ORIGINAL ARTICLE

Impact on biliary complications of donor abdominal aortic calcification among living donor liver transplantation: a retrospective study

Yuki Imaoka[1], Masahiro Ohira[1,2], Koki Sato[1], Shintaro Kuroda[1], Hiroyuki Tahara[1], Kentaro Ide[1], Tsuyoshi Kobayashi[1], Yuka Tanaka[1], Reo Kawano[3] & Hideki Ohdan[1]

1 Department of Gastroenterological and Transplant Surgery, Graduate School of Biomedical and Health Sciences, Hiroshima University, Hiroshima, Japan
2 Division of Regeneration and Medicine, Medical Center for Translational and Clinical Research, Hiroshima University Hospital, Hiroshima, Japan
3 Clinical Research Center in Hiroshima, Hiroshima University Hospital, Hiroshima, Japan

Correspondence
Masahiro Ohira MD, PhD, Department of Gastroenterological and Transplant Surgery, Graduate School of Biomedical and Health Sciences, Hiroshima University, 1-2-3, Kasumi, Minami-ku, Hiroshima 734-8551, Japan.
Tel.: +81-82-257-5222;
fax: +81-82-257-5224;
e-mail: mohira@hiroshima-u.ac.jp

SUMMARY

Abdominal aortic calcification (AAC) was reported as a poor prognostic factor among liver transplantation. However, donor AAC is not enough discussed. We analyzed the impact of the donor AAC level on graft function on outcomes following living donor liver transplantation (LDLT). A total of 133 consecutive patients who had undergone LDLT were divided into two groups (non-AAC group and AAC group) according to their donor AAC level by plain computed tomography. The rate of postoperative biliary complications (BC) was significantly higher in AAC group ($N = 17$) than in non-AAC group ($N = 116$; HR, 2.77; 95% CI, 1.32–5.83; $P = 0.0008$). The Cox proportional hazards regression model revealed that donor AAC (HR, 4.15; 95% CI, 1.93–8.97; $P = 0.0003$) and right lobe graft (HR, 2.81; 95% CI, 1.41–5.61; $P = 0.003$) increased the risk of BC. Conversely, splenectomy (HR, 0.39; 95% CI, 0.16–0.92; $P = 0.03$) decreased the risk of BC after LDLT independently. The long-term survival was also significantly worse in AAC group than in non-AAC group (HR, 2.25; 95% CI, 1.04–4.89; $P = 0.04$). Donor AAC was an independent prognostic factor for BC among patients undergoing LDLT. Although further investigations are needed to verify our results, the levels of donor AAC could be a useful tool to identify the risks of BC and predict better outcomes following LDLT.

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Key words
biliary complication, hepatocellular carcinoma, independent prognostic factor, liver transplantation, right lobe graft

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Introduction

A high incidence of biliary complications (BCs) remains the most intractable problem associated with living donor liver transplantation (LDLT). BC rates in LDLT recipients have been reported to be as high as 20–30% or more [1]. Representative BCs include bile duct stenosis, biliary leakage, anastomotic leakage, and acute cholangitis [1,2].

Several studies have focused on the arterial supply probably because of its significant surgical implications in liver transplantation and development of ischemic changes and strictures in the bile duct because of vasculobiliary injuries [3,4]. Therefore, we hypothesized that

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the reduction of the arterial blood flow, especially in the hepatic artery of the graft, causes serious biliary complications in LDLT.

Aortic calcification is a well-known risk marker of arteriosclerosis in cardiovascular disease, as it has been associated with coronary artery disease and stroke in the general population [5–7]. Abdominal aortic calcification (AAC) is easily evaluated using abdominal computed tomography (CT) scans [8]. We supposed that the clinical implication of incidental AAC findings in LDLT donors would reflect the peripheral arterial blood flow, as well as the hepatic arterial flow, of the graft. In terms of renal transplantation, the first report demonstrated that living donors with an AAC score of >100 required close observation because they had a higher probability of delayed renal function recovery after donation [8]. We first reported that the recipients with an AAC score of >100 required precise management because they had a poor prognosis after liver transplantation [9]. On the other hand, the influence of higher AAC on living donors remains unknown.

