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ORIGINAL ARTICLE

Efficacy of Amiodarone Versus Amiodarone and Dexmedetomidine on Heart Rate and Rhythm Following Aortic Cross-Clamp Opening During Cardiopulmonary Bypass and the Early Postoperative Period in Patients Undergoing Valve Replacement Surgery With Rheumatic Mitral Valvular Disease and Chronic Atrial Fibrillation: A Prospective, Randomized, Controlled, Double-Blinded Trial

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Abstract

Background: Long-standing atrial fibrillation (AF) is a common arrhythmia, often associated with rheumatic valvular disease, particularly mitral valve disease, which results in left atrial enlargement. In developing nations such as India, AF is found almost exclusively in association with rheumatic mitral stenosis and mitral regurgitation. **Objective:** To assess the effectiveness of amiodarone and dexmedetomidine in the treatment of atrial fibrillation after cardiac surgery, especially in patients undergoing cardiopulmonary bypass (CPB). **Methods:** The study compared the effects of amiodarone, an antiarrhythmic drug classified as a class III agent, with those of dexmedetomidine, a selective α2-adrenergic agonist, on rate control and recovery of normal sinus rhythm (NSR) after cardiac surgery. Their pharmacological effects, including adrenergic stimulation inhibition, modulation of sodium, potassium, and calcium channels, and anti-inflammatory effects, were experimented upon in the treatment of postoperative AF. **Results:** Amiodarone exhibited a high rate of conversion and maintenance of NSR in 50–70% of patients with minimal hemodynamic instability. The combination of amiodarone and dexmedetomidine had an additive effect that improved postoperative AF control and prevented hemodynamic fluctuations. **Conclusion:** Both amiodarone and dexmedetomidine are useful in the treatment of postoperative AF; amiodarone is better at controlling rhythm, while dexmedetomidine is better at controlling inflammation and hemodynamic stability.

Keywords: Randomized, Valve Replacement Surgery, Amiodarone, Dexmedetomidine

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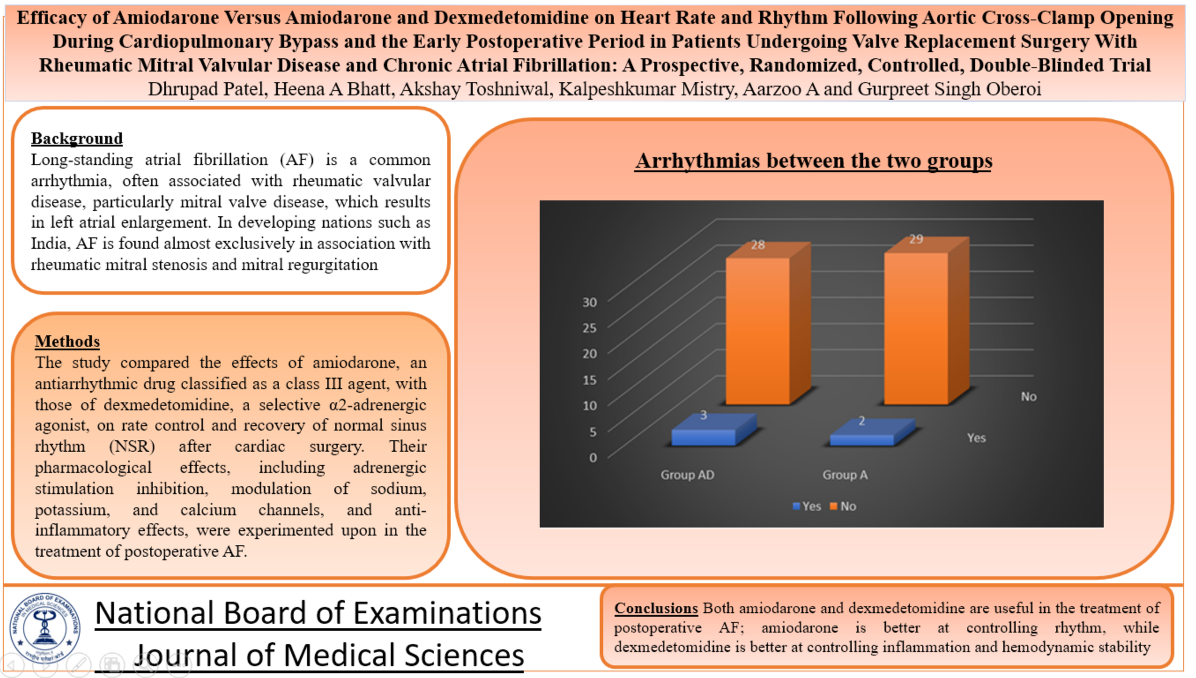
Graphical Abstract



