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Vitamin D level in relation to phonetic function among subacute stroke patients

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Abstract

There are many stroke patients with decreased phonation ability. Vitamin D is associated with weakness in muscle power and a decreased function of activity and is often accompanied by a deficiency of serum vitamin D in stroke patients. This study was conducted to evaluate the correlation between serum vitamin D level and phonetic function in subacute stroke patients. Among subacute stroke patients, patients with dysphonia were retrospectively recruited. Phonation function was assessed by acoustic analysis using the dysphonia severity index (DSI) and maximum phonation time for 4 corner vowels/a/,/i/,/u/, and/ae/. As a statistical method, the relationships of vitamin D levels with the maximum phonation time and DSI were evaluated using Pearson's correlation analysis and linear regression analysis. A total of 32 stroke patients with dysphonia were assessed. A positive correlation was found between vitamin D levels and the DSI of /a/, /u/, /i/, and/ae/. The DSI of/u/ was significantly lower in the group with vitamin D deficiency. Vitamin D level was associated with phonation function and its deficiency may be a factor in predicting phonation severity in stroke patients.

Abbreviations: DSI = dysphonia severity index, I-Low = lowest intensity, MPT = maximum phonation time, VDR = vitamin D receptor.

Keywords: dysphonia, maximum phonation time, stroke, vitamin D

1. Introduction

Dysphonia refers to disorders of the voice such as hoarseness, weakness, or fatigue.[1] After a stroke, including ischemic and hemorrhagic stroke, vocal fold paralysis, and vocalization-related muscle weakness may occur. The change in voice in stroke patients has various characteristics depending on the location of the brain lesion.[2] It is not common for vocal fold paralysis to occur directly from a stroke, but it is commonly associated with lesions in the brain stem area.[2] According to previous study, it is reported that vocal fold paresis occurs in about 20% of stroke patients.[3]

Vitamin D deficiency can be defined as a concentration of 25-hydroxyvitamin D (25(OH)D) in the blood of less than 20 ng/mL.[4] The 25(OH)D levels tend to be lower in stroke patients than in the healthy elderly population.[5] Vitamin D deficiency is associated with a decreased muscle power and function of activity;[6,7] several studies have shown a relationship between vitamin D deficiency and muscle function and endurance.[8,9] Vitamin D affects the synthesis of muscle cytoskeletal proteins such as calmodulin and insulin-like growth factor binding protein.[10,11] It affects the metabolism of phospholipids, which has the potential to affect muscle contraction, and stimulates muscle proliferation and differentiation.[12] It also controls cellular processes such as myogenesis and cell proliferation through activation of the mitogen-activated protein kinase signaling pathway.[13] Consequently, vitamin D deficiency is related to muscle fiber atrophy, fibrosis, and fat infiltration.[6] These findings cause muscle fatigue, prolong muscle relaxation time, and decrease maximal muscle contractility and physical activity. The laryngopharyngeal complex is a musculoskeletal system that is possibly just as susceptible to vitamin D deficiency-induced muscle dysfunction as other muscles. Since vocalization is achieved by complex interactions of neuromuscular processes, a decrease in laryngeal muscle function can lead to dysphonia.[14,15]Previous studies also reported that there was no significant difference in phonetic dysfunction in the group with vitamin D insufficiency and in the normal group.[15] However, there was a limitation in the previous study because it evaluated the phonatory symptoms via a questionnaire.

There has been no research on the relationship between vitamin D levels and parameters that measure phonation function through the maximum phonation time (MPT) and dysphonia severity index (DSI) in stroke patients. The MPT keeps vocalizations as long as possible, and thereby, provides an assessment of vocal fold function.[16] The vocal folds have several functions, such as the production of sound, regulation of airflow into the lungs, and protection of the airway from foreign materials. DSI was developed to quantitatively analyze phonetic functions of speech.[17] This indicator consists of the highest fundamental frequency (F0-High), lowest intensity (I-Low), MPT, and jitter. Due to the abnormality of the vocal fold coordination, the heterogeneity of the vocal fold inhibits the higher vibration rates

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and the F0-High decreases. It also increases the resistance of the vocal fold, making it require greater drive pressure to initiate and maintain vocalization.[18] Therefore, the I-low increases in patients with dysphonia. The jitter is an evaluation of perturbation, which measures the irregularity of vibration of the vocal fold. In dysphonic patients, it is difficult to maintain speech, and jitter increases due to irregular voice control. The muscles that control the vocal fold when vocalizing are different for each vowel.[19] Therefore, when performing MPT and DSI evaluation, it is helpful to evaluate not only the/a/ vowel but also corner vowels such as/i/,/u/, and/ae/.

