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Protective Effects of Quercetin and Sucrose on Cold Preservation Injury in Porcine Kidneys Donated After Cardiac Death

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Background: The University of Wisconsin (UW) solution is the gold standard for kidney preservation. Quercetin (QE), a widely used flavonoid, possesses strong antioxidant, anti-inflammatory, and anti-apoptotic properties. Sucrose (Suc) has shown beneficial effects as a supplement. The aim of this study was to investigate the protective effects of QE and Suc against cold injury in porcine kidneys and to evaluate whether their use improves ischemia-reperfusion (I/R) injury after simple CS.

Material/Methods: Kidney grafts were procured after 30 min of warm ischemia, followed by preservation under 2 conditions for 22 h: Group 1, preserved with CS/UW solution (n = 4), and Group 2, preserved with CS/UW solution containing QE 33.1 μM and Suc 0.1 M (n = 6). All grafts were evaluated using an ex vivo isolated kidney reperfusion model with oxygenated, diluted autologous blood at room temperature.

Results: At the end of reperfusion, creatinine phosphokinase and lactate dehydrogenase levels were significantly lower in Group 2 than in Group 1. We observed more vacuolation in the tubular epithelial cell in Group 1 than in Group 2, and the tubular epithelium showed more irregularity and exfoliation in Group 1 than in Group 2. Additionally, Group 2 had a significantly higher number of anti-ERG-positive endothelial cell nuclei than Group 1 at the end of reperfusion.

Conclusions: Our results suggest that the addition of QE and Suc to UW solutions can effectively prevent cold injury in DCD kidneys, with QE exerting this beneficial action, possibly by suppressing oxidative stress, and could potentially enhance kidney transplantation outcomes.

Keywords: Kidney • Nephrology • Organ Preservation • Transplantation

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Introduction

Shortages of organ donors is a universal problem in transplantation. The University of Wisconsin (UW) solution has been the gold standard for preserving the liver, kidneys, and pancreas since it was first developed by Belzer and Southard [1]. Quercetin (QE), a widely used flavonoid, possesses strong antioxidant [2], anti-inflammatory [3], and anti-apoptotic properties [4]. Some bioflavonoids have been reported to attenuate renal tubular cell injury during cold preservation in EuroCollins and UW solutions [5]. In addition, the beneficial effects of the preservation solution may be due to its ability to prevent tissue edema during cold storage (CS). Sucrose (Suc) has shown beneficial effects as a supplement [6]. The aim of this study was to investigate the protective effects of QE and Suc against cold injury in porcine kidneys and to evaluate whether their use improves ischemia-reperfusion (I/R) injury after simple CS. This study is investigating the mechanisms underlying the efficacy of QE and Suc by utilizing cytokines and immune tissues. We believe it will contribute to the development of new, more effective preservation solutions.

Material and Methods

Animals

The Institutional Animal Ethics Committee of the Clinical Research Center, Asahikawa Medical University, Japan (permit no. R4-089) approved all experimental procedures. We used domestic pigs (male, crossbred Large-Yorkshire, Landrace, and Duroc hogs, 2-3 months old, 30 kg; Taisetsu Sanroku-Sya, Asahikawa, Japan) in this study.

Midazolam (0.3 mg/kg), medetomidine hydrochloride (0.06 mg/kg), and butorphanol tartrate (0.03 mg/kg) were injected into porcine femoral muscle for anesthetic induction. The peripheral ear vein was cannulated, and Ringer's lactate solution was infused at a rate of 6.7 ~ 10 ml/kg/hr. Thiamylal sodium (4.2 mg/kg) was intravenously injected and intubation was performed. Anesthesia was maintained with propofol and oxygen (4 L/min). The internal jugular vein was then infused with 6% (w/v) hydroxyethyl starch (33.3 ml/kg/hr).

The domestic pigs underwent midline laparotomy. The right common iliac artery was isolated and a catheter was inserted to procure ~ 400 mL of autologous whole blood. The kidney ureter, artery, and vein were identified, and the blood flow cut off. We intravenously infused potassium chloride (2 mEq/kg) and cardiac arrest was induced. The kidneys were kept under warm ischemic time (WIT) conditions for 30 min which simulated the kidney donation after circulatory death (DCD). The kidney graft was retrieved and washed from the renal artery with 200 mL of each preservation solution at 4 °C as a back-table procedure. The kidney grafts were preserved under CS for 22 hours at 4 °C.

