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Odors and Scents Trigger Vocal Cord Dysfunction

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Abstract

Vocal cord dysfunction (VCD), is an extrathoracic upper airway disorder characterized by a paradoxical inspiratory closure of the vocal cords; it is not an intrathoracic lung condition. Some cases of VCD are solely recognized by an odor or scent without toxicological identification or quantitative documentation of an airborne exposure. In such cases, recognition of an odorant/scent causes the vocal cords' muscles to attain a spasmodic tight closure, especially during inspiration and sometimes during exhalation. Breathing against an obstructed glottis as well as hyperventilation from anxiety, panic and/or fear of personal harm worsens the laryngeal spasm. There is an accompanying voice change since normal vibrations of the edges of the two vocal cords are unattainable.VCD is identified by diagnostic studies such as spirometry, which reveals flattening of the inspiratory loop of the flow-volume curve. Making a correct diagnosis of VCD is imperative because there are unfavorable therapeutic and economic consequences including recurring emergency department visits, sustained corticosteroid and bronchodilator administration, multiple hospitalizations, and an unnecessary physician therapeutic intercession. Successful therapy and management of VCD requires an otolaryngologist assessment and/or speech therapy intervention.

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INTRODUCTION

The upper airways (i.e., nose, oral cavity, pharynx, and larynx) are divorced from the most distal intrathoracic bronchi/bronchioles/acini of the pulmonary system [1,2].The upper airways is where inhaled air is humidified, warmed, or cooled. It is imperative for human speech [3]. The extrathoracic location offers a pathway for inhaled oxygen to move downwards until reaching the distal lung acini where gas exchange takes place.

The extrathoracic glottis is that part of the larynx containing the vocal cords and the opening between them [4]. Abrupt spasmodic closure of the vocal cords occurs in the absence of local laryngeal disease occurs during an attack of vocal cords dysfunction (VCD) [2,5,6-10]. Two opposing glottic folds/cords attain a spasmodic closure during inspiration and at times during exhalation [8,9].The laryngeal obstruction temporarily ceases respiration and prevents external solid matter from air reaching the lower airways.

VCD is often misdiagnosed as bronchial asthma, an intrathoracic condition [11]. Inappropriate anti-asthma therapy is usually introduced. VCD is also mistaken for reactive airways dysfunction syndrome (RADS), a type of acute irritant-induced asthma, an intrathoracic disorder affecting the tracheobronchial tree [12]. RADS is always caused by a high-level/massive irritant gas, vapor, or fume exposure. Onset of symptoms occurs within 24 hours. Prompt medical help is a necessity.

A patient with VCD inaccurately receives treatment with asthma and/or anaphylaxis medications including aerosol bronchodilators, Epi-Pen injections, and oral or parenteral corticosteroids because of a mistaken diagnosis. More aggressive anti-asthma therapy does not correct the clinical manifestation of VCD. Reaching a correct diagnosis of VCD is imperative because there are adverse therapeutic and economic consequences such as repeated Emergency Department visits, multiple hospitalizations, continuous administration of medications, and unwarranted physician interventions including endotracheal intubation [13-15].

The precise pathophysiology of VCD is unknown but in certaininstances, the acute attacks of VCD are linked to an assumed exposure recognized by an odor or scent [13,17]. This causal connection represents the basis of this manuscript.

METHODS

Fourteen individual, seven women and seven men, ages 27 to 64 years old were assessed. Each person associated their acute respiratory complaints with an exposure recognized by an odor or scent. Detailed exposure and medical histories were taken. Physical examinations were conducted. Available Material Safety Data Sheets were surveyed. No quantitative air quality parameters, at the time of evaluation, were conducted. Normal appearing chest X-rays were assessed. Spirometry (FEV_1, FVC, FEV_1/FVC%, flow-volume curves, etc.) were analyzed. Endoscopy and methacholine challenges were achieved in several cases.

