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The roles of superficial anastomoses in twin-twin transfusion syndrome

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ABSTRACT

Objective: We aimed to evaluate whether types of vascular anastomoses affect fetal demise (FD) in twin-twin transfusion syndrome (TTTS) after fetoscopic laser photocoagulation (FLP).

Methods: All TTTS patients who underwent FLP in our institution from 2005 to 2017 were included. We described vascular anastomoses during FLP as either arterio-arterial (AA), veno-venous (VV), or arterial-venous (AV), and abnormal fetal Doppler waveforms before FLP. We also analyzed risk factors for FD following FLP.

Results: In total, 184 TTTS placentas following FLP were analyzed (36 cases of donor-only FD, 12 cases of recipient-only FD, 3 cases of double FD, and 133 cases of both alive). AA anastomoses prevalence, absent or reverse end-diastolic velocity of umbilical artery (UAAREDV) of donor before FLP, gestational age at the time of FLP and operation time of FLP were independent risk factors for donor FD. VV anastomoses prevalence and UAAREDV of recipient before FLP were independent risk factors for recipient FD. VV anastomoses prevalence was higher in double FD cases than in FD of one twin or both alive ($p = 0.002$). AV anastomoses number and number of cases with more AV anastomoses from donor to recipient were not significantly different between FD and non-FD cases.

Discussion: AA anastomoses are associated FD of the donor following FLP and may protect the donor from hypovolemia before FLP. VV anastomoses are detected more frequently in FD of the recipient and double FD placentas following FLP and may rescue hypervolemia of the recipient or circulations of both fetuses before FLP.

1. Introduction

The roles of superficial anastomoses in twin-twin transfusion syndrome (TTTS) have been reported in previous studies. Arterio-arterial (AA) anastomoses may rescue a fetus during blood transfusion owing to a blood pressure imbalance between both twins when the AA anastomoses behave as functional arterial-venous (AV) anastomoses [1–3]. However, the roles of veno-venous (VV) anastomoses still remain unclear.

Theoretically, superficial anastomoses, such as AA and VV anastomoses, allow bi-directional blood flow via these arterial-venous (AV) branches and could act as functional AV anastomoses [4]. The prevalence of each type of anastomosis in placentas with TTTS has been reported in several studies. AA anastomoses were present in approximately 20–46% of patients with TTTS [4–6], while VV anastomoses were present in approximately 16–42% of patients. VV anastomoses have been reported to have a role in the development of TTTS in several studies [5–7]. However, those studies excluded cases with fetoscopic laser photocoagulation (FLP). Previous studies regarding TTTS cases with FLP reported that the prevalence of VV anastomoses is lower in TTTS than in non-TTTS cases [8].

The aim of this study was to evaluate whether vascular anastomoses, especially VV anastomoses, affect the rate of fetal demise (FD) following FLP and to consider what the roles of vascular anastomoses are in TTTS before FLP.

2. Methods

All consecutive TTTS patients who underwent FLP in our institution from 2005 to 2017 were included in this retrospective cohort study. We described vascular anastomoses as either AA, VV, or AV. We also described abnormal fetal Doppler waveforms before FLP, stage of TTTS, and whether there were growth restrictions of the twins before FLP. We also analyzed risk factors for FD following FLP.

Before FLP, we calculated estimated fetal weight with transabdominal ultrasound and assessed fetal body weight discordance using the formula: (estimated body weight of recipient – estimated body weight of donor)/estimated body weight of the recipient. There were no cases in which the estimated body weight of the donor was higher than that of the recipient. We described any abnormal fetal Doppler

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Table 1
Characteristics.

