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ORIGINAL PAPER

Intraoperative vasoplegic syndrome in patients with fulminant myocarditis on ventricular assist device placement

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Abstract

Purpose Fulminant myocarditis is uncommon, but life-threatening, and some patients need mechanical circulatory support. This study was performed to evaluate how different types of mechanical circulatory support—biventricular assist device (BiVAD) or left ventricular assist device (LVAD) placement—affect intraoperative hemodynamic status.

Methods From January 2013 to September 2016, the patients who underwent BiVAD or LVAD placement for fulminant myocarditis were analyzed. The mean arterial pressure (MAP), mean pulmonary arterial pressure, central venous pressure (CVP), vasoactive score, and inotropic score were recorded at five time points: after the induction of anesthesia; at weaning, 30 min after weaning, and 60 min after weaning from cardiopulmonary bypass (CPB); and at the end of surgery. The vasoactive and inotropic scores were calculated as follows: vasoactive score = norepinephrine (μg/kg/min) × 100 + milrinone (μg/kg/min) × 10 + olprinone (μg/kg/min) × 25: inotropic score = dopamine (μg/kg/min) × 1 + dobutamine (μg/kg/min) × 1 + epinephrine (μg/kg/min) × 100.

Results We enrolled 16 patients of fulminant myocarditis. Ten of them underwent BiVAD placement, and the other underwent LVAD placement. After weaning from CPB, the BiVAD group had a significantly lower MAP but no difference in CVP. The vasoactive score was significantly higher in the BiVAD group at weaning of CPB ($p = 0.015$), 30 min after weaning ($p = 0.004$), 60 min after weaning ($p = 0.005$), and at the end of surgery ($p < 0.016$).

Conclusion Patients with BiVAD placement required more vasoactive support to maintain optimal hemodynamic status compared with those with LVAD placement. This result indicates that BiVAD placement was more associated with vasoplegic syndrome.

Keywords Fulminant myocarditis · Ventricular assist device · Vasoplegic syndrome

Introduction

Fulminant myocarditis is a life-threatening syndrome characterized by rapidly progressive cardiogenic shock and high mortality without appropriate mechanical circulatory support [1, 2]. The rapid adoption of venoarterial extracorporeal membrane oxygenation (ECMO) has improved survival rates; however, mortality remains high, ranging from 28 to 42% [3–5]. In patients in whom hemodynamic stability cannot be achieved with veno-arterial ECMO alone, ventricular assist devices (VADs), which fully compensate for impaired cardiac function, can be used to support cardiocirculatory function. Early support with a VAD improves outcomes for these patients and results in lower mortality rates of 0–23% [6, 7]. We have also experienced increased numbers of successful VAD placements for them during the last several years.

Patients with fulminant myocarditis who undergo VAD placement have both ventricles' heart failure and progressing multi-organ failure; therefore, almost all patients require a bi-ventricular assist device (BiVAD) [8, 9]. However, we have encountered some patients with fulminant myocarditis who survived with a left VAD (LVAD) alone.

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There is little research to examine whether BiVAD placement can maintain better hemodynamic status than LVAD placement for patients with fulminant myocarditis. Therefore, we conducted this retrospective analysis to find how BiVAD placement affects their hemodynamic status and requires vasoactive inotropic support compared with LVAD placement alone.

Methods

Patient collection

This retrospective analysis involved the patients with fulminant myocarditis with cardiac shock undergoing VAD placement from 1 January 2013 to 31 September 2016. Fulminant myocarditis was diagnosed by both clinical symptoms and histological analysis.

We retrospectively collected the patients' demographic data, the duration between symptom onset and operation, preoperative use of an intra-aortic balloon pump, ECMO support, continuous hemodiafiltration, type of VAD, ventilation, and preoperative left ejection fraction as measured by transthoracic echocardiography.

Perioperative management

The procedure of either BiVAD or LVAD alone placement was decided preoperatively. Our cardiovascular surgeons selected one of these procedures on the basis of their experience because criteria for LVAD or BiVAD selection in patients with fulminant myocarditis had not yet been established. In the LVAD group, paracorporeal pulsatile-flow VAD (Nipro VAD; Nipro, Osaka, Japan) was used to support the left ventricle because implantable VADs are only allowed as a bridge to transplantation in Japan. None of the patients in this study were considered candidates for transplantation before the operation. The inflow cannula of the VAD was placed in the left ventricle and the outflow cannula was inserted into the ascending aorta. In the BiVAD group, the same type VAD was used for the left ventricle and a centrifugal pump with an oxygenator was added to support right ventricular function; the inflow cannula was placed in the right atrium and the outflow cannula was inserted into the trunk of the pulmonary artery.

