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# The incidence of enlarged vestibular aqueduct among hearing-impaired children: hospital-based tertiary care referral center

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## Abstract

**Background:** The most common radiologically detectable congenital inner ear anomaly is an enlarged vestibular aqueduct (EVA), which is associated with varying degrees of hearing loss and vestibular disorders. The purpose of this study was to ascertain the enlarged vestibular aqueduct incidence in hearing-impaired children sent to a tertiary care referral center, as well as to describe the audiologic pattern of EVA in those individuals.

**Results:** In a retrospective study of 3765 hearing-impaired children aged 1 to 10 years, 450 had EVA (11.95%). The EVA was more prevalent in female populations (54.4%). Head trauma was present in 26.7% of cases; a positive family history of EVA was present in 17.8%. The most common associated syndrome was Pendred syndrome. Progressive hearing loss was observed in 25.6% of patients, fluctuant hearing loss in 19.1%, and sudden hearing loss in 2% of the patients.

**Conclusion:** The most prevalent inner ear anomaly is an enlarged vestibular aqueduct, contributing to 11.9% of the research study. The most common presentation of EVA is hearing loss, which may be stationary, progressive, fluctuant, or sudden onset. CT scan is considered the gold standard radiological test to diagnose EVA in the sagittal or parasagittal plane. Head trauma should be avoided in children with EVA, and close follow-up is essential.

**Keywords:** Enlarged vestibular aqueduct, Incidence, Fluctuating hearing loss, Progressive hearing loss, Asymmetrical hearing loss

## Background

The enlarged vestibular aqueduct (EVA) syndrome is one of the most often detected congenital inner ear defects on radiography [1]. EVA is distinguished by anomalies in the inner ear's structure as well as the auditory and vestibular systems' physiology, clinical presentation varies, and hearing loss may be variable, gradually progressive, or abrupt in onset. Vestibular disorders are classified according to their severity, ranging from slight imbalance to episodic vertigo [2].

The vestibular aqueduct (VA) is a bony canal that runs from the vestibule's medial wall to the petrous pyramid's cerebellar face. It is made up of a vein, an artery, and an endolymphatic duct. When measured in the middle portion between the external aperture and the common crus, the vestibular aqueduct has an average diameter of 0.62 mm [3].

Valvassori and Clemis demonstrated their first description of EVA as a vestibular aqueduct associated with an anterior-posterior diameter of more than 1.5 mm [3]. Zalzal et al. categorized EVA as a vestibular aqueduct anterior-posterior diameter more than 1.4 mm [4]. Boston et al. have recently evaluated 107 children with SNHL for the definition of EVA relying on CT and characterized EVA as being more than 1.9 mm in operculum and/or above 0.9 mm in midpoint [5].

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The vestibular aqueduct expansion is often seen as a consequence of a developmental arrest around the pregnancy's 5th week [6]. The majority of authors assumed that swelling of the bony aqueduct is induced by aberrant growth of the endolymphatic duct and sac throughout embryo development, and that hearing loss is congenital [7–10].

EVA is often found in conjunction with other inner ear abnormalities, the most common of which is an abnormally large vestibule, an enlarged semicircular canal, or a hypoplastic cochlea [11, 12]. Enlarged vestibular aqueduct can be isolated [13], or part of a syndromic form of SNHL, such as Pendred's syndrome (PS) [14, 15], branchiootorenal (BOR) syndrome [16], and distal renal tubular acidosis (dRTA) [17].

In terms of genetics, EVA is hypothesized to be genetically an autosomal recessive trait [18]. The nonsyndromic SNHL locus correlated with EVA has been subsequently localized to the same 19th chromosomal regions as the PS locus, 7q31 [19, 20]. Additionally, it has been found that the PDS gene, which encodes PS, is altered in individuals with EVA who have nonsyndromic SNHL [20–22].