The objective of this study was to identify the influence of the donor AAC on outcomes after LDLT. We also aimed to assess the association between AAC and preoperative complications after LDLT, especially BC.

Patients and methods

Study patients

A total of 133 LDLTs have been performed in our unit from April 2008 to March 2018. All recipients and donors were adult. All recipients were observed longer than one year. Data concerning recipients at the time of transplant (recipient age, donor age gender, Model for End-Stage Liver Disease [10] score, Child–Pugh classification, graft-to-recipient weight ratio (GRWR), ABO incompatibilities, HBs antigen, HCV antibody, recipient AAC, donor AAC, diabetes, presence of hepatocellular carcinoma, operation time, bleeding volume, portal vein pressure, presence of splenectomy, and portal pressure at LDLT, presence of multiple bile duct, and diameter of hepatic artery and bile duct, graft types) were collected from electronic records. The rate of postoperative complications and perioperative complications [including BC, cytomegalovirus infection, perioperative bleeding, bloodstream infection (BSI), hepatic artery thrombosis (HAT), clinical acute rejection (AR), and refractory ascites] were also collected from electronic records after LDLT. No data were derived from transplants involving organs obtained from executed prisoners. Furthermore, this study conformed to the 1975 Declaration of Helsinki as reflected in an approval by the appropriate institutional review committee. (E-1410).

Definition of BCs

BCs were defined according to the criteria based on the Clavien–Dindo classification [11], including bile duct stenosis, bile leak, acute cholangitis, and stones. The biliary complication was classified as Grade III or higher if invasive treatment and examination occurred. The acute cholangitis was defined by Tokyo Guideline [12,13]. We considered severe cholangitis as one which needed invasive treatment, including a biliary drainage.

AAC levels

CT angiographies were performed using a standardized examination protocol on a 320-detector row CT scanner (Aquilion ONE ViSION; Toshiba Medical Systems, Tochigi, Japan) from 2013. AAC score was calculated using AZE VirtualPlace Lexus64 Anatomia (AZE Inc., Tokyo, Japan). Using the Agatston method [14], the AAC volume was automatically calculated for calcifications located in the abdominal aorta (from the origin of the renal artery to the iliac bifurcation) with attenuation greater than the predefined 130 Hounsfield unit (HU) level [14]. AAC levels were categorized two groups (non-AAC group: 0 mm^3, AAC group: 0 mm^3 <).

Procedures in LDLT

The procedures for donor evaluation, donor surgery, recipient surgery, and perioperative management followed in our hospital have been described in previously published study [15]. In the brief, we performed duct-to-duct biliary reconstruction. An end-to-end anastomosis between the graft and recipient bile ducts was performed using an interrupted 6-0 PDS, beginning from the posterior wall and terminating at the anterior wall. A stent tube was routinely placed through the anastomosis as a splint and was pulled out through the common bile duct above the duodenum. In all cases, the portal vein pressure (PVP) was measured intra-operatively after allograft implantation. After biliary reconstruction, if the PVP was higher than 15 mmHg, we decided to add splenectomy to control the portal hypertension.

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Liver Doppler ultrasound examination

We evaluated the blood flow to the graft on postoperative day 1, using the Doppler ultrasound examination in 93 LDLT recipients after 2009. Resistance index (RI), peak systolic velocity (PSV), and end-diastolic velocity (EDV) were investigated at the position of the hepatic artery anastomosis, and the portal vein flow (PVF) was checked at the level of the portal trunk.

Statistical analysis

All data were retrospectively analyzed. Data for categorical variables are expressed as numbers and percentages. For continuous variables, data are reported as the median with interquartile range (IQR). Differences in ACC group and non-ACC group were compared with Mann–Whitney test. Fisher's exact test was used for categorical variables. Univariate and multivariate Cox regression analyses were performed to assess the association of the incidence rate of BC with all the variables: recipient age, donor age, gender, MELD score, GRWR, ABO incompatibilities, HBs antigen, HCV antibody, recipient AAC, donor AAC, presence of hepatocellular carcinoma, operation time, cold ischemic time, bleeding volume, presence of splenectomy, presence of multiple bile duct, diameter of hepatic artery and bile duct, graft types, and CMV infection after LDLT. All variables were included in the multivariate models and the backward elimination method with removal criterion $P = 0.05$ was used to select covariates. The incidence rate of BC and overall observed survival were evaluated using the Kaplan–Meier method and the log-rank test. All statistical analyses were two-sided, and P-values < 0.05 were considered statistically significant. Analyses were performed using JMP statistical software (JMP® 14; SAS Institute Inc., Cary, NC, USA).