In the operating room, the patients were monitored using five-lead electrocardiography, a radial artery line, pulse oximetry, capnography, and body thermometer. Their central venous pressure (CVP) and pulmonary arterial pressure (PAP) were also monitored using a central venous catheter and pulmonary artery catheters (Swan-Ganz CCO/VIP; Edwards Lifesciences LLC, Irvine, CA USA). Transesophageal echocardiography was used for all patients. Only rocuronium and fentanyl were used for the induction of anesthesia because they were already intubated and sedated by midazolam. Anesthesia was maintained by propofol at 5 mg/kg/h, remifentanil at 0.3–0.35 μg/kg/min, fentanyl at 10–20 μg/kg, and rocuronium at 0.3–0.5 mg/kg/h. With respect to perioperative inotrope management, we have neither institutional guidelines nor specified algorithms dictating inotropic support in our institution. The perioperative use and discontinuation of inotropes to optimize the cardiopulmonary status depended on the attending anesthesiologist, but both groups were managed in the same hemodynamic goals; mean arterial pressure (MAP) ranging between 60 and 80 mmHg and CVP less than 20 mmHg under adequate volume status evaluated by transesophageal echocardiography. Blood gas data such as lactate and hemoglobin were also evaluated and corrected to optimize the hemodynamic status. The patients were treated with following vasoactive agents and inotropes as perioperative therapy: dopamine, dobutamine, epinephrine, norepinephrine, milrinone, and olprinone.

We retrospectively collected hemodynamic data from the anesthesia records. MAP, mean PAP, CVP, vasoactive score, and inotropic score were collected at five time points: after induction of anesthesia, at weaning from cardiopulmonary bypass (CPB), 30 min after weaning from CPB, 60 min after weaning from CPB, and at the end of surgery.

Vasoactive and inotropic scores were calculated using the following formulas in accordance with previous studies [10–12]:

$$\begin{aligned}\text{Vasoactive score} = {} & \text{norepinephrine (μg/kg/min)} \times 100 + \text{milrinone (μg/kg/min)} \\ & \times 10 + \text{olprinone (μ g/kg/min)} \times 25.\end{aligned}$$

$$\begin{aligned}\text{Inotropic score} = {} & \text{dopamine (μg/kg/min)} \times 1 + \text{dobutamine (μg/kg/min)} \\ & \times 1 + \text{epinephrine (μg/kg/min)} \times 100.\end{aligned}$$

Statistical analysis

We compared the perioperative MAP, CVP, PAP, vasoactive score and inotropic score between patients who underwent BiVAD and LVAD placement. Categorical

variables were summarized as frequencies and percentages and continuous variables as medians and 25th and 75th percentiles. The Friedman test and Mann–Whitney *U* test were used for continuous variables, and Fisher's exact test was used for continuous variables and categorical variables. All statistical analyses were performed using R statistics (R.3.3.0) (Institute for Statistics and Mathematics, Wirtschaftsuniversität Wien).

Ethical standard

This study was reviewed and approved by the ethics committee of the National Cerebral and Cardiovascular Center (Suita, Osaka, Japan) on January 27, 2017 (N28-137). Written informed consent was obtained from all subjects.

Results

Patient characteristics

We enrolled 16 patients of fulminant myocarditis during the examined period. Ten of the 16 patients underwent BiVAD placement and the remaining six underwent LVAD placement alone. Before the onset of fulminant myocarditis, four patients had a history of hypertension, and two patients had paroxysmal atrial fibrillation (one patient had both). The other 11 patients were healthy and did not have any past medical history. No significant differences in the patients' clinical characteristics were noted between the BiVAD and LVAD groups (Table 1). All patients underwent intubation and insertion of an intra-aortic balloon pump, and 15 patients were supported by venoarterial ECMO. Half of the patients in each group required continuous hemodiafiltration, and the other patients also had acute kidney injury (mean serum creatinine, 1.68 mg/dl). Laboratory data also showed that all patients had liver failure as demonstrated by elevated aminotransferase and bilirubin concentrations, low platelet counts, and prolonged prothrombin times. We were unable to evaluate pulmonary function and pulmonary vascular resistance because most patients were undergoing ECMO upon admission to our institution and data from previous hospitals were lacking.

