

Intra-Aortic Balloon Pump Reduces in-Hospital Mortality in Patients with Fulminant Myocarditis

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Abstract

A life support-based comprehensive treatment regimen, in which an intra-aortic balloon pump (IABP) unloads the left ventricle, has reduced the mortality of patients with fulminant myocarditis (FM). This study evaluated the efficacy of IABP in the treatment of patients with FM. A total of 100 patients diagnosed with FM were divided into two groups depending on the application of IABPs. 57 (57%) received IABPs within 30 min after admission (IABP group), whereas 43 (43%) did not (non-IABP group). In IABP group, 24.6% (14/57) died, compared to 34.9% (15/43) of those without IABPs ($P < 0.01$). The mean blood pressure (MBP), averaged heart rate (AHR), and left ventricular ejection fraction (LVEF) were similar between the two groups on admission. However, compared to the non-IABP group, MBP and LVEF were increased and AHR was significantly in IABP group. IABPs reduced the in-hospital mortality risk and improved the cardiac dysfunction in patients with FM.

Background

Fulminant myocarditis (FM) is a life-threatening disease that typically manifests with nonspecific flu-like symptoms and rapidly progresses to severe hemodynamic instability, lethal ventricular arrhythmia and death¹. FM has multiple causes but is commonly associated with viral infections and considered a sequela of viral infection^{2,3}. Due to non-specific clinical features in the early stages, correct and timely diagnosis of FM is often difficult, leading to misdiagnosis in a significant number of patients. Likewise, treatment of patients with FM is also challenging because of a lack of specific therapy⁴. Moreover, there is no reliable predictive risk factors to guide the prevention of FM.

Despite the recent therapeutic advances, including the use of inotropic agents and unloading of the ventricles, FM mortality of patients with FM remains high, particularly in those who suffer cardiac arrest^{5,6}. We have recently implemented an aggressive approach for treatment of FM, referred to as “Life Support-Based Comprehensive Treatment Regimen”, which includes the use of mechanical life support (positive pressure respiration and IABP with or without extracorporeal membrane oxygenation [ECMO]), immunomodulation therapy using both glucocorticoids and immunoglobulins, as well as administration of neuraminidase inhibitors⁷⁻⁹. Utilizing this approach, we reported a marked reduction of in-hospital mortality of FM from 48.2–3.7% compared to conventional therapy. The new approach emphasized unloading of the left ventricle (LV), which is the prevailing strategy for treatment of FM with hemodynamic instability (hypotension and shock)^{10,11}. A commonly-used mechanical circulatory support device decreases LV afterload using intra-aortic balloon pumps (IABPs). However, data on the beneficial effects of IABPs in reducing in-hospital and 30-day mortality are mostly in patients with acute myocardial infarction and not compelling^{12,13}. Likewise, while IABP is generally used in patients with FM and hemodynamic instability, its clinical impact remains unclear. Therefore, the purpose of this study was to determine the impact of using IABPs on in-hospital mortality and cardiac function in patients with FM.

Methods

This study was funded by the Nature Science Foundation of China. The executive committee designed the study and analyzed the data. All protocols and methods were approved by the ethics committees of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology (HUST) and conducted in accordance with the principles of the Declaration of Helsinki and the International Conference on Harmonization Guidelines for Good Clinical Practice. Written informed consent was obtained from the participants or their relatives, as appropriate, before enrolment of patients in the study.

Enrolment of patients with FM

We enrolled 100 patients between the ages 18 to 76 years, who were admitted Tongji Hospital, from September 2008 to January 2019. Baseline characteristics of patients are presented in Table 1. Past medical history, laboratory tests, special tests, diagnosis, and treatment were extracted from medical records by two independent investigators.

