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Case Report

Test the Ischemic Atrial Fibrillation with Oxygen Therapy, a Case Report

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Received: October 16, 2018; **Published:** November 27, 2018

Abstract

Introduction: The problem in dealing with the cases of arrhythmia like atrial fibrillation is an ignorance of disease pathogenesis rather than the role of hypoxia in induction or precipitation of arrhythmia. The current case story started when an elder male patient presented to emergency department with acute atrial fibrillation (AF) and rapid ventricular rate.

Methods: Therapeutic trial depending on pathogenesis of atrial fibrillation was the study method that used in this study. My patient was previously managed for ischemic heart disease. Dramatic unexpected slowing of ventricular rate happened occurred after Oxygen (O_2) inhalation alone.

Conclusion: The aim of study directed towards clinical improvement of patient symptoms and termination or at least reduction of rate of arrhythmia like atrial fibrillation. Generally, hypoxemia and ischemia implicated in the pathophysiology of arrhythmia such as atrial fibrillation. Patient symptoms relieved with controlled the current arrhythmia. So, why don't use O2 as a therapeutic trial for ischemic type atrial fibrillation?

Keywords: *Test The Ischemic Atrial Fibrillation; Atrial Fibrillation; Oxygen Therapy*

Abbreviations

ACS: Acute Coronary Syndromes; AF: Atrial Fibrillation; AR: aortic Regurgitation; CRP: C-Reactive Protein; ECG: Electrocardiogram; HF: Heart Failure; HFNC: High-Flow Nasal Cannula Oxygen; ICU: Intensive Care Unit; TR: Tricuspid Regurgitation

Introduction

Atrial fibrillation (AF) is the most common sustained and serious cardiac arrhythmia among patients presenting at the emergency department [1-3]. Men are more often affected than women [4]. The lifetime risk of developing AF is about 25% in those who have reached the age of 40 [4]. Unfortunately, AF tachyarrhythmia is increasing in prevalence with age [2] from, < 0.5% at 40 - 50 years, to 5 - 15% at 80 years [4], More than 10% of AF cases are diagnosed in people over the age of 75 years rising to 23% in people over the age of 80 years [2]. The incidence of AF seem to be increasing (13% in the past two decades) [4]. AF is a supraventricular tachyarrhythmia occurring in 1 - 2% of the general population, characterized by predominantly uncoordinated atrial activation with subsequent deterioration of atrial mechanical function [2]. Age, hypertension, diabetes, valvular heart disease, obesity, weight loss, chronic obstructive pulmonary disease, sleep apnea, chronic kidney disease, and acute heart failure (HF) are risk factors associated with developing AF [3,5]. Acute hypoxia is

induced during coronary occlusion or when oxygen supply does not meet demand and can trigger cardiac arrhythmia [6]. The critically ill patient is particularly susceptible to arrhythmias given the metabolic, ischemic, and neurohormonal stressors present in the critical care unit [7]. The experimental basis of arrhythmias in the setting of acute myocardial ischemia and chronic myocardial infarction is described [8]. Patients may experience a variety of symptoms of AF [2]. Up to 90% of AF events may be symptomless [2]. The most common symptoms include palpitations, shortness of breath, tiredness or fatigue, generalized weakness, poor exercise intolerance, dizziness or light-headedness, and an irregularly irregular pulse [2]. Atrial fibrillation is associated mainly with stroke, heart failure and sudden death [2,5]. AF confers a 5-fold risk of stroke, and one in five of all strokes is attributed to this arrhythmia [6]. the risk of death from AF-related stroke is doubled and the cost of care is increased 1.5-fold [4]. The established risk scoring system of stroke in patients with AF is using cardiac failure, hypertension, age, diabetes, stroke (CHADS2) for risk assessment [9]. Hemodynamic instability and thrombo-embolic events related to AF result in significant morbidity, mortality, and economic cost [10]. Electrocardiogram study (ECG) is necessary in order to diagnose AF [4]. Management of AF patients is aimed at reducing the symptoms and at preventing the severe complications associated with AF [4]. Prevention of AF-related complications relies on anti-thrombotic therapy, control of ventricular rate, and adequate therapy of concomitant cardiac diseases [4]. These therapies may already alleviate symptoms; however, significant relief may require additional rhythm control therapy by cardioversion, anti-arrhythmic drug therapy, or ablation therapy [4]. Rate control to a target heart rate of less than 110 beats per minute is recommended in most people [11].

