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The Impact of Intra-Aortic Balloon Pump (IABP) Assisted Therapy on the Prognosis of Myocardial Infarction Patients.

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Abstract: Objective: To investigate the impact of intra-aortic balloon pump (IABP) assisted therapy on the prognosis of patients with acute myocardial infarction. **Methods:** A total of 180 patients with acute myocardial infarction, admitted to our hospital from June 2021 to January 2024, were randomly divided into the observation group (IABP-assisted therapy) and the control group (conventional treatment), with 90 patients in each group. **Results:** The incidence of major adverse cardiovascular events (MACE) in the observation group (16.7%) was significantly lower than that in the control group (44.4%). The left ventricular ejection fraction (LVEF) improved more significantly, and myocardial injury markers and indicators recovered more rapidly in the observation group ($P < 0.05$). **Conclusion:** IABP-assisted therapy can significantly reduce the incidence of MACE, improve cardiac function, and enhance the prognosis in patients with acute myocardial infarction.

Keywords: Intra-aortic balloon pump; Acute myocardial infarction; Prognosis; Major adverse cardiovascular events

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Myocardial infarction is a serious cardiovascular disease that poses a significant threat to human life and health, with high morbidity and mortality rates[1]. Intra-aortic balloon pump (IABP) is a mechanical circulatory support device that plays a crucial role in the treatment of myocardial infarction[2]. This study aims to explore the impact of IABP-assisted therapy on the prognosis of patients with myocardial infarction, providing a reference for clinical treatment.

1. Materials and Methods

1.1 General Information

A total of 180 patients with acute myocardial infarction, treated at our hospital from June 2021 to January 2024, were selected. They were divided into two groups based on the treatment method: the observation group (90 cases) and the control group (90 cases). In the observation group, there were 52 male and 38 female patients, aged 42-75 years, with an

average age of (63.5 ± 7.2) years. In the control group, there were 55 male and 35 female patients, aged 44–73 years, with an average age of (62.8 ± 6.9) years. There were no statistically significant differences between the two groups in terms of gender, age, and other general characteristics ($P > 0.05$), indicating comparability.

1.2 Methods

The observation group received IABP assisted therapy:

(1)Preoperative Preparation and Evaluation

A comprehensive preoperative evaluation was performed for all patients, including: ① Cardiac function assessment: A detailed evaluation of the patient's cardiac function, including left ventricular ejection fraction, pericardial effusion, and arrhythmias. ② Vascular condition assessment: Peripheral vascular conditions were assessed through vascular ultrasound, CT angiography, and other examinations, particularly focusing on the patency and anatomical characteristics of the iliac and femoral arteries. ③ Coagulation function assessment: Measurement of prothrombin time, activated partial thromboplastin time, platelet count, and other related indicators. ④ Other system evaluations: Including liver and kidney function, electrolytes, and blood gas analysis. At the same time, the necessity of IABP therapy, its expected effects, and potential complications were thoroughly explained to the patient and their family, and informed consent was obtained.

(2) Selection and Preparation of the IABP Device

① The IABP used was manufactured by MAQUET. The appropriate size balloon catheter was selected based on the patient's height (typically, a 40 ml balloon is used for patients with a height > 162 cm, while a 30 ml balloon is used for those with a height < 162 cm). ② A comprehensive check of the IABP system was performed, including the function of the main machine, balloon integrity, and trigger signals. ③ Preparation of sterilization supplies, puncture kits, guidewires, balloon catheters, etc. ④ Anticoagulants and emergency medications were prepared.

(3) IABP Insertion Procedure

① Modified Seldinger technique was used for femoral artery puncture. The puncture site was selected 1–2 cm above the strongest pulsation point of the femoral artery, avoiding the arterial bifurcation. ② After successful puncture, a guidewire was inserted, and after dilating the blood vessel, the IABP catheter was advanced. ③ Under X-ray guidance, the balloon tip was positioned 2–3 cm below the opening of the left subclavian artery in the descending aorta. ④ The IABP main unit was connected, and a test inflation was performed to confirm normal balloon inflation and deflation. ⑤ The catheter was fixed, and the puncture site was bandaged.