Study Design

The kidney grafts procured after 30 min of WIT were preserved under 2 conditions for 22 hours: Group 1, preserved with UW solution (n = 4) and Group 2, preserved with UW solution containing QE 33.1 μM and Suc 0.1 M (n = 6). Quercetin was dissolved in dimethyl sulfoxide (DMSO) to a concentration of 100 mg/mL. One milliliter of this solution, diluted to 10 mg/mL with DMSO, was dissolved in 1 L of deionized water to achieve a concentration of 33.1 μM.

Isolated Ex Vivo Reperfusion Model

Kidney function was evaluated using an isolated kidney reperfusion model. The grafts were then reperfused with oxygenated diluted autologous blood at room temperature. Reperfusion of the renal artery was set at 90-100 mm Hg and ~ 20 ml/min. The reperfusion fluid was autologous whole blood containing 500 mL saline, 250 mL dextran, 60 mL calcium gluconate hydrate, 22 mL 8.5% bicarbonate, and heparin. Heparin levels were adjusted to an activated clotting time of 300 s. The hematocrit was maintained at ~ 10-12%. The oxygenator was regulated to achieve physiological blood gas values (pO_2, ~ 290-300 mm Hg; pCO_2, 30-50 mm Hg).

Biochemistry

Blood urea nitrogen (BUN), creatinine (Cr), creatinine phosphokinase (CPK), and lactate dehydrogenase (LDH) levels in the perfusate were measured every 60 min during reperfusion to determine the viability of the preserved kidney grafts using standard biochemical methods. In addition, lactate levels were measured every 60 min during reperfusion using a blood gas analyzer (i-STAT 1 analyzer; Abbott Japan LLC, Tokyo, Japan).

Histopathological Evaluation

A kidney wedge biopsy was performed immediately after laparotomy, before and after preservation, and 1 and 3 h after isolated ex vivo reperfusion. The kidney tissue was fixed in 10% phosphate-buffered formalin and embedded in paraffin. Each sample was sectioned into 3 μm slices, dehydrated using ethanol, and hematoxylin-eosin staining was performed.

ERG immunohistochemistry was performed to assess endothelial cell damage in glomerular and peritubular capillary networks. Additionally, CD42b immunohistochemistry was performed to assess platelet aggregation in the glomerular tufts and around the renal tubules.

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Table 1. PCR primers.

Primer	Forward	Reverse
GAPDH	AGGAGTAAGAGCCCCTGGAC	GTGTGTTGGGGGATCGAGT
TNF-α	TTGTCGCTACATCGCTGAAC	CCAGTAGGGCGGTTACAGAC
IFN-γ	TTCAGCTTTGCGTGACTTTG	TGCATTAAAATAGTCCTTTAGGATCG
Caspase-3	GAATGGCATGTCGATCTGGT	TTGTGAAGGTCTCCCTGAGATT

A primary rabbit anti-ERG monoclonal antibody was used, obtained from Nichirei Bioscience Co.,

Ltd. (clone EP111; Tokyo, Japan) and a rabbit anti-CD42b polyclonal antibody from the Proteintech Group, Inc. (GeneID 2811; IL, United States). Envision-labeled polymer reagent (DAKO, Glostrup, Denmark) was used as the secondary antibody. ImageJ (U.S. National Institute of Health, Bethesda) was used to quantify the anti-ERG and CD42b positive area. ImageJ software was used, obtainable from https://imagej.nih.gov/ij/download.html. The number of anti-ERG staining-positive endothelial cell nuclei and the positive area of anti-CD42b staining were counted automatically in at least 10 randomly selected non-overlapping fields using the ImageJ software (Colour Deconvolution, green; Type, 8-bit; Threshold, 25-70, Analyzed Particles, 45).

Gene Expression

Quantitative reverse transcription polymerase chain reaction was used to determine the gene expression of endothelial-specific, inflammatory, and apoptotic proteins. Renal parenchyma biopsies were performed after laparotomy, before and after preservation, and 3 hours after reperfusion. The samples were stored at -80 °C until analysis. Total RNA was extracted from snap-frozen kidney biopsies using an miRNeasy Micro Kit (Qiagen, Valencia, CA, USA). RNA concentration was determined using a spectrophotometer (NanoDrop 2000; NanoDrop Technologies, Wilmington, DE, USA). Equal amounts of RNA were converted to complementary DNA using the Transcriptior First Standard cDNA Synthesis Kit (Roche, Basel, Switzerland), and complementary DNA levels were measured using a LightCycler 480 System II (Roche, Basel, Switzerland). The relative expression of the mRNA of interest was normalized to the housekeeping gene—glyceraldehyde-3-phosphate dehydrogenase (GAPDH). Data are presented as the relative quantification of GAPDH. **Table 1** summarizes sense and antisense primer sequences.