Oxygen therapy (O_2) is a standard component of treatment in patients with acute heart disease [12]. Hypoxemic patients benefit from O_2 insufflations; because hypoxia can induce general and brain ischemia [12]. Supplemental O_2 may increase O_2 delivery to ischemic myocardium and hence reduce myocardial injury supported by laboratory studies [13]. In addition to that, O_2 administration may reduce the incidence of unsuspected hypoxemia in patients with acute coronary syndromes (ACS) [14]. During hypoxia and ischemia, a fall in the intracellular ATP concentrations mediates the opening of ATP-sensitive potassium channels, which in turn causes hyper-polarization of the vascular smooth muscle cells and vasodilatation [15]. Animal studies have shown that K+/ATP channels play an important role in regulating coronary artery blood flow at rest and during hypoxia and ischemia [15]. One of the consequences of tachycardia is increased myocardial O_2 demand. This can cause angina or ischemic chest pain, particularly in patients having underlying coronary artery disease [16]. Griffin and his colleges (1990) found that, hypoxia was common during endoscopic retrograde cholangiopancreatography and occurred within the first 15 minutes [17]. Tachycardia occurred during these hypoxic episodes suggest that, the O_2 desaturation is clinically important [17]. O_2 Desaturation associated with tachycardia can result in myocardial ischemia in patients with border- line coronary perfusion [17].

However, high flow O_2 administration may not increase tissue O_2 delivery in non-hypoxemic patients [14]. In AF, chest discomfort generally results from inadequate blood flow to meet the needs of the heart; called angina [18]. This could be due to an increase in the heart's need for O_2 and/or a decrease in the heart's supply of blood and O_2 [18]. In some cases, chest pain is due to the rapid heart rate itself or perhaps due to stretching of the heart's chambers [14]. In addition to that, chest discomfort can result from worsening heart failure [18]. In the susceptible heart, acute mismatch between myocardial O_2 demand and supply could provoke myocardial ischemia or trigger arrhythmia [19]. High-flow nasal cannula Oxygen (HFNC) along with high quality sleep could reduce the release of inflammatory marker C-reactive protein (CRP), which is an independent predictor of atrial fibrillation [20].

Case Presentation

A seventy three year-old married heavy-smoker male Egyptian farmer presented to the emergency department with acute onset of palpitations, rapid breathing, and dizziness. The patient denied any previous similar attack. There were past history of recurrent angina and ischemic heart disease. Upon examination, patient appeared irritable, sweaty, and tachypnic. His vital signs were as follows: blood pressure: 140/80 mmHg, pulse rate: 175/minute; irregular, temperature: 36.7°C, respiratory rate: 24/min, initial pulse oximetry: 91%,

and absent 'a' waves in jugular venous pressure exam. Irregular tachycardia was heard on auscultation. No more relevant other clinical data was obtained from history or after examination. The initial workup was an ECG; that showed atrial fibrillation with rapid ventricular rate (180 bpm) (Figure 1). Patient was admitted to the critical care unit and initially managed with O_2 inhalation using nasal cannula in rate of 5 L/min. The heart rate started to slow down gradually within 10 minutes of O_2 inhalation as shown by ICU monitoring. ECG tracing was taken at that moment. Ventricular rate reached 120 bpm (Figure 2A). The monitoring physician advised for continuation of O_2 inhalation with close follow-up for patient's vital signs. Dramatic improvement of patient associated symptoms like palpitations, rapid breathing, and dizziness had happened after O_2 inhalation. The ECG was repeated 5 hours later (Figure 2B). Ventricular rate was 120 bpm. Troponin test, D-dimer level, electrolyte levels, complete blood count, thyroid studies, and random blood sugar were done for the patient. No abnormal results was detected in these investigations. Echocardiography showed septal hypokinesia, diastolic dysfunction, sclerotic aortic valve, trivial both AR and TR with normal ejection fraction (62%). Complete recovery had achieved and the patient was discharged within 12 hours of ICU admission with standard treatment of AF and ischemic heart disease (bisoprolol; 5 mg, amiodarone; 200 mg digoxin; 0.125 mg, warfarin; 1 mg, and aspirin; 75 mg) for follow-up. Atrial flutter, atrial tachycardia, atrioventricular nodal reentry tachycardia, sinus node dysfunction, ventricular fibrillation, and ventricular tachycardia, implicated in the case differential diagnoses.

