ORIGINAL ARTICLE

Clinical Significance of Vestibular Aqueduct's Width in Pediatric Patients with Sensorineural Hearing Loss

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OBJECTIVE: To assess the clinical significance of vestibular aqueduct (VA) width measurement on high resolution CT images in pediatric patients with sensorineural hearing loss (SNHL).

MATERIALS AND METHODS: 34 pediatric patients with sensorineural hearing loss (Group I) and 42 controls without SNHL (Group II) underwent otorhinolaryngological, otoneurological examinations, full audiological, sinusoidal harmonic acceleration evaluation and high resolution CT scan of temporal bone. VA width was measured at coronal and axial planes (at both midpoint and opercular levels).

RESULTS: In control group, vestibular aqueduct mean width was [0.80 and 0.71] at right and left midpoint, [1.6 mm] at opercular level and (1.4 mm at right and 1.5 at left side) on coronal image. Enlarged vestibular aqueduct was in nineteen (55.9%) patients with SNHL. VA mean width was [2.62 and 2.35] at midpoint, [3.31 and 3.27 mm] at opercular level, and (2.8 mm and 3.24) on coronal images at right and left sides respectively.

Sinusoidal Harmonic Acceleration (SHA) was abnormal in (47%) of children with SNHL; (68.8%) of them had enlarged VA.

CONCLUSION: VA is enlarged; if it exceeded (1.2), (2.1) and (1.5) mm at midpoint, opercular level and on coronal images respectively. Incidence of enlarged vestibular aqueduct is higher with severe to profound hearing loss. Incidence of gain abnormality in rotational testing is common in patients with enlarged vestibular aqueduct. Most children with enlarged vestibular aqueduct have combined audio-vestibular dysfunction.

The vestibular aqueduct (VA) is a small bony canal of labyrinthine capsule linking the medial wall of the vestibule of the inner ear to the posterior part of the petrous temporal bone. It houses the endolymphatic duct and includes part of the endolymphatic sac [1].

Large vestibular aqueduct syndrome is one of the common CT findings in children with SNHL [2]. Other deformities of otic capsule can also be found in up to 25% of young patients with unexplained hearing loss [3].

From a clinical standpoint, large vestibular aqueduct syndrome (LVAs) seems to be a unique clinical condition characterized by SNHL, frequently progressive, in children whose temporal bone CT studies demonstrate an enlarged VA. Accompanying abnormalities of the cochlea, modiolus, vestibule, and semicircular canals are frequent [4] .Several authors have independently reported a relationship among head trauma, changes in barometric pressure, and progression of SNHL in this syndrome [5, 6]. Although the risk remains undefined, most otolaryngologists caution patients with LVAs against high-risk activities, such as participation in contact sports, and advise the use of protective headgear when participating in moderate-risk activities [7].

High-resolution temporal bone computed tomography (CT) has been the first-line imaging study of choice obtained by pediatric otolaryngologists in the workup of children with all types of SNHL, including symmetric, asymmetric, and unilateral SNHL [8 -12]. Enlarged vestibular aqueduct is the most usual abnormality of the inner ear, which can be diagnosed with CT [13].

Valvassori and Clemis [14] measured the VAs of 3700 patients by using hypocycloidal polytomography and reported that a midpoint VA width of greater than 1.5 mm is abnormal.

Our aim was to assess the clinical significance of vestibular aqueduct width measurement on high-resolution CT images in pediatric patients with sensorineural hearing loss and correlate it to audiometric and vestibular functions.

MATERIALS AND METHODS

This was a prospective study done at a tertiary referral University Hospital from, March 2007 to February 2008. All patient were signed an informative consent prior to examination.

Patients were classified into two groups.

Group I: 34 children [28 males and 6 females] had idiopathic SNHL; their ages ranged from one to 16.5 years mean (5.8 ± SD 3.6).

Their SNHL was severe in [12] and severe to profound SNHL in [22].

Patients with other causes of SNHL as (infection, prematurity, neonatal hyperbiliruminemia, ototoxic drugs, trauma, genetic disorders as (CHARGE, Alport, etc.) were excluded from the study.

Group II: included 42 age -matched controls [20 males and 22 females] referred to the CT unit for assessment of otomastoiditis, otalgia, otitis externa, or cholesteatoma without SNHL proved by audiological assessment. Their age ranged from one to 18 years (mean 10.2 ± SD 5.2).