Okamoto et al. proposed that hearing loss in EVA could be caused by increased cerebrospinal fluid pressure, which damages hair cells [23]. Other investigations indicate that hyperosmolar fluid may backflow into the cochlea, disrupting the auditory hair cells [24, 25]. Vestibular dysfunction in EVA has the possibility to have the same causes as hearing dysfunction, and some propose that hyperosmotic fluid backflow into the cochlear duct's basal end can result in vertigo [9, 26]. Another mechanism of harm could be vestibular hair cell degeneration as a result of chemical and osmotic imbalances [27].

According to the literature, EVA is linked to progressive or fluctuating sensorineural hearing loss, which can be triggered by minor head trauma [28, 29]. According to Valvassori, it is associated with conductive component of hearing loss, which he explained by the opposition to the stapes movement caused by increased endolymph pressure within the typical ear cavity of the middle ear [30].

According to Govaerts et al., mixed hearing loss was found in 90% of cases, and it was hypothesized that the conductive component was of pure cochlear origin which is characteristic of this disease [12]. However, Boston et al. reported that only 39% of EVA ears had a mixed hearing loss and mostly had progressive course [5]. According to Govaerts et al., progressive hearing loss occurs at a rate of 4 dB/year on average [12].

Sudden SNHL has also been revealed in some children with EVA following minor head trauma, so dangerous activities and contact heavy sports should be avoided in those children [31, 32].

### The aim of work

To evaluate the incidence of enlarged vestibular aqueduct (EVA) among hearing-impaired children in tertiary care referral audio-vestibular medicine unit, as well as to describe the different audiologic pictures of EVA among those children.

### Methods

The study included a retrospective analysis of 3765 hearing-impaired children aged 1 to 10 years old attending the audio-vestibular medicine unit, between 1st December 2017 and 30th January 2021; 450 children had EVA (11.95%) based on the radiological and suspected audiological diagnosis.

All children in the study were subjected to the following:

- Personal history, onset, course, and duration of hearing loss; prenatal, neonatal, and postnatal events; developmental history, timing, and course of hearing loss; consanguinity and family history of hearing loss; history of head trauma; and vestibular complaints are all taken from the parents or children.
- General evaluation includes thyroid examination, thyroid profile, and ultrasonography if needed in suspected Pendred syndrome.
- Otological examination: Aimed to ensure normal otoscopic examination, no occlusion, infections, congenital abnormalities, or other abnormalities in the external auditory canal.
- Audiological evaluation in the form of the following:
  - A. Sound field audiometry: Intended for children under 3 years of age with warble tones of 0.5, 1, 2, and 4 kHz in a sound field environment, presented and calibrated at 45° azimuth. Reflexive and behavioral responses observed during testing were either alert (increase and decrease in movement, searching or locating the sound source, listening) or reflex (startle, arousal from sleep, blinking).
  - B. Play audiometry, or conventional audiometry, was used to assess the hearing threshold for air conduction between 250 and 8000 Hz and bone conduction between 500 and 4000 Hz, based on the children's age and reliability. The test was conducted twice in play audiometry to achieve a consistent and accurate test consequence [33].
  - C. Speech audiometry, including speech reception thresholds for children using Arabic spondee words [34] and speech recognition scores using Arabic kindergarten phonetically balanced words (list of 25 words) [35]. The audiometry was done

using Interacoustics AC 40 two-channel pure tone audiometer with a locally manufactured sound-treated booth.