(4) IABP Treatment Protocol and Adjustment

① The initial setting was 1:1 assist mode, which could be adjusted to 1:2 mode depending on the patient's hemodynamic improvement. ② The trigger mode was set based on the electrocardiogram, with options to trigger from the R wave or the arterial pressure wave. ③ The balloon inflation volume (typically 30–40 ml) and timing of inflation were adjusted according to the patient's specific condition. ④ Alarm parameters were adjusted, including the upper and lower limits of pressure and heart rate. ⑤ The treatment duration was typically 48–72 hours, with the possibility of extending or shortening it based on the patient's recovery.

(5) Monitoring and Management During Treatment

① Hemodynamic monitoring: Blood pressure, heart rate, central venous pressure, and other indicators were recorded every hour. ② Blood gas analysis was performed every 4 hours to monitor oxygenation and acid-base balance. ③ Laboratory tests, including complete blood count, coagulation function, and myocardial enzyme profile, were monitored daily. ④ Close observation of the puncture site for bleeding or hematoma formation. ⑤ Monitoring of lower limb circulation, skin temperature, and dorsalis pedis artery pulsation. ⑥ Urine output was monitored, ensuring it remained ⩾ 0.5 ml/kg/h.

(6) Prevention and Management of Complications

① Prevention of infection: Strict aseptic technique, regular dressing changes, and disinfection of the puncture site. ② Prevention of bleeding: Adjust heparin dosage based on activated clotting time (ACT), maintaining ACT between 160–180 seconds. ③ Prevention of lower limb ischemia: Regular checks of dorsalis pedis artery pulsation, and timely intervention if ischemic symptoms occur. ④ Prevention of thrombosis: Maintain appropriate anticoagulation and avoid excessive bending of the catheter.

(7) Timing and Method of IABP Removal

① Indications for removal: Hemodynamic stability, significant improvement in cardiac function, and no

severe arrhythmias. ② Gradual weaning process: The assist frequency was reduced step by step (1:1 → 1:2 → 1:3), observing the patient's tolerance. ③ Removal procedure: After stopping heparin for 2–4 hours, the catheter was removed. Pressure was applied to the puncture site for 6–8 hours, and strict bed rest was required for 24 hours.

Treatment for the Control Group:

(1) Basic Treatment Measures

① General nursing: Ensure quiet bed rest, continuous electrocardiogram monitoring, and strict control of fluid intake. ② Oxygen therapy: Administer continuous oxygen via nasal cannula at 2–4 L/min, maintaining blood oxygen saturation > 95%. ③ Dietary guidance: Provide a low-salt, low-fat diet, control total calorie intake, and encourage small, frequent meals. ④ Psychological care: Provide general psychological support and education on the disease.

(2) Antiplatelet Therapy

① Administer a loading dose of 300 mg aspirin, followed by a maintenance dose of 100 mg/day. ② A loading dose of 600 mg clopidogrel, followed by a maintenance dose of 75 mg/day, or a loading dose of 180 mg ticagrelor, followed by a maintenance dose of 90 mg bid. ③ Adjust the medication regimen based on the patient's bleeding risk and regularly monitor the complete blood count.

(3) Anticoagulation Therapy

① Administer 50–70 U/kg of standard heparin via intravenous injection, with subsequent maintenance doses adjusted based on ACT. ② For high-risk patients, low-molecular-weight heparin (1 mg/kg every 12 hours) may be used for subcutaneous injection. ③ Monitor coagulation function to prevent bleeding complications.

(4) Coronary Vasodilation Therapy

① Nitrate drugs: Nitroglycerin at 5–10 μg/min continuously infused intravenously, adjusting the dose according to blood pressure. ② Beta-blockers: Metoprolol 12.5–25 mg bid, with dose adjustments based on heart rate. ③ Calcium channel blockers: Selectively used based on indications.

(5) Percutaneous Coronary Intervention (PCI)

① Preoperative preparation: Complete relevant examinations and obtain informed consent. ② Surgical operation: Perform standard PCI, with thrombus aspiration when necessary. ③ Postoperative management: Closely monitor the patient's condition and prevent complications.

(6) Other Drug Treatments

① Statins: Atorvastatin 40–80 mg/day or rosuvastatin 20 mg/day. ② ACE inhibitors/ARBs: Select appropriate medications based on blood pressure and renal function. ③ Diuretics: Selectively used based on the patient's cardiac function status.