Perioperative data analysis

There was no conversion from LVAD alone to BiVAD placement during the operations; however, two patients in the LVAD group underwent tricuspid annuloplasty because severe tricuspid regurgitation was detected on the transesophageal echocardiogram at the end of CPB. Nitric oxide inhalation was used for all patients in the LVAD group and for eight of 10 (80%) in the BiVAD group. We

Table 1 Preoperative patient characteristics

	BiVAD group ($n=10$)	LVAD group ($n=6$)	p value
Sex (male)	8 (80.0)	3 (50.0)	0.210
Age (years)	43.8 ± 16.2	60.2 ± 12.6	0.054
BMI (kg/m^2)	18.6 ± 2.7	19.6 ± 3.9	0.216
Preoperative IABP support	10 (100)	6 (100)	1.000
Preoperative mechanical ventilation	10 (100)	6 (100)	1.000
Preoperative ECMO	10 (100)	5 (83.3)	
Preoperative CHDF	5 (50.0)	4 (66.7)	0.515
White blood cell count(/ml)	11,100 ± 4681	11,550 ± 3234	0.757
Hemoglobin (g/dl)	9.25 ± 1.50	10.00 ± 1.40	0.374
Platelet count ($\times 10^4$/ml)	7.80 ± 4.22	8.65 ± 2.63	0.641
AST (IU/l)	339.0 (205.2, 410.0)	106.0 (84.8, 3008.8)	0.157
ALT (IU/l)	132.5 (88.3, 625.3)	177.0 (99.3, 1139.0)	0.959
Total bilirubin (mg/dl)	5.60 (2.30, 7.85)	2.05 (1.12, 5.00)	0.404
Creatinine (mg/dl)	1.37 (0.95, 1.69)	1.33 (0.79, 2.86)	0.837
PT-INR	1.17 (1.15, 1.32)	1.27 (1.09, 2.48)	0.604
BNP (pg/ml)	603.5 (241.3, 834.3)	687.5 (444.3, 1325.3)	0.534
CKMB (ng/ml)	145.0 (101.0, 227.0)	16.00 (15.3, 27.3)	0.099

Data are presented as mean ± SD, or number (%), or mean (1st quartile–3rd quartile). Fisher's exact test or Chi-squared test was used for categorical variables

BiVAD biventricular assist device, *LVAD* left ventricular assist device, *BMI* body mass index, *IABP* intra-aortic balloon pump, *ECMO* extracorporeal membrane oxygenation, *CHDF* continuous hemodiafiltration, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase, *PT-INR* prothrombin time-international normalized ratio, *BNP* brain natriuretic peptides, *CKMB* creatine kinase-myocardial band

Table 2 Preoperative variables and intraoperative characteristics in both groups

	BiVAD group ($n=10$)	LVAD group ($n=6$)	p value
Anesthesia time (min)	368 (344, 388)	375 (336, 451)	0.718
Operation time (min)	290 (253, 300)	310 (272, 365)	0.293
CPB time (min)	101 (94, 108)	104 (79, 120)	0.679
Days from onset to operation	7 (5.5, 11.0)	13.5 (11.5, 23.0)	0.091
Days from VA-ECMO to operation	4.00 (2.00, 6.75)	1.50 (1.00, 4.25)	0.128
RBC transfusion (ml)	1540 (980, 1960)	2800 (2520, 3360)	0.024
FFP transfusion (ml)	1590 (1260, 2460)	2355 (2130, 3030)	0.334
Platelet transfusion (ml)	525 (425, 734)	575 (500, 650)	0.959
NO use	8 (80%)	6 (100%)	0.762

Data are presented as mean (1st quartile–3rd quartile), or number (%). Fisher's exact test or Chi-squared test was used for categorical variables

BiVAD biventricular assist device, *LVAD* left ventricular assist device, *CPB* cardiopulmonary bypass, *VA-ECMO* venoarterial extracorporeal membrane oxygenation, *RBC* red blood cells, *FFP* fresh frozen plasma, *NO* nitric oxide

were unable to ascertain why two patients were managed without nitric oxide inhalation. The anesthesia time and the operation time had no significant differences between BiVAD and LVAD placement (Table 2). There were also no significant differences in the transfusion volumes. We also collected the blood loss data; however, we did not document them because some records of blood loss in LVAD group were not reliable.

