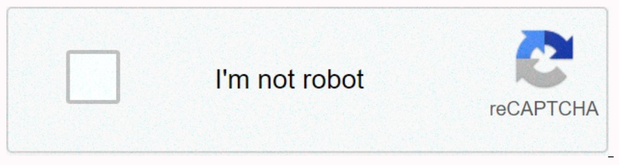

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External auditory meatus radiograph

1.Zimmerman, H.M.: Panel on Nervous System Tumors: Proceedings of the National Cancer Conference, 1949, New York: American Cancer Society, p. 227-243. Google Scholar 2.Carbtree, J.A. and House, W.F.: X-ray Diagnostics of Acoustic Neuroma, Arch. of Otolaryngology, Vol. 80, 695 – 1964. Google Scholar 3.Camp, J.D. and Cilley, E.J. cited (2). Google Scholar 4.Gupta, S.K.: Radiology of the Internal Auditory Canal, Indian Journal of Otolaryngology, Vol. 180–185, December 1971.5.Valvassori, G. E.: Radiological diagnosis of acoustic neuromas. Arch. of Otolarynaology, Vol. 83, 582-587, June 1966.CAS Google Scholar 1.Johnston, T.B. & Whillis J.: Gray's Anatomy, Longmans, Green & Co. London, New York, Toronto; Ed. 29; 1947. Google Scholar 2.Clark, K.C.: Location in Radiography, London, Ilford Limited, Wm. H. Boston Ltd., 8th Edition.3.Shambaugh, Jr. George E.: Ear Surgery, W.B. Saunders Company, Philadelphia & London, 2nd Edition, 1969. Google Scholar 4.Merrill, Viruta: Atlas of Roentgenographic Positions: Vol. Two. C.V. Mosby Company, Saint Louis, 1967. Google Scholar 5.Hitselberger, W.E. and House, W.L. Acoustic Neuroma: Adaptation of polytomography and Iofendylate to early diagnosis of acoustic tumors. Amer. Surg. 33: 791–796 (October)1967.CAS Google Scholar 6.Britton, B. Hill, Jr., Hitselberger. W. E. and Hurley, BJ: Iofendylate Examination of posterior Fossa in the diagnosis of cerebral angular tumors, Arch. ef Otolaryngology, Vol. 88, 608-617, Dec., 1968. Google Scholar Page 2 quotes are counted are provided from Web Science and CrossRef. The number may vary depending on the service and subject to the availability of their data. The number is updated daily once available. Published Online:July 1 1985 are more examining normal anatomy and discussing the characteristic findings of soft tissue abnormalities of the external auditory system (EAC). Indications for computed tomography (CT) of the time bone have been significantly expanded with the inclusion of abnormalities of the soft tissues of the outer ear and the auditory canal. Soft tissue EAC abnormalities that can be evaluated for CT, include athesia, oedema, bleeding, fracture, post-traumatic or infection-induced keloid, malignant external otitis, hemangioma, lymphangioma, papilloma, keratosis obturans, acquired cholesteatoma, adenoma, ceruminoma, fibroma, mixed tumor, sarcoma, and basal cells, squamous cells, or adenocystic carcinoma. CT scans of 25 patients who had EAC soft tissue abnormalities were reviewed. Clinical data correlated with radiographic findings. We concluded that CT is the best overall radiographic modality for assessing the extent and nature of EAC soft tissue abnormalities. Relevant clinical information that is useful in patient management is added by this technique. Posted in Print: 1985 Metrics Downloaded 925 Times External Auditory Canal and Pinna Valerie L. Jewells, Mauricio Castillo, and Craig Buchman Outer Ear, composed of pinna or ear canal and external auditory canal (EAC), has a different embryological origin than the middle and inner ears. This unique embryonic origin in conjunction with the superficial location of pinny and EAC results in a different pathological spectrum compared to the spectrum involving the middle and inner ears. Despite a simple visualization of these external structures by an otolaryngologist, a radiologist can help with important information for surgical planning and help in determining the type and extent of pathology. Intracranial complications resulting from pinna and EAC disorders can also be evaluated by a radiologist. Imaging techniques In our institution, we conduct time computed bone tomography (CT) studies more frequently than magnetic resonance imaging (MRI) studies to investigate EAC due to CT superiority in assessing bone erosion. When using a CT scanner, submilimeters (0.6 to 0.75 mm) are commonly obtained and processed by axial images and processed by bone and soft tissue algorithms. Although direct coronal imaging can be performed using a similar protocol, coronal and sagittal reconstructions can also be achieved at 0.75 mm intervals with limited radiation exposure, which is particularly desirable in children. For congenital lesions, non-comertal studies with bone windows will suffice for most patients. For some inflammatory and neoplastic lesions, contrast material and images processed with soft tissue window settings may be provided. MRI is usually used to assess the spread of tumor or inflammatory processes intracranial. Although lesions that spread through bones to the medial crayfish may be evident in upgraded coronal and sagittal images, MRI is generally better for evaluating soft tissues, brain and dural enlargement. Subtle dural enhancement may be the first sign of intracranial disability. MRI is also sensitive to the detection of bone marrow edema, and it can occasionally show bony involvement not suspected of CT in cases of cancer. Our MRI protocol includes sagittal T1 weighted images (thickness 4 mm), axial T1 weighted images (thickness 2 mm), axial T2 weighted images (thickness 2 mm) and axial FLAIR (liquid weakened by inverse restoration of T2 weighted images, thickness 4 mm), all obtained before contrast administration. After administration of gadolinia contrast, we obtain T1-weighted (3 mm thick) images in axial and coronal planes through areas of interest with and without fat suppression techniques. In addition, 4 to 5 mm thick axial and coronal postcontrast T1-weighted images are obtained to include the entire brain. Images that use High resolution T2-weighted techniques (constructive interference at steady state; ciss) may be performed if an anatomical assessment of structures in the inner ear or inner auditory canal is required. In patients with facial paralyzing, it is necessary that imaging studies include the entire course of facial nerves. Anatomy at birth, tympanic membrane (TM), ossicles, and otousicles are already adult in size, but the EAC grows gradually until the age of 9, when it reaches its typical S-shaped course and adult size. The EAC extends from meatus pinna to TM, its mean boundary that separates the outer from the middle ear. The purpose of pinna and EAC is likely to collect and funnel sound to TM as a resonating tube resulting in a 10 to 20 dB gain.1 Thus, athesia and EAC stenosis can result in conductive hearing loss of up to 60 dB, manifested as an air-bone gap for audiological testing. Adult EAC is ~ 25 mm and air filled in normal condition. EAC is lined with a squamous epithelium, which is continuous with the skin of the pinna. The channel travels inferiorly a posteriorly to TM in a slightly S-shaped course. The resulting cross-section has a roughly oblique oval configuration.2 The channel has the widest diameter at the side/superficial end adage to the pin, where it flips in the tube bell. EAC has two parts: the lateral part (one third) is fibrokartilaginous and demonstrates an incomplete elastic cartilage ring with a length of 8 mm. Inferiorly in the fibrocartilaginous part there are two deficiencies called santorini cracks that increase the elasticity of the superficial parts of the EAC, but can also allow transmission of infection and malignancy.1 The fibrocartilaginous part has thick skin with hair follicles and cerumen glands. The medial two-thirds of the EAC, adthing to TM, is a sedate part that is surrounded by a membranous bone and lacks the cerumen of the gland and hair follicles, and is lined with a thin layer of skin. The lack of thick skin and subcutaneous tissue makes this part of the canal wonderfully sensitive to touch during clinical examination. The EAC has two areas of narrowing: one on isthmus (between fibrokartilaginous and bony parts) and one distal and adjacent to TM. The medial bony part may have an ante area of dehiscence called foramen huschke. The bony part of the EAC is surrounded by squamous and mastoid segments of the time bone superiorally and posteriorly, while the ante and lower parts of bone EAC are surrounded by the tympanic part of the time bone.1 TM is located obliquely at the distal end of the EAC, conical shape and measures a diameter of 9 to 10 mm. It has peripheral anulus that bind to the tympanic ring in a well-defined bony sulcus. This fibrocartilaginous ring has an area of lack superiorly, above the short (or lateral) process of malleus, called notch Rivinus. From this notch, the anterior and posterior malleable ligaments are formed, forming folds in the TM, dating back to the attachment of the lateral process malleus. TM consists of both pars flaccida and pars tensa servings. Pars tensa consists of three layers: the inner epithelial layer of the mucous membrane, the central fibrous layer and the outer squamous epithelial layer. The inner fibrous layer gives this part of the TM a stiffer structure than the pars flaccida. Pars flaccida is a significantly smaller area than the pars tensa and is better placed above the short malleus process, covering the bony notch of Rivinius. Pars flaccida tm is thinner and therefore more flexible, and is the place of origin of many primary, acquired cholesteatomas (CHs) of the middle ear.3 Arterial delivery to the EAC is through the branches of the external carotid artery, not the posterior auter, superficial temporal and internal arteries. Venous drainage from this area is through postauricular and superficial temporal veins into the sigmoid sinus and internal and external cervical veins.1 Lymphatic drainage is lectures into the preauricular nodes, posteriorly into the mastoid nodes, and inferiorly into the subparotide nodes. All these node stations drain into digestion nodes. The innervation of the outer ear is from multiple somatosensory nerves, including crab nerves (CN) V, VII, IX, and X, as well as cervical nerves C2 and C3. Trigeminal nerve (CN V) fibers arise through the madibular division as the auriculotemporal nerve. Recent studies regarding the somatosensory contribution of CN VII to the external auditory tube have shown these branches arise in the descending or mastoid part of the facial nerve before leaving CN VII from the stylomastoid foramen. The Cn IX post, which supplies the medial aspect of TM, arises inferiorly in the hypothympanum of tympanic canaliculus. Similarly, CN