Liver Stiffness is Reduced to Normal After Successful Renal Transplantation: A Prospective Cohort Study

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Background: **Liver stiffness (LS) may be falsely elevated in patients on maintenance hemodialysis (MHD) due to fluid overload. We measured LS change by transient elastography (TE) in MHD patients before and after successful renal transplantation.** ***Method:*** **Adults on ≥2 years of MHD, without additional risk factors for liver fibrosis or fluid overload, and planned for renal transplantation were prospectively recruited. LS was measured on two occasions, i.e., within two weeks before transplantation (pre-Tx LS) and after ≥ 3 months after successful transplantation (post-Tx LS). The participants with pre-Tx LS ≤ 7.0 KPa and >7.0 KPa were classified as "Group I" and "Group II," respectively. Categorical and numerical data are expressed as ratio/proportions and mean (SD), respectively.** ***Results:*** **Paired data from 43 participants (males 42 [97.7%]; age 32 [11] years) were analyzed. The pre-Tx and post-Tx LS of the entire cohort, measured at 307 (198) days of interval, were 8.5 (7.3) KPa and 6.7 (3.1) KPa, respectively. Before transplantation, 21 (49%) participants belonged to Group II and 22 (51%) to Group I. Among the Group II participants, 12 (57%) showed LS normalization after 312 (182) days of transplantation. Of the 22 participants in Group I, three (13.6%) showed LS elevation to >7.0 KPa after 303 (217) days of transplantation. The mean LS changes among the overall cohort, Group II, and Group I were −1.8 KPa, −4.1 KPa, and +0.2 KPa, respectively.** ***Conclusion:*** **LS in people on MHD may be falsely elevated, which is likely to normalize after successful renal transplantation.** **(J Clin Exp Hepatol 2022;12:1445–1450)**

Patients with significant liver fibrosis have reduced liver function[1] and are at high risk for prolonged hospital stay, postoperative complications, and death following any major surgery such as organ transplantation.[2] People on maintenance hemodialysis (MHD) have a high prevalence of hepatitis C virus (HCV) infection[3] and high risk for the progression of liver fibrosis than those with normal renal function.[4] All those with suspected liver disease need evaluation for the severity of liver fibrosis before renal transplantation.

Liver biopsy, the gold standard for the assessment of liver fibrosis, is largely replaced by non-invasive methods of fibrosis assessment.[5] Such non-invasive methods include blood tests, radiological evaluation, or elastography.[6] Transient elastography (TE) is one of the most contemporary non-invasive techniques for fibrosis assessment. The TE measures the liver stiffness (LS) as a surrogate marker of liver fibrosis, and the stiffness is expressed in KiloPascal (KPa). The LS may also be increased by several non-hepatic factors such as post-prandial state,[7] acute inflammation in the liver,[8] biliary obstruction,[9] and hepatic congestion in the presence of fluid overload, such as congestive cardiac failure or end-stage renal failure.[10] The application of non-invasive methods for fibrosis assessment has certain limitations in patients on MHD.[11]

People on MHD are in a state of fluid overload which may result in falsely elevated TE value and overestimation of liver fibrosis. Such a false elevation in TE may lead to either unnecessary liver biopsy to assess liver fibrosis or deferment of transplant surgery. The data on the performance of TE for liver fibrosis assessment in the MHD population is controversial. Multiple studies have attempted to study the effect of fluid overload on LS by comparing the TE before and after a single session of hemodialysis. A single session of hemodialysis may not immediately restore the euvolemic state and may fail to normalize the falsely elevated LS. True euvolemia could only be assured after the achievement of normal renal function following successful renal transplantation.

We aimed to study the change in LS, as measured by TE, in MHD patients after successful renal transplantation and achievement of normal renal function.

Keywords: hemodialysis, maintenance hemodialysis, chronic kidney disease, end-stage renal disease, liver stiffness measurement, fibroscan, liver fibrosis, renal transplantation

