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Case Report Open Access

Acute Atrial Fibrillation Developed by the Inhalation of Chlorine Gas

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Abstract

Inhalation of chlorine gas is reported to cause upper and lower respiratory tract irritation, bronchospasm, respiratory failure and tachycardia. 31-year-old male patient was exposed to hydrochloric acid and chlorine bleach inhalation for 20 minutes. 48 hours after exposure, he was admitted to the emergency room with the complaints of increasing dyspnea and palpitation. The patient was tachypneic and tachycardic. His electrocardiogram revealed an atrial fibrillation at a rate of 145/min. The hemodynamics of the patient were stable, his hemogram, cardiac enzymes and other biochemical parameter measurements were normal. The patient was diagnosed with acute atrial fibrillation, he did not have any organic cardiac disease, as he was unresponsive to the administration of parenteral propafenone and metoprolol, he was treated with amiodarone infusion. 13 hours after the administration of amiodarone, his heart converted back to sinus rhythm. His control electrocardiogram and echocardiogram did not reveal any other pathology. He was discharged with metoprolol maintenance treatment.

Introduction

Chlorine gas inhalation is one of the most common among inhalation type intoxications. Chlorine was cited in almost one third of the morbidity cases following acute irritant exposure involving both adults and children. Toxic effects after inhalation exposure are usually mild to moderate, and death is uncommon. Large amounts of chlorine are produced in the industrial sector, and potential exists for accidental or deliberate release [1,2]. In a study recruited in Erzurum in Turkey, 64% of chlorine gas related respiratory tract intoxications occur during cleaning of the bathrooms and kitchens. A mixture of sodium hypochlorite (bleach, 40%) and hydrochloric acid (18%) is commonly used as a household cleaning solution [3]. These types of intoxications result from the inhalation of the toxic fume produced by mixing hypochloride (HOCI) and hydrochloric acid (HCI) [4-6]. Chlorine gas inhalation causes hypoxia, generation of free oxygen radicals (FORs) and increased sympathetic activation [1]. The extent and severity of injuriy from chlorine exposure depends upon duration of the exposure and concentration of the gas, individual susceptibility, water content of the exposed tissues and minute ventilation of the person exposed. Clinical presentation may be a spectrum of mucosal irritation to respiratory insufficiency requiring mechanical ventilation. There is a positive correlation between severity of symptoms and dose of exposure. A short exposure to 1-5 ppm doses will result in mucosal and ophthalmological irritation, while an exposure to 15-30 ppm causes cough, burning and choking; and a 30min exposure to 430 ppm may cause death.

Acute dyspnea, discomfort in the chest, cough, tachypnea, wheezing, vertigo, vomiting, irritation of the eye and tachycardia are commonly reported symptoms and signs [8]. The cardiac effect was commonly reported as sinusal tachycardia. Atrial fibrillation (AF) is the most common type of cardiac arrhythmia reported in the general population with an incidence of 1-2%, despite different factors were reported as responsible in its etiology [9], chlorine gas inhalation was not listed. That is why we here present a case of acute atrial fibrillation developing after the inhalation of chlorine gas.

Case Report

A 31 year-old male, without a documentation of previous ilness, drug use or addiction and physical complaints or sypmtoms, was admitted to the emergency department with tachycardia and dyspnea. During his interview, he reported exposure to chlorine bleach (HOCl) and hydrochloric acid (HCl) mixture for about 15-20 minutes after which he developed dyspnea, cough, tachycardia and burning in the eyes. His blood pressure was 120/80 mmHg, heart rate 120/min, and respiratory rate and peripheral oxygen saturation were 24/min and 95% respectively. On his physical examination there was mild conjunctivitis with red eyes and his laryngeal examination revealed normal vocal cords and airways.

During his observation in the emergency room under oxygen inhalation, ECG revealed sinusal tachycardia, his cardiac enzyme (CK-MB, troponin I) measurements and lung X-rays did not show any pathology and he was discharged after 6 hours. The next day, his dyspnea improved, but his tachycardia persisted while becoming more severe. Upon readmission to emergency, he was admitted to coronary intensive care unit. When he was admitted to the coronary unit, he was conscious, cooperative, tachypneic and tachycardic. In his personal history, he did not have any special characteristics, as a cardiovascular risk factor he smoked 10 packs/year and had a family history of coronary heart disease. Upon physical examination, the arterial blood pressure was 110/70mmHg, heart rate was 145/min-arrhythmic, respiratory rate was 22/min. Lungs were normal upon auscultation and they were equally participating in respiration. Blood gases analysis was not done since his peripheric oxygen saturation

