Cervical spine diseases as a cause of vertigo systematic review of the evidence

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BPPV: Benign paroxysmal positional vertigo.
CNS: Central nervous system.
DHI: Dizziness Handicap Inventory.
MRI: Magnetic resonance imaging.
OAE: Oto acoustic emission.
ROM: Range of motion.
RVAO: Rotational vertebral artery occlusion.
SNAGs: Sustained natural apophysial glides.
SPNT: Smooth pursuit neck torsion.
TCD: Trans cranial Doppler.
VA: Vertebral artery.
VAS: Visual analogue scale.
VEMP: Vestibular evoked myogenic potientials.
VOR: Vestibulo ocular reflex.
WAD: Whiplash associated disorder.

Cervical vertigo

Definition

Vertigo is a specific type of dizziness; a major symptom of balance disorder, it is the sensation of spinning or swaying while the body is stationary with respect to the earth or surroundings.

Vertigo is a symptom common to pathologies or dysfunction of a number of structures, such as the labyrinth, vestibular nerve, and afferent nerves from cervical structures (peripheral vertigo) or brain stem, cerebellum, and other supratentorial structures (central vertigo).

Mixed vertigo occurs when both peripheral and central structures are affected, as in the cervical syndrome. Additionally, vertigo may be present because of metabolic, hematologic, endocrine, and other systemic diseases. **(*Troost*.,1980).**

Cervicogenic vertigo is defined as a sensation of rotation resulting from an alteration of the neck proprioceptive afferent of the upper cervical spine, as a consequence of their association with the vestibular nucleus, patients frequently experience a sensation of rotation or falling when they turn or flex and extend their head. **(*Biemond and De Jong*.,1969).**

Vertigo resulting from cervical pathologies was first described in 1858 by Claude Bernard. It has been attributed to many causes and several mechanisms***. (Brandt and Baloh.,2005).***

Vascular risk factors lead to atherosclerosis and thromboembolism of the vertebrobasilar system. Strong vascular risk factors are age, hypertension, diabetes mellitus, hyperlipidemia, and cigarette smoking. ***(Bulsara et al., 2006).***

Of particular significance due to its commonness is unilateral or bilateral rotational vertebral artery occlusion (RVAO) in cervical spondylosis***. (Cagnie et al., 2005).***

Cervical osteophytes can press on the vertebral artery causing its occlusion during head turning to the same or opposite side***. (Ogino et al., 2001).***

Even though in the past the phenomenon of cervical vertigo was considered a myth by some due to the commonness of both vertigo and cervical spondylosis particularly in the elderly, recent studies using neurovascular imaging techniques have established the veracity of the association between vertigo and cervical spondylosis expressing as RVAO***. (Citow and Macdonald.,1999).***

RVAO is important in those who have vascular risk factors that may compromise the integrity of the circle of Willis, particularly in the elderly***. (Brandt and Baloh., 2005).***

The resultant vertebrobasilar ischemia presents with variable features of cerebellar, pontine, medullary, mesencephalic, and occipital lobe dysfunction. ***(Kuether et al.,1997).***

The common presentation with vertigo may be due to the fact that the vascular supply to the vestibulocochlear organ being an end artery, may be more susceptible to vertebrobasilar insufficiency***. (Morales et al.,1990).***

Etiology and Pathophysiology:

Three mechanisms are usually suggested to explain the pathophysiology of cervical vertigo:

- **Vascular compression**: Mechanical compression, tension, dissection, or stenosis of one or both vertebral arteries as they pass through the cervical region will cause decreased blood flow and can also result in symptoms of presyncopal dizziness.

  Faulty head and neck posture, congenital deformities of the bones and tissues of the upper cervical spine, and traumatic or degenerative instabilities are among the causes of the mechanical compromise that could result in decreased vertebrobasilar blood flow**.** ***(Huijbregts and Vidal.,2004).***

- **Vasomotor changes caused by irritation of the cervical sympathetic chain:** The cervical sympathetic ganglia lie parallel to the spinal cord traversing along blood vessels and muscles antero-lateral to the vertebral bodies.

