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Assessment of delayed graft function using susceptibility-weighted imaging in the early period after kidney transplantation: a feasibility study

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Abstract

Purpose: This study aimed to explore the feasibility of susceptibility-weighted imaging (SWI) for evaluating delayed graft function (DGF) during the early posttransplantation period.
Methods: Sixty-nine recipients who accepted allograft renal transplantation underwent SWI during the second posttransplantation week. Renal allograft function was estimated via the glomerular filtration rate. Recipients with and without DGF were identified. For each transplanted kidney, the presence of abnormal signal intensity lesions (ASILs), excluding benign lesions, on SWI was assessed. Renal allograft function was compared between the recipients with and without ASILs. The correlation between ASILs and renal allograft function was tested by Spearman's rank correlation analysis.
Results: Thirty-four recipients were diagnosed with DGF, while 35 recipients showed no DGF. In the DGF group, 16 recipients had low-intensity ASILs, primarily at the corticomedullary junction of transplanted kidneys on SWI, and no ASILs were found in 18 recipients. In the non-DGF group, none of the recipients showed ASILs on SWI. In the DGF group, the renal allograft function among the 16 recipients with low-intensity ASILs was significantly lower than that among the other 18 recipients (8.5 ± 4.2 vs. 19.7 ± 9.7 mL/min, $P < 0.001$). The presence of low-intensity ASILs on SWI showed a moderate negative correlation with renal allograft function in recipients with DGF ($r = -0.553$, $P = 0.001$).
Conclusion: SWI can be used to evaluate DGF in the early post-kidney transplantation period.

Key words: Susceptibility-weighted imaging—Diagnostic imaging—Delayed graft function—Kidney transplantation—Magnetic resonance imaging

Delayed graft function (DGF), defined as the necessity of dialysis during the first week post transplantation, is the most common immediate complication affecting kidney allografts in the early posttransplantation period [1–3]. DGF is associated with an increased short-term risk of graft loss, acute rejection, and hospitalization as well as high cost [1, 3]. The incidence of DGF ranges from 4% to 10% in living transplants and from 5% to 50% in deceased donor kidney transplant recipients [4]. Therefore, early and safe detection of DGF in a transplanted kidney is very critical for timely treatment [5]. Percutaneous renal transplant biopsy is the gold standard in the diagnosis of allograft function, but it is an invasive operation and can cause many serious complications [6]. In contrast, functional magnetic resonance imaging (MRI) has many advantages, as it is noninvasive, repeatable, and requires no ionizing radiation or contrast medium administration.

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Susceptibility-weighted imaging (SWI) is an emerging functional MRI technique that utilizes the susceptibility differences between various tissues to generate a novel type of contrast [7]. This methodology consists of both magnitude and phase images and provides exquisite sensitivity to susceptibility changes, such as those caused by blood metabolites (oxyhemoglobin, deoxyhemoglobin, methemoglobin, and hemosiderin), iron, and calcification [8–10]. The original SWI applied in brain imaging was traditionally performed using a long-TE, high-resolution, fully flow-compensated, and three-dimensional (3D) gradient-echo (GRE) sequence [8]. Because SWI is sensitive to patient motion, some technical barriers prevented the application of the original 3D SWI in the abdomen [10]. One important example is breathing artifacts generated from long acquisition times [9, 10]. Recently, a new multi-breath-hold 2D SWI sequence has been developed, and it has been successfully used to evaluate lesions in the liver and kidney [9–11].

However, to date, the application of SWI in assessing transplanted kidneys has not been reported. The purpose of this paper was to study the feasibility of using 2D SWI to evaluate DGF in the early period after kidney transplantation.

Material and methods

Subjects

Our institutional research ethics board approved the retrospective study (the approval number: KJK-2011-108). Informed consent was obtained from all patients prior to the examination. A total of 75 patients who underwent allograft renal transplantation for the first time from July 2013 to November 2016 were reviewed. Six cases were excluded due to allograft function injury caused by immunosuppression ($n = 2$), acute rejection of the renal allograft ($n = 1$), and unavailability of images ($n = 3$). Finally, 69 recipients [44 males and 25 females; median age (range), 40 (22–63) years] with 69 transplanted kidneys were included in this study.

Clinical data, laboratory data, and DGF diagnostic standard

The recipients' clinical and laboratory data were collected. Serum creatinine and cystatin C were acquired daily during the first two weeks after kidney transplantation. Renal allograft function was determined by estimating the glomerular filtration rate (GFR). The estimation of GFR (eGFR) was based on serum creatinine and cystatin C levels according to the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) creatinine-cystatin C equation (Table 1) [12]. DGF was defined as a lack of decrease in serum creatinine by at least 10% daily on 3 consecutive days or the need for more than 1 dialysis treatment within the first week post transplant [1].