- D. Immittance: Using Interacoustics AT 235, single-frequency tympanometry to assess the condition of the middle ear
- E. Evoked audiometry for infants and younger kids, tested while sleeping, either naturally or under sedation with chloral hydrate (0.5 cc/kg). The Interacoustics Eclipse 25 platform-evoked potential system was used for the tests.
- I. Auditory brainstem response (ABR): Was conducted using rarefaction click stimuli delivered via insert phone at a repetition rate of 21.1 p/s and at intensity level of 100 dB nHL. It was filtered between 30 Hz for high freq. and 1500 Hz for low freq. and gain factor of  $\times 100,000$  times, collected in a 15-ms time window, with a mean of more than 4000 sweeps per run. To get an ABR threshold, we start at 100 dB HL and gradual decrease in 20 dB steps; the stimulus level was decreased until no detected waves were obtained. It was elevated significantly by 10 dB up to the observation of the response. When no ABR response was obtained at the highest possible level, the run is repeated once [36].
- II. Auditory steady-state response (ASSR): The test stimuli were presented as modulated pure tones in both ears at rates of 74, 81, 88, and 95 Hz via insert earphones at frequencies of 500, 1000, 2000, and 4000 Hz. Modulated tones were given at high rates in order to establish a strong signal-to-noise ratio to get an accurate response. Each signal was frequency and amplitude modulated and delivered independently to each ear. Employed a carrier tone with an amplitude-modulated depth of 100% and a modulated width of 10% of the carrier tone to maximize response amplitude. ASSRs were measured at 100 dB HL or carrier frequency range of 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz. To get an ASSRs, the stimulus level was reduced in 10 dB steps until no response was detectable and then increased incrementally by 5 dB until the action was observed. The run was repeated if no ASSR was obtained at the maximum presentation level [37, 38].

The rationale of using both ABR and ASSR aimed to provide a frequency-specific and reliable threshold to get an accurate degrees of hearing loss at different frequencies. ASSR can be done and give information about the

presence of useful residual hearing mainly at low-frequency region, while ABR can give the information at high-frequency regions (2–4 KHz).

- CT scan of the petrous bone: Multiplanar reformations, specifically in the sagittal or parasagittal planes, may be even more beneficial in determining the whole diameter of the VA.

The definition of EVA on CT scan according to Cincinnati criteria is as follows: VA is abnormal if operculum width  $\geq 2$  mm and VA midpoint width  $\geq 1$  mm [5].

#### Data analysis and power of the study

The Statistical Package for Social Science (SPSS v. 19, IBM, Chicago, IL, USA) was utilized to evaluate the gathered data. Data have been presented using the mean  $\pm$  standard deviation (SD) and the frequency and percent for continuous and categorical variables, respectively.  $P$ -value  $\leq 0.05$  was considered significant.

#### Results

In retrospective analysis of the audiological profiles of 3765 hearing-impaired children aged from 1 to 10 years old attending the audio-vestibular unit from 1st December 2017 to 30th January 2021, 450 children had EVA (11.95%) based on the radiological and suspected audiological diagnosis. The EVA was more common in the female populations (54.4%), but difference between females and males was not significant. A history of head trauma was present in 26.7% of the EVA patients and positive family history of EVA in 17.8%. Numerous syndromes were associated with EVAS in the study, including Pendred, brachiootorenal, renal tubular acidosis, Waardenburg, and CHARGE syndromes, as shown in Table 1.

Vestibular complaints were observed in 100 children and mainly in the form of dizziness and unsteadiness, and few elder children experienced true vestibular spinning attacks. The vestibular complaints and vestibular evaluation for children with EVAS behind the scope of this paper will be discussed in details in another study.

The goal of the audiological evaluation was to provide a frequency-specific and reliable threshold to get an accurate degree of hearing loss. Conventional audiometry was done for 210 children, play audiometry was done for 140 children, two sessions were done to ensure reliable and specific thresholds, and, lastly, evoked audiometry (ABR, ASSR) was done for 100 infants and young kids. The hearing evaluation was done to get an accurate degree of hearing loss, and there were variable degrees of hearing loss, as shown in Table 2.

**Table 1** Demographic data of the studied patients

Demographic data	No. (450)	(%)
<b>Age (years)</b> Mean $\pm$ SD (range)	6.35 $\pm$ 2.00 (1–10)	
<b>Gender</b>		
Male	205	45.6
Female	245	54.4
<b>Family history of EVA</b>		
Yes	80	17.8
No	370	82.2
<b>History of hearing deterioration</b>		
Yes	200	44.4
No	250	53.6
<b>History of head trauma</b>		
Yes	120	26.7
No	330	71.3
<b>History of vestibular complaints</b>		
Yes	100	22.3
No	350	77.7
<b>Pattern of EVA</b>		
Isolated EVA	346	76.9
Part of Mondini anomalies	104	23.1
<b>Associated syndromes with EVA</b>	<b>62</b>	<b>13.8</b>
Pendred's syndrome (PS)	35	7.8
Branchiootorenal syndrome (BOR)	12	2.7
Distal renal tubular acidosis (RTA)	5	1.1
Waardenburg syndrome	6	1.3
CHARGE	4	0.9

