Audio-Vestibular Evaluation in Patients with Cervicogenic Dizziness

Original Article

Somaya Yosef Mahmoud[1], Ossama Ahmed Sobhy[2], Mohammed Bassiouny Atallah[3], Yousra Hisham Abdelfattah[4], Mayada Abdelsalam Elsherif[5]

[1,2,3,5]Audiovestibular Unit, Department of Otorhinolaryngology, [4]Physical Medicine, Rheumatology and Rehabilitation, Faculty of Medicine, Alexandria University, Alexandria, Egypt.

ABSTRACT

Background: Cervicogenic dizziness has many potential mechanisms and is usually a diagnosis of exclusion. Otoacoustic emissions are used to assess cochlear function. Inner ear disorders commonly affect both cochlea and vestibular labyrinth. Posturography is used to evaluate the incorporation of sensory inputs; visual, vestibular and somatosensory which maintain posture and can be used in patients with cervicogenic dizziness.

Objective: Analyze audiological, vestibular and postural findings in patients with spondylo-degenerative changes of the cervical spine with and without dizziness.

Patients and Methods: This study was carried out on 70 patients with spondylo-degenerative changes of the cervical spine; 35 associated with dizziness and 35 without dizziness as a control group. Radiological assessment of the cervical spine was done and cervical degenerative index was used to assess severity. Basic audiological evaluation and videonystagmography were done to exclude peripheral and central vestibular lesions. Self-report of dizziness was measured using Dizziness Handicap Inventory. Otoacoustic emissions and posturography were assessed in all patients.

Results: CDI total score was significantly higher in patients with dizziness. A positive correlation between CDI score and DHI scores was found. Lower OAES amplitudes were found in the patients with dizziness. SOT scores were lower in dizziness group compared to no-dizziness group.

Conclusion: Severity of cervicogenic dizziness could be associated with severity of radiographic findings. Two possible mechanisms may have a role in cervicogenic dizziness include vascular compression mechanism, which was explained by otoacoustic emissions results. Another probable mechanism is neck proprioceptors damage resulting in postural problems in those patients.

Key Words: Cervical Degenerative Index (CDI), cervicogenic dizziness, computerized dynamic posturography, otoacoustic emissions, spondylo-degenerative changes.

Received: 4 January 2022, **Accepted:** 21 February 2022

Corresponding Author: Somaya Yosef Mahmoud, MSc, Audiovestibular Unit, Department of Otorhinolaryngology, Faculty of Medicine, Alexandria University, Alexandria, Egypt, **Tel.:** 01022767519, **E-mail**: somaya.yosif@yahoo.com

ISSN: 2090-0740, 2022

INTRODUCTION

Cervicogenic dizziness (CGD) is currently used in practice to describe the dizziness arise from the cervical spine. It is also named cervicogenic vertigo, proprioceptive vertigo and cervical dizziness.[1, 2]

Balance results from integration of sensory inputs; visual, vestibular and proprioceptive which send signals to the CNS via specific afferent pathways and any dysfunction in these sensory inputs (such as cervical proprioception) or asymmetry in the afferents would lead to sense of imbalance or dizziness.[3, 4]

The etiology of CGD is not clear.[4] There are many causes and many potential mechanisms include: vertebra-basilar insufficiency "VBI" and or abnormal sensory input from neck proprioceptors.[5]

Otoacoustic emissions (OAEs) are used to assess cochlear function, because the cochlea and vestibular labyrinth share common blood supply, inner ear disorders commonly affect both structures resulting in dizziness and imbalance, hearing loss and tinnitus may present.[6]

Computerized dynamic posturography test (CDP) is used to differentiate between different mechanisms controlling posture; sensory, motor, and central mechanisms.[7] Sensory organization test (SOT) is one exclusive test of CDP which is used to quantify the involvement of the different sensory inputs; visual, proprioceptive, and vestibular which are needed to maintain posture.[8]

 DOI: 10.21608/ejentas.2022.114706.1459

Thus, finding a relationship between dizziness and abnormal vestibular function originate from cervical spine may provide a good diagnostic tools and effective rehabilitation for its management beside the traditional procedures.

Also, radiological assessment of those patients could show spondylo-degenerative changes, disk abutting cervical cord and stiffness of the neck.[9, 10]

Finding a relation between the SOT parameters and the radiological findings in such patient could help to understand the possible pathophysiology.

AIM OF THE WORK:

Analyze audiological, vestibular and postural findings in patients having cervical spine spondylo-degenerative changes with and without dizziness.

PATIENTS AND METHODS:

Subjects: :

A case-control study was carried out on 70 patients having cervical spine spondylo-degenerative changes with and without dizziness who were selected from Alexandria main university hospital clinics from physical medicine and rheumatology department and Audiology unit, divided into:

Group 1: consisted of 35 patients having cervical spine spondylo-degenerative changes and complaining of dizziness.

Group 2: consisted of 35 patients having cervical spine spondylo-degenerative changes not complaining of dizziness as a control group. Both groups were matched by age and sex. Patients with other neurootological disorders were excluded from this study.

METHODS

All subjects in this study signed an informed consent. Complete history was taken from all participants, Basic audiological assessment including: pure tone audiometry test, speech audiometry and immittance test and physical examination were done for study and control groups. Self-reported dizziness handicap inventory was completed. Complete video-nystagmography test to exclude patients with peripheral and central vestibular lesions was done.

