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Review Article

Anatomy of the vestibular aqueduct and its clinical importance: a review

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ABSTRACT

The vestibular aqueduct (VA) is a bony channel that houses the endolymphatic duct, linking the inner ear to the endolymphatic sac located in the posterior cranial fossa. The VA plays a crucial role in the metabolism and pressure buffer of the inner ear, and its enlargement can result in hearing impairment and balance disorders. The human VA often shows anatomical variations and even imaging can be difficult to appreciate, so clinicians need more data on the normal anatomy of VA for better understanding of radiological evaluation of enlarged VA. Enlarged vestibular adequate syndrome (EVAS) is reported to be one of the commonest anomalies of the inner ear associated with sensorineural hearing loss, although the exact mechanism of such loss is unclear. The EVAS can cause progressive sensorineural hearing loss (SNHL), in addition to sudden SNHL, conductive hearing loss, and mixed hearing loss. Mutations in a gene called SLC26A4 (formerly known as the PDS gene) are the primary cause of EVA and hearing loss. EVAS can be isolated or associated with cochlear malformation such as incomplete partition. There are no established criteria for diagnosis of EVAS. With advancement in imaging techniques, this VA and its anomalies are gaining greater interest among clinicians in recent years. High resolution computed tomography (HRCT) of temporal bone or MRI are helpful for the diagnosis of EVAS, with comparison to the adjacent posterior semicircular canal.

Keywords: Vestibular aqueduct, Anatomy, Enlarged vestibular aqueduct syndrome, Sensorineural hearing loss, Computed tomography

INTRODUCTION

The vestibular aqueduct (VA) is a tiny bony channel within the labyrinthine capsule that links the inner wall of the vestibule to the posterior part of the petrous part of the temporal bone.[1] It contains a portion of the endolymphatic sac and the endolymphatic duct.[2] From the vestibule's medial wall, it runs inferiorly and posteriorly alongside the common crus to the posterior fossa dura, which is anterior to the sigmoid sinus.[3] Both auditory and vestibular dysfunction have been associated with enlarged VA.[1] An enlarged vestibular aqueduct (EVA) is the most frequently observed congenital abnormality in children experiencing hearing loss. CT scans are commonly employed to assess the VA's width.[4] Large VA syndrome is one of the common findings in CT scan in children with sensorineural hearing loss.[5] When children with hearing loss are evaluated radiographically, the EVA is still the most frequently observed inner ear defect.[6] Individuals with EVA are vulnerable for perilymph gusher during cochleostomy in cochlear implant surgery or during stapedotomy in otosclerosis cases.[6] The objective of this review article is to discuss on the details of the clinical aspects of VA.

METHODS

A search was conducted for research articles on the anatomy of vestibular aqueduct and its clinical importance using various methods. This began with

searching online databases such as Scopus, PubMed, Medline, and Google Scholar. A search strategy was created based on the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analysis) guidelines. Articles eligible for review were selected by screening the title. Then, every abstract was carefully studied to confirm the eligibility criteria. The search approach found published article abstracts, and citations were used to manually find more research publications. The suitability of observational studies, comparative studies, case series, case reports, and randomized controlled trials for inclusion in this review was evaluated. A total of 72 articles (25 case reports, 19 case series, and 28 original articles) were found across various databases, with 56 being included in this review (Figure 1). This article discusses the history, anatomy, enlarged VA, clinical presentations, grading of large VA, investigations, VA with Meniere's disease, enlarged VA and cochlear implantation, and treatment of enlarged VA along with clinical perspectives of VA.

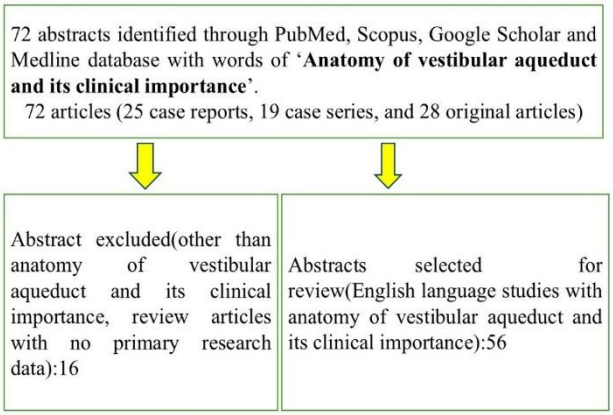

Figure 1: Methods of literature search.

