Psychogenic Respiratory Distress: A Case of Paradoxical Vocal Cord Dysfunction and Literature Review

Raphael J. Leo, M.D., and Ramesh Konakanchi, D.O.

Background: Pulmonary disease such as asthma is a psychosomatic disorder vulnerable to exacerbations precipitated by psychological factors. A case is described in which a patient thought to have treatment-refractory asthma was discovered to have a conversion reaction, specifically paradoxical vocal cord dysfunction (PVCD), characterized by abnormal vocal cord adduction during inspiration.

Data Sources: Reports of PVCD were located using a MEDLINE search and review of bibliographies. MEDLINE (English language only) was searched from 1966 through December 1998 using the terms *functional asthma, functional upper airway obstruction, laryngeal diseases, Munchausen's stridor, paradoxical vocal cord dysfunction, psychogenic stridor, respiratory stridor, vocal cord dysfunction,* and *vocal cord paralysis*. A total of 170 cases of PVCD were reviewed.

Study Findings: PVCD appears to be significantly more common among females. PVCD spans all age groups, including pediatric, adolescent, and adult patients. PVCD was most often misdiagnosed as asthma or upper airway disease. Because patients present with atypical and/or refractory symptoms, several diagnostic tests are employed to evaluate patients with PVCD; laryngoscopy is the most common. Direct visualization of abnormal vocal cord movement is the most definitive means of establishing the diagnosis of PVCD. A number of psychiatric disturbances are related to PVCD, including conversion and anxiety disorders. PVCD is associated with severe psychosocial stress and difficulties with modulation of intense emotional states.

Conclusions: Psychogenic respiratory distress produced by PVCD can be easily misdiagnosed as severe or refractory asthma or other pulmonary disease states. Recognition of PVCD is important to avoid unnecessary medications and invasive treatments. Primary care physicians can detect cases of PVCD by attending to clinical symptoms, implementing appropriate laboratory investigations, and examining the psychological covariates of the disorder. Psychotherapy and speech therapy are effective in treating most cases of PVCD.

(Primary Care Companion J Clin Psychiatry 1999;1:39–46)

Received Feb. 19, 1999; accepted March 15, 1999. From the Department of Psychiatry, School of Medicine and Biomedical Sciences, State University of New York, Buffalo.

The authors thank Patricia Danaher, M.D.; Tim Fox, M.D.; and Constance Sherry, R.N., M.S., for their assistance in the preparation of this article.

Reprint requests to: Raphael J. Leo, M.D., Department of Psychiatry, School of Medicine and Biomedical Sciences, State University of New York, Erie County Medical Center, 462 Grider St., Buffalo, NY 14215.

Primary care physicians are at the forefront of managing asthma and respiratory distress. Referral to a pulmonology specialist is apt to occur when the physician confronts atypical symptoms and patients who are refractory to treatment. A subset of patients may display symptoms of respiratory distress with a psychological basis, requiring psychological interventions. The primary care physician can implement some of these interventions and be instrumental in ensuring appropriate psychiatric treatment.

Presented with Dora, a friend's daughter who suffered from dyspnea, Freud embarked upon explaining how physical symptoms were related to unconscious processes.[1] This perspective continues to influence the contemporary understanding of psychosomatic disease states. Pulmonary disease (e.g., asthma), while classically considered an example of a psychosomatic disorder vulnerable to psychological factors, can also present as a conversion reaction. Conversion disorders are characterized by alterations in physical functioning that are rooted in a psychological conflict and have no known physiologic basis. We describe a case of a patient with a history of treatment-refractory asthma whose symptoms could not fully be accounted for by asthma. Psychiatric evaluation was requested, and with corroborating data provided by pulmonary and otolaryngology evaluations, the diagnosis of a conversion reaction, i.e., paradoxical vocal cord dysfunction (PVCD), was made.

