Chronic Subjective Dizziness

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KEYWORDS

• Dizziness • Vertigo • Anxiety • Migraine

The symptom of dizziness represents a nonspecific complaint that has a broad differential diagnosis. Otolaryngologists naturally focus on the inner ear as the source of the complaint. However, if a patient with dizziness is to be diagnosed accurately and treated effectively, then it is incumbent on the clinician to recognize that dizziness can also be a manifestation of underlying neurological, cardiovascular or psychiatric pathology. In addition, it has long been recognized that psychopathology can produce a sensation of dizziness.[1,2] It is helpful to approach a patient complaining of dizziness in a manner analogous to the approach taken when evaluating a patient presenting with chest pain. Although the initial reflex may be to focus on a cardiac etiology; gastrointestinal, musculoskeletal, and psychologic pathology may also result in chest pain.

It cannot be overemphasized that eliciting a precise description of the dizziness from the patient is the critical factor in delineating the specific diagnosis.[3,4] The specific symptoms described by the patient allow the clinician to categorize the of dizziness (eg, true vertigo, lightheadedness, presyncope, positional imbalance, ataxia); the severity of the dizziness; factors that provoke or ameliorate the dizziness; and any associated symptoms. At times, this description of dizziness is precise and easy to elicit. There are patients, however, who despite the interviewer's best efforts can only describe a vague sensation that defies a precise medical definition. Rather than become frustrated with the patient's inability to articulate a clear description of his or her dizziness, Barber[1] pointed out that such vague symptoms may be evidence, within the first few minutes of the interview, of a psychiatric etiology. Such vague symptoms of chronic heavy headedness, lightheadedness, tightness in the head, and the floor rising and falling are the hallmark of what he and others referred to as ''psychogenic dizziness.''

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Recognizing that psychiatric factors may have an important role in certain symptoms of dizziness is an important step in counseling patients and avoiding unnecessary medical and surgical procedures. This level of understanding of the disease process, however, has certain limitations. Too often patients are left with the impression that psychogenic dizziness is a diagnosis of exclusion. Simply referring to a process as psychogenic provides no insight as to which of the myriad of potential psychiatric diagnoses are responsible for the symptoms. Furthermore, psychiatric processes may be a cause or consequence of dizziness. They may trigger sensations of dizziness or sustain chronic symptoms following transient medical events. Because successful treatment is predicated on an accurate diagnosis, the need for better diagnostic precision is required.

Approximately 10 years ago the authors set out to better define the entity that was referred to as ''psychogenic dizziness.'' To that end, they endeavored to (1) provide an accurate and reproducible set of diagnostic criteria; (2) delineate the underlying etiologies for the disorder (eg, provide specific psychiatric and medical diagnoses and understand their potential interactions); (3) establish an effective treatment strategy for these patients; and (4) provide a theoretic framework for understanding this disorder that facilitates future research. Their work has resulted in the definition of a clinical entity they refer to as ''chronic subjective dizziness'' (CSD).

CHRONIC SUBJECTIVE DIZZINESS: SYMPTOMATOLOGY

Patients diagnosed with CSD present with a similar symptom complex:[5,6]

1. Persistent (>3 months) sensation of nonvertiginous dizziness that may include one or more of the following vague descriptors
   a. Lightheadedness
   b. Heavy headedness
   c. A feeling of imbalance that frequently is not apparent to others
   d. A feeling that the ''inside of their head'' is spinning in the absence of any perception of movement of the visual surround
   e. A feeling that the floor is moving from underneath them
   f. A feeling of disassociation from one's environment
2. Chronic hypersensitivity to one's own motion or the movement of objects in the environment
3. Exacerbation of symptoms in settings with complex visual stimuli, such as grocery stores or shopping malls, or when performing precision visual tasks (eg, working on a computer)

DEMOGRAPHICS

The age range of patients diagnosed with CSD spans from adolescence to late adulthood, but patients are typically between 40 and 50 years of age. Most of the patients (65% to 70%) are women.

CHRONIC SUBJECTIVE DIZZINESS: PATHOGENESIS

Most patients with CSD (93%) have a psychiatric disorder that contributes significantly to their symptoms.[5–8] Based on current classifications in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders*, anxiety disorders are by far the most common psychiatric pathology identified, including generalized anxiety disorder, panic or phobic disorders, or minor anxiety (ie, anxiety not otherwise specified in *Diagnostic and Statistical Manual of Mental Disorders-IV*). In a small minority of

patients, additional psychiatric pathology was identified including depression, post-traumatic stress disorder, hypochondriasis, and conversion disorder.