HISTORY

The VA has been documented since 17th century.[7] The first description of VA was done by Neapolitan anatomist Domenico Cotugno, which was named as Aqueduct of Cotunnius.[7] Years later, the first enlarged inner ear structure was documented by Mondini in samples of dissected temporal bones.[8] Bast and Anson reported the VA morphology and its topographic relation to the other external ear structures.[9] The first radiologic study on VA and its abnormalities was done by Clemis and Valvassori.[10] After one-decade, same authors reported the first clinical description of a congenital disease characterized by a large VA, seen in CT scan.[10] The EVA was first documented by Mondini in 1791, during performing a temporal bone dissection.[11] Mondini documented a specific malformation of cochlea with incomplete cochlear partition (hypoplastic modiolus), short cochlear duct with flat cochlea, auditory and vestibular organs that were immature, a dilated vestibule, semicircular canals that were wide, small, or missing, an endolymphatic sac that was bulbous, and a large VA. Instead of 2.5 turns, the cochlea had 1.5 turns with an absent interscalar septum between the middle and apical turn.[11] Embryologically, this refers to arrest in development during 7th week of gestation.[11]

ANATOMY

The VA is a J shaped bony canal measuring approximately 5 to 7 mm in length.[12] The VA is localized in the posterior segment of the medial wall of the petrous part of the temporal bone. The VA is parallel to the petrous apex in contrast to the cochlear aqueduct, which lies perpendicular to the petrous apex. [12] The VA extends from the medial side of the vestibule to the petrous part of the temporal bone and contains the endolymphatic duct, which is lined with cuboidal or low-columnar epithelial cells.[12] The vestibular aqueduct (VA) features an opening that extends to the posterior surface of the structure and is divided into two main segments. The first is the proximal segment, or isthmus, which is narrowed at the internal opening and measures approximately 1.5 mm in length and 0.3 mm in diameter.[12] This part is located at the anteromedial wall of the vestibule. The second is the distal segment, which has a triangular shape with its apex connecting to the isthmus and widening toward its base, ending at an external opening that ranges from 0.5 mm to 5 mm in diameter.[12] Morphologically, this segment resembles an inverted "J." The normal diameter of the VA varies between 0.4 mm and 1.0 mm, allowing the passage of a small vein. 12This vein contains a tubular extension of the membranous labyrinth and the endolymphatic duct, which terminates in a sac-like structure situated within the cranial cavity between the layers of the dura mater. [13] Along its course, the endolymphatic duct progressively narrows until it becomes the endolymphatic sac, inner ear vestibule, and cochlea. [14] The initial segment of VA is narrow and often called as isthmus and contains parts of the endolymphatic duct (the most proximal portion is found in the vestibule). The distal segment of VA is anatomically very variable and contains the intra-osseous part of the endolymphatic sax. The extraosseous part is seen in a dural pouch on the posterior slope of the pyramid near the sigmoid sinus. The human endolymphatic duct contains active epithelial elements surrounded by a rich vascular plexus connected to the vein of the VA. One report showed that the peri-endolymphatic duct channels extending from the proximal sac to the supporting tissue of the saccule and speculated regarding their involvement in fluid and potassium hydrodynamics.[15] Since the VA is typically well defined distally, in contrast to the proximal segment (isthmus), which is frequently invisible due to its narrowing lumen and is also hidden by the neighbouring crus commune, Alvarenga et al. exclusively examined the distal segment of the VA.[16]