All patients were submitted to otorhinolaryngological and otoneurological examinations. Full audiological evaluation was done to all children via play audiometry using air conduction thresholds (0.25-8 KHz), bone conduction thresholds (0.5-4KHz), speech audiometry including speech reception thresholds and word discrimination scores. Immittancemetry including tympanometry and acoustic reflex thresholds was also done. Finally Auditory brainstem response (ABR) was done to infants who were unable to perform play audiometry. The level of hearing impairment was classified according to the criteria recommended by WHO [15]. Vestibular evaluation in the form of Sinusoidal harmonic acceleration (SHA) was performed (0.01-0.64Hz). The child was seated on his mothers lap. Prior to testing the head of the child was restrained. The child's head was flexed 30 degrees so that rotation occurs in the plane of the horizontal

semicircular canal. After proper calibration, rotation performed at acceleration frequencies ranging from 0.01 to 0.64 Hz. The chair was rotated with maximum velocity of 60 degrees / second at each test frequency. Infra red lenses were mounted on the chair to allowing monitoring of eye movement, head position and the patient condition during the test. Gain, phase and symmetry were calculated by the equipment and compared with age matched norms of the Rotary chair Micromedical Meta 4.

All our patients underwent HRCT of temporal bone using a spiral CT HiSpeed Advantage scanner (General Electric Corp., Milwaukee, WI, USA).

Transverse and coronal images were obtained. Eleven patients of ***group I*** and four patients of ***group II*** were not able to tolerate direct coronal scanning.

Scanning parameters were 120 kV, 180 mAs, 1-second rotation time, 1-mm section thickness, 1-mm collimation, 0.5 reconstruction increment, and 1-mm table feed per rotation, 512 x 512 matrixes, and 9-cm field of view. Transverse scans were acquired parallel to the hard palate and inferior to the orbit. Therefore, the cornea was not in the primary x-ray beam of the CT scanner. The coronal scans were acquired perpendicular to the transverse images. All images were displayed at bone window.

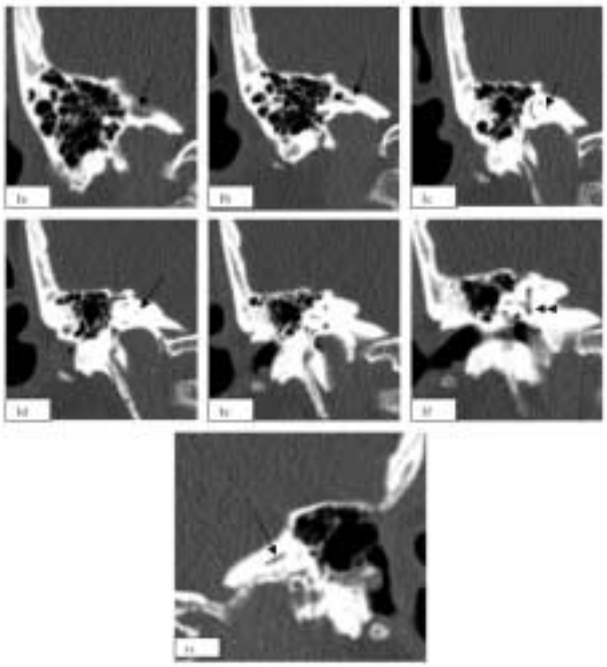

Figure-1: HRCT coronal images in 16 years old male patient without SNHL. They show normal course and size (1.1mm) of right bony vestibular aqueduct (black arrow) in six consecutive images from posterior (a) to its entrance in the vestibule (double headed black arrow) in f. VA width was (1.1) mm in b. This level was the one we standardized in our measurement on coronal images. (g) Left side of the same patient at the level we opted to measure VA (black arrow). White arrow in (c) is the posterior semicircular canal.

