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ORIGINAL RESEARCH

Role of Ultrasonography and Elastography in Renal Parenchymal Disease in Diabetic and Hypertensive Patients

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ABSTRACT

BACKGROUND: In this study, we wanted to compare the renal parenchymal stiffness between healthy subjects and patients with chronic kidney disease due to type II diabetes mellitus and hypertension, using shear wave elastography. We also wanted to do the staging of chronic kidney disease based on renal parenchymal stiffness and study its correlation with estimated glomerular filtration rate (eGFR).

METHODS: This was a hospital-based study conducted among 140 patients who underwent ultrasound examination in the Department of Radiodiagnosis, R.L. Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar, after obtaining clearance from the institutional ethics committee and written informed consent from the study participants.

RESULTS: A significant negative linear association between "YM readings and eGFR" was found using the "Spearman correlation coefficient ($r = -0.668$, $p < 0.001$)". 5 CKD (9.71 ± 2.61) patients followed by Stage 4 (8.85 ± 1.74), Stage 3 (7.58 ± 1.26), Stage 2 (6.36 ± 1.28) and Stage 1 (3.85 ± 0.30). Tukey post-hoc multiple comparison test revealed that there was a statistically significant difference in means between stages 1, 3 and 5, and stages 2, 4 and 5. However, the ability to differentiate between individual stages was poor. Setting a threshold between healthy and unhealthy renal parenchyma could aid in the early detection and treatment of CKD.

CONCLUSION: SWE was more effective in identifying CKD than "renal length and cortical thickness". A cut-off value of 4.44 kPa was used to determine whether a kidney was diseased or not.

KEYWORDS: Electrography, Ultrasound, CKD, Diabetes, Hypertension, Correlation

INTRODUCTION

Patients with T2DM for an extended period frequently get diabetic kidney disease (DKD).[1] If prompt diagnosis and therapy are given, it can be managed or even reversed. Early on albuminuria, estimated glomerular filtration rate (eGFR), and serum creatinine were less accurate indications.[2] Before nephropathy, cortical cells undergo the initial pathophysiologic alterations.[3] The basal membrane thickening is the first histologic change, and in the subsequent three to five years, the afferent and efferent arterioles are hyalinized. Within 15 years of the beginning of the disease, the mesangial volume would have increased.[3] Early diagnoses are important for DM patients' prognosis because 20–30% of them eventually

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develop nephropathy.[3] Biopsy offers a conclusive diagnosis, but it also comes with potentially fatal risks. Both MRI and CT can assess the kidney's morphology and functional state. But they come with some drawbacks, like greater expenses, extended appointment times, radiation exposure and CIN. The evaluation of the kidney with ultrasound (US) is non-invasive, accessible, affordable, and routinely employed. US results like decreased renal size, increased parenchymal echogenicity, and parenchymal thickness, may be beneficial, particularly in advanced stages.[4] Although reversible, the early phases are where the majority of diagnostic issues manifest. The aforementioned criteria are unreliable and may stay within normal limits in hyper-infiltration stages.[5] A non-invasive technique is necessary for the early phases of DKD evaluation. The 2nd and 3rd most frequent reason for RRT in Europe, the USA, and Japan, respectively, is "hypertensive nephroangiosclerosis (HN)"[6] which represents the progression of arterial hypertension.[6-8] Epidemiological statistics showed that over the previous 20 years, HN has drastically increased in Europe and the USA. About 15% of new cases of "end-stage renal disease (ESRD)" in Europe and 28% of new cases with the condition in the US had HN as the cause.[9-10] According to Mahmoodi et al., there is a direct link between cardiovascular illness, renal involvement, and high blood pressure.[11] Microalbuminuria and macroalbuminuria are related to cardiovascular events.[12] Micro / macroalbuminuria is also responsible for CKD.[13] Fewer patients get benefited from the histopathological study, where the diagnosis of HN is made based on a routine test. As a result, epidemiological data from various medical facilities varied greatly "in Europe, between 5 and 33% of ESRD cases had HN as the cause".[10] Vasculature, glomeruli, and tubulointerstitium are all involved in kidney injury brought on by hypertension. Intrarenal arteries exhibit media thickness due to smooth muscle cell hyperplasia and increasing intimal thickening and fibrosis brought on by collagen deposition. A hyalinization process is visible in afferent arterioles. Glomerular involvement can have a variety of morphologies, including normal, ischemic, destroyed, collapsed capillaries, or hypertrophied. Tubular atrophy and interstitial fibrosis are two additional characteristics of hypertensive kidney damage. These histological alterations develop from "asymptomatic organ damage to symptomatic organ damage", with the appearance of CKD. However, if kidney involvement is detected early and the patients receive the proper treatment, its progression might be halted.[9,14] Renal function is impacted by these changes in renal morphology. The initial alterations that characterise the developing kidney injury in HN are an increase in albuminuria and a decrease in glomerular filtration rate.[14] However, ultrasonographic "B-mode and Doppler" studies also play a significant role in the diagnosis of HN in addition to serum and urine biomarkers. B-mode ultrasonography assesses the morphology and location of the kidneys - "kidney length, parenchyma thickness and echogenicity". But regrettably, abnormalities in ultrasonography only become apparent later in the course of the disease.[9,14,15]