MRI examination

Conventional MRI and SWI were performed in all recipients during the second week (day 8–day 14) after transplantation. The scan range covered the area in the pelvic cavity from the upper pole to the lower pole of the transplanted kidneys. All MRI images were acquired on a 3T MRI scanner (Magnetom Verio, Siemens Healthcare, Erlangen, Germany) with a standard 12-channel body matrix coil.

The conventional MRI and SWI protocols are shown in Table 2.

Imaging analysis

Two genitourinary radiologists with more than 10 years of diagnostic experience analyzed all the MRI images on a commercially available workstation (Syngo, Workstation, Siemens, Erlangen, Germany). The radiologists were blinded to all the clinical and laboratory data. In ambiguous cases, the reviewers reached a consensus in their decision.

For each transplanted kidney recipient, conventional MRI and SWI images were successively assessed. (1) First, the presence of benign lesions, such as simple or complicated cysts and angiomyolipomas, within the transplanted kidney were assessed on conventional MRI. Simple cysts were diagnosed if a lesion possessed low signal intensity on T1WI but high signal intensity on T2WI, with or without fat suppression. Complicated cysts were diagnosed if a lesion possessed high signal intensity on T1WI as well as T2WI with or without fat suppression. Angiomyolipoma was diagnosed if a lesion had fat tissue with high signal intensity on both T1WI and T2WI but low signal intensity on T2WI with fat suppression. (2) After these benign lesions were excluded, the presence of abnormal signal intensity lesions (ASILs) within the transplanted kidney was assessed on conventional MRI images. (3) In addition to conventional MRI, the presence of ASILs within the transplanted kidney was assessed on SWI images, with the exclusion of the benign lesions mentioned above. If ASILs were found in a transplanted kidney, the location and signal intensity (compared with the renal cortex) were assessed.

The signal intensities of ASILs and the renal cortex in each transplanted kidney with ASILs on SWI wereS manually measured by drawing a suitable region of interest (ROI) on SWI images at the same slice, avoiding the border of the transplanted kidney. When measuring the ASIL signal intensity, a ROI was drawn along the edge of every lesion. To minimize error, all measurements were repeated three times for every lesion, and the data were averaged. Then, the mean signal intensity of all

Table 1. CKD-EPI creatinine-cystatin C equation for estimating GFR [12]

Sex	Serum creatinine	Serum cystatin C	Equation for estimating GFR
Female	≤ 0.7	≤ 0.8	$130 \times (\mathrm{Scr}/0.7)^{-0.248} \times (\mathrm{Scys}/0.8)^{-0.375} \times 0.995^{\mathrm{Age}}$
		> 0.8	$130 \times (\mathrm{Scr}/0.7)^{-0.248} \times (\mathrm{Scys}/0.8)^{-0.711} \times 0.995^{\mathrm{Age}}$
Female	> 0.7	≤ 0.8	$130 \times (\mathrm{Scr}/0.7)^{-0.601} \times (\mathrm{Scys}/0.8)^{-0.375} \times 0.995^{\mathrm{Age}}$
		> 0.8	$130 \times (\mathrm{Scr}/0.7)^{-0.601} \times (\mathrm{Scys}/0.8)^{-0.711} \times 0.995^{\mathrm{Age}}$
Male	≤ 0.9	≤ 0.8	$135 \times (\mathrm{Scr}/0.7)^{-0.207} \times (\mathrm{Scys}/0.8)^{-0.375} \times 0.995^{\mathrm{Age}}$
		> 0.8	$135 \times (\mathrm{Scr}/0.7)^{-0.207} \times (\mathrm{Scys}/0.8)^{-0.711} \times 0.995^{\mathrm{Age}}$
Male	< 0.9	≤ 0.8	$135 \times (\mathrm{Scr}/0.7)^{-0.601} \times (\mathrm{Scys}/0.8)^{-0.375} \times 0.995^{\mathrm{Age}}$
		> 0.8	$135 \times (\mathrm{Scr}/0.7)^{-0.601} \times (\mathrm{Scys}/0.8)^{-0.711} \times 0.995^{\mathrm{Age}}$

CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; GFR, glomerular filtration rate; Scr, serum creatinine; Scys, serum cystatin C