EVA

One of the most common inner ear malformations is enlarged VA (EVA).[17] The basic cause for deformity of

the VA is the abnormal growth of its contents such as the endolymphatic sac and canal.[18] The EVA may be associated with enlarged horizontal semicircular canal or Mondini's dysplasia, syndromic deafness as in CHARGE syndrome, Alagille syndrome, von Hipple-Lindau disease or Pendred's syndrome.[19,20] The EVA is associated with Pendred's syndrome and with mutations of SLC26A4(PDS) gene.[21] This gene encodes the pendrin which is an important protein involved in the cellular transport of chloride, iodine, and bicarbonate anions.[22] The mutations in SLC26A4 can result in Pendred's syndrome as well as non-syndromic recessive deafness (DFNB4).[22] Enlarged VA is also associated with distal renal tubular acidosis, Wardenburg's syndrome, X-linked congenital mixed hearing loss, branchio-oto-renal syndrome, oto-facio-cervical syndrome, and Noonan's syndrome.[23,24] The enlarged vestibular aqueduct syndrome (EVAS) is an important cause of third mobile window syndrome due to a larger than normal vestibular acting as a path for acoustic energy to be shunted from the cochlea.[25] The criteria used for diagnosis of enlarged vestibular aqueduct is typically the midpoint of the VA being 1.5mm or greater.[25] There are different criteria stated for EVA. The common criteria which gained acceptance for EVA include Valvassori and Clemis criteria, Cincinnati criteria, and Wilson criteria. Valvassori and Clemis criteria (1978) is the most accepted criteria for diagnosis of EVA. [25] According to this criteria, VA is said to be enlarged if the width measured at the midpoint of its course from the vestibule to the opening(operculum) in the posterior cranial fossa is more than 1.5mm. 1,25 Cincinnati criteria stated that VA is to be considered enlarged if the width measured at the midpoint is greater than 0.9mm or at the operculum is greater than 1.9mm.[26] Wilson criteria for EVA defined as any segment of the VA twice that of the adjacent posterior semicircular canal.[27] Valvassori and Clemis measured the VA of 3700 patients with help of hypocycloidal polytomography and documented that a midpoint VA width of greater than 1.5mm is abnormal.[2] The association of SNHL and enlarged VA is explained by the fact that if the VA is enlarged, the endolymphatic duct and sac often grow large too. This leads to disturbance in normal ionic homeostasis of the inner ear.[28] The most well-known cause for enlarged VA and hearing impairment is mutations to a gene called as SLC26A4 (also called PDS gene) on chromosome 7. 28 A study showed that stationary SNHL among patients with enlarged VA that progress with minor head trauma.[29] On the contrary, another study reported that patients with complex of enlarged VA, Mondini dysplasia, large vestibule, and semicircular canal dysplasia revealed a significantly higher incidence of fluctuating hearing loss (93%) and a better hearing level in comparison to those with other malformations.[30] Children with enlarged VA are instructed to avoid contact sports that can lead to head injury; and avoid situations that can result in barotraumas.[1] There is no universally accepted size-based criteria for enlargement of VA, but a rough rule of thumb if the VA is wider than width of the posterior semicircular canal.[31]

Grading of EVA

The VA enlargement is classified into five grades (Table 1).[28] This classification is based on the measurement from the intermediate point between the external opening and the common crus area. Grade I:lumen of the VA is usually seen in the temporal bone cortex; Grade II: lumen of the VA is seen close to the common crus; Grade III: lumen of the VA is larger than common crura, but it is not seen in the topography of the vestibule output; Grade IV: the internal part of the VA is seen and its diameter in the output topography is smaller or equal to the common crura; Grade :the internal part of the VA is seen and the diameter of the output topography is larger than the diameter of the common crura.

Table 1: Antonelli's classification of enlarged vestibular aqueduct.