The Relationship of Chronic Subjective Dizziness and Other Neuro-Otologic and Neurologic Disorders

The accurate diagnosis of a patient with the complaint of dizziness is dependent on obtaining a precise history and description of the symptoms. It was found that CSD often occurred in patients with a history of a physical neuro-otologic illness (eg, vestibular neuronitis or benign positional vertigo) or neurologic disorder (eg, migraines, postconcussional syndrome).[6] It is particularly important for the clinician to differentiate between symptoms of active neuro-otologic disease (eg, vertigo) and symptoms of CSD (eg, chronic nonvertiginous dizziness). Making this distinction is of immense practical significance. For example, a patient with known Meniere's disease may present complaining of persistent dizziness. Given the patient's known diagnosis, a superficial history of the present complaint may lead the clinician to recommend a vestibuloablative therapy for refractory Meniere's disease. This is more than acceptable if the patient is suffering from vertigo. However, if the patient has actually developed more vague and persistent symptoms consistent with CSD (not an uncommon scenario in patients with Meniere's disease), then vestibuloablative therapy that induces a unilateral vestibular loss could increase his or her anxiety and symptoms. Such a patient might feel worse after treatment, making for an unhappy patient and a frustrated physician. Similarly, in a subset of patients, an episode of vertigo as experienced in benign positional vertigo or vestibular neuronitis may trigger an anxiety response with prolonged symptoms of CSD long after the actual vertigo has resolved.[9] These symptoms persist unless the underlying psychopathology is addressed.

The authors studied the relationship of CSD with other neuro-otologic and neurologic disorders and were able to derive the following classification system.[6,8]

CHRONIC SUBJECTIVE DIZZINESS WITH ANXIETY

A minority of patients with CSD have a primary anxiety disorder. Most have anxiety disorders that are triggered by or coexist with a neuro-otologic illness. The authors found that patients with CSD had one of the following three patterns of presentation.

Otogenic Chronic Subjective Dizziness

These patients had no history of anxiety disorder before developing an acute vestibular insult or other similar pathology (eg, vestibular neuronitis, benign positional vertigo, transient ischemic attack). Their neuro-otologic illness precipitated the onset of anxiety.

Psychogenic Chronic Subjective Dizziness

These patients had no physical disorders including no history of a vestibular disorder. They developed dizziness during the course of their primary anxiety disorder.

Interactive Chronic Subjective Dizziness

These patients had a history of an anxiety disorder or diathesis before the onset of any symptoms of dizziness. They developed CSD and a worsening of their anxiety disorder subsequent to an acute and transient episode of true vertigo or medical condition causing dizziness.

The authors' results indicated that patients were distributed equally between these groups. They reinforce the concept of a bidirectional relationship between dizziness

and anxiety: dizziness can cause anxiety and anxiety can cause dizziness. Only one-third of these patients had pure anxiety disorders with no history of physical vestibular disorders. Nevertheless, it must be emphasized that in all these patients, psychiatric processes played the principal role in sustaining both symptoms and functional impairment. Regardless of the subgroup, addressing the psychiatric symptoms in these patients is the key to therapeutic success.

CHRONIC SUBJECTIVE DIZZINESS WITH MIGRAINE

Patients with migraines have an increased incidence of vestibular complaints and anxiety disorders.[10] It was found that close to 20% of patients with CSD had active migraines.[6] This is in keeping with other studies looking at the association between migraines, vestibular complaints, and anxiety. At present, there is general agreement that in patients with these overlapping symptoms, therapeutic interventions must be directed at all the contributing factors, including the migraine headaches, the anxiety disorder, and the vestibular complaints.

CHRONIC SUBJECTIVE DIZZINESS AND OTHER NEUROLOGIC DISORDERS

Patients with postconcussional syndrome represent an enormously challenging group of patients that present with a variety of complaints, including chronic dizziness, depression and irritability, headache, insomnia, and difficulties with memory and concentration. Fifteen percent of the authors' patients with CSD had traumatic brain injury.[6]

Patients with dysautonomias represent a small but significant subgroup of the CSD population.[6,11,12] These patients who have alterations in central neurovascular control can manifest symptoms typical of CSD. At initial evaluation, they can be differentiated from the other subgroups based upon the stimuli that provoke their symptoms. Dysautonomia patients develop CSD with exertion (eg, when engaging in some form of aerobic exercise). Environments that provoke symptoms in CSD with anxiety patients (grocery stores, shopping malls, and so forth) are not as likely to evoke symptoms in patients with dysautonomias.