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remained normal (SpO2 > 95%). Cardiac S1, S2 were normal, there was no S3, S4 or additional sounds. Other system examinations were normal. Elektrocardiogram (ECG) showed AF with a ventricular rate of 145/min (Figure 1). In transthoracic echocardiography (TTE), ejection fraction was 60%, heart chambers, regional wall movements and the systolic and diastolic functions of the ventricles, valvular functions and pulmonary artery pressures were normal. Laboratory tests; CK: 74 U/l, CK-MB:14U/l (0-25 U/l), Troponin I:0,01 (>0,1ng/ml), hematocrit (41%), leukocyte, TSH, freeT3, free T4, biochemical renal and liver tests, lipids and electrolytes were normal. During his observation, CK-MB and troponin I measurements were repeated twice with 6 hour intervals yielding normal results. During his stay in the coronary intensive care unit, he was diagnosed as acute atrial fibrilation with rapid ventricular response two days after the initiation of his symptoms.

Following the parenteral administration of propafenone and metoprolol in the emergency room, he was unresponsive. So, he was given amiodarone infusion (900mg/day), enoxaparin 0,1mg/kg/day s.c twice a day and acetylsalicylic acid. While the rhythm was AF with a ventricular rate of 116-135/min, during the 13th hour of amiodarone treatment, he converted to sinus rhythm. ECG did not reveal any additional pathology (Figure 2A,B). Following amiodarone infusion, maintenance treatment was initiated with metoprolol succinate 50mg twice a day. In control ECG recordings, the rhythm continued as sinusal rhythm and no pathology was identified in the control echocardiogram.

The patient was asymptomatic and did not show any findings favoring systemic embolism. On the third day of his treatment, he was discharged with metoprolol succinate 2x50mg/day and acetylsalicylic acid 300mg/day. In his first month control, he was asymptomatic and had sinus rhythm, so the treatment was discontinued. Second and sixth month controls without medical treatment did not demonstrate any cardiac pathology.

Discussion

Chlorine gas inhalation leads to hypoxia, generation of free oxygen radicals (FORs), and increased sympathetic activity [2]. Factors involved in tissue injury resulting from short-term sublethal exposure to chlorine gas depend on the intensity and the duration of the exposure, the presence or absence of a preexisting cardiopulmonary disease [10]. Chlorine is highly reactive, and inhalation of the gas causes cellular damage to the respiratory tract, inflammation, pulmonary edema, and airway hyperreactivity [11]. Animal models for chlorine gas inhalation have demonstrated evidence of oxidative injury and inflammation. Early epithelial injury, airway hyperresponsiveness, and airway remodeling, likely diminishing over time, have been shown [1]. Free oxygen radicals generated by the contact of the chlorine gas with water damage cellular proteins, ensuing tissue injury results in a decrease in myocardial contractility and deterioration of myocardial functions. Moreover, bronchospasm that develops after the irritation of the respiratory tract causes hypoxemia triggering cardiac arrhythmias and myocardial ischemia. Exposure to chlorine gas increases sympathetic stimuli and O_2 consumption and hinders myocardial contractility and cardiac conductivity [1]. Dyspnea and accompanying tachycardia seen in our patient resulted from such effects. Exposure to chlorine gas affects several other organs than the respiratory system. As was the case in our patient, with short exposure to chlorine gas, rhinitis, conjunctivitis, tracheobronchitis, pneumonitis and pulmonary edema have been reported [4,13,14]. However, there are limited reports about cardiovascular and rhythm findings after chlorine gas intoxication. Babu et al. [15] and Segal et al. [8] reported sinusal tachycardia on ECG, Guloglu et al. [3] reported the presence of sinusal tachycardia, extrasystoles and ST depression. In their study Kose et al. [12] reported sinus tachycardia and significant increases in cardiac enzymes.