Introduction

Chronic atrial fibrillation (AF) is a common arrhythmia frequently associated with rheumatic valvular disease (RHD), particularly mitral valve disease, which causes left atrial enlargement in the long run [1]. The prevalence of AF is increasing across the globe, while an increase in age in Western nations is a predominant factor. Rheumatic mitral stenosis (MS) and mitral regurgitation (MR) are the main causes in emerging nations like India. Controlling the rate is essential because AF's rapid ventricular rate (VR) and irregular rhythm impair cardiac function. Restoration of the normal sinus rhythm (NSR) is particularly critical after cardiac surgery to reduce morbidity [2]. Atrial fibrosis is a key element in the multifactorial aetiology of atrial fibrillation (AF), which is responsible for the onset and maintenance of the arrhythmia. Atrial tamponade, epicardial inflammation, hypoxia, acidosis, electrolyte imbalances, and sympathetic nervous system stimulation are other contributing factors that are frequently observed in valvular surgeries. Given this complexity, combining drugs with different pharmacological effects may enhance the efficacy of AF management postoperatively. Beta-blockers and calcium channel blockers, which are frequently employed in the management of AF, are known to induce hemodynamic instability, especially in cardiac surgery [3].

Amiodarone, a class III antiarrhythmic drug, blocks adrenergic stimulation and influences sodium, potassium, and calcium channels, greatly extending action potential and repolarization. This is followed by decreased atrioventricular conduction and enhanced sinus node function, rendering it useful in rate control of the ventricle without much hemodynamic instability. Intravenous amiodarone has been found to be effective in converting and sustaining NSR in 50–70% of patients because of its quick onset of action. It is not administered orally because of its extensive first-pass metabolism, with an intravenous half-life of about six hours, and the kidneys mainly excrete its metabolites [4]. Dexmedetomidine is a selective α2-adrenergic agonist with sympatholytic action, which influences presynaptic and postsynaptic receptors in the central nervous system. It has sedative, analgesic, and anti-inflammatory properties, decreases heart rate and inhibits sinus and atrioventricular nodal function. By enhancing myocardial oxygen demand, it is a possible prophylactic treatment for postoperative AF [5].

Cardiopulmonary bypass (CPB) induces a systemic inflammatory reaction that may lead to profound organ dysfunction, with the additional risk of AF. Dexmedetomidine's sympatholytic and anti-inflammatory properties could have an additive effect when used in conjunction with amiodarone, enhancing rhythm control during CPB weaning [6]. The objective of this research is to compare the effectiveness of amiodarone with a combination of amiodarone and dexmedetomidine in the control of heart rate and rhythm after aortic cross-clamp opening during cardiopulmonary bypass and in the postoperative period. Patients with rheumatic mitral valvular disease complicated by persistent atrial fibrillation who are having valve replacement surgery are the focus of the study. The main goal is to compare the efficacy of intravenous (IV) amiodarone alone with IV amiodarone plus dexmedetomidine in maintaining rhythm and heart rate after opening of the aortic cross-clamp and during early postoperative hours. The secondary goals involve assessing the effects of these regimens on several hemodynamic variables, including heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure. The study will also assess whether pacing is necessary after opening the aortic cross-clamp during surgery and in the postoperative period.

Materials and Methods

The study was conducted as a prospective, randomized, controlled, double-blinded trial. Institutional Research Ethical Board clearance and written informed consent from the patient were obtained before starting the study at Geetanjali Medical College and Hospital, Udaipur. The study patients were those undergoing valve replacement surgery for rheumatic mitral valvular disease with chronic AF on CPB between January 2021 and July 2022.

Group randomization and blinding were secured by keeping the data collector and the patient unaware of the group assignment. The study drug was administered by an independent anesthesiologist who was not participating in the study, as per the assigned group. The pre-anesthetic check-up was routine one day prior to surgery, and the patients were kept nil per os for eight hours prior to the surgery. Randomization with the aid of a computer randomization schedule resulted in two equal groups of 31 patients each. After starting CPB, Group A received a bolus of amiodarone (3 mg/kg) in 20 mL of normal saline and a placebo in 20 mL of normal saline (NS). Amiodarone 0.4 mg/kg/h (450 mg in 50 mL NS) and a placebo infusion (50 mL NS) were then administered intravenously for 24 hours after surgery. A bolus of dexmedetomidine (2 mcg/kg) and amiodarone (3 mg/kg) in 20 mL NS was given to Group AD. Additionally, an IV infusion of dexmedetomidine 0.1 mcg/kg/h (100 mcg in 50 mL NS) and amiodarone 0.4 mg/kg/h

(450 mg in 50 mL NS) was administered for 24 hours after surgery.