X contributes to innervation to the medial aspect of EAC through the Arnold nerve, which enters the time bone through the mastoid canaliculus and exits through the tympanomastoid chiitheria.1 Embryology As with all discussions of embryology, we begin with a normal sequence of events occurring in the womb. Pharyngeal (or branch) arches arise from the migration of nerve crest cells to the fifth week of pregnancy. The first branch or maverular arch gives the origin of the maxilla, maxilla, zygomatic time bone, squamous time bone and mastification muscles, as well as the anterior abdomen of the digastric muscle, tensor tympani, tensor veli palatini and mylohyoid Also derived from the first arch are the head and neck malleus and the short process and body incus. Derivatives from the second branch arch include the long process of the incus superstructure and stapes, the smaller horn (cornea) and the upper edge of the hyoid bone, the styloid process, the stylohyoid ligament, the muscles of facial expression, the posterior abdomen of the digaspic muscle and the stapedius and stylohyoid muscles.4 The EAC arises from the ectoderm of the first and second branch arches and therefore consists of tissues from both the mandibular (first) and hyoid (second) arches. The formation of EAC occurs during the sixth week of fetal life as cleft invaginates. By this time, the tympanic cavity had already formed from the invagination of the first pharyngeal sac (tubotym panic break) during the fourth gestational week. TM forms from the meeting of these two invaginations as the collection of epithelial cells between them is canalized during the 26th century. The first three hillocks form a spiral and tragus, and the third through the sixth hillocks form the remaining auses. Therefore, abnormal development of hillock leads to microtia or pinna anotia, the severity of which is related to EAC canalization.6 Abnormal accessory hillocks can be seen in some patients as preauricular tags and/or cysts (Fig. 2.1). Pinna or auricle malformations occur in 1 in 12,500 births, and arise during the 3rd year of birth. As the bony EAC develops synchronously, but from another anlage, there may also be a small development of the bony canal.7 Fig. 2.1 Axial computed tomography scan shows incidentally found large bilateral preauricular cysts. These cysts can become symptomatic as a result of infection; However, many small remain clinically silent throughout the patient's life. Pathology Embryological microtia and external auditory dysplasia of the Mikrotia canal, which means small auricles, is more common in the Navajo and Japanese populations, as well as in fetuses exposed to thalidomide and retinoic acid.8 Mikrotia has three degrees of severity, according to classification Weerda:9 First degree: Microtia, pronounced ear, pocket ear, absence of upper spiral, absence of tragus, clefts, lobular deformation, and cup ear deformities type 1 and 2 Second degree: Dysplasia as a cup of ear deformity type 3 and mini-ear Third degree: Absence (i.e. annotation) of the normal ear structure (unilateral or bilateral), or severely dysplastic sewn inferiorly due to incomplete rise in the Mayer et al study, a third of patients had first or second degree microtiu and two thirds had third-degree microtiu. Seventy-five patients with mild microstia were associated with bony or cartilaginous EAC stenosis, and 75% of those with major microtia exhibit EAC atresia.10 Malthe development of the first brachial arch affecting the EAC may occur syndromically or nonsyndromically and may manifest as EAC athesia or stenosis, as well as duplicative anomalies, including cysts, sinuses and fistulas. These anomalies rarely occur together, but have been reported to coexist occasionally.11 Atresia EAC occurs in 1 in 10,000 births and more often includes the lateral membranous or fibrokartilabrosic part of the EAC as its bony part. EAC athesia may be associated with discharge in 18q and 66% of subjects with these discharges have congenital aural athesia or EAC stenosis.12 These anomalies can also be seen in Goldenhar, Treacher Collins, Pfeiffer and Rasmussen syndromes.13 In physical examination, the right ear is more frequently affected and patients have conductive hearing loss. Many of the severe cases may have mixed hearing loss due to associated internal ear malformations. As the EAC continues to grow during the first 2 years of life, the severity of the athesia may change during this time. Patients with EAC stenosis or athesia are usually present before discharge from the neonatal nursery because of either apparent visible pinna deformities or failure at the neonatal hearing screening examination (Fig. 2. 