Studies have shown that renal USG can detect changes in the size, echogenicity, and cortical thickness of the screened kidney. Shear wave elastography (SWE), an advanced, non-invasive, and straightforward sonographic technique, has been developed to quantitatively detect the onset of parenchymal fibrosis based on stiffness. In SWE, the tissues are bent temporarily by an acoustic radiation force applied by the transducer. The waves which get deformed, also known as shear waves, radiate perpendicular to the US beam and are measured in m/s and transformed into a "quantitative stiffness score in kPa using Young's modulus". Low speed signifies a soft medium, whereas high speed denotes a hard one. Shear wave elastography (SWE) has begun to be utilised on DM patients and has lately gained popularity as a method. A non-invasive, affordable, and reliable USG approach is shear wave elastography.[16-18] Systemic and demographic factors had little impact on cortical stiffness (CS), which was evaluated by SWE, and it is correlated with renal parenchyma disease and fibrosis.[16-19] Using colour duplex, both internal renal venous and arterial vascularisation are

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 02, 2023

measured using Doppler and power Doppler ultrasound, giving information regarding kidney function.[20,21] A novel ultrasonographic technique called "Acoustic Radiation Force Impulse Elastography (ARFI)", which measures elastic compliance changes as shear wave velocity, can be used to diagnose abnormal renal morphology. In ARFI, the transducer's acoustic pulses cause microscopic displacements (1–20 m) in the tissue being studied. Micrometric displacements are measured using the ROI's square shape. Shear waves are created by displacement and are propagated away from the ROI. These waves are collected by the same transducer and are displayed as m/s. ARFI does not differ from operators and is effective in deep organ analysis.[22-24] SWE measurements of nephrogenic cortical stiffness in patients with T2 DM and hypertension have been shown to increase.[18,25,26,27]

AIMS AND OBJECTIVES

In this study, we wanted to compare the renal parenchymal stiffness between healthy subjects and patients with chronic kidney disease due to type II diabetes mellitus and hypertension, using shear wave elastography. We also wanted to do the staging of chronic kidney disease based on renal parenchymal stiffness and study its correlation with estimated glomerular filtration rate (eGFR).

MATERIALS & METHODS

This was a hospital-based study conducted among 140 patients who presented with ultrasound examination to the Department of Radio-Diagnosis, R.L. Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar, after obtaining clearance from the institutional ethics committee and written informed consent from the study participants.

Inclusion Criteria

Controls - Healthy Volunteers

Healthy volunteers were chosen as controls with inclusion criteria as follows being age >18 yrs.

Cases-CKD patients

Cases included CKD patients referred to our department for imaging of kidneys with inclusion criteria as follows:

- Age >18 years
- Cases of chronic kidney disease secondary to type II diabetes mellitus or hypertension or both.

Exclusion Criteria

- BMI >35 or any condition that impedes visualization of kidneys.
- Diabetes mellitus, hypertension or any other systemic disease that might influence renal function.
- Presence of kidney lesions – renal cysts/stones/mass/HUN/solitary kidney.