Normally, the vocal cords abduct during inspiration and adduct during expiration. PVCD is characterized by an abnormal inspiratory closure of the vocal cords resulting in stridor. Patients with PVCD exhibit respiratory distress that is often confused with upper airway obstruction or asthma. Cases of PVCD have been described in the medical literature as far back as 1842.[2] A syndrome of la-

ryngeal spasm affecting hysterical females was described and termed "hysterical croup." The treatment included throwing cold water onto the face of the afflicted patient while administering ammonia salts to the nose. Subsequently, the famed Austin Flint described "laryngismus stridulous" in 1868 as a syndrome occurring in "neurotic" adults and children.[3] The correct diagnosis of psychological mimics of disorders such as asthma or upper airway obstruction has obvious practical significance. While advances have been made in the treatment of PVCD since its description in 1842, an exploration of the demographic, psychological, and management issues associated with PVCD warrant attention in primary care circles.

CASE REPORT

Ms. A, a 32-year-old woman, was admitted to the Family Medicine service, Erie County Medical Center, Buffalo, N.Y., having experienced dyspnea for 2 days. The patient gave a history of asthma 2 years in duration. She had been treated with multiple inhalers and prednisone, 25 mg/day. In the past year, she had 5 brief hospitalizations for "asthma" exacerbations. She never required ventilator support or intensive monitoring in a critical care setting. She presented with prominent stridor, wheezing, tachypnea, and cough without expectoration. Despite diaphoresis and tachypnea, her vital signs were otherwise normal and stable, and there was no pulsus paradoxus. She appeared to use accessory muscles of respiration at times. Inspiratory and expiratory wheezes were audible over the lung fields, but were particularly prominent over the larynx. Pulse oximetry revealed an O_2 saturation of 95% on room air. Chest x-ray findings showed no abnormalities. Her symptoms resolved with intravenous hydration and nebulized albuterol.

Four days after admission, with news that discharge was imminent, Ms. A had another dyspneic episode. Her symptoms were so dramatic that she was intubated and admitted to the intensive care unit (ICU) under the care of a pulmonologist. Because her wheezing was no longer audible, she was extubated shortly thereafter. While in the ICU, she had several recurrent bouts of respiratory distress, each lasting for 3 to 4 hours. Despite nonresponsiveness to intravenous aminophylline or nebulized inhalers, arterial blood gas analysis revealed no hypoxia or hypercarbia. Episodes of respiratory distress appeared to abate with staff support and were exacerbated by conversations about recent psychosocial stressors. However, she breathed comfortably when asleep. Fiber-optic rhinolaryngoscopy performed by the otolaryngologist revealed inspiratory adduction of the anterior vocal processes with a small posterior glottic opening, consistent with PVCD.

Psychiatric consultation was requested. The patient had limited social supports. One of her main supports, her younger brother, had recently died; her significant other was recently incarcerated. She was ambivalent about her children and reported feeling "alone" raising them. She was distressed that her children were "too young and too distracted" to be concerned about her, e.g., "nobody seemed to care how sick I was." Physical symptoms appeared to intensify when her limited supports were discussed. She vehemently denied any association between the severity of the "attacks" and perceived abandonment. She denied that her attacks were in any way related to worry, phobias, obsessions, compulsions, or reexperiencing of prior traumas. She denied a prior history of panic disorder or agoraphobia. The duration of her attacks was in excess of what would be expected for a panic attack.

On psychiatric evaluation, she was noted to be sitting surprisingly comfortably in bed (not in prototypical "tripod" position of respiratory distress) with neck flexion, wearing a venturi mask for supplemental oxygen. She had an audible stridor, but no use of accessory muscles or obvious tachypnea. Her speech was whispered, often interrupted by stridor and breath holding. She was unwilling to participate meaningfully in the interview and appeared annoyed that a psychiatric evaluation had been requested. She went into elaborate explanations to excuse herself from participating in the psychiatric assessment, ironically alleging that she could not breathe adequately to speak.

She denied symptoms of depression, and indicated her mood was "good." Her thoughts were goal-directed, although she lacked spontaneity. She denied experiencing any delusions, illusions, or hallucinations. She lacked fluctuation of consciousness, and she was consistently attentive to her surroundings, ruling out the possibility of a delirium. There were no deficits in cognitive functioning on mental status examination; however, judgment and insight were poor.