Sample size determination:

The sample size has been calculated from a prevalence of cardiac surgery of 2% for mitral valve disease with an absolute error of 7% at a 95% confidence level using the formula:

$$N=((Z\alpha + Z1-\beta)^2 * P(100-P))/E2$$

Where $Z\alpha=1.96$ at a 95% confidence level and $Z1-\beta=0.8413$ at 80% study power, the required sample size was calculated to be 31 patients in each group, making a total of 62 patients.

Adult patients aged 18 to 70 years and classified as ASA grade I, II, or III with BMI ≤28 kg/m² and an ejection fraction of ≥60% were studied. Patients who underwent valve replacement surgery with chronic AF were included. The exclusion criteria were baseline heart rate <50/minute, atrioventricular (AV) nodal block, thyroid dysfunction, abnormal liver function tests, serum creatinine >2 mg/dL, allergy to the study drugs, and pre-existing neurological, psychiatric, or respiratory illness.

Data collection method

As soon as the patient arrived in the operating room, initial measurements of heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), respiratory rate (RR), and oxygen saturation (SpO_2) were taken. An arterial line was inserted in the radial artery for invasive blood pressure monitoring, and a central venous catheter was inserted under local anaesthesia for central venous pressure monitoring. Baseline arterial blood gas (ABG) was analyzed. General anaesthesia was induced using midazolam (2 mg), fentanyl (5 µg/kg), etomidate (0.3 mg/kg), vecuronium (0.1 mg/kg), and lignocaine (1.5 mg/kg). Oxygen, air, and sevoflurane were used to maintain patients.

Intravenous heparin (400 IU/kg) was used to target an activated clotting time (ACT) of 480 seconds. The CPB circuit was primed with lactated Ringer's solution, sodium bicarbonate, mannitol, and heparin. After CPB initiation, the assigned study drugs were given over 10 minutes and subsequently by continuous infusion according to the group assignment. Haematocrit was 21–28% during CPB. Protection of the myocardium was done with antegrade cold cardioplegia (St. Thomas' solution-derived crystalloid-blood cardioplegic solution, 1:4 ratio, 20 mL/kg) at 20-minute intervals following aortic cross-clamping. The valve replacement was done under CPB with mild hypothermia, and all the patients were rewarmed to 37°C prior to weaning from bypass. The serum potassium was kept between 4 to 4.5 mEq/L in order to avoid arrhythmias secondary to electrolyte imbalance.

Once the aortic cross-clamp was released, the first cardiac rhythm was noted. If HR was <50/minute, epicardial pacing was started. In VF or VT, stepwise treatment with magnesium, lignocaine (1.5 mg/kg), and internal defibrillation (up to 50 J) was done. AV block was treated with AV sequential pacing and inotropic support with adrenaline (0.05–0.1 µg/kg/min) was started if SBP dropped below 90 mmHg.

After weaning from CPB, the residual effects of heparin were reversed using protamine sulfate (1:1 ratio to heparin). After surgical closure, patients were transported to the postoperative ICU and were observed for arrhythmias for 24 hours.

Important parameters monitored included rhythm and heart rate following aortic cross-clamp release, ventricular rate, pacing requirement, and DC cardioversion requirement. Continuous monitoring was done for heart rhythm, HR, SBP, DBP, MAP, SpO_2, and $EtCO_2$ for 24 hours after surgery.

Statistical analysis

Statistical analysis was performed with SPSS version 27.0. Continuous variables, including CPB time, aortic cross-clamp time, amount of cardioplegia utilized, and pre-aortic cross-clamp release potassium levels, were compared using the Student's t-test. Categorical variables were compared with the Chi-square test. A p-value <0.05 was taken to be statistically significant.