TREATMENT OF CHRONIC SUBJECTIVE DIZZINESS

Patients with CSD are frustrated and even, at times, desperate. They feel chronically ill and have invariably seen a number of physicians with no benefit. They have tried a variety of medications and other interventions to no avail. They do not receive benefit from traditional interventions for vestibular disease and, as such, some may believe that there is little that can be done to treat their condition. It is in this sense that their treatment may initially be considered palliative. With the interventions outlined below, however, most patients can see a dramatic improvement and even eradication of their symptoms.

Psychoeducation

This is a critical first step in the successful treatment of these patients.[6,13] Many of these patients believe that they have a physical disorder and are reluctant to accept the concept that their symptoms stem from a psychiatric process. As such, a significant period of time must be spent at the initial encounter to educate the patient as to why and how psychologic disease can produce and sustain physical symptoms. This is best performed by a medical professional who is familiar with both the medical and psychiatric aspects of these issues and can spend the required time with the patient.

Putting into place a proper patient education process is critical to the success of the other interventions described next.

Pharmacologic Interventions

The selective serotonin reuptake inhibitors are currently considered to be the first-line therapy for anxiety disorders. The authors and others have evaluated the effects of the selective serotonin reuptake inhibitors on patients with CSD. In a series of open label prospective studies, they found these drugs to be effective in the treatment of CSD.[8,14,15] Roughly 50% of patients studied had a complete remission of symptoms, with approximately 70% showing a significant positive effect. The specific drugs studied and their appropriate doses are listed in **Table 1**. It must be emphasized that when treatment is initiated with selective serotonin reuptake inhibitors, an initial increase in symptoms of anxiety may be observed in patients. This can lead to a premature termination of treatment. It is important to counsel the patient that these effects are typically temporary, and to initiate treatment with low doses that are increased slowly during the first weeks of therapy. Supplementation of the selective serotonin reuptake inhibitors with a benzodiazepine, such as clonazepam, may be beneficial during the initial weeks of treatment. Despite instituting these measures to maximize compliance, roughly 20% of patients in the authors' studies were intolerant of these medications. In patients with migraines and CSD, pharmacotherapy should be directed at addressing both the migraines and the CSD. This may be accomplished with one medication, such as a serotonin norepinephrine reuptake inhibitor (eg, venlafaxine HCl or duloxetine HCl) or possibly a tricyclic antidepressant (eg, nortriptyline). Head trauma patients have multiple central nervous system deficits, as outlined previously. They are best managed in a comprehensive head trauma program that combines pharmacotherapy, neuropsychologic testing and therapy, and physical and occupational therapy.

Behavioral Interventions

Cognitive behavioral therapy is a well-established and effective treatment for patients with anxiety disorders. Its efficacy in treating patients with CSD has not been well established; however, preliminary studies indicate it may be beneficial.[16] Cognitive behavioral therapy is of particular interest in patients with the interactive form of CSD, because they did not respond as well as the other groups of patients to pharmacotherapy alone.

Table 1
Doses of common selective serotonin reuptake inhibitors

Drug	Starting Daily Dose (mg)	Target Daily Dose by 4th Week (mg)	Subsequent Daily Increases (at 2—4-Week Intervals) (mg)	Maximum Daily Dose (mg)
Fluoxetine HCl	5–10	20	20	80
Sertraline HCl	12.5–25	50	50	200
Paroxetine HCl	5–10	20	20	60
Citalopram HBr	5–10	20	20	40
Escitalopram oxalate	5	10	10	20
Fluvoxamine maleate	25	150	50	300

Data from Staab JP, Ruckenstein MJ. Chronic dizziness and anxiety: effect of course of illness on treatment outcome. Arch Otolaryngol Head Neck Surg 2005;131:675–9.

Vestibular rehabilitation therapy in which patients are exposed to provocative stimuli in a controlled fashion may have a role for treatment of patients with CSD.[17] Some patients, however, report the exposure to these stimuli to be too uncomfortable and actually report a paradoxical increase in symptoms with vestibular rehabilitation therapy. If vestibular rehabilitation therapy is to be used in this patient group, then it should be administered by a therapist who is sensitive to the severe anxiety response that may be engendered in this patient group when exposed to provocative stimuli.

SUMMARY

Patients with chronic complaints of nonspecific dizziness can present frustrating diagnostic and therapeutic challenges. They seem to have no definite illnesses and seem to be beyond the scope of curative interventions. The authors' work, however, has expanded on previous studies and confirmed that anxiety-related processes cause or maintain symptoms in most cases. Recent research has shown that most patients with these symptoms can be helped by interventions directed at their underlying psychiatric disorders, including current methods of pharmacotherapy and psychotherapy. As a result, patients can be offered definitive, not just palliative, care.

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