ORIGINAL ARTICLE

Apparent diffusion coefficient of renal parenchyma and color Doppler ultrasound of intrarenal arteries in patients with cirrhosis related renal dysfunction

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KEYWORDS
Diffusion weighted imaging; Hepatorenal syndrome; Color Doppler ultrasound; Liver cirrhosis

Abstract Objectives: The aim of this work was to study the renal hemodynamic changes which occur with liver cirrhosis using diffusion weighted magnetic resonance imaging (DW-MRI) and renal color duplex Doppler ultrasound.

Patients and methods: Patients were divided into four groups: Group A: 15 cirrhotic patients with compensated liver cirrhosis, Group B: 15 cirrhotic patients with refractory ascites, Group C: 15 cirrhotic patients with hepatorenal syndrome, Group D: 10 healthy persons as a control. The apparent diffusion coefficient (ADCs) of the kidneys was calculated using low \(b\) values (ADC\(_{\text{low}}\)) and high \(b\) values (ADC\(_{\text{high}}\)). Color Doppler ultrasound was performed in interlobar and arcuate arteries to calculate resistive index (RI) and pulsatility index (PI) in all patients.

Results: ADC\(_{\text{low}}\) showed a statistically significant difference between patients with hepatorenal syndrome and other groups. Using ADC\(_{\text{high}}\) no significant difference between different groups was noted. RI and PI of both interlobar and arcuate arteries were significantly higher in all the patient groups than the control group (\(P < 0.0001\)). RI and PI of both interlobar and arcuate arteries were significantly higher in patients with hepatorenal syndrome.

Conclusion: Liver cirrhosis, even in the presence of refractory ascites, did not affect the ADC value of renal parenchyma, however ADC value is affected in renal parenchyma of patients with hepatorenal syndrome. Duplex-Doppler ultrasound of intrarenal arteries enables the early detection of renal hemodynamic disturbances in patients with liver cirrhosis.

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1. Introduction

The first description of disturbances in renal function in chronic liver diseases was made by Frerichs and Flint in two independent reports from the late nineteenth century (1). The impairment of kidney function is caused by severe renal...
arterial vasoconstriction due to complex changes in systemic hemodynamics (2) [Fig. 1] (3).

Retrospective studies have identified hepato-renal syndrome (HRS) in about 17% of the patients with ascites admitted to hospital and in more than 50% of deaths occurring among cirrhotic patients with liver failure (4).

The apparent diffusion coefficient (ADC), as a quantitative parameter calculated from diffusion-weighted magnetic resonance images, combines the effects of capillary perfusion and water diffusion in the extracellular extravascular space. Therefore, diffusion-weighted magnetic resonance imaging (DW-MRI) can be used to differentiate normal from abnormal tissue structure and might be useful in characterizing various renal abnormalities (5).

DW-MRI of renal disease is an evolving field and previous investigators have tried to investigate its role in characterization of focal renal lesions, parenchymal disease and renal infections (6–11). To the best of our knowledge, no previous study investigated its role in diagnosis of renal dysfunction in cirrhotic patients.

Duplex Doppler ultrasonography of the kidneys is an easy and non-invasive method to assess blood flow and arterial vascular resistance as a parameter for vasoconstriction (12,13). The arterial resistance index is the most widely used parameter to estimate the arteriolar vascular resistance (14).

The aim of this work is to study the renal hemodynamic changes which occur with liver cirrhosis using DW-MRI and renal color Doppler ultrasound for prediction and diagnosis of hepatorenal syndrome.

2. Patients and methods

This study included 45 patients with liver cirrhosis (27 males and 18 females) as a purposive non-probability sample. They were selected from those admitted to the Internal Medicine Department. In addition 10 healthy persons were selected as a control group. Written consents were taken from all the patients after thorough explanation and understanding of the study.

The study subjects were classified into 4 groups:

- **Group A**: included 15 cirrhotic patients with compensated liver cirrhosis and normal renal functions.
- **Group B**: included 15 cirrhotic patients with refractory ascites, and normal renal functions.
- **Group C**: included 15 cirrhotic patients with hepatorenal syndrome.
- **Group D**: included 10 healthy persons as the control group.