Results

Patient characteristics

The cohort included 73 men and 60 women with an overall median age (interquartile range) of 58 years (51.5–63 years) at transplantation. This study included 69 right lobe grafts and 64 left lobe grafts. A scatterplot shows the age and AAC distribution in Fig. 1. Spearman's rank correlation coefficient between AAC and age in recipients and donors were 0.39 and 0.39, respectively. We divided the patients into two groups according to the donor AAC levels (AAC group: 0 mm^3< and non-AAC group: 0 mm^3). We evaluated the data according to patient characteristics, surgical procedures, and the postoperative complications within one year. Table 1 summarizes this study population. In this population, AAC group was significantly older than non-

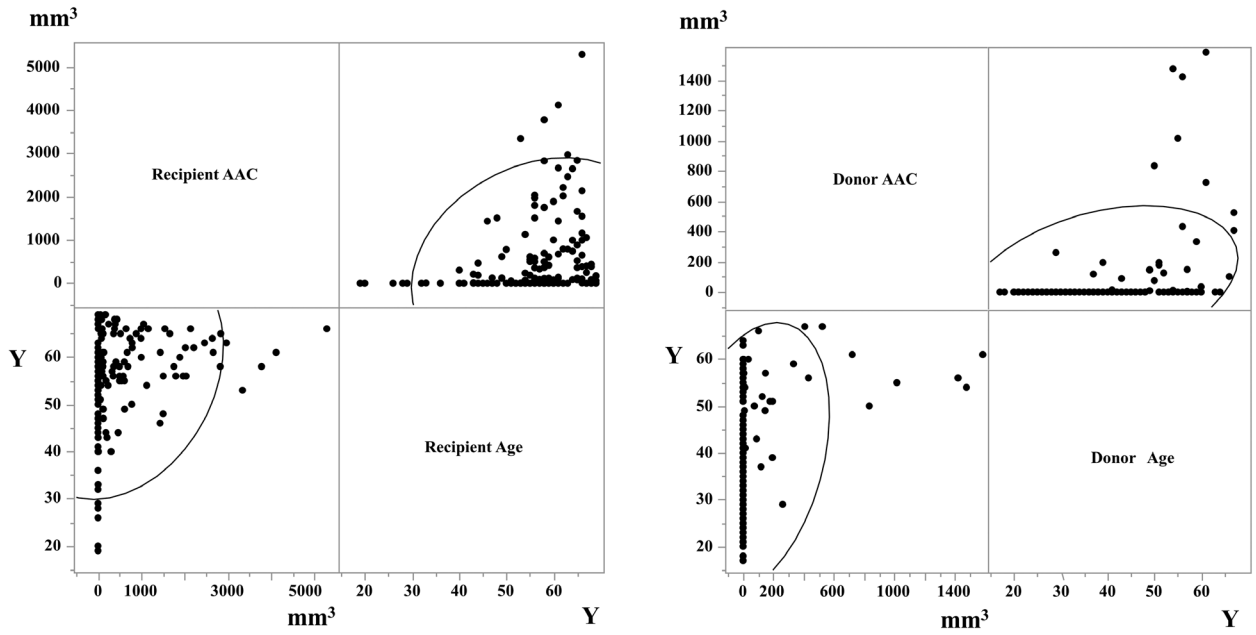

Figure 1 A scatterplot showing the age and AAC distribution. Spearman's rank correlation coefficient between AAC and age in recipients and donors were 0.39 and 0.39. AAC, abdominal aortic calcification.

Table 1. Patient characteristics in the AAC group and non-AAC group.