Received: 19.4.2022; *Accepted:* 2.6.2022; *Available online 9 June 2022*

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Abbreviations: ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CKD: Chronic kidney disease; HCV: Hepatitis C virus; MHD: Maintenance hemodialysis; TE: Transient elastography; LS: Liver stiffness

https://doi.org/10.1016/j.jceh.2022.06.002

METHOD

This prospective, observational study was conducted in the departments of Nephrology and Gastroenterology in Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India. The participants were recruited and followed from July 2018 to June 2021. Adults on MHD for ≥2 years with prospects for renal transplantation were included. Patients were excluded if they had either (i) coexisting cardiac diseases on clinical examination, electrocardiography, or echocardiographic evaluation; (ii) clinical, laboratory, or radiological evidence of liver or biliary disease (total serum bilirubin >2 mg/dL or serum alanine aminotransferase [ALT] > 80 IU/L, i.e., >2 times upper limits of normal); (iii) active or resolved hepatitis B or HCV infection, alcoholic liver disease, or pre-existing CLD; (iv) anti-HIV positivity; (v) focal lesion in the liver such as simple cyst; (vi) clinical or laboratory evidence of active bacterial or viral infections; (vii) patients on peritoneal dialysis; (viii) patients with ascites, regardless of its cause; (ix) history of significant alcohol intake (men >30 g/wk; women >20 g/wk); or (x) body mass index beyond the normal range of 18.5–25.0 kg/m^2.

Each participant underwent LS measurement on two occasions. The first TE measurement was done within two weeks prior to renal transplantation (pre-transplant observation, pre-Tx LS); the second measurement was done after ≥3 months of renal transplantation in those with normal functioning allograft (post-transplant observation, post-Tx LS). LS was measured with TE (FibroScan®, Echosens, France). All the LS measurements were done in the morning hours after overnight fasting by a single operator (TSN) who had the experience of >5000 examinations. All TE measurements were done within 24 h of their last hemodialysis session. To abide by the widely accepted international recommendations, an LS measurement was considered to be acceptable if it satisfied the following characteristics (i) a minimum of ten readings were taken with (ii) a success rate of ≥60% and (iii) ratio of interquartile range/median being ≤0.30. The median of the ten successful readings was taken as the patient's TE score.[12]

The summary analysis of global data on LS in healthy individual suggests mean TE up to 4.7 KPa is normal in people with BMI <30 kg/m^2.[13] The hepatology colleagues consider LS > 7.0 as evidence suggestive of significant liver fibrosis.[14] Accepting a more conservative approach to making our results more reliable, we classified the participants with pre-Tx LS ≤ 7.0 KPa and >7.0 KPa as "Group I" and "Group II," respectively.

We also calculated two other indices for the assessment of liver fibrosis (aspartate aminotransferase [AST] platelet ratio, AST-platelet ratio index [APRI], and fibrosis-4 index [FIB-4]).[6] Their calculation needs laboratory values for ALT, AST, and platelet counts. All the necessary laboratory investigations were done in the fasting state on the day of LS measurement. The APRI and FIB-4 >1.5 and 3.25 are universally accepted as an indicator of significant fibrosis.[15]

Statistical analysis

The LS, in patients on MHD without any evidence of liver disease, is reported as 7.7 ± 2.3 KPa.[16] We estimated the required sample size, assuming the mean difference of 1.0 ± 2.3 KPa in paired observation taken before and after renal transplantation, as 43 pairs to achieve a power of 80% and a level of significance of 5% using two-sided tests of significance. Assuming a 10% data loss due to various reasons, we plan to include 47 patients. Categorical and numerical data are expressed as ratios/proportions and mean (standard deviation, SD). The categorical variable was compared using the chi-square test. The unpaired and paired numerical data are compared using a t-test. Data were analyzed with STATA/IC 16.1 (StataCorp. 2019. Stata Statistical Software: Release 16. College Station, TX: StataCorp LLC).

The study was approved by the institute ethics committee (2017-171-IMP-99B), and the participants were enrolled after obtaining written consent. The study was performed in accordance with the 1964 Declaration of Helsinki and its later amendments.

RESULTS

Of the 78 patients enrolled, 45 could undergo renal transplantation—two patients who had organ rejection after transplantation were excluded. Data from the remaining 43 patients, who achieved normal renal function after transplantation, were included in the analysis. Most of the participants were young males (males 42 [97.7%]; age 32 [11] years).

Pre-transplant clinical characteristics, laboratory parameters, and liver fibrosis parameters of the participants are summarized in Table 1. Among 43 patients, 22 participants (51.2%) with normal LS were included in Group I, whereas 21 participants (48.8%) with elevated LS were included in Group II. The characteristics of the participants in Group I and Group II were comparable (Table 1). The participants were reassessed after 307 (198) days after renal transplantation. The changes in laboratory parameters, an indicator of liver fibrosis, and LS scores are summarized in Table 2.