Sample Size

OpenEpi version 3.01 was used to determine the sample size (Open Source Epidemiologic Statistics for Public Health). The sample size was calculated using OpenEpi software version 3.01 (Open Source Epidemiologic Statistics for Public Health). Assuming an alpha error of 5% (95% confidence limit), Power of 80% (β=0.20) and the ratio of cases and controls to be 1:1, the minimum required sample size was calculated to be 70 in each group and the total sample size was 140 (70 healthy controls and 70 CKD patients).

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 02, 2023

Sample size (n) = $\frac{Z^2(P*Q)}{d^2}$

Where
Z is the value for the Confidence Interval
D is the absolute precision
P is the expected proportion (p = 0.70) q=1-p (q = 0.30)

Statistical Methods

Data was entered in MS Excel and analyzed using SPSS software. Results were presented as tables.

RESULTS

Group Normal Spearman's Rho	**Correlations**	**Age**	**SWE Avg**
Age	Correlation Coefficient	1.000	.170
	Sig.(2-tailed)	.	.161
	N	70	70
SWE Avg	Correlation Coefficient	.170	1.000
	Sig.(2-tailed)	.161	.
	N	70	70
Spearman's rho correlation between age and YM measurements among controls			
Group CKD Spearman's Rho	**Correlations**	**Age**	**SWE Avg**
Age	Correlation Coefficient	1.000	.293*
	Sig.(2-tailed)	.	.014
	N	70	70
SWE Avg	Correlation Coefficient	.293*	1.000
	Sig.(2-tailed)	.014	.
	N	70	70
Spearman's rho correlation between age and YM measurements among CKD group			
Table 1			

YM measurements showed no significant correlation with age among the controls, but showed a moderate positive correlation with age among the CKD group (r = 0.293, p < 0.014)

Group CKD Spearman's Rho	**Correlations**	**eGFR**	**SWE Avg**
eGFR	Correlation Coefficient	1.000	-.668**
	Sig.(2-tailed)	.	.000
	N	70	70
SWE Avg	Correlation Coefficient	-.668**	1.000
	Sig.(2-tailed)	.000	.
	N	70	70
****Correlation is significant at the 0.01 level(2-tailed)**			
Spearman's rho correlation between eGFR and YM measurements among CKD group			

	Controls	**CKD**	**t**	**p-value**
YM (kPa)	3.51 ± 1.56	7.96 ± 2.41	-12.95	0.001
Kidney length (cm)	9.3 ± 0.87	8.50 ± 1.82	3.44	0.001
Comparison of means of YM measurements, kidney length among controls and CKD group				
Table 2				

The YM measurements and eGFR had a substantial negative linear association, according to the "Spearman correlation coefficient" (r = 0.668, p 0.001).

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 02, 2023

BMI was assessed by comparing the two groups by "independent variable t-test". YM measurements were greater in the CKD group (7.96 2.41) compared to the control (3.51 1.56), showing increased stiffness within the CKD group, and were statistically significant with a p-value of 0.001 in the comparison of mean YM measurements between the CKD and control groups. Mean kidney length was higher in controls (9.3 ± 0.87) as compared to the CKD group (8.50 ± 1.82), and mean BMI was higher in the CKD group as compared to controls.

Area Under the Curve				
Test Result Variable(s): SWE-Avg				
Area	**Std. Error**	**Asymptomatic Sig.[b]**	**Asymptomatic 95% Confidence Interval**	
			Lower Bound	**Upper Bound**
.940	.019	.000	.903	.977
AUROC of YM in distinguishing between CKD and control groups				
CKD Stage		**Mean YM (kPa)**		**Std deviation**
Stage 1		3.85		0.30
Stage 2		6.36		1.28
Stage 3		7.58		1.26
Stage 4		8.85		1.74
Stage 5		9.71		2.61
Mean YM values of CKD stages				
Table 3				

ROC curves were used to assess the mean YM measurements between the control and patient groups. SWE had a 0.94 area under the ROC curve. We determined a cut-off value for YM measurements of 4.44 kPa, below which a kidney without disease was recommended. This resulted in sensitivity and specificity which were, 90.0% and 77.1% respectively.

