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ORIGINAL ARTICLE

Effects of Radiotherapy and Chemotherapy on Vestibular Function in Patients with Head and Neck Cancer

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ABSTRACT

Background: The ototoxicity of both chemotherapy and radiotherapy has been studied. Little is known regarding their impact on the vestibular system, particularly, the laboratory vestibular testing that evaluates the effects of both chemotherapy and radiotherapy on the peripheral and central vestibular system. Consequently, the current research was designed to study Videonystagmography (VNG) and video head impulse test (vHIT)in patients with head and neck cancer receiving chemotherapy and radiotherapy for early diagnosis of vestibularsystem abnormalities.

Methods: This study included two groups with head and neck cancer: group I included 19 patients receiving chemotherapy and group II included 19 patients receiving radiotherapy. Basic audiological evaluation, VNG, and vHIT were conducted on all patients before treatment and 3 months following treatment.

Results: Regarding pure tone audiometry, there was a statistically significant increase in hearing threshold at high frequencies in patients receiving chemotherapy and radiotherapy before and after treatment. As regards the VNG test, there was a statistically significant impairment in saccade, smooth pursuit, optokinetic, and caloric tests in patients before and after receiving chemotherapy**.** However, in patients receiving radiotherapy, there was no statistically significant difference in the VNG test battery except for increased value of caloric weakness. vHIT abnormalities were reported in patients of both groups.

Conclusions:
This study demonstrated the ototoxic and vestibulotoxic effects of both chemotherapy and radiotherapy in patients with head and neck cancer.

Keywords:
Chemotherapy; Radiotherapy; Vestibular; Ototoxicity.

INTRODUCTION

Head and neck cancer is the sixth most common cancer in the world. More than a half million people develop head and neck cancer every year, and more than a quarter million die as a result. Surgery, chemotherapy, and radiotherapy are used as adjuvant or definitive treatments for head and neck cancer, either separately or in combination [1]. Any of these therapeutic approaches could have an impact on the auditory system and cause temporary or permanent hearing loss [2].Surgical treatment that involves neck dissection and tumor excision may result in hearing loss. On the other hand, because of their central location, the temporal bone and ear are frequently in radiation fields for skin cancers related to the nasopharynx, oropharynx, parotid gland, and periauricular area. Radiation toxicities, both acute and late, can affect any portion of the ear [3].

Furthermore, cisplatin is a highly efficient chemotherapeutic treatment against a range of life-threatening malignancies, but its ototoxicity is significant, limiting its use and dosage [4]. Ototoxicity is defined as drug-induced damage to the inner ear structures, which might include cochlear dysfunction, vestibular dysfunction, or both [5]. Cisplatin treatment has been linked to varying degrees of irreversible hearing loss, with reported rates ranging from 50-90% depending on patient demographics, medication dosage, and changes in tools and grading systems [6].

Vestibular dysfunction can significantly reduce quality of life [7] and impose a significant economic burden on individuals and society [8].According to recent studies, balance problems such as falls and mobility handicap are more frequent among those who have survived cancer than those without cancer [9]. Falling is a major cause of morbidity and mortality in the general population, which makes this significant [10].As a result, there is a need to raise awareness of balance problems among this susceptible population of cancer patients in order to provide effective preventative and intervention strategies [11].

The vestibular system function is evaluated using a variety of trials and tests. The results of which enableaccurate diagnosis of the underlying condition. Electrophysiologic tests, such as videonystagmography (VNG), are crucial in the diagnosis of vertigo and in distinguishing between central and peripheral vestibular system dysfunctions [12].To improve the sensitivity and specificity of the assessment battery in vestibular pathology distinction, recent tests were introduced. The video head impulse test (vHIT) is one of these tests. The vHIT is distinguished by its ease of use, fastness, practicality, and its ability to assess each semicircular canal separately [13].

Various studies of audiovestibular impairment in patients undergoing chemotherapy and radiotherapy have yielded inconsistent results. Consequently, the purpose of this study was to test audiovestibular function in patients having chemotherapy and radiotherapyfor head and neck cancers.

METHODS

Participants:

The Research Ethics Committee at the Faculty of Medicine, Zagazig University Hospitals approved this study with the number 9023-12-10-2021. Written informed consent was obtained from all participants after the test procedures had been explained. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

This observationalfollow-up study included adults of both genders with head and neck cancer. They ranged in age from 20 to 50 years. They divided into two groups according to sample size: group I included 19 patients who received chemotherapy and group II included 19 patients who received radiotherapy. This study was doneintheAudio-Vestibular Medicine Unit, ENTDepartment, Faculty of Medicine. Patients referred from the Clinical Oncology and Nuclear Medicine Department.