Subjects	AAC group (*N* = 17)	Non-AAC group (*N* = 116)	*P*-value
Gender: male/female	4/13	69/47	0.01
Recipient age (years)	59 (53–64.5)	58 (51.25–62.75)	0.41
Donor age (years)	55 (42–59.5)	36 (28–44.75)	0.0001
MELD (points)	16 (14–22.5)	16 (9–21.5)	0.42
Child–Pugh classification			
A	2	9	0.39
B	3	32	
C	12	75	
GRWR (%)	0.91 (0.83–1.00)	0.86 (0.75–100)	0.19
ABO incompatibilities +/−	0/17	16/100	0.22
Hepatitis virus			
Nonvirus	10	56	0.19
HCV	3	45	
HBV	4	15	
DM +/−	1/16	26/90	0.19
CIT (min)	77 (46–106)	59 (46–98)	0.08
Operation time (min)	692 (663–800)	763.5 (695.25–840)	0.23
Bleeding volume (ml)	3580 (2898–4595)	3800 (2540–5964)	0.58
HCC +/−	9/8	52/64	0.61
Recipient AAC (mm^3)	98 (0-648)	57 (0-609)	0.70
Multiple bile ducts +/−	2/17	30/86	0.36
Diameter of hepatic artery (mm)	3 (2.5–4)	3 (3–4)	0.30
Diameter of bile duct (mm)	6 (5–8)	6 (5–7)	0.68
Left lobe graft/right lobe graft	12/5	52/64	0.07
Biliary complication	9 (52.9%)	29 (25.0%)	0.02
Bile duct stenosis	3 (17.7%)	21 (18.1%)	
Bile leak	3 (17.7%)	9 (7.8%)	
Severe cholangitis	4 (23.5%)	3 (2.6%)	
Refractory ascites within 14 days	8 (47.1%)	20 (17.2%)	0.01
Postoperative hemorrhage	3 (17.7%)	24 (20.7%)	1.00
Bloodstream infection within 30 days	6 (35.3%)	24 (20.7%)	0.21
Hepatic artery thrombosis	0 (0%)	0 (0%)	–
CMV infection	6 (35.3%)	59 (50.9%)	0.30
Clinical rejection	5 (29.4%)	28 (24.1%)	0.76

AAC, abdominal aortic calcification; CIT, cold ischemic time; CMV, cytomegalovirus; DM, diabetes mellitus; GRWR, graft-to-recipient weight ratio; HBV, hepatitis B virus; HCC, hepatocellular carcinoma; HCV, hepatitis C virus; MELD, Model for End-Stage Liver Disease.

Median (interquartile range).

AAC group (55 vs. 36 years, $P = 0.0001$). In addition, AAC group had a significantly larger proportion of female recipients compared with non-AAC group ($P = 0.01$, respectively). The rate of BCs within one year after LDLT was significantly higher in AAC group than in non-AAC group (52.9% vs. 25.0%, $P = 0.02$). The rate of bile leakage in AAC group was 17.7% and 18.1% in non-AAC group. The rate of bile duct stenosis in AAC group was 31.3% and 25.0% in non-AAC group. The rate of severe cholangitis which needs the invasive therapy was higher in AAC group compared with non-AAC group (23.5% vs. 2.6%). Refractory ascites occurred more frequently after LT in AAC group compared with non-AAC group. (47.1% vs. 17.2%, $P = 0.01$).

Risk factors for BCs

Next, we analyzed the risk factors for BCs in univariable and multivariable analyses (Table 2). Multivariate analysis revealed that donor AAC (HR, 4.15; 95% CI, 1.93–8.97; $P = 0.0003$), small-sized bile duct (<4 mm; HR, 2.23; 95% CI, 1.04–4.77; $P = 0.04$), and right lobe graft (HR, 2.81; 95% CI, 1.41–5.61; $P = 0.003$) increased the

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Table 2. Risk factors for biliary complications.