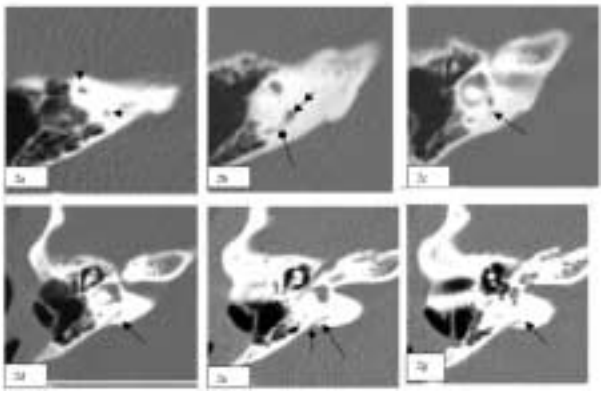

Figure-2: HRCT axial images in 10 years old male patient without SNHL showing normal course and size of the bony vestibular aqueduct (black arrow) at six consecutive levels measuring (1.4 mm) at opercular plane and (0.8 mm) at its midpoint. Its external aperture is marked by (short rhomboid shaped head black arrow). Superior semicircular canals (short black arrows in (a). Double-headed arrow in (b) is crus commune, and the rounded head arrow is posterior semicircular canal.

We used both coronal (Figure 1) and axial (Figure 2) images to measure vestibular aqueduct's width. The way we opted to measure the VA on coronal images is shown in (Figure 3-b); which was a midway between the midpoint and opercular levels. We follow Vijayasekaran et al,[7] method to measure the VA width on axial plane. On axial images vestibular aqueduct was measured at its midpoint (which is the halfway

between the origin and aperture), and at opercular level (which was done by drawing a line from the opercular edge laterally to the posterior wall of the petrous bone) (Figure 3a). Measurements were done at one level on axial images except in two patients. Two radiologists made measurements, with images enlarged with the use of workstation software.

Data Management and Statistical Analysis: Data collected was revised and introduced to a PC for statistical manipulation and analysis. Categorical data was described in term of frequency (number and percentages), while interval data was presented as mean and standard deviation. Association between two categorical variables was tested using chi-square. Comparison between two groups' means was evaluated using student t test. Pearson correlation coefficient described the correlation between three interval variables. P value was always set at 0.05 and all statistical analyses were performed using the 11th version of SPSS

.

RESULTS

Seventy-six patients were recruited in our study. 152 ears were examined. VA identification rate was higher on axial compared to coronal images. However, VA was identifiable only on coronal scans in 2 patients.

In control group (***Group II)***, vestibular aqueduct width ranged from (0.6 to 1.1 mm, mean 0.8 ± SD 0.2) at its midpoint on right side and ranged from (0.4 to 1 mm, mean 0.71 ± SD 0.2) at its midpoint on left side.

It ranged from (1.2 to 2 mm, mean 1.6 ± SD 0.3) at its operculum on right side and ranged from (0.8 to 2 mm, mean 1.6 ± SD 0.4) at its operculum on left side.

On coronal image VA, width was equal to the mean measurement at midpoint and operculum on axial images. It ranged from (1- 2 mm, mean 1.4 ± SD 0.5 on right side and 1.5 ± SD 0.4 on left side).

In ***Group I*** enlarged bony vestibular aqueduct was seen in nineteen (55.9 %) out of the thirty- four children with SNHL. The enlargement was bilateral in sixteen patients (Figure 4) and unilateral in three children.

Its width ranged from (1.4 - 5.3 mm, mean 2.62 ± SD 1.2) at its midpoint on right side and from (1.2 - 5.3 mm, mean 2.35 ± SD 1.1) on left side.

It ranged from (2.1 - 8.4 mm, mean 3.31 ± SD 1.8) at its operculum on right side and from (2.2 - 6 mm, mean 3.27 ± SD 1.3) on left side. On coronal images, it ranged from (1.6 -7 mm mean 2.8 ± SD 2.0 on right side and mean 3.24 ± SD 1.7 on left side) (Table 1).

There was significant correlation between measurements at midpoint and opercular level on both right and left sides while, there was not

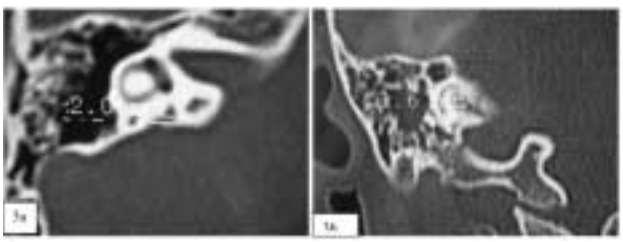

Figure-3: (a) Axial HRCT image in a patient with enlarged VA shows the way we used to measure the VA at both midpoints and opercular levels. (b): Coronal HRCT in another patient with enlarged VA show the level and the way we used to measure the VA.