One-way analysis of variance (ANOVA) test was used to see the changes in mean YM values according to the CKD stages. The mean values of YM were found to be higher in Stage 5 CKD (9.71 ± 2.61) patients followed by Stage 4 (8.85 ± 1.74), Stage 3 (7.58 ± 1.26), Stage 2 (6.36 ± 1.28) and Stage 1 (3.85 ± 0.30).

Multiple Comparisons						
Dependent Variable: SWE-Avg						
Tukey HSD						
(I) Stage	**(J) Stage**	**Mean Difference (IJ)**	**Std. Error**	**Sig.**	**95% Confidence Interval**	
					Lower Bound	**Upper Bound**
1.00	2.00	-2.50554	.95157	.076	-5.1755	.1644
	3.00	-3.73150*	.92646	.001	-6.3310	-1.1320
	4.00	-4.99956*	.91412	.000	-7.5644	-2.4347
	5.00	-5.85511*	.91412	.000	-8.4200	-3.2902
2.00	1.00	2.50554	.95157	.076	-.1644	5.1755
	3.00	-1.22596	.67519	.374	-3.1204	.6685
	4.00	-2.49402*	.65816	.003	-4.3407	-.6473
	5.00	-3.34957*	.65816	.000	-5.1963	-1.5029
3.00	1.00	3.73150*	.92646	.001	1.1320	6.3310
	2.00	1.22596	.67519	.374	-.6685	3.1204
	4.00	-1.26806	.62130	.259	-3.0113	.4752
	5.00	-2.12361*	.62130	.009	-3.8669	-.3803
4.00	1.00	4.99956*	.91412	.000	2.4347	7.5644

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 02, 2023

	2.00	2.49402*	.65816	.003	.6473	4.3407
	3.00	1.26806	.62130	.259	-.4752	3.0113
	5.00	-.85556	.60275	.618	-2.5468	.8357
5.00	1.00	5.85511*	.91412	.000	3.2902	8.4200
	2.00	3.34957*	.65816	.000	1.5029	5.1963
	3.00	2.12361*	.62130	.009	.3803	3.8669
	4.00	.85556	.60275	.618	-.8357	2.5468
*. **The mean difference is significant at the 0.05 level.**						
Tukey post-hoc multiple comparison tests between individual CKD stages						
Table 4						

Tukey post-hoc multiple comparison tests between individual CKD stages. Post hoc Tukey significant difference tests were used to see the changes in mean YM values according to the CKD stages. "Tukey post-hoc multiple comparison" test revealed that there was a statistically significant difference in means between stages 1, 3 and 5, and stages 2, 4 and 5, no other significant changes were observed in between the other CKD stages.

Our findings indicated that as the stage of CKD increases, the CS increases up till CKD 5. To an extent, reversible and non-reversible stages may be differentiated by the stiffness values which are significantly different between CKD stage 2 v/s 5 and 3 v/s 5. However, the ability to differentiate between individual stages was poor.

Measured mean values of YM were lower in the CKD group that had higher eGFR, with the exception being stage 1 which had a higher YM value than stage 2.

DISCUSSION

Relationship between YM measurements and age and eGFR

A progressive build-up of harmful connective tissue in the kidney parenchyma known as tubulointerstitial renal fibrosis appears to be the main factor contributing to the decline in renal function. A falling eGFR as a result of progressive interstitial injury suggests an inverse relationship between serum creatinine and eGFR.[28] Plasma proteins may be pushed out into the tubule and urine by hyperfiltration, resulting in tubulointerstitial injury at the glomerulus.[29] Inflammation and fibrosis may develop as a result of protein reuptake at the tubules. The shear wave travels less swiftly in fibrotic tissue.[30,31] eGFR was inversely linked with the amount of renal fibrosis, which in itself is relevant to the transmission of shear waves.