Intraoperative hemodynamic status, vasoactive score, and inotropic score

At the point of anesthetic induction, there was no difference in the MAP, CVP, mean PAP, vasoactive score, and inotropic score between the two groups (Fig. 1). In both groups, the MAP ranged from 60 to 80 mmHg (Fig. 1a) and the CVP ranged from 10 to 15 cmH_2O (Fig. 1b). The CVP did not change throughout the operation, and it had no significant difference between the BiVAD and LVAD groups; however, the MAP was higher in the LVAD than BiVAD group 60 min after CPB ($p=0.005$) and the mean PAP was lower 30 min after CPB ($p=0.011$). As shown in Fig. 2, vasoactive scores were higher in the BiVAD group at the end of CPB ($p=0.014$), 30 min after it ($p=0.043$), 60 min after it ($p=0.005$), and at the end of surgery ($p=0.016$) (Fig. 2a). Norepinephrine was the main agent accounting for this difference, whereas inotropic scores did not change during separation from CPB and did not differ significantly between the groups (Fig. 2b).

Discussion

In this study, we found that the patients with BiVAD placement were associated with lower MAP and requirement for more vasoactive support than the patients with LVAD placement alone. The CVP had no significant difference, and the mean PAP of BiVAD placement group was slightly higher than that of LVAD placement. This might be because the pulmonary blood flow and the pulmonary vascular resistance of BiVAD group were greater than those of LVAD group. Then, we mainly discuss the reason for the difference of vasoactive support between two groups.

We found that the amount of norepinephrine was responsible for this difference. Each attending anesthesiologist used norepinephrine as a vasopressor to maintain blood pressure and systemic vascular resistance within the normal range. This result indicates that the patients with BiVAD placement are more likely to cause vasodilation than the patients with LVAD placement. To the best of our knowledge, no previous studies have reported this phenomenon.

The hemodynamic statuses of patients in the BiVAD group were close to vasoplegic syndrome, which is characterized by abnormally low systemic vascular resistance with normal cardiac output and caused by septic shock, post-cardiopulmonary bypass, burns, or trauma [13]. A multiple logistic regression analysis identified risk factors for vasoplegic syndrome after cardiac surgery: temperature, duration of cardiopulmonary bypass, total cardioplegic volume infused, reduced left ventricular function, and preoperative treatment with angiotensin-converting enzyme inhibitors [14]; however, none of these factors differed significantly between the two groups in this study. A retrospective research also analyzed risk factors of vasoplegia following VAD implantation: INTERMACS profile, CVP, systolic blood pressure and intraoperative CPB time [15], but neither of them were different between the LVAD and the BiVAD group in our study. The result of our study implies that BiVAD placement is an independent risk factor of vasoplegic syndrome. Although the sample size of this study did not have enough statistical power for identifying the risk factor, we here suggest a possible explanation for the association