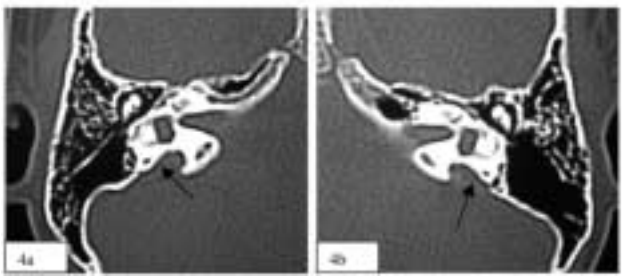

Figure-4: HRCT axial image in 11 years male patient with bilateral SNHL show enlargement of bony vestibular aqueduct on both sides of average size (4.2 mm) at opercular plane and (2.3 mm) at its midpoint.

significant correlation between measurements on coronal and axial planes (Table 2).

Incidence of enlarged vestibular aqueduct was higher with severe to profound hearing loss (Table 3).

Sinusoidal Harmonic Acceleration (SHA) was abnormal in (16) 47 % of total children with SNHL, eleven (68.8%) of them had enlarged vestibular aqueduct. Incidence of gain abnormality in rotational testing was common in patients with enlarged vestibular aqueduct. The higher number of patients and percentage in (Table 4) is due to suffering of two children from combined abnormal gain and phase.

There was no significant correlation between VA width and audiometric results (Table 5).

In addition, no significant correlation was found between VA width and vestibular function results (Table 6).

Table-1: VA Measurements on right and left sides in Cases and Controls

	Group I **N=19** **Mean (SD) in mm**	***Group II*** **N=42** **Mean (SD) in mm**	**P value**
Axial Mid Point			
* Right	2.62 (1.2)	0.80 (0.2)	<0.01* S
* Left	2.35 (1.1)	0.71 (0.2)	<0.01* S
Axial Operculum			
* Right	3.31 (1.8)	1.6 (0.3)>	<0.01* S
* Left	3.27 (1.3)	1.6 (0.4)	<0.01* S
Coronal			
* Right	2.8 (2.0)	1.4 (0.5)	NS
* Left	3.24 (1.7)	1.5 (0.4)	NS

VA: Vestibular aqueduct

* S: Significant

NS: Not significant

Table-2: Correlation of Measurements at different Points and Planes of Examination

	Right Axial Mid Point	**Right Axial Operculum**
Right Axial Operculum	0.779**	
Right Coronal	0.121	0.230
	Left Axial Mid Point	Left Axial Operculum
Left Axial Operculum	0.885**	
Left Coronal	0.331*	0.254

** p<0.01 (Significant),

* p<0.05 (Not significant)

Table-3: Prevalence of enlarged vestibular aqueduct in patients with SNHL (Group I)

	Enlarged Vestibular aqueduct	
Degree of SNHL	**No of patients**	**%**
Severe	4	11.7 %
Severe to profound	15	44.2%
Total	**19**	**55.9%**

SNHL: sensorineural hearing loss

Table-4: Results of sinusoidal harmonic acceleration (SHA) in patients with enlarged vestibular aqueduct.

	Enlarged Vestibular aqueduct	
Abnormal SHA	**No of patients**	**%**
Gain	9	26%
Phase	7	20%
Symmetry	2	6%
Total	**16**	**47%**

Table-5: Correlation of measurements at different points of examination with audiometric results at different PTA by dB

	PTA by dB			
	500	**1000**	**2000**	**4000**
Axial Mid Point				
* Right	-0.393	-0.450	-0.379	-0.339
* Left	-0.394	-0.453	-0.382	-0.341
Axial Operculum				
* Right	-0.389	-0.445	-0.374	-0.333
* Left	-0.390	-0.450	-0.379	-0.339
Coronal				
* Right	-0.498	-0.478	-0.428	-0.486
* Left	-0.495	-0.477	-0.428	-0.487

PTA: play therapy audiometry

dB: Decibel

DISCUSSION

The vestibular aqueduct (VA) is a bony canal extending from the medial wall of the vestibule, coursing posteriorly and inferiorly parallel to the common crus to the posterior fossa dura anterior to the sigmoid sinus, the VA houses the endolymphatic duct and part of the endolymphatic sac [13].

Alvarenga et al, [16] have studied only the VA distal segment, because VA is usually well defined distally contrarily to the proximal segment (isthmus) that is frequently not visualized due its lumen narrowing, besides being obscured by the contiguous crus commune.