	at least one FD n = 51	both alive n = 133	p-value
Maternal Age (years)	30.6 ± 4.2	30.7 ± 5.4	0.153
Maternal Body height (cm)	157.6 ± 5.0	157.9 ± 4.9	0.695
Maternal Body weight (kg)	54.2 ± 7.1	54.4 ± 6.9	0.860
Nulliparity	31 (61%)	67 (50%)	0.367
Onset of TTTS (weeks)	19.4 ± 2.4	20.2 ± 2.6	0.034
GW at FLP (weeks)	19.8 ± 2.5	20.8 ± 2.4	0.017
Body weight discordance (> 25%) before FLP	38 (75%)	90 (68%)	0.695
GW at delivery (weeks)	32.4 ± 4.6	32.9 ± 5.5	0.523
Birth weight of ex-donor (g) n = 145	1749.5 ± 906.5	1464.2 ± 652.7	0.308
Birth weight of ex-recipient (g) n = 169	2124.2 ± 892.7	1809.2 ± 682.8	0.055
FLP-delivery (weeks)	11.6 ± 5.4	13.1 ± 6.3	0.114
Complete occlusion of anastomoses n = 152[a]	34/34 (100%)[a]	114/118 (97%)[a]	0.575

Data are presented as mean ± SD or number (%).
FD, fetal demise; TTTS, twin-twin transfusion syndrome; GW, gestational week; FLP, fetoscopic laser photocoagulation; FLP-delivery; time to delivery from FLP.
[a] The rate of complete occlusion cases was assessed by cases that could be evaluated for occlusions.

waveforms of the umbilical artery, umbilical vein, and ductus venosus before FLP.

We recorded the number and type of anastomoses as either AA, VV, or AV and also recorded the direction of the AV anastomoses during FLP. Following delivery, we checked for occlusions of the intertwin vascular anastomoses by injecting colored dye or air into the umbilical vessels except for the placentas that could not be evaluated for occlusions because of infarction after FD or destruction of the placenta. Incomplete occlusions of the intertwin vascular anastomoses were defined in the cases that there were any residual anastomoses by injecting colored dye or air.

Following FLP, all pairs of twins were assigned to the following groups: FD of the donor, FD of the recipient, FD of both fetuses, and both alive. The FD of the donor and FD of the recipient groups included FD of both fetuses. FLP in all cases were performed with both twins alive and all FD cases were identified after FLP. We instituted a control group for FD of donor (defined as the combination of cases where both were alive and cases where there was FD of recipient only), a control group for FD of recipient (defined as the combination of cases where both were alive and cases where there was FD of donor only), and a control group for FD of both (defined as the combination of cases where both were alive and cases where was FD of one twin).

The chi-square test was used to analyze categorical variables and the *t*-test or Mann-Whitney test were used to analyze continuous variables as required. Significance was defined as $p < 0.05$. We analyzed the risk factors for FD after FLP using multiple logistic analysis. Statistical analysis was performed using SPSS Statistics version 16.0J (SPSS Inc., Shibuya-Ku, Japan).

This study was approved by the institutional research review board and informed consent was sought and received from participating mothers.

3. Results

A total of 184 TTTS placentas were analyzed following FLP. There were 36 cases of FD of the donor only, 12 cases of FD of the recipient only, 3 cases of double FD, and 133 cases of both alive.

Characteristics are shown in Table 1. Comparisons were made between patients with at least one FD and where both fetuses survived. There was slightly difference in the onset of TTTS ($p = 0.034$) and gestational week of FLP ($p = 0.017$). There were no statistically significant differences in the other characteristics between groups. Thirty-two cases of occlusions of the intertwin vascular anastomoses could not be evaluated because of infarction after FD or destruction of the placenta. Thirty-four cases of at least one FD and 118 cases of non-FD could be evaluated occlusions of the intertwin vascular anastomoses. The total number of complete occlusions of anastomoses was found in 97% of cases (148/152). There were no significant difference in the percent of complete occlusion anastomoses between groups.

The presence of vascular anastomoses by stage of TTTS is shown in Table 2. The presence of AA anastomoses was significantly higher in TTTS stage III atypical than in any other stages. The presence of VV anastomoses was not significantly different between each of the stages. Sixty-four cases (35%) had a majority of AV anastomoses shunting from the donor to recipient compared to shunting in the opposite direction. There were no significant differences between the number of cases at other stages. The number of FD of donor cases was higher in stage III atypical than in any other stage.

The prevalence of AA anastomoses was higher in the FD of the donor group than in the control ($p < 0.001$) (Table 3). Multiple logistic analysis revealed that the prevalence of AA anastomoses (odds ratio [OR]: 3.9, 95% confidence interval [CI]: 1.69–9.06, $p = 0.001$),

Table 2
The presence of vascular anastomoses and FD according to stages of TTTS.