Table 2. Sequences and parameters for sample renal MRI protocols

Parameters	Conventional MRI			SWI
	T2-weighted HASTE	T2-weighted HASTE	T1-weighted GRE	GRE
Plane orientation	Coronal	Transverse	Transverse	Transverse
Acquisition type	2D	2D	2D	2D
Repetition time (ms)	1400	1800	161	162
Echo time (ms)	91	96	2.5	10.3
Field of view (mm)	380 × 380	285 × 380	285 × 380	285 × 380
Matrix	117 × 256	168 × 320	180 × 320	187 × 384
Slice thickness (mm)	4	5	5	5
Interslice gap (mm)	1.95	1.0	1.0	1.0
Flip angle (°)	160	150	70	20
Bandwidth (Hz/pixel)	781	500	270	620
Acquisition time (s)	34	39	16	53
Physiology control	Breath-hold	Breath-hold	Breath-hold	Breath-hold
No. of breath-holds	2	3	1	3

HASTE, half acquisition single-shot turbo spin echo; GRE, gradient-recalled echo; *D*, dimension; SWI, susceptibility-weighted imaging; No., number

lesions was taken as the signal intensity of the ASILs for each transplanted kidney. When measuring the signal intensity of the renal cortex, a round or oval ROI was drawn in the anterior, middle, and posterior cortex of the transplanted kidney, and the mean of the three data points was assessed as the signal intensity of the renal cortex for the transplanted kidney.

Statistical analysis

All statistical analyses were performed using SPSS 22.0 (SPSS, Inc., Chicago, IL, USA) and GraphPad Prism 5.0 (Graphpad software Inc., San Diego, CA, USA). Measurement data were expressed as the means ± standard deviation ($x \pm$ SD). The *t* test was used to compare the means of two groups. The chi-squared test was used to compare the ratio between different groups. The correlation between two variables was tested by Spearman's rank correlation, and the symbol "r" was used to represent the correlation coefficient [13]. The correlation was interpreted as follows: $r > 0$ was considered a positive correlation; $r < 0$ was considered a negative correlation; $|r| = 1$ was considered a perfect correlation; $0.7 \le |r| < 1$ was considered a high correlation; $0.4 \le |r| < 0.7$ was considered a moderate correlation; $|r| < 0.4$ was considered a low correlation; and $r = 0$ was considered zero correlation. $P < 0.05$ was considered statistically significant.

Results

Clinical data, laboratory data, and DGF diagnosis

Thirty-four recipients with DGF and 35 recipients without DGF were diagnosed, and the incidence rate of DGF was 49.3% (34/69). Recipient clinical and laboratory data are summarized in Table 3.

Qualitative analysis

Six transplanted kidneys had simple cysts, and 1 transplanted kidney had a complicated cyst; all of these lesions were excluded from the ASILs in this study.

In the DGF group ($n = 34$), two transplanted kidneys had low-intensity ASILs that were primarily located at the corticomedullary junction on both conventional MRI (T2WI) and SWI (Fig. 1); 14 transplanted kidneys had low-intensity ASILs that were primarily located at

Table 3. Patient clinical and laboratory data

Data	Non-DGF (n = 35)	DGF (n = 34)	P value
Recipient data			
Age (year)	39.0 ± 9.8	41.2 ± 10.1	
Gender (male)	19/35 (54.3%)	25/34 (73.5%)	0.096
Transplantation details			
Deceased donor kidney	30/35 (85.7%)	34/34 (100%)	0.054
Clinical and laboratory data post transplantation			
Maximum transverse diameter of transplanted kidney (cm)	7.8 ± 1.2	8.2 ± 1.3	0.230
eGFR, 10th day (mL/min)	59.5 ± 22.0	14.4 ± 9.4	< 0.001
Hemodialysis during the first week	0/35 (0%)	30/34 (88.2%)	< 0.001