She was treated with individual psychotherapy, including relaxation training. Therapy was directed at addressing the links between physical symptoms and her perceived level of abandonment and distress, fostering enhanced coping, and developing alternate means of mobilizing increased supports when she felt abandoned. She was also treated with speech therapy. She responded well to these interventions. She no longer required oxygen supplementation, inhalers, or steroids. There were no recurrences of dyspneic episodes. She was discharged with follow-up involving both psychotherapy as well as speech therapy. At 6-month follow-up, she continued to do well without any recurrences of dyspnea. While she no longer required speech therapy, she continued to participate in outpatient psychotherapy.

DATA SOURCES

Case reports and case series reporting vocal cord dysfunction were generated by a MEDLINE search and re-

view of the bibliographies from each article retrieved. MEDLINE (English language only) was searched from 1966 through December 1998 using the terms *functional asthma, functional upper airway obstruction, laryngeal diseases, Munchausen's stridor, paradoxical vocal cord dysfunction, psychogenic stridor, respiratory stridor, vocal cord dysfunction,* and *vocal cord paralysis*. The individual cases were then examined for consistency with a diagnosis of PVCD. Other cases depicting functional airway disorders not involving the cords, such as psychogenic pharyngeal constriction,[4] were not included in this review. Similarly, suspected cases of vocal cord dysfunction that were subsequently found to have a remediable organic etiology or that referred to functional disturbances mimicking asthma but were not explicitly defined as psychogenic were also not included.[5–8] Documents and articles that lacked descriptions of respiratory symptoms or specific patient cases were excluded.[9] Seventy-two articles were obtained, reporting 170 cases of vocal cord dysfunction.[10–81]

STUDY FINDINGS

Of the 171 cases of PVCD, including the present case, 39 referred to males, 132 referred to females. A chi-square analysis testing the hypothesis of equal frequencies of expected cases of PVCD among males and females in the 171 cases revealed statistically significant differences in observed frequencies ($\chi^2 = 26.08$, p = .000001).

The mean ± SD age for patients experiencing PVCD was 26.1 ± 13.5 years. For 10 patients with PVCD, ages were not reported.[52,75] The mean ± SD age for male patients was 22.9 ± 15.1 years and for females, 27.1 ± 13 years; however, these ages were not significantly different (t = 1.64, df = 159, p = .10).

PVCD presented with symptoms of wheezing and dyspnea suggestive of asthma in 74 cases (43.3%).[a] In 49 cases (28.7%), patients with PVCD presented with stridor suggestive of upper airway disease.[b] Less common modes of presentation included exercise-induced asthma or bronchospasm (N = 23, 13.5%)[32,37,41,43,54,60,61]; laryngeal spasm/stridor (N = 5, 2.9%)[11,57,58]; aphonia with upper respiratory infection (N = 3, 1.8%)[33,62,80]; anaphylaxis (N = 3, 1.8%)[54,66]; 2 cases each (1.2%) for episodic dyspnea,[21,24] acute respiratory distress,[38,65] and chronic obstructive pulmonary disease[54]; and 1 case (0.6%) each for postoperative respiratory distress,[12] upper respiratory infection,[33] rhinitis,[60] and hoarseness.[54] In 4 cases (2.3%), the presumed clinical diagnosis was not specified.[44,63,72]

Patients were treated with several medication regimens for presumed asthma and upper airway obstruction. The medications employed consisted of aminophylline/theophylline (N = 21, 12.3%),[a] β-agonists and bronchodilators (N = 67, 39.2%),[b] steroids (N = 56, 32.7%),[c] ipratropium (N = 3, 1.8%),[26,51] cromolyn sodium (N = 10, 5.8%),[13,15,35,36,43,60,65] epinephrine (N = 11, 6.4%),[d] and antihistamines (N = 8, 4.7%).[43,54,60,66,70] Botulinum toxin injection was employed in 2 cases (1.2%).[36,39] In 29 cases (17%), intubation or tracheostomy was employed.[e] In 1 case, intubation was employed 13 times in a 10-year span[23]; and in 2 cases, tracheostomy was maintained 19 months before decannulation was possible.[11,74] Two patients required permanent tracheostomies,[29,52] and 1 patient required surgical intervention to improve vocal cord movement.[39]

The time between initial presentation and final diagnosis of PVCD was determinable in 96 cases.[f] The mean latency until diagnosis of PVCD was 2.7 years (range, 2 hours[27] to 13 years[17]).