	I n = 31	II n = 39	III		IV n = 18	total n = 184	p-value
			c n = 66	a n = 30			
AA	13 (42%)	10 (26%)	14[a] (21%)	18[b] (60%)	4 (22%)	59 (32%)	0.002
VV	6 (19%)	4 (10%)	6 (9%)	5 (17%)	4 (22%)	25 (14%)	0.850
AA + VV	5 (16%)	2 (5%)	2 (3%)	3 (10%)	2 (11%)	14 (8%)	0.624
DR > RD	9 (29%)	15 (38%)	20 (30%)	11 (37%)	9 (50%)	64 (35%)	0.533
FD of donor	3 (10%)	7 (18%)	13 (20%)	13[c] (43%)	3 (17%)	39 (21%)	0.019
FD of recipient	4 (12%)	1 (3%)	4 (6%)	2 (7%)	4 (22%)	15 (8%)	0.077
FD of both	1 (3%)	1 (3%)	0 (0%)	1 (3%)	0 (0%)	3 (2%)	0.620

Data are presented as number (%).
c, stage III classical that donor has non-visible bladder and abnormal waveform Doppler; a, stage III atypical that donor has visible bladder and abnormal waveform Doppler.
FD, fetal demise; TTTS, twin-twin transfusion syndrome; AA, arterio-arterial anastomoses; VV, veno-venous anastomoses; AA + VV, cases that have both AA and VV; DR, AV anastomoses to recipient from donor; RD, AV anastomoses to donor from recipient; DR > RD, a majority of DR as compared to RD.
[a] adjusted residual = −2.4.
[b] adjusted residual = 3.6.
[c] adjusted residual = 3.2.

Table 3
The type of vascular anastomoses and abnormal fetal Doppler waveforms before FLP in FD of donor, FD of recipient and both FD.

	FD of donor n = 39	control (both alive and FD of recipient only) n = 145	p-value
AA n = 59	23 (59%)	36 (25%)	< 0.001
FLP-FD < 24 h	13		
24 h-7d	3		
7-14 d	1		
> 14 d	1		
VV n = 25	6 (15%)	19 (13%)	0.712
FLP-FD < 24 h	3		
24 h-7d	1		
7-14 d	1		
> 14 d	1		
AA + VV n = 14	4 (10%)	10 (7%)	0.500
The number of AV anastomoses from donor to recipient	3 (0–10)	3 (0–13)	0.705
from recipient to donor	4 (1–14)	4 (0–18)	0.362
DR > RD n = 64	15 (38%)	49 (34%)	0.587
Abnormal waveform Doppler of donor	25 (64%)	38 (26%)	< 0.001
UA AREDV	25 (64%)	35 (24%)	< 0.001
DV absent or reverse	4 (10%)	5 (3%)	0.097
UV pulsation	1 (3%)	5 (3%)	1.000
Abnormal waveform Doppler of recipient	12 (31%)	55 (38%)	0.409
UA AREDV	0 (0%)	11 (8%)	0.124
DV absent or reverse	7 (18%)	42 (29%)	0.167
UV pulsation	7 (18%)	38 (26%)	0.287
	FD of recipient n = 15	control (both alive and FD of donor only) n = 169	p-value
AA n = 59	6 (40%)	53 (31%)	0.566
FLP-FD < 24 h	2		
24 h-7d	2		
7-14 d	1		
> 14 d	1		
VV n = 25	6 (40%)	19 (11%)	0.007
FLP-FD < 24 h	0		
24 h-7d	4		
7-14 d	1		
> 14 d	1		
AA + VV n = 14	3 (20%)	11 (7%)	0.092
the number of AV anastomoses from donor to recipient	3 (1–11)	3 (0–13)	0.810
from recipient to donor	3 (0–10)	4 (0–18)	0.550
DR > RD n = 64	7 (47%)	57 (34%)	0.313
Abnormal waveform Doppler of donor	3 (20%)	60 (36%)	0.225
UA AREDV	3 (20%)	57 (34%)	0.392
DV absent or reverse	0 (0%)	9 (5%)	1.000
UV pulsation	0 (0%)	6 (4%)	1.000
Abnormal waveform Doppler of recipient	8 (53%)	59 (35%)	0.155
	FD of donor n = 39	control (both alive and FD of recipient only) n = 145	p-value
UA AREDV	3 (20%)	8 (5%)	0.049
DV absent or reverse	5 (33%)	44 (26%)	0.549
UV pulsation	5 (33%)	40 (24%)	0.530
	FD of both n = 3	control (both alive and FD of one twin) n = 181	p-value
AA n = 59	2 (67%)	57 (31%)	0.241
FLP-FD < 24 h	0		
24 h-7d	0		
7-14 d	0		
> 14 d	2		
VV n = 25	3 (100%)	22 (12%)	0.002
FLP-FD < 24 h	0		
24 h-7d	0		
7-14 d	1		
> 14 d	2		
AA + VV n = 14	2 (67%)	12 (7%)	0.016
the number of AV anastomoses from donor to recipient	1 (1–6)	3 (0–13)	0.270
from recipient to donor	2 (1–10)	4 (0–18)	0.629
DR > RD n = 64	1 (33%)	63 (35%)	1.000
Abnormal waveform Doppler of donor	1 (33%)	62 (34%)	1.000
UA AREDV	1 (33%)	59 (33%)	1.000
DV absent or reverse	0 (0%)	9 (5%)	1.000
UV pulsation	0 (0%)	6 (3%)	1.000
Abnormal waveform Doppler of recipient	0 (0%)	67 (37%)	0.555
UA AREDV	0 (0%)	11 (6%)	1.000
DV absent or reverse	0 (0%)	49 (27%)	0.566
UV pulsation	0 (0%)	45 (25%)	1.000