DGF, delayed graft function; eGFR, estimated glomerular filtration rate

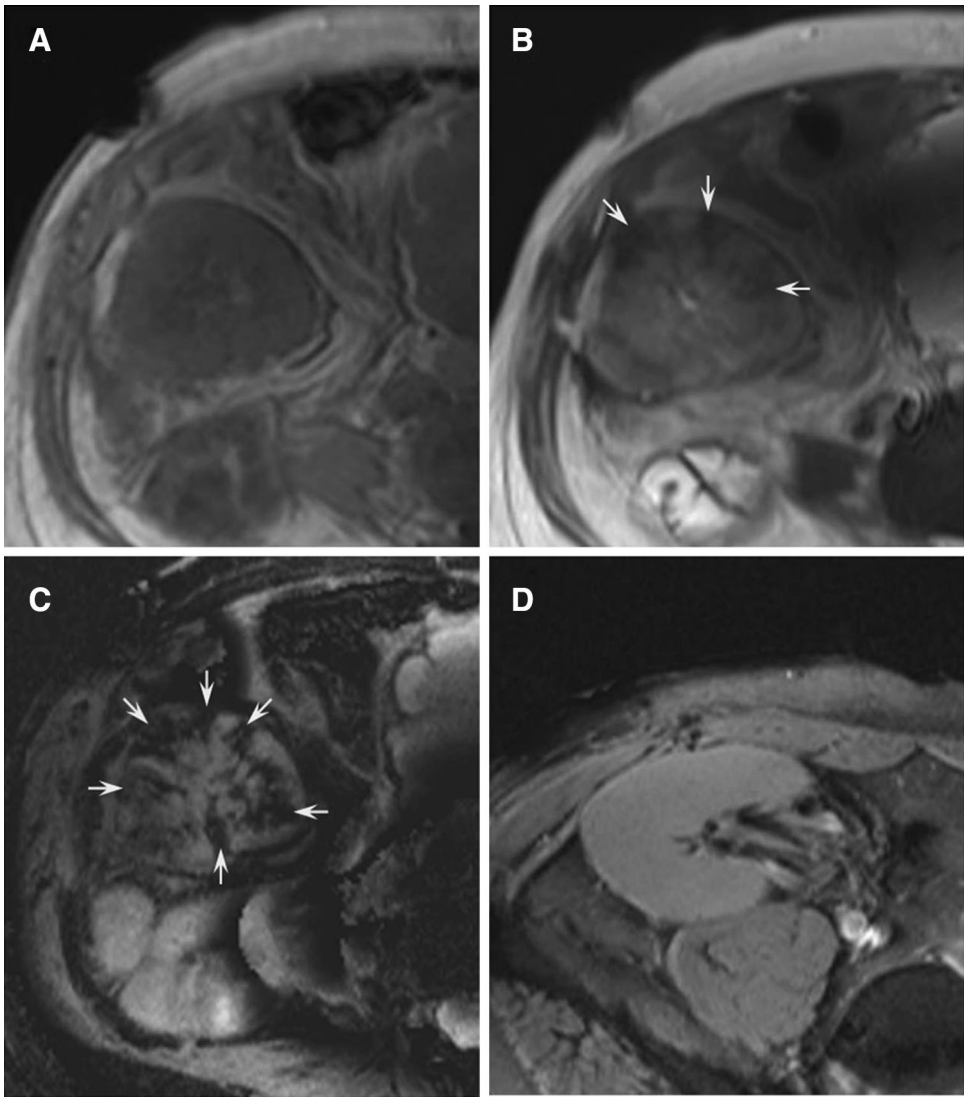

Fig. 1. **A**–**C** A 55-year-old female patient with delayed graft function in the early period after kidney transplantation. On T1WI (**A**), no ASILs were seen in the transplanted kidney. On both T2WI (**B**) and SWI (**C**), low-intensity ASILs (arrows), primarily located at the corticomedullary junction of the transplanted kidney, were found. (**D**) A 49-year-old male patient with non-delayed graft function in the early period after kidney transplantation. On SWI (**D**), no ASILs were seen in the transplanted kidney.

the corticomedullary junction on only SWI and not conventional MRI (Figs. 2, 3); and 18 recipients did not have ASILs on conventional MRI or SWI. In the non-DGF group ($n = 35$), no ASILs were found in the transplanted kidneys on either conventional MRI or SWI.

Quantitative analysis

In the DGF group, the detection rate of low-intensity ASILs was significantly higher on SWI than on conventional MRI [47.1% (16/34) vs. 5.9% (2/34), $P < 0.001$]. Renal allograft function measured by eGFR among the 16 recipients with low-intensity ASILs in the transplanted kidneys on SWI was significantly lower than that among the 18 recipients who did not have a low-intensity ASIL in the transplanted kidneys on SWI (8.5 ± 4.2 vs. 19.7 ± 9.7 mL/min, $P < 0.001$) (Fig. 4). There was a moderate negative correlation between the presence of low-intensity ASILs in transplanted kidneys on SWI and renal allograft function, as measured by eGFR, in recipients with DGF ($r = -0.553$, $P = 0.001$).

In the 16 recipients with DGF found to have a low-intensity ASIL in their transplanted kidneys on SWI, the signal intensity of the low-intensity ASIL was significantly lower than that of the renal cortex on SWI (112.6 ± 52.3 vs. 175.2 ± 50.6, $P = 0.002$).