Hearing loss was categorized into mild, moderate, moderately severe, severe, and profound hearing loss, according to Clark [39]:

- Normal hearing (0–15 dB HL)
- Slight or minimal hearing loss (16–25 dB HL)
- HL in the mild range (26–40 dB HL)
- HL in the moderate range (41–55 dB HL)
- HL in the moderately severe range (56–70 dB HL)
- HL in the severe range (71–90 dB HL)
- HL in the profound range (more than 91 dB HL)

Asymmetrical hearing loss is described as the difference in hearing loss between ears that are larger than 15 dB at 0.5, 1, and 2 kHz or greater than 20 dB at 3, 4, and 6 kHz on an audiogram [40]. Asymmetrical hearing loss is shown in Figs. 1 and 2.

The course of HL was classified into *stationary* hearing loss, which means the degree of hearing is still the same over time with no or little change. *Progressive* hearing loss means hearing loss becomes worse over time. *Fluctuant* HL means hearing loss changes over

**Table 2** Characteristics of hearing loss

Character	No. (450)	(%)
<b>Diagnosis of hearing loss</b>		
Conventional audiometry	210	46.7
Play audiometry	140	31.3
Evoked audiometry (ABR, ASSR)	100	22.2
<b>Type of hearing loss</b>		
Mixed HL (ABG at 500, 1000 Hz)	70	15.6
SNHL	380	84.4
<b>Degree of SNHL</b>		
Mild SNHL	10	2.2
Moderate SNHL	90	20
Moderately severe SNHL	110	24.4
Severe SNHL	90	20
Profound SNHL	80	17.8
<b>Degree of MHL</b>		
Moderate MHL	12	2.7
Moderately severe MHL	22	4.9
Severe MHL	28	6.2
Profound MHL	8	1.8
<b>Symmetry of SNHL</b>		
Symmetrical	270	60
Asymmetrical	180	40
<b>Course of hearing loss</b>		
Stationary	240	53.3
Progressive	115	25.6
Fluctuant	86	19.1
Sudden SNHL	9	2

time, sometimes gets better, and sometimes gets worse [39].

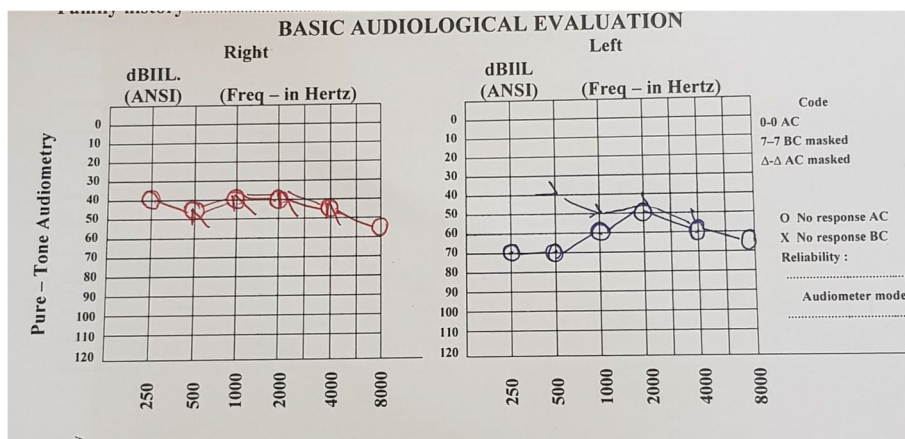
Sudden SNHL (SSNHL) is characterized as a sensori-neural hearing loss of 30 dB or more over 3 consecutive audiometric frequencies starting less than 3 days [41].

