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The Potential Value of Red Cell Distribution Width in Predicting the Transition from Compensated into Decompensated Liver Cirrhosis

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Abstract

Background: Systemic inflammatory response syndrome is relatively common in patients with complicated cirrhosis. Red cell distribution width (RDW), is known to be an inflammatory response marker which is easily evaluated from blood samples.

Aim of Study: The aim of this study was to evaluate the role of RDW in the prediction of liver decompensation in a previously compensated liver cirrhosis.

Patients and Methods: This study was conducted as a retrospective study, which included 401 patients with established diagnosis of liver cirrhosis. The patients were divided into 2 groups; compensated control group which included 100 patients with compensated liver cirrhosis and 301 patients with newly decompensated cirrhosis those patients were subdivided, according to the form of hepatic decompensation, into 3 groups; first hepatic encephalopathy group included 136 patients, first hematemesis group (84 patients) and newly developed ascites group which included (81 patients). Medical records of the patients were retrospectively reviewed for medical history, clinical and laboratory characteristics on admission.

Results: RDW was significantly high in hepatic decompensated group as compared with compensated control group (t-test=18.398), (p-value=0.001). The frequency of developing decompensation was statistically significant higher among patients who had high RDW (96.9%) than those who had normal RDW (36.6%) (p-value=0.001). RDW at a cutoff point ≥13.55, the sensitivity and specificity for prediction of hepatic decompensation were 86.4% and 85% respectively with area under the curve (AUC)=0.944, confidence interval (CI): 0.924-0.964) (p-value=0.001).

Conclusion: RDW may serve as a potential predictor of liver decompensation and therefore a prognostic indicator forcompensated liver disease.

Key Words: *Red Cell – Liver Cirrhosis.*

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Introduction

LIVER cirrhosis, the end stage for most chronic liver diseases, is the 14th leading cause of death worldwide, resulting in approximately 1.3 million deaths annually. The prognosis in cirrhotic patients varies widely, with the reported 1 year mortality rate ranging from 1% to 57%, depending on the occurrence of clinical decompensating events **[1]**. Therefore, it is important to establish an accurate assessment system for the early prediction of decompensating events and prognosis **[2]**. Systemic inflammatory response syndrome is relatively common in patients with complicated cirrhosis and is increasingly recognized to play an important role in the development and progression of liver cirrhosis. Red cell distribution width (RDW) is known to be inflammatory response markers which are easily evaluated from blood samples **[3]**. The aim of this study is to evaluate the role of RDW in prediction of complications of liver cirrhosis.

Patients and Methods

This study was conducted as a retrospective study which included 401 patients with established diagnosis of liver cirrhosis who were admitted to National liver Institute Hospital, Menoufia University in the period from 2020 to 2023. The patients were divided into 2 groups; compensated control group which included 100 patients with compensated liver cirrhosis and 301 patients with newly decompensated cirrhosis those patients were subdivided into 3 groups; first hepatic encephalopathy

included 136 patients who were admitted for their first hepatic encephalopathy, first hematemesis group which included 84 patients who were admitted for their first hematemesis and recent ascites group which included 81 who were admitted for their newly developed ascites. Medical records of the patients were retrospectively reviewed for medical history, clinical and laboratory characteristics on admission.

Exclusion criteria: Patients under 18 year old, previous or recurrent hepatic decompensation features, blood transfusion within 4 months before admission, End-stage renal disease and hepatic or extrahepatic malignancy.

Valid consents were obtained from patients before involvement in the study. All study procedures were carried out and approved by the Ethical Committee of National liver Institute, Menoufia University and in accordance with the declaration of Helsinki. Participant's names were kept on database and linked only with a study identification number for this research.

The following data were reported for each patient; laboratory investigations including; complete blood count (CBC), Alanine transaminase (ALT), Aspartate transaminase (AST), alkaline phosphatase (ALP), gamma glutmale transferase (GGT), Serum Albumin, serum Bilirubin (total & direct), Prothrombin time, Concentration and I.N.R., viral Markers (HCV Ab, HbsAg), creatinine, urea, Serum sodium and serum potassium. All patients were categorized according to child pugh score and model of end stage liver disease (MELD) Score. Imaging including; abdominal ultrasonography to determine; liver: Size, surface, echo pattern of the liver, PV diameter, Spleen; Size, Splenic vein diameter, Portosystemic collaterals and Ascites. Esophagogastroduodenoscopy was done to all patients with firstvariceal hemorrhage to diagnosis, grade and management of the esophageal varices.