		Univariable analysis			Multivariable analysis		
Subject	*N* = 131	HR	CI	*P*-value	HR	CI	*P*-value
Male	73	1.18	0.63–2.20	0.60			
Recipient age >60 years	51	0.80	0.42–1.52	0.50			
Donor age >50 years	27	1.19	0.55–2.57	0.66			
MELD >20 (points)	38	1.17	0.59–2.35	0.64			
GWRW >0.8	90	1.46	0.73–2.93	0.27			
ABO incompatibilities	16	1.55	0.63–3.56	0.35			
HCV antibody positive	49	1.09	0.59–2.04	0.76			
HBV antigen positive	19	1.26	0.56–2.85	0.57			
Recipient AAC >0 mm^3	54	0.99	0.53–1.84	0.98			
Donor AAC >0 mm^3	17	2.77	1.32–5.83	0.008	4.15	1.93–8.97	0.0003
CIT >70 min	75	1.57	0.85–2.90	0.15			
Operation time >6 h	86	1.00	0.52–1.91	1.00			
Bleeding volume >5 l	46	0.87	0.45–1.67	0.75			
Right lobe graft	69	2.50	1.28–4.92	0.008	2.81	1.41–5.61	0.003
Diameter of hepatic artery <3 mm	81	1.52	0.78–2.93	0.21			
Diameter of bile duct <4 mm	19	2.13	1.02–4.48	0.04	2.23	1.04–4.77	0.04
Multiple bile duct	32	1.71	0.90–3.26	0.10			
Splenectomy	34	0.41	0.17–0.99	0.03	0.39	0.16–0.92	0.03
CMV infection after LDLT	65	0.89	0.48–1.64	0.70			

AAC, abdominal aortic calcification; CIT, cold ischemic time; CMV, cytomegalovirus; GRWR, graft-to-recipient weight ratio; HBV, hepatitis B virus; HCV, hepatitis C virus; LDLT, living donor liver transplantation; MELD, Model for End-Stage Liver Disease.

risk of BCs. On the other hand, splenectomy (HR, 0.39; 95% CI, 0.16–0.92; $P = 0.03$) decreased the risk of BCs after LDLT, independently. The donor age was not an independent risk factor for BCs.

The cumulative BC rate and long-term survival according to AAC levels

The cumulative BC rate was shown in Fig. 2. AAC group had a significantly higher risk of BC compared with non-AAC group (Fig. 2: HR; 2.27, 95% CI; 1.93–8.97, $P = 0.008$). The 1-, 3-, and 5-year BC rates were 27.1%, 30.4%, and 30.4% in non-AAC group, respectively, and were 66.1%, 66.1%, and 66.1% in AAC group, respectively ($P = 0.005$). In the Kaplan–Meier survival curve analysis, the long-term survival was significantly worse in AAC group than in non-AAC group (Fig. 3, HR; 2.25, 95% CI; 1.04–4.89, $P = 0.04$). The 1-, 3-, and 5-year survival rates were 84.4%, 79.9%, and 75.8% in non-AAC group, respectively, and were 58.8%, 58.8%, and 49.0% in AAC group, respectively ($P = 0.04$).

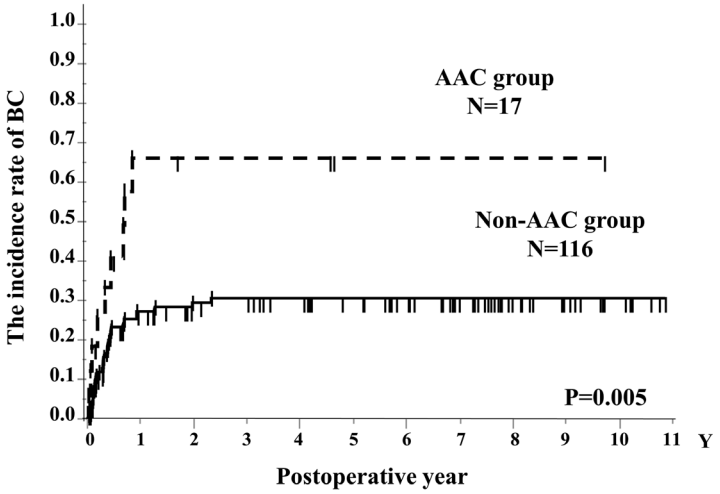
The Doppler ultrasound examination findings

We evaluated the blood flow to the graft on postoperative day 1 according to donor AAC levels, using the Doppler ultrasound examination in 93 LDLT recipients after 2009. The results of the Doppler ultrasound examination were summarized in Fig. 4. The RI in patients with AAC donors was significantly higher than in those with non-AAC donors (0.79 vs. 0.73, $P = 0.0014$). In addition, the EDV in patients with AAC donors was significantly lower than that in patients with non-AAC donors (7 cm/s vs. 11.3 cm/s, $P = 0.001$). There were no significant differences for PSV and portal vein flow.