In Group II, LS was normalized in 12 of the 21 participants (57.1%) participants on repeat assessment performed after a mean (SD) 312 (182) days of transplantation. In Group I, three of the 22 (13.6%) showed elevation of LS beyond 7.0 KPa on repeat measurement after a mean (SD) 303 (217) days of transplantation (Fig. 1). The mean reduction in LS values among the overall cohort

Table 1 Pre-transplant Clinical Characteristics, Laboratory Investigations, and Indices of Liver Fibrosis in Study Participants.

Parameters	All the participants (n = 43)	Subgroups		
		Group I: Participants with LS ≤ 7.0 Kilopascal (n = 22)	Group II: Participants with LS > 7.0 Kilopascal (n = 21)	*P* value
Males	42 (97)	21 (95)	21 (100)	1.00
Age (years)	32 (11)	29 (9)	36 (11)	0.02
Hemoglobin (g/dL)	10 (2.5)	10.5 (2.1)	9.8 (2.8)	0.23
Platelet ($\times 10^9$/L)	176 (49)	169 (56)	184 (41)	0.34
Creatinine (mg/dL)	8.0 (3.1)	8.0 (3.1)	7.2 (2.9)	0.12
Total bilirubin (mg/dL)	0.6 (0.3)	0.6 (0.3)	0.7 (0.3)	0.47
ALT (IU/L)	21 (14)	22 (12)	21 (16)	0.81
AST (IU/L)	23 (11)	23 (10)	23 (13)	0.88
Albumin (g/dL)	4.4 (0.7)	4.5 (0.6)	4.2 (0.7)	0.13
Liver stiffness (KPa)	8.5 (7.3)	5.5 (1.0)	11.8 (9.5)	<0.01
Controlled attenuation parameter (dB/m)	201 (40)	200 (27)	203 (51)	0.77
APRI	0.35 (0.20)	0.38 (0.21)	0.32 (0.20)	0.39
FIB-4	0.97 (0.49)	0.91 (0.46)	1.03 (0.52)	0.42
Interval between transplant and repeat measurement (days)	307 (198)	303 (217)	312 (182)	0.89
Etiology of kidney disease				0.45
CGN	22 (51)	10 (45)	12 (57)	
DN	13 (30)	7 (32)	5 (24)	
CIN	8 (19)	5 (23)	4 (19)	

Note: Data are presented as number (%) or mean (SD). ALT, alanine aminotransferase; AST, aspartate aminotransferase; APRI, AST-platelet ratio index; FIB-4, fibrosis-4 index; CGN, chronic glomerulonephritis; DN, diabetic nephropathy; IGN, interstitial glomerulonephritis. groups are compared using chi-square and t-test.

and Group II were 1.8 KPa and 4.1 KPa, respectively. The mean decrease in stiffness in Group II was significantly higher than in Group I (Table 2). The mean increase in LS in Group I was +0.2 KPa.

The median (interquartile range, IQR) LS of the cohort before and after transplantation were 6.9 (5.5–8.4) and 6.0 (4.6–7.8), respectively. The median interval between the repeated measurements was 246 (119–470) days. The

Table 2 Comparison of Laboratory Parameters, Liver Fibrosis Indices, and Liver Stiffness Before and After Kidney Transplantation.

Parameters	Overall (n = 43)			Group I: Participants with LS ≤ 7.0 Kilopascal (n = 22)			Group II: Participants with LS > 7.0 Kilopascal (n = 21)		
	Before Tx	After Tx	*P*-value	Before Tx	After Tx	*P* -value	Before Tx	After Tx	*P* -value
Platelet ($\times 10^9$/L)	176 (49)	159 (48)	0.02	169 (56)	150 (45)	0.12	184 (41)	169 (52)	0.10
ALT (IU/L)	21 (14)	30 (18)	0.02	22 (12)	33 (20)	0.04	21 (16)	26 (14)	0.28
AST (IU/L)	23 (11)	26 (15)	0.27	23 (10)	27 (16)	0.30	23 (13)	25 (15)	0.60
Creatinine (mg/dL)	8.0 (3.1)	1.4 (0.5)	<0.01	8.0 (3.1)	1.4 (0.5)	<0.01	7.2 (2.9)	1.4 (0.6)	<0.01
Liver stiffness (KPa)	8.5 (7.3)	6.7 (3.1)	0.02	5.5 (1.0)	5.7 (1.2)	0.42	11.8 (9.5)	7.7 (4.0)	0.01
Controlled attenuation parameter (dB/m)	201 (40)	220 (45)	0.01	200 (27)	205 (25)	0.50	203 (51)	236 (56)	<0.01
APRI	0.35 (0.20)	0.52 (0.71)	0.11	0.38 (0.21)	0.65 (0.96)	0.20	0.32 (0.20)	0.39 (0.23)	0.22
FIB-4	0.97 (0.49)	1.08 (0.67)	0.16	0.91 (0.46)	0.96 (0.44)	0.58	1.03 (0.52)	1.20 (0.84)	0.18

