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Acute Chlorine Gas Poisoning and Ecg Changes

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1. Abstract

1.1. Introduction: Chlorine is a yellow-green gas at room temperature. It is an extremely reactive element and a strong oxidizing agent. It has intermediate water solubility and it can cause acute damage to upper and lower respiratory tract. It can be detected easily because of its strong odor (1,2). Common cause of chlorine gas poisoning these days are accidental industrial exposure. Toxicity of chlorine depends upon the dose and duration of exposure.

1.2. Objectives of the Study: 1) To assess the frequency of hyperacute T waves on electro cephalogram (ECG) among patients diagnosed with chlorine gas poisoning. 2) To evaluate the relationship between incidence of hyperacute T waves and the troponin I levels among patients.

1.3. Methodology: A prospective observational study was conducted between October and November 2020 for a duration of 1 month at Liaquat National Hospital, Karachi. All patients between ages of 18-65 years diagnosed with chlorine gas poisoning presented to the hospital were included in the study. All patients with inconclusive diagnosis or those with diabetes mellitus, hypertension, chronic kidney, or liver disease, were excluded from the study. The demographic data, clinical characteristics and ECG findings were retrieved by reviewing patients file retrospectively. Data was compiled and analyzed using statistical package for social sciences (SPSS) version 25. A p-value of < 0.05 was considered as statistically significant.

1.4. Results: A total of 37 patients were diagnosed with chlorine poisoning. 27 (73%) patients had mild, 8 (21.6%) had moderate, and 2/37 had severe symptoms i.e. ARDS, edema, or pneumonitis. Out of 37 patients, 36 (97.3%) had hyperacute T waves on ECG and troponin I was positive in only one patient. The one patient in which troponin I was positive presented with severe symptoms of chlorine poisoning albeit the association was not significant ($p=0.054$). No significant association was found between ECG changes and the severity of chlorine poisoning ($p>0.05$).

2. Introduction

Chlorine is a yellow-green gas at room temperature. It is an extremely reactive element and a strong oxidizing agent. It has intermediate water solubility and it can cause acute damage to upper and lower respiratory tract. It can be detected easily because of its strong odor [1-2]. Common cause of chlorine gas poisoning these days are accidental industrial exposure. Toxicity of chlorine depends upon the dose and duration of exposure [2]. Inhalation poisoning substances are usually divided into three categories, simple asphyxiants, pulmonary irritants and chemical axphyxiants. Chlorine gas belongs to pulmonary irritants and it is considered as the most common toxic inhalant.

Low level poisoning at concentrations of 1 to 3 ppm chlorine gas act as an eye and oral mucosa membrane irritant. At high concentration of up to 15 ppm it produces pulmonary symptoms and it can be fatal at concentration of 450ppm [3]. The source of exposure includes industrial or chemical accidents, school chemistry lab or release of gas from swimming pool [2-5]. Findings due to chlorine gas poisoning include conjunctival irritation, sore throat, rhinorrhea, lacrimation, dyspnea, laryngeal edema and stridor [6]. Acute manifestation can be tracheitis and dyspnea but in the long run it can permanently damage the lungs [7-8]. Complication includes ARDS or respiratory failure [2, 4, 9].

Chlorine gas cause myocardial depression. In world war chlorine gas was used as chemical weapon and it causes severe myocardial depression and Right heart dilatation [10]. Cardiac dysfunction

could result from pulmonary hypertension and release of vasoactive mediators [11]. Multiple human and animal studies showed the involvement of cardiovascular system in chlorine poisoning [12-14]. In previous study they have reported different ECG changes including ST depletion, sinusoidal tachycardia and extrasystoles [15]. Due to the discrepancies in the previous literature, the present study was undertaken to observe and assess the changes such as hyper acute T waves among the patients who suffered from chlorine gas poisoning and its role as the early indicator of cardiac involvement. We wanted to assess the relationship between incidence of hyperacute T waves and the cardiac damage as indicated by troponin I levels.