There were no reports about acute onset, treatment- resistant atrial fibrillation. Together with the respiratory and cardiac symptoms our patient was diagnosed as new-onset AF due to toxic gas inhalation. In

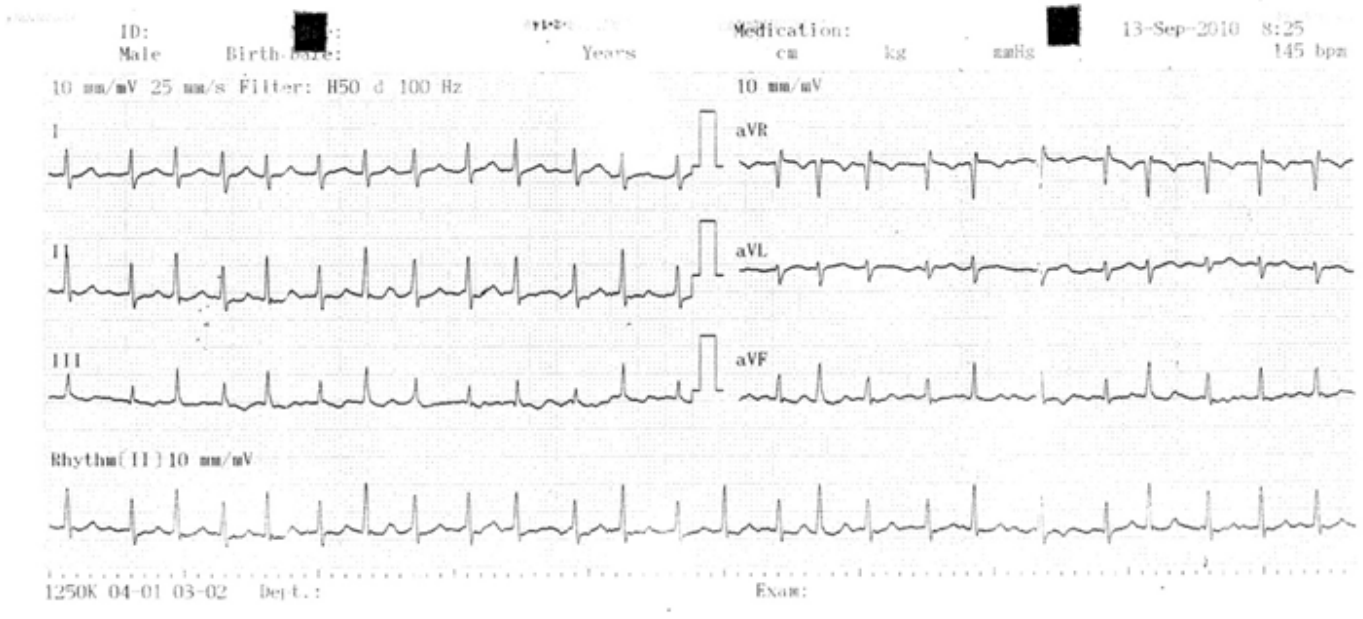

Figure 1: Acut Atriyal Fibrillation.