Results

Table 1. Demographic variables between groups

Parameters	Group AD	Group A	p-value
Age (years)	52.39±14.35	48.32±12.09	0.232
Males	13 (41.93%)	12 (37.20%)	0.999
Females	18 (58.06%)	19 (61.29%)	
Weight (kgs)	62.48±7.58	65.64±8.47	0.166
Height (cms)	166.09±8.01	168.32±10.01	0.337
BMI	23.0±3.35	23.0±2.93	1

Table 1 shows the various demographic variables like age, gender (male, female), weight, height and BMI in groups AD and A and were found to be statistically non-significant (p>0.05).

Table 2. Comparison of HR at various time intervals between groups

Duration	Group AD		Group A		t-test	p-value
	Mean	SD	Mean	SD		
5 minutes	89.41	6.58	87.41	8.02	-1.07	0.28
10 minutes	86.77	5.2	85.61	8.62	-0.6	0.52
15 minutes	82.64	7.66	82.51	9.12	-0.06	0.95

30 minutes	69.58	10.57	77.29	7.73	-3.278	0.002*
1 Hour	66.39	11.56	75.13	9.77	-3.215	0.002*
2 Hours	66.55	10.74	75.13	9.07	-3.398	0.001*
6 Hours	64.48	10.3	74.61	9.33	-4.058	<0.001*
16 Hours	65.93	9.68	73.68	9.15	-3.239	0.002*
24 Hours	63.35	6.35	72.29	8.45	-4.709	<0.002*

Table 2 shows the comparison of HR of patients in groups AD and A at different time intervals. It was found that there was statistical significance between both groups in the time intervals 30 minutes, 1 hour, 2 hours, and 6 hours post-aortic cross-clamp opening and postoperatively at 16 and 24 hours, respectively (p <0.05). Still, there was no significance between time intervals of 5 minutes, 10 minutes, and 15 minutes, respectively (p>0.05), in the ACC opening.

Table 3. Comparison of SBP between two groups at various time intervals

Duration	**Group AD**		**Group A**		**t-test**	**p-value**
	Mean	**SD**	**Mean**	**SD**		
5 minutes	130.25	11.11	128.22	6.63	-0.87	0.385
10 minutes	129.71	11.98	128.93	6	-0.32	0.748
15 minutes	130.16	12.34	129.45	5.13	0.26	0.768
30 minutes	121.61	14.08	137.9	12.03	-4.89	<0.001*
1 Hour	124.39	14.35	139.03	11.95	-4.37	<0.001*
2 Hours	122.71	14.56	140.45	12.21	-5.19	<0.001*
6 Hours	123.23	13.79	140.35	10.02	-5.59	<0.001*
16 Hours	121.87	11.67	137.93	13.11	-5.09	<0.001*
24 Hours	121.77	13.19	135.09	11.46	-4.24	<0.001*

Table 3 shows the comparison of SBP in patients between the AD and A groups. There was statistical significance between both groups in the time intervals 30 minutes, 1 hour, 2 hours, and 6 hours post-aortic cross-clamp opening and postoperatively at 16 and 24 hours, respectively ($p<0.05$). There was no significance between time intervals of 5 minutes, 10 minutes and 15 minutes, respectively ($p>0.05$) of ACC opening.

Table 4. Comparison of DBP between two groups at different time intervals

Duration	Group AD		Group A		t-test	p-value
	Mean	SD	Mean	SD		
5 minutes	76.03	3.57	75.48	4.79	0.51	0.611
10 minutes	75.51	4.97	75.38	5.86	-0.09	0.925
15 minutes	74.06	4.44	76.32	6.02	1.68	0.098
30 minutes	62.7	8.04	76.45	4.47	-8.32	<0.001*
1 Hour	63.58	8.09	75.48	4.35	-7.21	<0.001*
2 Hours	61.22	7.21	75.51	4.86	-9.15	<0.001*
6 Hours	61.74	8.09	76.32	4.39	-8.82	<0.001*
16 Hours	60.54	6.4	75.64	4.71	-10.58	<0.001*
24 Hours	60.93	6.71	74.45	4.96	-9.02	<0.001*

Table 4 shows the comparison of DBP in patients between groups AD and A. There was statistical significance between both groups in the time intervals 30 minutes, 1 hour, 2 hours, and 6 hours post-aortic cross-clamp opening and postoperatively at 16 and 24 hours, respectively ($p<0.05$). There was no significance between time intervals of 5 minutes, 10 minutes, and 15 minutes ($p>0.05$) for the ACC opening.