  The superior cervical ganglion, the largest of the cervical sympathetic ganglia and formed by coalescence of the cranial four sympathetic ganglia, is located at the level of C2-C3**.**

  Upper cervical dysfunction has been hypothesized to negatively impact this ganglion. This might affect the sympathetic innervations of both the vertebral and internal carotid arteries with subsequent posterior circulation hypoperfusion resulting in complaints of presyncopal dizziness. ***(Fitz-Ritson.,1991).***

- **Abnormal sensory input from neck proprioceptors:** Proprioceptors are nerves that sense movement and vibration. Sensory information from the neck is combined with vestibular and visual information to determine the position of the head on the neck, and postural adjustment.

  Cervicogenic dizziness results from abnormal input into the vestibular nuclei from the proprioceptors of the upper cervical region.

  The contribution of the upper cervical spine to this etiology, lies in the intricate neurological connection between the dorsal roots of the spinal nerves Cl -C3 and their synapses with the vestibular nucleus. ***(Brunarski .,1988).***

The reflexes responsible for maintaining balance are regulated through the stimulation of the afferent nerve fibers of the semicircular canals, the proprioceptive input from the eye muscles and the afferents from the cervical spine.

Hulse (1983), reported that 50% of the proprioceptors are found in the joint capsules of the cervical vertebrae C1- C3.

Furthermore, the interconnections between the cervical proprioceptors and the vestibular nuclei may contribute to a cyclic pattern, such that cervical muscle spasms contribute to dizziness and dizziness contributes to muscle spasm, although the causal relationship is unclear. ***(Furman and Cass.,1996).***

The above neurological characteristics may help explain why traumatic, degenerative, inflammatory or mechanical derangements of the cervical spine can affect the mechanoreceptor system and give rise to vertigo.

In one-third of the patients suffering from cervical disequilibrium, the onset was attributed to trauma, usually whiplash, in one-third the onset was insidious, and in the remaining third several causes were reported, including manual therapy to the upper cervical spine. ***(Hulse.,1983).***

Clinical picture:

The clinical picture of patients suffering from cervicogenic vertigo tends to be vague .

The following symptoms are indicative of a diagnosis of cervicogenic vertigo:

- Pain or discomfort in the cervical region, especially following trauma, muscle tenderness and stiffness.
- Dizziness that can be provoked by certain head positions or movements.
- Dizziness of short duration and decreasing intensity.
- Persistent occipital region headache.
- Limited cervical spine range of motion (ROM).
- Jaw pain.
- Upper extremity radicular symptoms.

The symptoms may be worsened with computer use, reading or sustained neck position. ***(Wrisley et al.,2000).***

Differential diagnosis :

Cervical vertigo needs to be distinguished from other causes of vertigo such as :

- Labyrinthine:

-Benign paroxysmal positional vertigo.
-Post-traumatic otolith vertigo.
-Perilymph fistula. ***(Lempert et al.,1995).***

- Vestibular nerve:

-Unilateral vestibular failure (e.g, vestibular neuritis).
-Bilateral vestibular failure.
-Vestibular paroxysmia. ***(Brandt and Dieterich.,1994).***
-Nerve compression by cerebellopontine angle mass.

- Ocular motor:

-Extraocular eye muscle or gaze paresis.

- Central vestibular:

-Central positional nystagmus /vertigo.
-Migraine without aura.
-Migraine with aura (basilar migraine, vestibular migraine) .
-Vestibulocerebellar ataxia. ***(Brandt.,1999).***

- Vascular:

-Rotational vertebral artery occlusion (RVAO).
-Carotid sinus syndrome. ***(Strupp et al.,2000).***

- Intoxication:

-Positional alcohol nystagmus /vertigo.
-Drugs (eg, antiepileptics).

<u>Diagnostic maneuvers:</u>

There is no consensus on how to diagnose cervical vertigo. ***(Brandt., 1996).***

Cervicogenic dizziness is a diagnosis of exclusion (i.e. the diagnosis is usually based on the elimination of the other competing diagnosis, such as vestibular or central nervous system pathologies). The development of a robust clinical diagnostic test for cervicogenic dizziness has been elusive.