Table 1. Baseline characteristics of the enrolled patients

Variables	Non-IABP (N=49)	IABP (N=57)	P-value
Male	25(51.0%)	26(41.9%)	0.443
Age	36.1±14.5	38.42±14.9	0.403
SBP	85.0±3.5	83.8±3.1	0.741
DBP	51.2±2.2	53.3±7.8	0.721
MBP	63.9±2.0	62.7±7.4	0.471
Heart rate	105.7±5.9	102.7±3.9	0.134
Temperature	37.1±1.3	36.7±1.0	0.086
Characteristics of ECG			0.217
Conduction block	16 (32.7%)	19 (33.3%)	
VT/VF	10 (20.4%)	7 (12.3%)	
Others	20 (40.8%)	36 (63.2%)	
cTnl	19381.0	20431.0	0.248
BNP	9468.7	9928.5	0.549
LV (mm)	47.9±7.5	47.8±6.5	0.423
EF (%)	40.9±14.5	39.9±12.8	0.378
Scr (umol/L)	165.5±23.7	155.7±20.8	0.195
PMH			
Hypertension	7	6	
CHD	3	3	
AF	2	1	

SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, diastolic blood pressure; HR, heart rate; BT, body temperature; VT, ventricular tachycardia; VF, ; ventricular fibrillation; CTnl, Cardiac troponin I; BNP, B-type natriuretic peptide; LV, left ventricle; EF, ejection fraction; Scr, serum creatine; PMH, past medical history.; CHD, coronary heart disease; AF, atrial fibrillation.

All patients were clinically diagnosed with FM according to the diagnostic criteria described previously ⁸. All patients met the following key diagnostic criteria: (1) Acute onset of cardiac symptoms such as dyspnea, palpitations, chest pain, and/or syncope after a recent history of viral infection; (2) Clinical signs of cardiogenic shock (e.g., systolic blood pressure \leq 90 mmHg or mean arterial pressure $<$ 70 mmHg or systolic blood pressure decrease $>$ 40 mmHg from the baseline healthy state) or hypotension associated

with signs of impaired sufficient organic perfusion (cyanosis, cold extremities, oliguria, and/or changes in mental status); (3) Striking increase in serum concentrations of myocardial injury and dysfunction biomarkers, such as troponin I and B-type natriuretic peptide or N-terminal pro-B-type natriuretic peptide (NT-pro-BNP); (4) Imaging examinations showing notable diffused left ventricular hypokinesia and markedly decreased left ventricular ejection fractions (LVEF < 45%); (5) coronary angiographic examinations showing normal or mild stenosis of the coronary artery. The exclusion criteria included (1) Patients with high suspicion of acute coronary syndrome (ACS), in whom the diagnosis of ACS could not be confirmed because of contra-indication to coronary angiography; (2) sepsis-associated myocardial injury and (3) myocardial injury induced by chemotherapeutical agents, poisons, hemodynamic instability or shock caused by hypovolemia.

IABP indications and procedure

The indications for IABP implantation included cardiogenic shock complicated by multiple organ dysfunction, cardiogenic shock associated with ventricular arrhythmias, or cardiac arrest. Patients with FM in the non-IABP group received vasopressors and inotropic agents such as catecholamines (norepinephrine, dopamine, and dobutamine) adjusted to maintain a mean arterial blood pressure of 60 mmHg. The IABP was applied through cannulation of the left or right femoral artery. Heparin was continuously administered during the IABP procedure to avoid cannular occlusion by intracavitary thrombus.

Pharmacological therapy

The dosage and duration of each medication were recorded in patients of both groups. In brief, norepinephrine and/or dopamine were used to increase the blood pressure. Immunoglobins and corticosteroids were used to modulate the immune response. Neuraminidase inhibitors, including oseltamivir, were used to reduce viral load virus-induced myocardial attack and subsequent myocardial injuries. Other medications including antibiotics and supportive agents were also administered. The vasoactive and inotropic scores were calculated using the following formulae as described previously to quantitatively evaluate the dosages of each patient ¹⁴: Vasoactive score = norepinephrine ($\mu\text{g}/\text{kg}/\text{min}$) \times 100 + milrinone ($\mu\text{g}/\text{kg}/\text{min}$) \times 10 + olprinone ($\mu\text{g}/\text{kg}/\text{min}$) \times 25, and inotropic score = dopamine ($\mu\text{g}/\text{kg}/\text{min}$) \times 1 + dobutamine ($\mu\text{g}/\text{kg}/\text{min}$) \times 1 + epinephrine ($\mu\text{g}/\text{kg}/\text{min}$) \times 100.