Statistical Analyses

We presented the results as the mean ± standard deviation. Statistical analyses were performed using Microsoft Excel 2013 (Microsoft Corp., Redmond, WA, USA). The results were analyzed using the *t* test and a repeated-measures ANOVA. *P* values < 0.05 were considered statistically significant.

Results

BUN, Cr, CPK, LDH, and Lactate in the Perfusate Solution

No BUN level differences were observed between the groups. At the end of reperfusion, the BUN levels in Groups 1 and 2 were 4.30 ± 0.56 and 4.65 ± 1.01 mg/dl, respectively ($P = 0.598$) (**Figure 1A**). Cr levels did not differ between groups. At the end of reperfusion, the Cr levels in Groups 1 and 2 were 0.45 ± 0.07 and 0.49 ± 0.45 mg/dl, respectively ($P = 0.431$) (**Figure 1B**).

At 0, 1, and 2 h, and at the end of reperfusion, CPK levels were significantly lower in Group 2 at 313.16 ± 171.12, 391.00 ± 184.33, 449.33 ± 189.95, and 504.66 ± 200.33 IU/L, respectively, than in Group 1 at 1249.75 ± 188.50, 1409.25 ± 204.43, 1490.25 ± 149.65, and 1504.00 ± 94.92 IU/L, respectively (Group 2 vs Group 1; $P < 0.005$, $P < 0.005$, $P < 0.005$, $P < 0.005$) (**Figure 1C**).

At 0, 1 h, and the end of reperfusion, the LDH levels were significantly lower in Group 2 than in Group 1, at 154.16 ± 25.54 vs 215.55 ± 35.42, 233.33 ± 61.90 vs 411.00 ± 101.88, and 573.66 ± 151.52 vs 836.00 ± 141.08 mg/dl (Group 2 vs Group 1; $P < 0.05$, $P < 0.05$, $P < 0.05$). The LDH levels at 2 h were not different between the groups (634.00 ± 166.12 [Group 1] vs 420.50 ± 141.77 mg/dl [Group 2]; $P = 0.080$) (**Figure 1D**). No differences were observed in lactate levels between the groups. At the end of reperfusion, the lactate levels in Groups 1 and 2 were 10.84 ± 0.86 and 10.38 ± 0.69 mg/dl, respectively ($P = 0.611$) (**Figure 1E**).

Histological Findings During Reperfusion

Hematoxylin-eosin staining revealed that at the end of reperfusion, there was more vacuolation in tubular epithelial cells in Group 1 than in Group 2. In addition, the tubular epithelium showed more irregularity and exfoliation in Group 1 than in Group 2. In Group 1, there was more interstitial inflammation and hemorrhaging than in Group 2 (**Figure 2A, 2B**).

