“ROLE OF HIGH RESOLUTION COMPUTED TOMOGRAPHY IN EVALUATION OF PATHOLOGIES OF TEMPORAL BONE”

By

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ABSTRACT

BACKGROUND & OBJECTIVES

Pathology of the ear is one of the most common reasons of visiting an otolaryngologist, with inflammatory conditions being predominant. The evaluation and diagnosis of complex lesions of the temporal bone is challenging task both for the radiologist as well as otolaryngologist. Earlier, clinical examination was used along with X- rays for the diagnosis. However with increasing prevalence of infective pathologies of ear, this approach proved inadequate.

Complicated anatomical structure of middle and inner ear makes radiographic assessment of temporal bone difficult. CT has the advantage of producing images with higher contrast and a better spatial resolution. High resolution CT (HRCT) images are obtained with thin sections and special bony algorithm for high details. HRCT, a modification of routine CT, provides a direct visual window into the temporal bone providing minute structural details. It is an excellent tool for evaluation of the middle ear diseases and adjacent bone and has the advantage of being devoid of artifacts from superimposition of structures. HRCT also helps in accurate assessment of pathology prior to surgical exploration regarding location, extent and complication of the disease.

This study is undertaken to study congenital anomalies, infective, trauma and and neoplastic pathologies of the temporal bone along with their complications on HRCT.

AIMS & OBJECTIVES OF THE STUDY:

1. To study the extent of middle ear infections and their complications.

2. To study the congenital anomalies of the ear according to compartment involvement.
3. To evaluate changes in temporal bone due to trauma.
4. To characterize neoplasms and assess their extent of involvement in temporal bone.

SOURCE OF DATA:

Data for the study is collected from the patients attending/referred to the Radiology department of B.L.D.E.U's Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur who fulfill the inclusion criteria.

METHOD OF COLLECTION OF DATA:

The study was done on patients, who visited the Department of Radio Diagnosis during the period from OCTOBER 2015 to MAY 2017. Consent taken for each case.

RESULT: In our study series of 48 cases, we got 26 cases of infective etiology, 12 cases of traumatic etiology, 8 cases of neoplasms and 2 cases of congenital anomalies. Majority of the temporal bone pathologies included infections (72%). Among infective conditions, cholestetoma was commonest with more preponderance in young age. Trauma accounted for 14% followed by neoplasms (10%) and congenital anomalies (6%).

INTERPRETATION: HRCT is a revolutionary imaging modality that helps in evaluating the normal anatomical structures, normal variants, distribution features, localization and assessing the extent of various pathologies affecting the temporal bone.

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INTRODUCTION

The ability to image the human central nervous system non-invasively has completely changed the diagnostic approach to pathology of the brain.

Many imaging modalities are available for the evaluation of the temporal bone, including plain radiographs, angiography, cerebrospinal fluid (CSF) analysis, air and non-ionic contrast cisternography, computed tomography (CT), and magnetic resonance imaging (MRI). CT and MRI are currently the most widely used techniques and have largely replaced the other modalities.

Conventional radiography has been of value in screening the entire temporal bone. It produces a composite single plane image of a tridimensional temporal bone resulting in superimposition where larger and denser structures obscure smaller and less denser ones.

MRI has expanded the range of pathology that can be accurately evaluated because it can image many soft tissue entities not visible by other techniques. MRI studies can also be extremely useful in the evaluation of blood vessel related disorders of the temporal bone.

Angiography is still the "gold standard" for vascular evaluation, and interventional angiography can be used in treatment of vascular lesions of the temporal bone. Each technique has its own advantages and disadvantages, and often more than one examination is necessary for a complete temporal bone evaluation.

Ear pathology is a common reason of visiting an otolaryngologist, with inflammatory conditions being predominant. The evaluation and diagnosis of complex lesions of the temporal bone is challenging task both for the radiologist as well as

otolaryngologist. Earlier, clinical examination was used alone for the diagnosis. However with increasing prevalence of infective pathologies of ear, this approach proved inadequate.

Complicated anatomical structure of middle and inner ear makes radiographic assessment of temporal bone difficult. CT has the advantage of producing images with higher contrast and a better spatial resolution. High resolution CT (HRCT) images are obtained with thin sections and special bony algorithm for high details.[1] HRCT, a modification of routine CT, provides a direct visual window into the temporal bone providing minute structural details. It is an excellent tool for evaluation of the middle ear diseases and adjacent bone and has the advantage of being devoid of artifacts from superimposition of structures. HRCT also helps in accurate assessment of pathology prior to surgical exploration regarding location, extent and complication of the disease.

The purpose of the study is primarily to understand the capability of HRCT in diagnosis and detection of pathologies of the temporal bone.

AIMS AND OBJECTIVES

1. To study the extent of middle ear infections and their complications.
2. To study the congenital anomalies of the ear according to compartment involvement.
3. To evaluate importance of HRCT in temporal bone trauma.
4. To evaluate HRCT appearances of neoplasms involving temporal bone.

METHODOLOGY

This study evaluating the efficacy of CT in the diagnosis of temporal bone pathologies was done on 48 cases. This study was conducted during the period from OCTOBER 2015 to MAY 2017 in Radiology department B.L.D.E.U's Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

Source of Data:

The main source of data for this study are patients from B.L.D.E.U's Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

SAMPLE SIZE:

Based on the incidence of patients for inactive chronic otitis media (2%).

At 95% confidence level and 4 % allowable error the sample size is =48

$$n = \frac{z\sim^2 p(100-p)}{D^2}$$

Hence a minimum of 48 cases will be included in the study.

SELECTION OF PATIENTS:

INCLUSION CRITERIA:

Patients who are clinically suspected of having features like hearing loss, ear discharge, bleeding from ears, otalgia, vertigo, post auricular swelling, fever, facial nerve weakness, headache, ear pain, tinnitus or diplopia.

EXCLUSION CRITERIA:

1. Patients with electric devices at the skull base, such as cochlear implants, were excluded from the study.

2. Patients who are pregnant.

CT Machine:

All the HRCT scans were performed at our institute on SIEMENS SOMATOM SCOPE 32 slice CT scan, which is modified third generation machine, available at Radiology department B.L.D.E.U's Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

Patients were scanned in the axial and coronal (supine or prone) axes. Scout films were taken routinely in all patients before starting the scan. Scanning commenced from the lower margin of the external auditory meatus and extended upward to the arcuate eminence of the superior semicircular canal as seen on lateral topogram.

Slight extension of the head was given to avoid gantry tilt and thereby protect the lens from radiation. Coronal images were obtained perpendicular to the axial plane from the cochlea to the posterior semicircular canal.

Contiguous 0.6 mm mm thick slices were obtained using an ultra high algorithm with a scan time of 4 seconds at a 133KV tube voltage. The mA selected was 70.

At 133KV, the noise level is low, bone penetration is better and there is minimal beam hardening.

At 70 mA, the soft tissue differentiation is better. A long scan time of 4 seconds increases the image sharpness but there is a greater probability of motion artifacts.

Intravenous Contrast was Administered to Study

1. Hypervascular lesions like glomus tumours
2. Cerebello pontine angle masses
3. Intracranial or extracranial extension of middle ear disease

Preparation of Patients:

Prior to performing the scan particularly in infants and children less than six years, sedation was usually required. The purpose of sedation was to avoid motion artifact and to ensure a CT scan of diagnostic quality.

From six years onwards the need for sedation generally decreased. Sedatives used in our institution were Pedichloryl syrup administered orally or Injection Midaz administered intravenously in the dose of 0.1 – 0.3 mg/kg dose.

Patients were kept nil orally 4 hours prior to the procedure to avoid complications of contrast In infants the last feed before the procedure was omitted.

HRCT Technique:

CT excels in the evaluation of disorders that primarily affect air spaces or cortical bone.

The optimal technique for HRCT was described in detail by Shaffer and Turski. Gantry angulations for axial and coronal scans have been suggested for evaluating specific intratemporal structures.

If the goal of a temporal bone CT study is to focus on the otic capsule, cortical plates, ossicles and the air spaces alone, then high resolution bone algorithm techniques may be adequate. However, if it is also important to evaluate the soft tissues, as in the case of a patient with cancer of external auditory canal, then it may be necessary to use intravenous contrast and techniques similar to those used for a brain or soft tissue neck study.

HRCT comprises the use of a thin collimation, a high spatial frequency algorithm, smallest practical FOV (15 to 20cm) and a large reconstruction matrix (512 x 512). With 1 cm collimation the volume averaging within the plane of scan reduces the ability of CT to resolve small structures significantly. Therefore, scanning with thin collimation is essential.