Grading	Features
Grade I	Contents of the VA is only visible in the temporal bone
Grade II	Contents of VA is visible close to common crus
Grade III	Contents extend beyond the common crus, but not visible at the exit to the vestibule
Grade IV	Internal part of VA visible and its diameter at the exit to the vestibule is smaller than or equal to the common crus
Grade V	Internal part of VA is visible and its diameter at the exit to the vestibule is larger than diameter of the common crus

CLINICAL REPORT

EVA is a congenital anomaly of temporal bone affecting both auditory and vestibular systems, resulting in SNHL and disequilibrium at a very early age in the affected individuals.[32] Though females seen to possess greater enlarged VA, this difference does not extend to hearing loss.[33] LVA syndrome seems to be a unique clinical entity where patient present with progressive SNHL.[1] Some patient of EVAS complaints of hearing loss and even not present any other audiological signs or symptoms. Pure tone audiogram is helpful to confirm the hearing loss in patients with EVAS. Speech reception threshold (SRT) often shows poor thresholds. The type of hearing loss in EVAS is a point of controversy. All three types of hearing loss such as sensorineural, mixed, and conductive type of hearing loss have been documented in this clinical condition, though pure conductive hearing

loss is the rarest form reported. While sensorineural hearing loss is the most frequently observed in cases of EVAS, some researchers suggest that nearly all patients with an enlarged VA exhibit an air-bone gap, especially at lower frequencies.[34] Variations in findings across studies may be due to incomplete or inadequate audiometric evaluations. [34] ABR testing is commonly used to evaluate the hearing among many pediatric patients, but it presents challenges in accurately assessing low-frequency hearing through bone conduction. This can often lead to an overdiagnosis of SNHL rather than identifying a mixed hearing loss. Additionally, the presence of severe hearing loss in both ears in many patients creates a masking dilemma, which further complicates the interpretation of results. Patients of EVAS also frequently present vestibular complaints such as vertigo, motor delay, and imbalance.[35]

Investigations of EVA

The anatomical anomalies of temporal bone have increasingly been considered as an important cause for deafness.[36] The high-resolution CT scan showed the abnormalities in up to 37% of pediatric patients with previously unexplained SNHL.[37] The EVA is associated with abnormalities of the cochleovestibule.[38] High-resolution temporal bone computed tomography (HRCT) is the preferred initial imaging method for evaluating patients with sensorineural hearing loss, whether the loss is symmetric, asymmetric, or unilateral.[39] EVA (Figure 2) is the most common anomalies of inner ear, which can be diagnosed with HRCT.[40] The VA is an osseous canal approximately 10mm of length, localized in the posterior segment of the medial wall of the petrous part of the temporal bone. Investigations such as MRI and CT scans of inner ear are helpful to diagnose the enlarged VA. High resolution temporal bone CT scan has been the first line imaging study of choice obtained by pediatric otolaryngologists in the workup of children with SNHL, including asymmetric, symmetric, and unilateral SNHL.[41] Enlarged VA is the most common abnormality of the inner ear, which can be diagnosed by CT scan.[42] On coronal CT images, the VA can be followed from its posterior to anterior course.[43] The isthmus is represented by the anterior parts, emphasizing its close connection to the common crus. On the other hand, the distal part of the VA is reflected in the posterior coronal pictures, which show its enlarged triangular shape and its entry into the posterior cranial fossa. When the VA's diameter on a CT or MRI scan is more than 1.5 mm at the midpoint between its start in the vestibule and its terminus in the posterior fossa (operculum), it is deemed enlarged.[44] Patients of LVA is often show sensorineural hearing loss in audiogram.[45] However, sometimes patients present conductive hearing loss. The exact mechanism for conductive hearing loss in LVAS is not known clearly. According to one study, the low-frequency air-bone gap observed in LVAS may be explained by the "third window" phenomenon in the inner ear.[46] In this context, the enlarged vestibular aqueduct functions as a third window, altering the compliance of the auditory system. This results in acoustic energy from stapes movement being diverted away from the cochlea and instead entering the vestibule. In cases of LVAS, pure tone audiometry (PTA) often reveals mixed hearing loss characterized by a low-frequency air-bone gap and severe high-frequency hearing loss. In many instances, the hearing loss progresses rapidly to a severe or profound sensorineural hearing loss (SNHL), making it difficult or impossible to measure bone conduction thresholds and effectively eliminating the observed air-bone gap. [47] So, patients of LVA with isolated conductive hearing loss is uncommon.[46] With advancement of newborn auditory screening, EVA is most often diagnosed in childhood. High resolution CT can visualize the anatomical anomalies of VA.