Statistical analysis

Continuous variables are expressed as the means \pm SD. Comparisons between 2 groups were tested by an unpaired t test or Mann-Whitney U test. Categorical variables were reported as the number and percentage in each group and were analyzed with the Fisher exact test. $P < 0.05$ was considered statistically significant.

Results

Baseline Characteristics of Patients with FM

A total of 100 patients, comprised of 51 male and 49 female patients with FM were enrolled in the study. Fifty-seven patients met the criteria, described above, and underwent IABP implantation. Two patients (3.5%) had a history of coronary heart disease (CHD), 11 patients (19.3%) had a history of hypertension, and 2 patients (3.5%) had paroxysmal atrial fibrillation (one patient had atrial fibrillation and hypertension). Among the patients who did not receive IABPs, 7 had a history of hypertension (16.3%) and 3 had a history of CHD (7%). The medications of the patients in two groups were similar. The remaining patients were healthy and did not have any past medical history. No significant differences in patients' clinical characteristics were noted between those in the IABP and non-IABP groups (Table 1). All patients in the IABP group underwent intubation and insertion of an IABP with a suitable size of balloon according to their body height. Thirty-seven patients (64.9%) in the IABP group and 26 (60.5%) in the non-IABP group received hemofiltration; the other patients also had acute kidney injury.

Effects of IABPs on in-Hospital Mortality and Length of Hospital Stay

Patients with FM who did not undergo IABPs implantation had a significantly higher crude in-hospital mortality rate than those in the IABP group (Fig. 1A). Among the 57 patients who received IABPs, 24.6% (14/57) died, compared to 34.9% (15/43) in the non-IABP group ($P < 0.01$). Cox regression analysis and complementary Kaplan-Meier curves indicated a 29.5% reduction in in-hospital mortality risk of patients with FM who were treated with IABP. The most common cause of death was multi-organ dysfunction in both IABP ($n = 10$) and non-IABP ($n = 12$) patients. Patients in the IABP group had a shorter hospital stay by 2 days as compared to those in the non-IABP group (17.4 vs. 15.4 days, $P < 0.05$) (Fig. 1B), indicating that IABP implantation promoted recovery of patients with FM and shortened their hospital stays.

Effects of IABPs on Hemodynamic Parameters

Before IABP implantation, there was no difference in the systolic blood pressure (SBP), diastolic blood pressure (DBP), mean blood pressure (MBP), vasoactive score, and inotropic score between the two groups (Table 1 and Fig. 2). Baseline MBP of the non-IABP and IABP groups was 63.9 ± 2.0 and 62.8 ± 1.4 mmHg, respectively (Table 1). All patients were in cardiogenic shock. However, 30 min after IABP implantation, both SBP and DBP increased dramatically compared to their baseline values as well as compared to the corresponding values in patients in the non-IABP group IABPs (Fig. 2A, B and C). The average systolic, diastolic and mean blood pressures were quickly increased after 30 min of IABP by 11.1, 8.3 and 8.7 mmHg, respectively. Although baseline HRs in both groups were similar, it was significantly lower 30 min after IABP implantation, compared to a slight reduction in the non- IABP group (Fig. 2D).

The vasoactive scores were similar in both groups on admission but were much lower at 30 min, 6 h, and 24h and 3days after IABP implantation (Fig. 2E and F). Norepinephrine and dopamine were the main agents accounting for this difference between the groups. IABP implantation significantly reduced the dose of vasoactive drugs needed for the maintenance of BP.

IABPs Attenuated Heart Failure in Patients with FM

Twenty-four hours after the application of IABP, the EF of patients was increased markedly compared to the baseline levels (Fig. 3A). The baseline serum cardiac troponin I (cTnI) and NT-pro-BNP levels of both groups were strikingly elevated (Table 1). There were no significant differences between the two groups at the baseline (Table 1). However, cTnI and NT-pro-BNP levels were reduced significantly 3 days after treatment in both groups (Fig. 3B and C). The reduction of cTnI and NT-pro-BNP was similar between the two groups, indicating that IABP implantation had no effect on the reduction of myocardial biomarkers.