This study included histopathologically confirmed cases of head and neck malignancies in patients with head and neck cancers receiving radiotherapy or chemotherapy. It included cases with Karnofsky's score ≥80%. This score is astandard method for measurement of the ability of cancer patients to do ordinary tasks. The Kasnofsky performance status scores range from 0 to 100. The high scores mean that the patient is better able to do daily activities.

On the other hand, both groups did not includepatients with conductive hearing loss, a history of systemic diseases (e.g. uncontrolled hypertension, renalfailure, or hepatic failure), and a history suggesting vestibular neuritis, labyrinthitis, migraine, Meniere's disease, or any central nervous system disorder.

***Procedure*:**

1. Chemotherapy and radiotherapy regimen:

- All patients in group I received chemotherapy in the induction or metastatic setting (a platinum-containing chemotherapeutic regimen like cisplatin100mg/m^2 every 3 weeks or 30mg /m^2 every week or carboplatin AUC 5 every 3 weeks).
- Patients in group II were treated with definitive or adjuvant conformal three-dimensionalradiotherapy. The patients underwent computerized tomography (CT) simulation, in the supine position with head and neck fixation using athermoplastic mask. Multi-slice CTwas done every 3mm. linear accelerator was used with energy 6MV or 15 MV. Phase I was delivered using a total dose of 50 Gray (Gy)to the primary site and neck lymphatics by conventional fractionation (1.8-2 Gy per fraction, 5 times per week). Phase II the primary site or tumor bed and any positive neck nodes were boosted with 16 -20 Gy.

2. *Assessment*:

Before starting either radiotherapy or chemotherapyin all cases, the patient characteristics

(name, age, andsex of the patient), type of cancer, plan of treatment(radiotherapy/chemotherapy), and dose of radiotherapy and chemotherapy received wererecorded.All patients in this study were submitted to full history taking, basic audiological evaluation,and vestibular assessment, including VNG and vHIT. These tests were done before the treatment started and three months following treatment.

A- Basic audiological evaluation:

Pure-tone audiometry, using Amplaid 311 audiometer, included air and bone conduction estimated hearing threshold; air conduction in the frequency range of 250 to 8000 Hz and bone conduction from 500 to 4000 Hz. Speech audiometry included speech reception threshold (SRT) using Arabic spondee words and word discrimination score (WDS) using Arabic phonetically balanced words. Immittancemetry,using MADSEN, Zodiac 901immittancemeter, included both tympanometry and acoustic reflex threshold.

B- Vestibular evaluation including:

I.VNG

It was done using the VNG system, Ulmar, version 0.1. This test included: spontaneous nystagmus, gaze-evoked nystagmus, oculomotor tests (saccade, smooth pursuit, and optokinetic tests), positional, Dix-Hallpike, and caloric tests.

II.vHIT

ThevHIT was done usingEYE SEECAM vHIT from Interacoustics. Three planes were used to do the head impulses [horizontal, right anterior-left posterior (RALP), and left anterior-right posterior (LARP)]. Every direction and plane had examined with at least five head impulses.

The parameters measured in vHIT included the gain and refixation Saccade.The Gain representsthe ratio between eye velocity and headvelocity. The gain was considered normal when it was more than0.75 for anterior and posterior semicircular canals (SCCs) and more than 0.80 for horizontal SCCs without the presence of saccade. Conversely, the gain was considered abnormal if the reduced gain in at least one canalwas associated with the presence of saccade [14].A saccade isthe repositioning of the eyes on the target. Overt and covert saccades are the two types of saccades that may occur.Overt saccadeoccursafter the head impulse while the covert saccade occurs during head impulses [15].

STATISTICAL ANALYSIS

Analysis of the data was done using IMP SPSS version 26.0. Mean and standard deviation were used when presenting the continuous variables. Qualitative data were presented as frequencies and percentages. Paired t-test was used for comparison of the quantitative variable between the baseline and three months after treatment. When the p-value≤0.05, it was considered significant.

RESULTS

Two groups were included in this study: group I (patients receiving chemotherapy) and group II (patients receiving radiotherapy). The group receiving chemotherapy included 19 patients (9 females and 10males) and the mean age was 37±7.9 years.Thegroup receiving radiotherapy included 19 patients (8females and 11 males) and the mean age was 40.2±8.2 years. Table 1 shows the tumor localization and dose ofbothradiotherapy and chemotherapy for patients with head and neck cancer.