Data are presented as median (range) or number (%).
FLP, fetoscopic laser photocoagulation; FD, fetal demise; FLP-FD, time to fetal demise from FLP; AA, arterio-arterial anastomoses; VV, veno-venous anastomoses; AA + VV, cases that have both AA and VV; AV, arterio-venous anastomoses; DR, AV anastomoses to recipient from donor; RD, AV anastomoses to donor from recipient; DR > RD, a majority of DR as compared to RD; UA, umbilical artery; AREDV, absent or reverse end-diastolic velocity; DV, ductus venosus; UV, umbilical vein.

absent or reverse end-diastolic velocity of umbilical artery of the donor before FLP (OR: 4.6, 95% CI: 2.04–10.59, p < 0.001), gestational weeks at the time of FLP (OR: 0.8, 95% CI: 0.63–0.91, p = 0.004) and operation time of FLP (OR: 1.0, 95% CI: 1.00–1.04, p = 0.040) were independent risk factors for FD of the donor following FLP (Table 4). Earlier gestational weeks of FLP was an independent risk factor for FD of the donor. Operation time of FLP was not a large risk factor with an OR of 1.0.

VV anastomoses were present in 14% (25/184) of all cases (Table 2). There were present in 18% (9/51) of cases with at least one FD and in 12% (16/133) of non-FD cases. Also, the prevalence of VV anastomoses was higher in FD of the recipient and double FD cases than in the control (p = 0.007, 0.002) (Table 3). Multiple logistic analysis revealed that the prevalence of VV anastomoses (odds ratio [OR]: 6.1, 95% confidence interval [CI]: 1.85–20.02, p = 0.003) and absent or reverse end-diastolic velocity of umbilical artery of the recipient before FLP (OR: 6.5, 95% CI: 1.40–30.48, p = 0.017) were independent risk

Table 4
Multiple logistic analysis about FD of donor and FD of recipient.