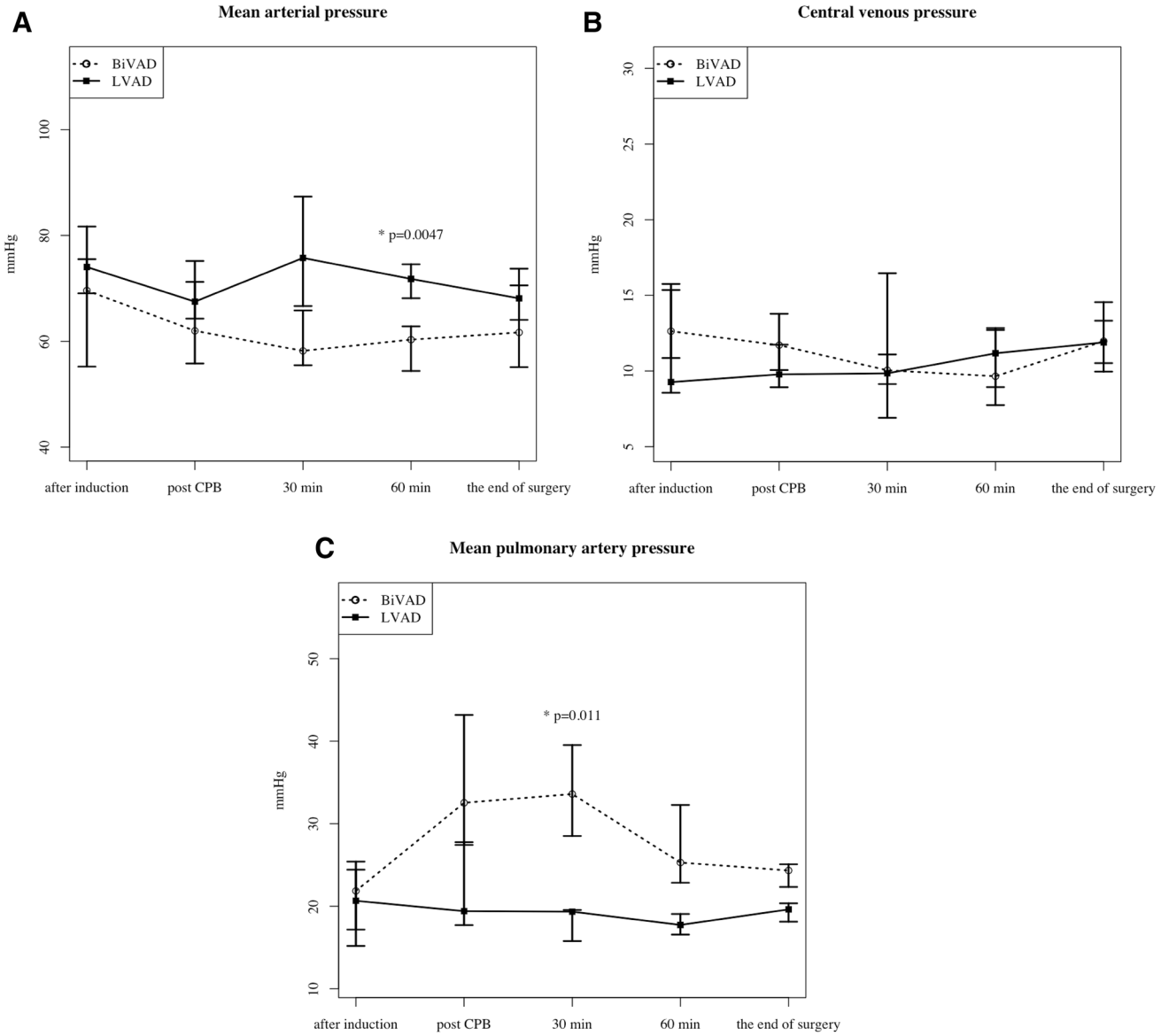

Fig. 1 Intraoperative hemodynamic status. Mean arterial pressure (**a**), mean central venous pressure (**b**), and mean pulmonary artery pressure (**c**) during the operation. *BiVAD* biventricular assist device, *LVAD* left ventricular assist device, *CPB* cardiopulmonary bypass

between vasoplegic syndrome and BiVAD placement on the basis of studying its pathophysiology.

The possible causality is that additional right VAD with membrane oxygenator in BiVAD group induces an inflammatory response and dilating peripheral vessels. Previous studies have shown that percutaneous cardiopulmonary support induces an inflammatory response due to endothelial cell activation [16, 17]. An inflammatory response increases NO production and Prostacyclin (PGI2) causing vasodilation and inhibits endothelin 1 acting as a vasoconstrictor, which results in vasoplegic syndrome. To prove this causality, we should have checked the inflammation marker during the operation.

Another possibility is that severe preoperative hypotension causes depletion of arginine vasopressin (AVP) in patients with a BiVAD. In addition to inflammation per se, arterial hypotension induced by low cardiac output also stimulates AVP release, resulting in its depletion [18], which in turn causes vasoplegic syndrome; however, we did not examine preoperative AVP concentrations in our patients, nor could we collect their preoperative hemodynamic status because they were transported from the other hospitals. Another investigation should be performed in the future to prove that the association between the treatment for acute fulminant myocarditis and vasoplegic syndrome.