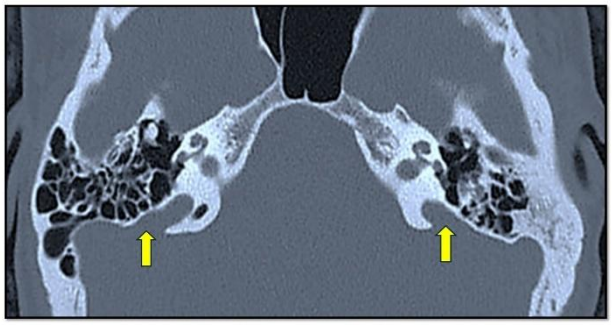

Figure 2: HRCT scan (coronal view) of temporal bone showing bilateral EVA.

VA and Meniere's disease

Valvassori first documented Meniere's like symptoms in patient with EVA.[48] The VA is an inner ear structure that connects the endolymphatic sac to the vestibule and has been associated with Meniere's disease.[49] There are two categories of Meniere's disease such as hypoplastic and degenerative types reported on the basis of differences in pathology affecting the VA or endolymphatic sac and associated differences in clinical manifestations. Meniere's disease with hypoplastic pathology presents with a VA that ends prematurely in the operculum and is associated with bilateral Meniere's disease and increased severity of inner ear swelling called endolymphatic hydrops. Meniere's disease with degenerative pathology presents with a damaged epithelium of the endolymphatic sac and is associated with unilateral Meniere's disease and an increased duration of disease onset.[50]

EVA and cochlear implantation

Children with congenital deafness are usually benefitted by performing early cochlear implantation.[51] Children with EVA often present progressive hearing loss, also benefited from cochlear implantation with understanding of outcomes in this groups.[52] The treating option for hearing loss caused by EVA has progressed over time, with cochlear implantation now considered a viable

option for children who meet the appropriate criteria.[53] The concern for cochlear implantation with EVA is persistent cerebrospinal fluid leak, incomplete electrode insertion, and postoperative meningitis.[52] The surgical technique of cochlear implantation in patients with EVA is the same as in patients with anatomically normal inner ear. However, surgeon should be prepared to face perilymph gusher or oozing.[54] In patients with EVA, perilymph is usually under pressure. Once the cochlea fenestration is made, oozing or gusher of perilymph occur. To minimize additional perilymph leakage, the cochleostomy site is sealed using a small piece of temporalis muscle along with fibrin glue.[55]

Treatment of EVA

There is no proven effective treatment for reducing hearing associated with EVA or in slowing its progression. Although some recommend steroids to treat sudden SNHL, there are no studies to show its effectiveness. The surgical method to drain the liquid from the endolymphatic sac or duct is not only ineffective in treating EVA, also it can be harmful.[55] To reduce the chance of progression of hearing loss, individuals with EVA should avoid contact sports that may lead to head injury; and avoid conditions that result in barotrauma (rapid changes in air pressure such as scuba diving or hyperbaric oxygen treatment). If an individual with EVA, the risk of hearing loss associated with air travel can be minimized by taking nasal decongestants if suffering from nasal or sinus congestion as in cold or flu.[56]

CONCLUSION

The VA is an important inner ear structure that extends from the vestibule to the posterior cranial fossa. It houses the endolymphatic duct, which terminates in the endolymphatic sac located within the bony operculum. Improved radiological investigations with newly developed 3D models may enhance the understanding of the anatomy and its anomaly of the VA. The morphology and course of the VA in the temporal bone might be associated with different clinical manifestations. Large VA is the most common inner ear dysplasia seen among patients with hearing loss. Clinicians should keep in mind about the LVA for the differential diagnosis of SNHL, progressive mixed hearing loss, and genetic syndrome for deafness.

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