Discussion

Our data demonstrated that early application of IABP in the treatment of FM restores hemodynamic stability via unloading of the left ventricle, improves left ventricular function, shortens hospital stay, and improves survival. The findings advocate for early application of IABP in treatment of patients with FM and hemodynamic instability and/or arrhythmias.

The presentation of FM varies from cardiogenic shock to sudden death, while the complications include fatal arrhythmias, atrioventricular (AV) blocks, congestive heart failure, cardiac tamponade, acute renal failure, respiratory dysfunction, and multisystem organ failure. The diagnosis of FM is commonly based on comprehensive considerations of patients' medical history, clinical manifestations, and accessory examinations^{15, 16}. Patients who presented with a new onset of acute and severe heart failure, typically following a bout of viral infection, requiring parenteral inotropic or mechanical circulatory support are diagnosed as FM⁵. The most common cause of FM is considered to be enteroviral infection, such as coxsackievirus. New causes of FM, including H1N1 influenza and drug-related eosinophilic myocarditis^{17, 18} have emerged as the prevalence of enteroviral infection has waned.

The risk of in-hospital death was reduced by 29.5% in patients who received IABPs as compared to those who did not. In addition, the use of IABP significantly decreased the dose of vasoactive drugs, including norepinephrine and dopamine. The latter drugs may be detrimental in patients with FM^{10, 11}. Overall, the consensus is to minimize the use of vasoactive drugs in the treatment of FM⁸. Despite the lower doses of norepinephrine and dopamine in patients with FM, hemodynamic indices were more stable in patients with IABP than those without, indicating a beneficial effect of IABP in restoration of hemodynamic in FM patients.

Cardiac function improved after 24 hours of IABP application, as evidenced by a notable increase in echocardiographic LVEF from 37.5–45.4%, indicating that mechanical life support unloaded and enhanced the working efficiency of the heart. Although the LVEF was similar in both groups before discharge, application of IABP was associated with a higher and an earlier improvement in LVEF, an important determinant of survival in patients with FM¹⁹. The use of IABPs had no significant effect on cTnI and BNP levels in FM patients.

FM is an excessive inflammatory response process in the heart mostly due to a viral infection, which causes acute-onset severe heart failure^{20, 21}. Although viral infection is the cause of most cases, over-

activated immune responses and inflammatory cascades are the key pathogenic mechanisms for the initiation and development of FM^{9,22–24}. Unlike the irreversible death of the myocardium that occurs in ischemic heart disease, inflammatory response-induced myocardial injury is in part a reversible pathological process⁹. Thus, the pathophysiology of fulminant myocarditis involves direct myocardial injury caused by viral pathogens and indirect injury caused by the overwhelming immunologic responses and acute inflammatory shock⁹. Therefore, temporary mechanical circulatory support to unload the heart could lead to a better outcome as the inflammation gradually subsides.

In summary, our data suggest that early IABP implantation reduced in-hospital mortality in patients with FM. It also improved hemodynamic instability and attenuated cardiac dysfunction. Our data suggest that patients with FM should receive IABP support as early as possible, unless it is contraindicated.

Abbreviations

ACS

acute coronary syndrome; AHR:averaged heart rate; AV:atrioventricular; CHD:coronary heart disease; cTnl:cardiac troponin I; DBP:diastolic blood pressure; ECMO:extracorporeal membrane oxygenation; FM:fulminant myocarditis; IABP:intra-aortic balloon pump; NT-pro-BNP:N-terminal pro-B-type natriuretic peptide; MBP:mean blood pressure; LV:left ventricle; LVEF:left ventricular ejection fraction; SBP:systolic blood pressure.

Declarations

Ethics approval and consent to participate

The study protocol was approved by the ethics committees of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology (HUST) and conducted in accordance with the principles of the Declaration of Helsinki and the International Conference on Harmonization Guidelines for Good Clinical Practice.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Conflicts of interest

None declared.