The objective of this study was to analyze the relationships between vitamin D levels and the DSI and MPT in subacute stroke patients to see if there was a correlation between phonetic function and vitamin D levels.

2. Methods

2.1. Subjects

From September 2019 to April 2020, a retrospective study was conducted on subacute stroke patients with dysphonia admitted at the Kyung Hee University Hospital at Gangdong. Using the GRBAS scale,[20] one speech therapist and 2 rehabilitation specialists evaluated dysphonia. Patients with grade 2 or higher were included in the evaluation. Patients who underwent a voice assessment and were tested for blood vitamin D levels were selected. Using the multidimensional voice program Model 3950 (Kay Pentax, Montvale, NJ), voices were analyzed. The voice input was obtained using an SM48 microphone (SHURE, Niles, IL). The background noise level (less than 30 dB) was maintained through monitoring and the mouth-to-microphone distance was maintained at 4 cm. The inclusion criteria were the following: no history of stroke, no previous vitamin D supplementation, Patients within 3 months of onset of stroke, a Mini-Mental State Examination score of 20 or higher to have a cognitive function capable of coordinating speech evaluation, and over 20 years old. Exclusion criteria included: existing history of speech disorder due to causes other than stroke, aphasia, tracheostomy, history of hip or spine fracture, history of steroid therapy that may be a secondary cause of vitamin D deficiency.[21] To analyze the differences in phonetic function between subgroups according to brain lesions, the brainstem lesion group and the non-brainstem lesion group were divided. In addition, although vitamin D levels are a continuous variable, groups were divided and compared to analyze whether there was a difference in phonetic function between the vitamin D deficiency group and the normal group. The study was conducted according to a protocol approved by the Institutional Review Board of Kyung Hee University Hospital at Gangdong, Korea (Institutional Review Board approval number: 2020-06-006).

2.2. Maximum phonation time

After a maximum inspiration, the study participants vocalized the 4 corner vowels/a/,/ae/,/i/, and/u/ for as long as possible. Participants were instructed to speak at a comfortable loudness that is used in everyday life. The timeframe wherein the vocalization was sustained at a constant intensity with an audible sound was recorded. Participants were asked to sit in a comfortable position, and the test was conducted 3 times. The longest of the 3 results was recorded in the MPT.[16] MPT was measured in seconds (s).

2.3. Jitter

The participants were instructed to continue vocalization on/a/,/ae/,/i/ and/u/ vowels for 3 seconds at comfortable voice strength and pitch. The voice test was repeated 3 times, and jitter was recorded at 5 kHz for each vowel. The multidimensional voice program was then used to assess jitter.[22]

2.4. Highest fundamental frequency

The participants were asked to vocalize/a/,/ae/,/i/, and/u/ vowels to a habitual pitch used in their daily lives and then directed to gradually raise this pitch to the highest possible frequency. Afterwards, using the multidimensional voice program, the F0-High was obtained.[22]

2.5. Minimum intensity

The participants were asked to vocalize the vowels/a/,/ae/,/i/, and/u/ vowels to a habitual pitch used in their daily lives, and then directed to decrease the intensity gradually for 5 seconds to the level of whispering. The I-Low was then measured.[22]

2.6. Dysphonia severity index

A more negative DSI value indicates more severe dysphonia, and a more positive value indicates a better phonic function. Severe patients may have values lower than -5, and normal individuals may have values higher than + 5.[17] The F0-High, MPT, I-Low, and Jitters are subjected to acoustic analysis through a multidimensional voice program. The DSI is then calculated using the following formula.[23]

$$DSI = 0.13 \times MPT + 0.0053 \times F0\text{-}High — 0.26 \times I\text{-}Low — 1.18 \times Jitter + 12.4$$

2.7. Serum vitamin D level

Serum 25(OH)D measurements were taken using the Architect 25-OH D vitamin kit (Abbott Diagnostics, Lake Forest, IL), which was collected within 3 months of stroke onset.