Exclusion criteria for this study include patients with clinical or laboratory evidence of diabetes mellitus or hypertension and patients known to have nephropathies. There was no history of recent nephrotoxic drugs uptake in all of our study groups.

![Fig. 1](image_url) Pathogenesis of circulatory abnormalities and renal failure in cirrhosis [adopted from Ginès and Schrier (3)].
The diagnosis of liver cirrhosis was based upon typical clinical and sonographic findings and laboratory investigations including liver and renal function tests.

2.1. Ultrasonographic examination

Ultrasonographic examination was performed for all the patient and control groups. The equipment used was GE E8 ultrasound. Doppler signals were taken from inter-lobar arteries and arcuate arteries in the cortex of both kidneys. Color Doppler ultrasound was used to help to identify the arteries.

The following parameters were calculated from each inter-lobar artery and arcuate artery: peak systolic velocity, end diastolic velocity, resistive index and pulsatility indices.

2.2. MR imaging

MR examination was done for all patients, using 1.5 Tesla system (Signa, GE medical system, Milwaukee, WI, USA). A body coil was used. Conventional MRI sequences; T1W axial and fat-suppressed (FS) T2W axial and coronal sequences, were acquired.

2.3. DW MR imaging

Respiratory triggered FS (spectral fat suppression) spin echo–echo planar imaging (SE-EPI) axial diffusion-weighted sequence at \( b \)-values of 0.50 and 100 s/mm\(^2\) (ADC\(_{\text{low}}\)) and \( b \) values of 400, 500 and 800 s/mm\(^2\) (ADC\(_{\text{high}}\)) was done. The following parameters were used: EPI factor = 95, TR/TE = 1600/62 ms, flip angle = 90°, slice thickness = 7 mm, distance factor = 30%, number of averages = 6, receiver bandwidth = 1735 Hz/pixel, field of view = 249 × 380, matrix = 94 × 192, acquisition time = 2–4 min (depending on patient’s respiratory cycle). Trace DW images and ADC maps were derived automatically on a voxel-by-voxel basis. Good-quality DW images and ADC maps could be obtained in all the patients.

![Image](image-url)

Fig. 2 Apparent diffusion coefficient (ADC) map, with (a) ADC\(_{\text{low}}\) and (b) (ADC\(_{\text{high}}\)) \( b \) value in patient of the control group. ADC\(_{\text{low}}\) was 2.81 (×10\(^{-3}\) mm\(^2\)/s), ADC\(_{\text{high}}\) was 2.14 (×10\(^{-3}\) mm\(^2\)/s).

### Table 1

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study groups (( n = 5 ) 5)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group A (( n = 15 ))</td>
<td></td>
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<tr>
<td></td>
<td>Group B (( n = 15 ))</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Group C (( n = 15 ))</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control group (( n = 10 ))</td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Age (years)</td>
<td>Range</td>
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</tr>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>37.5 ± 4.2</td>
</tr>
<tr>
<td>Sex</td>
<td>Male</td>
<td>10 66.7</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>5 33.3</td>
</tr>
</tbody>
</table>

Group A = patients with compensated liver cirrhosis.
Group B = patients with refractory ascites and normal renal functions.
Group C = patients suffering from hepatorenal syndrome.
* Significant (\( P < 0.05 \)).
2.4. Image analysis

Regions of interest (ROIs) for quantitative measurement of ADC were placed on a commercial workstation (Fig. 2). To measure the ADC of renal parenchyma, circular ROIs were placed on the renal parenchyma, without any preference for the cortex/medulla. Three ROIs were placed—one each in the upper pole, inter-polar region, and lower pole—and the mean ADC was calculated. The images show apparent diffusion coefficient (ADC) maps with different b-values: (a) b-value 50, (b) b-value 400, and (c) b-value 800, in a patient of the compensated cirrhotic group. ADC_low was 2.78 ($\times 10^{-3}$ mm$^2$/s), ADC_high was 2.12 ($\times 10^{-3}$ mm$^2$/s).