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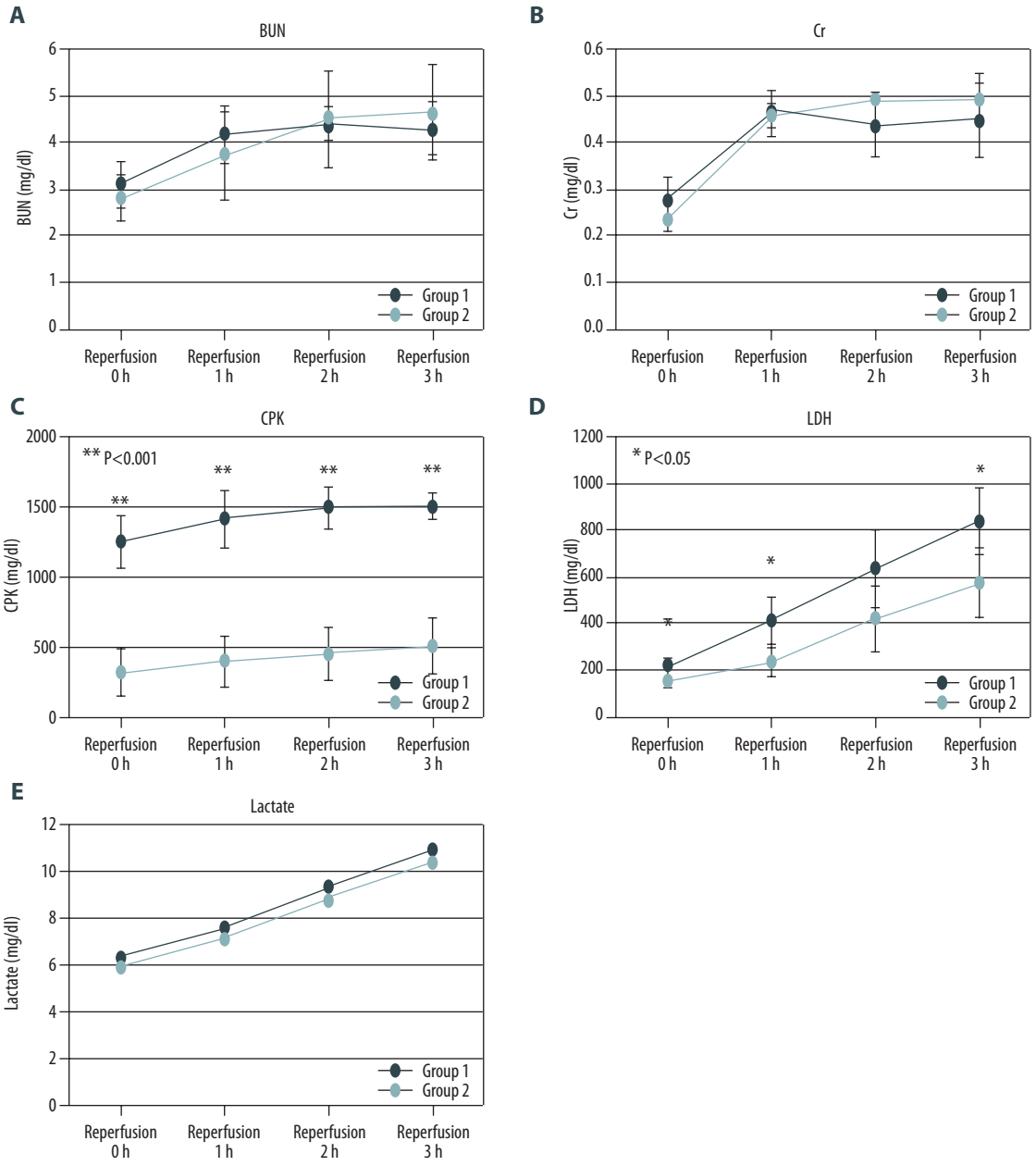

Figure 1. Laboratory test results. (**A**) BUN (mg/dl), (**B**) Cr (mg/dl), (**C**) CPK (IU/L), (**D**) LDH (mg/dl), and (**E**) lactate (mg/dl) levels (mean ± SD) in the perfusate solution before, and 1, 2, and 3 h after reperfusion.

ERG and CD42b Immunohistochemistry

At the end of reperfusion, Group 2 had significantly higher numbers of anti-ERG staining-positive endothelial cell nuclei than Group 1 (433.70 ± 151.69 [Group 2] vs 378.02 ± 119.29 [Group 1] per field; $P = 0.046$) (**Figure 3A-3C**). However, the positive areas of anti-CD42b staining at the end of reperfusion were not different between the groups (22087.62 ± 14258.80 [Group 1] vs 17176.51 ± 10944.07 [Group 2] per field; $P = 0.073$) (**Figure 4A-4C**).

Gene Expression

There were no differences in gene expression between groups. At the end of reperfusion, Groups 1 and 2 did not differ in the expression of the tumor necrosis factor (TNF-α) (0.2223 ± 0.0749 vs 0.1423 ± 0.0579; $P = 0.108$; **Figure 5A**), interferon-γ (IFN-γ) (0.0797 ± 0.0406 vs 0.0447 ± 0.0183; $P = 0.096$; **Figure 5B**), and caspase-3 (cas-3) (0.4381 ± 0.1785 vs 0.3269 ± 0.1156; $P = 0.354$; **Figure 5C**).