Regarding pure tone audiometry, there was astatistically significant differencein 2,4, and 8 kHz before and three months after receiving chemotherapy (Table 2). In patients receiving radiotherapy, there was astatistically significant difference in 4 and 8 kHz (Table 3). There was no statistically significant difference in SRT and WDsbefore and after receiving both chemotherapy and radiotherapy.

As regards the VNG test, there was a significant difference in saccade (velocity and accuracy), smooth pursuit(0.4 and 0.6 Hz), optokinetic (OPK) nystagmus, and unilateral caloric weakness in patients before and three months after receiving chemotherapy (Table 4).No reported cases with spontaneous, gaze-evoked, positional, or positioning nystagmus. In patients receiving radiotherapy, there was no significant difference in theVNG test battery except caloric weakness (Table 5).

The comparison of vHITbefore and after receiving chemotherapy revealed astatistically significant difference in lateral, posterior, and anterior canal gain (Table 6). In patients receiving radiotherapy, there was asignificant difference in Rt and Ltlateral and Lt anterior canal gain (Table 7). Overt or covert saccades were associated with reduced canal gain in both groups.

The number and percentage of patients with SNHLfollowing treatment in the chemotherapy groupwere 11 cases out of 19(58%), while in the radiotherapy group were 8 out of 19 (42%), andthe hearing loss was bilateral and symmetrical. Vestibular abnormalities were reported in 10 patients (52%) with chemotherapyand 7 patients (36%) with radiotherapy. Regarding VNG

abnormalities in the chemotherapy group, there were three cases with central, two with peripheral, and five with combined central and peripheral vestibular dysfunction.In the radiotherapy group, there were two cases with central and five with peripheral vestibular dysfunction. vHIT abnormalities were reported in nine patients with chemotherapy and six cases with radiotherapy.

Table (1): Tumor localization for patients receiving chemotherapy and radiotherapy.

Tumor localization	Number of radiotherapy patients	Radiation dose (gray)	Number of chemotherapy Patients	Chemotherapy dose range
Oropharyngeal	4	60	3	Cisplatin (160 -180) mg every 3 weeks
Oral cavity	3	63	4	Cisplatin (50 -70)mg every week
Sinonasal	2	66	2	Carboplatin 150 mg every week
Laryngeal	6	60	5	Cisplatin (120 -135) mg every 3 weeks
Hypopharyngeal	4	66	5	Carboplatin (150 -190) mg every 3 weeks

Table (2): Comparison of pure tone audiometry before and after receiving chemotherapy.

Pure tone threshold (dBHL)		Baseline mean ± SD	3 months follow-up mean ± SD	Paired t-test	p-value
250	Rt	12.4±5.5	13.6±4.6	1.6	0.11
	Lt	13.2±6.1	13.7±4.1	0.6	0.5
500	Rt	12.2±6.3	13.8±5.1	0.7	0.6
	Lt	13.5±4.9	13.8±5.2	0.6	0.5
1000	Rt	12.8.3±4.7	14.9±6.3	0.8	0.7
	Lt	11.9±3.8	14.8±7.2	0.6	0.5
2000	Rt	11.6±5.4	25.8±6.5	5.7	<0.001**
	Lt	12.5±5.2	22.8±7.2	5.9	<0.001**
4000	Rt	13.8±4.6	40.5±11.6	6.3	<0.001**
	Lt	14.1±5.1	39.1±10.2	5.9	<0.001**
8000	Rt	13.2±5.7	38.6±9.1	6.8	<0.001**
	Lt	15.3±6.4	42.7±9.3	7.5	<0.001**

**Statistically highly significant difference (p< 0.001)

Table (3):Comparison ofpure tone audiometry before and after receiving radiotherapy.

Pure tone threshold (dBHL)		Baseline mean ± SD	3 months follow up mean ± SD	Paired t-test	p-value
250	Rt	11.1±5.2	12.5±3.6	0.7	0.6
	Lt	12.2±5.1	13.6±4.4	1.6	0.1
500	Rt	12.3±4.3	13.2±5.2	0.6	0.5
	Lt	11.5±4.9	12.8±4.1	0.8	0.7
1000	Rt	12.8.3±4.5	13.9±3.1	1.1	0.2

	Lt	10.9±3.8	11.8±3.8	0.9	0.3
2000	Rt	11.6±5	13.7±3.5	1.7	0.8
	Lt	12±5.1	14.8±4.2	1.6	0.1
4000	Rt	13.4±4	27.5±11.6	5.8	<0.001**
	Lt	14±5.1	35.1±10.2	6.6	<0.001**
8000	Rt	14.2±5.6	36.6±9.1	6.8	<0.001**
	Lt	16.3±6.4	38.7±9.3	6.9	<0.001**

**Statistically highly significant difference (p < 0.001)

Table (4): Comparison of VNG before and after receiving chemotherapy.