Fig. 3  Apparent diffusion coefficient (ADC) map, with (a) b value 50, (b) b value 400 and (c) b value 800 in patient of the compensated cirrhotic group. ADC_low was 2.78 ($\times 10^{-3}$ mm$^2$/s), ADC_high was 2.12 ($\times 10^{-3}$ mm$^2$/s).
of these three values was calculated. The ADC values were expressed as mean ± standard deviation in the form of $A \times 10^{-3}\text{mm}^2/\text{s}$ up to four decimal places. We did not evaluate ADC values in the renal cortex and medulla separately because as pointed out by previous studies, it may be difficult to position the ROI cursor accurately in these areas.

### 2.5. Statistical analysis

Data were checked, coded, entered and analyzed by using SPSS (The Statistical Package for Social Sciences) version 16.0 software. Mean, standard deviation, frequency distribution (minimal and maximal), Independent $t$-test, and One-way ANOVA were used for quantitative data to test significance of differences between the mean values of the study variables for comparison between more than two groups. Pearson correlation coefficient was used for determination of the correlation between the age, sex of different groups and the resistive index. The significance level was adopted at $P < 0.05$.

### 3. Results

#### 3.1. The study subjects were classified into the following groups

1. **Group A**: included patients with compensated liver cirrhosis. Their ages ranged from 30 to 45 years old with mean $= 37.5\text{ years} \pm 4.2$. They were 10 males and 5 females.

2. **Group B**: included patients with refractory ascites and normal renal functions. Their ages ranged from 40 to 63 years with mean $= 52 \pm 3.6$. They were 8 males and 7 females.

3. **Group C**: included patients suffering from hepatorenal syndrome. Their ages ranged from 42 to 65 years with mean $= 51 \pm 5.3$. They were 9 males and 6 females.

4. **Group D**: included ten healthy persons as a control group, their ages ranged from 22 to 43 years with mean $= 32 \pm 7.1$. They were 6 males and 4 females (Table 1).

The mean $ADClow$ in the renal parenchyma of the control group in the current study was $2.87 \pm 0.21 (\times10^{-3}\text{mm}^2/\text{s})$ (Fig. 2), while the mean $ADChigh$ was $2.16 \pm 0.13 (\times10^{-3}\text{mm}^2/\text{s})$, with no significant difference with patients with the cirrhotic group (Fig. 3). The mean $ADClow$ in the renal parenchyma of the patients with hepatorenal syndrome in the current study was $2.31 \pm 0.08 (\times10^{-3}\text{mm}^2/\text{s})$ (Fig. 4), while the mean $ADChigh$ was $2.10 \pm 0.11 (\times10^{-3}\text{mm}^2/\text{s})$ (Table 2).

There was no significant difference in the mean ADC value of renal parenchyma between different groups using high $b$ value.

### Table 2 ADC in patient groups and control group.

<table>
<thead>
<tr>
<th></th>
<th>Group A ($n = 15$)</th>
<th>Group B ($n = 15$)</th>
<th>Group C ($n = 15$)</th>
<th>Group D ($n = 10$)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$ADClow$ ($\times10^{-3}\text{mm}^2/\text{s}$)</td>
<td>$2.83 \pm 0.11$</td>
<td>$2.72 \pm 0.22$</td>
<td>$2.31 \pm 0.08$</td>
<td>$2.87 \pm 0.21$</td>
<td>A vs D = 0.84</td>
</tr>
<tr>
<td>$ADChigh$ ($\times10^{-3}\text{mm}^2/\text{s}$)</td>
<td>$2.14 \pm 0.13$</td>
<td>$2.11 \pm 0.12$</td>
<td>$2.10 \pm 0.11$</td>
<td>$2.15 \pm 0.13$</td>
<td>A vs B, $P = 0.95$</td>
</tr>
</tbody>
</table>

* Significant ($P < 0.05$).
Using ADC_{low}, the mean ADC value of renal parenchyma in patients with frank HRS (Group C) was significantly lower than other groups (Fig. 4). On the other hand, even with ADC_{low}, there was no significant difference between the ADC values of patients with liver cirrhosis (Group A), and patients with refractory ascites (Group B).