Table 5. Comparison of MAP between two groups at different time intervals

Duration	Group AD		Group A		t-test	p-value
	Mean	SD	Mean	SD		
5 minutes	94.1	3.72	93.06	3.81	-1.09	0.2802
10 minutes	93.58	5.81	93.23	4.37	-0.26	0.7933
15 minutes	92.76	3.85	94.03	4.79	1.14	0.2553
30 minutes	82.34	6.74	96.93	4.49	-10.37	<0.001*
1 Hour	83.84	7.57	96.66	4.56	-10.46	<0.001*
2 Hours	81.72	6.19	97.16	5.47	-11.91	<0.001*
6 Hours	82.23	6.82	97.66	4.87	-11.62	<0.001*
16 Hours	80.98	5.28	96.4	5.39	-11.38	<0.001*
24 Hours	81.21	6.32	94.66	5.06	-9.25	<0.001*

Table 5 shows the comparison of MAP in patients between groups AD and A. There was statistical significance between both groups in the time intervals 30 minutes, 1 hour, 2 hours, and 6 hours post-aortic cross-clamp opening and postoperatively at 16 and 24 hours, respectively ($p<0.05$). There was no significance between time intervals of 5 minutes, 10 minutes, and 15 minutes ($p>0.05$) for the ACC opening.

Table 6. Comparison of SpO2 between two groups at different time intervals

Duration	Group AD		Group A		t-test	p-value
	Mean	SD	Mean	SD		
5 minutes	98.25	1.36	98.25	1.34	0	1
10 minutes	97.96	1.3	98.41	1.28	-1.37	0.179
15 minutes	97.96	1.6	97.8	1.47	-0.01	0.99

30 minutes	98.45	1.5	98.16	1.29	0.82	0.42
1 Hour	97.93	1.31	98	1.46	-0.2	0.843
2 Hours	98.45	1.41	98.77	0.8	-1.1	0.27
6 Hours	98.9	0.7	99.12	0.84	1.14	0.256
16 Hours	98.12	1.56	97.93	1.28	0.52	0.6
24 Hours	98.06	1.23	98	1.43	0.18	0.86

Table 6 shows the comparison of SpO2 of patients in groups AD and A at different time intervals, and it was found that there was no significance between time intervals of 5 minutes, 10 minutes, 15 minutes, 30 minutes, 1 hour, 2 hours, 6 hours post aortic cross-clamp opening and postoperatively at 16hours, 24hourss respectively ($p>0.05$).

Table 7. Incidence of arrhythmias between the two groups

VT/VF	Group AD	Group A	p-value
Yes	3	2	0.6
No	28	29	

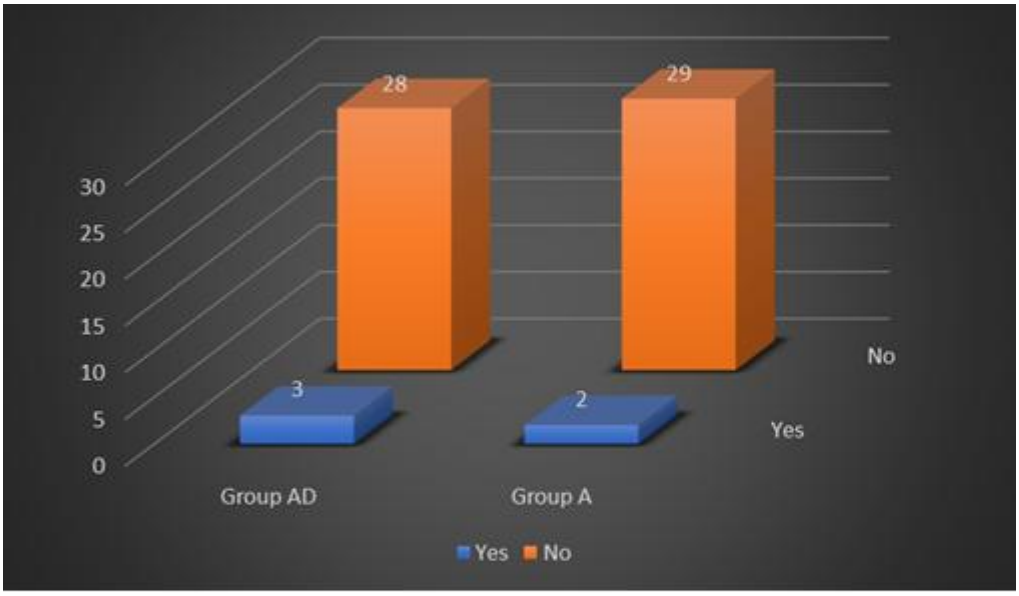


Figure 1. Graphical presentation of arrhythmias between the two groups

Table 7 and Figure 1 shows the incidence of arrhythmias in patients who had ventricular tachycardia (VT) and ventricular fibrillation (VF) post aortic cross-clamp opening between groups AD [n=3, 9.6%] and A [n=2, 2.45%] which was statistically non-significant (p>0.05).