A high spatial frequency algorithm reduces image smoothening and increases spatial resolution, making structures appear sharper. This also increases the noise present in the image, which is reduced by increasing the KVp and MAs setting.

CT images are usually acquired or displayed in axial and coronal planes. For axial imaging, sections are made in a plane rotated 30^0 superior to the anthropologic base line. Scan produced in this plane display the temporal bone structures to good

advantage. This plane allows separation of individual component of the temporal bone so that they are better visualized in their entirety, with less of overlap and fewer partial volume imaging artifacts.

Retrospective image targeting and reconstruction of the other side from stored raw data, significantly reduces image pixel size and increases spatial resolution.

The important patient factor influencing HRCT is motion. Therefore, patients were instructed to be motionless during the procedure.

For contrast enhancement, a bolus injection of Iohexol USP was given in the dose of 300 mg lodine/kg of body weight.

This was given just before the contrast enhancement CT procedure.

Statistical Analysis:

All the data were expressed in numbers and percentages.

REVIEW OF LITERATURE

BASIC ANATOMY

Embryology & Development:

Development of inner ear structures (vestibule, semicircular canals, porus acousticus, cochlea) is independent of middle ear (ossicles, mastoid antrum, tympanic membrane, middle ear air spaces) and external ear (external auditory canal (EAC). This is the reason why external and middle ear abnormalities are independent of inner ear abnormalities and vice-versa. Mechanical sound conducting structures of the external & middle ear get developed from the branchial arches viz. first and second. [(2)] The neural sound perceiving apparatus of the inner ear develop from the ectodermal otocyst. The rest of the ear develop from the adjacent mesenchyme. [(3)]

1st and 2nd branchial arches mold two significant cartilaginous structures. The head of malleus & short process of incus develops from Meckel's cartilage which is developed from the 1st branchial arch. [(4)] 2nd arch forms Reichert's cartilage, from which remaining part of malleus and incus, styloid & stapes superstructure develop. Stapes footplate is a bilaminar structure, with an outer portion developing from Reichert's cartilage and an inner portion developing from the ectodermal otocyst.

The antrum of mastoid & middle ear cavity are filled with fluid till birth. Normal first breathing and crying causes filling of the air in middle ear cavity and Eustachian tube.

The middle ear cavity and mastoid antrum are fluid filled until birth.The

neonate's initial crying and breathing fill the Eustachian tube system and the middle ear with air. The mastoid air cells develop as sac like extensions from the mastoid antrum, commencing at about the time of birth and continuing for several years. There is extensive variation in the degree of pneumatization. Incomplete pneumatization of the air cells of mastoid may be caused by lack of proper function of the Eustachian tube during early life.

ANATOMY

There are two temporal bones which are placed at the base of skull laterally and comprises of following parts [(5)]

- Squamous
- Mastoid
- Petrous
- Tympanic
- Styloid process

The squamous portion is easily seen on routine skull films. The styloid process can be studied in a prone Townes projection.

The tympanic and petro-mastoid parts are of primary interest and will be discussed here in detail.

SQUAMOUS:

The squamous portion forms the anterolateral, thin shell like part of the bone from which arises the zygomatic process. The temporalis muscle is attached to the external surface.

The inner surface is concave and irregular. Meningeal vessels groove the inner surface. The superior border gives articulation to parietal bone & anteroinferior border with the greater wing of sphenoid. (5)

STYLOID PROCESS:

The styloid process is 2.5cm long and projects downward and forward, anterior to the stylomastoid foramen.

MASTOID:

The mastoid portion is hollowed to form a number of mastoid air cells[1].The largest air cell which is situated in the upper and anterior part is the antrum.It communicates with the remaining air cells and attic by a narrow channel called aditus ad antrum. In the supero-anterior aspect of the bone these cells are large and irregular, towards the middle they diminish in size and in the apexes they are small. (5)

PETROUS PORTION:

The petrous portion is a three sided pyramid resting on its side, wedged between the sphenoid and occipital bones with its long axis 45^0 to sagittal plane. Its base is lateral and apex is directed medially. The apex has a shallow depression medially where the semilunar ganglion lies. (Meckel's cave).

Middle cranial fossa is separated from petrous portion by its anterior surface. In mid portion , arcuate eminence formed by underlying superior semicircular canal. The tegmen tympani separates the tympanic cavity & cranial cavity. (6)

Posterior surface forms bony demarcation between posterior fossa and the tympanic cavity. It is more vertical. Near its centre is the internal auditory meatus

which is the passage for VII & VIII cranial nerves. Lamina spirales is a bony plate which closes the internal auditory canal on lateral aspect and seperates the vestibule and fundus of canal. Fundus is further divided by a bony crest cristafalciformis into the smaller upper and the larger lower compartment. Postero-inferior to the internal aqueduct, superiorly and inferiorly are the respective petrosal sinuses.

THE TYMPANIC PORTION:

It is a 'C' shaped curved plate which acts as anterior wall, floor and postero-inferior part of external auditory canal. At medial end tympanic membrane lodges in the tympanic sulcus. The lateral border forms a large part of the margins of the opening of external canal. [7]

THE EXTERNAL AUDITORY CANAL:

It comprises of a lateral fibro-cartilagenous part and a medial bony part. The osseous part is a bony canal 16mm long and is directed downwards, forwards and inward. On sagittal scan the canal appears elliptical or oval in shape & its long axis is directed slightly posteriorly & inferiorly. [5]

The orientation of tympanic membrane is oblique such that the inferior and anterior walls of external auditory canal are longer. It forms the medial boundary of the external auditory canal, separating canal from middle ear cavity. It has two parts

a. Pars tensa
b. Pars flaccida

THE MIDDLE EAR: [5]

- It is an irregular cavity between external and internal ear within temporal bone.
- It is mainly filled with air coming from the nasopharynx via Eustachian tube.

- It is transversed by an ossicular chain, connecting the lateral and medial walls.

CONSISTS OF THREE PARTS:

a. Mesotympanum
b. Attic
c. Hypotympanum

ROOF OR TEGMEN WALL:

The tegmen tympani is a plate of bone that originates from the petrous part. It separates middle cranial fossa from tympanic cavity. In children, lateral margin of tegmen tympani may be unossified and allows passage of infection from middle ear to epidural space. [8]

FLOOR/ JUGULAR WALL:

Floor is formed by a bony thin plate separating the hypotympanum and internal jugular vein.

Jugular foramen is a complex canal coursing anteriorly, laterally and inferiorly to exit from the skull base. It has a smaller anterior compartment (pars nervosa) and larger posterior compartment (parsvascularis). The terminal portion of the sigmoid sinus flows anteriorly to enter the jugular foramen (pars vascularis), turns laterally to expand and form the jugular bulb and then drains inferiorly into the

internal jugular vein.

The Carotico-Jugular spine is a vertically oriented plate which separates the jugular foramen from the carotid canal.

Posterior or the Mastoid wall:

It has the aditus ad antrum superiorly communicating epitympanic recess and antrum of mastoid. [9]

Pyramidal eminence is a 'W' shaped elevation situated behind the oval window and gives origin to the stapedius muscle. It divides the posterior wall into two recesses.

- The facial recess between pyramidal eminence medially and bony tympanic annulus laterally.
- The sinus tympani between the labyrinthine wall medially and pyramidal eminence laterally.
- The incudal fossa is a shallow depression in the epitympanum for the attachment of the posterior ligament of the short process of incus.

CAROTID /ANTERIOR WALL:

It's wider in the superior aspect as compared to lower and is corresponding to carotid canal. A thin cortical bony plate separates them. Internal carotid artery is in close relation to the horizontal vertical segments of anterior wall. Superiorly are the orifices for origin of semicanals of tensor tympani and Eustachian tube.

EUSTACHIAN TUBE:

Tympanic cavity communicates to nasopharynx through this tube. It is 3.5cm and is directed downward, forward and medially.

It has both osseous and cartilaginous parts. The pharyngeal opening of the cartilaginous portion is 'C' shaped and can open its lumen maximally during swallowing. This helps to ensure that the middle ear and pharyngeal air pressures are equilibrated during swallowing.

MEMBRANOUS/ LATERAL WALL:

It is made by tympanic membrane. It is lodged in the tympanic ring. It is directed downwards and medially with an angle of 50^0 to the floor of external auditory canal. It is divided into two parts by the manubrium of the malleus. The superior pars flaccida and the inferior pars tensa.

LABYRINTHINE/ MEDIAL WALL: [10]

It lies in between inner and middle ear. Anterior limb of lateral semicircular canal produces a prominence below which & anteriorly intratympanic part of 7^{th} nerve produces prominence. Terminus of septum canalis musculotuborii lies anterior to this, which lies in region of position of geniculum of facial nerve.