Vertebral artery test:

By using VNG goggles, the head of the patient was turned to one side and the patient was in supine or upright position for 30 seconds. The examiner observed for signs of VBI as (diplopia, dizziness, dysarthria and nystagmus. If a nystagmus occurred, the head was turned again to the center and observe the disappearance of the nystagmus. The test was repeated for confirmation.[11]

Radiological assessment of the cervical spine by:

Plain x-ray of cervical spine was done (AP, lateral and dynamic: flexion and extension views). Grading of cervical degenerative changes was done by cervical degenerative index score (CDI) to quantify the radiological changes.[12, 13]

Four factors were considered and a quantitative score for these factors was calculated. They included: narrowing of disc space, osteophytes formation, endplate/facet sclerosis and olisthesis. At each level (C2/C3 to C6/C7), the 4 factors were added resulting in the overall cumulative score of CDI. The normal appearance was given‘‘0’’ score while severe spine changes were given ‘‘3’’ score. Quantitative criteria for each factor are shown in (Table 1).

Otoacoustic emissions (OAEs), TEOAES and DPOAES, were used to assess cochlear function and to detect inner ear ischemia. The test was done in a quiet environment (a sound booth), patients were instructed to sit quietly in a chair near the equipment.

The recommended test settings for TEOAE acquisition by manufacture: Stimulus: 75 microsecond click, rate: 19.30 per second, sweeps: 1024, intensity: 85 to 90 dB SPL and passing Criteria: 3 out of 5 passing with 6 dB SNR minimum and 90% Correlation.

The recommended test settings for DPOAE by manufacture: frequencies range: from 500 to 6000 Hz, F2/F1 ratio: 1.22, L1: 65dB SPL and L2: 55dB SPL and passing Criteria ≥ 65%. A DP-Gram presents the response intensity at each frequency and the level of noise around it.

Computerized dynamic posturography (CDP):

Using SYNAPSYS Posturography System (SPS) to assess postural instability and mainly by a Sensory Organization Test (SOT).

The complete protocol of SOT of SPS Posturography exposes the patient to six sensory conditions. They were presented beginning with the simplest, eyes open on platform, and ending with the most challenging in which the patient stands on foam surface with closed eyes. Each of the six conditions was performed in two trials, each of 20seconds.

Fixed force platform with eyes open (EO), Fixed force platform, eyes closed (EC), Fixed force platform, vision erroneous (VE), sway platform, (EO), sway platform, (EC) and sway platform (VE).

Antero-posterior (AP) and medio-lateral (ML) center of pressure sway were plotted for the test six conditions. Each test trial lasted for 20 seconds with a rest for about 20 seconds before the next trial and then the two trials average was used to compare results.

Ratios between the equilibrium scores of the sensory conditions were calculated by CDP system creating five sensory scores in both in both (AP) and (ML) translations including: somesthetic, visual, vestibular, preferential and global scores.

Somesthetic score = Condition 2 / Condition 1

Visual score = Condition 4 / Condition 1

Vestibular score = Condition 5 / Condition 1

Preferential = Condition 3 + Condition 6 / Condition 2 + Condition 5

The global score is a weighted average of balance scores on the 6 conditions and represents the overall level of balance. These sensory scores in the software are plotted in a vertical bar chart. Each of the vertical bars presents in green color if the score is more than the reference value and in red color else. (Figure 1).

Table 1: Cervical Degenerative Index (CDI).

	CDI score			
Factor	0	1	2	3
Disc space narrowing (%)	None–25	25–50	50–75	75–100
End plate sclerosis	None	Minimal	Moderate	Severe
Osteophytes	None	Small, <2 mm	Moderate, 2–4 mm	Large, >4 mm
Listhesis	None	<3 mm	3–5 mm	>5 mm

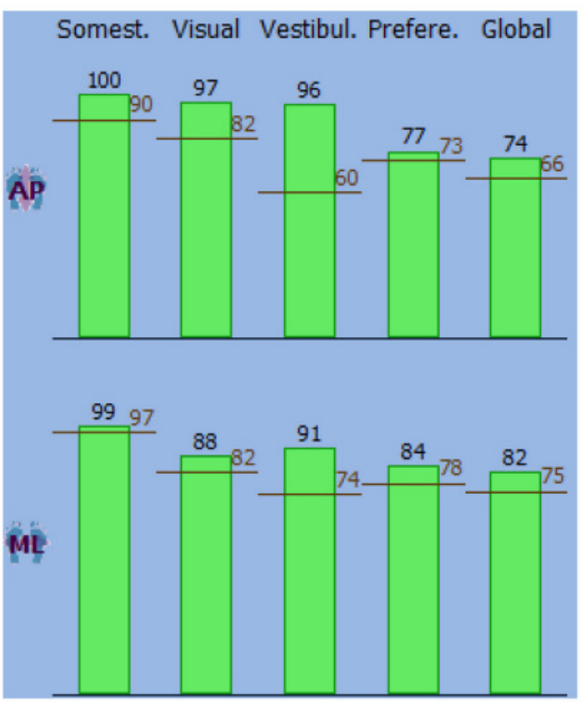

Fig. 1: The sensory organization test (SOT) results in a normal patient

Statistical Analysis:

Data were analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). Qualitative data were described using number and percent. Shapiro-Wilk test was used to verify the normality of distribution Quantitative data were described using range (minimum and maximum), mean, standard deviation and median. Significance of the obtained results was judged at the 5% level. Chi-square, Fisher's Exact or Monte Carlo correction and Student t-test were used for statistical analysis. Spearman coefficient was used to correlate between two distributed abnormally quantitative variables.