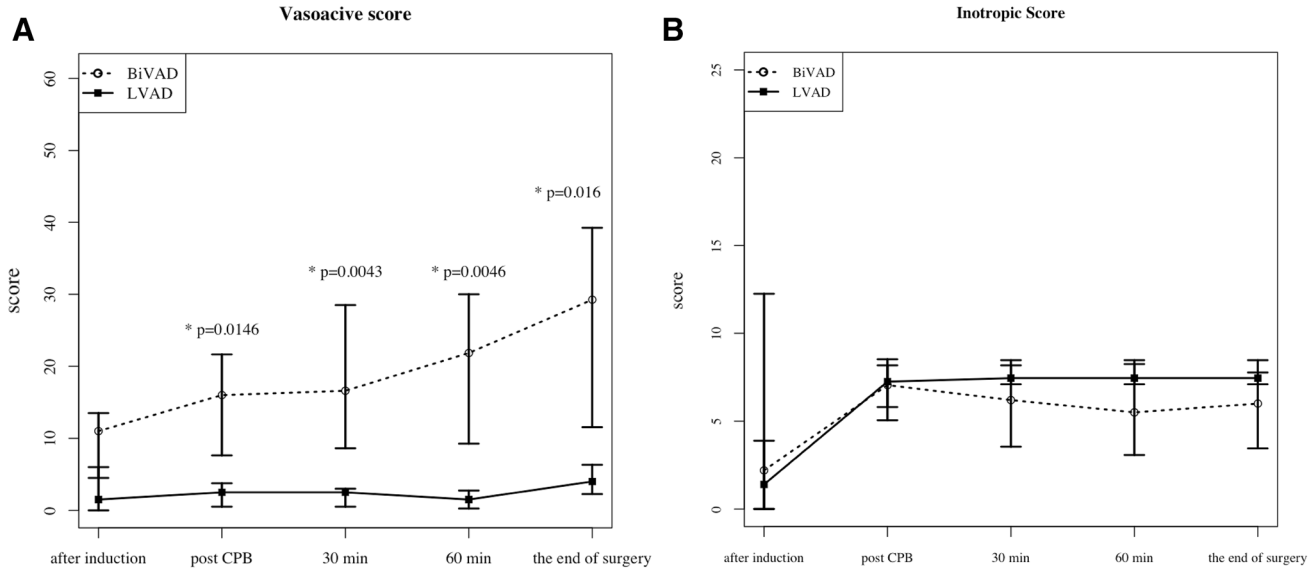

Fig. 2 Intraoperative vasoactive scores (**a**) and inotropic scores (**b**). Vasoactive scores = norepinephrine (μg/kg/min) × 100 + milrinone (μg/kg/min) × 10 + olprinone (μg/kg/min) × 25, inotropic scores = dopamine (μg/kg/min) × 1 + dobutamine (μg/kg/min) × 1 + epinephrine (μg/kg/min) × 100. *BiVAD* biventricular assist device, *LVAD* left ventricular assist device, *CPB* cardiopulmonary bypass

Vasopressin and methylene blue are well-known vasoactive agents that are used to treat vasoplegic syndrome that is resistant to other agents. However, none of our patients received vasopressin. Even though norepinephrine is also useful to individuals with vasoplegic syndrome, we should have considered administering vasopressin to some of our patients because its intraoperative usage has been shown to improve outcomes [19], and it could be added in the form of "vasopressin (units/kg/min) × 10,000" to vasoactive score as previously described [10].

From 2016, another mechanical circulatory support devices such as Impella (Abiomed, Danvers, MA, USA) have become available in Japan. Some case reports have documented the effectiveness of these devices in patients with fulminant myocarditis [20, 21]. Although these new devices are now available, our study is noteworthy in that we found that biventricular support may cause vasodilation.

Limitations

Our study has several limitations. First, we lacked data on pulmonary vascular resistance and right ventricular ejection fraction, which are essential for evaluating right ventricular function and determining whether LVAD placement alone is appropriate, because all study patients had been transported from other hospitals with IABP and venoarterial ECMO and we did not have access to their preoperative data. We were also unable to collect precise SvO_2 and cardiac output data despite placement of Swan–Ganz catheters. Second, this was a single-center retrospective study; therefore, we cannot generalize our results to all patients with fulminant myocarditis undergoing LVAD placement. The use of inotropes differs among various hospitals and centers, which makes difficult to make conclusions regarding the best usage of inotropic medicine. Third, our sample size was small and some records of blood loss were inaccurate. Because few patients for fulminant myocarditis need VAD support, a multicenter or international registry system is necessary.

Conclusion

We experienced 16 fulminant myocarditis patients undergoing VAD placement. Compared with LVAD placement alone, BiVAD placement was associated with a higher requirement for vasoactive support. This result indicates that BiVAD placement induced vasoplegic syndrome.

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