3. Methodology

A prospective observational study was conducted between October and November 2020 for a duration of 1 month at Liaquat National Hospital, Karachi. After procuring ethical clearance from the institutional review board of Liaquat National Hospital, data collection was initiated. A total of 37 number of patients were included in the study using a non-probability convenience sampling technique. All patients between ages of 18 and 65 years, with diagnosed case of inhalational chlorine gas poisoning presenting to the emergency department within 12 hours of the onset of adverse symptoms were included in the study. All patients with inconclusive diagnosis or those with diabetes mellitus, hypertension, chronic kidney, or liver disease, were excluded from the study. The demographic data, clinical characteristics and ECG findings were retrieved by reviewing patients file retrospectively. The severity of symptoms of chlorine poisoning were stratified into three groups: i) Mild Symptoms indicated by conjunctival irritation and oral mucosal membrane burning, ii) Moderate Symptoms indicated by sore throat and dyspnea (shortness of breath), and iii) Severe Symptoms denoted by dyspnea, edema, pneumonitis, and Acute Respiratory Distress Syndrome (ARDS).

Data was compiled and analyzed using statistical package for social sciences (SPSS) version 25. Mean and standard deviations were calculated for the quantitative variables. Frequencies and percentages were calculated for the qualitative variables. The severity of symptoms and its correlations with the ECG findings (hyper acute T waves) were observed. Chi square test was applied for finding association between these categorical variables taking p-value of ≤ 0.05 as statistically significant.

4. Results

A total of 37 patients who were eligible as per inclusion and exclusion criteria were included in the study. All patients had a presenting history of accidental exposure to chlorine gas. All participants were males and the majority of them were in their middle age as depicted by figure 1.

A total of 33/37 patients presented to the emergency room within 12 hours out of which four patients arrived with delayed presentation (more than six hours of onset of symptoms of chlorine gas poisoning). 27/37 (73%) patients had mild symptoms including irritation and mucosal membrane burning, 8 (21.6%) had moderate symptoms of shortness of breath and sore throat, while 2/37 had severe symptoms i.e. ARDS, edema, or pneumonitis as depicted by table 1.

A total of 9/37 (24.3%) patients were admitted to the hospital for further assessment and observation. Five of these were admitted to the ward setup while other four patients were in critical condition so were shifted to the intensive care unit (ICU) at LNH.

Out of 37 patients 36 (97.3%) had hyperacute T waves on ECG and troponin I was positive in only one patient as shown in table 1.

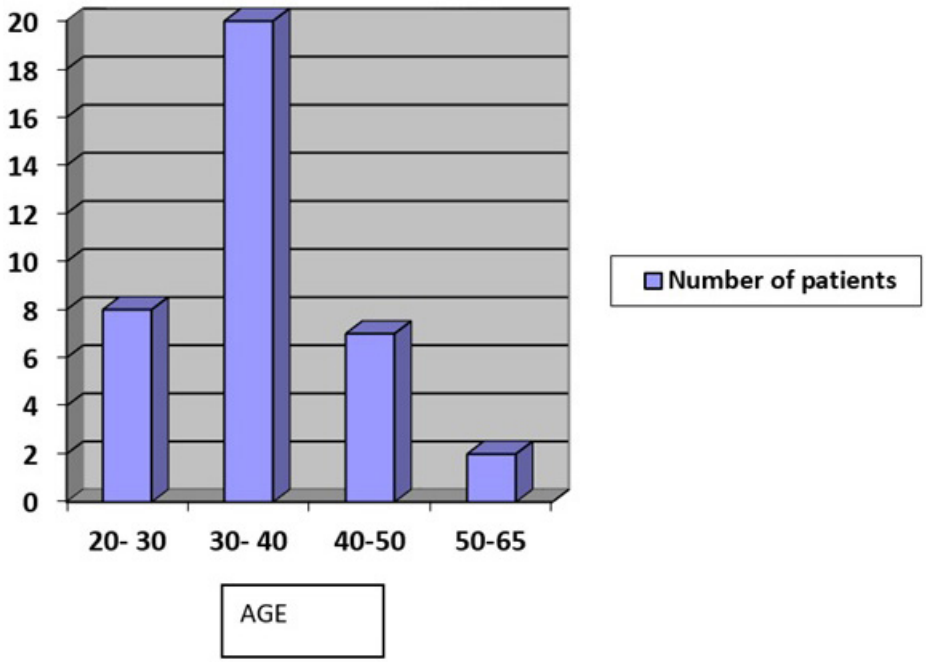

Figure 1: Bar chart of number of patients in respective age group.