RESULTS:

A total number of 70 patients having cervical spine spondylo-degenerative changes with and without dizziness were included in this study. They were divided into: Group 1: consisted of 35 patients complaining of dizziness (15 male and 20 female), their mean age was 51.26 ± 7.79 years .Group 2: consisted of 35 patients with no dizziness as a control group (20 male and 15 female), their mean age was 48.06 ± 8.66 years. No significant statistical difference was found between study and control groups regarding age and sex. ($p > 0.05$)

Results of basic audiological evaluation shown normal hearing sensitivity in speech frequencies (500, 1000 & 2000 Hz), some individuals had mild high frequency sloping SNHL. Excellent speech discrimination and normal middle ear function were detected in both groups and no statistical significant difference between them. As regards physical examination, vertebral artery test was carried out for both groups with no abnormal findings (negative) in the control no-dizziness group. In dizziness group, 11 patients showed positive abnormal findings (vertigo, nystagmus or blurring of vision).

Dizziness handicap inventory results for the dizziness group:

Descriptive analysis of the DHI scores of the studied cases with dizziness in different domains was done. Mean for total scores was (65.43 ± 10.81), total physical scores (15.83 ± 2.93), total emotional scores (23.14 ± 5.30) and for total functional scores (26.46 ± 4.03).

Radiological results

The radiological assessment of the cervical spine by Plain x-ray (AP, lateral and dynamic: flexion and extension views) was carried out upon all participants and grading of cervical abnormalities by Cervical degenerative index score (CDI) to quantify the radiological changes was calculated. Patients in dizziness group showed significantly higher scores as regard disc space narrowing, osteophytes and overall cumulative scores when compared to the no-dizziness group. (*P < 0.001*)

As regard total scores of sclerosis and olithesis there was no statistically significant difference a between the two studied groups. (*P= 0. 082* and 0.107 respectively)

Figure 2 shows an example of a case of spondylo-degenerative cervical spine changes.

Otoacoustic emission results:

No failed response was observed in both groups, however, in dizziness group 11 patients (31.4%) pass totally, unilateral partial pass in 17 (48.6%) patients and bilateral partial pass in 7 (20.0%) of patients. In the control group 24 patients (68.6%) pass totally, unilateral partial pass in 9 (25.7%) patients and bilateral partial pass in 2 (5.7%) of patients. These results were statistical significant. (*p≤ 0.05*)

Tables (2 & 3) show results of TEOAE of the two studied groups. Dizziness group showed significantly lower overall reproducibility (%) compared to no-dizziness group (mean 86.43 ± 8.65 and 89.50 ± 7.75) respectively. (*P=0.029*)

As regard TEOAE amplitude, Patients in dizziness group showed significantly lower amplitude compared to no dizziness group at (2 & 4) KHz. (*P= 0.002* and *P<0.001*) While there was no statistically significant difference between the two studied groups at 1 KHz. (*P= 0.108*)

Table (4) shows the results of DPOAES (F2) of the two studied groups. Dizziness group showed significantly lower DPOAES level compared to no-dizziness group at (4 & 6) KHz. (*P<0.001*)

While there was no statistically significant difference between the two studied groups at (1 & 2) KHz (*P= 0.152* and 0.415) respectively.

Sensory organization test results:

Figures (3 & 4) show comparison of sensory balance scores of complete static sensory organization test in antero-posterior and medio-lateral translation respectively between the two studied groups. All scores, except vestibular, were statistically significant lower in the dizziness group than the no-dizziness group. (*p=<0.001*)

Correlations:

There was statistically significant positive correlation between overall cumulative score of CDI and DHI total scores in dizziness group (*P<0.001*) (r=0.787). It means that increasing the CDI of the radiological findings leads to increasing in self-report of dizziness and performance by patients.

Correlations were done between the radiological findings and SOT scores shown in (Figures 5-10); there was statistically significant negative correlation between overall cumulative score of CDI and all conditions of SOT except the preferential scores in ML translation.