Table 3 showed that the bilateral lesions in EVAS cases were 91.1%, and the diameter of EVA was ranged from 2 to 7 mm.

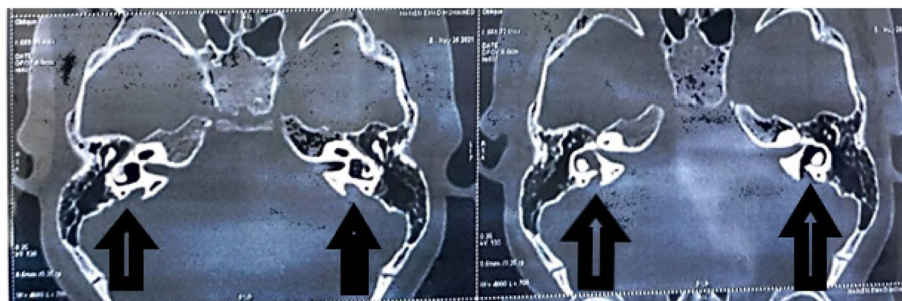
Follow-up of the hearing-impaired children with EVA revealed that 180 children were enrolled in a cochlear implant program funded by the Health Insurance Authority under 5 years old; the rest of the children were fitted with hearing aids as shown in Table 4.

## Discussion

Every year, 1 to 3 children of 1000 are diagnosed with hearing loss through newborn hearing assessment [42]. The incidence of permanent hearing loss in the school-age population has been calculated to be approximately 10–15 per 1000 people [43]. This increase was associated with variables such as differences in the hearing loss definition, progressive and late onset hearing losses that can



**Fig. 1** Example of hearing threshold of a child aged 7 years old in both right and left ears with an air-bone gap mainly at 0.5, 1 KHz in the left ear despite the normal middle ear compliance and the presence of acoustic reflexes. It showed air-bone gap mainly at 0.5 and 1 KHz in the left ear despite the normal middle ear compliance and the presence of acoustic reflexes



**Fig. 2** CT scan of petrous bone showed enlarged vestibular aqueduct in a 9-year-old female with Pendred syndrome; the black arrows showed the EVA

**Table 3** CT Petrous pattern of EVA

	No. (450)	(%)
Laterality of the lesion		
Bilateral	410	91.1
Unilateral	40	8.9
Diameter of EVA (mm)	4.8 ± 2.1 (2-7)	
Mean ± SD (range)		

**Table 4** Prognosis of EVA patients

Prognosis	No. (450)	%
Hearing aid	270	60
Cochlear implant	180	40

be inherited or induced by the environment. Congenital enlarged vestibular aqueduct syndrome (EVA) is a major cause of progressive hearing loss [3].

The current study showed that the EVA had an incidence of about 11.95% among hearing-impaired children during the time of the study in the tertiary care referral unit. EVA is the most common abnormal inner ear anomalies. Its prevalence rate in pediatric patients ranges between 5 and 15% [32, 44]. This study showed a higher prevalence of EVA among female population (54.4%), as shown in Table 1. The findings agreed with the findings of Valvassori and Clemis, who reported that female patients were more prone to EVA than male patients at a rate of 2:3 [3]. In addition, Noguchi et al. studied 380 patients with EVA, 221 (58.2%) of whom were female, suggesting female preponderance [45]. Madden et al. reported that 77 EVA patients had a male-to-female ratio which was 1:1.5 [32].

Also, 17.8% of EVA children had a family history of the disease. Only a few studies have reported familial cases of EVA. Griffith et al. assumed that enlarged vestibular condition was reported in two siblings with healthy parents on the assumption that the pattern was a recessive chromosome heritage or that the mom was the carrier of the germs [18]. In agreement with Griffith et al., Goh et al. reported EVA in two sisters with healthy parents and proposed an autosomal recessive inheritance pattern; when EVA appeared in one of the children, it was assumed that screening of the offspring would be favorable [32, 46].