VNGtestbattery	Parameters	Baseline mean ± SD	Follow up mean ± SD	Paired t-test	p-value
Saccade	**Rt latency**	197(29)	230(30)	1.1	0.2
	Rt velocity	352(31)	279(40.5)	2.7	0.01*
	Rt accuracy	90(5.7)	70.7(5.5)	4.7	<0.001**
	Lt latency	198(27)	204(28.5)	1	0.3
	Lt velocity	355(34)	250(45)	4.7	<0.001**
	Lt accuracy	90.1(5.75)	72.5(5.6)	5	<0.001**
Smooth pursuit	**Rt 0.3 Hz gain**	0.99(0.2)	0.9(0.25)	0.9	0.3
	Rt0.4 Hz gain	0.97(0.23)	0.85(0.3)	0.59	0.5
	Rt 0.6 Hz gain	0.8(0.25)	0.62(0.31)	6.1	<0.001**
	Lt 0.3 Hz gain	0.98(0.21)	0.94(0.22)	1	0.30
	Lt 0.4 Hz gain	0.96(0.2)	0.9(0.29)	0.8	0.44
	Lt0.6 Hz gain	0.84(0.27)	0.6(0.28)	6	<0.001**
OPK	**Rt gain**	0.91(0.16)	0.7(0.3)	5.5	<0.001**
	Lt gain	0.94(0.17)	0.68(0.32)	5.6	<0.001**
UW		10(4.1)	25.7(8.8)	8	<0.001**
DP		9.1(3.4)	9.7(3.6)	0.6	0.94

**Statistically highly significant difference (p<0.001).
Abbreviations:OPK:optokinetic,UW:unilateral weakness, DP: Directional preponderance.

Table (5): Comparison of VNG before and after receiving radiotherapy.

VNGtest battery	Parameters	Baseline mean ± SD	Follow up mean ± SD	Paired t-test	p-value
Saccade	**Rt latency**	195(27)	204(30)	1.3	0.19
	Rt velocity	350(30)	343(32.5)	0.9	0.31
	Rt accuracy	91(4.7)	89.7(5.5)	0.6	0.22
	Lt latency	197(28)	204(28.5)	1.2	0.21
	Lt velocity	352(31)	345(31)	0.9	0.35
	Lt accuracy	91.1(5.7)	89.5(5.6)	0.4	0.61
Smooth pursuit	**Rt 0.3 Hz gain**	0.99(0.23)	0.95(0.25)	0.7	0.46
	Rt0.4 Hz gain	0.97(0.2)	0.94(0.24)	0.5	0.6
	Rt 0.6 Hz gain	0.95(0.25)	0.92(0.26)	0.48	0.62
	Lt 0.3 Hz gain	0.97(0.24)	0.94(0.22)	0.8	0.44
	Lt 0.4 Hz gain	0.95(0.2)	0.92(0.23)	0.8	0.44
	Lt0.6 Hz gain	0.94(0.23)	0.88(0.24)	1	0.29
OPK	**Rt gain**	0.91(0.16)	0.89(0.2)	0.4	0.64
	Lt gain	0.94(0.18)	0.91(0.21)	0.13	0.39
UW		11(4.2)	20.7(6.1)	6	<0.001**
DP		9.5(3.5)	10.7(3.8)	0.5	0.64

**Statistically highly significant difference (p< 0.001).
Abbreviations: OPK: optokinetic,UW: unilateral weakness, DP: Directional preponderance.

Table (6): Comparison of vHIT before and after receiving chemotherapy.

vHIT	Baseline	Follow up	Pairedt-test	p-value
Rt lateral gain	0.9±0.15	0.68±0.2	7.5	<0.001**
Rt anterior gain	0.95±0.11	0.72±0.22	6.7	<0.001**
Rt posterior gain	0.98±0.19	0.68±0.24	8.1	<0.001**
Lt lateral gain	0.91±0.21	0.64±0.29	7.4	<0.001**
Lt anterior gain	0.95 ±0.19	0.65±0.28	8	<0.001**
Lt posterior gain	0.93±0.16	0.67±0.29	6.9	<0.001**

**Statistically highly significant difference (p< 0.001)

Table (7): Comparison of vHITbefore and after receiving radiotherapy.

vHIT	Baseline	Follow up	Pairedt-test	p-value
Rt lateral gain	0.89±0.16	0.6±0.28	7	<0.001**
Rt anterior gain	0.97±0.1	0.96±0.18	1.2	0.2
Rt posterior gain	0.95±0.19	0.94±0.12	0.9	0.3
Lt lateral gain	0.9±0.2	0.61±0.26	7.1	<0.001**
Lt anterior gain	0.91 ±0.18	0.63±0.28	7.3	<0.001**
Lt posterior gain	0.89±0.15	0.9±0.14	1.9	0.6

**Statistically highly significant difference (p< 0.001)

DISCUSSION

This study investigated the cochlear and vestibular function in patients receiving chemotherapy and radiotherapy in patients with head and neck cancer.

