Clinics of Surgery

Case Report ISSN: 2638-1451 | Volume 10

A Patient Complained with Severe Faint Attacks after the Successful Heart-Transplantation

Bingxu C[#], Jie S[#], Qinjing G, Ming J, Yuangang Q, Zhenyu J and Jie C*

Department of Cardiology, The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine), Youdian Road No. 54,Hangzhou, Zhejiang Province, China; Key Laboratory of Integrative Chinese and Western Medicine for the Diagnosis and Treatment of Circulatory Diseases of Zhejiang Province, Hangzhou, Zhejiang Province, China

[#]Chen Bingxu and Shi Jie are Co-authors

***Corresponding author:**

Chen Jie,
Department of Cardiology, The First Affiliated Hospital of Zhejiang Chinese Medical University (Zhejiang Provincial Hospital of Chinese Medicine), Youdian Road No. 54,Hangzhou, Zhejiang Province, China; Key Laboratory of Integrative Chinese and Western Medicine for the Diagnosis and Treatment of Circulatory Diseases of Zhejiang Province, Hangzhou, Zhejiang Province, China

Received: 05 Oct 2023
Accepted: 06 Nov 2023
Published: 14 Nov 2023
J Short Name: COS

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Citation:

Jie C. A Patient Complained with Severe Faint Attacks after the Successful Heart-Transplantation. Clin Surg. 2023; 10(5): 1-4

Keywords:

Heart transplantation; Cardiac rehabilitation; Severe hypotension; External counterpulsation

1. Case Report

A 71-year-old patient came to our clinic for several episodes of faint attacks, he had a successful heart transplantation operation 3 months ago for end-stage dilated cardiomyopathy and followed the regular medicine plan for immunology rejection reaction. We checked his vital sign immediately and found his blood pressure was just 79/35mmhg, and he was admitted for the further treatment.

His admission vital sign showed that BP 70/37mmhg, HR93bpm, RR 19/minute, T 36.2 Celsus. we also did the laboratory test for him routinely, and the result showed his white blood count was 11.11*10^9/L, hemoglobin was 104g/L, C reactive protein was 3.74 mg/L(0-8), BNP268ng/L(0-100), TNI0.055ug/L(0-0.026), Crwas218umol/L(29-104), Na was 137.7mmol/L(137-147), K was 3.28mmol/L(3.5-5.3), Tacrolimus concentration was 1.5ug/L, ESR was 2 mm/h(0-15) ALT was 36 U/L(15-40) AST was 84U/L(9-50).ECG was nonspecific, ecocardiography revealed that ejection fraction was 56%, and other laboratory test was normal.

We prescribed the Dopamine, Norepinephrine and Lactated Ringer's Solution for him to elevate the blood pressure immediately. Although it worked, his blood pressure was unstable and it would drop sharply when we tried to slow down the DA and NE velocity slightly. We get into the trouble that we can't withdraw any vasopressor at all and he might be in hospital for a long time. Then, we tried to perform the External Counterpulsation under the protection of vasopressor daily, and his blood pressure could maintain between the 95-120/65-85 mmhg and reported no discomfort during the process. After 3 days EECp treatment, we decided to discontinue the vasopressor tentatively under the ECG monitoring, fortunately, his blood pressure was stabilize between 95-110/60-80 mmhg.after that, we continued the EECp for him regularly until he finally discharged within the normal BP range (Figure 1-3).

Figure 1: Laboratory test result at admission

Laboratory test at admission	value	Normal range
WBC	12.6	3.5-9.5^9/L
Neutrophils percent	77.2	40-75%
hemoglobin	119	130-175g/L
C-reactive protein	9.7	0-8mg/L
procalcitonin	0.176	0-0.046ug/L
creatinine	218	59-104umol/L
glomerular filtration rate	25.48	
alanine transaminase	84	15-40U/L
Aspartate Transaminase	36	9-50U/L
sodium	137.7	137-147mmol/L
potassium	3.28	3.5-5.3mmol/L
chlorine	102.3	99-110mmol/L
D-dimer	5.24	0-0.55mg/L
BNP	268.6	268.6ng/L
TroponinI	0.055	0-0.026ng/L
Tacrolimus concentration	1.5	ug/L