Table 8. Incidence of Rhythm between two groups

Rhythm	Group AD	Group A	Chi-Square Value	p-value
SR	27	19	4.128	0.042*
AF	4	12		

Table 8 and Figure 2 show the incidence of rhythm, such as NSR and AF, in patients between groups AD [n=27, 87.1%] and A [n=19, 61.3%], which was statistically significant (p<0.05).

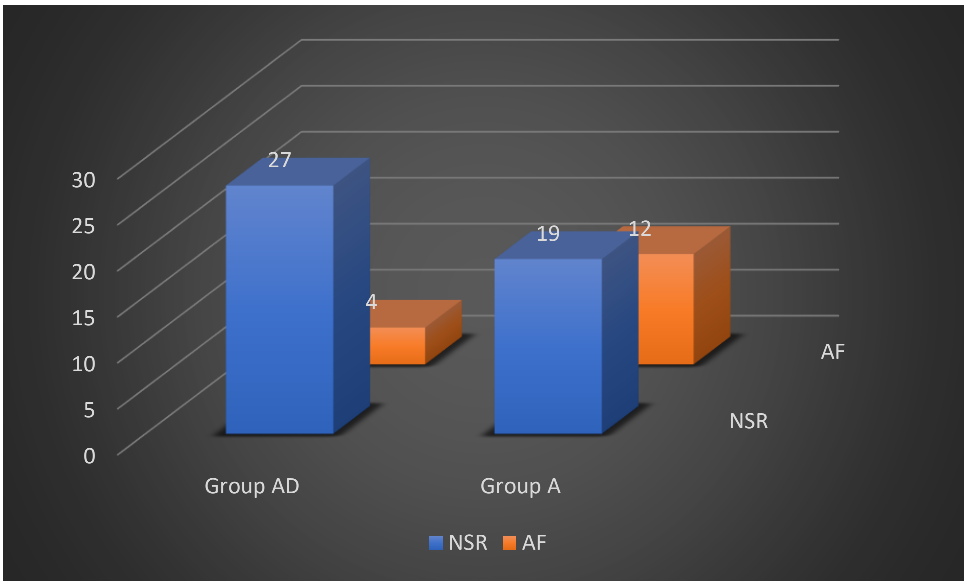


Figure 2. Graphical comparison of Sinus rhythm and Atrial fibrillation between two groups

Table 9. Comparison of incidence of need for pacing between two groups

Need for Pacing	Group AD	Group A	Chi-Square Value	p-value
Yes	11	3	4.521	0.033*
No	20	28		

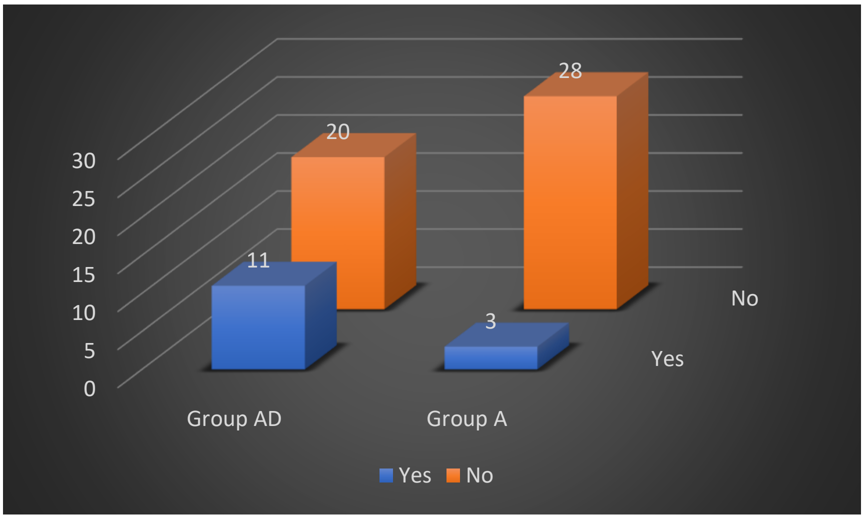


Figure 3. Comparison of incidence of need for pacing between two groups

Table 9 and Figure 3 show the comparison of the incidence of the need for pacing in patients, which was found to be statistically significant between groups AD (n=11, 35.5%) and A (n=3, 9.7%), respectively ($p<0.05$).