2). There is a three-stage severity classification system for EAC athesia as follows: 14 Fig. 2.2 A side photo of the patient shows a microtea with deformed residual pinnom and athesia of the external auditory canal. (See colour plate Fig. 2.2) Mild athesia of EAC: Normal ausesthesia, minimal ossicular deformation and normal middle ear cavity Mild Athesia EAC: Small rudimentary pinna, small cavity of the middle ear, severely deformed ossicles and aberant course of the seventh crab nerve (Fig. 2.2) Severe Athesia EAC: Missing ear shield and only a small cleft of the middle ear cavity with missing ossicles. This severe form can also have internal ear deformities. There is also a classification by location and severity range for EAC athesia as follows: 15 Type A (meat): Fibrokartilaginous with only a small Type B TM (partial): Fibrokartilaginous and osseous. The nucleus may be solid, or the manubrium may be short or curved type C (total): EAC is absent, and there is a bony atretic plate with a normal middle ear size cavity. Malleus is usually fused to the side wall. Type D (hypopneumatic sum): The cavity of the middle ear is small and there is little or no mastoid pneumatization. The facial nerve is aberant (a common form with mandibular dysostosis). The non-phyndromic causes of EAC stenosis and athesia are caused by failure of canalization. This failure may be complete, resulting in athesia or incomplete, resulting in stenosis (Fig. 2.3). It is good that severe microtia is often observed with EAC athesia, while milder microtia is usually observed with EAC stenosis (Fig. 2.4). Related dysplasia ossicles occurs in 98% of patients and 72% of them may include all ossicles, including stapes. Accompanying round window athesia is found in 6% of patients and labyrinth malformations in 13%, but not atresia oval window.10,16 Common ossicular abnormalities include short or absent manubrium malleus, more pronounced deformation of the head malleus than incus, and deformed stapes crura. Ossicular fusion was observed in 54% of patients with EAC athesia and most commonly involves malleoincudal joint (76%), followed by ossicles fusion to plate atresia. 2.3 (A.B) Computed tomography (CT) three-dimensional reformation in a patient with non-štyndromic bilateral external sound channel (EAC) atresia. In atypical EAC athesia, a zygomatic arc and a tonsils usually form, while in the syndromic atresia of the EAC they may be hypoplastic or even absent. (C) A coronal CT scan shows a thin atresia plate, a small cavity of the middle ear, missing ossicles, and soft tissue filling the epitympanic space. Coronal computed tomography (CT) shows severe right-sided soft tissue and distal bony external sound stenosis of the canal. The long process of incus is slightly deformed, but not fused. The size of the cavity of the middle ear is normal. (B) Left CT shows similar findings in this patient with bilateral microthips. The atresia plate may be bony or membranous (soft tissue) and of variable thickness (Fig. 2.5). Also, the cavity of the middle ear may be small (68%) and the facial nerve can be displaced.17,18 Uncommonly, accessory ossicles can be seen with type 2 first branch cleft anomalies.19 The association of incus and malleus abnormalities with EAC stenosis/athesia occurs due to the common (i.e. branch) embryological origin of all ossicles except stapes footplate. EAC atresia and mallear manubrium development are linked, a finding that was supported by a knockout mouse model.20 Also associated with mikrotia and EAC atresia are poor mastoid pneumatization, mandibular condyle dysplasia, zygomatic arch defects (50%), eustachian tube dysplasia (20 to 40%), tensor tympani muscular hypoplasia (20 to 40%), oval window absence (33%), labyrinth dysplasia (13%), and/or round window absence (6%). Related hypoplasia or aplasia of the inner carotid artery is rare.10 There may be associated CH medial atresia plate. More often, CH may develop in the EAC stenosis medium (Fig. 2.6). The presence of CH correlates with a smaller EAC size, so the speed of CH is 50% if the EAC is 4 mm or less in diameter.21 Fig. 2.5 (A) Coronal computed tomography scan shows right-hand membranosis atresiu absence of a bony atresi plate). Malleus and incus are deformed and fused laterally. The cavity of the middle ear is slightly small. (B) Similar findings were observed on the left side. The deformed incus is laterally muted. 2.6 (A) Axial computed tomography (CT) scan shows bone stenosis of the medial external auditory canal (EAC) with a small soft tissue mass media that has proven to be cholestatoid. (B) Axial CT in another patient exhibits bone stenosis of the lateral aspect of the EAC with a large mass of medial soft tissue, also turned out to be cholesteatoma. Of the syndromes that result in microtiu and EAC atresia, Goldenhar syndrome or hemifatial microsomia is the most common and is also the second most common craniofarial anomaly after cleft lip and palate. There are four main components of Goldenhar syndrome: ear deformity, EAC athesia, malformation of the tympanic cavity and ossicular abnormalities causing conductive hearing loss. Sensorineural hearing loss also occasionally occurs due to stria vascularis and semicirural canal or cochlear abnormalities, as well as hypoplastic or atheric oval windows.21.22 Pfeiffer syndrome is another cause of EAC stenosis/atresia resulting in moderate to severe conductive or mixed hearing loss in patients with kranio-facial abnormalities. In addition to stenosis and/or athesia of EAC, hypoplasia of the middle ear cavity and occasionally hypoplastic ossicles. Typically, inner ear anatomy is normal, but middle ear effusion is often seen. Therefore, when