RESULTS

The mean age of men was greater (50 years old), compared to the mean age of the women (38 years old). All individual recognized an odoror scent, which triggered acute respiratory complaints. Besides difficulty in breathing, there was persistent coughing. Most of the afflicted described a discomforting feeling or a constrictive sensation in their neck or upper trachea. Occasionally, there was difficulty in swallowing. Voice was always altered; there was full loss, a change, or hoarseness of the voice.

The VCD attack was typically fleeting, lasting several minutes or less unlike asthma. Not all patients revealed inspiratory wheezing/stridor, especially if acute VCD was short lived. The chest examination became unremarkable during an asymptomatic period. Auscultation of the chest during an attack typically depicted inspiratory "wheezing" or "stridor."Routine chest X-rays were unremarkable. Spirometry was judged consistent with extrathoracic obstruction with flattening of the inspiratory loop of the flow volume curve. Visualization of the vocal folds by flexible, transnasal fiber-optic laryngoscopy and/or provocative testing was not employed in the present investigation. The claimed odorants causing VCD (in this study) are listed in Table 1.

DISCUSSION

Indicators connecting VCD to an odorant/scent trigger include the following: **1-**The patient perceives an odor or scent that triggers acute respiratory complaints. **2**-Crucial support for a significant airborne exposure is lacking. The precise constituent(s) of the exposure and its duration are lacking. There is no accurate knowledge as to how much of an airborne constituent(s) was/were delivered. Was it a massive airborne irritant exposure consistent with RADS?Uncovering chemical and physical properties of the alleged exposure (i.e., vapor pressure, pH, and degree of irritancy) is beneficial. There is help assessing a Material Safety Data Sheets (MSDS). **3**-There is ambiguous clinical data. If a substantial airborne irritant exposure occurs, then the sites of the eyes, nose, and throat are initially encountered. Eye tearing and injection is anticipated. The throat and nasal mucosal surfaces are inflamed and/or painful. The chest x-ray shows no parenchymal involvement. A normal % oxygen saturation lessens the likelihood of an intrathoracic target. Oxygen saturation will be reduced (≤ 94%) with an intrathoracic disorder. The clinical presentation of VCD is that of airway obstruction with inspiratory wheezing/stridor, breathlessness, and coughing. The latter combinations are easy to confuse with symptoms due to bronchial asthma. Vocal cords' closure resolves after a short-lived attack of VCD unlike asthma, which takes hours or days to resolve. The clinical criteria for a diagnosis of RADS are not met. There is an absence of an obstructive airways display by pulmonary function testing. Asthma and RADS are obstructive lung disorders. Spirometric measurement of $FEV_1$is reduced in proportion to a fall in FVC with VCD. FEV_1/FVC% is ≥70%.There is flattening of the inspiratory loop of the flow-volume tracing as shown in Figure 1

The low prevalence rate of vocal cord dysfunction hinders a clearer understanding of the entity. The exact mechanism explaining VCD is currently unknown. Table 2 lists origins of VCD published in the scientific literature.

When first recognized in the 19th century, vocal cord dysfunction was considered a disorder occurring among "hysterical" persons [18-20]. Nearly a century later, the condition of "Munchausen's stridor" [21] was coined for a 33 year old woman hospitalized 15 times for VCD. Reports depicted VCD as being a psychological illness, a factitious entity, a hysterical neurosis, or a somatoform disorder with "a loss of or alteration in physical functioning" [18-22]. In the 21st century, the pathophysiologic mechanism of VCD remains enigmatic with is no biochemical, physiologic, or structural abnormalities known to be consequential [8]. However, a perceived "exposure," recognized by the existence of an odor/scent, can trigger acute vocal cord spasm. It is imperative to recognized odorant induced VCD. Early recognition will limit frequent emergency department visits, numerous hospitalizations, uninterrupted administration of medications, and unnecessary physician interventions.

