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A STUDY ON ROLE OF INTERVENOUS AMIODARONE IN ACUTE ONSET ATRIAL FIBRILLATION

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Abstract
Introduction: *Intravenous amiodarone is a recognised treatment alternative for the conversion of atrial fibrillation to sinus rhythm.* ***Aims and objectives:*** *The basic aim of the study is to analyse the role of intervenous amiodarone in acute onset atrial fibrillation.* ***Material and methods:*** *This cross sectional study was conducted in THQ Hospital, Fort Abbas during March 2019 to July 2019. The data was collected from 80 patients. Eligible patients had continuous monitoring of their ECG. In addition, a 12-lead ECG and blood pressure measurements were performed initially, after 1, 8 and 24 hours, and then every 24 hours for 3 consecutive days.* ***Results::*** *The data was collected from 80 patients. The majority were patients with chronic atrial fibrillation, atrial fibrillation of unknown age, or of more than 48 hours duration. Twenty-six patients presented with ROAF, 20 of whom (77%) were eligible for intravenous amiodarone. Six patients (23%) were excluded for one or more of the following reasons: two patients had already been on amiodarone treatment, and one patient each because of end-stage-renal failure, severe hypotension, and large left atrial thrombus revealed in initial echocardiogram; one patient refused therapy.* ***Conclusion:*** *It is concluded that intravenous amiodarone was feasible and relatively safe. However, the acute conversion rate was disappointing. It is suggested that intravenous amiodarone is probably more effective in patients with rapid ROAF and good left ventricular function.*

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INTRODUCTION:

Intravenous amiodarone is a recognised treatment alternative for the conversion of atrial fibrillation to sinus rhythm. Consensus regarding dosage and duration of treatment has not been reached. Recent-onset atrial fibrillation (ROAF) is a common finding in patients admitted to general internal medicine departments [1]. The optimal way to safely convert this arrhythmia to sinus rhythm has not been elucidated and has been the subject of much debate. The conventional use of class 1A anti-arrhythmic drugs, such as quinidine or procaineamide, has recently been relinquished, due to potentially dangerous pro-arrhythmogenicity [2]. Moreover, even type 1C anti-arrhythmic drugs, such as propafenone and flecainide, which are highly effective in converting ROAF to sinus rhythm, carry the pro-arrhythmic risk of transformation of atrial fibrillation to flutter with 1:1 atrioventricular conduction and haemodynamic compromise [3].

Intravenous infusion of amiodarone has been shown to be useful, though with variable efficacy, for control of ventricular rate and conversion of ROAF. However, adverse reactions such as phlebitis, bradycardia, AV block, hypotension, aggravation of heart failure, torsade de pointes and death, have been reported with short-term administration of amiodarone. Subsequently, it has been suggested that intravenous amiodarone should be used cautiously, as it may have lethal complications [4]. Therefore, most studies of the feasibility of acute amiodarone loading in ROAF, have been carried out in coronary care units or in highly selected patients. Moreover, previous studies have focused on the acute (24-hour) effect of amiodarone and data on longer periods of observation in ROAF patients treated with intravenous amiodarone are incomplete [5].

Aims and objectives

The basic aim of the study is to analyse the role of intervenous amiodarone in acute onset atrial fibrillation.

MATERIAL AND METHODS:

This cross sectional study was conducted in THQ Hospital, Fort Abbas during March 2019 to July 2019. The data was collected from 80 patients. Patients were excluded for any of the following reasons: pulmonary oedema, hypotension (systolic blood pressure <90 mmHg), mean ventricular rate during atrial fibrillation less than 70 beats/min, previous or current ECG evidence of ventricular pre-excitation, previous evidence of second- or third-degree atrioventricular block, hypokalaemia (serum potassium < 3.5 mmol/l), severe hypoxia (O_2sat <90%), acidosis (pH<7.35), significant liver disease or hepatitis, renal failure (serum creatinine >3 mg/dl), known thyroid dysfunction, patients already taking amiodarone and patients with an atrial thrombus on echocardiography.