Note: Data are presented as number (%) or mean (SD). ALT, alanine aminotransferase; AST, aspartate aminotransferase; APRI, AST-platelet ratio index; FIB-4, fibrosis-4 index; Tx, transplantation. groups are compared using non-parametric Wilcoxon signed rank test.

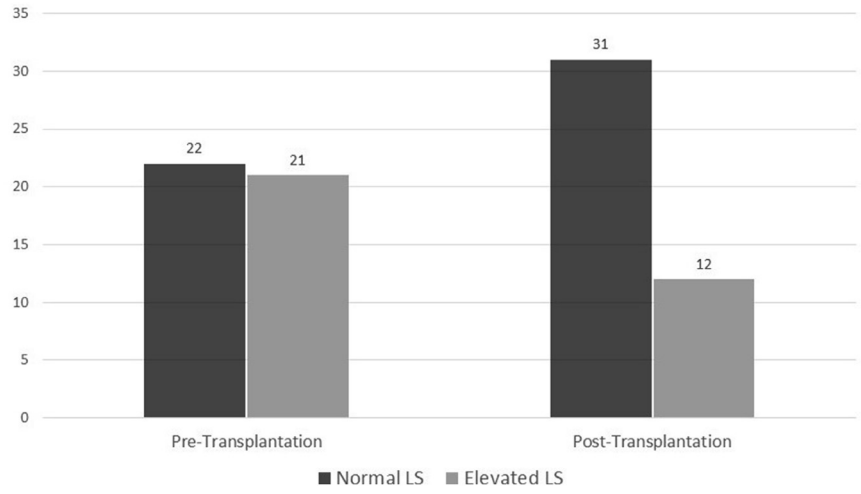

Figure 1 Number of participants with normal liver stiffness (≤7.0 KPa) and elevated liver stiffness (>7.0 KPa) before and after successful renal transplantation.

median LS in Group I, measured after an interval of 268 (99–470) days, was changed from 5.6 (4.9–6.1) KPa to 5.8 (4.5–6.3) KPa. Similarly, the median LS in Group II, measured after an interval of 246 (175–460) days, was changed from 8.4 (7.7–11.1) KPa to 6.3 (4.9–8.5) KPa.

Pre-transplant APRI and FIB-4 and post-transplant FIB-4 for none of the participants were suggestive of significant liver fibrosis. For one participant, the post-transplant APRI was increased from 0.35 to 4.8.

DISCUSSION

We followed a cohort of 43 MHD patients and reported the changes in their non-invasive parameters of liver fibrosis, including LS as assessed by TE, APRI, and FIB-4. These parameters were evaluated after a mean interval of 307 (198) days after successful kidney transplantation. Pre-transplant LS were elevated among half of the participants. The mean value of TE in Group II participants was reduced from 11.8 KPa before transplant to 7.7 KPa after transplant ($P < 0.01$) on repeat measurement after a mean interval of 312 (182) days.

Liver biopsy, the gold standard for liver fibrosis assessment, has several disadvantages. Some of the major disadvantages are invasive nature, need for hospitalization, inherent financial expenses, pain during the procedure, requires expertise to perform and interpret the biopsy, and finite risk of complications including death.[5] The risk of bleeding following liver biopsy is further increased by secondary to uremia in MHD patients.[5] The use of non-invasive methods and indices has almost replaced liver biopsy for the assessment of liver fibrosis. Three non-invasive methods, namely APRI, FIB-4, and LS measurement by TE, are the most widely and extensively validated and have the best diagnostic performances.[6]

The calculation of APRI and FIB-4 indices require serum ALT and AST levels. The serum levels of ALT and AST are relatively lower among MHD patients[17] than those with normal renal function, which underestimates the fibrosis scores and limits their application to the dialysis population. In a cohort of 165 dialysis patients with chronic HCV infection, the cut-off to define cirrhosis by APRI was 0.28 and 1.91, respectively,[18] which are much lower than 2.0 and 3.25 recommended for those with normal renal function.[19]