Oval window niche lies immediately inferior to canal of the facial nerve canal, which consists oval window at its medial terminus. The promontory is a convex bulge formed due to otic capsule on cochlear basal turn.

Below and behind promontory lies round window niche leading to round window. Posterior to promontory is subiculum promontorii which forms inferior border of tympanic sinus.

THE TYMPANIC CAVITY: [11]

It consists of three parts:

Mesotympanum	:	Medial to the tympanic membrane
Epitympanum/attic	:	Above tympanic membrane level
Hypotympanum	:	Inferior & medial extension of mesotympanum

Contents:

1. Auditory ossicles
2. Ligaments and muscles
3. Facial nerve

AUDITORY OSSICLES: [5]

- The ossicular chain is placed in between medial and lateral wall of middle ear cleft and acts as a sound conducting medium.
- Parts of malleus are anterior process, lateral process, head and neck.
- Head lies in the epitympanum.
- The neck (manubrium) attaches to tympanic membrane.
- Lateral process abuts tympanic membrane below pars flaccida.
- Anterior process is a very small spicule of bone.

INCUS:

It consists of a two processes & body.

The body has an anterior concavo-convex facet, which articulates with the head of malleus.

The short process is placed horizontally and directed backwards. It is attached to the incudal fossa. The long process descends parallel to the manubrium and forms lenticular process.

STAPES:

Consists of 2 crura, foot plate and head. It has articulation with lenticular process of incus. Neck is constricted. Stapedius muscle is inserted on its posterior aspect.

The anterior and posterior crura diverge from neck & meet the foot plate. The foot plate covers the oval window. The ossicles are attached to walls with muscles and ligaments.

LIGAMENTS AND MUSCLES:

The anterior malleolar ligament commences from the neck of malleus and is inserted over the carotid wall.

The superior malleolar ligament is attached from the roof of epitympanum to the head of malleus.

Posterior incudal ligament connects the short crus of incus to the posterior wall of incudal fossa.

The annular ligament at the base of stapes encircles it along margin of oval window.

Tensor tympani muscle is in an osseous compartment above the Eustachian tube. It takes a sharp bend around the processus cochleaformis and is inserted over the neck of malleus.

Stapedius muscle arises hollow cavity from interior of pyramidal eminence and its insertion is at neck of stapes.

FACIAL NERVE:

Facial nerve emerges from the brainstem by a sensory and a motor root; leaving the brain stem at pons in inferior aspect medial to VIIIth nerve. Intracranial segment is 23-25 mm long.

The internal auditory canal segment is 7-8mm and lies above the cochlear nerve. The labyrinthine segment is 3-4mm and passes forward and laterally in its bony canal (fallopian canal). Laterally it angulates forward perpendicular to the petrous till geniculate ganglion where its direction reverses. This is the first knee or genu. **(12)**

The tympanic segment is 12mm long and passes posteriorly and laterally over the medial wall in middle ear. It lies below bulge of the lateral semicircular canal.

At level of sinus tympani facial nerve assumes a vertical position & forms second genu. It runs along posterior wall & exits through stylomastoid foramen. This mastoid segment is 15-20mm in length.

The three important branches are -

Greater superficial petrosal nerve

The nerve to the stapedius

The chorda tympani

THE INNER EAR:

The bony labyrinth consists of cochlea, semicircular canals & vestibule. Vestibule is an ovoid perilymphatic space, 4mm in diameter, opening into cochlea anteriorly and semicircular canals posteriorly .

Vestibule has Two Openings.

- Oval window - for communication with the foot plate of stapes.
- Vestibular aqueduct - bony canal which extends posteriorly from vestibule medial wall to the posterior surface of the petrous pyramid. Aqueduct is inverted 'J' shaped. The proximal 'isthmus' arches medial to the crus and measures 03mm in diameter. Outer aperture measures 2-6mm in diameter.

SEMICIRCULAR CANALS:

There are three canals communicating with the vestibule. Each canal makes $2/3^{rd}$ circle. All are enlarged on anterior aspect to form the ampulla. The non-ampullary ends of the superior and posterior canals join forms common crus.

Arcuate eminence is a ridge present on the anterior surface of petrous part which is a part of superior semicircular canal. The lateral semicircular canal projects as ridge at the medial attic wall.

Superior & posterior canals are aligned in a vertical perpendicular orientation. Superior semicircular canal is placed at an angle of 45^0 to the midsagittal

plane antero laterally and is directed postero- laterally at a corresponding angle. The posterior semicircular canal is similarly placed with the angle directed postero-laterally.

The lateral semicircular canal does not occupy a horizontal plane and for this reason the older terminology has been discarded. Its anterior limb lies in a plane higher than that of posterior limb, making at angle of 30^0 with the horizontal. In the erect position therefore the neck would have to be fixed about 30^0 for the lateral semicircular canal to be 'horizontal'.

COCHLEA: [13]

Cochlea has central conical axis - modiolus and bony canal wound spirally around it for 2 ½ turns[7]. The 1st turn bulges along the medial wall to form the promontory.

The cochlear aqueduct is a well corticated notch medial to the pars nervous and inferior to porus acousticus. It serves as a potential communication between the sub arachnoid space and inner ear perilymph.

TABLE 1 - Best projection for imaging anatomy of temporal bone

S.No	Structure	Axial	Coronal	Both
1	Carotid canal			+
2	Jugular foramen	+		
3	Cochlear aqueduct			+
4	Internal auditory meatus			+
5	Inner ear			
	Cochlea and vestibule			+
	Lateral semicircular canal			+
	Semicircular canals		+	
6	Facial nerves			
	Labyrinthine and tympanic segment	+		
	Genu			+
	Mastoid segment		+	
7	Prussak's space attic		+	
8	Scutum		+	
9	Aditus, antrum and central mastoid tract	+	+	
10	Tegmen tympani		+	

11	Tensor tympani muscle	+		
12	Sigmoid sinus groove	+		
13	Tensor tympani tendon			+
14	Fossa incudus	+		
15	Round window		+	
16	Facial recess	+		
17	Pyramidal eminence	+		
18	Sinus tympani	+		
19	Lateral malleolar ligament		+	
20	Superior malleolar ligament		+	
21	Anterior malleolar ligament	+		
22	Patterns of pneumatization	+		
23	Tympanic membrane		+	
24	Anterior epitympanic recess	+		
25	Posticulus		+	
26	Subiculum		+	
27	Stapes foot plate			+
28	Stapes super structure	+		
29	Incus lenticular process	+		
30	Incus long process		+	
31	Incus body	+		
32	Malleus head			+
33	Malleus manubrium		+	
34	Malleus neck			+
35	Malleus lateral (short) process		+	
36	Malleoincudal articulation	+		
37	Incudo stapedial articulation	+		
38	Stapediovestibular region	+		

NORMAL HRCT ANATOMY OF TEMPORAL BONE

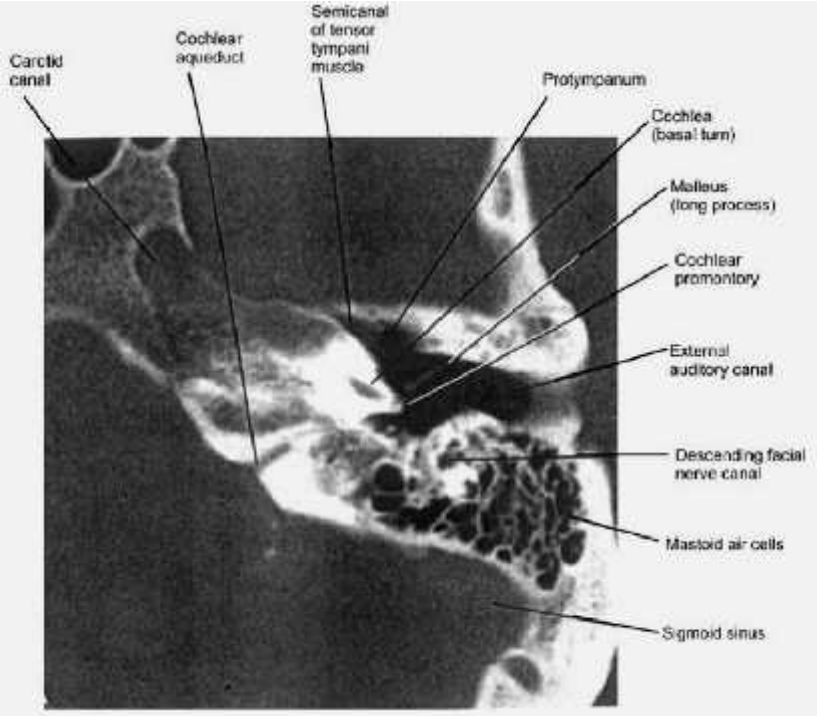


Figure 1: Axial View, Normal Anatomy

Axial CT section. This is the most inferior CT axial sections. The external auditor canal and long process of malleus are seen. The tympanic membrane is too thin to be visible. The cochlear promontory and cochlear basal turn are visible. Thin, linear semicanal for tensor tympani muscle is seen interposed between the protympanum and the carotid canal. The cochlear duct is seen arching from posterior fossa towards cochlear basal turn. The descending facial nerve canal, sigmoid sinus, and the mastoid air cells are shown.