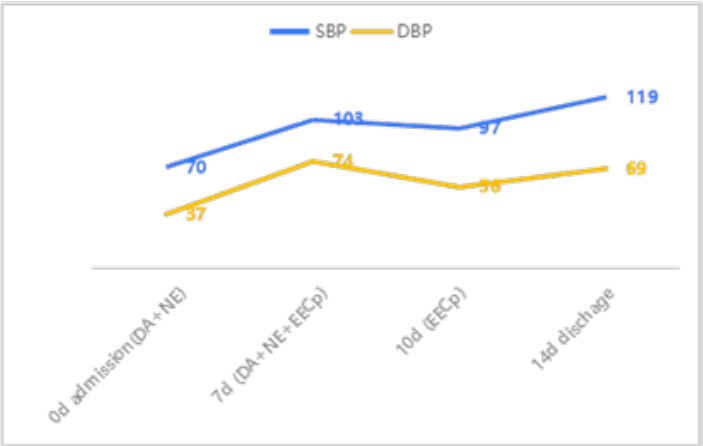

Figure 2: his blood pressure variation during admission

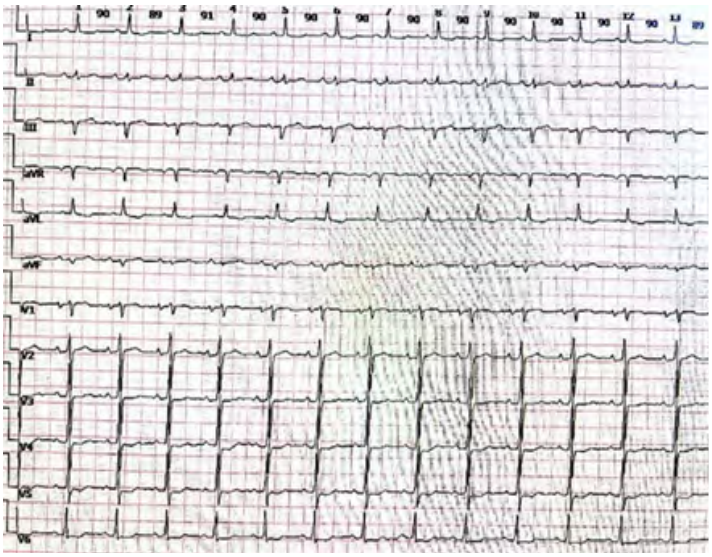

Figure 3: his ECG at admission

2. Discussion

End stage heart failure has been the challenge problem worldwide and it could be 25% by 2030 [1] Heart transplantation(HTx) has been gold standard to treat end-stage heart failure and has stabilized 5500 procedure annually recent years 1, and it has greatly improved the survival and life quality for these patients who were historically bedridden with the poor life expectancy together with the widespread use of automatic implantable cardioverter defibrillators, ventricular assist devices (VAD), and improved care of end-stage heart failure [2, 3].

However, heart transplantation complication has been the troublesome problem for their following rehabilitation even after the successful operation.one of them is hypertension which has multiple underlying reasons, like ventricle dysfunction, pronounced inflammatory responses or vasoplegic syndrome. some study showed that ventricle dysfunction incidence which was mainly caused by the primary graft dysfunction was up to 60.6% high [4-8], it could be diagnosed by the echocardiography that often showed the limited systolic capability with the ejection fraction < 45% and also accompany with the ascending BNP9. as for this patient, his EF was 78.6% and his BNP was 268 ng/L just slightly higher than normal at admission. henceforth, we exclude the primary graft dysfunction as the main diagnose for him. The other possible reason was profound inflammatory reaction after operation, especially for those patients who have not received the adequate induction [9, 10], but our patient has received the induction and maintenance properly and take the tacrolimus and mecophenolate regularly with the appropriate monitoring drug concentration. Another likely cause was vasoplegic syndrome that was common after transplantation which its pathophysiology was still unclear. Vasoplegic syndrome is a form of vasodilatory shock and is characterized by severe arterial hypotension (MAP<70 mmHg), typically despite preserved cardiac output, that is usually refractory to high doses of vasopressors, and some studies showed there was 5-25% incidence after cardiopulmonary bypass [10-12]. Batchelor et al revealed the prevalence and outcomes of vasoplegic syndrome after heart transplantation in a retrospective review of a 135 patients cohort study at their hospital between 2015-2020 was 66% [13]. and it also accompany with high incidence of mortality in hospital which nearly approximate to the 25% according to Byrne et al investigation [14]. henceforth, we diagnosed him with the vasoplegic syndrome according to his reliance to the high concentration vasopressor in the early hospital stage.its mechanism remained poorly understand, Alfirevic A et al thought it linked to the activation of inflammatory cytokines as the systemic response to the reperfusion and heart failure statue [15]. Therefore, several institutes tried the methylene blue to correct the dysregulation of nitric oxide synthesis and vascular smooth muscle guanylate cyclase activation that was caused by inflammatory cytokines [16, 17], however, their treatment effect was controversial and it's still the grand challenge for clinical practice. Back to our patient, we performed the vasopressor at first to stabilize his blood pressure, but we got into the dilemma that we can't withdraw any vasopressor because of his brittle blood pressure maintenance. His blood pressure dropped sharply as soon as we slowed down the injection velocity not mention to stop it. We also tried the stretch stock and concentrated saline solution, but it didn't work. Considering that EECp could improve the endothelial function, we decided to try it for him tentatively. At first, we performed EECp under the protection of vasopressor, and his blood pressure was slightly elevated during the procedure, thus we attempted slowed down the vasopressor velocity gradually. On the third day we decided to discontinue the vasopressor transiently for a while, fortunately his blood pressure maintained 90-95/ 60-70mmhg without any vasopressor. After we did the regularly EECp and monitored his blood pressure daily the next 7days, he successfully discharged with a stable blood pressure.

Enhanced external counterpulsation is a noninvasive technique designed to increase myocardial perfusion and reduce cardiac workload in patients with coronary artery disease in the first place, and some study showed it could improve the vascular endothelial

function by reducing the vascular stiffness [18-19]. Picard F et al reveal that it could treat the hypertension besides anti-hypertensive medicine. Braith et al showed that EECp could activate the NO production form endothelial tissue to lower blood pressure [20]. however, our patients showed another possibility that it might be able to stable the refractory hypotension after some operations. It might relate with the recovery of vascular endothelial function by balancing the ratio of nitro oxide and endothelin concentration according to some investigations [21-22]. therefore, it could be a potential approach to treat the thorny vasoplegic syndrome after heart transplantation.

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