TE measures LS and has gained momentum for fibrosis assessment in patients with normal function. For the population with normal renal function, significant fibrosis, severe fibrosis, and cirrhosis are given by TE value ≥ 7.0 KPa, ≥9.5 KPa, ≥12.5 KPa, respectively.[14] LS is falsely elevated in the presence of hepatic congestion secondary to biliary obstruction, back pressure due to congestive cardiac failure, edema secondary to acute hepatitis insult, or volume overload. Advanced renal failure is a state of chronic volume overload and can falsely increase the LS. However, the diagnostic performance of TE is found to be comparable to liver biopsy in MHD patients with liver disease.[20] The data on LS in MHD patients without liver disease are minimal. In a recent study of 171 MHD patients without risk factors for liver disease, 21% of participants had LS suggestive of advanced fibrosis.[21] However, the fibrosis was not assessed with liver biopsy in this study. Another study, although it included only 17 participants, found LS suggestive of significant fibrosis in 41%.[22]

The data from previous studies performed in MHD patients with chronic viral hepatitis are inconsistent about the effect of a single session of hemodialysis on LS. Studies have shown that following a single session of hemodialysis, the LS may reduce,[23] remain unchanged,[24] or increase.[22] All

these studies had attempted to study the effect of volume overload on LS by removing the fluid following a single session of hemodialysis. A hemodialysis session may not be adequate to restore the euvolemia; further, it may take time to reduce the LS after the restoration of euvolemia.

Our study is the first to report the change in LS after successful renal transplantation and normalization of renal function. Our data suggested that the LS of the participants who had normal stiffness before transplantation does not change after transplantation. In contrast, the LS of those with high values before transplantation is reduced to normal after transplantation in a proportion of patients. The other liver fibrosis indices, such as APRI and FIB-4, do not change after transplantation, which further supports the hypothesis that liver fibrosis was unlikely to be responsible for the elevation of LS among people on MHD. Our data suggest that in almost half of MHD patients with elevated LS, the stiffness seems to be falsely elevated, and liver biopsy may be avoided in them.

Our study has a limitation of the long interval between the two LS measurements. We had planned for a repeat LS measurement after three months of renal transplantation, but it was delayed due to the ongoing COVID-19 pandemic. The long interval could have led to liver steatosis, which is reflected in the increased controlled attenuation parameter (CAP). New-onset fatty liver disease is common among renal transplant recipients.[25] Steatosis is likely to increase the LS, which will lead to underestimation of the reduction in LS following transplantation which makes our results more robust to believe.

In conclusion, LS in people on MHD may be falsely elevated due to chronic fluid overload, and it does not necessarily suggest significant liver fibrosis. The LS is likely to normalize in many of them who follow renal transplantation.

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CONFLICTS OF INTEREST

The authors have none to declare.

ACKNOWLEDGEMENTS

None.

FUNDING

Dr. Dharmendra Singh Bhadauria has received an intramural research grant from Sanjay Gandhi Postgraduate Institute of Medical Sciences (2017-171-IMP-99B). Prachi Tiwari received financial support from the Indian Council of Medical Research by extramural research grant no VIR/18/2018/ECD-I.