Data were collected and entered to the computer using SPSS (Statistical Package for Social Science) program for statistical analysis (version 21) (IBM Corp., Released 2012). The results were considered significant when the probability of error is less than 5% (p-value ≤0.05).

Results

The mean age was 60.15±6.87, 59.89±6.64, 58.51±7.08, and 47.5±5.77 years among the first encephalopathy, first hematemesis, newly developed ascites, and control groups respectively. 251 participants were males and 150 were females. Regarding the complete blood picture, among the three decompensated groups (the first encephalopathy, first hematemesis, newly developed ascites), and control groups respectively. The mean level of Hb was 9.68±1.06, 8.6±0.79, 9.76±0.93, and 12.36±0.89g/dl respectively. The mean RDW level was 18.97±2.52, 17.92±3.02, 16.66±2.67, and 12.69±0.96 fL respectively. The rest of hematological, biochemical or laboratory data are shown in (Table 1).

Table (1): Demographic characteristics and laboratory data of the studied patients.

		Decompensated liver disease			Compensated Control Group (n=100) N (%)
		First Hepatic Encephalopathy (n=136) N (%)	First Hematemesis (n=84) N (%)	Recent Ascites (n=81) N (%)	
Age	Mean ± SD	60.15±6.87	59.89±6.64	58.51±7.08	47.5±5.77
Gender	Male (n=251) Female (n=150)	85 (62.5) 51 (37.5)	51 (60.7) 33 (39.3)	53 (65.4) 28 (34.6)	62 (62.0) 38 (38.0)
Hb	Mean ± SD	9.68±1.06	8.6±0.79	9.76±0.93	12.36±0.89
RDW	Mean ± SD	18.97±2.52	17.92±3.02	16.66±2.67	12.69±0.96
RDW	Normal (n=145) High (n=256)	16 (11.8) 120 (88.2)	16 (19.0) 68 (81.0)	21 (25.9) 60 (74.1)	92 (92.0) 8 (8.0)
Platelets	Mean ± SD	83.15±18.15	86.7±12.53	90.48±16.18	175.39±10.98
WBCs	Mean ± SD	4.9±1.35	4.76±1.08	4.98±1.07	5.11±0.87
INR	Mean ± SD	1.52±0.28	1.48±0.19	1.29±0.17	1.04±0.05
Total bilirubin	Mean ± SD	2.66±0.54	2.57±0.63	2.0±0.47	1.09±0.03
Creatinine	Mean ± SD	1.2±0.28	1.11±0.27	1.17±0.22	0.95±0.13
Na	Mean ± SD	131.71±5.17	134.27±4.22	132.0±4.76	138.8±2.49
K	Mean ± SD	3.19±0.51	3.29±0.42	3.47±0.42	4.04±0.1

The mean MELD score was 18.31±3.78, 17.96±4.14, 17.54±4.23, and 7.32±0.86 among the first encephalopathy, first hematemesis, newly developed ascites, and control groups respectively (Fig. 1).

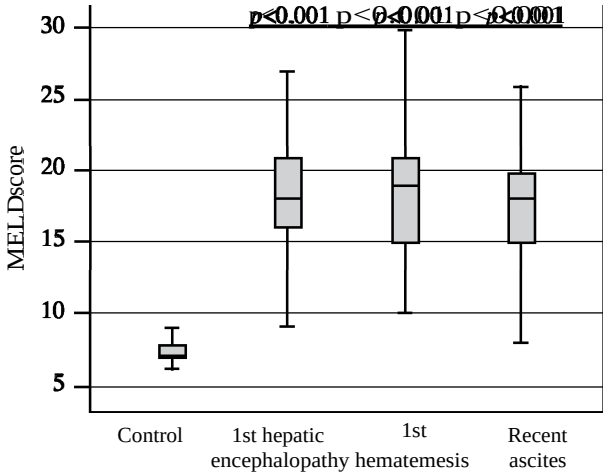

Fig. (1): MELD score of the studied patients.

