight, iPTH, serum creatinine, URR and serum ferritin in hemodialysis patients.

We recommend that more evaluation is needed by more studies on larger groups of hemodialysis patients and to use more sensitive indices of viral load using HCV RNA PCR, liver fibroscan or liver biopsy in the future studies for more accurate results.
Also we recommend reducing risk of insulin resistance among hemodialysis patients by encouraging weight reduction in overweight patients, proper control of secondary hyperparathyroidism, increasing efficiency of dialysis and avoiding unnecessary excess IV iron replacement in hemodialysis patients.

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