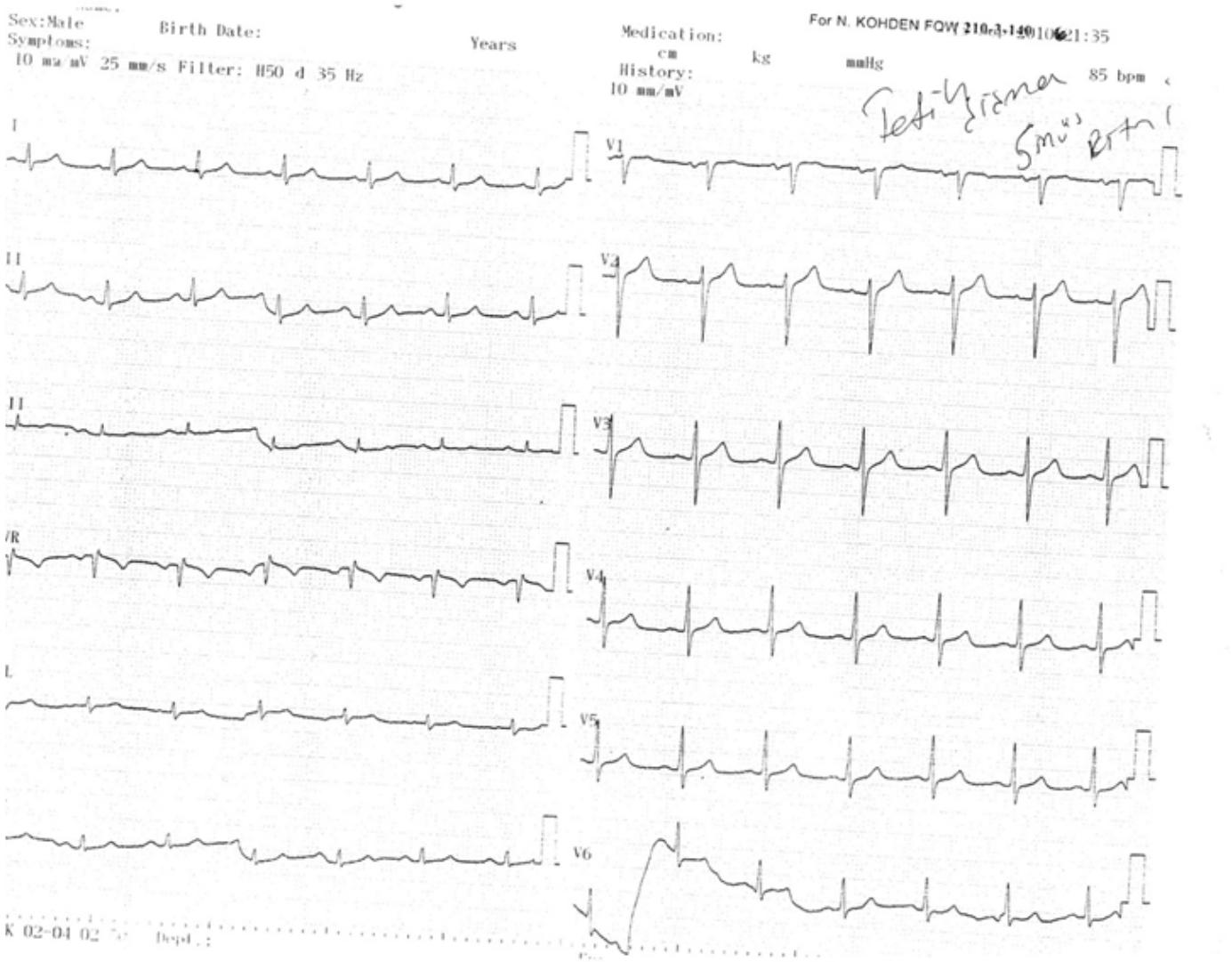

Figure 2a: Normal Sinus Rythim.

the etiology, there was no organic heart disease (ischemic heart disease, valvular disease, cardiomyopathy, hypertension, etc.) or thyroid disease that might result in metabolic AF. Among the examinations aimed at identifying the etiology, chest X-ray, TTE, biochemical tests and thyroid function tests were normal. It is well-known that chlorine gas inhalation leads to hypoxia, generation of free oxygen radicals (FORs), and increased

sympathetic activity [8]. But the patient was not hypoxic and oxidative stress markers and antioxidant levels were not evaluated in our case, so we propose that his resultant AF may be due to increased sympathetic activity. Because, for the AF in our case there was no predisposing factor or structural cardiac abnormality. Following exposure to chlorine gas, the cases respond differently to the administered treatment, this was explained by the differences in the duration of exposure, the presence of underlying systemic diseases and chronic repiratory diseases [12,16]. The cases are frequently discharged within 48 hours [17], however there might be persistent or delayed-onset symptoms. This was proven by demonstrating the presence of long-term histological changes in the lungs of the rats. These findings suggest that the people using these cleaning agents are confronted with the risk of harming themselves, and the victims of chlorine gas injury should be reexamined at a later period since they may have pulmonary damage even after 45 days following the exposure [18].

Most of the patients are discharged from the emergency room after they improve. Those who require hospitalization (19.8%) need an average hospital stay of 1.4 days [19]. Our case was initially discharged upon resolution of his symptoms. It is worthwhile to pay attention to the reappearance of his symptoms which included atrial fibrillation. For certain symptoms that have delayed-onset, appropriate follow-up was recommended to determine their resolution [5].

Such a situation makes us consider that the increases in the sympathetic activity and oxygen consumption result from tissue injury [1,2]. Use of anti-arrhythmics [20] that are effective for the increases in sympathetic activity did not generate the desired response in our case. This should make us understand that hypoxia