It is important to take a detailed history and perform a comprehensive examination in order to rule out other causes of dizziness. A negative history screen was assumed to exclude vertebrobasilar involvement. A negative sit-to-stand test was used to exclude neurocardiogenic syncope. Negative limb ataxia tests served to exclude CNS involvement. A negative response on the sensitive Hallpike-Dix maneuver excluded BPPV. Positive findings on the VOR test (vestibulo-ocular reflex), the tests for saccadic and smooth pursuits eye movements, and the Romberg test suggested a sensory processing disorder but not a CNS lesion. **(Vidal and Huijbregts.,2005).**

Therefore, the diagnosis of cervicogenic dizziness is suggested by:
(1) a close temporal relationship between neck pain and symptoms of dizziness, including time of onset and occurrence of episodes.
(2) previous neck injury or pathology.
(3) elimination of other causes of dizziness. ***(Furman and Cass.,1996).***

If cervical vertigo still seems likely after excluding reasonable alternatives, we have to look for positive confirmation.

The neck torsion nystagmus test, or head-fixed, body-turned maneuver is considered by some to identify cervicogenic dizziness. Theoretically, the neck proprioceptors are stimulated while the inner ear structures remain at their resting state. This test requires the head of the patient to be stabilized while the body is rotated underneath. ***(Phillipszoon.,1963).***

Routine studies in working up cervical vertigo include:

1. Plain radiographs of the cervical spine: may show flat lordosis, discopathy, osteophytes and sclerosis. ***(Olszewski et al.,2006).***

2. MRI neck and brain: provides information on brain ischemia, infarction, it may not reveal the specific cause of dizziness ***(Colledge et al.,2002).***

3. VEMP (vestibular-evoked myogenic potentials): assess function of the saccule, the inferior vestibular nerve and descending vestibular pathways from the medulla to the neck muscles**.**

4. CT-angiography: if MRI neck is negative or there is strong suspicion of vascular etiology, it demonstrates the nature, cause and location of the vertebral artery compression and shows the vascular anatomy of the anterior and posterior circulation ***(Bulsara et al.,2006).***

5. Posturography : with the head held in different angles on the neck has been used in an attempt to diagnose cervical vertigo.

6. Vertebral Doppler : is useful in demonstrating RVAO.

7. Fluoroscopy of the neck : may be used in persons with abnormal flexion –extension views. ***(Sakaguchi et al.,2003).***

These studies are done to rule out other causes of vertigo:

1. Audiogram.

2. OAE (oto-acoustic emission).

Management:

Conservative approach consists of control of vascular risk factors and neck immobilization either by instructing the patient to refrain from excessive head turning or by the use of cervical collar. ***(Dabus et al.,2007).***

The intervention for cervicogenic dizziness has included manual therapy (mobilization and manipulation), mechanical traction, physical modalities, postural reeducation, active range of motion, massage, balance retraining, trigger point injection,and
muscle relaxants. ***(Biesinger.,1988).***

In addition to manipulation, soft tissue therapy should also be directed to the suboccipital musculature, which is frequently
hypertonic***. (Brunarski.,1988).***

Stretching and strengthening exercises may prove to be effective in the short- and long-term care of these patients. A carefully planned program that involves exercises progressing from flexibility to strengthening to endurance, emphasizing the suboccipital muscles, should be given***. (Fitz-Ritson.,1990).***

When cervicogenic vertigo is associated with benign positional vertigo, vestibular habituation training should be part of the plan of management. ***(Bourgeois and Dehaene.,1988).***

Medical management may include anti-inflammatory drugs which may facilitate healing by reducing inflammation and fibrotic constriction of the vertebral artery. Also antidepressants may be used for chronic pain ***.(Borg-Stein et al.,2001).***

Surgical treatment, where indicated, must be tailored to the identified cause of the obstruction.

Surgery is indicated when symptoms are incapacitating and recurrent and diagnostic tests clearly demonstrate a hemodynamically significant compression of the VA as being responsible for the symptoms. ***(Vilela et al.,2005).***

Options include fascial decompression, vertebral artery decompression (anterior and posterior approach, osteophytectomy C1-C2 fusion, decompressive foraminotomy, decompressive transverse foramenectomy and discectomy**.** ***(Bulsara et al.,2006).***

Intraoperative and postoperative dynamic angiography and dynamic trans cranial Doppler (TCD) ultrasound may be useful in the evaluation of the outcome of the surgery. Overall success rate for surgery was put at 85% by Kuether and collaege. ***(1997).***

Prevention:

Care should be taken to avoid RVAO in patients at risk during cervical manipulation and perhaps during tracheal intubation. ***(Weintraub and Khoury.,1998).***

Those at risk can be identified by clinical or Doppler ultrasound screening for RVAO. Proper management of RVAO can prevent stroke. ***(Childs et al.,2005).***