Discussion

In this study, donor AAC was an independent risk factor for BC after LDLT. However, no publications have demonstrated the difference in BCs based on the degree of donor ACC. This study is the first to demonstrate the influence of donor AAC on the risk of BC after LDLT.

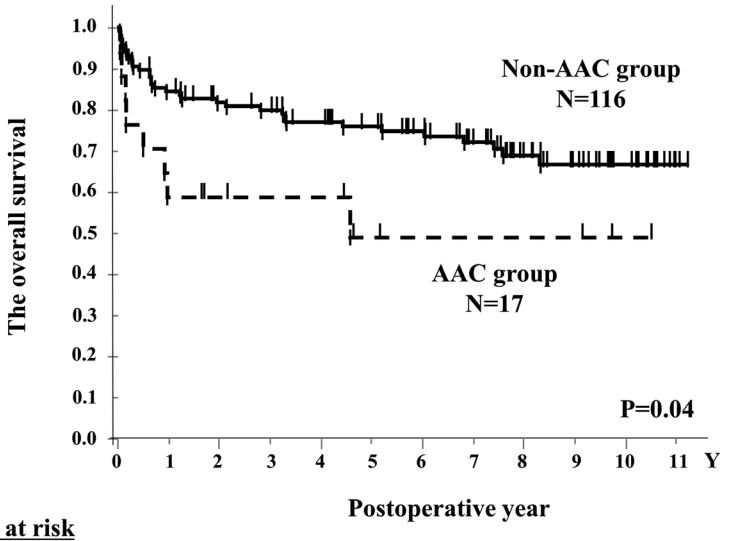
Aortic calcification is associated with coronary artery disease and stroke in the general population [5–7]. The relationship between abdominal aortic and coronary artery calcification has been demonstrated using AAC in chronic kidney disease patients [16]. Along with the advancement of imaging devices, the AAC can be calculated and quantified accurately and automatically [8]. In the field of gastrointestinal surgery, only one study has



Patients at risk

Non-AAC group	116	64	56	43	20	7
AAC group	17	4	4	2	2	1

Figure 2 The cumulative BC rate according to donor AAC. AAC group had a significantly higher risk of BC compared with non-AAC group (HR; 2.27, 95% CI; 1.93–8.97, $P = 0.008$). AAC, abdominal aortic calcification; BC, biliary complication.



Patients at risk

Non-AAC group	116	89	78	62	34	17
AAC group	17	9	8	4	4	2

Figure 3 The overall survival according to donor AAC. The long-term survival was significantly worse in AAC group than in non-AAC group (HR; 2.25, 95% CI; 1.04–4.89, $P = 0.04$). AAC, abdominal aortic calcification.

reported that high AAC was a risk factor for clinically relevant postoperative pancreatic fistula in elderly patients who underwent pancreaticoduodenectomy [17]. We previously reported the deleterious effects of high recipient AAC by demonstrating that a high recipient AAC level was a risk factor for a poor prognosis after