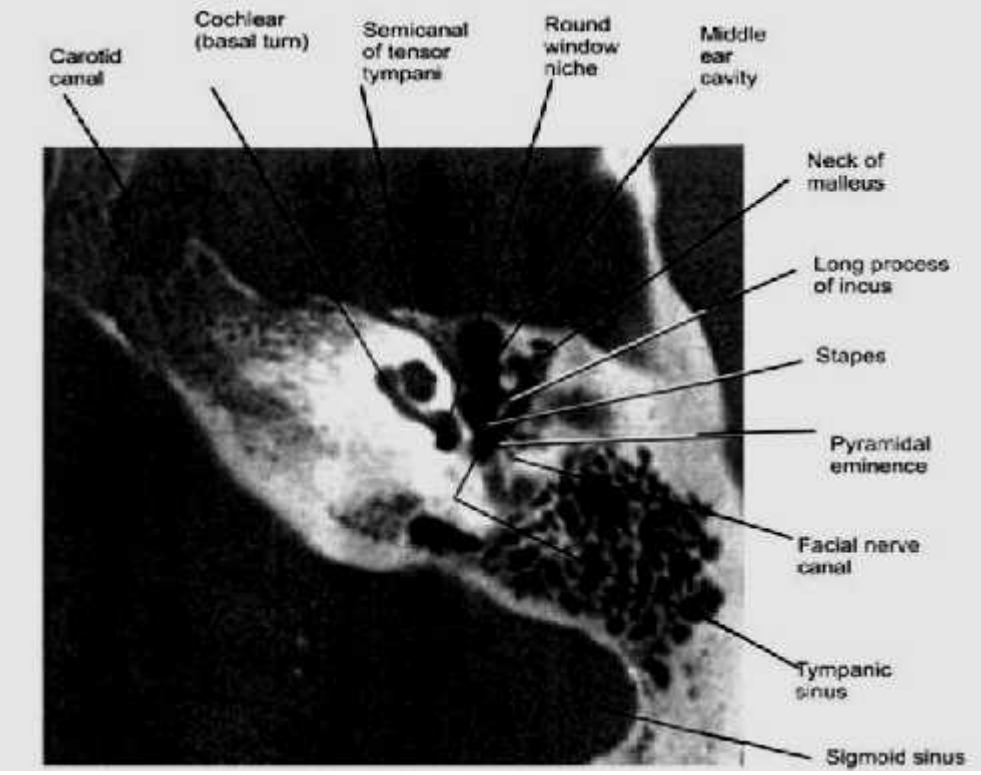


Figure 2: Axial view, Normal Anatomy

Axial CT is centered at neck of malleus in epitympanic space. The adjacent long process of malleus & stapes, are both visible posteriorly. The semicanal of tensor tympani muscle is seen as a linear lucency just lateral to the cochlea. The ligaments are not visible. The three turns of the cochlea are visible as well as the air space related to round window niche. Desending facial nerve canal is seen posteriorly of tympanic sinus & anterior to air cells of mastoid. Other labeled structures include the petrous apex, carotid canal, and the sigmoid sinus.

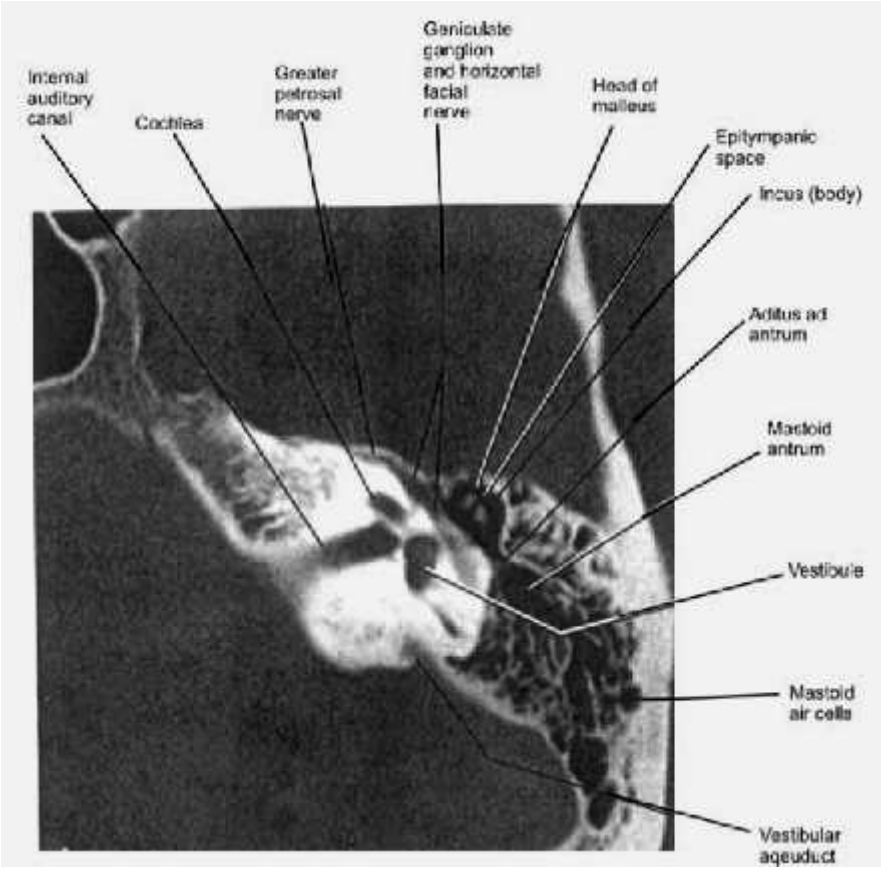


Figure 3: Axial view, Normal Anatomy

Axial CT image centered over facial nerve canal. Geniculate ganglion & horizontal portions of facial nerve canal are seen continuous with the internal auditory canal. Superior portion of cochlea and vestibule are visible. Opening of the endolymphatic sac & vestibular aqueduct is seen like thin slit along the posterior margin just medial to mastoid air cells. The epitympanic space holds the head of the malleus and the body of the incus and continues posteriorly with aditus and antrum and the mastoid antrum.

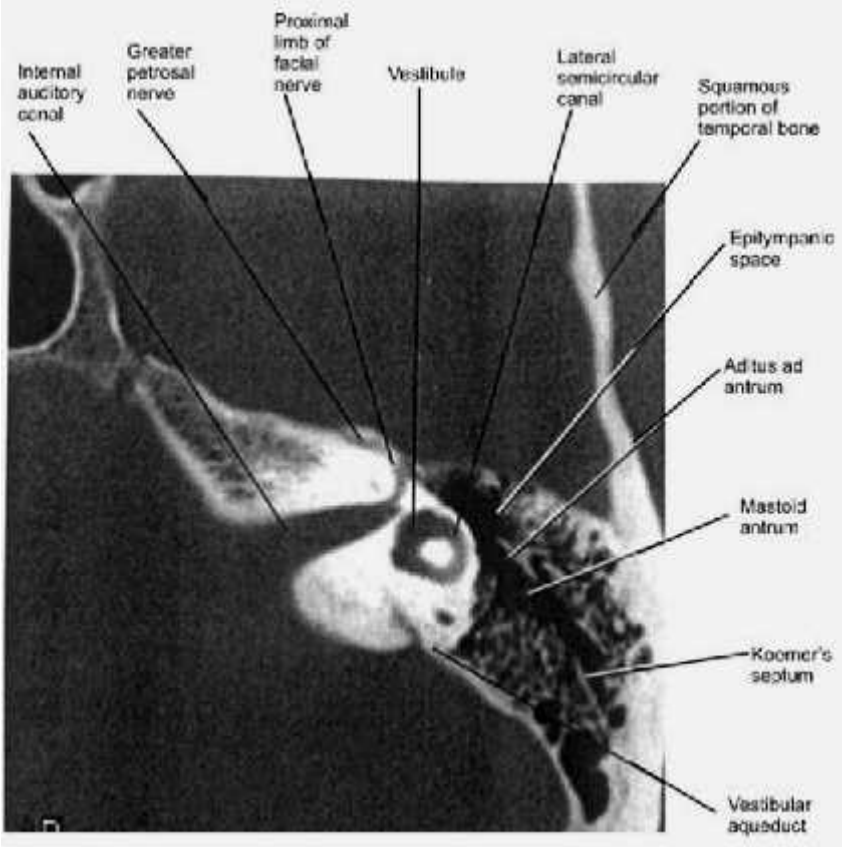


Figure 4: Axial view

Axial CT section of complete internal auditory canal, ending at proximal limb of geniculate segment of facial nerve canal anteriorly. The vestibule and complete lateral semicircular canal are visible. Posterior semicircular canal are visible. The posterior semicircular canal and vestibular aqueduct are seen posteriorly. Portion of the epitympanic space, the aditus ad antrum, and the mastoid antrum are seen in continuity. Korner's septum is also visible. The squamous part of temporal bone is seen anterior to mastoid & petrous segments.