We agree with Murray et al, [17] that the vestibular aqueduct could be traced along its course from posterior to anterior on coronal CT images. The anterior images correspond to the isthmus and clearly show its proximity to the common crus. The coronal posterior measurements correspond to the distal segment of the vestibular aqueduct and demonstrate its widened triangular shape as well as its aperture at the posterior fossa. In their series, they were able to measure the vestibular aqueduct on 100% of the anterior coronal views, 77% of the midisthmus axial CT images, and 53% of posterior coronal CT images. In our study, fifteen patients did not tolerate the direct coronal scans and this explain the lower identification rates on coronal scans (27.8%) compared to (77.8%) on

Table-6: Correlation of measurements at different points of examination with vestibular function (Rotatory Chair Gain)

	Rotatory Chair Gain				
	0.01	**0.02**	**0.04**	**0.08**	**0.16**
Axial Mid Point					
* Right	0.350	0.424	0.315	0.505	0.466
* Left	0.351	0.427	0.318	0.505	0.466
Axial Operculum					
* Right	0.346	0.415	0.311	0.502	0.463
* Left	0.348	0.427	0.315	0.501	0.463
Coronal					
* Right	0.411	0.432	0.308	0.534	0.467
* Left	0.410	0.435	0.307	0.532	0.466

PTA: play therapy audiometry

dB: Decibel

axial scans. We did not follow their way in VA measurement as the level we opted to measure the VA was the best to delineate it, it is seen uniform, long and clear at this level (Figure 1-b and g) which was midway between the midpoint and operculum at axial scans.

The non-visualization of VA does not mean necessarily that it is absent [18]; besides, other technical factors or anatomical characteristics [19-22], as well as the influence of the partial volume effect [23] may be involved.

Valvassori and Clemis [14] measured the VAs of 3700 patients by using hypocycloidal polytomography and reported that a midpoint VA width of greater than 1.5 mm is abnormal. This value is still generally accepted today. In agreement with their results, in our study all patients with enlarged VA their VA width were > 1.2 mm.

Simons et al, [24] studied 131 children with unilateral or asymmetrical SNHL; the prevalence of abnormal CT findings was seen in 35% of patients with unilateral SNHL, compared with 7% to 44% in the literature, [9, 12, 25, 26] and in 52% of subjects with asymmetric bilateral SNHL. CT abnormalities have been found in their series to be about 28% of children with bilateral SNHL (either symmetric or asymmetric) [11] For all subjects with SNHL in their study, the overall prevalence of abnormal CT findings was 41%, compared with an overall prevalence of 13% to 37% in the literature, [8,9,10, 26,27]. In our study, abnormal CT findings were seen in 55.9 % of our patients that were higher than that mentioned in the literature, moreover on individual basis (64.7%) of them had severe to profound SNHL which prove the correlation between the degree of SNHL and VA width. Yet no statistical significant correlation was found between Pure Tone thresholds and vestibular aqueduct diameter.

The association between idiopathic SNHL in patients with enlarged vestibular aqueduct was explained by the fact that if the vestibular aqueduct is enlarged, the endolymphatic duct and sac usually grow large too. This results in disturbance in the normal ionic homestasis of the inner ear (Berrttini et al.) [28], the most well known cause of enlarged VA and hearing loss is mutations to a gene known as SLC26A4 (also referred to as the PDS gene) on chromosome 7.

Colvin et al., [29] reported stationary SNHL in patients with enlarged VA which progress with minor head trauma. On the contrary, Wu et al., [30] reported that patients with complex of enlarged vestibular aqueduct, Mondini dysplasia, large vestibule, and semicircular canal dysplasia demonstrated a significantly higher incidence of fluctuating hearing loss (93%) and a better

hearing level compared with those with other malformations. In our study, children with enlarged vestibular aqueduct syndrome were instructed to avoid contact sports that might lead to head injury; and avoid situations that can lead to barotraumas.

In agreement with Grimmer and Hedlund [31] who reported that nearly 50% of children with enlarged VA experienced vertigo and vestibular symptoms with equal frequency when compared to adult patients. In our study, Sinusoidal Harmonic Acceleration (SHA) was abnormal in (16 children) 47 % of total children with SNHL. Eleven (68.8%) of them had enlarged vestibular aqueduct. This reflects the association between cochlear and vestibular dysfunction in children with enlarged vestibular aqueduct

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