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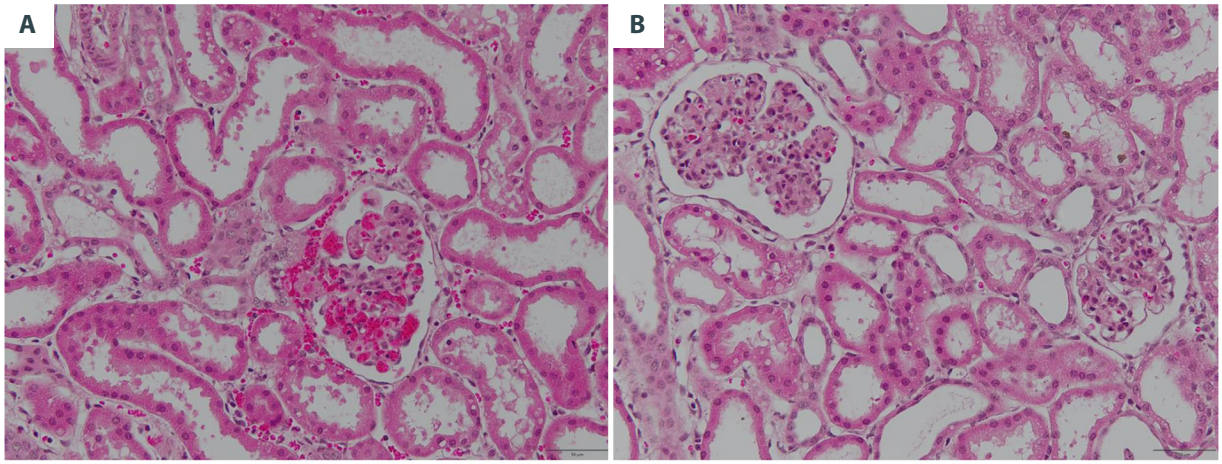

Figure 2. Hematoxylin-eosin staining at the end of reperfusion. (**A**) Group 1. (**B**) Group 2.