patients with Pfeiffer syndrome receive a CT scan for craniofarial anomalies, a time bone examination should also be performed. Eighty-five percent of these patients are located with bilateral microtia, as well as bilateral abnormalities of EAC, TM, ossicles and the central ear cavity. These patients will also have midface hypoplasia and micrognathia.24,25 Pierre Robin syndrome (also known as Pierre Robin sequence) demonstrates retrognathia, glossoptosis, and bilateral cleft palates, as well as ear abnormalities. Ear abnormalities include abnormal pinna and ossicles, as well as abnormal stapes footplates in 50% of patients. Aplasia of the lateral semicirular ducts (LSCC), large vestibular acroducts, and unusually large otoconie are also seen, but not EAC atresia or stenosis.26 Because the middle ear arises from the branch arches, while the inner ear is not, labyrinth abnormalities such as aplasia and division deficiencies are usually not associated with microtia and EAC athesia/stenosis. When interpreting CT time bone studies conducted in patients with congenital abnormalities, our report addresses the following: The extent of EAC athesia and its nature (membranous, bony, combination of both) Thickness of atresia plate Amount of mastoid pneumatization Normal or abnormal medial crayl foss (especially if too low) Temporomandibular location of the joint Presence or absence of CH (Fig. 2.7) Ossicular fusion– incudomal and incudostapedial maintenance of joints and if ossicly melted to the Atresian plate (Fig. 2.8). (Rotation of the long incus process resulting in a bold angle to the lenticular process should also be indicated and the incudomal and incudostapedial joints are visible on the same axial slice.) Stapes present, dysplastic, or missing (may be useful information, if a prosthesis location is necessary) Size of the oval window (normal size is 2 mm in vertical diameter)27,28 Central ear cavity size in all three axes (if < 3 mm width from lateral edge to cochlear protrusion, surgery may be excluded)28 Internal ear structures and size of internal auditory canal (IAC; rarely affected EAC dysplasia)28 Internal ear structures and size of internal auditory canal (IAC; rarely affected EAC dysplasia , but if abnormally may exclude surgery. If the IAC is small, there may be associated with a sochlear nerve deficiency, but a lack of the parachutic nerve may also be present with a normal IAC size, and MRI may be indicated for the assessment of the condition of the cochlear nerve).29,30 The course of the facial nerve with particular attention to its horizontal part of the middle ear and its descending part (location of the stylomastoid foramen) (Fig. 2.9) Three-dimensional computed tomography (CT) surface that is plotted in a patient with hemifacial microsome , shows a missing external sound channel, a hypoplastic zygomatic arc and a thin, small vertical tidibular ramus. (B) In another patient even with a hemifatial microsome, a CT scan surface rendering of bones shows a missing EAC, partly lacking a zygomatic arc, and a hypoplastic ipsilateral mandible. (C) In the same patient, the CT surface rendering of the skin shows hypoplasia of the right side of the face and the missing pinna on that side. (See colour plate Fig. 2.7.) Coronal computed tomography shows severe bony stenosis and membranosis athesia. The small external auditory canal is vertically oriented. Malleus and incus are muted and deformed, and this ossicular mass is fused laterally. The facial nerve is directly behind the deformed ossicles, and the medial ear cavity is small. Drawing showing microtiu, bony athesia and deformed ossicles. The facial nerve descends forward under the lateral semicirural canal and in front of the stapes, and so is prone to injuries when drilling the atresia plate to gain access to the cavity of the middle ear. (Courtesy of Kind Adjrding Suresh Mukherji, MD, Ann Arbor, MI.) (See colour plate Fig. 2.9) Surgical reconstruction for ear and EAC atresia is considered separately. Usually, microtia is a cosmetic procedure that is performed before entering primary school. Different and materials have been used, but currently autogenic rib notching using multistage surgery produces excellent results when performed by a highly experienced surgeon. Others use alloplastic materials with very good results as well. The formation of an EAC with an intact conductive auditory mechanism usually follows ear surgery, so there is no interference with the heating of the skin flaps and implanted framework. Reconstruction of the EAC is carried out either to improve hearing or when CH is available. Bilateral involvement therefore constitutes a clear indication for surgery. After early identification, these children are suitable with a bone guidance hearing aid to ensure normal auditory receptive abilities prior to surgery. With this device, speech and language acquisition usually proceed normally. Atresiaplasty in unilateral cases is reserved for those patients where the anatomy is favorable, family expectations are appropriate and the child is very cooperative. Favorable anatomy is generally present when the morphology of the inner ear and facial nerve is normal, sleep