Based on the combination of clinical data, laboratory data, and well-known definition of DGF as the diag-

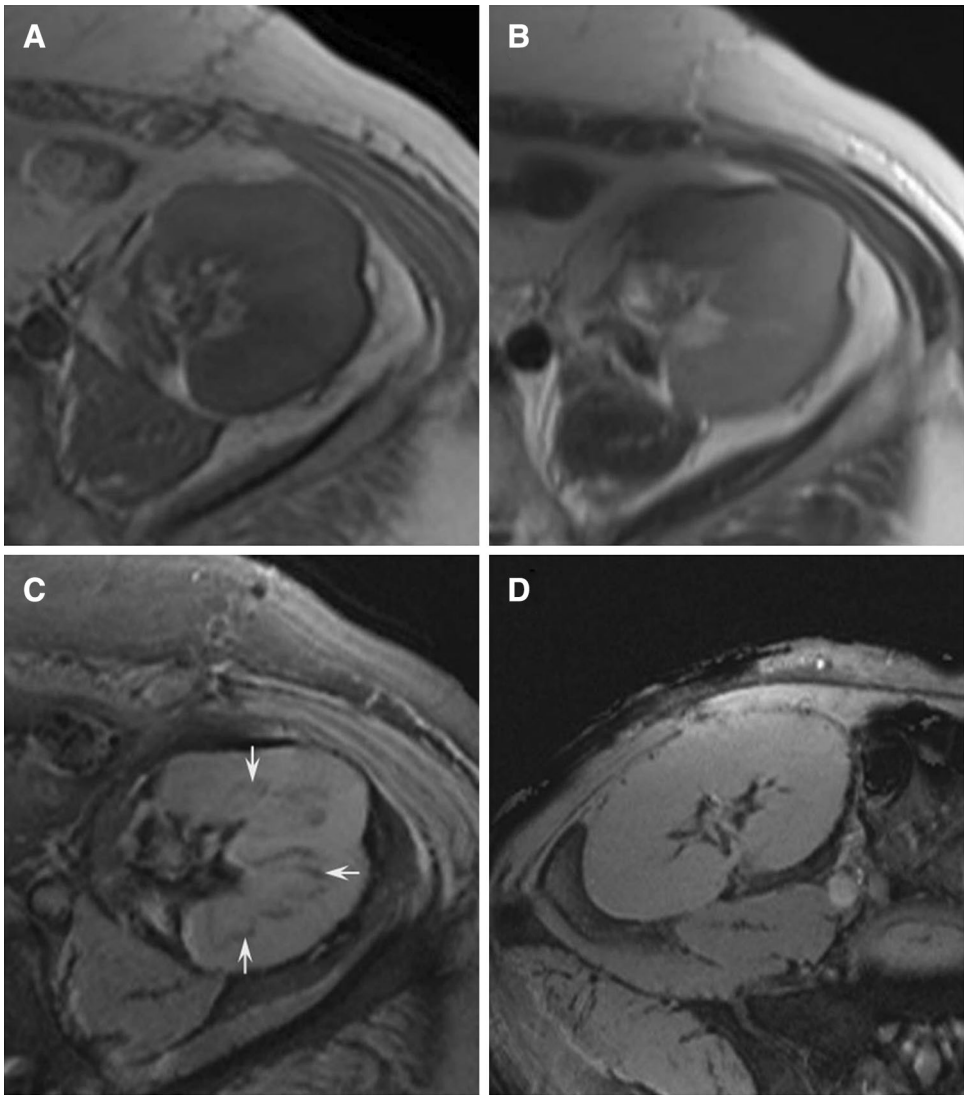

Fig. 2. (**A**–**C**) A 61-year-old male patient with delayed graft function in the early period after kidney transplantation. On both T1WI (**A**) and T2WI (**B**), no ASILs were seen in the transplanted kidney. On SWI (**C**), low-intensity ASILs (arrows), primarily located at the corticomedullary junction of the transplanted kidney, were found. (**D**) A 42-year-old male patient with non-delayed graft function in the early period after kidney transplantation. On SWI (**D**), no ASILs were seen in the transplanted kidney.