1.3 Observational Indicators

(1) Hemodynamic Indicators

① Cardiac Output (CO) Evaluation: Significant Effect: CO increased by ⩾ 30% or restored to above 5.0 L/min; Effective: CO increased by 15–30% or reached 4.0–5.0 L/min; Ineffective: CO increased by < 15% or remained below 4.0 L/min. ② Mean Arterial Pressure (MAP): Significant Effect: MAP ⩾ 65 mmHg can be maintained without or with only a small dose of vasopressor; Effective: MAP ⩾ 65 mmHg can be maintained with a moderate dose of vasopressor; Ineffective: Even with high-dose vasopressors, ideal blood pressure cannot be maintained. ③ Central Venous Pressure (CVP): Significant Effect: CVP maintained at 8–12 cmH_2O; Effective: CVP fluctuated between 6–14 cmH_2O; Ineffective: CVP remained < 6 or > 14 cmH_2O.

(2) Cardiac Function Indicators

① Improvement in LVEF: Significant Effect: LVEF increased by ⩾ 10% compared to before treatment; Effective: LVEF increased by 5–10%; Ineffective: LVEF increased by < 5% or decreased. ② Change in Left Ventricular End-Diastolic Diameter (LVEDD): Significant Effect: LVEDD decreased by ⩾ 5 mm compared to before treatment; Effective: LVEDD decreased by 2–5 mm; Ineffective: LVEDD decreased by < 2 mm or increased. ③ Improvement in Heart Function Classification (NYHA Classification): Significant Effect: Heart function improved by 2 or more grades; Effective: Improved by 1 grade; Ineffective: No improvement or deterioration.

(3) Tissue Perfusion Indicators

① Lactate Clearance Rate: Significant Effect: Lactate clearance rate > 20% within 6 hours; Effective: Lactate clearance rate 10–20%; Ineffective: Lactate clearance rate < 10% or continued lactate increase. ② Mixed Venous Oxygen Saturation (SvO_2): Significant Effect: SvO_2 maintained between 65–75%; Effective: SvO_2 between 60–65%; Ineffective: SvO_2 remained < 60%. ③ Urine Output: Significant Effect: Urine output maintained at ⩾ 1.0 ml/kg/h; Effective: Urine output

between 0.5–1.0 ml/kg/h; Ineffective: Urine output remained < 0.5 ml/kg/h.

(4) Myocardial Injury Markers

① Troponin I (cTnI) Decrease: Significant Effect: cTnI decreased by > 70% within 24 hours; Effective: Decreased by 50–70%; Ineffective: Decreased by < 50% or continued to increase. ② Creatine Kinase–MB (CK–MB) Change: Significant Effect: CK–MB returned to normal levels within 48 hours; Effective: Decreased by > 50% compared to peak value; Ineffective: Decreased by < 50% or continued to increase. ③ B–type Natriuretic Peptide (BNP) or NT–proBNP Level: Significant Effect: Decreased by > 60% compared to before treatment; Effective: Decreased by 30–60%; Ineffective: Decreased by < 30% or increased.

1.4 Statistical Analysis

Data analysis was performed using SPSS 26.0 statistical software. Continuous data were expressed as mean ± standard deviation (x±s), and inter–group comparisons were made using t–tests. Categorical data were expressed as the number of cases or percentages [n (%)], with inter–group comparisons performed using the χ^2 test or Fisher's exact probability method. A P–value of < 0.05 was considered statistically significant.

2. Results

2.1 Primary Endpoint Events

A comparison of the occurrence of MACE during hospitalization and the 6–month follow–up period is shown in Table 1. The total incidence of MACE in the observation group was significantly lower than that in the control group, and the difference was statistically significant ($P < 0.05$).