2.8. Modified Barthel Index

The ten-item Modified Barthel Index (MBI) scale was used to evaluate the patient's activities of daily living, which included bathing, eating, modifying, and dressing.[24] The total score ranges from 0 to 100, with lower scores indicating less independence.

2.9. Statistical analysis

Statistical analyses of variables were performed using SPSS version 25.0 for Windows (IBM Corp., Armonk, NY). The Kolmogorov-Smirnov test was conducted to establish the normal distribution of the data, and the Levene test was conducted to examine the homogeneity of the variance. An independent *t* test was performed to compare the MPT and DSI in subgroups according to the location of stroke lesion. Through the Pearson's correlation analysis, the association of serum vitamin D levels with the DSI and MPT was analyzed. Linear regression analysis was performed to determine the effect of serum vitamin D levels on the MPT and DSI. A *P*-value less than .05 was considered statistically significant in all statistical tests.

3. Results

3.1. Participants baseline characteristics

Among the patients hospitalized for stroke in our rehabilitation department, 34 patients with symptoms of dysphonia were included in the study. Of these patients, 18 were male and 16 were female, and their average age was 65.52 ± 13.72 years (range from 33 to 88 years). With regards to the type of stroke, there were 22 patients with ischemic stroke and 12 patients with hemorrhagic stroke. Furthermore, there were 22 patients with brain stem lesions and 12 patients with non-brain stem lesions. The mean values of the MPT, DSI, MBI and Mini-Mental State Examination data are shown in Table 1.

3.2. Comparison of the MPT and DSI among subgroups

The DSI of/a/,/i/, and/u/ showed statistically significant differences between groups, with lower results in the brain stem lesion subgroup (Table 2). The DSI of/u/ showed statistically significant differences between groups, with lower results in the vitamin D deficiency group (Table 3).

3.3. Correlation of the MPT and DSI of/a/,/u/,/i/, and/ae/ vowels with vitamin D level

As a result of the correlation analysis, serum vitamin D level was statistically related to the DSI of/a/ (R = 0.380),/u/ (R = 0.639),/i/ (R = 0.486), and/ae/ (R = 0.375) (Table 4). However, it had no statistically significant association with the MPT of/a/,/u/,/i/, and/ae/ (Table 5). MBI was statistically related to the DSI of/a/ (R = 0.161),/u/ (R = 0.262),/i/ (R = 0.310), and/ae/ (R = 0.163) (Table 4). However, it had no statistically significant association with the MPT of/a/,/u/,/i/, and/ae/ (Table 5).

Table 1

Clinical manifestation of patients.

Characteristic	Value
Age (yr)	65.52 ± 13.72
Sex	
Male	18 (52.90)
Female	16 (47.10)
Type of stroke	
Ischemic	22 (64.70)
Hemorrhagic	12 (35.30)
Lesion location	
Brain stem	22 (64.70)
Nonbrain stem	12 (35.30)
MBI	42.93 ± 25.96
MMSE	24.44 ± 3.49
Vitamin D	19.65 ± 9.23
MPT of/a/	10.73 ± 7.68
MPT of/i/	9.82 ± 6.68
MPT of/u/	9.45 ± 6.41
MPT of/ae/	9.44 ± 6.56
DSI of/a/	−1.85 ± 2.68
DSI of/i/	−2.59 ± 2.48
DSI of/u/	−2.55 ± 2.74
DSI of/ae/	−2.81 ± 2.26

Values are presented as the mean ± standard deviation or number (%).
DSI = dysphonia severity index, MBI = modified Barthel index, MMSE = Mini-Mental State Examination, MPT = maximal phonation time.

Table 2

Comparison of the MPT and DSI among subgroups categorized by lesion location.

	Brain stem (n = 22)	Nonbrain stem (n = 12)	*P* value
Vitamin D	16.49 ± 5.78	25.46 ± 11.61	.055
MPT of/a/	9.15 ± 5.98	13.63 ± 9.71	.165
MPT of/i/	8.14 ± 5.00	12.91 ± 8.33	.044
MPT of/u/	7.99 ± 4.84	12.13 ± 8.16	.072
MPT of/ae/	8.68 ± 6.21	10.82 ± 7.22	.372
DSI of/a/	−2.6164 ± 1.72611*	−0.4675 ± 3.57146*	.024
DSI of/i/	−3.3555 ± 2.09465*	−1.1958 ± 2.63031*	.013
DSI of/u/	−3.3059 ± 2.51828*	−1.1667 ± 2.68497*	.027
DSI of/ae/	−3.0318 ± 2.06103	−2.4175 ± 2.65008	.458

Values are presented as the mean ± standard deviation.
DSI = dysphonia severity index, MPT = maximal phonation time.
*P < .05.