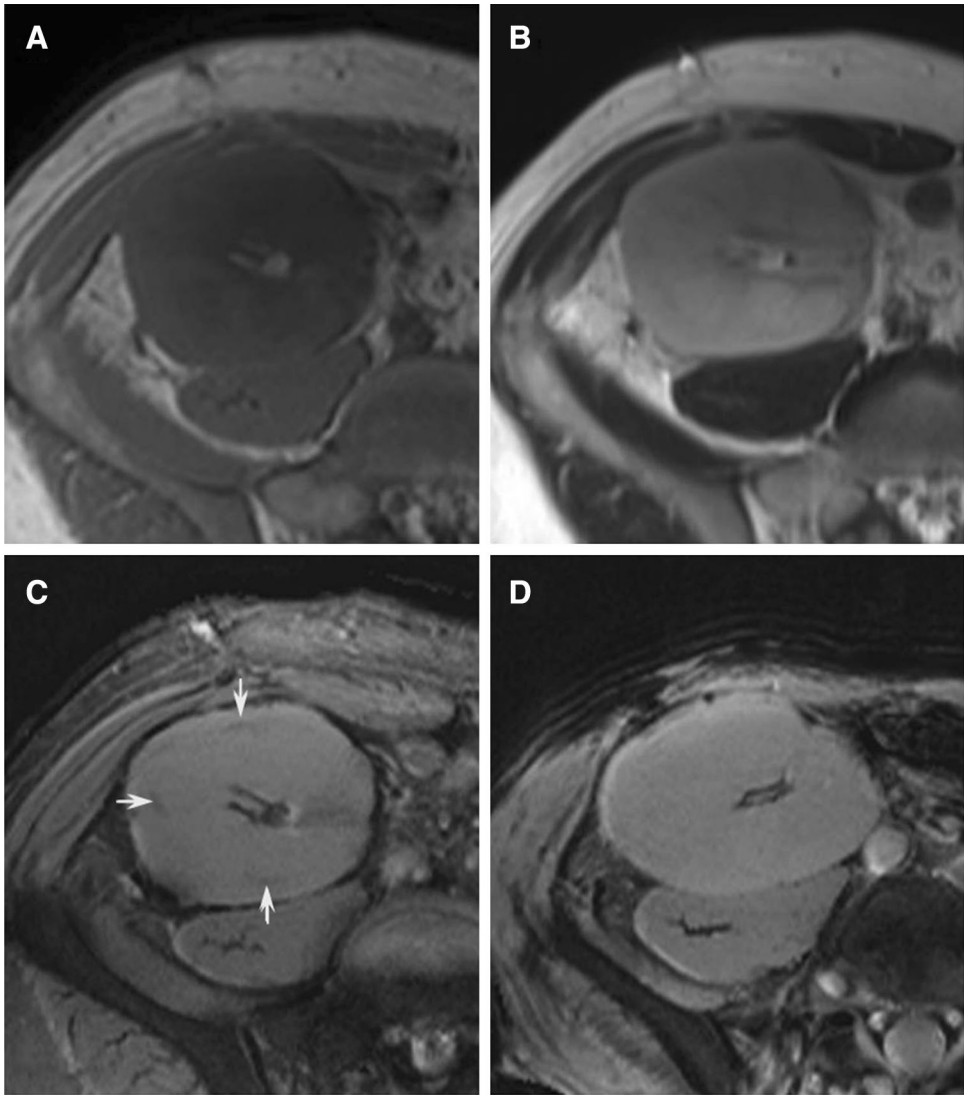

Fig. 3. (**A**–**C**) A 35-year-old male patient with delayed graft function in the early period after kidney transplantation. On both T1WI (**A**) and T2WI (**B**), no ASILs were seen in the transplanted kidney. On SWI (**C**), low-intensity ASILs (arrows), primarily located at the corticomedullary junction of the transplanted kidney, were found. (**D**) A 44-year-old male patient with non-delayed graft function in the early period after kidney transplantation. On SWI (**D**), no ASILs were seen in the transplanted kidney.

nostic standard of DGF, the sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of SWI for diagnosing DGF by detecting low-intensity ASILs primarily located at the corticomedullary junction were 47.1% (16/34), 100% (35/35), 100% (16/16), and 66.0% (35/53), respectively.

Discussion

This study demonstrated SWI can be used to evaluate the DGF in the early period after kidney transplantation, which is significantly better in evaluating DGF than conventional MRI. The detection of low-intensity ASILs (primarily located at the corticomedullary junction) on SWI had a specificity and PPV of 100% for diagnosing DGF for both measures.