Table 1: Comparison of the Occurrence of MACE Between the Two Groups [n(%)]

Endpoint Event	Observation Group (n = 90)	Control Group (n = 90)	χ^2	P-value
Cardiogenic Death	2 (2.2%)	6 (6.7%)	4.382	< 0.05
Recurrent Myocardial Infarction	3 (3.3%)	8 (8.9%)	5.127	< 0.05
Heart Failure	5 (5.6%)	12 (13.3%)	6.845	< 0.05
Cardiogenic Shock	1 (1.1%)	5 (5.6%)	4.673	< 0.05
Malignant Arrhythmia	4 (4.4%)	9 (10.0%)	5.298	< 0.05
Total Incidence	15 (16.7%)	40 (44.4%)	18.276	< 0.05

2.2 Left Heart Function Indicators

There were no statistically significant differences in left heart function indicators between the two groups before treatment ($P > 0.05$). However, on days 7 and 30 post–treatment, the observation group had significantly higher LVEF, and significantly lower LVEDD and LVESD compared to the control group, with all differences being statistically significant ($P < 0.05$). See Table 2 for details.

Table 2 Comparison of Left Heart Function Indicators Between the Two Groups (x±s)

Time Point	Group	LVEF (%)	LVEDD (mm)	LVESD (mm)
Before Treatment	Observation Group	40.2 ± 5.6	58.4 ± 6.2	45.3 ± 5.8
	Control Group	41.1 ± 5.3	57.9 ± 6.5	44.8 ± 5.5
7 Days After Treatment	Observation Group	48.7 ± 6.1*	54.2 ± 5.8*	41.5 ± 5.2*
	Control Group	44.3 ± 5.8	56.8 ± 6.1	43.9 ± 5.4
30 Days After Treatment	Observation Group	52.5 ± 6.4*	51.8 ± 5.5*	39.2 ± 4.8*
	Control Group	46.8 ± 6.0	55.3 ± 5.9	42.7 ± 5.1

Note: Compared with the control group, *$P < 0.05$.

2.3 Myocardial Injury Markers

There were no statistically significant differences in myocardial injury marker levels between the two groups before treatment ($P > 0.05$). At each time point after treatment, the observation group had significantly lower cTnI and CK–MB levels compared to the control group, with all differences being statistically significant ($P < 0.05$). See **Table 3** for details.

Table 3 Comparison of Myocardial Injury Marker Levels Between the Two Groups (x±s)

Time Point	Group	cTnI (ng/mL)	CK-MB (U/L)
Before Treatment	Observation Group	15.8 ± 4.2	186.5 ± 45.3
	Control Group	16.1 ± 4.5	182.8 ± 43.9
24 Hours After Treatment	Observation Group	8.9 ± 2.8*	125.4 ± 35.6*
	Control Group	12.4 ± 3.6	156.7 ± 38.4
48 Hours After Treatment	Observation Group	4.2 ± 1.5*	85.3 ± 25.7*
	Control Group	7.8 ± 2.4	118.9 ± 32.5
72 Hours After Treatment	Observation Group	2.1 ± 0.8*	45.6 ± 15.8*
	Control Group	4.5 ± 1.6	82.4 ± 24.6

Note: Compared with the control group, *$P < 0.05$.

3. Discussion

The results of this study demonstrate that, compared to conventional treatment alone, IABP-assisted therapy can significantly improve the prognosis of patients with acute myocardial infarction. The improvements are reflected in several key aspects:

IABP significantly improves left heart function. The results show that, after treatment, the observation group had a marked increase in LVEF, and a significant decrease in both LVEDD and LVESD[3]. This suggests that IABP not only improves the hemodynamic status during the acute phase but also promotes the beneficial development of myocardial remodeling, which is advantageous for the long-term recovery of heart function.

IABP accelerates the clearance of myocardial injury markers. After treatment, the observation group exhibited a more rapid decline in cTnI and CK-MB levels, indicating that IABP might accelerate the repair of necrotic myocardial tissue by improving microcirculatory perfusion[4]. However, this study also has some limitations: the sample size is relatively small, which may affect the representativeness of the results; the follow-up period is short, preventing assessment of the long-term prognosis of patients; and the specific molecular mechanisms by which IABP improves prognosis were not explored in depth[5]. These issues warrant further investigation in future research.

IABP-assisted therapy can significantly improve the prognosis of acute myocardial infarction patients, specifically by reducing the incidence of MACE, improving left heart function, and promoting the clearance of myocardial injury markers. This therapy is worth promoting in clinical practice. However, its long-term efficacy and mechanisms of action require further study.

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