Diagnostic testing commonly included pulmonary function testing in 108 cases (63.2%),[g] laryngoscopy in 126 (73.7%),[h] bronchoscopy in 43 (25.1%),[i] fluoroscopy in 2 (1.2%),[49,68] methacholine challenge tests in 44 (25.7%),[j] and histamine-, acetylcholine-, exercise-, or cold-induced provocation tests in 24 (14%).[19,20,43,51,54,60,62,78]

Otolaryngology evaluation was obtained in 26 cases (15.2%),[k] and neurology consultation was obtained in 9 cases (5.3%).[11,16,21,27,30,36,72] Psychological or psychiatric evaluation was requested in 84 cases (49.1%). Psychiatric diagnoses were varied and included conversion reactions (N = 20, 11.7%),[l] anxiety disorder (N = 18, 10.5%),[m] histrionic and other personality disorders (N = 11, 6.4%),[n] parent-child and other family/school conflicts (N = 7, 4.1%),[10,11,36,63] depression (N = 6, 3.5%),[13,14,31,37,47] psychosomatic disorder (N = 4, 2.3%),[25,42] factitious disorder (N = 4, 2.3%),[41,47,57,74] and somatization disorder (N = 2, 1.2%).[74] In 12 cases (7%), no formal psychiatric diagnosis

[a]References 10, 13, 14–17, 19, 20, 22, 23, 25, 26, 29, 31, 35, 40, 41, 45, 47, 49, 51–55, 59, 60, 66–68, 75, 77, 78, 81.
[b]References 21, 27–29, 31, 33, 34, 36, 39, 56, 64, 66, 69, 70, 71.
[a]References 13, 14, 17, 20, 29, 35, 38, 42, 43, 49, 50, 57, 60, 62, 65, 69, 79.
[b]References 10, 13, 15, 17–23, 26, 28, 29, 34–37, 41–45, 47, 49, 51–53, 56, 59, 60, 62, 65–68, 75, 77, 81.
[c]References 10, 12, 17, 19, 20–23, 29, 35, 37–39, 42, 44, 45, 47, 49, 51–55, 60, 62, 64, 66–70, 78, 80, 81.
[d]References 12, 30, 38, 45, 48, 54, 56, 57, 72, 77, 81.
[e]References 11, 12, 16–18, 23, 27–29, 30, 33, 38, 39, 46, 48, 50–52, 66, 71, 74, 78, 79.
[f]References 10–12, 14–20, 22, 23, 25–29, 31–36, 38–45, 47–49, 51, 56–58, 60, 62–72, 74, 77–81.
[g]References 10, 11, 13, 14, 16–20, 22–26, 28, 29, 31, 32, 35, 37–43, 45, 47, 50–52, 54, 56–58, 60, 62, 66–70, 74, 75, 78, 79, 81.
[h]References 11–21, 23, 25–39, 41, 42, 45–50, 54, 57–67, 70–76, 78–80.
[i]References 10, 11, 13, 16, 18, 22, 24, 28, 34–36, 38–40, 46, 47, 50–53, 56–58, 62, 63, 69, 74, 76, 78, 79, 81.
[j]References 16, 17, 20, 22, 24–26, 35, 37, 38, 43, 47, 48, 54, 60, 66, 75.
[k]References 12, 14, 21, 26, 31, 33, 36, 46, 49, 53, 57, 59, 61, 63, 66, 71, 73, 76, 79, 80.
[l]References 17, 21, 26, 27, 34, 41, 47, 56, 66, 70, 79, 80.
[m]References 10, 15, 20, 30, 31, 35, 37, 38, 62, 73, 75, 77.
[n]References 11, 18, 22, 34, 37, 64, 67, 74, 79.

was discernible, although psychological precipitants to PVCD were implicated.[10,27,54]