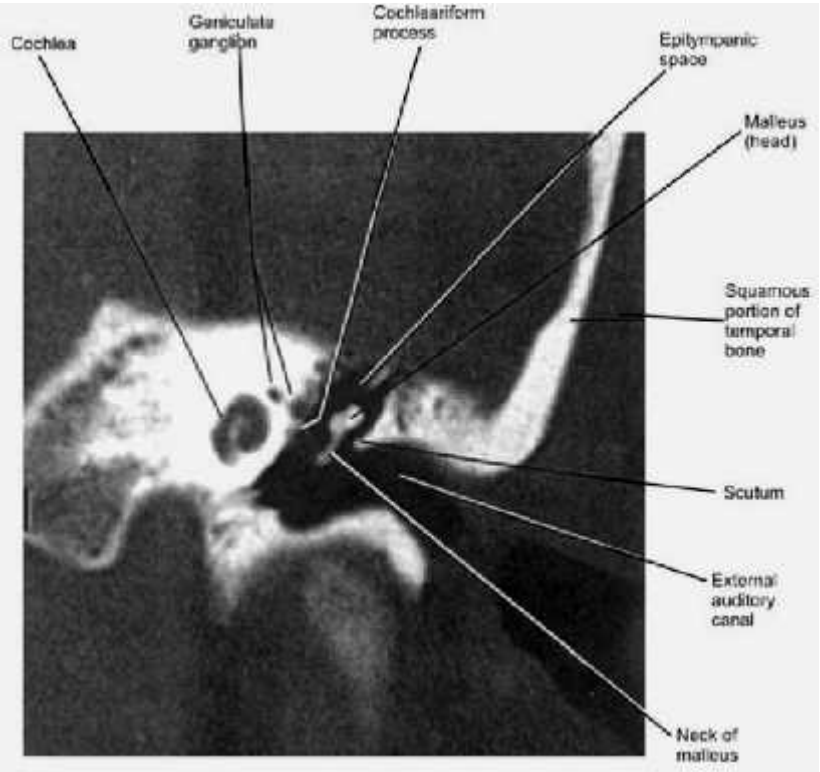


Figure 5: Axial view

Coronal CT section. This is the most anterior CT section of left side. Scutum forming medial margin of external auditory canal and adjacently head & long process of malleus are seen. The epitympanic space supports the ossicles. The external auditory canal appears continuous with the middle ear. Tympanic membrane is frequently not visible when normal. Abnormal thick tympanic membranes are easier to image. There small canals are just medial and superior to the anterior cochlea. These include the proximal limb of the geniculate ganglion, distal limb of the geniculate ganglion, and the semicanal of the tensor tympani muscle.

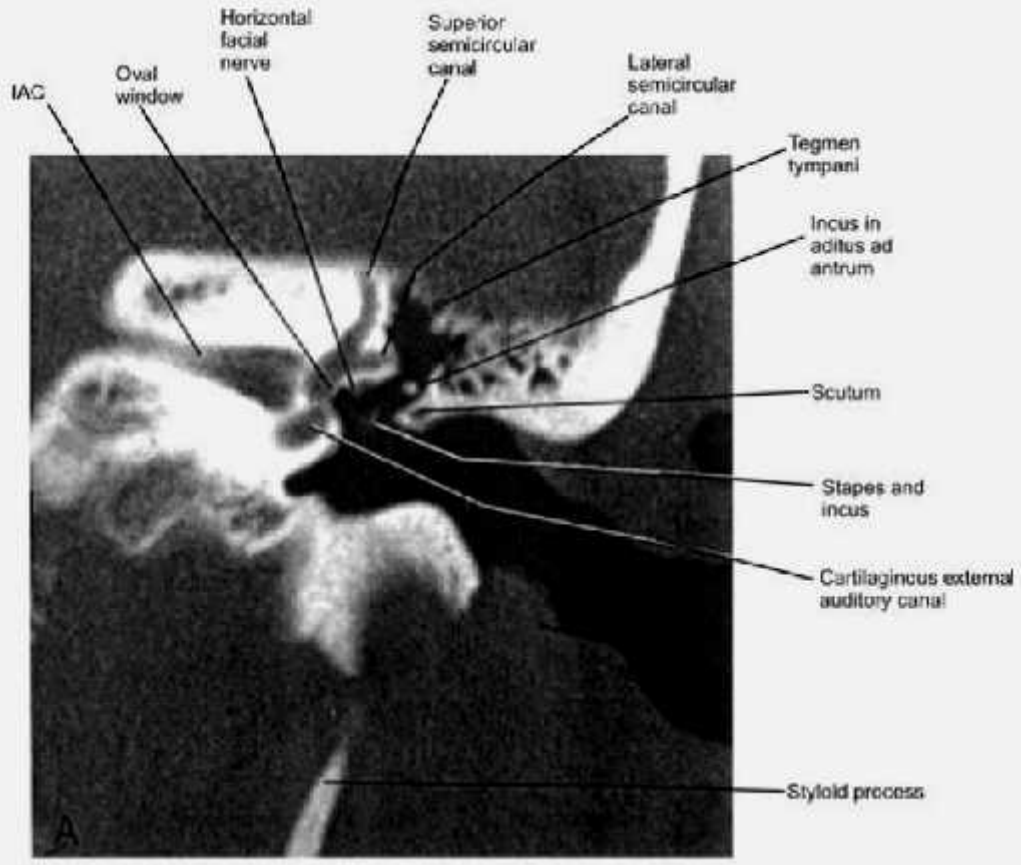


Figure 6: Coronal view, Normal Anatomy

Coronal CT section centered at oval window. The adjacent stapes & incudostapedial joint are visible as an L shaped structure overlying the oval window. The facial nerve (horizontal portion) is below the lateral semicircular canal. Between the scutum and the lateral semicircular canal lies the short process of the incus. The internal, middle ear, external auditory canal (EAC), stapes and cochlear basal turn are shown. Only the cartilaginous segment of the EAC is seen as a soft tissue component.