bone pneumatization is good and the ossicular chain is only slightly deformed. Jahrsdorfer et al24 has set up a classification system to assist decision-making in this regard. For atresiaplasty, EAC is created by drilling the posterior glenoid fossa and before the mastoid air cells, entering the epitympanum and an excellent aspect of the middle ear space just below the level of the medial craywood fossa dura.28 The ossicular chain is mobilized from the atresia plate, and TM is created using temporalis fascia grapht. Finally, the canal is lined with skin graft with split thickness, and carbonoplasty is formed by connecting the skin graft to the newly formed carotene hole.14 Good hearing results (air-bone gap of 25 dB) can usually be obtained in 75% of patients and depend on favorable anatomical factors. In cases where good hearing is not realized from the procedure, a conventional hearing aid can be used with excellent results. In children in whom anatomical factors exclude EAC reconstruction or the risks appear unacceptable to the family, an osseointegrated cochlear stimulator (BAHA, Cochlear Corp., Englewood, CO) may be considered. Postoperative complications from reconstruction of EAC atresia include facial paralysis, sensorineural hearing loss, conductive hearing loss, TM perforation, cerebrosietic mucus leakage (CSF) and slashed stenosis secondary to bony resupply or soft tissue stenosis. This latest complication is generally considered to be the most common.31 The most common complication of EAC athesia surgery is a cheek nerve injury. Although rare, the facial nerve is at risk of injury in both its descending segment and in the extracranal part. In the intratemporal part, the front displacement of the descending is the norm, making it most vulnerable when drilling the sound system inferiorly. The extratemporal part of the facial nerve is at risk of injury during caroplasty, especially when the cartilaginous framework of the microtia repair lies in front of a newly formed sound system that requires mobilization for alignment. Other complications of atresiaplasty include persistent conductive hearing loss from sound system stenosis, TM perforation or lateralization, and discontinuity or fixation of the ossicular chain. Sensorineural hearing loss can occur from inadvertent labyrinth injury when drilling around the ossicular chain. We recently started using a laser to lyse the final attachments of the ossicular chain to avoid such trauma caused by the drill.30 Fig. This is a

proven type 1 branch cleft cyst that has spread to the EAC through a defect in its floor. Brachial cleft cyst Brachial cleft cyst (BCC) is a congenital lesion that can occur in the EAC area and is the cause of one third of childhood non-neural lesions in the area of the esoteric gland requiring surgery.32 BCC result from insufficient involuction of structures from the first to fourth branch arch and are classified by location. Pathologically, BCC consist of a thin fibrous pseudocapsule with a central spinoid epithelium and occasionally lymphocytic and germ tissues. Most BCC are simple cysts (two-thirds) with a thick content of the mucous membrane, without skin or airway opening, and 3 cm or less in size. Due to their histological components, BCCs are similar in their imaging appearance, regardless of their location. Rarely, BCC undergoes a malignant transformation into squamous cell carcinoma.33 For physical examination, BCC is compressible due to its fluid components and usually painless. These lesions tend to increase with upper respiratory tract infections due to lymphoid secretions from follicles in the cyst wall and can be painful even in the absence of infection. BcC is easy to evaluate ct. Their appearance is that of a well-bound mass with fluid-like components centrally (Fig. 2.10).34 On MRI, BCC is a low T1- and high T2-weighted signal and may show an increase in rim after administration of contrast. An increased protein content may lead to higher T1 signal weighing and occasionally low T2 strength of the weighted signal. In the absence of infection, FLAIR imaging will demonstrate the central fluid will have a low signal. If there is an accompanying infection, surrounding soft tissue edemia (fat stranding) will be present on the CT and MRI and the rim tends to be thicker, nodular and increase. MRI can be useful for localized sinus/fistula placement. CT better evaluates bone abnormalities.35 Fistulography may also be better assess the course, anatomy and topography of the fistulous tract, which helps to improve the rate of complete resection of the fistula associated with BCC.36 In this chapter, we focus on the first BCC because they occur in the EAC area. The first BCC are less common than second BCC cysts and account for less than 8% of all BCC. The first BCC arises at the periurivascular site, often parallels the EAC in orientation, and can be coated with the esoteric gland. The congenital tract or sinus of the BCC can interact with the EAC. Due to this embryological development, the first BCC may be associated with other first cleft anomalies, including heterotopic tissue of the salivary glands, which is predisposed to malignant transformation37 