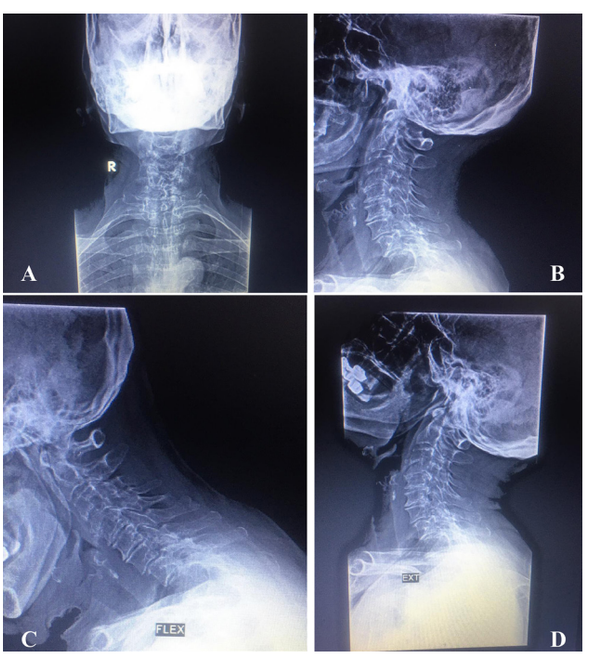

Fig. 2: Plain x-ray (AP, lateral and dynamic: flexion and extension views) of a case of spondylo-degenerative cervical spine changes

Table 2: Comparison of the TEOAE overall reproducibility (%) between the two studied groups

TEOAE overall repro %	Dizziness (n=70)	No dizziness (n=70)	T	P
Min. – Max.	65.0 98.0 –	75.0 98.0 –		
Mean ± SD.	86.43 8.65 ±	89.50 7.75 ±	2.213*	0.029*
Median (IQR)	86.0 (79.0 96.0 –)	93.0 (82.0 96.0 –)		

IQR: Inter quartile range SD: Standard deviation
t:Student t-test
p: p value for comparing between the studied groups
*: Statistically significant at $p \leq 0.05$

Table 3: Comparison of the TEOAE amplitude (dBSPL) between the two studied groups

TEOAE Amplitude (dBSPL)	Dizziness (n=70)	No dizziness (n=70)	U	P
1KHz				
Min. – Max.	-5.0 20.30 –	2.40 25.50 –		
Mean ± SD.	8.79 4.97 ±	10.13 4.99 ±	2065.50	0.108
Median (IQR)	8.0 (5.60–11.30)	10.0 (5.0–12.0)		
2KHz				
Min. – Max.	-9.0 17.60 –	-8.0 18.70 –		
Mean ± SD.	3.17 7.15 ±	6.95 7.44 ±	1722.50*	0.002*
Median (IQR)	4.50 (-4.0–8.50)	8.25 (2.0–14.50)		
4 KHz				
Min. – Max.	-18.0 10.50 –	-15.0 18.0 –		
Mean ± SD.	-7.75 6.90 ±	1.73 9.78 ±	1095.0*	<0.001*
Median (IQR)	-8.60 (-12.80– -5.0)	2.0 (-5.0 – 9.50)		

IQR: Inter quartile range SD: Standard deviation
U: Mann Whitney test
p: p value for comparing between the studied groups
*: Statistically significant at $p \leq 0.05$

Table 4: Comparison of the DPOAES level (dBSPL) between the two studied groups

DPOAES level (dBSPL)	Dizziness (n=70)	No dizziness (n=70)	U	P
1KHz				
Min. – Max.	-8.0 18.0 –	-12.0 17.60 –		
Mean ± SD.	7.40 5.88 ±	8.71 5.98 ±	2107.0	0.152
Median (IQR)	8.0 (5.0 12.0 –)	10.0 (5.0 12.0 –)		
2KHz				
Min. – Max.	-13.0 14.0 –	-13.0 13.0 –		
Mean ± SD.	3.0 6.88 ±	3.88 6.69 ±	2255.0	0.415
Median (IQR)	5.0 (8.0 – 1.0–)	5.0 (1.0 10.0 –)		
4 KHz				
Min. – Max.	-17.0 7.0 –	-16.0 14.0 –		
Mean ± SD.	-7.05 6.34 ±	1.51 7.35 ±	914.50*	<0.001*
Median (IQR)	2.30– – 12.0–) 8.0–)	2.75 (7.30 – 1.0–)		
6 KHz				
Min. – Max.	-20.0 -1.0 –	-19.50 6.0 –		
Mean ± SD.	-14.93 4.16 ±	-6.03 7.30 ±	825.0*	<0.001*
Median (IQR)	-16.20 (13.0– –18.0–)	-4.0 (0.0 – 12.50–)		

IQR: Inter quartile range SD: Standard deviation
U: Mann Whitney test
p: p value for comparing between the studied groups
*: Statistically significant at $p \leq 0.05$