Once asthma and upper airway obstruction were ruled out, a variety of treatment measures were employed in the cases reviewed here. Administration of a helium-oxygen mixture was employed in 11 cases (6.4%),[12,16,17,19,28,36,40] but was ineffective in 1 case.[36] Placebo treatments, such as saline injections, were employed and effective in 2 cases (1.2%).[33,56] Treatment often consisted of psychotherapy and speech therapy. Speech therapy was invoked in 42 cases (24.6%).[a] However, speech therapy alone was deemed ineffective in 6 cases (6/42, 14.3%)[16,20,29,51] and partially effective in 3 cases (3/42, 7.1%).[51,71,81] Psychiatric interventions took the form of pharmacotherapy (N = 34, 19.9%),[b] individual psychotherapy (N = 45, 26.3%),[c] family therapy (N = 2, 1.2%),[13,26] pharmacotherapy plus psychotherapy (N = 14, 8.2%),[14,16,29,30,47,50,70,79] and psychiatric inpatient care (N = 2, 1.2%).[34,66] Individual therapy took the form of relaxation training in 5 cases,[45,48,50,54] biofeedback in 4 cases,[29,36,66,70] behavior modification in 7 cases,[75] and hypnosis in 2 cases.[29] Psychiatric referral was made in 6 cases (3.5%), but there was no mention of whether patients followed through with therapy. Psychiatric intervention was offered to 12 additional patients (7%), but was declined.[d]

DISCUSSION

PVCD, produced by abnormal vocal cord adduction during inspiration, presents with signs of respiratory distress suggestive of airway obstruction or asthma. The alarming features of respiratory stridor, wheezing, tachypnea, and the fear of respiratory failure naturally prompt a number of aggressive interventions and investigations. Once physical abnormalities have been appropriately ruled out, attention is directed at determination of the "functional" basis for the disorder.

PVCD can occur in children,[e] adolescents,[f] and adults.[g] In the cases reviewed here, females were represented significantly more often than males, suggesting that PVCD predominantly affects females. However, one can only speculate on the influence of gender. Certainly, this finding is consistent with the observation of an increased prevalence of conversion disorders, somatoform disorders, and anxiety among female patients.[83,84] On the other hand, the significantly different gender frequencies observed here may simply reflect biases in diagnosis, with fewer males recognized as demonstrating psychogenic respiratory distress or asthma.

Often, such patients come to medical attention through the emergency room[21,30,31,40,59,73,79] or contact with internal or pediatric medicine, including critical care settings as well as pulmonary clinics.[a] As in our case, such patients often have frequent hospital presentations.[b] Occasionally, episodes of functional vocal cord disturbances are encountered postoperatively.[12,28,46,61,76,80]

For many cases cited here, there is a long latency between symptom onset and diagnosis of PVCD. Several exacerbations and remissions characterize the disorder. For some, the episodes are short-lived, respond favorably to brief interventions, and are merely presumed to reflect asthma exacerbations, anaphylaxis, and so on. Only atypical patterns of symptoms, variable or poor response to treatment, and/or multiple exacerbations prompt further investigations leading to the diagnosis of PVCD.

Patients with PVCD are often misdiagnosed with refractory asthma. PVCD is suspected when inconsistencies with the clinical picture of asthma are encountered. As in our case, PVCD is differentiated from asthma by the absence of nocturnal symptoms (in the course of a physical abnormality, the relaxed vocal apparatus would be expected to worsen breathing ability)[13,19,31,37,69]; localization of wheezing to the upper chest and throat (which disappears with intubation)[70,77]; lack of sputum production[9,63]; and often, although not always,[11,16,48,85] normal blood gas values despite marked symptoms of respiratory distress.[19,38,56] Patients may demonstrate neck flexion (in contrast to the extension often encountered in asthma)[34] and speak in complete sentences despite the obvious respiratory difficulties.[34,42] PVCD is suggested when airway difficulties are precipitated by suggestion[34] and when patients demonstrate exacerbations related to stress. Psychogenic causes are also supported by symptoms that abate with distraction, support, and reassurance or placebo use.[33,56,73,76]