The frequency of developing decompensation was higher among patients who had high RDW (96.9%) than those who had normal RDW (36.6%) and RDW was higher among first time ofdecompensation than those without previous decompensation. Moreover, the frequency of developing first hepatic encephalopathy, first hematemesis andnewly developed ascites was higher among patients who had high RDW (93.8%, 89.5%, and 88.2% respectively) than those who had normal RDW (14.8%, 14.8%, 18.6%). RDW was higher among first hepatic encephalopathy, first hematemesis and newly developed ascites than those without previous attacks (p-value=0.001 for each) (Table 2 & Fig. 2).

Table (2): Relation between RDW and the development of hepatic decompensation.

Parameters	First Hepatic Encephalopathy	Compensated Control Group	Test of significance (p-value)
RDW:			
Min-Max	12.5-26	11-14.9	t-test = –23.699
Mean ± SD	18.97±2.52	12.69±0.96	(<0.001)**
Median (IQR)	19.15 (18.43-20.5)	12.65 (11.83-13.4)	
Normal	16 (11.8%)	92 (92.0%)	$\chi 2$ = 149.468
High	120 (88.2%)	8 (8.0%)	(<0.001)**
	First Hematemesis		
RDW:			
Min-Max	12.5-27	11-14.9	t-test = –16.353
Mean ± SD	17.92±3.02	12.69±0.96	(<0.001)**
Median (IQR)	18.1 (16.35-20.08)	12.65 (11.83-13.4)	
Normal	16 (19.0%)	92 (92.0%)	$\chi 2$ = 100.216
High	68 (81.0%)	8 (8.0%)	(<0.001)**
	Recent Ascites		
RDW:			
Min-Max	12.5-22	11-14.9	t-test = –13.833
Mean ± SD	16.66±2.67	12.69±0.96	(<0.001)**
Median (IQR)	16.5 (13.75-18.15)	12.65 (11.83-13.4)	
Normal	21 (25.9)	92 (92.0%)	$\chi 2$ = 83.299
High	60 (74.1)	8 (8.0%)	(<0.001)**
	First attack of decompensation		
RDW:			
Min-Max	12.5-27	11-14.9	t-test = –18.398
Mean ± SD	18.05±2.86	12.69±0.96	(<0.001)**
Median (IQR)	18.6 (16.5-20.0)	12.65 (11.83-13.4)	
Normal	53 (36.6%)	92 (63.4%)	$\chi 2$ = 179.952
High	248 (96.9%)	8 (3.1%)	(<0.001)**

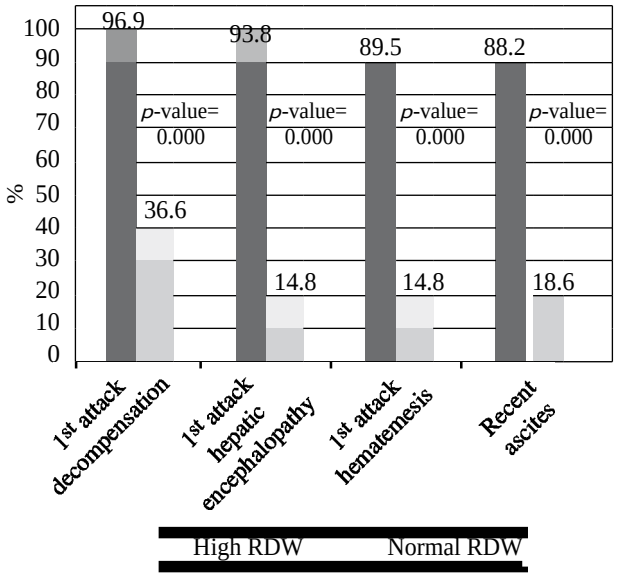

Fig. (2): Relation between RDW and the development of hepatic decompensation.