**Table 3**  Measurements of resistive index (RI) of patient groups and control group.

<table>
<thead>
<tr>
<th>Group</th>
<th>(n = 15)</th>
<th>(n = 15)</th>
<th>(n = 15)</th>
<th>(n = 10)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Range</td>
<td>Range</td>
<td>Range</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Interlobar artery</td>
<td>0.57-0.72</td>
<td>0.75-0.82</td>
<td>0.75-0.82</td>
<td>0.50-0.56</td>
<td>0.0001*</td>
</tr>
<tr>
<td></td>
<td>0.638 ± 0.037</td>
<td>0.78 ± 0.023</td>
<td>0.787 ± 0.017</td>
<td>0.538 ± 0.011</td>
<td>A vs B, P = 0.0001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>A vs C, P = 0.0001*</td>
</tr>
<tr>
<td>Arcuate artery</td>
<td>0.55-0.70</td>
<td>0.74-0.80</td>
<td>0.74-0.80</td>
<td>0.50-0.54</td>
<td>0.0001*</td>
</tr>
<tr>
<td></td>
<td>0.621 ± 0.037</td>
<td>0.768 ± 0.021</td>
<td>0.766 ± 0.016</td>
<td>0.526 ± 0.008</td>
<td>B vs C, P = 0.480</td>
</tr>
</tbody>
</table>

* Significant (P < 0.05).

**Table 4**  Measurements of pulsatility index (PI) of patient groups and control group.

<table>
<thead>
<tr>
<th>Group</th>
<th>(n = 15)</th>
<th>(n = 15)</th>
<th>(n = 15)</th>
<th>(n = 10)</th>
<th>P value</th>
</tr>
</thead>
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<tr>
<td></td>
<td>Range</td>
<td>Range</td>
<td>Range</td>
<td>Range</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Interlobar artery</td>
<td>0.90–1.40</td>
<td>1.60–2.40</td>
<td>1.70–2.40</td>
<td>0.75–0.81</td>
<td>0.0001*</td>
</tr>
<tr>
<td></td>
<td>1.167 ± 0.146</td>
<td>1.94 ± 0.257</td>
<td>1.94 ± 0.214</td>
<td>0.776 ± 0.019</td>
<td>A vs B, P = 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>A vs C, P = 0.001*</td>
</tr>
<tr>
<td>Arcuate artery</td>
<td>0.93–0.130</td>
<td>1.60–2.30</td>
<td>1.54–2.25</td>
<td>0.75–0.80</td>
<td>0.0001*</td>
</tr>
<tr>
<td></td>
<td>1.07 ± 0.107</td>
<td>1.81 ± 0.236</td>
<td>1.81 ± 0.206</td>
<td>0.768 ± 0.017</td>
<td>B vs C, P = 0.970</td>
</tr>
</tbody>
</table>

* Significant (P < 0.05).

**Fig. 5**  Color duplex scanning of intrarenal artery in a patient of the control group, RI = 0.53.
Also, there was no significant difference in ADC of the kidneys with high resistive index (>0.7) and the kidneys with normal resistive index (<0.7) (Table 5).

As regards renal Doppler RI measurements, RI of both interlobar and arcuate arteries was significantly higher in all patient groups than the control group ($P < 0.0001$). RI of both interlobar and arcuate arteries was significantly higher in patients with hepatorenal syndrome than patients with compensated cirrhosis ($P < 0.0001$) (Table 3) (Figs. 4 and 5).

As regards renal Doppler PI measurements, PI of both interlobar and arcuate arteries was significantly higher in all patient groups than the control group ($P < 0.0001$). PI of both interlobar and arcuate arteries was significantly higher in patients with refractory ascites than patients with compensated cirrhosis ($P < 0.0001$) (Table 4). But there were no significant changes of PI between patients with refractory ascites and patients with hepatorenal syndrome (Table 4).