There is a higher rate of occurrence of acute VCD attacks among persons repeatedly visiting Emergency Departments due to sudden-onset shortness of breath [14]. The prevalence of VCD among children and adolescents, hospitalized because of asthma, is elevated [18,29,30]. Emergency care provider may

Table 1: Claimed Odorants Triggering Vcd.
"Moldy" odor claimed causing an office/building-related illness after visualization of "black" mold on surfaces (two cases).
Chlorine odor emitted from a swimming pool.
Odor developing during the use of a cleaning chemical that causes symptoms.
Scentemanating from distantly applied hydrochloric acid.
Smell noted while applying an odorous adhesive glue (not superglue).
Odor from ananhydrous ammonia exposure.
Smell during the application of a floor stripping chemical.
Odor following the discharged of a fire extinguisher containing ammonium-containing powder.
Odor originating from airborne nonspecific dust in a workplace.
Smell coming from remotely located welding fumes.
Unknown odor in a physician's office.
Residual smell declared present within an empty truck's interior.
Proclaimed odor stemming from steam in the air.

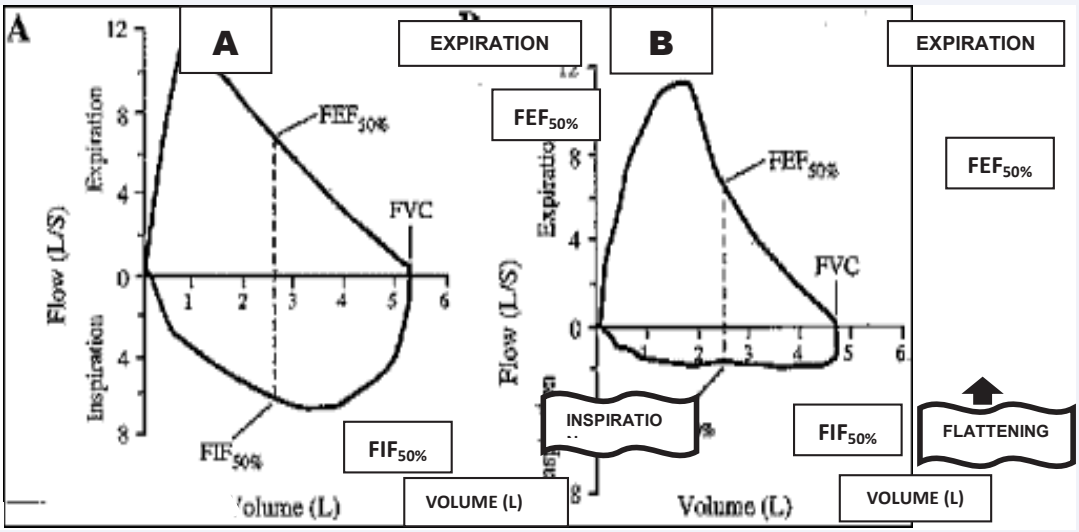

Figure 1 shows an example of a flow-volume loop in a normal subject (A) and in a patient with VCD (B). Note the blunting and flattening of the inspiratory loop of the flow-volume curve (arrow). Sometimes, there is a saw-tooth inspiratory flow pattern (6); the latter is not shown in the figure. The ratio of the spirometric forced inspiratory flow at 50% of the forced vital capacity (FIF50) divided by the maximal forced expiratory flow at 50% of forced vital capacity (FEF50) is reduced.

Table 2: Causes Associated With VCD.
Psychological/stress
Military recruits and active duty soldiers
Male & female athletes competing in cold outdoors
Elite cyclists
Exercise-related challenge
Methacholine challenge testing
Incorrect diagnosis of asthma
Incorrect diagnosis of reactive airways dysfunction syndrome (RADS)
Irritable Larynx Syndrome
GERD (Gastroesophageal Reflux)
Irritant-induced VCD
Sudden death occurrence in infants
Chlorine gas (Cl_2) inhalation
Water damaged building
Eucalyptus exposure
Smoke and particulates emitted from fires
Latex exposure
A worker in a corn field
Experience with episodic coup
Sodium metabisulphite in the fishing industry
Former World Trade Center rescue and recovery workers
Prolonged intubation
Central nervous system neurological disorder
Amyotrophic lateral sclerosis muscular dysfunction
Calcium deficiency
Post-operative complication
Occurrence after thyroidectomy
Happening after implanted vagal nerve stimulator
Administration of Botulinum toxin

inappropriately institute asthma therapies under theses clinical circumstances. Differentiating between asthma and VCD is crucial. Table 3 depicts differences between VCD and asthma.