History of hearing deterioration and head trauma was reported in children with EVA. Hearing deterioration was present in 44.4%, and trauma was reported in 26.7%. Smith and Van Camp observed that EVA almost invariably causes a progressive or variable hearing loss. Such progression and fluctuation are often reported following head trauma and injuries [47]. The gradual nature of EVA hearing loss is a major issue for patients and family members. Recommendations should emphasize the need to maintain residual hearing by eliminating activities that might contribute to head damage, such as the sport of high impact or barotrauma, as dive in scuba, and wearing appropriate headgear while taking possible dangers in biking, skating, or skiing [48]. Since hearing loss commonly escalates from a severe to a profound level, it is necessary that hearing is regularly checked [48].

Vestibular complaints account for 22.3% of EVA children in the current study, manifested as dizziness, unsteadiness, and a few vertiginous spells. According to Grimmer and Hedlund, vestibular symptoms were reported in roughly half of the individuals associated with EVA and associated with vertigo and other vestibular symptoms occurring at nearly equal rates in pediatric patients [49]. In agreement with the Grimmer study, Berrettini et al. investigated vestibular symptoms in 15 EVA patients and found that seven (47%) complained of vestibular disturbance [50]. The overall prevalence of vestibular symptoms in EVA individuals ranges from 12 to 71% [11, 51, 52].

However, these findings contradicted the findings of Noguchi et al. who reported that vertigo/dizziness/imbalance accounts for approximately 8.8% of EVA patients [44]. Furthermore, Madden et al. reported that vestibular symptoms were present in only three (4%) of the studied patients with EVA [45].

According to the current study, isolated EVA occurred in 76.9%, and 23.1% was part of Mondini anomalies. EVA had associated with syndromes in 13.8% of all cases. EVA is the most prevalent internal ear abnormality correlated with SNHL as per evidence (Online Mendelian Inheritance in Man [OMIM]). It can happen on its own

or in conjunction with other syndromes such as Pendred syndrome (PS), branchiootorenal syndrome, CHARGE syndrome, and Waardenburg syndrome [53, 54]. Around one-third of participants who had EVAs were subsequently diagnosed with PS in a recent NIDCD research [55]. PS is recessively inherited and induced by SLC26A4 gene mutations located on chromosome 7 [47]. With or without Mondini malformation, EVA is reported to occur in 80% of PS patients and is almost always bilateral; half of them will have congenital, severe to profound SNHL [47, 55].

According to Hamid, EVA has also been linked to branchiootorenal (BOR) syndrome, which affects the anatomy of the ears, kidneys, and neck [56]. According to Stinckens et al., three of the twelve (25%) affected BOR patients had a widened vestibular aqueduct and progressive sensorineural hearing loss [16]. Berrettini et al. describe a relationship between dRTA, SNHL, and EVA in two patients [17]. Shingo et al. reported that patients with RTA have progressive SNHL due to EVA [57]. Branchiootorenal syndrome, CHARGE, Waardenburg syndrome, Pendred syndrome, and distal tubular acidosis have been documented by Pryor et al. In such cases, progressive or fluctuating SNHL was observed, requiring close follow-up and monitoring, as well as proper adjustment of their hearing aids [54].

During a hearing evaluation, 70 children (15.6%) were found to have mixed hearing loss with an air-bone gap at 500 and 1000 Hz despite normal middle ear function, while 84.4% of the children had SNHL. Both mixed and SNHL children had varying degrees of hearing loss that range from mild to profound degrees in SNHL and from moderate to profound degrees in MHL. Hearing asymmetry was found in 40% of the cases. Over the course of the study, 53.3% of cases had a stationary hearing, 25.6% had a progressive hearing, 19.1% had a fluctuant hearing, and 2% had sudden hearing loss. EVA was suspected due to the presence of ABG despite normal ME function, asymmetry of hearing loss, and hearing fluctuation and was later confirmed by the radiological investigation. This false or pseudo-conductive hearing loss is due to enhance bone conduction through the third window phenomena [58, 59].