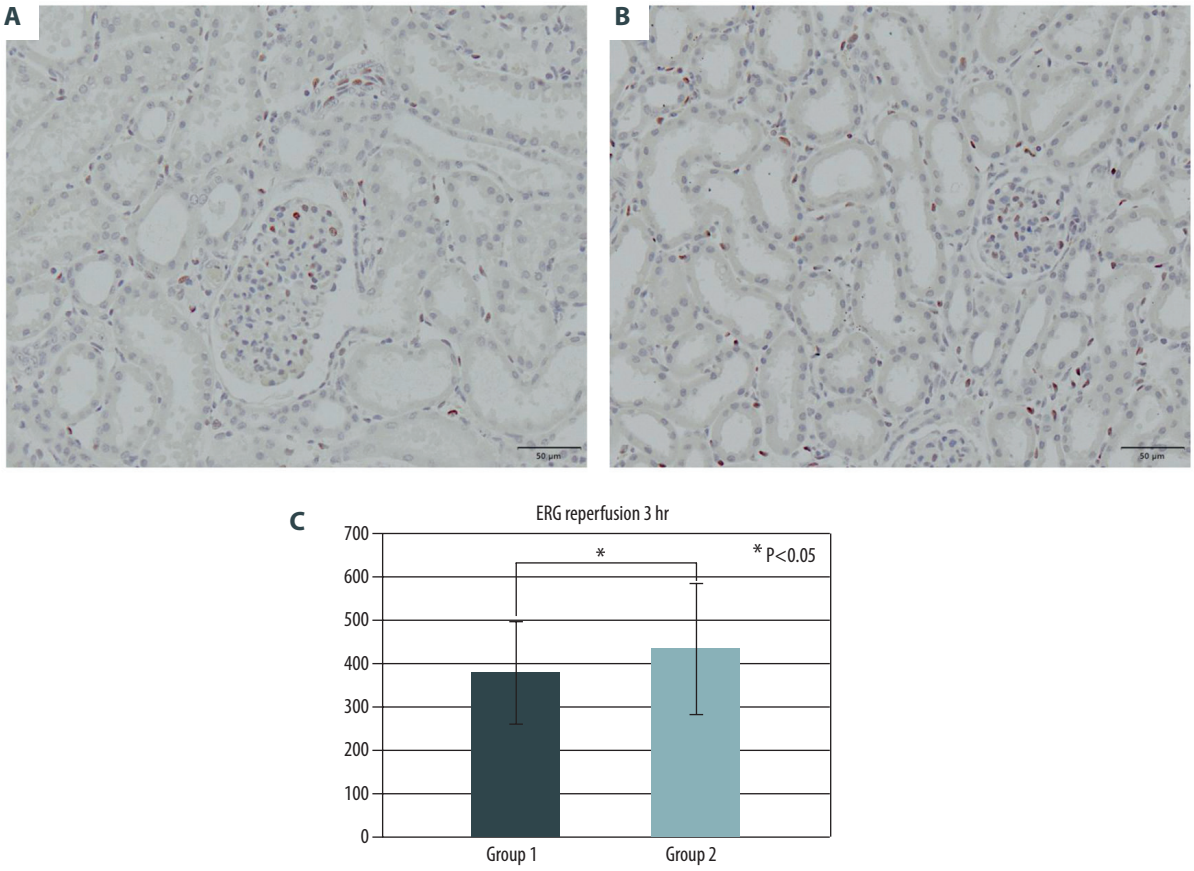

Figure 3. ERG immunohistochemistry at the end of reperfusion. (**A**) Group 1. (**B**) Group 2. (**C**) Quantification of anti-ERG positive area in Groups 1 and 2.

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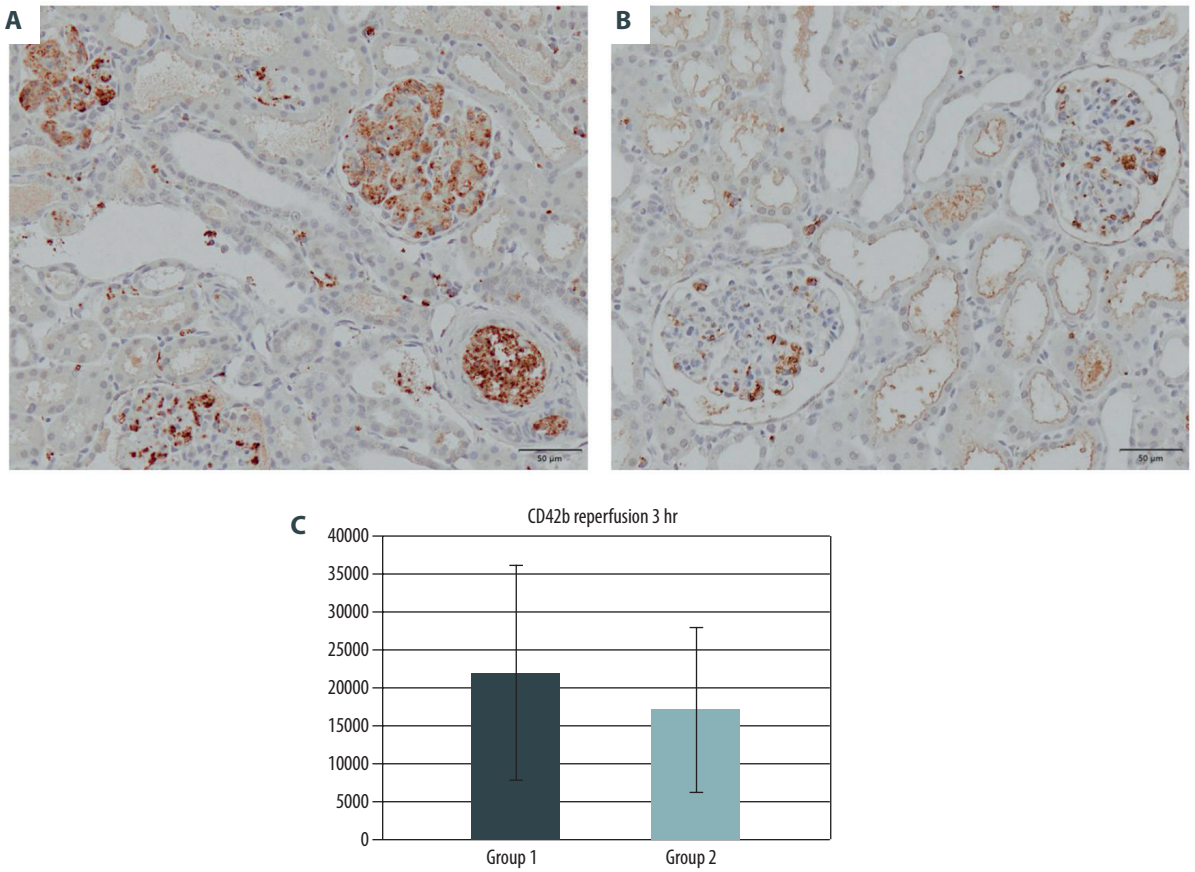

Figure 4. CD42b immunohistochemistry at the end of reperfusion. (**A**) Group 1. (**B**) Group 2. (**C**) Quantification of anti-CD42b positive area in Groups 1 and 2.