### 4. Discussion

Advanced chronic liver disease is responsible for a significant number of physiological changes that affect the renal hemodynamics and renal function (15) (Fig. 6).

The revised definition states that HRS is a potentially reversible syndrome occurring in patients with cirrhosis, ascites and liver failure. It is characterized by impaired renal function, marked alterations in the cardiovascular function and over-activity of the endogenous vasoactive systems. The resulting vasoconstriction in the kidney causes low glomerular filtration rate (GFR), also, there is a decreased vascular resistance due to splanchnic and peripheral arterial vasodilatation (16).

In the literature, there are some articles about DW-MRI of the kidneys in systemic diseases as hypertension (17) and familial Mediterranean fever (18). At high $b$ values, ADC is dominated only by diffusion effects, and the DWI is influenced by perfusion effects at lower diffusion factors ($b$ values) (19). In the current study, we calculated ADC at both low and high $b$ values.

The hydration state of the patient has been previously described as having an effect on the resulting ADC, because the hydrated kidneys have shown a higher ADC than have the dehydrated kidneys (20). This finding has not be confirmed by recent studies (21), in the current study, we did not consider the hydration state of the patient.

Early reports suggested the importance of measurement of ADC in the cortex and medulla separately (22–24), but it may be difficult to position the ROI cursor accurately in these areas as pointed out by recent reports (25,26), in the current study, we did not evaluate ADC values in the renal cortex and medulla separately.

The mean ADC in normal renal parenchyma in studies use whole renal parenchyma ranged from $2.26 \pm 0.36 \times 10^{-3} \text{mm}^2/\text{s}$ to $3.54 \pm 0.47 \times 10^{-3} \text{mm}^2/\text{s}$ (21,27). In the current study, the mean $\text{ADC}_{\text{low}}$ was $2.87 \pm 0.21 \times 10^{-3} \text{mm}^2/\text{s}$, while the mean $\text{ADC}_{\text{high}}$ was $2.16 \pm 0.13 \times 10^{-3} \text{mm}^2/\text{s}$ in agreement with the previous reports.

In the current study, there was a statistically significant difference between $\text{ADC}_{\text{low}}$ in patients with hepatorenal syndrome, and control group, patient with compensated cirrhosis and patients with cirrhosis and ascites ($P = 0.01$), while there was no significant difference between the control

<table>
<thead>
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<th></th>
<th>ADC$_{\text{low}}$</th>
<th>ADC$_{\text{high}}$</th>
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<tbody>
<tr>
<td>High RI &gt; 0.7</td>
<td>2.65 ± 0.18</td>
<td>2.12 ± 0.12</td>
</tr>
<tr>
<td>Low RI &lt; 0.7</td>
<td>2.68 ± 0.22</td>
<td>2.12 ± 0.82</td>
</tr>
<tr>
<td>$P$</td>
<td>0.45</td>
<td>0.78</td>
</tr>
</tbody>
</table>

*Significant ($P < 0.05$).

![Fig. 6](image) Color duplex scanning of intrarenal artery in a patient of the cirrhotic group, RI = 0.7.
group and cirrhotic patients with and without ascites with normal renal function tests. This means that the ADC of the kidneys is not affected in cirrhotic patients unless hepatorenal syndrome has complicated the case.

On the other hand, there was no significant difference between $\text{ADC}_{\text{high}}$ of renal parenchyma between all the groups. This probably reflects the fact that the use of high $b$ values provides information on microscopic water motion in the extracellular extravascular space, which approximates the true diffusion of the tissue (28). With lower $b$ values, however, an additional effect of movement within vascular and tubular structures is seen, effect is usually called the perfusion contribution. Thus, in the case of a $b$ value range starting above 200 s/mm$^2$, the resulting ADC approximates true diffusion. On the other hand, low $b$ values ($< 100$ s/mm$^2$) are strongly influenced by perfusion effects and only a little by diffusion (29).

Using color Doppler ultrasound, the evaluation of renal hemodynamics in patients with liver cirrhosis is mainly based on the index of resistance of the renal arteries (30,31). In the current study, renal duplex Doppler ultrasonography was done in the right or left kidney at the interlobar and arcuate arteries to measure the resistive index and the pulsatility index.