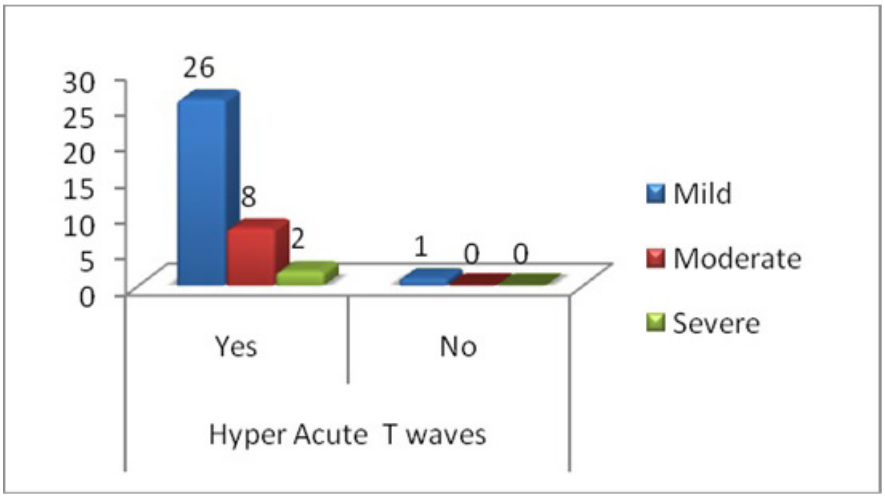

Figure 2: Bar chart showing association of symptoms with T waves.

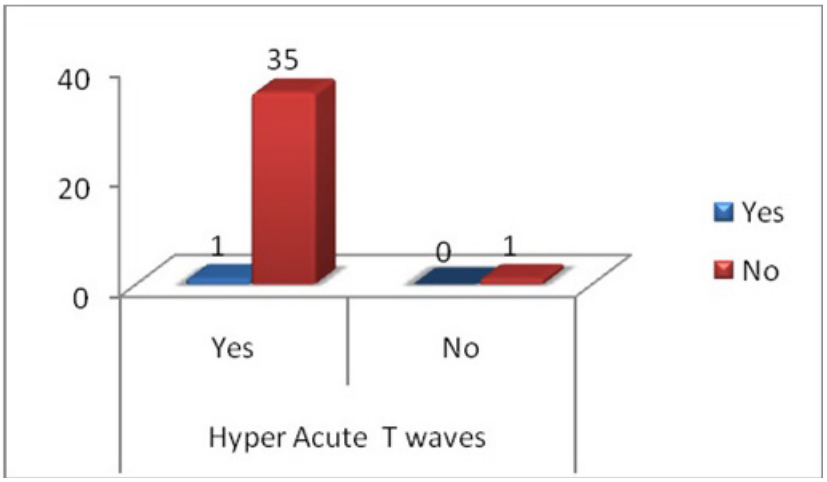

Figure 3: Bar chart showing association of Troponin I with T waves.

Table 1: Demographic of patients

Characteristics	**n (%)**
Patient arrived	
Within 6 hours	33(89.2)
After 6 hours	4(10.8)
Symptoms	
Mild	27(73)
Moderate	8(21.6)
Severe	2(5.4)
Hyper Acute -T waves	
Yes	36(97.3)
No	1(2.7)
Troponin I positive	
Yes	1(2.7)
No	36(97.3)
Admission	
Yes	9(24.3)
No	28(75.7)
Admitted to ward	
Yes	5(13.5)
Monitoring setup	
Yes	4(10.8)
Outcome	
Discharge	27(73)
Admission	9(24.3)
LAMA	1(2.7)

Table 2: Association of clinical characteristics of patients with incidence of hyperacute T waves on electro cephalogram (ECG)

Characteristics	Total n (%)	Hyper Acute T waves		P-value
		Yes	No	
Admission				
Yes	9(24.3)	9(25)	0(0)	1
No	28(75.7)	27(75)	1(100)	
Symptoms				
Mild	27(73)	26(72.2)	1(100)	1
Moderate	8(21.6)	8(22.2)	0(0)	
Severe	2(5.4)	2(5.6)	0(0)	
Patient arrived				
Within 6 hrs	33(89.2)	33(91.7)	0(0)	0.1
After 6 hrs	4(10.8)	3(8.3)	1(100)	
Troponin I positive				
Yes	1(2.7)	1(2.8)	0(0)	1
No	36(97.3)	35(97.2)	1(100)	
Outcome				
Discharge	27(73)	26(72.2)	1(100)	1
Admission	9(24.3)	9(25)	0(0)	
LAMA	1(2.7)	1(2.8)	0(0)	
*Significant at p-value <0.05				

In this study there was no significant association between incidence of hyperacute T waves on ECG of patients and the severity of chlorine poisoning, nevertheless, ECG changes were observed in majority of the patients as stated earlier.