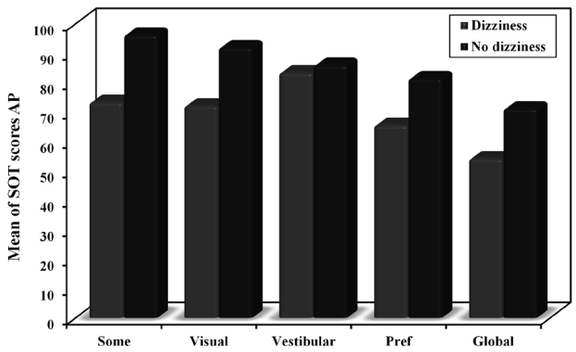

Fig. 3: Comparison of the SOT scores in AP translation of the two studied groups

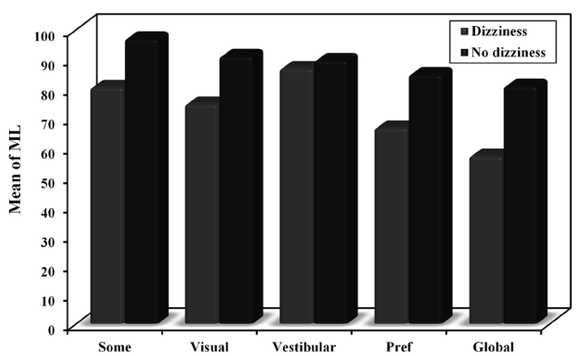

Fig. 4: Comparison of the SOT scores in ML translation of the two studied groups

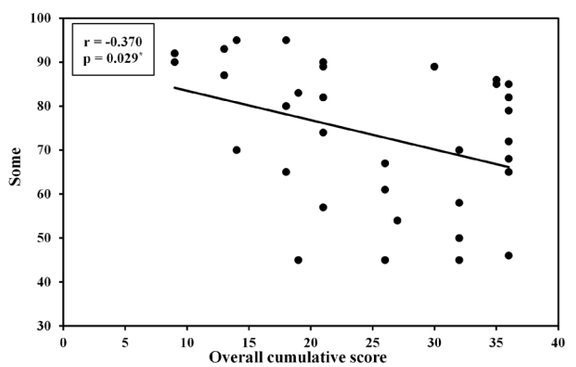

Fig. 5: Correlation between overall cumulative score and SOT scores AP translation (Some) in dizziness group (n = 35)

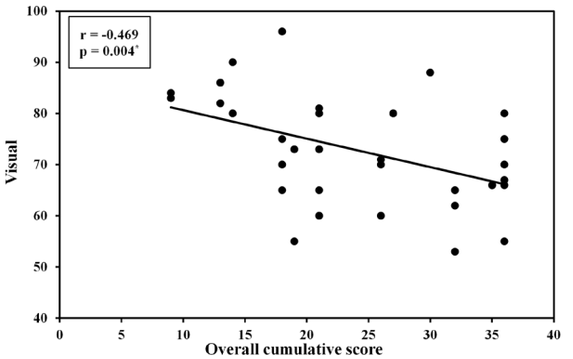

Fig. 6: Correlation between overall cumulative score and SOT scores AP translation (Visual) in dizziness group (n = 35)

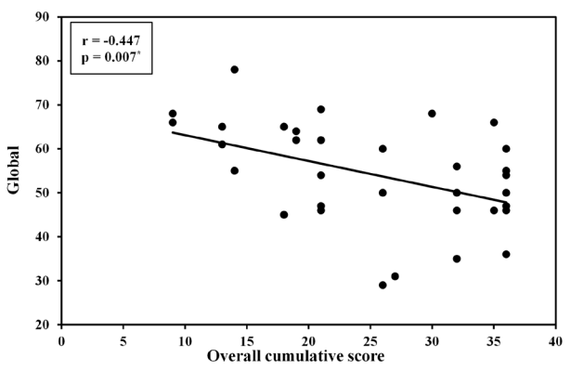

Fig. 7: Correlation between overall cumulative score and SOT scores AP translation (Global) in dizziness group (n = 35)

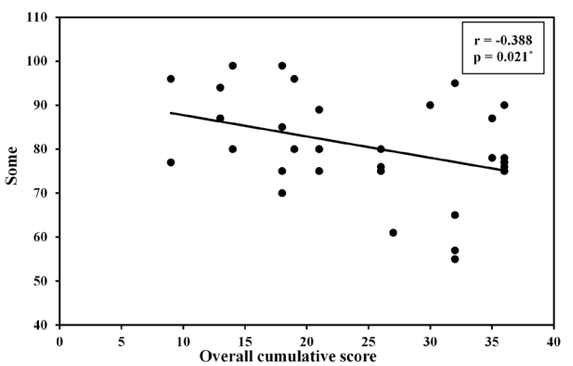

Fig. 8: Correlation between overall cumulative score and SOT scores in ML translation (Some) in dizziness group (n = 35)

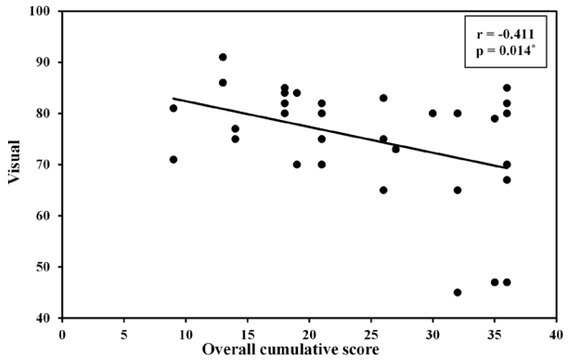

Fig. 9: Correlation between overall cumulative score and SOT scores in ML translation (Visual) in dizziness group (n = 35)

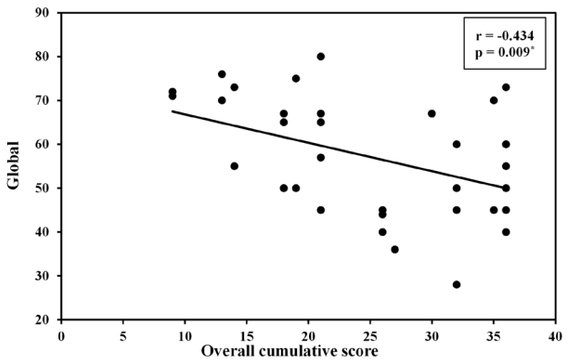

Fig. 10: Correlation between overall cumulative score and SOT scores in ML translation (Global) in dizziness group (n = 35)