The failure to respond favorably to conventional treatments, e.g., bronchodilators and steroids, or invasive procedures, also suggests a functional etiology.[9] It should be noted that lack of response to conventional treatments has led to intensification of treatment, invasive studies, and intubation or tracheostomy. It is noteworthy that in 14 cases (8.2%), patients reportedly experienced toxic effects of steroids, were cushingoid, or were steroid-dependent.[c]

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[a]References 10, 13, 16–20, 23, 29, 32, 35, 41, 48, 51, 52, 55, 60, 65, 67, 71, 77, 78, 81.
[b]References 12–14, 16, 18, 22, 28–30, 33, 36, 42, 47, 50, 51, 53, 57–59, 61–63, 66, 70–74, 79, 81.
[c]References 17, 20, 25, 29, 31, 36, 43, 45, 48, 52, 54, 56, 57, 60, 62, 66, 67, 75.
[d]References 18, 20, 25, 34, 35, 43, 56, 69, 71.
[e]References 10, 21, 31, 32, 37, 38, 41, 44, 45, 49, 56, 59, 63, 71, 73.
[f]References 10, 13, 15, 17, 18, 21, 26, 31, 33–37, 40, 41, 43, 47, 50, 51, 53, 54, 56, 63–65, 67–69, 71–73, 77, 78, 81, 82.
[g]References 11, 12, 14, 16–20, 22–25, 27–30, 33, 35, 39, 42, 43, 46–48, 50–52, 54, 55, 57, 58, 60–62, 64, 66, 70, 71, 73, 74, 76, 78–81.

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[a]References 10, 13, 16, 19, 23, 24, 26, 32, 38, 43, 49, 50, 54, 62, 67, 70, 77, 78, 81.
[b]References 9, 14, 15, 19, 22, 25–27, 29, 30, 36, 38–40, 42, 45, 47, 48, 51, 62, 66, 70, 71.
[c]References 17, 19, 29, 35, 37, 42, 52, 62, 70, 81.

While diagnosis of PVCD has been possible based on clinical grounds, corroborating evidence is generally obtained through laboratory investigations. The absence of immunoglobulin and complement levels argues against allergic reactions.[13,57,70] The absence of characteristic abnormalities on spirometry refutes asthma, chronic obstructive pulmonary disease, and upper airway obstruction.[22,31,38,57,62] Furthermore, provocative tests can clarify psychogenic etiologies; for example, methacholine- or histamine-challenge testing would induce bronchospasm in asthmatics, but would fail to do so for patients with PVCD.[17,19,43,65] The most definitive evaluation comes from direct visualization of abnormal vocal cord movement during endoscopic examination.[17,36,42,43,49,56,78] When laryngoscopy is impossible, an alternative, i.e., ultrasonography, can be a less invasive means of demonstrating abnormal vocal cord movement.[67,86]

In the postoperative setting, a functional basis for respiratory symptoms is established if symptoms of vocal cord adduction are relieved by anesthesia. Anesthesia would lead to worsening of symptoms in upper airway disease or asthma.[16,74] Helium-oxygen therapy is not effective in the treatment of asthma and/or lower airway disease states. Yet, the effect of that treatment in reducing symptoms of PVCD is profound and aids in clarifying the diagnosis.[12,16,17,19,28,40] The expedient response of reduced respiratory distress in patients experiencing psychogenic respiratory distress may help to foster an understanding or recognition of the relationship between psychological correlates and PVCD.

Once a psychogenic cause is suspected, the beleaguered clinician may be apt to impugn the character and motives of the "difficult" patient. Although malingering may be suspected, the literature suggests that PVCD patients are unable to produce vocal cord adduction voluntarily.[17–19,24,56,66] Furthermore, feigned results on spirometry and laryngoscopy cannot be consistently replicated.[17,24] Yet, controversy attends this matter, since some patients with PVCD were considered to derive secondary gains or needed to be in "the sick role," e.g., to have factitious asthma.[41,47,57,74] One patient was reportedly capable of reproducing symptoms of respiratory distress at will when under duress.[68]