REFERENCES

1. Tsochatzis EA, Bosch J, Burroughs AK. Liver cirrhosis. *Lancet (London, England)*. 2014 May 17;383:1749–1761.
2. O'Leary JG, Yachimski PS, Friedman LS. Surgery in the patient with liver disease. *Clin Liver Dis*. 2009 May;13:211–231.
3. Goel A, Seguy N, Aggarwal R. Burden of hepatitis C virus infection in India: a systematic review and meta-analysis. *J Gastroenterol Hepatol*. 2019 Feb;34:321–329.
4. Espinosa M, Martin-Malo A, Alvarez de Lara MA, et al. Risk of death and liver cirrhosis in anti-HCV-positive long-term haemodialysis patients. *Nephrol Dial Transplant*. 2001 Aug;16:1669–1674. official publication of the European Dialysis and Transplant Association - European Renal Association.
5. Rockey DC, Caldwell SH, Goodman ZD, et al. Liver biopsy. *Hepatology*. 2009 Mar;49:1017–1044.
6. EASL-ALEH Clinical Practice Guidelines. Non-invasive tests for evaluation of liver disease severity and prognosis. *J Hepatol*. 2015 Jul;63:237–264.
7. Alvarez D, Orozco F, Mella JM, et al. Meal ingestion markedly increases liver stiffness suggesting the need for liver stiffness determination in fasting conditions. *Gastroenterol Hepatol*. 2015 Aug-Sep;38:431–435.
8. Arena U, Vizzutti F, Corti G, et al. Acute viral hepatitis increases liver stiffness values measured by transient elastography. *Hepatology*. 2008 Feb;47:380–384.
9. Millonig G, Reimann FM, Friedrich S, et al. Extrahepatic cholestasis increases liver stiffness (FibroScan) irrespective of fibrosis. *Hepatology*. 2008 Nov;48:1718–1723.
10. Perazzo H, Veloso VG, Grinsztejn B, et al. Factors that could impact on liver fibrosis staging by transient elastography. *Int J Hepatol*. 2015;2015624596.
11. Goel A, Bhadauria DS, Aggarwal R. Hepatitis C virus infection and chronic renal disease: a review. *Indian J Gastroenterol: official journal of the Indian Society of Gastroenterology*. 2018 Nov;37:492–503.
12. Castera L, Foucher J, Bernard PH, et al. Pitfalls of liver stiffness measurement: a 5-year prospective study of 13,369 examinations. *Hepatology*. 2010 Mar;51:828–835.
13. Bazerbachi F, Haffar S, Wang Z, et al. Range of normal liver stiffness and factors associated with increased stiffness measurements in apparently healthy individuals. *Clin Gastroenterol*

Hepatol. 2019 Jan;17:54–64. : the official clinical practice journal of the American Gastroenterological Association.
14. Castera L, Forns X, Alberti A. Non-invasive evaluation of liver fibrosis using transient elastography. *J Hepatol*. 2008 May;48:835–847.
15. Sterling RK, Lissen E, Clumeck N, et al. Development of a simple noninvasive index to predict significant fibrosis in patients with HIV/HCV coinfection. *Hepatology*. 2006 Jun;43:1317–1325.
16. Zjačić Puljiz D, Delić Jukić IK, Puljiz M, et al. Which factors influence liver stiffness measured by real-time two dimensional shear wave elastography in patients on maintenance hemodialysis? *Croat Med J*. 2021 Feb 28;62:34–43.
17. Sette LH, Almeida Lopes EP. Liver enzymes serum levels in patients with chronic kidney disease on hemodialysis: a comprehensive review. *Clinics*. 2014;69:271–278.
18. Lee JJ, Wei YJ, Lin MY, et al. The applicability of non-invasive methods for assessing liver fibrosis in hemodialysis patients with chronic hepatitis C. *PLoS One*. 2020;15e0242601.
19. European Association for the Study of the Liver. Electronic address eee, European association for the study of the L. EASL recommendations on treatment of hepatitis C 2018. *J Hepatol*. 2018 Aug;69:461–511.
20. Liu CH, Liang CC, Huang KW, et al. Transient elastography to assess hepatic fibrosis in hemodialysis chronic hepatitis C patients. *Clin J Am Soc Nephrol : CJASN*. 2011 May;6:1057–1065.
21. Syed T, Chadha N, Kumar D, et al. Non-invasive assessment of liver fibrosis and steatosis in end-stage renal disease patients undergoing renal transplant evaluation. *Gastroenterol Res*. 2021 Aug;14:244–251.
22. Kellner P, Anadol E, Huneburg R, et al. The effect of hemodialysis on liver stiffness measurement: a single-center series. *Eur J Gastroenterol Hepatol*. 2013 Mar;25:368–372.
23. Taneja S, Borkakoty A, Rathi S, et al. Assessment of liver fibrosis by transient elastography should Be done after hemodialysis in end stage renal disease patients with liver disease. *Dig Dis Sci*. 2017 Nov;62:3186–3192.
24. Khunpakdee N, Jayanama K, Kaewdoung P, et al. Transient elastography in end-stage renal disease patients on hemodialysis: the effect of net fluid withdrawal. *Blood Purif*. 2015;40:256–259.
25. Mikolasevic I, Racki S, Lukenda V, et al. Nonalcoholic Fatty liver disease in renal transplant recipients proven by transient elastography. *Transplant Proc*. 2014 Jun;46:1347–1352.