This study on 45 adult patients, with compensated and decompensated liver cirrhosis with and without ascites, showed that mean renal arterial RI for these patients was higher than for the 10 healthy control subjects. Furthermore, RI was higher in cirrhotic patients with refractory ascites than in those without ascites. These results suggest that the degree of renal vasoconstriction varies with the severity of ascites. At the different stages of liver cirrhosis there may be varying degrees of renal vasoconstriction which can lead to a decrease in renal blood flow, resulting in oliguria and anuria.

The mean RI in patients with hepatorenal failure was 0.787 ± 0.017, and PI was 1.94 ± 0.214 which is significantly different from the mean RI and PI in the control group and patients with compensated cirrhosis ($P < 0.0001$), but no significant difference with patients with refractory ascites. These results are in agreement with a recent study by Wang et al. (32), who found a mean renal arterial RI 0.74 ± 0.02 in patients with de-compensated livers. Also, Colli et al., (33) have shown that RI is significantly higher in de-compensated non azotemic cirrhotic patients with ascites than in compensated cirrhotic patients without ascites.

The increase in renal vascular RI in cirrhotic patients with ascites could be explained by a physiological homeostatic response to vascular underfilling occurring in ascitic patients. When the vascular underfilling is moderate, the renal vasoactive substances are effectively counterbalanced by increased renal synthesis of prostaglandins so that renal blood flow and GER remain normal. In contrast, when the vascular underfilling is severe, intense stimulation of endogenous vasoconstrictor systems occurs, producing renal vasoconstriction and impairment of renal blood flow and GFR (34). Paternon et al., (35) concluded that intrarenal blood flow is preserved in cirrhotic patients by intrarenal mechanisms until the ascites becomes refractory. When this regulation fails renal ischemia causes tubular necrosis, azotaemia and oliguric renal failure.

Also our results are in agreement with Götzberger et al., (36) who found that RI was significantly higher in ascitic patients compared to non-ascitic patients (0.74 vs 0.67, $P < 0.01$) and in non-ascitic patients with liver cirrhosis than in control subjects (0.67 vs 0.62, $P < 0.01$). They concluded that intra-renal RI measurement is a predictor of renal vasoconstriction and serves to detect early renal function impairment in cirrhotic patients.

Several studies have shown that a normal mean renal RI is approximately 0.60 for subjects without preexisting renal disease (37). In general, 0.70 is now considered to be the upper threshold of the normal RI in adults (38,39).

In the current study, the ADC values of patients with high resistive index $> 0.7$ did not differ from those of patients with normal intra-renal resistive index $< 0.7$. In previous studies, there was no significant correlation between hypertension and ADC values (17), or between ADC values and early obstruction (40).

Though there was no affection of ADC values in the kidneys in the early phases of cirrhosis and ascites, still this study has significant clinical implication. The color Doppler ultrasound of intrarenal arteries is significant in early detection of the renal impairment, as the resistive index and pulsatility index increase early before the development of HRS. On the other hand, color Doppler ultrasound cannot differentiate between the patient with true HRS and patients with just refractory ascites, because in both patients there is high RI. DWI can differentiate between patients with true HRS and those who do not have HRS, because ADC value is affected only in patients with HRS. This may have a significant impact in the management of the patients.

5. Conclusion

Both DW-MRI and color Doppler ultrasound are useful tools in the management of patients with liver cirrhosis and suspected hepatorenal syndrome. DWI can be useful in confirmation of diagnosis in patients with suspected HRS. Duplex-Doppler ultrasound of intra-renal arteries is a simple, effective and non-invasive method which enables the early detection of renal hemodynamic disturbances in patients with liver cirrhosis. We recommend integration of Doppler ultrasound of the intra-renal arteries in the routine ultrasound examination of patients with liver cirrhosis, and DWI of renal parenchyma is recommended in patients suspected to have HRS.

Conflict of interest

There is no conflict of interest to declare.

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