FD of donor	OR	95%CI	p-value
Onset of TTTS	–	–	0.937
Gestational weeks of FLP	0.8	0.63–0.91	0.004
Operation time of FLP	1.0	1.00–1.04	0.040
The presence of AA anastomoses	3.9	1.69–9.06	0.001
UA AREDV of donor before FLP	4.6	2.04–10.59	< 0.001
FD of recipient	**OR**	**95%CI**	**p-value**
The presence of VV anastomoses	6.1	1.85–20.02	0.003
UA AREDV of recipient before FLP	6.5	1.40–30.48	0.017

FD, fetal demise; TTTS, twin-twin transfusion syndrome; FLP, fetoscopic laser photocoagulation; AA, arterio-arterial anastomoses; UA, umbilical artery; AREDV, absent or reverse end-diastolic velocity; VV, veno-venous anastomoses; OR, odds ratio; CI, confidence interval.

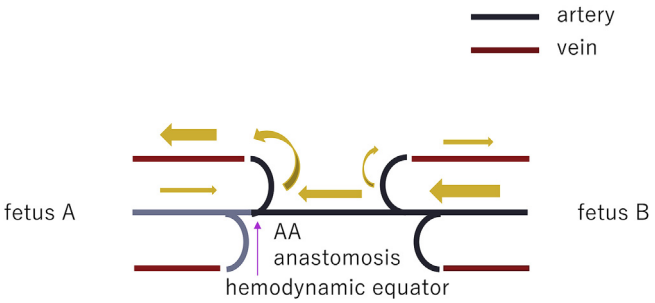

Fig. 1. When the arterial blood pressure of fetus B is higher than that of fetus A, and the hemodynamic equator of fetus B reaches an arterial branch of fetus A, AA anastomoses carry blood to fetus A from fetus B as a functional AV anastomoses. Partial blood volume flows back to fetus A.

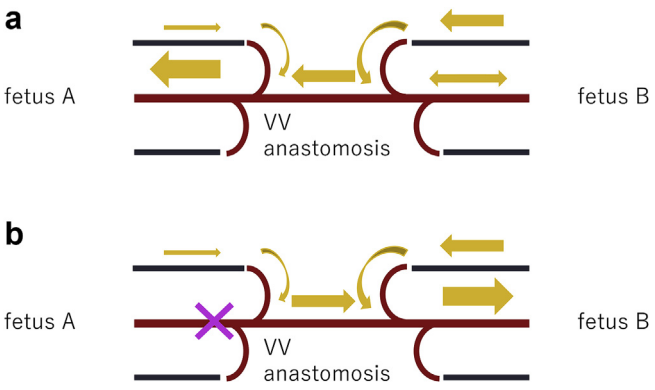

Fig. 2. a. VV anastomoses does not have hemodynamic equator. When the venous blood pressure of fetus B is higher than that of fetus A, VV anastomoses carry blood passively to fetus A from fetus B according to the pressure gradient of both venous blood pressures. Almost the entire blood volume in the VV anastomoses flows to fetus A.**b**. When the venous blood vessel of fetus A is compressed by an external impact (X), the VV anastomoses carries blood to fetus B. Almost the entire blood volume in the VV anastomoses flows to fetus B.

factors for FD of the recipient following FLP (Table 4).

The presence of AV anastomoses from either the donor to the recipient or from the recipient to the donor was not significantly different between FD and non-FD cases (Table 3).

4. Discussion

In TTTS cases, AA anastomoses are associated with FD of the donor following FLP and may play a protective role against hypovolemia or hypotension in the donor. VV anastomoses, however, can be associated with FD of the recipient and double FD placentas following FLP and may rescue the recipient from hypervolemia or hypertension, or may help maintain circulations in both fetuses in TTTS before FLP.

Previous studies have shown that AA anastomoses may serve as a protective factor against TTTS [1–3,9,10]. When AA anastomoses behave as functional AV anastomoses, it may rescue or reverse the transfusion of blood from one twin to the other due to an arterial blood pressure imbalance between both twins through AA anastomoses (Fig. 1) [4].