Table 10. Incidence of postoperative complications between two groups

Post Operative Complications	Group AD	Group A
Arrhythmia	6	7
Bradycardia	6	2
Hypotension	4	1

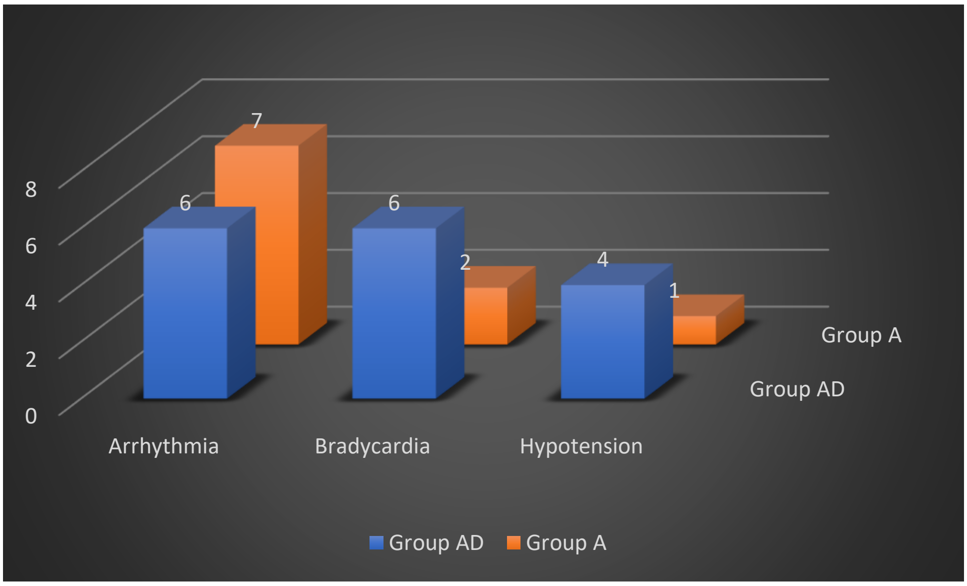


Figure 4. Incidence of postoperative complications between two groups

Table 10 and Figure 4 show the incidence of postoperative complications like arrhythmia, bradycardia and hypotension in patients between two groups, AD and A.

Discussion

Postoperative AF is a frequent and clinically relevant morbidity after MVR, especially for RHD. AF is related to higher morbidity, such as thromboembolic complications, hemodynamic compromise, increased duration of hospital stay, and more postoperative adverse events. Effective control and prevention of postoperative AF, despite evolving surgical and perioperative care measures, remain a persistent challenge. In most instances, AF continues even after the treatment of structural heart disease, which requires pharmacologic and non-pharmacologic therapies for successful rhythm control [7].

Treatment of postoperative AF is either rate control, which reduces the ventricular rate but permits AF to continue, or rhythm control, which seeks to restore and sustain sinus rhythm. Whereas rate control is the preferred choice in stable hemodynamic patients, rhythm control is an option for symptomatic AF, hemodynamic instability, or when maintaining sinus rhythm long-term. Of the antiarrhythmic drugs on hand, amiodarone is most commonly utilized because of its wide-range electrophysiological properties, which include extension of the action potential, blockade of various ion channels, and inhibition of ectopic atrial activity. Yet, amiodarone by itself is not always adequate, especially in those with high sympathetic drive or recurrent AF. Its delayed action and possible side effects, including bradycardia and hypotension, can also restrict its use in some clinical situations [8,9].

Dexmedetomidine, a very selective α2-adrenoceptor agonist, has received growing interest in the perioperative care of cardiac surgery patients because of its distinct pharmacological characteristics. It produces sedation, analgesia, and anxiolysis without causing severe respiratory depression. Most significantly, it possesses sympatholytic activity by preventing the release of catecholamines, thereby attenuating the hyperadrenergic state associated with cardiac surgery. The sympatholytic action leads to reduced heart rate and improved hemodynamic stability, and dexmedetomidine is a valuable adjunct in patients with arrhythmias. Since it is potent to modulate autonomic tone and to suppress overactive sympathetic activity, dexmedetomidine is presumed to enhance the therapeutic effectiveness of amiodarone in rhythm control by reducing the inducers of AF, such as tachycardia and hemodynamic instability [6,10].