GFR is usually considered the best index of renal allograft function [14]. It is well-known when the GFR is lower, renal allograft function is worse. GFR can be measured accurately by using exogenous markers, such as inulin clearance and radiolabeled isotopes, but these methods are laborious as well as expensive and rarely used in clinical practice [14]. Thus, endogenous markers, such as serum creatinine and cystatin C, are increasingly used to evaluate renal function [14]. The CKD-EPI creatinine-cystatin C equation has been proven to be more accurate than equations based on either of these markers alone for estimating GFR [12]. Our results indicate that

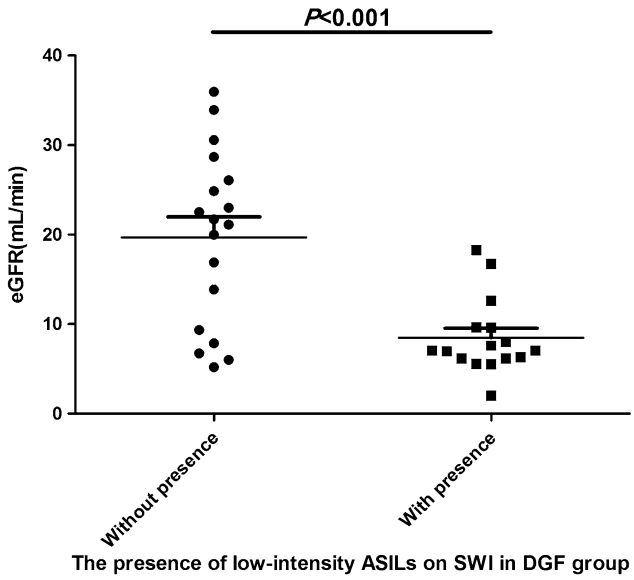

Fig. 4. The columnar scatter-plot shows that the eGFR value of DGF recipients with low-intensity ASILs in the transplanted kidneys on SWI was significantly lower than that without low-intensity ASILs.

allograft function in the DGF group was significantly worse than that in the non-DGF group, which in turn verified the accuracy of grouping DGF and non-DGF by means of combining the clinical data, laboratory data, and well-known definition of DGF.

Many studies have noted that severe ischemia-reperfusion injury (IRI) is the primary cause of DGF in kidney transplantation [15–17]. However, there are currently no effective therapies to prevent IRI [17]. Among multiple factors that leading to renal IRI, the extended cold ischemia time is considered the primary cause. Currently, static cold storage is the most common means for preserving donor kidneys, as it is simple and low cost, but this storage technique inevitably results in cold ischemia in donor kidneys [17]. During the cold ischemia period, the metabolic rate is decreased, which allows for prolonged preservation of the donor kidneys until transplantation [13]. Because kidneys from deceased donors generally need more cold ischemia time for preservation than those from living donors, the incidence of DGF in deceased donor kidneys is higher than that in living donor kidneys [4, 15]. The DGF incidence (49.3%) in this study was similar to the value reported in other studies [2, 4, 15].

Sixteen transplanted kidneys in the DGF group had low-intensity ASILs with a fuzzy boundary primarily located at the corticomedullary junction on SWI, and the signal intensity of the low-intensity ASIL was significantly lower than that of the renal cortex. The pathophysiological and histopathological changes caused by IRI were the primary causes of low-intensity ASILs on SWI among patients with DGF.

The pathophysiological changes in IRI are summarized as follows. First, donor kidneys suffer ischemic injury before transplantation. The ischemia results in insufficient oxygen and nutrient supplies, which leads to a decrease in oxidative metabolism, accumulation of metabolic waste products, and depletion of adenosine triphosphate [13]. Hypoxic and anoxic cell injuries trigger the inflammatory response in ischemic kidneys. Second, donor kidneys experience reperfusion injury after transplantation. Reperfusion gives rise to rewarming, reoxygenation, and a return to aerobic metabolism in the transplanted kidney [13]. Reactive oxygen species are rapidly generated, which directly injures the cytoskeletal and functional cellular components, even resulting in cell death [13]. The inflammatory response accelerates upon reperfusion with the upregulation of cytokines, release of chemokines, and activation of the complement system, which leads to the recruitment and migration of leukocytes [4, 18]. During reperfusion, renal tissue injury is further exacerbated by the inflammatory response. This series of pathophysiological changes results in a variation in the deoxy-to-oxy-hemoglobin ratio in the vessels of the renal cortex and medulla [19]. Deoxyhemoglobin is paramagnetic, but oxyhemoglobin is diamagnetic [19]. The variation in the deoxy-to-oxy-hemoglobin ratio results in local magnetic field inhomogeneity. Then, the local magnetic field inhomogeneity further leads to phase and susceptibility differences between the vessels and their surrounding tissue in the renal parenchyma. These local phase and susceptibility differences can be easily detected by SWI, manifesting as low-intensity ASILs [10, 19].