In linear regression analysis, vitamin D level was a significant predictor of the DSI of/a/ (β = 0.361, P = .042, R^2 = 0.101),/u/ (β = 0.590, P < .001, R^2 = 0.327),/i/ (β = 0.485, P = .005, R^2 = 0.209), and/ae/ (β = 0.417, P = .017, R^2 = 0.164) (Table 6).

4. Discussion

In this study of stroke patients with dysphonia, the association of serum vitamin D levels with the MPT and DSI measured by acoustic analysis. Vitamin D level and MBI were significantly and positively correlated with DSI. Vitamin D level was also found to be a significant factor influencing DSI. These results show that serum vitamin D levels in stroke patients are related to the DSI as reflection of phonetic function. On the other hand, vitamin D levels were found to have no significant association with the MPT; this may be because the MPT reflects not only vocal fold function but also pulmonary function as well.[16] In stroke patients, pulmonary function may be deteriorated by many factors, such as long bedtime and muscle weakness.[25,26] Because our analysis uses DSI values which can objectively evaluate phonetic function, the correlation between phonetic function and serum vitamin D level could be confirmed. In addition, the DSI for/u/ and/i/ showed strong correlations with vitamin D levels. This seems to be similar to our previous findings that high vowels like/u/ and/i/ more sensitively reflect phonation function compared to low vowels like/a/ and/ae/.[27] High vowels such as/u/ and/i/ have high supraglottic impedance, slow intraoral pressure discharge, and increased larynx position, which increases the tension in the vocal fold.[27] The comparison of brain stem and non-brain stem lesion subgroups showed that the DSI was significantly larger in the brain stem lesion subgroups, similar to previous studies.[2,28,29]

Vitamin D has an important effect on musculoskeletal function related to physical performance[30]; it is also associated with muscle strength, endurance, fatigability, and frailty.[8,9] Several mechanisms have been proposed, involving genomic and non-genomic pathways. Calcitriol, or 1,25-(OH)-2D, activates

Table 3

Comparison of the MPT and DSI among subgroups categorized by serum vitamin D level.

	Vitamin D deficiency group (n = 21)	Normal group (n = 13)	*P* value
MPT of/a/	10.24 ± 6.56	11.53 ± 9.44	.642
MPT of/i/	9.39 ± 5.78	10.52 ± 8.10	.639
MPT of/u/	8.78 ± 4.86	10.53 ± 8.46	.447
MPT of/ae/	9.16 ± 5.76	9.88 ± 7.92	.762
DSI of/a/	−2.5230 ± 2.4705	−0.7846 ± 2.7752	.066
DSI of/i/	−3.6760 ± 2.0380	−0.7327 ± 2.8188	.198
DSI of/u/	−3.0296 ± 2.2927**	−1.8885 ± 2.7206**	.001
DSI of/ae/	−3.1383 ± 1.9242	−2.2952 ± 2.7327	.299

Values are presented as the mean ± SD.
MPT, maximal phonation time; DSI, dysphonia severity index.
**P < .01.

Table 4

Correlation analysis of serum vitamin D level with the DSI.

	DSI of/a/	DSI of/u/	DSI of/i/	DSI of/ae/
Vitamin D	R = 0.380*	R = 0.639**	R = 0.486**	R = 0.375*
	P = .027	P < .001	P = .004	P = .029
MBI	R = 0.161*	R = 0.262**	R = 0.310**	R = 0.163*
	P = .042	P = .008	P = .003	P = .041

DSI = dysphonia severity index, MBI = modified Barthel index; r, correlation coefficient.
*P < .05, **P < .01.

Table 5

Correlation analysis of serum vitamin D level with the MPT.

