Progressive Vocal Cord Dysfunction Subsequent to a Chlorine Gas Exposure

*Patrick F. Allan, *Sahar Abouchahine, †Lee Harvis, and ‡Michael J. Morris

San Antonio, Texas

Summary: Chlorine gas inhalation, similar to other toxic gas exposures, can impart a variety of effects to the entire airway ranging from mucous membrane irritation to acute respiratory distress syndrome. The extent and location of damage is determined by numerous situational factors such as the duration of exposure, quantity of gas released, environmental factors, and instituted chemical defense measures. Reactive airways dysfunction and nonspecific bronchial hyperresponsiveness are commonly reported as sequelae to chlorine exposure. This article constitutes the first case of a single antecedent chlorine exposure inducing progressive vocal cord dysfunction.

Key Words: Chlorine—Airway irritant—Vocal cord dysfunction—Dyspnea.

INTRODUCTION

Dyspnea, cough, and chest pain are common manifestations of vocal cord dysfunction syndrome (VCD), which is a condition characterized by paradoxical vocal cord closure during inspiration. Laryngoscopy serves as the gold standard means of achieving the diagnosis through the visualization of vocal cord adduction during inhalation maneuvers.[1] Potential origins for VCD include gastroesophageal reflux disease, exercise, upper respiratory tract infections, asthma, and psychological factors. Ammonia, flux fumes, smoke, pungent odors, and dust are irritants that have also been associated with either the induction or exacerbation of ''irritant'' associated VCD (IVCD). It is unclear whether a single high-level or chronic low-level exposure to such agents gives rise to IVCD or, alternatively, results in discernable clinical aspects of a VCD expression of a patient.[2] Depending on chemical properties and situational factors associated with a single high-concentration exposure, such agents as nitrogen dioxide or ammonia have been noted to induce a variety of upper respiratory tract disorders such as rhinitis or ''reactive upper airway dysfunction syndrome'' and/or VCD.[3] We report the first case of a temporal association between exposure to a single incident involving a large-volume chlorine chemical spill and the eventual diagnosis of VCD.

Accepted for publication April 15, 2005.

The opinions or assertions contained herein are the private views of the authors and are not to be construed as reflecting the views of the U.S. Department of the Army, Department of the Air Force, or Department of Defense.

From the *Pulmonary Medicine Flight, Wilford Hall Medical Center, Lackland AFB, San Antonio, Texas; †Brooks City Base, San Antonio, Texas; and ‡Pulmonary/Critical Care Medicine, Brooke Army Medical Center, Fort Sam Houston, San Antonio, Texas.

Address correspondence and reprint requests to Michael J. Morris, Pulmonary/Critical Care (MCHE-MDP), Brooke Army Medical Center, 3851 Roger Brooke Drive, Fort Sam Houston, TX 78234. E-mail: michael.morris@amedd.army.mil

Journal of Voice, Vol. 20, No. 2, pp. 291–296
0892-1997/$32.00
© 2006 The Voice Foundation
doi:10.1016/j.jvoice.2005.04.003

CASE HISTORY

A 49-year-old Caucasian male firefighter was evaluated in the Pulmonary Clinic at Wilford Hall Medical Center for potential adverse health effects of a recent chemical spill. He had been in his usual good state of health without any symptoms of dyspnea, cough, wheezing, or chest pain until he responded to an incident involving the collision of two trains in a rural area near San Antonio. The collision led to the derailment of both trains and the release of approximately 15,000 gallons of chlorine gas. His role in the incident as first responder was the immediate evacuation of passengers and crew on the train and those civilians near the train collision area. Upon arrival to the collision site, he noted a large yellow cloud consuming the local area. He did not wear protective respiratory gear on initial arrival, but upon hearing of the potential chemical release, he donned a fire department-provisioned respirator during the estimated 6 hours of the operation. As the rescue effort continued, he noted a progressive eye and throat irritation. The heavy chemical suit and elevated daytime temperatures compounded by the demands and intensity of his activities eventually led to heat exhaustion. He was transported by emergency medical services to the Wilford Hall Medical Center Emergency Department (ED).

During transport and evaluation in the ED, he noted a worsening scratchy irritation in his throat accompanied by vocal hoarseness. He was eventually discharged home without receiving any directed therapy for his throat irritation. Over the ensuing 24 hours, his pharyngeal symptoms receded but he noted the appearance of yellow-white blisters on the back of his tongue and worsening dyspnea. He initially ignored the blisters, but after several days, he became annoyed by them and manually ruptured the lesions. A debilitating sense of exhaustion and fatigue superimposed on minimal dyspnea at rest pervaded his course from the day after ED discharge to our initial evaluation 3 weeks after his chlorine gas exposure. These symptoms significantly impaired his ability to participate as vigorously as he once had with firefighting activities.

At the initial pulmonary appointment 3 weeks after the incident, the patient demonstrated a normal forced expiratory volume in 1 second (FEV_1)/forced vital capacity (FVC) ratio on spirometry as well as normal lung volumes by plethysmography and carbon monoxide diffusion capacity. However, inspection of the flow volume loop (FVL) on spirometry revealed inspiratory loop truncation suggestive of variable extrathoracic obstruction. Questionnaire evaluation performed serially over the ensuing weeks disclosed worsening dyspnea related to the postchlorine-incident baseline (Table 1). Serial pulmonary function testing (PFT) in each of the subsequent 2 weeks showed worsening inspiratory loop truncation parallel to his dyspnea scores (Table 2). The 6-minute walk test (6MWT) distance of 1433 to 1386 ft was largely preserved over the same 2 weeks from his baseline assessment (Table 1). During the 3-week evaluation (6 weeks after the chemical spill), the patient presented with complaints of 3 days of acute on chronic (his postchlorine gas exposure baseline) dyspnea, and nonexertional, bilateral parasternal chest tightness. He did not complete a respiratory questionnaire during this visit. He could not comply with PFT, but his 6MWT had shortened to 1020 ft and he had to stop twice to

TABLE 1. *Serial 6-Minute Walk Testing (6MWT) and Dyspnea Questionnaire Results*

Timeline	Baseline (3 Weeks Postexposure)	Week 4	IVCD Diagnosis	Week 5 (Two Days After Acute VCD Episode)	Week 6
6MWT—distance	1433	1386	1020	1224	1055
6MWT—rest stops	0	0	2	2	0
6MWT—Spo2 nadir	96%	94%	96%	95%	94%
UCSD SOBQ (range 0–120)	69	74	ND	109	88

Abbreviations: Sp02, fingertip oxyhemoglobin saturation; SpO2, oxyhemoglobin saturation; UCSD SOBQ, University of California, San Diego shortness-of-breath questionnaire.
Notes: The UCSD SOBQ score ranges from 0 (no dyspnea when performing tasks) to 120 (severe dyspnea when performing tasks).

Download English Version:

https://daneshyari.com/en/article/1102456

Download Persian Version:

https://daneshyari.com/article/1102456

Daneshyari.com