Acute tubular necrosis (ATN) is considered the major histopathological change in DGF [15]. ATN primarily results in death and the absorption of tubular epithelial cells, focal expansion of proximal tubules, partial occlusion of tubular lumens by cellular debris and multiple mitoses, endothelial injury, and interstitial hemorrhage [4, 15]. Interstitial hemorrhage is composed of various types of blood metabolites [8, 10]. In hemorrhagic lesions, deoxyhemoglobin has a marked hypo-intense signal and decreased T2*, and hemosiderin can generate a strong susceptibility effect leading to phase and susceptibility variation and a T2*-related signal decrease [8, 10]. It is generally known that the sensitivity of T2* to hemorrhage is enhanced with SWI [8]. Hemorrhage can be sensitively detected by SWI, appearing as low-intensity ASILs [10].

There were still 18 transplanted kidneys in the DGF group that didn't have low-intensity ASILs on SWI. The allograft function in these 18 recipients was proved to be significantly better than that in the 16 recipients with DGF who had low-intensity ASILs. There was a moderate negative correlation between the presence of low-intensity ASILs in the transplanted kidney on SWI and

allograft function in recipients with DGF. For DGF, the presence of low-intensity ASILs in the transplanted kidneys on SWI is not only a result of quantitative and qualitative changes in pathophysiology and histopathology caused by IRI but also a reflection of worsening allograft function. The sensitivity of SWI in diagnosing DGF was 47.1%, but the diagnostic specificity and PPV were both 100%. Moreover, once DGF is diagnosed using SWI, it is highly likely that allograft function is poor, and the recipient should be treated as soon as possible.

The low-intensity ASILs in transplanted kidneys with DGF on SWI were all primarily located at the corticomedullary junction, resulting from the unique structure and function of the kidney. In the normal physiological state, blood flow to the renal cortex usually far exceeds the metabolic needs of the kidney, whereas the renal medulla has a decreased blood supply and is relatively hypoxic [20]. The oxygen tension (P_{O_2}) ranges from 40–50 mmHg in the renal cortex and 10–15 mmHg in the renal medulla [21]. As a result, an obvious P_{O_2} gradient difference exists in the corticomedullary junction [22]. When DGF occurs in transplanted kidneys, the corticomedullary junction suffers the most from the hypoxic stimulation, inflammatory response, and cytokine release.

The clinical applicability of SWI assessing DGF in the early period after kidney transplantation: (1) The chief indication for performing SWI: recipients who need the dialysis during the first week post transplantation; (2) The optimal timing for performing SWI: within the second week (day 8–day 14) after transplantation; (3) Assessing process: in clinical practice, if a low-intensity ASIL primarily located at the corticomedullary junction of the transplanted kidneys on SWI is found on a recipient, he or she can be diagnosed with DGF. Moreover, according to our further study results, ''the presence of low-intensity ASILs on SWI'' suggests that the allograft function in these recipients is likely to be very poor, requiring immediate clinical intervention. This study shows SWI can be used for diagnosing DGF in the early period after kidney transplantation.

This study has some limitations. First, not all patients underwent percutaneous renal biopsy, as this invasive operation may cause many serious complications. The clinical data, laboratory data, and definition of DGF were used to group all cases. The grouping results were shown to be correct according to eGFR data, which demonstrated that allograft function in the DGF group was significantly lower than that in the non-DGF group. Second, in theory, MR examinations should be performed as early as possible after kidney transplantation. However, recipients must remain on bed rest during the first week. Thus, MR examinations were performed only in the second week after transplantation, which was the earliest time at which the procedure could be performed in practice. Third, the sample size of the DGF group was small. These preliminary results require validation with a larger sample size in the future.

In conclusion, SWI can be used to evaluate DGF during the early posttransplantation period. DGF can present with or without a low-intensity ASIL, which was primarily located at the corticomedullary junction of the transplanted kidneys on SWI. The presence of low-intensity ASILs on SWI showed a moderate negative correlation with renal allograft function in recipients with DGF. The recipients who have low-intensity ASILs in the transplanted kidneys on SWI can be diagnosed with DGF, and their allograft function are likely very poor, requiring immediate clinical treatment.

Compliance with ethical standards

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Conflict of interest All authors declare that they have no conflicts of interest.

Ethical approval All research procedures were conducted in accordance with the Declaration of Helsinki. The local ethics committee approved this retrospective study.

Informed consent Informed consent was obtained from all participants included in the study.

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