Spencer suggests that the cause of fluctuation in EVAS may relate to pressure fluctuations in the inner ear because of a larger vestibular aqueduct that may cause hearing loss through the wear and tear mechanism [60].

Alemi and Chan concluded that progressive sensorineural hearing loss is common in EVAS, and the mechanism of progression is unknown [61]. It was speculated that HL with EVAS after relatively minor head trauma could be caused by a rupture of the intracochlear membrane, resulting in endolymph and perilymph mixing. A

defect or tear in an area of congenital weakness in the basilar membrane or Reissner's membrane is thought to result from abnormal transmission of CSF pressure to the inner ear via large VA [62].

As Madden explained, sudden hearing loss in EVA is usually related to trauma [32]. Noguchi also reported that sudden HL occurred in 5.3% of patients because of head trauma and 5.0 because of upper respiratory infection [45].

Table 3 showed that EVA had a bilateral incidence of 91.1%. Noguchi also reported that 91.1% of the patients in his study (346/380) had bilateral EVA [45]. Also, Smith and Van Camp reported that EVA is generally bilateral and less frequently occurred as a unilateral presentation [47].

The diameter of the EVA in the study ranged from 4.8 ± 2.1 mm. Valvassori and Clemis define EVA if a diameter is larger than 1.5 mm [3]. Zalzal et al. described EVA as a vestibular aqueduct with a diameter higher than 1.4 mm [4]. Boston et al. characterized EVA in the operculum as being higher than 1.9 mm and/or in the medium of 0.9 mm [5].

Table 4 showed that follow-up of the hearing-impaired children with EVA revealed that 180 children were enrolled in a cochlear implant program funded by the Health Insurance Authority under 5 years old; the rest of the children were fitted with hearing aids. The CI children were of severe and profound degrees and of progressive course; they fulfilled the criterion for the CI proposed by the Health Insurance Authority, and they are doing well on the follow-up visits. While the rest of the children were in close monitoring, the baseline audiograms and the aided response were measured with adjustment of hearing aid if needed in every visit.

## Conclusion

The enlarged vestibular aqueduct in the study is 11.9% of inner ear anomalies. The most common presentation of EVA is hearing loss, which may be stationary, progressive, fluctuant, or of sudden onset. CT scan is considered the gold standard radiological test to diagnose EVA in the sagittal or parasagittal plane. Head trauma should be avoided in children with EVA, and close follow-up is essential.

## Abbreviations

EVA: Enlarged vestibular aqueduct; VA: Vestibular aqueduct; SNHL: Sensorineural hearing loss; PS: Pendred's syndrome; BOR: Branchiootorenal syndrome; dRTA: Distal renal tubular acidosis; TM: Tympanic membrane; ABR: Auditory brainstem response; ASSR: Auditory steady-state response; SPSS: Statistical Package for Social Science; USA: United State of America; SD: Standard deviation; OMIM: Online Mendelian Inheritance in Man.

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## Author's contributions

The author declares that he is the single author of this manuscript. The author read and approved the final manuscript.

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None

## Availability of data and materials

Data of the current study is available on reasonable request from the author.

## Declarations

### Ethics approval and consent to participate

The present study was approved by the Al-Azhar Medical Research Ethics Committee. The research ethics committee at Faculty of Medicine Al-Azhar University (Assiut) is independent organized committee operating according to international guidelines including the Declaration of Helsinki, Islamic organization for medical science, World health organization and international council on harmonization and good clinical practice. The committee's reference number: the number of meeting code is 3-2021 and the number of paper code is 8. The updated ethical code is MSR/AZ.AST./ENT030/45/212/10/2022 and the approval date is 11-10-2022. Consent for participate: informed verbal consent from the parents of the children under the age of 16 years old. As the study was a retrospective analysis of the patient's data in tertiary care unit, only verbal consent from the patients upon primary investigation.

### Consent for publication

Written informed consent for publication from the parent of the child with CT petrous image in the manuscript.

### Competing interests

The author declares no competing interests.

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