DISCUSSION

There are many potential mechanisms of CGD; there is no consensus on the diagnosis of CGD. Cervical spine was assessed by plain x-ray (AP, lateral and dynamic: flexion and extension views). The significant higher CDI scores in the dizziness than the control no-dizziness group could explain the probable pathophysiology of CGD; as the vestibular labyrinth, vestibulo-cochlear nerve up to cerebral lobes are supplied by the vertebro-basilar circulation, so, cervical osteophytes and disc space narrowing may compress VA when turning the head leading to VBI leading to dizziness.[14-17]

The vascular supply to the vestibulo-cochlear organ is an end-artery. Thus, it depends on vertebro-basilar circulation leading to increased vulnerability to VBI. Neurons, axons, and hair cells in the vestibulo-cochlear system are responding to ischemia by depolarization leading to transient hyper-excitability and ectopic discharges which is presented by dizziness.[18]

Disc space narrowing, stenosis of cervical spine, which is spinal canal narrowing and/or spinal nerve root passages in the neck results in compression of ascending or descending tracts in the spinal cord that interact with vestibulo-spinal projections, vestibular nucleus and cerebellum. This is a common mechanism of CGD supporting the nerve compression theory.

In agreement with our study, Chetan Shende *et al* in a prospective-observational study supported the finding s in the present study.

The severity of spondylytic changes was higher according to CDI scores in the dizziness group than the control group. This is a possible justification for CGD and why some patients with spondylo-degenerative changes in the cervical spine complain of dizziness and some do not.

Self-report of dizziness and performance measurement using Arabic version of Dizziness Handicap Inventory questionnaire (DHI), which comprises 25 questions designed to assess the physical, functional and emotional restrictions of patients on a three point scale.[19] Several studies in literature used Dizziness Handicap Inventory in patients with CGD.[20-22]

A positive correlation between overall cumulative score of CDI and DHI total scores in dizziness group was found. It means that increasing the CDI of the radiological findings leads to increasing in self-report of dizziness and performance by patients.

On physical examination, vertebral artery test (VAT) was carried out for both studied groups with no abnormal findings (negative) in the no-dizziness group while in dizziness group, 11 patients revealed positive abnormal findings (vertigo, nystagmus or blurring of vision). The positive test was common in patients with more severe radiological findings and could be explained by compression on VA on rotation the head leading to VBI.

In agreement with our study, a study was carried by Kotait *et al*.[23] in 2017 who conducted a study on a group consisted of 32 patients with diagnosed cervicogenic vertigo and a control group consisted of 20 normal subjects and found that on physical examination, positive vertebral artery test in five patients of study group, these findings support our present study.

Otoacoustic emissions (OAEs) are present in individuals with normal hearing and are no longer produced when the hearing loss is greater than 30 dB by TEOAEs. DPOAEs are shown in individuals with hearing loss up to 50 dB. Cochlear function changes are likely to be detected by the OAE test before the

hearing loss is presented by audiogram. Thus, OAEs measurement was used to assess cochlear function.[24]

The present study showed that the dizziness group had statistically significant lower overall reproducibility (%) of TEOAEs compared to no-dizziness group. (Table 2)

As regard TEOAE amplitude, patients in dizziness group showed significantly lower amplitude compared to the no-dizziness group at (2 & 4) KHz. On the other hand, there was no statistically significant difference between the two studied groups at 1 KHz. (Table 3)

Additionally, dizziness group showed significantly lower DPOAES level compared to the no-dizziness group at (4 & 6) KHz, while there was no statistically significant difference between the two studied groups at (1&2) KHz. (Table 4)

These results mean that the cochlear function is affected mainly in the group of patients with dizziness rather than the other no- dizziness group and the affection is mainly at high frequencies rather than low frequencies obtained both TEOAEs and DPOAEs as high frequencies at cochlear basal portion are more vulnerable to metabolic and ischemic changes than apical low frequencies. Region of cochlea tuned for high frequencies is more vulnerable than the low frequencies region and this could explain the more vulnerability of deficit blood supply.

A study conducted by Li and Zhong,[25] in 1998 about the spectral analysis of distrot-product otoacoustic emissions (DPOAE) confirmed that those patients with mild dysfunction of cochlea can be detected by the amplitude and spectral analysis of DPOAE, which may be useful to differentiate the region of VBI.