	MPT of/a/	MPA of/u/	MPT of/i/	MPT of/ae/
Vitamin D	$R = 0.105$	$R = 0.254$	$R = 0.147$	$R = 0.052$
	$P = .556$	$P = .147$	$P = .407$	$P = .770$
MBI	$R = 0.035$	$R = 0.139$	$R = 0.076$	$R = 0.215$
	$P = .847$	$P = .449$	$P = .678$	$P = .238$

MBI = modified Barthel index; *r*, correlation coefficient, MPT = maximal phonation time.
*$P < .05$, **$P < .01$.

Table 6

Linear regression analysis of factors correlated with the DSI.

	Standardized β	*P* value	Adjusted R^2
DSI of/a/			0.101
Vitamin D	0.361	.042*	
MBI	0.005	.978	
DSI of/u/			0.327
Vitamin D	0.590	<.001**	
MBI	0.207	.166	
DSI of/i/			0.209
Vitamin D	0.485	.005**	
MBI	0.023	.891	
DSI of/ae/			0.164
Vitamin D	0.417	.017*	
MBI	0.261	.125	

Variables are based on their order of listing in multiple linear regression analysis.
DSI, dysphonia severity index; MBI, modified Barthel index.
*$P < .05$, **$P < .01$.

the nuclear vitamin D receptor (VDR), and through a transcription factor, this initiates the genomic pathway and increases the synthesis of muscle cell skeletal proteins, similar to calmodulin and growth factor binding proteins which are important for musculoskeletal function. Moreover, the activation of VDR is thought to affect phospholipid metabolism, an important process in muscle contraction. On the other hand, the nongenomic pathway is initiated due to the activated plasma membrane VDR. Signal pathway sequencing in the nongenomic pathway is faster than the protein synthesis time in the genomic pathway. The activation of the mitogen-activated protein kinase signaling pathway also explains the action of vitamin D, which affects the musculoskeletal system. This pathway is involved in the process of muscle differentiation and proliferation. In our findings, muscles take longer to relax, resulting in maximized contraction and, eventually, reduced physical performance.

The length and tension of the vocal fold muscles depend on the type of vowel. The fundamental frequency (F0) varies depending on the horizontal position of the hyoid bone and the vertical position of the thyroid cartilage, which has different characteristics for each vowel.[31,32] When vocalizing high vowels such as /u/ and/i/, tongue muscles such as the geniohyoid and genioglossus contract. The hyoid bone is pulled forward and the thyroid cartilage is tilted upwards and forwards. Therefore, as the vocal fold is pulled forward, there is vertical tension of the vocal fold. From the superior laryngeal structure, the laryngeal nerve is activated by sensory stimulation, and the F0 increases by increasing the tension of the vocal fold.[33–35] Because the tension of the vocal fold increases with the change of the horizontal position of the larynx, the onset time of vocalizing high vowels is longer than when vocalizing low vowels.[31]

In addition, studies by vowel electroglottography have reported that the low vowel/a/ increases the closed quotient compared to the high vowel/i/.[30] The narrowed oral cavity lowers the pressure of the air passing through the vocal fold and slows the contact speed of the vocal fold.[30] When vocalizing low vowels, the larynx rises as the hyoglossus contracts. On the other hand, when vocalizing high vowels, the genioglossus contracts, raising the tongue dorsum, the hyoid bone descends, and the tongue base indirectly lowers the larynx. As a result, when the vocal fold vibrates, the vertical contact surface becomes larger and the closed quotient increases. Therefore, it can be thought that the evaluation of high vowels like/i/ and/u/ is more sensitive to vocal function and stability than is the evaluation of low vowels like/a/ and/ae/.

The phonation process is driven by complex neurological and skeletal system functions, and vitamin D deficiency can affect this complex neuroskeletal system.[36] Therefore, these results indicate that treating vitamin D deficiency may be helpful in the treatment of dysphonia in stroke patients.

This study has several limitations. First, it is a retrospective study with a small number of participants. Prospective studies are needed, and studies on phonation functions before and after treatment through vitamin D replacement will be needed. Second, vitamin D measurements were performed within 3 months of the onset of stroke, but it was not known whether the vitamin D deficiency had persisted for a long time before the onset. Finally, factors that can influence dysphonia, such as smoking, were not considered.

In conclusion, serum vitamin D level seems to be related to the phonation function in stroke patients, and these results may be evidence that the measurement of serum vitamin D level in stroke patients is helpful for rehabilitation.

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