We found the presence of AA anastomoses was higher in the stage III atypical (critical abnormal fetal doppler in either twin with visible bladder of donor) subgroup of TTTS than in other stages [4]. Therefore, this finding shows that AA anastomoses potentially play an etiological role against the advancement of TTTS [4]. The incidence of AA anastomoses in stage III classical (critical abnormal fetal doppler in either twin without visible bladder of donor) and atypical subgroups of TTTS have been reported to be 13–18% and 48–73%, respectively [4,11]. In this study, the incidence of AA anastomoses was 21% in the stage III classical subgroup and 60% in the stage III atypical subgroup, which is similar to previous studies [4,11]. In TTTS, the donor may receive some blood from the recipient via AA anastomoses due to the arterial blood pressure difference because the arterial blood pressure of the donor may be theoretically lower than that of the recipient. It has been suggested that the bladder of donors may be visible despite hypovolemic circulation in the stage III atypical subgroup [4]. Furthermore, FD of donor could occur by the elimination of AA anastomoses by FLP and therefore rescue for circulation of the donor does not occur.

The roles of VV anastomoses, however, still remain unclear. Several studies have reported that the incidence of VV anastomoses in TTTS placentas (16–42%) is higher than that in non-TTTS placentas (7–25%) [5–7]. These findings suggested that VV anastomoses have a role in the development of TTTS [5–7]. However, these studies excluded TTTS cases with FLP. In contrast, another study reported that the number of VV anastomoses was 12% in TTTS in which FLP had been performed [8]. This may indicate that the presence of VV anastomoses is lower in severe TTTS cases that need FLP than in TTTS cases that do not require surgery. In this study, the prevalence of VV anastomoses was 14% in all TTTS cases with FLP, which is comparable to a previous study [8].

In previous studies, VV anastomoses carried unidirectional blood flow due to an inter-twin pressure gradient in venous circulation affected by an external impact, such as fetal position or velamentous umbilical cord insertion (Fig. 2b) [6,12]. Therefore, VV anastomoses were found to play a role in the development of TTTS [6]. In this study, the prevalence of VV anastomoses was significantly higher in the FD of the recipient and double FD groups compared to the control. This showed that VV anastomoses may rescue circulation of the recipient or both fetuses in TTTS before FLP, and that FD of the recipient or both fetuses occurs after ablation of the VV anastomoses. In TTTS, the venous blood pressure of the donor is theoretically lower than that of recipient and the blood of the recipient may be carried passively to the donor via VV anastomoses (Fig. 2a), rescuing the recipient from hypervolemia by reducing the venous return blood volume.

VV anastomoses could be involved in both the development and protection from TTTS. The role of VV anastomoses ultimately may be decided by the venous pressure gradient of both fetuses and external compression of the umbilical vein. VV anastomoses that are easily compressed may develop into TTTS and ultimately may lead to deterioration of TTTS by the same mechanism because the hypovolemic donor is compressed more easily than the hypervolemic recipient. However, if there is no external compression of the umbilical vein, the blood volume of umbilical vein in the recipient would be reduced owing to VV anastomoses, which may passively carry blood to the donor from the recipient according to the inter-twin venous pressure gradient.

Both AA and VV anastomoses may play different roles, although they behave similarly to AV anastomoses because they are superficial

anastomoses connecting each twins' blood vessels without an intervening cotyledonary capillary system. When AA anastomoses, affected by arterial blood pressure (afterload of the fetus), play the same role as AV anastomoses and carry blood to twin A from twin B, a portion of the blood volume may go back to twin B via the venous branch of the AA anastomoses (Fig. 1). Conversely, when VV anastomoses, affected by venous return (cardiac preload of the fetus), play the same role as AV anastomoses, almost all of the blood in VV anastomoses from the umbilical vein of twin B may flow to that of twin A (Fig. 2a). Therefore, VV anastomoses could carry more blood to the co-twin and rescue the blood volume of both fetuses, or especially in the hypervolemic recipient, more effectively than AA anastomoses.

A limitation of this study is that the accurate assessment of placental sharing discordance and umbilical cord insertion in all cases was impossible. Although they might affect FD after FLP, it is difficult to assess them of the demise twin because of infarction and maceration.

In conclusion, VV anastomoses are detected frequently in FD of recipient and double FD with TTTS placentas following FLP. They may rescue hypervolemia in the recipient or circulations of both fetuses before FLP. We need to evaluate additional cases and investigate the different role of each anastomoses in TTTS before and after development.

Conflicts of interest

There are no conflicts of interest to declare.

Declaration of interest

None.

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