Discussion

Kidney transplantation is an established treatment for end-stage renal disease. However, the shortage of kidney grafts has become a global problem. DCD and other donors have been used to expand the available donor pool [7,8]; however, an optimal preservation solution for marginal kidney grafts, such as DCD kidneys, has not yet been developed.

CS is the easiest and most widely used preservation method for kidney transplants. However, prolonged CS can lead to postoperative complications, early graft dysfunction, and poor post-transplantation outcomes [9,10]. This is because ischemia during storage is associated with oxidative stress and apoptosis. Oxidative stress results from an imbalance between the production of reactive oxygen species (ROS), the level of antioxidants, and the effectiveness of repair processes [11]. ROS-mediated lipid peroxidation is hypothesized to be an important cause of cell membrane damage during oxidative stress [12].

The UW solution, commonly used for preservation, has limitations in preventing oxidative injury; however, the addition of antioxidants to preservations has resulted in improved cell survival [13]. There is increasing interest in new preservation techniques that resolve these problems, improve organ quality, and decrease severe complications. To this end, various new organ preservation strategies have been examined [3].

QE is an excellent in vitro antioxidant and the most potent ROS scavenger in the flavonoid family [14]. Inhibition of NF-κB activation required for tissue repair has been suggested to prevent apoptosis associated with renal I/R injury [15,16]. Kinaci et al reported that NF-κB expression was significantly decreased by QE treatment in an I/R rat model [4]. Thus, QE treatment reduced renal I/R injury by reducing apoptosis. The levels of enzymes such as superoxide dismutase, catalase, and glutathione peroxidase are reportedly insufficient during I/R in association with abundant ROS formation [17,18]. Inal et al reported that the superoxide dismutase, catalase, and glutathione peroxidase levels in the renal cortex of the I/R group were significantly lower than those in the control group; however, the difference was not significant [17]. These findings suggest that QE exerts a protective effect against ROS production during renal I/R.

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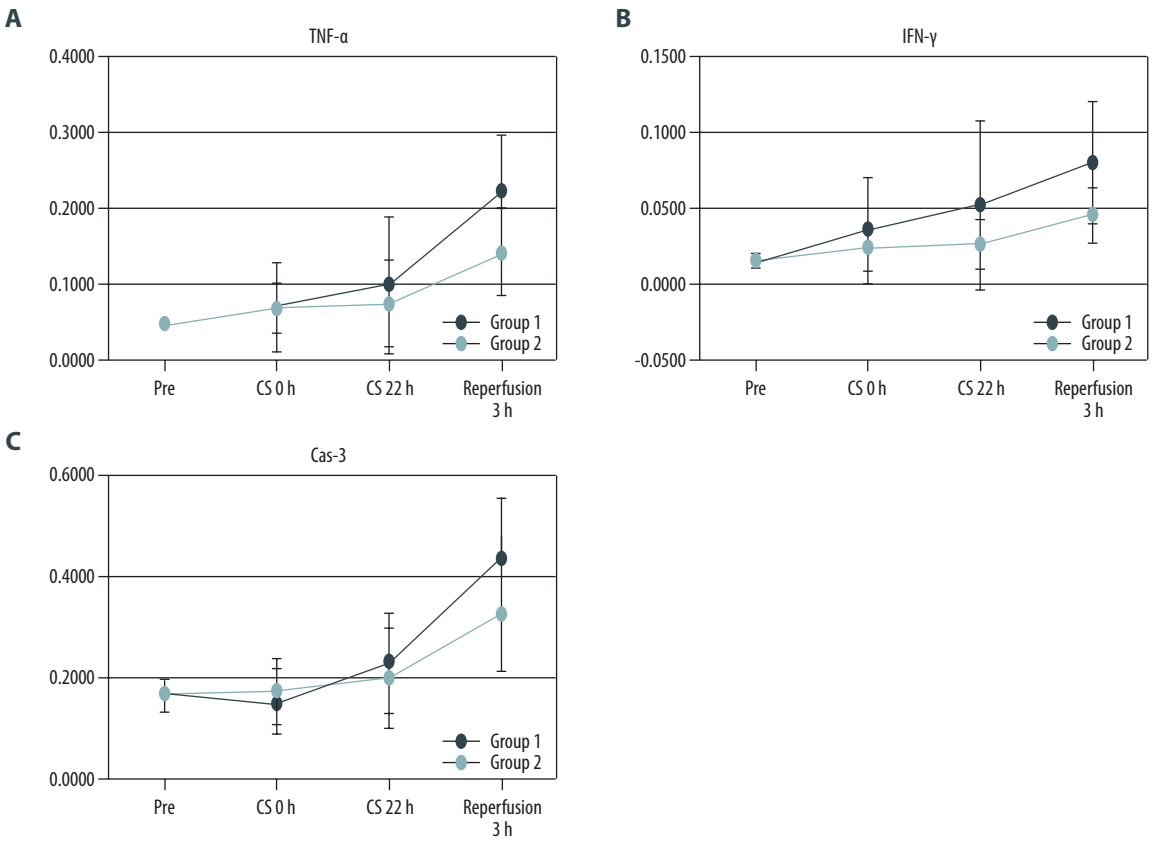