Because the cochlea and vestibular labyrinth share the common blood supply and lymphatic spaces, inner ear disorders commonly affect both structures, resulting in dizziness and imbalance, hearing loss and tinnitus may present. These findings can support the VBI and ischemic pathophysiology as a cause of CGD.[6]

The present study showed that the group with dizziness had statistically significant lower sensory balance scores than the group with no dizziness in both antero-posterior and medio-lateral translation in somatosensory, visual, preferential and global scores. On the other hand, there was no statistical significant difference in vestibular scores. (Figures 3 & 4)

Balance results from integration of sensory inputs from visual, vestibular and proprioceptive receptors then afferents are sent to CNS. The information integrated by CNS then efferent pathways from it control muscle tone and maintain the posture and balance. Dysfunction in these sensory organs (such as cervical proprioception) or asymmetry in the afferent pathway may result in sense of dizziness or imbalance.[3, 26]

Based on this principle, the high proprioceptors amount in the cervical muscles and ligaments[27, 28], with the normal function of the vestibular system,[29] cervical inputs acts as a reference of the body position to process vestibular information adequately.[5, 30] So, the cervical proprioceptive error leading to mismatch between proprioception from the cervical region, visual and vestibular sensations might be explained one cause of imbalance and dizziness in patients with spondylo-degenerative changes in cervical spine. Also, this may cause an uncertainty about body control, resembling somatoform dizziness, which may impact the quality of life.[31]

In this field, many studies have been conducted to assess postural disorders by posturography in patients with cervical problems.[22, 32-35]

Another explanation of stability disorders in patients with spondylo-degenerative disorders is related to the limitation in the mobility of cervical spine and then visual field restriction during head movement. Neck stiffness may lead to eye-head decoupling with loss of coupling function which means that whenever the eyes move some degrees in the horizontal plane, a reflex head movement shifts the visual target by the same degrees towards the fovea on the retina to get more detailed analysis.[36, 37] A study carried by Yahia *et al*.,[33] reported similar results.

Additionally, this study found a correlation between radiological findings and sensory balance scores (AP & ML). There was statistically significant negative correlation between overall cumulative score of CDI and all conditions of SOT except the preferential scores in ML translation. It means that increasing in CDI scores correlates with decreasing scores of SOT. (Figures 5-10)

And this may justify the abnormal findings of SOT scores in patients with spondylo-degenerative disorders complaining of dizziness rather than the other group of patients with no dizziness. The significant lower SOT score in patients with spondylo-degenerative disorders complaining of dizziness denoting the influence of cervical spine pathology on the postural stability.

CONCLUSION

The severity of cervicogenic dizziness could be associated with the severity of radiographic findings. Furthermore, two possible mechanisms may have a role in cervicogenic dizziness include vascular compression mechanism, vertebrobasilar insufficiency, which was explained by the results of otoacoustic emissions. Another probable mechanism is the damage to neck proprioceptive receptors resulting in instability and postural problems in those patients.

LIST OF ABBREVIATIONS:

CDI: Cervical degenerative index

CDP: Computerized dynamic posturography

CGD: Cervicogenic dizziness

DCD: Degenerative cervical spine disorder

DHI: Dizziness Handicap Inventory questionnaire

DPOAEs: Distortion product otoacoustic emissions

OAEs: Otoacoustic emissions

SOT: Sensory organization test

TEOAEs: Transient evoked otoacoustic emissions

VAT: Vertebral artery test

VBI: Vertebrobasilar insufficiency

VNG: Videonystagmography

CONFLICT OF INTEREST

There are no conflicts of interest.

REFERENCES

1. Herdman S. Vestibular Rehabilitation. 3rd ed. Philadelphia: Davis Company; 2007.
2. Lystad RP, Bell G, Bonnevie-Svendsen M, Carter CV. Manual therapy with and without vestibular rehabilitation for cervicogenic dizziness: a systematic review. Chiropr Man Therap 2011; 19(1):21.
3. St George RJ, Fitzpatrick RC. The sense of self-motion, orientation and balance explored by vestibular stimulation. J Physiol 2011; 589(Pt 4):807-813.
4. Grgić V. [Cervicogenic proprioceptive vertigo: etiopathogenesis, clinical manifestations, diagnosis and therapy with special emphasis on manual therapy]. Lijec Vjesn 2006; 128(9-10):288-295.
5. Hain TC. Cervicogenic causes of vertigo. Curr Opin Neurol 2015; 28(1):69-73.
6. Mazzoni A. The vascular anatomy of the vestibular labyrinth in man. Acta Otolaryngol Suppl 1990; 472:1-83.
7. Vanicek N, King SA, Gohil R, Chetter IC, Coughlin PA. Computerized dynamic posturography for postural control assessment in patients with intermittent claudication. J Vis Exp 2013; (82):e51077.
8. Black F. Clinical status of computerized dynamic posturography in neurotology. Opin Otolaryngol Head Neck Surg 2001; 9(1):314-318.
9. Brandt T, Bronstein AM. Cervical vertigo. J Neurol Neurosurg Psychiatry 2001; 71(1):8-12.
10. Heikkilä H, Johansson M, Wenngren BI. Effects of acupuncture, cervical manipulation and NSAID therapy on dizziness and impaired head repositioning of suspected cervical origin: a pilot study. Man Ther 2000; 5(3):151-157.
11. Hain M, Cherchi T. Provocative maneuvers for vestibular disorders. In: Eggers S, Zee D (eds). Vertigo and imbalance: clinical neurophysiology of the vestibular system. London: Elsevier; 2010. 300-315.
12. Gore DR, Sepic SB, Gardner GM. Roentgenographic findings of the cervical spine in asymptomatic people. Spine (Phila Pa 1976) 1986; 11(6):521-524.
13. Ofiram E, Garvey TA, Schwender JD, Denis F, Perra JH, Transfeldt EE, *et al*. Cervical degenerative index: a new quantitative radiographic scoring system for cervical spondylosis with interobserver and intraobserver reliability testing. J Orthop Traumatol 2009; 10(1):21-26.
14. Wrisley DM, Sparto PJ, Whitney SL, Furman JM. Cervicogenic dizziness: a review of diagnosis and treatment. J Orthop Sports Phys Ther 2000; 30(12):755-766.
15. Hedera P, Bujdáková J, Traubner P. Blood flow velocities in basilar artery during rotation of the head. Acta Neurol Scand 1993; 88(3):229-233.
16. Petersen B, von Maravic M, Zeller JA, Walker ML, Kömpf D, Kessler C. Basilar artery blood flow during head rotation in vertebrobasilar ischemia. Acta Neurol Scand 1996; 94(4):294-301.
17. Olszewski J, Zalewski P, Machała W, Gaszyński W. [Administration of the cervical torsion test by the