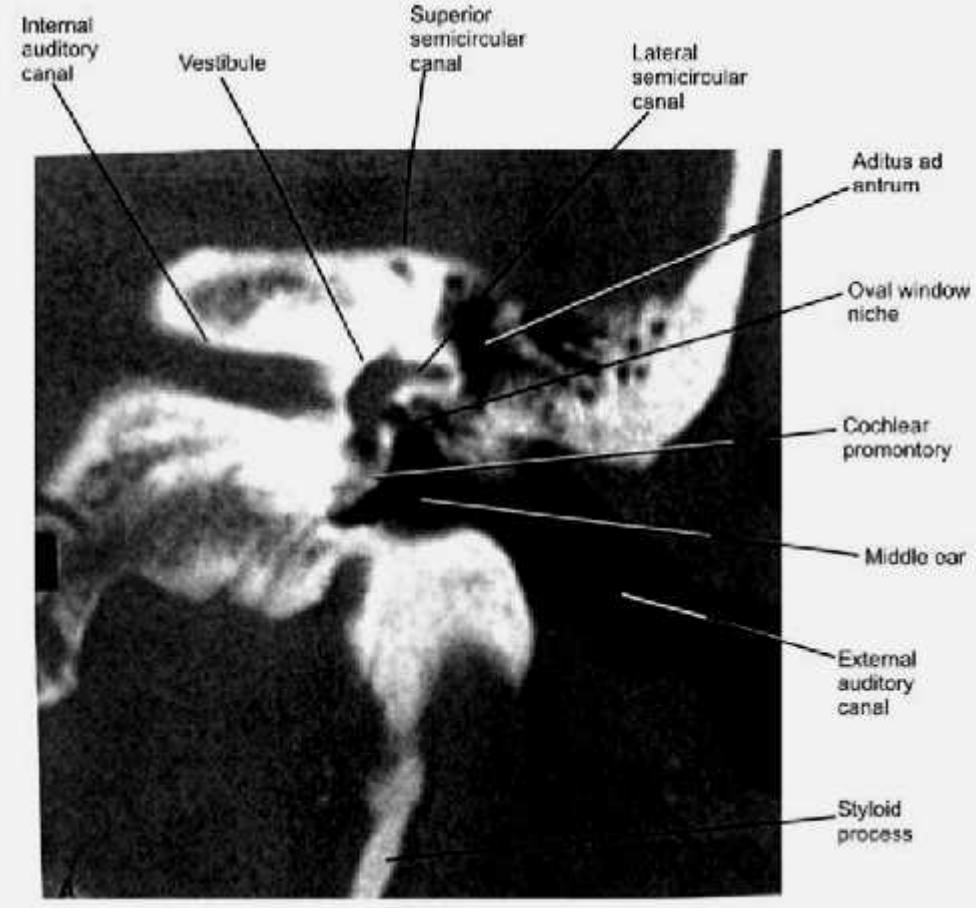


Figure 7: Coronal view, Normal Anatomy

Coronal CT section made through vestibule & posterior portion of oval window. The cochlear promontory and cochlear basal turn are seen. The aditus ad antrum lies lateral to lateral semi circular canal. A short portion of superior semicircular canal is visible. Middle ear, internal ear, styloid process, and external auditory canal are shown.

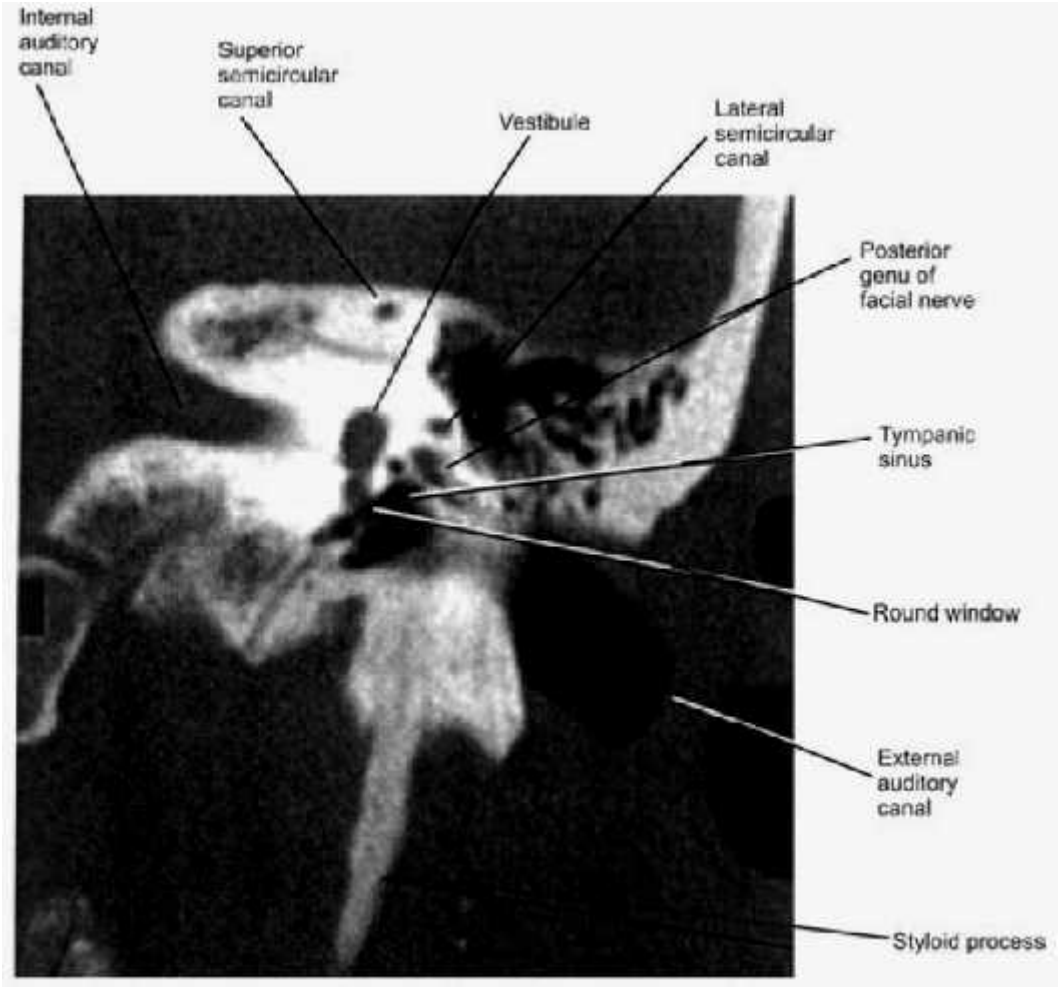


Figure 8: Coronal view, Normal anatomy

Posterior coronal section of CT of temporal bone centered at round window and the tympanic sinus. The air spaces extend all the way to the otic capsule membrane surfaces. The adjacent posterior genu of the facial nerve and vestibule are seen. Other depicted structures include the posterior internal auditory canal, superior semicircular canal, lateral semicircular canal, styloid process, and external auditory canal.

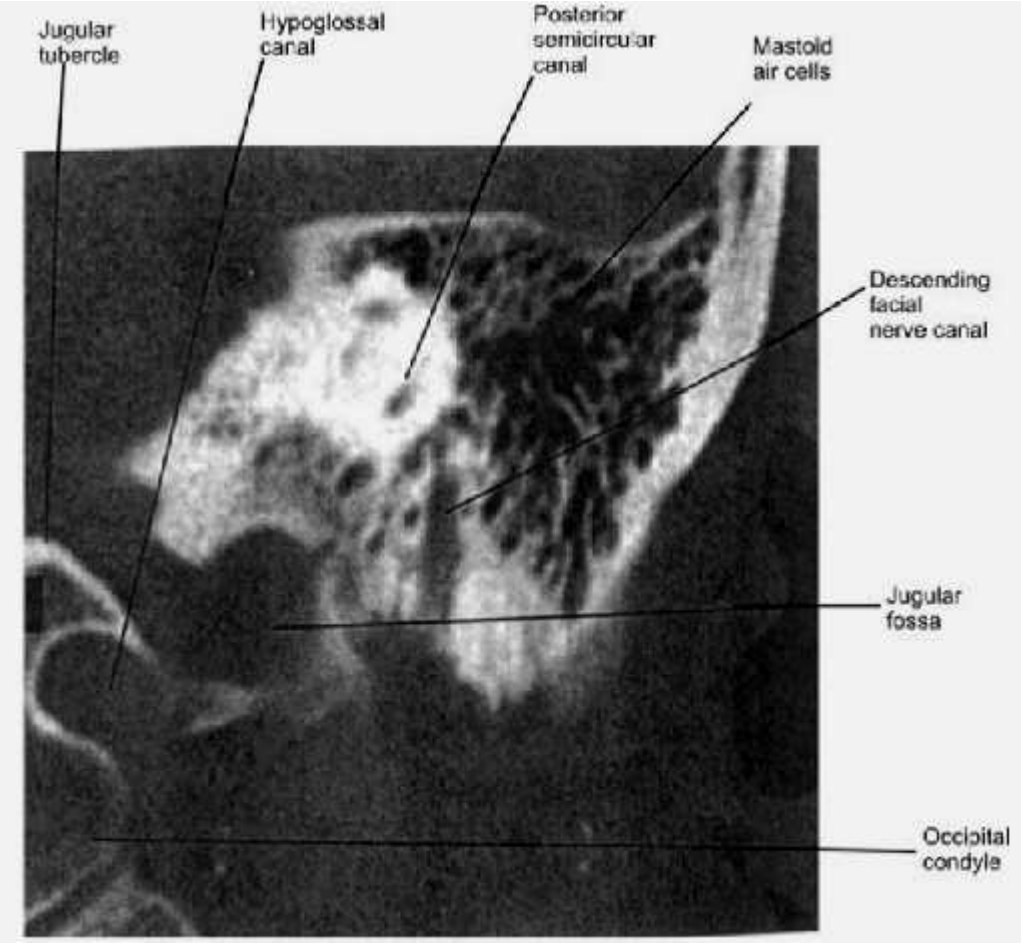


Figure 9: Coronal CT Section

This is the most posterior section of the temporal bone. Descending facial nerve canal lies between mastoid air cells & occipital condyle all appear on medial aspect of jugular fossa. Short segments of posterior semicircular canal are seen.