In the present study, as shown in Tables 2 and 3 during audiometric evaluation, the patients had normal hearing sensitivity before therapy. During subsequent follow-up at three months after treatment, there was hearing loss at high frequencies.

These findings corroborated previous studies, which reported that hearing loss was more common at high frequencies than at low frequencies [16, 17]. The first damage occurs at the base of the cochlea, where high-frequency sounds are processed. Subsequent exposure causes damage to proceed to the cochlear apex, where low-frequency sounds are processed [18].This damage results from the relatively low stores of glutathione (antioxidant agent) in the outer hair cell of the basal turn in comparison to the apical part. The less antioxidant capacity in the basal part leads to more susceptibility to ototoxicity [19].

Chemotherapy-induced toxicity is defined by the production of hazardous reactive oxygen species in the cochlea, which leads to loss of cochlear hair cells and damage to stria vascu **Zagazig** laris and the spiral ganglion. Cochlear hair cell damage is frequently bilateral, dose-dependent, and irreversible [20].

On the other hand, sensor neural hearing loss (SNHL) following radiation exposure varies from no hearing impairment to 54% involvement [21]. The precise cause ofSNHL is unknown; however, it is believed to be related to direct damage to the cochlear apparatus or damage to small capillaries that result in hypoxia affecting inner ear components. Also, radiation damage to the brainstem may indirectly cause hearing loss. Radiation-induced SNHL Frequently progresses over time and is irreversible. It might start during the acute phase of treatment or take years to manifest [22].

Cochleotoxicity following the treatment with chemotherapy and radiotherapy is well documented, but the potential for vestibulotoxicity is still unclear. Accordingly, the evaluation of vestibulotoxicity in the existing study included objective tests (VNG and vHIT).

As shown in Table (4), there were statistically significant differences as regards saccade, smooth pursuit, and OPK nystagmus in patients before and

three months after receiving chemotherapy. Moreover, patients receiving chemotherapy showed canal paresis, which reflects peripheral and central vestibular affection. On the other hand, inpatients receiving radiotherapy, there was no significant difference in the VNG test battery except for caloric weakness (Table 5) (only two patients had central affection).

Besides the peripheral end organs, additional parts of the central vestibular system may be affected by toxicity. Toxicity in the brainstem could thus explain the above-mentioned outcomes. On the one hand, the vestibular cores could be vulnerable to the toxicity caused by cisplatin. Additionally, radiation therapy may cause damage to the vestibular cores. For example, radio logically generated brainstem lesions and myelitis are documented consequences of chemo radiation in head and neck malignancies [23].

In contrast to our findings, Nilakhe [21] stated that none of the patients in his studydemonstrated canal paresis or directional preponderance as a result of radiation onvestibular function throughout the six-month follow-up period. Only two (4%) participants experienced vertigo, which was temporary and improved with treatment. This, could be due to the difference in radiation doses between this study and our study.

The vHIT provides an objective and quick measure of the vestibular-ocular reflex and efficiently assesses the dizzy patient to identify if dizziness is due to a vestibular disorder[24].Table (6) showedasignificant difference in the gain of the three semicircular canals before and three months after chemotherapy. In patients receiving radiotherapy, there was a significant difference in RT and Lt Lateral and Lt anterior canal gain (Table 7). Moreover, overt or covert saccades were associated with reduced canal gain. In agreement with these results, research by Hulse et al. [25] showed a statistically significant reduction in median gain vHIT in six weeks followingchemoradiation, and more refixation saccades were identified.

The current study has some limitations because it was done with a small group of patients and only a follow-up for three months. Follow-up for a long time may be needed becausechemotherapystays in the body for an extended period and shows if they might get other balance problems, like benign paroxysmal positional vertigo, and ensures early management of these problems.

CONCLUSIONS

Our findings demonstrated that chemotherapy and radiotherapy can produce auditory and vestibular impairment in patients with head and neck cancer. For medical and legal reasons, it's important to do both auditory and vestibular testing on susceptible populations.

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