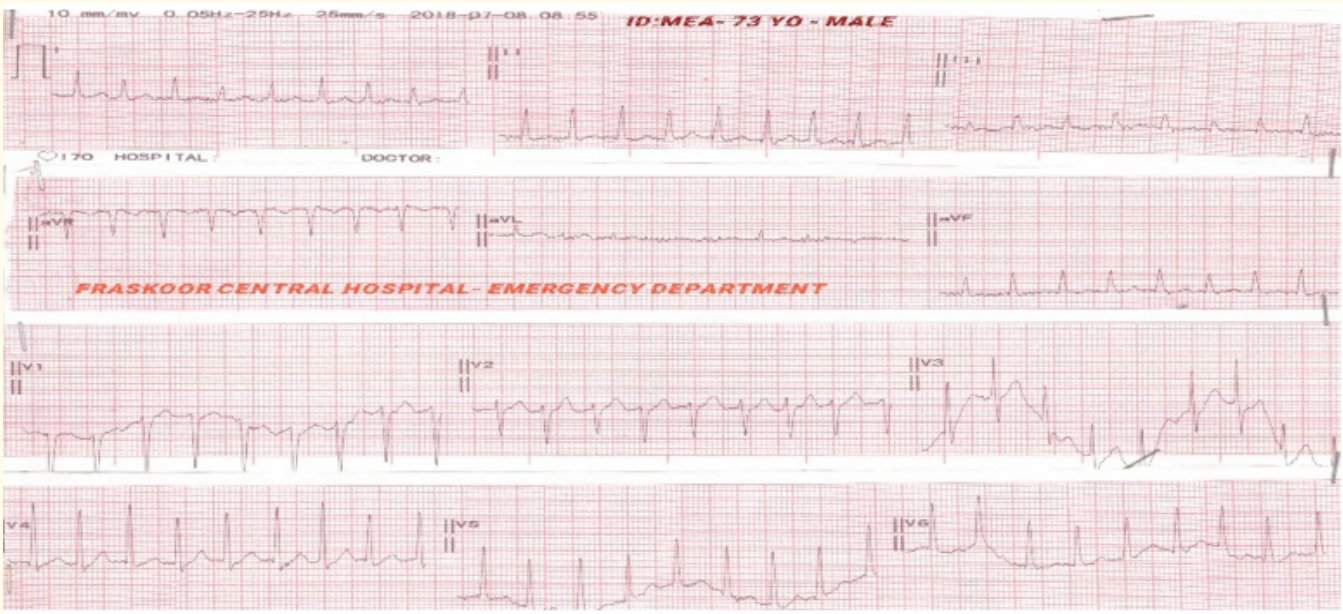

Figure 1: *ECG tracing of presentation in emergency room showing atrial fibrillation with rapid ventricular rate (180 bpm).*