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Authors' contributions

NZ, HS, WH, CL, DW, XW, JJ, DWW participated in the conception, drafting, and revision of the manuscript. All authors have read and approved the final manuscript.

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Figures

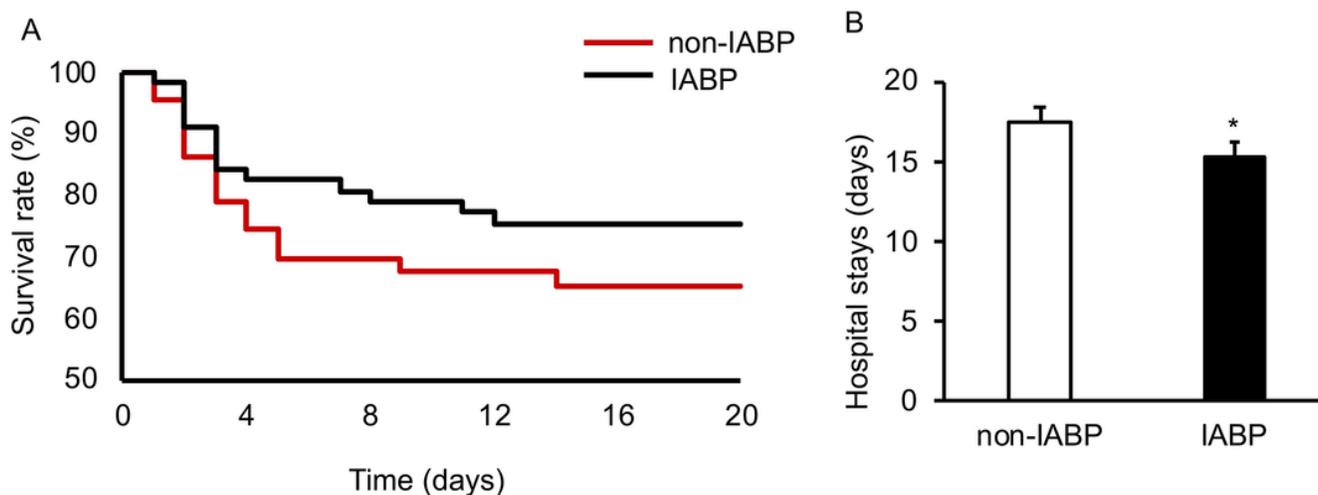


Figure 1

A, Kaplan-Meier curves of in-hospital mortality risk of patients with FM. B, The hospital stays of FM patients. *P < 0.05 compared to non-IABP group.