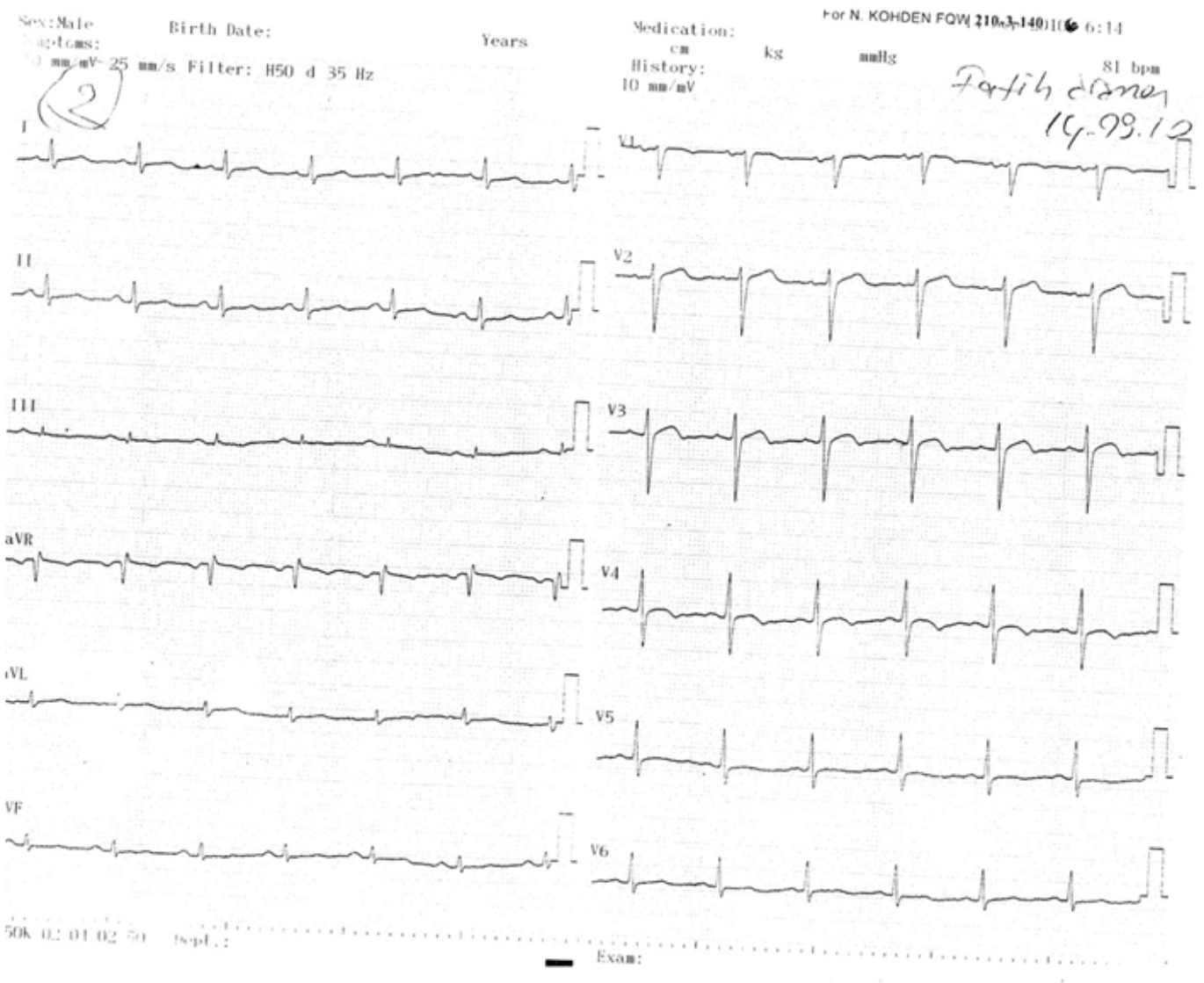

Figure 2b: Normal sinus rythim.

and epithelial damage should not be overlooked. For lung problems, beta agonist administration and humidified oxygen remains the mainstay of treatment; steroid therapy and bicarbonate inhalation are still inadequately supported [21]. There are also prospective studies emphasizing that nebulized sodium bicarbonate treatment is safe and has merits [19]. Beta antagonists are anticipated to be effective for AF, as our patient was unresponsive to this treatment, amiodarone was administered. Having a response to amiodarone should make us realize that different antiarrhythmics should be tried in the treatment of chlorine intoxication related AF and that this might increase the success of therapy.

Those who are exposed to chlorine gas should be warned about these potantial side effects. Such an awareness raising is important for early diagnosis and treatment. If the patients have underlying cardiac or systemic diseases, we should be more careful. In conclusion, we should keep in mind that exposure to chlorine gas might affect several systems but predominantly the respiratory system and might cause acute-onset AF. In environments and professions where chlorine gas exposure is a possibility, preventive and awareness-raising measures should be put into place. It is also important to train the emergency room doctors about the diversity of possible side effects, the time that elapses until discharge and the importance of long follow-ups.

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