Figure 5. Polymerase chain reaction. (**A**) TNF-α, (**B**) IFN-γ, and (**C**) caspase-3 value after laparotomy, before and after preservation, and 3 h after reperfusion.

Considering its metabolism in the organs and the stability of its effects, the ideal QE concentration in a rat model of liver cold preservation and orthotopic liver transplantation was determined to be 33.1 μmol/l [19]. Kato et al showed that aspartate aminotransferase and alanine aminotransferase levels were significantly lower in the group treated with UW containing QE and Suc than in the group treated with UW alone after orthotopic liver transplantation in rats [19]. In addition, Gochi et al reported that 33.1 μM as the QE concentration used in a porcine model of renal autologous transplantation and showed lower lipid peroxidation levels and renal resistance when QE and Suc were combined as a machine perfusion solution in hypothermic oxygenated perfusion [20]. Because QE is less expensive than other flavonoid compounds, it is considered suitable for clinical applications from an economic perspective.

Here, the protective effects of a preservation solution containing QE and Suc during cold storage were demonstrated using early kidney function tests. Soares et al evaluated the effects of alprostadil in an experimental model of I/R injury in rat renal tissue and reported that CPK levels in the alprostadil-treated group were significantly lower than those in the vehicle-treated group [21]. Ahlenstiel et al reported that the addition of bioflavonoids to preservation solutions significantly reduced cell injury during cold storage by measuring LDH release in cultured renal tubular cells [5,22]. Our study revealed that Group 2 had significantly better CPK and LDH levels than Group 1 during reperfusion.

Kidney transplantation is associated with a high likelihood of endothelial injury in allografts [23]. Given its intimate contact with the blood, the endothelium is the target of choice for I/R injury, which causes tubular and endothelial damage, particularly in the peritubular capillary network and induces microvascular rarefaction. Jansen et al found that renal I/R injury in mice led to increased platelet activation in the immediate proximity of necrotic cell casts [24].

ERG immunohistochemistry experiments showed that Group 2 had significantly more endothelial cells during reperfusion than Group 1. CD42b immunohistochemistry experiments showed more platelet aggregation during reperfusion in Group 1 than in Group 2, although no significant difference was observed. These results indicate that the addition of QE and Suc to the UW solution can improve I/R injury.

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IFN-γ, which is associated with macrophage activation and neutrophil recruitment, may amplify the immune response following kidney reperfusion, mediating the early I/R injury phase [25,26]. Additionally, early renal tissue TNF-α expression contributes to neutrophil infiltration and injury after renal I/R [27]. Yang et al showed that caspase-3 is a pivotal regulator of microvascular endothelial cell apoptosis, renal fibrosis, and microvascular injury after I/R [28]. Here, IFN-γ, TNF-α, and caspase-3 levels were lower in Group 2 than in Group 1, although there were no significant differences. These results also suggest that the addition of QE and Suc to UW solutions alleviated I/R injury.

This study has several limitations. First, there was a surgical bias in the experiments using porcine models, and to reduce this, the number of samples should be increased in future studies to stabilize surgical procedures. Secondly, the protective effect was limited by the short I/R period used here. Further studies are required to investigate the mechanisms of action of QE and Suc in UW solutions using transplantation models.

Limitations

1. Because HES is controversially associated with renal impairment and coagulopathy, this may have been a critical confounding factor in the renal ischemia-reperfusion model.
2. The validity of studies on platelets (CD42b) under heparinization may be insufficient.
3. No quantitative evaluation based on an established scoring system has been conducted.

Conclusions

We investigated the utility of flavonoids QE and Suc in kidney transplantation using a reperfusion model in CS. Our results suggest that the addition of QE and Suc to UW solutions can effectively prevent cold injury in DCD kidneys, with QE exerting this beneficial action, possibly by suppressing oxidative stress, and could potentially enhance kidney transplantation outcomes.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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