REVIEW OF LITERATURE OF THE PATHOLOGIES INVOLVING THE TEMPORAL BONE WOULD BE COVERED UNDER THE FOLLOWING HEADINGS:

- Congenital anomalies
- Trauma and fractures.
- Inflammatory diseases
- Neoplasms

CONGENITAL EAR ANOMALIES:

50% of the congenital pathologies affect the ear in the ear, neck & throat region according to Sylva Bartel-Friedrich & Cornelia Wulke. The incidence is approx. 1 in 3800 newborns. [16]

According to descriptive-observational study carried on 4800 births carried out by Sedighah Akhavan Karbasi et al in 2007, incidence of congenital ear malformations was about 1.81 %.[14]

ANOMALIES OF OUTER EAR:

Outer ear is made up of two parts viz auricle & external auditory canal. Auricle develops from 1st and 2nd brachial arch.

Congenital anomalies of external ear can be in the form of size, shape, position, pattern of pinna. Ear tags, sinuses and pits are also commonly found. Complete anotia with or without hypoplasia or atresia of externa auditory canal may be seen.

The external auditory canal develops from 1st brachial cleft. The tympanic cavity develops from 1st pharyngeal pouch. The meeting of these two results is formation of primitive tympanic membrane. (16)

Partial or complete failure of this canalization results in stenosis or atresia. Malformations of the outer ear are referred to as congenial aural dysplasias. (17)

EAR TAGS (16) :

Ear tags are nothing but abnormal growth of mandibular tissue along the groove of 1st branchial arch. Usually after this groove closes the hyoidal mandibular margin is shifted towards cheek. Therefore ear tags are seen between angle of the mouth & ear. Large ear tags are also called as 'cheel ears' or supernumerary pinna. In such patients special examination should be done to look for additional malformations of the middle & inner ear. **(16)**

EAR PITS & CYSTS (16)

They may be sometimes multiple or bilateral. Pits & cysts correspond to retentions of epithelium. They are lined by squamous epithelium. They may be found around crus helicis and preauricular regions.Rarely they may also be seen in upper neck regions.

Types:

1. **Type I** - "duplication" of the external auditory canal having lining of normal skin. They are usually post auricular as compared to pre-auricular and are parallel to external auditory canal. They have blind ending on lateral or on upper aspect of facial nerve.

2. **Type II** They are true external auditory canal doublings. They contain cartilage & lining is of normal skin. They have blind ending in region of transition of bony & cartilaginous part of external ear. Some of them can even open in post auricular region.

PINNA MALFORMATIONS [16,18]

Pinna malformations occur because of involvement of hillocks. Involvement may be single or multiple of them. Weerda has classified these malformations.[16]

• Dysplasia [18]

- Grade I (mild)– In this type mostly all the structures of pinna are normally recognizable. Only sometimes the use of skin or cartilage may be done for the purpose of reconstruction.
- Grade II(Moderate) - In this type only few structures of pinna are recognizable. It more frequently requires the use of skin or cartilage for the purpose of partial reconstruction.[18]
- Grade III (Severe)- In this type none of the structures of pinna are recognizable. It requires the use of skin or large amount of cartilage for the purpose of reconstruction. [18]

TABLE NO. 2 Weerda classification of various dysplasia grades with subgroups [16]

Grade of dysplasia	Subgroups
I: First-degree malformations	• Prominent ears • Macrotia • Cryptotia (pocket ear) • Coloboma (transverse cleft) • Scaphoid ear • Stahl ear • Satyr ear • Slight deformities (distinct Darwin tuberculum, absent crus of helix, deformities of tragus and antitragus • Deformities of lobe (fixed lobe, hyperplasia or hypoplasia or aplasia or cleft of lobe) • Cup ear deformity type I • Cup ear deformity type IIa • Cup ear deformity type IIb
II: Microtia grade II, second-degree malformations	• Cup ear deformity type III • Mini ear („concha-type microtia") ➤ Hypoplasia of upper pinna ➤ Hypoplasia of middle pinna ➤ Hypoplasia (or aplasia) of lower pinna often combined with dystopia and EAC stenosis, rarely with EAC atresia, ear drum malformations possible
III: Microtia grade III with anotia, third-degree malformations	• Unilateral microtia grade III („lobule-type microtia") • Bilateral microtia grade III • Anotia often with dystopia and EAC atresia

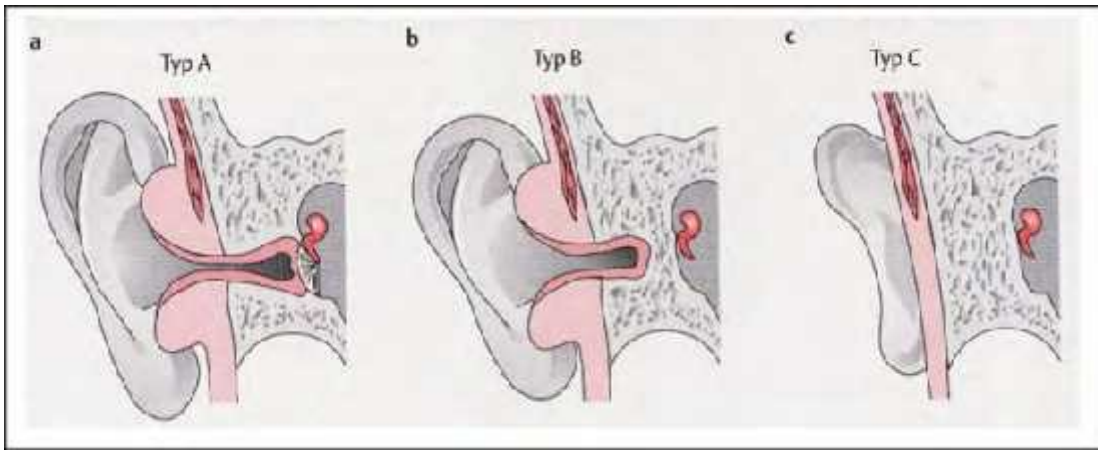


Figure 10 : EAC malformation according to Weerda. [16]

EAC MALFORMATIONS

Weerda classifies malformations of the external auditory canal into: [16]

- **Type A** - marked stenosis/narrowing of the external auditory canal. Overlying skin is intact.
- **Type B** –An atretic plate is present at medial aspect with incompletely developed external auditory canal.
- **Type C** Complete atresia of external auditory canal with bony involvement.

EAC & MIDDLE EAR MALFORMATIONS [16]

The closely interrelated development of the EAC and the middle ear led to the classification of the combined malformation termed atresia auris congenita according to Altmann.

HRCT findings:

- 1st degree malformation: Mild deformity of external auditory canal with normal/slightly hypoplastic tympanic cavity, deformed ossicles, and a well-aerated mastoid. [16]
- 2nd degree malformation:Intermediate deformities including blind ending/absent external auditory canal with narrow tympanic cavity, deformations & fixation of the ossicles with hypo-pneumatization of mastoid. [16]
- 3rd degree malformation: Severe deformities where external auditory canal is absent, hypoplasia of middle ear & severe deformity of ossicles. They may also be hypo-pnematisation. [16]

TYMPANIC RING APLASIA: [16]

HRCT findings: Completely absent EAC & tympanic membrane, with presence of a bony atretic plate. There will be fusion of neck of malleus to the plate. The facial nerve is anteriorly displaced. The tempero-mandibular joint is positioned posteriorly. [16]

MIDDLE EAR MALFORMATIONS [16]

Kösling describes following degrees of malformations

- Mild - normal configuration of the tympanic cavity along with dysplasia of ossicles.
- Moderate - hypoplastic tympanic cavity with aplastic/rudimentary ossicles.
- Severe – aplastic/cleft like tympanic cavity.

The involvement of *malleus is less* in isolated malformations of middle ear. Commonly encountered findings on HRCT are deformed or hypoplastic of the head/manubrium of malleus, abnormal fixity in epitympanic recess & abnormal malleoloincudal joint. Malleus may be even absent. [16]

Majority of malformations of incus are due to absent/hypoplastic long process with separated incudostapedial joint. Sometimes there may be variation in the long process & infrequently complete aplsia of incus may also be present. There may be bony fusion of incus & malleus. [16]

Stapes malformations are commonly seen as isolated “minor” deformities.Most commonly there is combined deformation of suprastructure of stapes and incus. [16]

CLASSIFICATION OF CONGENITAL INNER EAR ABNORMALITIES [20,21,22,23]

Jackler et al proposed the most commonly used classification of inner ear abnormalities, which is based on a linear developmental model towards normal anatomy and the most likely time at which developmental arrest occurs during embryogenesis. Sennaroglu et al further modified this classification. However, this classification has been challenged by other authors. [20,21,22,23]

MICHEL APLASIA: [16,20,24,25]

Michel aplasia is completely absent inner ear structures and is rare congenital pathology. Its etiology is due to arrest in development of otic placode in first trimester gestational period. Michel aplasia maybe associated with petrous bone aplasia, narrowed or aplastic internal auditory canal, hypoplastic middle ear & mastoid as well as variation of the jugular bulb. [20,21,22,23]

HRCT findings: Absent inner ear structures, may be associated with petrous bone aplasia, narrowed or aplastic internal auditory canal, hypoplastic middle ear & mastoid as well as variation of the jugular bulb.