Univariate logistic regression revealed age, history of DM, RDW, blood urea, serum creatinine, and Na as predictors for hepatic encephalopathy. Although, multivariate logistic regression revealed age, RDW, blood urea, and Na as predictors for hepatic encephalopathy. Univariate logistic regression revealed age, history of DM, RDW, WBCs, blood urea, serum creatinine, as predictors for hematemesis. Although, multivariate logistic regression revealed age, RDW and blood urea, as predictors for hematemesis. Univariate logistic regression revealed age, RDW, blood urea, Na and K as predictors for ascites. Although, multivariate logistic regression revealed age, RDW, blood urea, and K as predictors for ascites (Table 3).

Among hepatic encephalopathy patients, the mean level of MELD score was statistically significantly higher among patients who had high RDW (19.13±3.19) than those who had normal RDW (12.19±1.52). Moreover, there was statistically significant correlation between RDW level and MELD score (r=0.462). Among hematemesis patients, the mean level of MELD score was statistically significantly higher among patients who had high RDW (19.4±3.15) than those who had normal RDW (11.88±1.2). Moreover, there was statistically significant correlation between RDW level and MELD score (r=0. 579). Among ascites patients, the mean level of MELD score was statistically significantly higher among patients who had high RDW (19.0±3.33) than those who had normal RDW (13.38±3.8). Moreover, there was statistically significant correlation between RDW level and MELD score (r=0. 601) (Table 4).

Table (3): Univariate and multivariate logistic regression of parameters associated with hepatic decompensation.

	Crude OR	95% CI; LL-UL	p-value	Adjusted OR	95% CI; LL-UL	p-value
1st Hepatic encephalopathy:						
Age (years)	1.373	1.268-1.487	<0.001**	1.3	1.07-1.58	0.008**
History of DM	2.589	1.501-4.464	0.001**	0.522	0.061-4.486	0.554
RDW	3.136	2.184-4.503	<0.001**	2.259	1.143-4.464	0.019*
Urea	1.292	1.202-1.389	<0.001**	1.313	1.03-1.973	0.028*
Creatinine	676.662	97.042-4718.28	<0.001**	8.722	0.005-14477.582	0.567
Na	0.53	0.446-0.63	<0.001**	0.565	0.346-0.924	0.023*
1st Hematemesis:						
Age (years)	1.378	1.259-1.507	<0.001**	1.868	1.214-2.873	0.004**
RDW	3.303	2.261-4.825	<0.001**	6.348	1.69-23.843	0.006**
WBCs	0.686	0.499-0.942	0.02*	0.684	0.124-3.776	0.663
Urea	1.363	1.239-1.498	<0.001**	2.141	1.219-3.759	0.008**
Creatinine	63.706	9.956-407.643	<0.001**	0.746	0.0-193643.107	0.963
Newly developed ascites:						
Age (years)	1.323	1.22-1.435	<0.001**	1.609	1.138-2.275	0.007**
RDW	3.224	2.282-4.554	<0.001**	4.686	1.171-18.75	0.029*
Urea	1.293	1.193-1.402	<0.001**	1.724	1.141-2.605	0.01*
Na	0.643	0.568-0.727	<0.001**	0.805	0.56-1.159	0.243
K	0.016	0.005-0.054	<0.001**	0.002	0.0-0.384	0.02*

Table (4): Correlation between RDW and MELD score in patients with hepatic decompensation.

1st Hepatic encephalopathy	RDW (n=136)		Test of significance (p-value)
	Normal (n=16)	High (n=120)	
MELD score: Mean ± SD	12.19±1.52	19.13±3.19	t-test = –8.55 (<0.001)**
Correlation co efficient	r=0.462		(<0.001)**
1st Hematemesis	RDW (n=84)		Test of significance (p-value)
MELD score: Mean ± SD	11.88±1.2	19.4±3.15	t-test = –9.346 (<0.001)**
Correlation co efficient	r=0.579		(<0.001)**
Newly developed ascites	RDW (n=81)		Test of significance (p-value)
MELD score: Mean ± SD	13.38±3.8	19.0±3.33	t-test = –6.415 (<0.001)**
Correlation co efficient	r=0.601		(<0.001)**