The current research was designed to compare the therapeutic efficacy of amiodarone alone with amiodarone combined with dexmedetomidine in the maintenance of sinus rhythm and hemodynamic stability in MVR patients. The primary results revealed that the application of dexmedetomidine significantly improved the rate of maintenance of sinus rhythm, reduced heart rate variability, and facilitated improved blood pressure control, thereby improving overall hemodynamic stability. More of the amiodarone-dexmedetomidine patients were in sinus rhythm when the observation time was over. 87.1% remained in sinus rhythm, compared with 61.3% for the amiodarone-alone group. The number was statistically significant and represents the potential value of dexmedetomidine as a valuable adjunct to treatment for rhythm control after surgery. The increased rhythm stability in the dexmedetomidine group may be attributed to its sympatholytic effect that diminishes adrenergic drive, a strong predictor of postoperative AF initiation and sustenance. These results were similar to the findings of previous studies [11,12].

The other notable finding was the impact of dexmedetomidine on heart rate regulation. In the postoperative period, the amiodarone-dexmedetomidine group maintained lower heart rates compared with the amiodarone-alone group during the period. This would mean that dexmedetomidine's action to prevent inappropriate sympathetic stimulation played a critical role in maintaining cardiac function stably. Tachycardia is an adequately proven postoperative precipitant for AF, and management of tachycardia is crucial in attempting sinus rhythm maintenance. Due to heart rate variability, dexmedetomidine might have supported the generation of a more stable electrophysiological environment and thus eradicated the recurrence of AF. The results were similar to previous studies [12,13].

Blood pressure levels for the two groups also differed quite significantly. Reduced SBP, DBP and MAP at various postoperative intervals were noted among the patients given dexmedetomidine. This was to be expected as dexmedetomidine is capable of inhibiting sympathetic outflow and producing mild hypotension. No patient who received dexmedetomidine had severe hypotension necessitating stopping therapy, indicating that the decrease in blood pressure was near the upper end of the clinically acceptable range. The amiodarone group had more significant blood pressure variability, which could have caused a less stable postoperative course. The incidence of other arrhythmias, VF and VT, was minimally increased in the amiodarone-dexmedetomidine group, but this did not reach significance. This finding suggests that the amiodarone-dexmedetomidine combination did not increase the risk of lethal arrhythmias, an essential consideration in the evaluation of new rhythm control adjunctive treatments. Furthermore, the rate of other antiarrhythmic therapy, i.e., electrical cardioversion or rescue medication, was reduced with amiodarone-dexmedetomidine, a further indication of its rhythm-stabilizing ability. These were in accordance with previously done studies [14,15].

Hemodynamic side effects, including the need for vasopressor support, were less in the amiodarone-dexmedetomidine group. Fewer episodes of severe hypertension or hypotension needing treatment were noted in patients in this group. The reduced requirement for vasopressor therapy suggests that dexmedetomidine may assist in obtaining a more stable postoperative hemodynamic course by reducing abrupt blood pressure fluctuations. In addition, the anxiolytic and sedative effects of dexmedetomidine may have been responsible for avoiding stress-related hemodynamic alterations in the early postoperative period. The overall safety profile of the combination of dexmedetomidine-amiodarone was satisfactory. Although bradycardia occurred more frequently in the group treated with dexmedetomidine, it was not of sufficiently large clinical magnitude to warrant stopping the drugs. Severe bradycardia requiring pacing or urgent intervention did not occur in any patient. The finding is consistent with previous accounts that dexmedetomidine at therapeutic doses is not typically to blame for hemodynamically significant bradycardia in stable patients undergoing cardiac surgery [16–19].

Conclusion

The findings of the study introduce the potential advantages of the combination of dexmedetomidine and amiodarone in patients undergoing MVR. The combination was associated with an extremely high incidence of maintenance of sinus rhythm, improved heart rate and blood pressure control, lower hemodynamic complications, and reduced need for additional antiarrhythmic interventions. Importantly, no increased risk of significant arrhythmias or other severe adverse events was noted with the combination. These findings support the addition of dexmedetomidine to postoperative AF treatment protocols, particularly in high-risk patients for recurrent AF following MVR. Larger populations and longer follow-up are required to determine these benefits and individualize dosing regimens.

Statements and Declarations

Conflicts of interest

The authors declare that they do not have conflict of interest.

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