or CH.38 Although there are two possible subtypes of the first BCC, for each of them there are no strictly or precisely defined histological and anatomical properties. The probability of an associated sinus with the first BCC is 56% and the fistulous tract to the EAC at the cartilaginous-bony junction occurs in 31%, while the remaining first BCCs have simply a cystic appearance.39 The first BBC usually represents, while the patient is a child, one third of patients who become acute as a result of infection and two thirds representing asymptomatic swelling , but can also be found in adolescents or young adults if the lesion is purely cystic and not infected. Often BCC is treated with complete resection, to prevent recurrence and reduce complications.41 Since many BCC present as an abscess can be misdiagnosed and treated with a simple incision and drainage instead of complete excision.42 The surgeon must be careful not to injure the adjacent extratemporal part of the seventh crabase nerve.39,43 Due to the variable relationship of BCC to the facial nerve, imaging is important for surgical planning. Even with the identification of the facial nerve during surgery, there is a 21% incidence of temporary and 1% incidence of persistent facial palsy after resection of the first BCC. In addition, the likelihood of a facial nerve injury increases with the number of previous infections and operations.44 Recently, methods other than surgical excision for the treatment of BCC have been described. An endoscopic approach to BCC excision through a small transcervical incision has been described and is said to have minimal morbidity.45 However, data on the safety and efficacy of this approach are lacking. Also described is the use of ethanol injections for Sclerotherapy BCC.46 Differential diagnosis for BCC includes other cystic lesions such as lymphangiomas, which are usually multiloculated, nonenhancing, and transspatial lesions. Cystic lesions in the area of the anesthetic tail include cysts with AIDS, parotid cysts in Sjogren's syndrome, and less often, cysts in Reiter syndrome. Infected intraparotid or purulent crustacean lymph nodes or rarely intranodal necrotic metastasis from squamous cell or thyroid cancer should also be considered.47,48 Lymphoma, Both Hodgkin and the non-Hodgkin type are also in differential diagnosis, but usually show more mass/enlarged nodules.49 Another unusual entity in the differential is cystic schwannoma from the facial nerve.50 Cystic hygroma and cystic lymph angioma lymphangiomas occur less frequently than BCCs. Embryologically, lymphang can have two possible origins: they can arise secondary to the failure of embryonic fusion between the central venous system and lymphoid bags, or from sequestration of lymphoid bags. In both cases, the condition is associated with Turner and Noonan syndrome, as well as with fetal alcohol syndrome. Clinically, most cystic hygromas and lymphangiomas present in the first 2 years of life and rarely in adulthood.51 In physical examination, lymphangiomas are compressible masses, which usually include submandibular and posterior triangular areas, which can lead to airway obstruction. Lymph angiomas can also spread to the mucous surfaces of the oropharynx, tongue or airways. Pathologically, they consist of endothelial enlarged endolymphatic spaces with septations of variable thickness and occasionally vascular structures. Depending on the size of the lymphatic spaces in the lesion, there is a pathological continuum with cavernous hemangioma, capillary lymphangiomas (the least common form with the smallest spaces) and vasculolymphatic malformations. Different types of lesions cannot always be differentiated by imaging, but cystic hygroma is the most common type.52 Contrast administration is necessary to assess these lesions, as the presence of venous components may alter the surgical approach. In imaging, the most important feature of cystic hygraine or lymphangioma is the tendency to induce into multiple departments in a transspace way and surround normal anatomical structures such as muscles and blood vessels. Therefore, lymphangioma is less well bounded than BCC. Lymphangiomas are most common multilocular and nonenhancing, although they can rarely increase if infection overlaps. In addition, when the infection is present, it may spread beyond the lesion.53 For CT scans, these lesions may show fluid and fluid levels and occasionally exhibit venous (increasing) soft tissue strengthening components or areas. At MRI, lesions may have a simple cystic appearance, but if there has been prior bleeding or proteinaceous fluid, they will have high T1-weighted signal and/or fluid-fluid levels. The differential diagnosis of lymphangioma includes other slow-growing cervical masses, including schwannoma, haemangioma, vascular malformation and sublingual salivary musculature (i.e. ranula or pseudocyst). Acute swelling and faster presentation were observed in purulent lymphadenopathy secondary to sinusitis, odontogenic infection or abscess. BCC with overlapping infection is more common than lymphangioma. If there has been a rapid clinical change, such as swelling and/ or crab nerve deficit, a