examination of Doppler's blood flows in vertebral arteries and basilar artery in patients with degenerative cervical spine changes]. Otolaryngol Pol 1994; 48(6):549-555.

18. Machaly SA, Senna MK, Sadek AG. Vertigo is associated with advanced degenerative changes in patients with cervical spondylosis. Clin Rheumatol 2011; 30(12):1527-1534.

19. Alsanosi AA. Adaptation of the dizziness handicap inventory for use in the Arab population. Neurosciences (Riyadh) 2012; 17(2):139-144.

20. Reid SA, Callister R, Katekar MG, Treleaven JM. Utility of a brief assessment tool developed from the Dizziness Handicap Inventory to screen for Cervicogenic dizziness: A case control study. Musculoskelet Sci Pract 2017; 30:42-48.

21. L'Heureux-Lebeau B, Godbout A, Berbiche D, Saliba I. Evaluation of paraclinical tests in the diagnosis of cervicogenic dizziness. Otol Neurotol 2014; 35(10):1858-1865.

22. Grande-Alonso M, Moral Saiz B, Mínguez Zuazo A, Lerma Lara S, La Touche R. Biobehavioural analysis of the vestibular system and posture control in patients with cervicogenic dizziness. A cross-sectional study. Neurologia (Engl Ed) 2018; 33(2):98-106.

23. Kotait MA, Younes RL. Ocular vestibular evoked myogenic potentials (o-VEMPs) testing in cervicogenic vertigo and its relation to radiological findings: a correlation study. Hearing, Balance and Communication 2017; 15(4):235-243.

24. Durante A. Otoacoustic emissions. In: Bevilacqua M, Martinez M, Balen S, Pupo A, Reis A, Frota S (eds). Audiology Treaty. São Paulo, Brazil: Editora Santos; 2011. 145-158.

25. Li H, Zhong N. [Analysis of spectral history of distort-product otoacoustic emissions in subjects of transient vertebrobasilar ischemic vertigo]. Lin Chuang Er Bi Yan Hou Ke Za Zhi 1998; 12(5):217-220.

26. Reiley AS, Vickory FM, Funderburg SE, Cesario RA, Clendaniel RA. How to diagnose cervicogenic dizziness. Arch Physiother 2017; 7:12.

27. Kulkarni V, Chandy MJ, Babu KS. Quantitative study of muscle spindles in suboccipital muscles of human foetuses. Neurol India 2001; 49(4):355-359.

28. Boyd-Clark LC, Briggs CA, Galea MP. Muscle spindle distribution, morphology, and density in longus colli and multifidus muscles of the cervical spine. Spine (Phila Pa 1976) 2002; 27(7):694-701.

29. Cullen KE, Roy JE. Signal processing in the vestibular system during active versus passive head movements. J Neurophysiol 2004; 91(5):1919-1933.

30. Falla D. Unravelling the complexity of muscle impairment in chronic neck pain. Man Ther 2004; 9(3):125-133.

31. Magnusson M, Malmström EM. The conundrum of cervicogenic dizziness. Handb Clin Neurol 2016; 137:365-369.

32. Minguez-Zuazo A, Grande-Alonso M, Saiz BM, La Touche R, Lara SL. Therapeutic patient education and exercise therapy in patients with cervicogenic dizziness: a prospective case series clinical study. J Exerc Rehabil 2016; 12(3):216-225.

33. Yahia A, Ghroubi S, Jribi S, Mâlla J, Baklouti S, Ghorbel A, *et al*. Chronic neck pain and vertigo: Is a true balance disorder present? Ann Phys Rehabil Med 2009; 52(7-8):556-567.

34. Karlberg M, Johansson R, Magnusson M, Fransson PA. Dizziness of suspected cervical origin distinguished by posturographic assessment of human postural dynamics. J Vestib Res 1996; 6(1):37-47.

35. Reid SA, Callister R, Snodgrass SJ, Katekar MG, Rivett DA. Manual therapy for cervicogenic dizziness: Long-term outcomes of a randomised trial. Man Ther 2015; 20(1):148-156.

36. Duplan B, Lavignolle B. Posture humaine et rachis cervical. Rev Rhum 2008; 75(1):712–716.

37. Treleaven J. Dizziness, Unsteadiness, Visual Disturbances, and Sensorimotor Control in Traumatic Neck Pain. J Orthop Sports Phys Ther 2017; 47(7): 492-502.