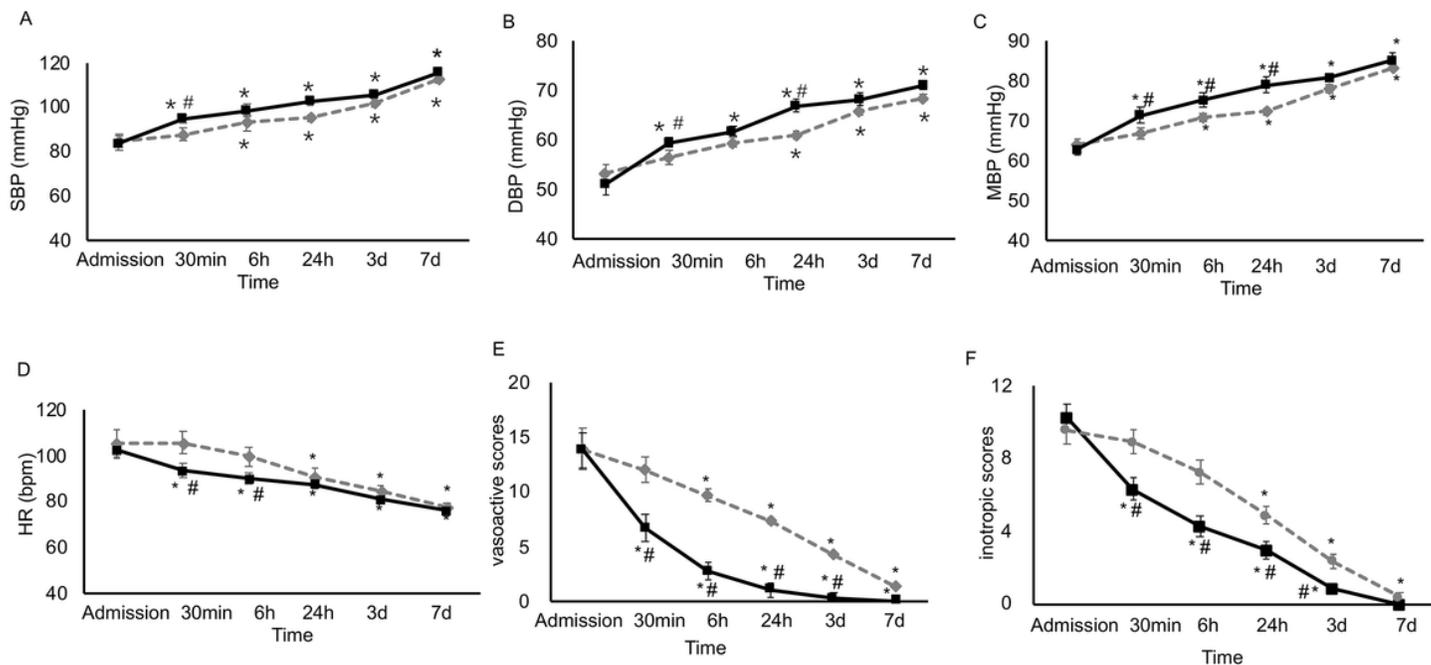


Figure 2

Changes in blood pressure, vasoactive score and inotropic score in each group of the patients after admission. SBP, systolic blood pressure. DBP, diastolic blood pressure, MBP, mean blood pressure. HR, heart rate. * $P < 0.05$ compared to baseline. # $P < 0.05$ compared to non-IABP group. Vasoactive score = norepinephrine ($\mu\text{g}/\text{kg}/\text{min}$) \times 100 + milrinone ($\mu\text{g}/\text{kg}/\text{min}$) \times 10 + olprinone ($\mu\text{g}/\text{kg}/\text{min}$) \times 25, and inotropic score = dopamine ($\mu\text{g}/\text{kg}/\text{min}$) \times 1 + dobutamine ($\mu\text{g}/\text{kg}/\text{min}$) \times 1 + epinephrine ($\mu\text{g}/\text{kg}/\text{min}$) \times 100.

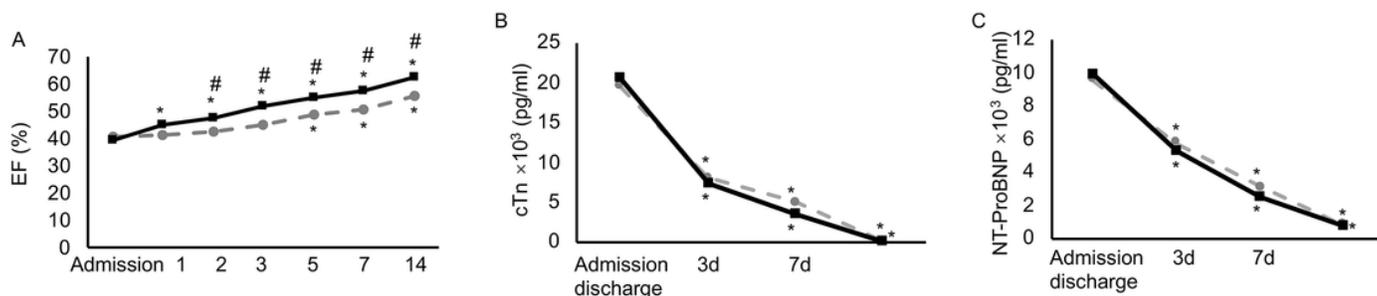


Figure 3

Changes of ejection fraction (EF), serum cardiac troponin I (cTnI) and NT-pro-BNP of patients in each group. *P <0.05 compared to baseline. #p<0.05 compared to non-IABP group.

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