The study revealed that YM measurements showed no correlation with age among the controls, but showed a moderate positive correlation with age among the CKD group ($r = 0.293$, $p < 0.014$) and Samir et al finding that there was no discernible relationship between YM measurement and age confirmed this. The study's tiny sample size may be one reason for this. However, Leong et al and Yang et al research revealed a substantial correlation between this observation and YM measurements and age. As kidneys became older, glomerulosclerosis, interstitial fibrosis, tubular atrophy, and arteriosclerosis began to emerge.[32]

In our research, the YM measurements and eGFR had a substantial "negative linear connection (Spearman coefficient: $r = 0.668$, p 0.001)". According to Hu et al., renal length and parenchymal thickness showed a lesser connection with eGFR than SWE does. Guo et al, who observed a "positive correlation between shear wave velocity (SWV) and eGFR", showed contrary results. It is still unknown why these differences exist.[33]

Comparison of mean of different parameters between CKD and control groups

Comparison of mean YM measurements between CKD and control groups revealed higher YM values in the CKD group (7.96 ± 2.41) compared to control (3.51 ± 1.56), indicating

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 02, 2023

increased stiffness within the CKD group, and was statistically "significant with a p-value of <0.001". Mean kidney length was higher in controls (9.3 ± 0 87) as compared to the CKD group (8.50 ± 1.82), and the mean BMI was higher in the CKD group. In contrast to the study by Leong et al., no discernible difference was found between the aforementioned groups. "One-way analysis of variance" revealed a significant difference in YM measurements (F = 90.188, p 0.0001).

ROC curve of YM in distinguishing between CKD and control groups

"ROC curves were used for the analysis of mean YM between the control and sick groups in our study". SWE had a 0.94 AUROC. We determined a cut-off value for YM measurements of 4.44 kPa, below which a kidney without disease was recommended. This produced results that were 90.0% and 77.1% more sensitive and specific than typical ultrasonography values. Leong also found comparable outcomes, with SWE having a greater area under the ROC curve (0.87) than measurements of "kidney length and cortical thickness" made using conventional ultrasonography. A "cut-off value of 4.31 kPa", with 80.3 % as sensitivity and 79.5% as the specificity indicates that a less value reflects a normal kidney. According to Bob et al., a kidney shear wave speed of 2.32 m/s foretells a drop in eGFR to 60 mL/min. However, this cut-off value has a low sensitivity (67.39%) and specificity (67.83%), making it challenging to predict renal involvement in diabetic individuals only using elastography.[34]

ROC curve of length in distinguishing CKD and controls

A predictor of CKD has also been found in the bipolar length of the kidney. In the present study, for the bipolar kidney length, we found the best possible cut-off of 9.0 cm with a sensitivity of 44.3% and specificity of 40.0% to differentiate control and cases. The AUROC was poor (0.363). However, compared to kidney volume, "Sanusi et al. claims that kidney length is not a reliable indicator of kidney abnormalities".[35]

Correlation between CKD stage and YM measurements

In our study, ANOVA tests were used to witness mean YM according to the CKD stages. The mean values of YM were found to be higher in Stage 5 CKD (9.71 ± 2.61) patients followed by Stage 4 (8.85 ± 1.74), Stage 3 (7.58 ± 1.26), Stage 2 (6.36 ± 1.28) and Stage 1 (3.85 ± 0.30).

Tukey post-hoc multiple comparison tests between individual CKD stages

"Post hoc Tukey significant difference tests were used to see the changes of mean YM values according to the CKD stages in the present study. "Tukey post-hoc multiple comparison tests" revealed that there was a statistically significant difference in means between stages 1, 3 and 5, and stages 2, 4 and 5, but there was no significant difference between the other CKD stages. Certain traditional renal USG results, such as decreased kidney size, decreased cortical thickness, and increased echogenicity in the cortex, may be indicative of parenchymal disease in the kidney. The use of cortical stiffness (CS) measurements from SWE tests has increased recently. The limitations of SWE include the test's sporadic availability in clinics and the absence of defined average results of CS in the patient population. Regular USG results do not include renal SWE data, and only specific diseases and research quantify CS levels. SWE is a non-invasive, cost-effective, and reliable USG test that can be used to assess tissue elasticity. Values for CS are given in kPa.[36] The most significant indicator of kidney disease is renal parenchymal fibrosis, which affects the mechanical characteristics of the kidneys and may be assessed objectively using SWE. It has been demonstrated that renal SWE examination helps stage diabetic nephropathy, determining renal fibrosis, identifying rejection of renal allografts, and in CKD patients.[37,38]