The sensitivity and specificity of RDW at a cut-off point ≥13.95 were 89% and 91% respectively for the prediction of hepatic encephalopathy with AUC=0.967 (CI: 0.947-0.986) (p-value=0.001). RDW at a cutoff point ≥13.55, the sensitivity and specificity for prediction of hematemesis were 84.5% and 85% respectively with AUC=0.94, CI: (0.906-0.973) (p-value=0.001). The sensitivity and specificity of RDW at a cutoff point ≥13.45 were 80.2% and 79% respectively for the prediction of ascites with AUC= 0.911 (CI: 0.869-0.952) (p-value =0.001). RDW at a cutoff point ≥13.55, the sensitivity and specificity for prediction of first liver decompensation(whether hepatic encephalopathy, hematemesis or ascites) were 86.4% and 85% respectively with area under the curve (AUC)=0.944, confidence interval (CI): 0.924-0.964) (p-value=0.001) (Table 5 & Fig. 3).

Table (5): Performance of RDW in the prediction of first hepatic decompensation.

	RDW Cutoff point	AUC	p	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
1st Hepatic encephalopathy	≥13.95	0.967 (0.947 – 0.986)	<0.001	89.0	91.0	93.08	85.85
1st Hematemesis	≥13.55	0.94 (0.906 – 0.973)	<0.001	84.5	85	82.56	86.73
Newly developed ascites	≥13.45	0.911 (0.869 – 0.952)	<0.001	80.2	79	75.58	83.16
1st Decompensation (overall)	≥13.55	0.944 (0.924 – 0.964)	<0.001	86.4	85	94.5	67.5

AUC: Area under the curve. PPV: Positive predictive value. NPV: Negative predictive value.

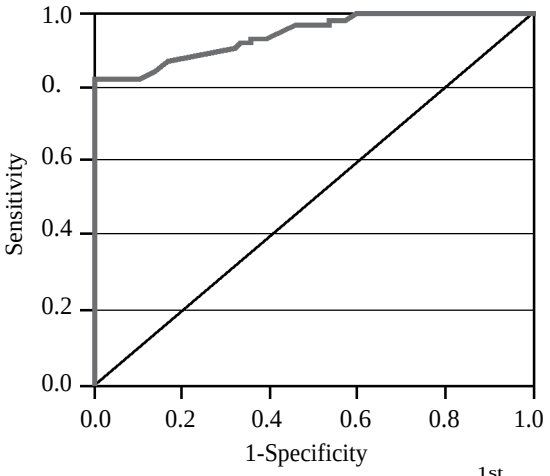

Fig. (3): ROC curve of RDW in the prediction of 1st hepatic decompensation.

Discussion

In the present study we noted that among the included groups males represented about 60% of them. A previous study, included 186 patients with liver cirrhosis, showed that male predominance of 2:1. The authors suggested that the possible reason for this is that men are involved in events that exposes them to risk factors that causes liver cirrhosis such as intravenous drug abuse and alcoholism **[4].**

In the present study we noted that hemoglobin level among the 3 decompensated groups was lower than the control compensated group. In accordance to our findings a previous study showed that anemia is a poor prognostic factor in patients with liver cirrhosis, and the severity of anemia increases with the stage of liver disease **[5].**

In the present study we noted that platelet count among the 3 decompensated groups was lower than the control compensated group. In accordance to our findings a previous study showed that decompensated cirrhosis is associated with low platelet count [6].

In the present study we noted that RDW values and percentage among the 3 decompensated groups were higher than the control compensated group. A previous study showed that elevated RDW values may associate with the severity of the liver disease [7]. Another study showed that RDW values were revealed to be a predictor of the degree of fibrosis in patients with chronic hepatitis C [8].

Meta-analyses by Cai and his colleagues [9] and Milas and his colleagues [10] confirmed that the progression of liver cirrhosis is accompanied by an increase in RDW and its elevation might even be a marker of poor prognosis. RDW was found to be a marker of increased mortality in alcoholic liver cirrhosis patients - independently of MELD score. Lately, RDW elevation was found to accompany severe inflammation and liver fibrosis in three independent studies on autoimmune hepatitis patients [11-13]. RDW was even proposed as a prognostic marker in the course of hepatocellular carcinoma [14,15].