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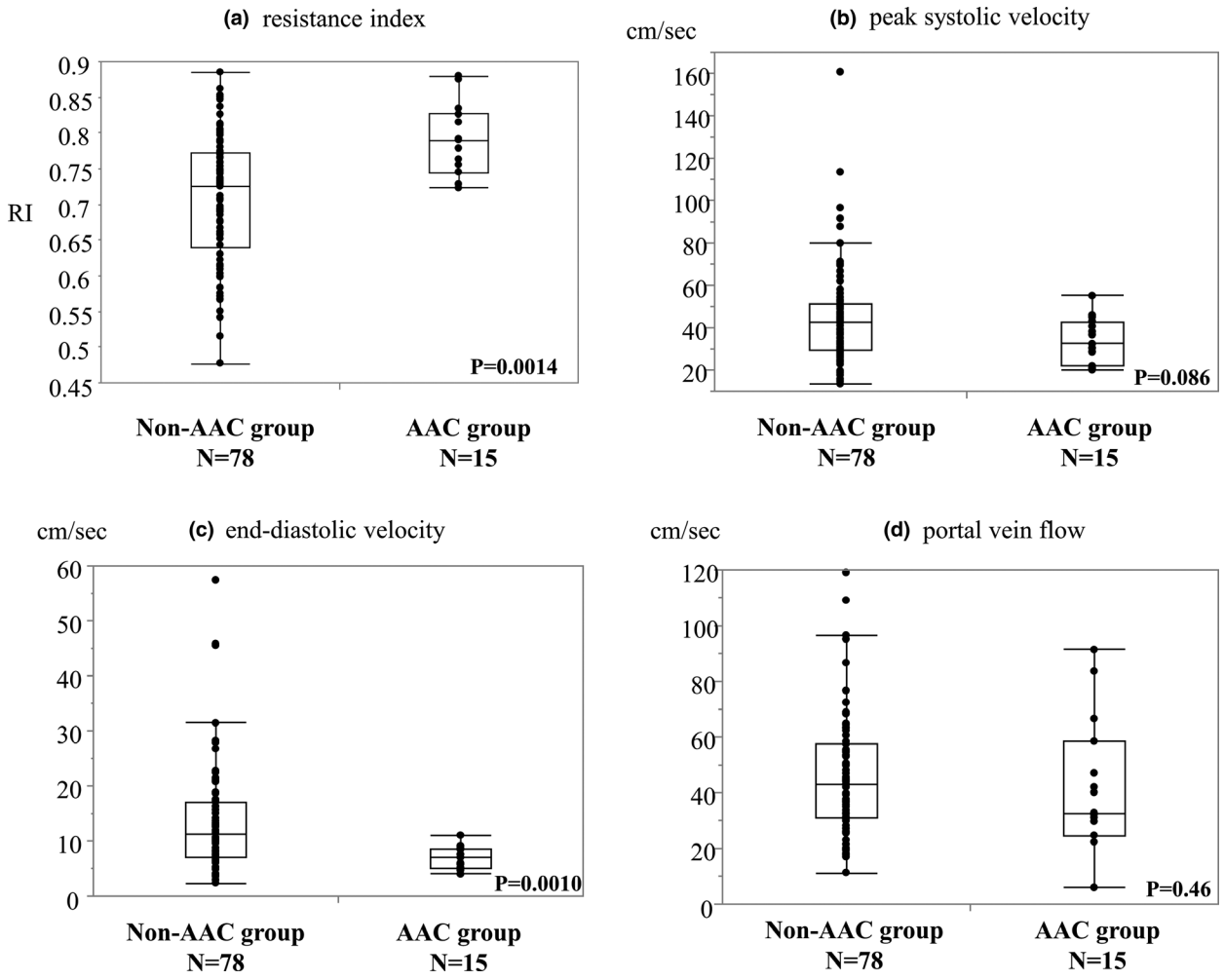

Figure 4 The Doppler ultrasound examination. The RI (resistance index) in patients with AAC donors was significantly higher than in those with non-AAC donors. In addition, the EDV (end-diastolic velocity) in patients with AAC donor group was significantly lower than that in patients with non-AAC donor group. Statistical differences were detected by the nonparametric Mann–Whitney *U*-test. AAC, abdominal aortic calcification; EDV, end-diastolic velocity; PSV, peak systolic velocity; PVF, portal vein flow; RI, resistance index. (a) Resistance index. (b) Peak systolic velocity. (c) End-diastolic velocity. (d) Portal view flow.

LT [9]. The previous results also suggested that chronic inflammation caused by arteriosclerosis may result in partial organ dysfunction [9].