vague process such as rhabdomyosarcoma, Histiocytosis of Langerhans cells, Ewing's sarcoma, osteogenic sarcoma or metastatic neuroblastoma should be considered. In our institution, most patients are evaluated with contrast-enhanced CT scan of the neck, since the diagnosis is not always known during presentation, and CT scans can exclude most of the other lesions listed above in the differential. Imaging prior to surgery allows staging of the lesion, determination of unilaterality or bilaterality, infra- or suprahyoid range and mediastinal involvement. These staging differences predict surgical outcomes as well as complication rates and morbidity.55 MRI reveals a related pathology that has not been seen in the U.S. in 20% of patients.56 MRI is also useful for assessing the amount of tracheal/respiratory tract compromise.57 Standard treatment for lymph angiomas is surgical. Recently, new types of interventions are used, including aspiration of the cavity guided by U.S. injection of bleomycin, which led to good reactions >i in more than 50% of patients. Cystic hygromas.60 In a minority of patients, lymphangiomas may disappear/involute on their own.61 Lymphangiomas are known to recur, especially if uncapsulated; therefore, they should be closely monitored regardless of the course of treatment used. Foramen of Huschke Approximately 4.6% of patients have an area of medial bony dehiscence involving anterior and lower EAC called foramen Huschke. Normally, foramen huschke smoothes in childhood or childhood as U-shaped EAC cartilage passes through fusion into a complete circle. On physical examination, the persistent foramen may be presented as a small polyp or dooter on the front wall of the EAC. These congenital fistulas are rare. Foramen is easily detected by high resolution CT, located posterior and medial temporomandibular joint (TMJ), and measures ∼3 to 4 mm in size. Patent foramen huschke is more common in women and can cause transient otorrhea from TMJ synovial fluid. Rarely, soft tissue posterior tmj meniscus can herniate into the EAC during mouth closure. Rarely, this tract can act as a portal spread of infection or tumour between EAC and TMJ. Its presence can be identified with high resolution (0.6 mm thick) CT imaging, is seen as bony EAC thinning (< 1.0 mm) predsane and inferiorly, and is usually bilateral. Normally, the foramen huschke closes at the age of 5 years and its endurance is an anatomical variant.62 Salivary otorrhea from huschke patent foramen represents a serous discharge from EAC that occurs more frequently with food. This fluid demonstrates the presence of amylase when it is antistained with iodine on a starchy agar plate. Sialography may show the presence of a fistula on the EAC or only chronic ethetode sialadenitis without a definitive fistula. The MRI reveals a clear T2-weighted signal in an adjacent ezote secondary to sialadenitis and/or fluid in the EAC. Usually the defect is repaired surgically using temporalis fascia and tragal perichondrial grafts.63 Inflammatory Otitis Externa There are six forms of otitis externa (OE; also known as external otitis); acute, chronic, eczematous (dermatitis, psoriasis, lupus or infantile eczema) fungal and necrotising/malignant. Acute uncomplicated external otitis Acute uncomplicated external otitis or swimmer's ear is the most common external infection of the ear. Patients with OE present with pain, erythema, swelling and severe tragal and ear tenderness of movement. Occasionally, conductive hearing loss may be present when swelling of the canal smoothed the patency of EAC.64.65 Most often this condition is caused by bacterial infection from Pseudomonas aeruginosa or Staphylococcus aureus. Otomycosis or fungal OE usually results from infection from either Candida or Aspergillus. Presentation usually follows the administration of several oral or topical antibacterial medicinal products and may be quite refractory to conventional therapy.66 There is also an increased risk of otomycosis in diabetic and/or immunocompomommitized patients.67 When otomycosis is complicated by perforated otitis media or inflammation of the tube, Therapy can be very challenging because most drugs that are active against fungal species have not been approved by the U.S. Food and Drug Administration or are safe for middle ear use. Less often, acute OE can be the result of a viral infection. Ramsay Hunt syndrome or herpes zoster oticus is due to infection of the seventh and eighth cranial nerves by reactivation of latent herpes zoster virus in the geniculate, spiral (i.e. sochear), and /or scarpa's (i.e. vestibular) ganglia. Classically, this disease presents with acute facial palsy and vesicular eruption in the distribution of somatosensory fibers of the facial nerve in the EAC and auterine.68 When the vestibulocochlear nerve is involved, sensorineural hearing loss and vertigo may be present to varying degrees. Treatment of this disorder requires systemic corticosteroid therapy and an antiviral medicinal product with efficacy against an illegal agent (i.e. valacyclovir). Only members of gold can continue reading. Sign in or sign up to continue

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