The exact mechanisms underlying the association between RDW values and the severity of liver diseases are largely unclear. It was reported that inflammation might contribute to elevated RDW values by not only impairing erythrocyte maturation but also causing immature erythrocytes to enter the blood flow. It is plausible that inflammatory cytokines such as tumor necrosis factor (TNF)-α, interleukin (IL)-1β, and IL-6 might inhibit iron metabolism and the production of erythropoietin, resulting in synthesis disorders or abnormal activity of erythropoietin [16]. These inflammatory factors can disrupt iron utilization and reduce the responsiveness of bone marrow in response to erythropoietin. These mechanisms support the hypothesis that a high RDW may be associated with a tendency for upper GI bleeding [17].

Oxidative stress is common in liver disease with the characteristic of impairment of balance between oxidants and antioxidant defenses. Moreover, oxidative stress has a profound influence on erythrocyte homeostasis and survival, and low serum antioxidant concentrations have been shown to be related to increased RDW levels [18]. Therefore, oxidative stress may be another mechanism that leads to increased RDW levels in liver disease. Additionally, poor nutritional status such as iron deficiency, vitamin B12 or folate deficiency during liver disease can also lead to ineffective RBC production, leading to elevated RDW levels [19].

Another proposed mechanism for liver injury could be increased intestinal iron absorption via the intestinal pathway and increased iron accumulation within Kupffer cells. This results in the formation of reactive oxygen species, causing lipid peroxidation, leading to cellular protein and DNA degradation, and causing upregulation and activation of hepatic stellate cells and smooth muscle actin, eventually causing hepatic fibrosis [20].

Although the exact mechanism of action remains unknown, a possible hypothesis could be linked to increased iron storage and impaired usage, leading to ineffective erythropoiesis and causing elevated RDW. Given its frequent use, it can be utilized as a marker in chronic liver disease [21].

In the present study we found that the 3 groups of decompensated liver cirrhosis showed lower values of albumin than compensated liver cirrhosis group. A previous study showed that low albumin levels were associated with decompensated liver cirrhosis [22].

In the present study we found that the 3 groups of decompensated liver cirrhosis showed higher values of PT and INR than compensated liver cirrhosis group. Kim and his colleagues reported that the international normalized ratio (INR) was increased in liver cirrhosis [23]. Another study found that INR was correlated independently and significantly with liver fibrosis in chronic hepatitis C patients [24].

In the present study we found that the 3 groups of decompensated liver cirrhosis showed higher values of urea and createnine than compensated liver cirrhosis group. A previous study showed that by multivariate analysis, high urea level remained as an independent factor associated with hepatic decompensation [25].

In the present study we found that the 3 groups of decompensated liver cirrhosis showed lower values of sodium and potassium (reaching their lowest values among HE group) than compensated liver cirrhosis group. A previous study reported that electrolyte derangements, mainly hyponatremia and hypokalemia, represent major precipitating factors for HE [26]. It was suggested that chronic hyponatremia makes the brain more vulnerable to an ammonia induced osmotic disturbance with subsequent brain swelling. Alterations of potassium could also influence HE by altering normal ammonia metabolism. Hypokalemia rises renal ammoniagenesis and seems to result in both urinary excretion and venous secretion of ammonia as seen in humans with chronic potassium depletion [27].

In the present study we found that the 3 groups of decompensated liver cirrhosis showed higher MELD score than compensated liver cirrhosis group. The MELD is based on three biochemical variables that are readily available, reproducible,

and objective. These include: serum bilirubin, serum creatinine, and the international normalized ratio (INR) of prothrombin time. The MELD scores have been widely used for the assessment of prognosis in liver cirrhosis **[28].**

In the present study on applying ROC curve to assess the ability of RDW for the prediction of first encephalopathy among liver disease patients, we found that RDW at cut off value of >_13.95, RDW has Sensitivity and Specificity of 89% and 91%, respectively. Also, we found that RDW at cut off value of >_13.55, RDW has Sensitivity and Specificity of 84.5% and 85%, respectively to predict of first hematemesis among liver disease patients. We found that RDW at cut off value of >_13.45, RDW has Sensitivity and Specificity of 80.2% and 79%, respectively to predict newly developed ascites among liver disease patient conclusively RDW-seems to be a strong predictor of first encephalopathy, first hematemesis and newly developed ascites among liver disease patients.