The previous studies showed several predictive factors for BC after LDLT, including postoperative bleeding requiring re-operation [18], prolonged CIT [1], right liver grafts [19], a small-sized duct (less than 4 mm in diameter) [2,20], cytomegalovirus infection [20], ductoplasty [19,20], HAT [19,20], and a graft presenting with multiple bile ducts [19,21]. In the present study, the results showed that the risk factors for BC were similar to the previous reports, except for donor AAC. Gender did not appear to be an independent risk factor for BCs. The donor AAC group had a larger number of females. Therefore, the smaller diameter of anatomical structures in females was considered as a risk factor for anastomotic complications in LT. However, the diameter of bile ducts was not significantly different between the non-AAC and AAC groups. Therefore, gender was not an independent risk factor for BCs in this study.

In terms of anatomical knowledge, minimized peeling around the common bile duct is important to prevent BC. Fine branches from the posterior–superior pancreaticoduodenal, retro-portal, gastroduodenal, hepatic, and cystic arteries form two plexuses to supply the bile ducts [3,4]. The paracholedochal plexus, as right and left marginal arteries, runs along the margins of the bile duct and the reticular epicholedochal plexus lies on the surface [3,4]. After biliary tract reconstruction, the blood flow from these blood vessels is interrupted, and the blood flow of the bile duct depends on the hepatic artery. A recent study showed the possible effect that

atherosclerosis could have on the common bile duct diameter by affecting its smooth muscle contractility and blood flow, when evaluating AAC [22]. We reported that recipient AAC had a relationship with low EDV and high RI among LT [23]. The analysis of the Doppler ultrasound examination in 93 LDLT recipients indicated that donor AAC showed a relationship with low EDV and high RI in the non-AAC group, reflected by the flow of the hepatic artery. On the contrary, donor AAC showed no relationship with PVF. The low flow of the hepatic artery possibly led to BC, such as bile duct stenosis and bile leak. Therefore, this study suggests that donor AAC is a potential index that reflects the peripheral arterial blood flow. The hepatic artery seems to play an important role in the blood supply to the bile duct. Among LDLT recipients with a high incidence of BC, donor AAC may be a predictive factor for BC.

Aged grafts are known to have poor survival, even after LDLT [24,25]. In our department, a multivariate logistic regression analysis indicated that a donor age ≥50 years remained an independent risk factor for poor survival after LDLT [23]. High donor AAC in LDLT may also reflect the arteriosclerosis of the graft. In our study, AAC group had a lower EDV and higher RI of the hepatic artery on postoperative day 1. The arteriosclerosis of the graft led to the increased vascular resistance and increased postoperative refractory ascites. AAC group included a larger number of the recipients with the portal hypertension. Postoperative refractory ascites are reported to be associated with reduced 1-year survival and increased postoperative complications [10]. However, there is no evidence to explain the higher portal pressure at LT in AAC group, because the portal vein does not usually calcify. In general, high PVP has a strong relation with GWRW and liver stiffness [26,27]. There was no significant deference in GWRW between two groups. We speculated that the high AAC was because of liver stiffness. Although larger cohorts are necessary to verify our results, our results indicate that the AAC of the donor is one of the effective markers to evaluate graft quality before LDLT. This study revealed donor AAC (HR, 4.15; 95% CI, 1.93–8.97; $P = 0.0003$) strongly increased the risk of BC compared with other factors. It is important to understand the potential risks, not only in terms of the age of the donor, but also in terms of vascular calcification. Our results indicated that the peripheral arterial blood flow of the graft could be affected by arteriosclerosis of the donor.

Although this study presents the impact of donor AAC in LDLT, the major limitation of this study was the small sample size. Larger cohorts are necessary to investigate donor AAC after LDLT and clear the mechanisms involved.

In conclusion, donor AAC was associated with BC in our retrospective study. Higher donor AAC was an independent prognostic factor for BC among LDLT. Although further investigations are needed to verify our results, donor AAC could be a new tool to identify the risks of BC and to predict better outcomes following LDLT.

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Authors contributions

Y.I. and M.O. performed the investigation, analyzed the data, and wrote the manuscript. K.S.,S.K., H.T., K.I., T.K.,Y.T. and R.K. performed the investigation and analyzed the data. H.O. designed the study.

Conflict of interest

The authors have declared no conflicts of interest.

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