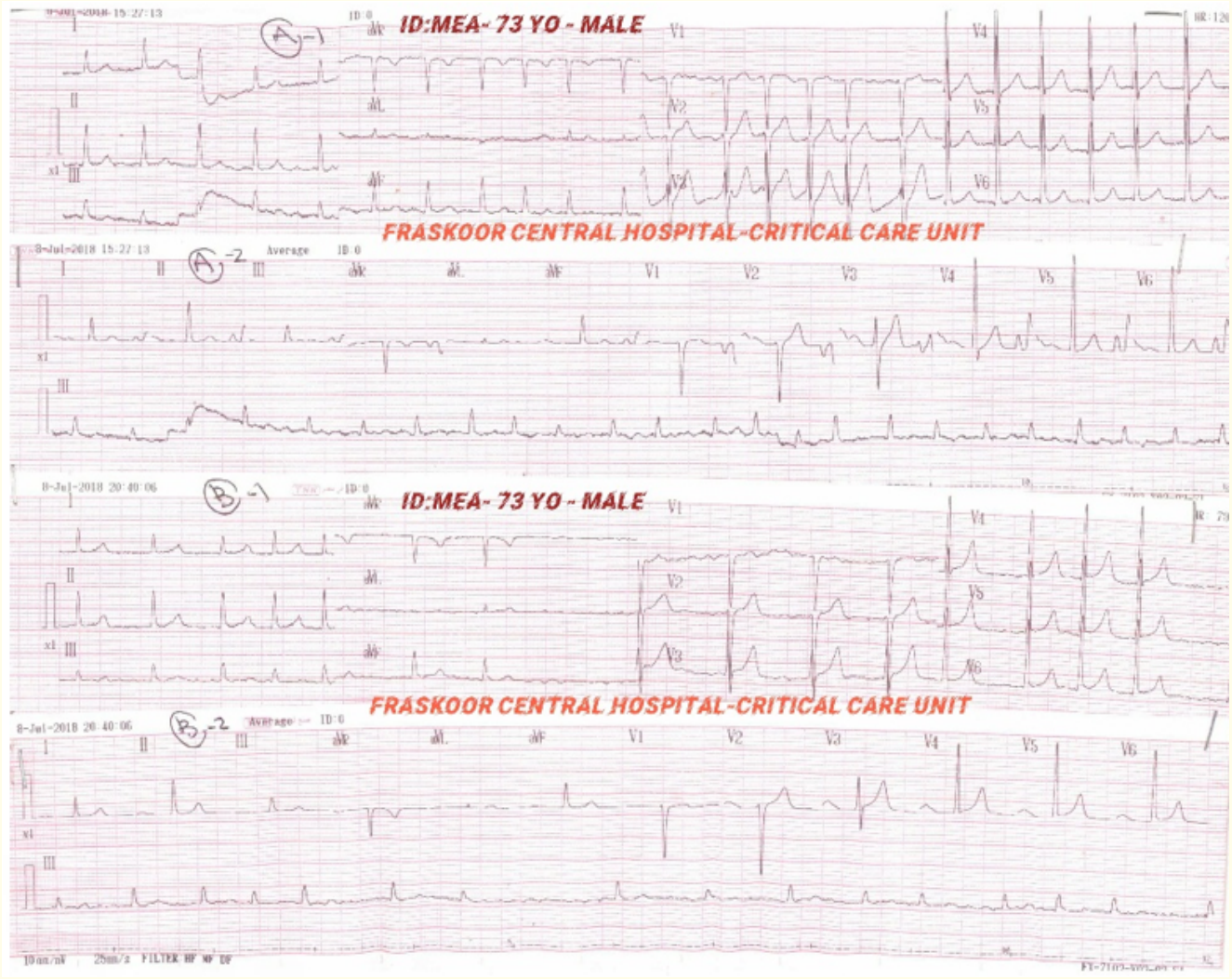

Figure 2: *ECG tracing 10 minutes after initiation of O_2 therapy in ICU showing atrial fibrillation with rapid ventricular rate (120 bpm) in "A" tracing. ECG after 5 hours of intermittent O_2 therapy showing controlled atrial fibrillation with ventricular rate (86 bpm) in "B" tracing.*

Discussion

- Overview of the case results included in dramatic response of atrial fibrillation to oxygen inhalation. The response in my case may undergo many factors. The main related factors are the evidence of hypoxia or ischemia. The response means positive rela tionship between hypoxic or ischemic related AF and oxygen response.
- I can't compare the current case study to other studies due to deficiency of the studies that only relay on using O2 therapy lonely in management of arrhythmia.

- Guidelines for using O_2 therapy in cases of arrhythmia in the emergency room and critical care unit suggested solutions for improved arrhythmogenic problems in my work hospital. Other hospitals can benefit from this research if shared and applied in their emergency room and critical care unit.
- Study question here; Why don't use O_2 therapy as a therapeutic test for possible ischemic or hypoxic related atrial fibrillation?
- The primary objective for my case study was termination or at least reduction of rate of arrhythmia like atrial fibrillation. But, the secondary objective was clinical improvement of patient symptoms.
- This case was a unique in the dramatic improvement of symptoms after O2 inhalation.
- Hypoxia and ischemia were implicated in the pathophysiology of the current case of AF. During the patient stay at the ICU, there was no use of anti-arrhythmic drugs, or anti-coagulants during the initial management of the case.
- The outcome with this current case comparing the traditional therapies for AF using rate control and rhythm control medications is very important.
- The adverse effects with using rate control and rhythm control medications are known and a well-defined hazardous. But, O_2 inhalation is often very safe.

Conclusion

- Hypoxia and ischemia were implicated in pathophysiology of the current atrial fibrillation.
- O_2 therapy was a crucial in AF management due to patient's ischemic history.
- The dramatic response of AF and its symptoms to O_2 therapy support the role of O_2 therapy in the initial therapeutic manage ment of ischemic AF or AF due to ischemic heart disease.
- That might lower the rates of use of anti-arrhythmic drugs in AF management, hence, their adverse effects.
- More research is needed for establishing protocols of O_2 therapy in the initial therapeutic management of ischemic AF.

Limitations of the Study

- There are no limitations for the study unless there are contraindications for oxygen therapy.
- However, hypoxic or ischemic related AF are preferable target for oxygen therapy.

Recommendations

- It is a recommended to start the oxygen therapy for any case of AF for few minutes before starting the standard therapy of AF.
- Also, widening the spectrum for the researching in this field is advisable.

Conflicts of Interest

No conflicts of interest.

Acknowledgement

I wish to thank Dr. Osama Maria, MD PhD, who edited the manuscript, and critical care unit team.

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Volume 2 Issue 4 December 2018