Psychiatric involvement is generally requested through consultation and/or psychiatric referral. Establishing a link between PVCD and certain psychiatric diagnoses is not possible given the limited psychiatric information provided in the cases cited here. Psychiatric diagnoses were varied, with conversion disorder the most commonly diagnosed. As is the case for PVCD, conversion is suspected when the physical findings, specifically respiratory difficulties, are not consistent with known neuroanatomic and physiologic principles. Clearly, respiratory distress and associated difficulties with speaking can serve the function of primary gain in temporarily resolving the conflicts of otherwise expressing anger or other unpleasant emotional states. In our case, Ms. A's respiratory distress and "inability to speak" bypassed unacceptable feelings, e.g., her ambivalence about the children and anger about the perceived lack of support from available sources. Despite overt respiratory distress, which would naturally be associated with alarm and psychological distress, our patient reported that her mood was "good" and often appeared to demonstrate an affective blandness to her situation. Similarly, in other cases cited here, situational conflicts were temporally related to respiratory disturbances and associated with indifference.[50,51,63,64]

Diagnosis of conversion becomes particularly difficult in those cases where PVCD was present in a patient with a history of confirmed asthma.[9,23,29,52,67,78] Such cases would be viewed as paralleling conversion reactions in other disease states, such as pseudoseizures occurring among patients with seizure disorder.[87] In our case, the diagnosis of asthma, with which the patient reported to have been diagnosed previously, was no longer entertained. It is not presumed that she deceived, but was in fact told this by previous treating sources. Based upon her history, prior episodes apparently distracted her from stressors and may have dissipated the stress of internal conflicts. In addition, she derived some clear secondary gains, for example, support from family.

Other psychiatric conditions should be entertained in the differential diagnosis of PVCD. The dyspnea, tachypnea, respiratory difficulties, and other similar symptoms (e.g., "a catch in the throat")[10] suggest panic or other acute anxiety disorders. It should be noted that some observers would disagree with the notion of a purely psychogenic basis for PVCD, despite inability to fully account for PVCD based upon physical findings.[5] In such cases, a diagnosis of "psychological factors affecting medical conditions" could be invoked, in that reactions to stress serve to exacerbate or mimic symptoms and/or render the individual "refractory" to treatment.[44]

PVCD is speculated to be associated with prior traumas, e.g., physical/sexual abuse,[9,14,25,77] and psychosocial stressors,[a] e.g., abandonment fears and family disputes. Of the cases reviewed here, some patients were characterized with difficulties with expression of, and modulation of, intense emotional states.[14,17,26,31,44,47,74] Hence, it is not surprising that patients with PVCD were characterized as dependent,[13,26,31,34,38,70,74] immature,[26,64,67] histrionic,[11,20] borderline,[74] narcissistic,[79] perfectionistic,[66] obsessional,[17,35] or as having feelings of inadequacy.[63] Symptoms such as those associated with PVCD can serve to express intense emotions indirectly.[47] For example, cases of PVCD had been related to the distress associated with

[a]References 10, 11, 26, 36, 41, 44, 45, 50, 54, 62–64, 66, 70, 72.

perceived parental expectations,[10,63] may serve to restore equilibrium in an otherwise chaotic home life,[11,27,36,50] may express feared abandonment or ambivalence about perceived dependency,[70] and may represent repressed anger.[31,47] Removed from the sources of conflict, the symptoms of PVCD no longer serve a purpose in the hospital setting. Interestingly, in our case the symptoms abated quickly with hospitalization, but dramatically recurred with an anticipated discharge.

Primary care physicians can assist the patient to recognize the relationship between symptoms and psychological distress and facilitate acceptance of the diagnosis of PVCD. With this acceptance, the patient may be amenable to speech therapy and psychotherapy instead of vain attempts at pursuing intensification of "conventional" treatments.

In cases where psychiatric intervention is not immediately available and only referral to a psychiatrist is made, efforts should be made to ensure follow-up. Patients may be unable or unwilling to relinquish the diagnosis of a "purely" physical disorder. Clearly, several reports indicate that patients were resistant to the idea of the psychogenic origins to their respiratory difficulties. In some cases, patients refused speech therapy and/or psychotherapy, abandoned treatment, or sought treatment elsewhere with clinicians who perpetuated the notion of a physical disorder.[35,43] The problem may be related to the manner in which psychological underpinnings to the physical symptoms are explained; for example, the patient accused of feigning symptoms is likely to be less than receptive to psychotherapy. Any "confrontation" of patients would need to invoke ego strengths, which allow for "face-saving" symptom relief. In addition, family members and support systems may be invested in insisting upon a physical condition since exploration of psychological covariates may lead to an indictment of problems in those relationships.