Eligible patients had continuous monitoring of their ECG. In addition, a 12-lead ECG and blood pressure measurements were performed initially, after 1, 8 and 24 hours, and then every 24 hours for 3 consecutive days. The blood pressure was measured thrice daily thereafter. When patients converted to sinus rhythm, a 12-lead ECG was done immediately. Sustained conversion was defined as stable sinus rhythm that lasted for at least 24 hours and was also evident on discharge. All patients were examined twice daily by the treating physicians. Further data obtained on each patient included clinical history, results of physical examination, chest X-rays and trans-thoracic echocardiography.

Statistical analysis

Quantitative data were expressed as means ± SD. For the comparison of continuous variables, two-tailed Student *t*-test was used. Comparisons of proportions were done by Chi-square or Fisher's exact test.

RESULTS:

The data was collected from 80 patients. The majority were patients with chronic atrial fibrillation, atrial fibrillation of unknown age, or of more than 48 hours duration. Twenty-six patients presented with ROAF, 20 of whom (77%) were eligible for intravenous amiodarone. Six patients (23%) were excluded for one or more of the following reasons: two patients had already been on amiodarone treatment, and one patient each because of end-stage-renal failure, severe hypotension, and large left atrial thrombus revealed in initial echocardiogram; one patient refused therapy. Four of these six patients were discharged with sinus rhythm; they were treated with digoxin (one patient), propranolol (one patient), verapamil.

Table 01: Amiodarone effects on ventricular response rates. The values presented in the table are the mean ± SD ventricular response rate per minute.

		Time (hours after amiodarone intravenous loading)				
		0	**1**	**8**	**24**	**p-Value****
All patients	113±30	100±24	82±19	78±17	0.07; 0.001	
Group I	130±29	108±27	83±21	79±22	0.035; 0.001	
Group II	92±17	91±17	88±18	77±10	0.42; 0.4	
p-Value	0.002	0.05	0.24	0.45		

DISCUSSION:

Therapeutic goals for atrial fibrillation include ventricular rate control, stroke prevention, conversion to normal sinus rhythm and maintenance of normal sinus rhythm. The optimal strategy of rate versus rhythm control for acute management of patients with atrial fibrillation is a continuous debate [6]. However, selected patients may require acute treatment with antiarrhythmic agents for conversion of symptomatic atrial fibrillation episodes to normal sinus rhythm. For conversion of atrial fibrillation to normal sinus rhythm, amiodarone is effective and relatively rapid acting and is recommended by some authorities as a first line drug [7].

The relatively modest efficacy of intravenous amiodarone in conversion of ROAF to sinus rhythm in the present report is in accordance with the results of a recent study which compared intravenous amiodarone and placebo (both groups received digoxin), showing a non-significant difference between amiodarone and control in conversion rates (68% *vs* 60% after 24 hours). Similarly, Donovan *et al* detected no differences in conversion rates of ROAF between amiodarone (59%) and placebo (56%) after 8 hours of observation [8].

Atrial fibrillation is the most common arrhythmia and it accounts for about one third of hospitalization for arrhythmia. It involves the upper chambers (atria) of the 1 heart and the ventricular response is grossly irregularly irregular. Paroxysm of atrial fibrillation often precedes the 2 onset of sustained atrial fibrillation [9]. Its incidence increases with age. It occurs in rheumatic heart disease, atrial septal 3 defect, hypertension, mitral valve proplaspse and rarely runs in families. The patients can present with palpitation, general feeling of discomfort, diuresis or can present as heart failure and pulmonary oedema or stroke. Atrial fibrillation can lead to atrial electrical and mechanical remodeling leading to tachycardia induced cardiomyopathy. Patients with atrial fibrillation are more prone to 5 develop thromboembolism especially with history of heart failure, left ventricular dysfunction and left atrial dilatation [10].

CONCLUSION:

It is concluded that intravenous amiodarone was feasible and relatively safe. However, the acute conversion rate was disappointing. It is suggested that intravenous amiodarone is probably more effective in patients with rapid ROAF and good left ventricular function.

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