Table 3. Association of clinical characteristics of patients with the severity of symptoms

Characteristics	Total n (%)	Symptoms Mild	Moderate	Severe	P-value
Admission					
Yes	9(24.3)	0(0)	7(87.5)	2(100)	<0.001*
No	28(75.7)	27(100)	1(12.5)	0(0)	
Patient arrived					
Within 6 hrs	33(89.2)	24(88.9)	7(87.5)	2(100)	1.000**
After 6 hrs	4(10.8)	3(11.1)	1(12.5)	0(0)	
Troponin I positive					
Yes	1(2.7)	0(0)	0(0)	1(50)	0.054
No	36(97.3)	27(100)	8(100)	1(50)	
Monitoring setup					
Yes	4(10.8)	0(0)	2(25)	2(100)	<0.001*
No (non monitoring setup)	5(13.5)	0(0)	5(62.5)	0(0)	
Outcome					
Discharge	27(73)	26(96.3)	1(12.5)	0(0)	<0.001*
Admission	9(24.3)	0(0)	7(87.5)	2(100)	
LAMA	1(2.7)	1(3.7)	0(0)	0(0)	
*Significant at p-value <0.05					

The one patient in which troponin I was positive presented with severe symptoms of chlorine poisoning albeit the association was not significant (p=0.054).

5. Discussion

Chlorine gas is a most common pulmonary irritant. It is very volatile and has moderate solubility in water. It primarily effects the respiratory system [1, 2]. The set of symptoms starts from irritation of mucosal membranes to fatal pneumonitis and Acute respiratory distress syndrome. Chlorine gas is been commonly used in various aspects of industries and its exposure to human environment is relatively frequent [5, 13]. This gas also effects the cardiovascular system. As mentioned in previous studies chlorine poisoning is attributed to the myocardia depression and ECG changes that includes ST depression, sinus tachycardia and extrasystoles [11, 14].

Chlorine gas released free oxygen species that was thought to be the cause of cellular damage. But recent studies have shown that oxidization of different components of cell results in damage to tissue. When chlorine reacts with water it formed an acid which irritates the mucosal and conjunctival membranes and cause burning sensations. Experimental studies showed that majority of gas is absorbed from the upper respiratory mucosa and only 5 percent is absorbed from the lower respiratory mucosa [16]. It was observed in World War 1 that chlorine poisoning causes Right sided heart dilation as described above.

In an experimental animal study chlorine poisoning cause release of calcium in myocytes that cause impairment in cytoskeleton and causes myocardial dysfunction that results in depression of systolic and diastolic blood pressure [17].

In our study we observed that after an industrial accident and exposure to chlorine gas poisoning the majority only developed mild symptoms and required no additional treatment while a small percentage of patients experienced more severe symptoms of the poisoning and required admission and supportive treatment. Almost all patients had ECG changes indicating the hyperacute T waves. Troponin I was not a prominent finding. As mentioned above previous studies showed some ECG changes in patients with chlorine poisoning but by far our knowledge none of the studies mentioned about hyperacute T waves in ECG. T waves represent ventricular repolarization and hyperacute T waves are usually present in coronary artery occlusion either by a mechanical obstruction or vasospasms. This change in ECG can be a predictor of outcome in patients of chlorine poisoning with myocardial dysfunction.

Due to a small sample size, the findings of the current study cannot be generalized to a larger population. Hence, future research should come up with better and more elaborated study designs keeping the current findings as a foundation and recruit a larger sample size from different tertiary care centers of Pakistan.

6. Conclusion

The present study indicated that there may be an unexplored association of severity of chlorine gas poisoning and cardiovascular damage as indicated by the changes detected on the electro cephalograms. It is further advised to perform an ECG in patients diagnosed with chlorine gas inhalation poisoning to observe any early manifestations of cardiac damage.

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