4-There are always voice issues during an acute attack of VCD. Dysphonia, hoarseness, and/or impairment in the ability to produce voice sounds is because vocal folds do not oscillate effectively when they are under marked vocal folds' tension from VCD. **5-**A clinical unresponsiveness to therapies is observed. There is an inexplicable failure of clinical improvement even with sustained aggressive treatment when VCD is mistaken for asthma [6,9].

Transnasal fiber-optic laryngoscopy demonstrates approximation of the anterior two-thirds of the vocal cords with posterior, diamond-shaped chinking [6-9]. Provocative measures are required to verify VCD after a short-lived resolution of respiratory complaints. The patient is instructed to sniff, perform sequential phonation, undergo rapid panting, and take repetitive deep breaths [23]. Aerosolized methacholine, inhaled mannitol powder, accomplishing strenuous exercise on a treadmill or bicycle ergometer, breathing in refrigerated cold air, or inhaling a perfume or cleaning agents containing chlorine or ammonia are provocation approaches [23].

Perception of an odorant can trigger VCD. Smell is a phenomenon caused by simulation of the olfactory organs. This sensory manifestation is influenced by emotion and memory. Odors and scents elicit an acute VCD attack if there is a worrisome belief the environment is dangerous even when the concentration of an airborne chemical is far below the expected toxicity level [24-29]. Neural signals passing from the olfactory receptors to the olfactory cortex and other regions of the brain heighten the sensitivity of laryngeal reflexes [9,30,31].

VCD emerges among exercising recruits and active duty military personnel suspected of suffering from asthma

Table 3: Contrasting Facts between Asthma and VCD.

FACT	ASTHMA	VCD
SITE	intrathoracic; bronchi	extrathoracic; neck/throat;
DYSPNEA DEVELOPMENT	usually expiration but may be both inspiratory and expiration	usually inspiration; stridor; can be expiratory
WHEEZING	often expiratory	usually inspiratory
DURATION	variable, minutes, hours, or days	short, seconds to a few minutes
RESPONSE TO ODORANT	sometimes	usually
COUGH	during exercise	during a trigger
SPIROMETRY	expiratory airflow obstruction; decreased FEV_1/FVC%; inspiratory loop of flow-volume curve normal	"restriction; normal FEV_1/FVC%; inspiratory loop of flow-volume curve shows flattening
ENDOSCOPY	bronchial mucosal erythema, edema, and secretions; normal moving vocal cords	Adduction of the anterior 2/3 of the vocal cords; posterior chinking
INHALED DRUG THERAPY	effective	ineffective

[9,10,31]. US Olympic male and female athletes demonstrate exercise-induced inspiratory stridor when competing ina cold and dry ambient environment [32-36]. Competitive swimmers and swimming pool participants develop VCD [37-39].When the swimming pool disinfectant hat is used is added sodiumhypochlorite, the swimming pool water instantly produces innocuous hypochlorous acid not chlorine gas, which is rarely utilized for disinfecting swimming pools [39]. An exposure to chlorine gas does lead to VCD [40].There are reports of VCD precipitated by eucalyptus exposure, water damage buildings, irritants, from working in a corn field, and by former World Trade Center rescue and recovery workers and volunteers [41-44].

Symptoms of VCD significantly recuperate with interventional speech therapy [45]. Phonatory tests, video stroboscopy and laryngeal image analysis are tests available for the VCD investigation [46,47]. Psychological and psychiatric management can include behavioral, psychodynamic, and/or pharmacological treatment modalities. Marital or family counseling may be beneficial. For patients with a significant mood or anxiety disorder, antidepressant oranxiolytic treatment can be added to treatment.

CONFLICT OF INTEREST

I certify that I (Stuart M. Brooks, MD), am the guarantor and only corresponding author who holds no potential conflicts of interest; no sources of funding and support; no information on statistical analyses; correct name/participation/degree/ institution of the only author. I also certify that I did not accept compensation for inclusion of any of the statements contained in the manuscript.

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