In the acute setting, the primary care physician can educate the patient to employ panting when dyspneic episodes recur since doing so is effective in relieving PVCD.[58] In addition, placing the patient in the "sniff" position can also facilitate improved breathing, particularly if the patient tends to assume neck flexion during acute episodes.[76] The role of distraction warrants staff education as well. Anxiolytics and other psychotropics, such as antidepressants, may also be indicated,[a] although the role of psychotropics has as yet to be determined.[82] The clinician may need to educate the patient about the psychological underpinnings of PVCD. Education of families, other supports, and medical personnel may reduce the resentment and negativity conveyed by them toward the patient, which could predispose the patient to distress and may perpetuate symptoms. Lastly, patients may need assistance with secondary disturbances arising from the diagnosis of PVCD. Some patients, when learning that the respiratory distress is functional as opposed to asthma or another condition, experience confusion, doubt, depression, and a sense of loss.[89,90]

Psychotherapy may help to clarify and remedy some of those factors perpetuating and maintaining symptoms of PVCD. In many of the reports reviewed here, the descriptions of the extent of, and issues related to, therapy were often not described. Psychotherapeutic interventions have, according to the literature, varied in terms of modality, duration, frequency, and efficacy. Modes of therapy included supportive therapy,[36] hypnosis,[29,72] biofeedback training,[29,36,66,70] and marital and family therapy.[13,26] Through biofeedback and self-hypnosis, patients can recognize heightened distress and relax themselves. The presumption is that it is impossible to be simultaneously aroused and relaxed, so that relaxation thereby mitigates symptom severity or precludes the full spectrum of PVCD symptoms. In some cases, the efficacy of psychotherapy was questionable,[20,29,75] but in other cases, the effect was remarkable, notable after one or two sessions.[15] For most patients, however, the interventions required extended therapy.

Speech therapy is effective in treating PVCD as well. The speech pathologist encourages the patient to employ abdominal or diaphragmatic breathing and eliminate shallow breathing; relax the throat during quiet respiration; relax the neck, shoulders, and chest; and interrupt sensations stimulating cough by swallowing and relaxed breathing.[17,91] The efficacy of speech therapy is thought to involve strengthening the vocal apparatus. Similar to biofeedback, the substitution of exercises when vocal cords adduct, e.g., panting and diaphragmatic breathing, can mitigate or prevent episodes of respiratory distress.

As with psychotherapy, speech therapy did not always render patients symptom-free[16,20,29,51] and was only moderately effective for others.[51,71,81] In part, this may have been due to patient refusal or lack of compliance with treatment. Long-term outcome assessments in the cases reported in the literature were often lacking. In other cases, patients unresponsive to speech therapy responded to psychotherapy instead.[13,51] It appears that, for most cases, treatment response is best when speech and psychotherapy are combined.[92] It should be noted that PVCD could persist on a long-term basis despite speech and psychotherapy.[29]

PVCD is not a benign condition, and its psychological underpinnings are complex. The recognition of PVCD is important to avoid unnecessary medications, prevent intubation and tracheostomy, and to secure appropriate psychiatric and speech therapies.[9,17,23,93] PVCD presents a diagnostic and treatment dilemma to the primary care physician. Further collaboration with psychiatric consul-

[a]References 13, 14, 18, 22, 33, 62, 63, 79, 88.

tants is required to identify cases of PVCD and clarify the psychological correlates of the disorder. The literature is limited by a lack of prospective studies comparing patients with PVCD with control subjects to allow for clarification of causal psychosocial and psychological variables. In addition, establishing psychiatric diagnosis and, more importantly, effective treatment for PVCD will be possible. Consultation with the psychiatrist and speech pathologist can be pivotal to symptom resolution and prevention of recurrences.

Drug names: cromolyn (Intal and others), ipratropium (Atrovent and others), prednisone (Sterapred and others).

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