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 02, 2023

Our findings indicated that as the stage of CKD increases, the CS increases up till CKD 5. To an extent, reversible and non-reversible stages may be differentiated by the stiffness values which are significantly different between CKD stage 2 v/s 5 and 3 v/s 5. However, the ability to differentiate between individual stages was poor. Leong concluded that the test also revealed that because of the significant variation between the groups, it was challenging to discriminate between CKD 3rd, 4th, and 5th stages based on their YM measures. In a study involving individuals with diabetic nephropathy, Hassan et al. discovered a substantial reduction in renal cortical thickness. According to the same study, grade 4 CKD patients' renal cortical thickness was lower than that of grade 3 CKD patients. Similar findings were made by Koc and Sumbul et al., "who discovered that patients with type 2 DM had increased cortical thickness in addition to normal renal function".[39]

This supports Soldo et al. in the literature.[40] Increased cortical stiffness results from the nephropathy that long-term diabetics experience. The relationship between increasing "renal cortical parenchymal thickness" and CS is a result of nephron hypertrophy and increased CS from increased filtration.

Measured mean values of YM were lower in the CKD group that had higher eGFR, with the exception being stage 1 which had a higher YM value than stage 2. This is in accordance with the study of Leong et al. Tukey post hoc analysis showed that the group with greater eGFR had lower YM readings.

Although the SWE results are promising, it is important to be aware of this novel technique's limitations, including bladder distension, intra- and inter-observer variation, and the position of the ROI. However, a bladder that is excessively distended and has transmitted backpressure could result in a false-positive diagnosis of obstructive hydronephrosis. According to a study by Sohn et al, hydronephrosis-related increased pelvic pressure may exacerbate renal parenchymal stiffness.[41]

Nephrogenic tissue is anisotropic; as a result, not all axis orientations have the same qualities.[42] "The Henle and vasa recta in the medulla", as well as the collecting ducts in the cortex and medulla, may respond differently to the placement of ultrasound beams on account of the varied shear wave propagation axes. As a result, our findings demonstrated a considerable difference in YM readings when the ROI's location was modified, thereby changing the orientation of the beam to the tissue. Given that the ROI box location had a substantial impact on YM measurements, it is important to choose a fixed location for the ROI box during image capture to provide accurate and repeatable results, particularly when determining the usual range of stiffness for a given tissue. Because the renal medulla and sinus are easily excluded from the ROI box when it is positioned in the middle of the kidney during image capture, we advise doing so.

Preventing nephropathy brought on by diabetes is one of the most crucial objectives in DM care. Although the mainstays of treatment for achieving this goal are blood sugar and blood pressure control, it is still challenging to prevent this consequence. Reactive oxygen products, glycolyzed lipids, and elevated glucose levels were the main Metabolic issues that lead to increased generation of inflammatory cells and fibrosis.[43-44] Glomerulosclerosis and acute interstitial fibrosis are caused by nephropathy, which also damages mesangial, endothelial, and epithelial cells.

Interstitial fibrosis is the key identifying characteristic of nephropathy brought on by DM.[45] Before the onset of nephropathy, it is critical to identify alterations in the mesangium, endothelial, and epithelial cells. Early identification of microalbuminuria is crucial for detecting diabetic nephropathy.[46,47] Although invasive, histological evaluation with kidney biopsy demonstrates the continuing fibrosis but cannot be employed. Non-invasive examinations have been favoured for this reason. SWE is a potential, non-invasive

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 02, 2023

examination that can be utilised for this reason since it provides an objective indication of renal elasticity or tissue stiffness.

CONCLUSION

SWE performed better than traditional ultrasonography in evaluating CKD. Patients who had T2 DM had considerably higher cortical stiffness values when measured with SWE. 4.44 kPa was selected as the cut-off value to distinguish between kidneys with illness and those without. Despite its shortcomings, SWE-derived estimations of renal stiffness are a reliable, inexpensive technique for non-invasively adding diagnostic information to CKD.

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ISSN: 0975-3583,0976-2833 VOL14, ISSUE 02, 2023

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