A previous study found that RDW is significantly low in patients with no hepatic encephalopathy, but with no significant correlation with the grade of encephalopathy. Increased resistance to portal blood flow due to alteration of the hepatic architecture leads to dilatation of PV, splenomegaly, and formation of esophageal and gastric varices, variceal hemorrhage, ascites, hypersplenism, encephalopathy, etc. In cirrhosis, increased intrahepatic vascular resistance is thought to be located mainly in the hepatic sinusoids **[29].** A previous study suggested that RDW is a predictor of risk for GI bleeding **[17].**

In the present study, logistic regression analysis showed that age, urea and sodium were strong predictors of hepatic encephalopathy A previous study showed that, serum bilirubin serum creatinine and serum sodium were independently associated with the development of HE **[30].** In patients with cirrhosis, the kidneys are considered to be important for ammonia metabolism. In rats with acute and chronic liver failure, the kidneys switch from ammonia production to ammonia removal. Therefore, the presence of renal failure may impair the capacity of the kidneys to remove ammonia from the circulation. The results of this study, together with previous data, suggest that the impairment of renal function is an important risk factor of HE in cirrhosis **[31].** A study published by Riggio and his colleagues showed that low serum sodium was an independent factor related with the development of overt HE **[32].**

In the present study, predictors logistic regression analysis showed that urea and potassium were strong predictors of ascites. This can be explained as ascites in hepatic cirrhosis mainly establishes due to impaired kidney sodium excretion which disrupts the sodium balance and as a consequence fluid retention arises, leading to the expansion of extracellular fluid volume. The decreased excretion of sodium is due primarily to arterial vasodilation, which triggers neurohormonal responses such as the renin-angiotensin-aldosterone system (RAAS) and the sympathetic nervous system; responses that cause kidney vasoconstriction and sodium retention **[33].**

Conclusion: Our study documented a strong relationship between RDW and the development of first hepatic decompensation. So, RDW may serve as a potential predictor of liver decompensationand therefore a prognostic indicator for compensated liver disease.

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دور عرض توزيع خلايا الدم الحمراء فى توقع تحول التليف الكبدى من تعويضى إلى تليف لاتعويضى

المقدمة: عرض توزيع الخلايا الحمراء هو مقياس آلى لعدم تجانس أحجام خلايا الدم الحمراء (مثل كثرة الكريات) ويتم إجراؤه بشكل روتينى كجزء من تعداد خلايا الدم الكامل. يستخدم عرض توزيع الخلايا الحمراء فى التشخيص التفريقى لفقر الدم.

فى الآونة الأخيرة، أظهرت سلسلة من الدراسات أن عرض توزيع الخلايا الحمراء يمكن أن يكون بمثابة مؤشر جديد ومستقل للتشخيص لدى المرضى الذين يعانون من أمراض القلب والأوعية الدموية (مثل قصور القلب، وأمراض الشريان التاجي المستقرة، واحتشاء عضلة القلب الحاد، والسكتات الدماغية وارتفاع ضغط الدم الرئوى). كما تبين أن ارتفاع قيم عرض توزيع الخلايا الحمراء يرتبط بزيادة خطر الوفاة بين عامة السكان.

ومع ذلك، على حد علمنا، لم يتم تحديد دور قيمعرض توزيع الخلايا الحمراء التى تتنبأ بتعويض الكبد في تليف الكبد بشكل جيد. تم تصميم هذه الدراسة لدراسة دورعرض توزيع الخلايا الحمراء كمؤشر موسع للتنبؤ بتعويض الكبد وارتفاع ضغط الدم البابى لدى مرضى التليف الكبدى مما سيؤدى إلى تحسين كفاءة التشخيص.

الهدف من العمل: الهدف من هذه الدراسة هو تقييم دورعرض توزيع الخلايا الحمراء فى التنبؤ بمضاعفات تليف الكبد.

وبعد تحليل البيانات التى تم جمعها، تبين أن:

قيم عرض توزيع الخلايا الحمراء والنسبة المئوية لارتفاع عرض توزيع الخلايا الحمراء بين المجموعات الثلاث غير المعوضة كانت أعلى من مجموعة التحكم المعوضة.