COCHLEAR APLASIA [20,25]

Developmental arrest in 3rd week of gestation results in cochlear aplasia.

HRCT findings: There may be either dilation or hypoplasia of vestibule &

semicircular canals. Sometimes they also may be normal [20,25]

COCHLEAR HYPOPLASIA [20]

Failure of development at 6th gestational week causes hypoplasia of cochlea. [20]

HRCT findings: The differentiation of cochlea and vestibule is possible but cochlear size is less than normal.

COMMON CAVITY: [20]

In this type of malformation, arrest of growth takes place at 4th gestational week and it represents a common cavity with undifferentiated vestibule & cochlea. [20]

HRCT findings: One common cavity with undifferentiated vestibule & cochlea.

INCOMPLETE PARTITION TYPE 1 [20,23]

It is known as a cystic cochlea-vestibular type of malformation in which the cochlea is devoid of bony modiolus which results in empty cystic cochlea. It may be coexisting with cystic vestibule with dilatation due to arrest in development at 5th gestational week. [20,23]

INCOMPLETE PARTITION TYPE 2 [20,23]

In this group, there are one & half turns in cochlea. There is non-differentiation of apex & middle cochlea to form cystic apex. There might be enlargement of vestibular aqueduct. It occurs due to arrest in development at 7th gestational week. Third deformity is also known as mondini deformity. [20,23]

SEMICIRCULAR CANAL ABNORMALITIES

The semicircular canals can either be absent, hypoplastic or enlarged. Short lateral semicircular canal with confluent vestibule is most commonly seen. The semicircular canals develop at 6 to 8 gestational weeks & are completely developed by 22nd gestational weeks.

Axial and coronal images show both lateral semicircular canals are undifferentiated from the vestibules.

HRCT findings: Semicircular canals appear hypoplastic or absent on axial and coronal images.

ENLARGED VESTIBULAR AQUEDUCT

It is commonly defined as having width larger than 1.5 mm. This measurement is taken at centre of external aperture & common crus. Valvassori & Clemis first described this pathology as most common inner ear abnormality associated with sensorineural hearing loss.

HRCT findings: Vestibular aqueduct enlarged in width, measuring greater than 1.5 mm.

TABLE 3. Malformations of cochlea – as described by Sennaroglu [24]

Cochlear Malformations	**Configuration**
Michel deformity (arrest: 3rd week)	Completely absent cochlea & vestibular structures; often aplastic internal carotid canal; absent vestibular aqueduct
Aplasia of cochlea (arrest: late 3rd week)	Absence of cochlea, normal/dilated/hypoplastic vestibule and semicircular canal system; often enlarged internal auditory canal; mostly normal vestibular aqueduct.
Common cavity (arrest: 4th week)	Cochlea and vestibule build a common space without internal architecture, normal, deformed or absent semicircular canal system; internal auditory canal more enlarged than narrow; mostly normal vestibular aqueduct.
Incomplete partitionType 1 (cystic cochleovestibular malformation) (arrest; 5th week)	Cystically enlarged cochlea without internal architecture; dilated vestibule,mostly enlarged internal auditory canal; absent/ dilated / normal semicircular canal system Normal vestibular aqueduct
Cochlear hypoplasia (Arrest: 6th week)	Distinctly recognizable separation of cochlear and vestibular structures; small cochlear bud, absent or hypoplastic vestibule and semicircular canal system; narrow or normal internal auditory canal; normal internal vestibular aqueduct.
Incomplete Partition type II (Mondini deformity) (Arrest ; 7th week	Cochlea with 1 ½ turns, cystically dilated middle and apical turn(Cystic apex), nearly normal size cochlea: slightly dilated vestibule; normal semicircular canal; enlarged vestibular aqueduct.
Normal (No arrest)	Regular cochlear and vestibular structures and normal IAC & vestibular aqueduct.

INTERNAL AUDITORY CANAL & COCHLEAR NERVE ANOMALIES [24]

- Normal measurement of IAC is 2-8 mm. If diameter is less than 2 mm it is considered as stenotic.

  HRCT findings The IAC less than 2 mm and stenotic.

- The aperture of cochlea is a canal near internal auditory canal through which the cochlear nerve passes . If the location of cochlear aperture is replaced by bone, the condition is described as hypoplasia of the bony canal of the cochlear nerve [24]

  HRCT findings: Absent cochlear aperture or site filled with bone density tissue.

Cochlear nerve anomalies are divided into three types:

- Type 1:- Anomaly of cochlear nerve, stenosis of internal auditory canal with absence of 8th nerve.
- Type2:-Common vestibulocochlear nerve with hypoplastic/aplastic cochlear branch.
  - Type 2A malformation in association with other inner ear abnormalities,
  - Type 2B malformation when occurs as isolated type [24]

INFLAMMATORY CONDITIONS

Infections of the temporal bone involve following regions

- External ear

- Middle ear & mastoid
- Inner ear
- Petrous apex

EXTERNAL EAR

NECROTIZING EXTERNAL OTITIS [26] :

It is an infection which is commonly seen in immuno-compromised people and elderly people with diabetes mellitus. It involves temporal & its adjacent bones and is rare complication of external otitis. HRCT helps in determining the extent, location and involvement of the lesion. It usually affects the temporal bone first followed by mastoid & petrous apex. Thickening of the mucosa of EAC & auricle is noted with enhancement on post contrast study. It may also show osteomyelitic type of appearance of surrounding bones. There may be infiltration into temporo-mandibular fat pad. [26]

EAC MEDIAL CANAL FIBROSIS [26]

It is an acquired atresia of meatus which is characterized by fibrous tissue formation on the medial aspect of EAC.

HRCT findings – it appears as soft tissue density lesion seen throughout the length of bony EAC, against the tympanic membrane, with no bony erosions. [26]

EAR WAX (CERUMEN): [26]

Its also called as cerumen. It is yellow waxy substance which is secreted in the ear canal. It has a protective function against from insects,fungi,bacteria & water. Its impaction can cause blockage of EAC or even hinder in hearing.

HRCT findings- Hypodense lesion filling the EAC. Fat attenuation within the lesion and the presence of a rim of air around the lesion confirm the diagnosis. [26]

EXTERNAL AUDITORY CANAL CHOLESTEATOMA: [26]

Acquired type of cholesteatoma is an inflamatory etiology which is seen mostly in middle ear. Its incicence in external ear is rare & is 1 in 1000 new patients in ENT clinics. They typically present with ear discharge and pain due to invaded skin tissue in external ear.

HRCT findings - appears as fat density lesion with bony erosions and intramural bony fragments. Usually inferior and/or posterior walls are involved. [26]

MIDDLE EAR

EUSTACHIAN TUBE DYSFUNCTION [27]

Dysfunction of Eustachian tube is excessive secretion of mucoid and serous nature in middle ear cavity. There may be no associated inflammatory changes. It is commonly seen in children but may also be present in adults. Predisposing factors include adenoid hypertrophy or neoplasm of nasopharynx.

They may present with as bulge in tympanic membrane with absent inflammatory signs. Later fluid secretions may become thick called 'glue ear'.

HRCT findings – Homogenous opacification of cavity of middle ear & mastoid air cells with no evidence of erosion of ossicles. [27]

Acute middle ear infection (acute otitis media, acute otomastoiditis):

They present as pain in ear, ear discharge with hearing loss of conductive type. Otoscopic examination shows bulging of tympanic membrane [30,31] . Differentiation of secretory otitis & acute otitis media is important. [28] .It may also show resorption of matoid septate with disease progression [32] .

HRCT findings – The most important finding on HRCT is erosive changes in mastoid septae & cortex. Later complications like abscess &sinus thrombosis may be seen. [27]

CHRONIC MIDDLE EAR INFECTION/INFLAMMATION

Chronic middle ear infection generally presents with hearing loss of conductive type, vertigo,pain & otorrhea. It occurs when there is persistence of inflammatory etiology

beyong 6 weeks perforating through tympanic membrane. It may even extend intracranially [33,34].

Best diagnostic modality is HRCT. Ear ossicles and middle ear cavities should be well examined. It may even proceed to cholesteatoma [34,35] .

CHRONIC OTITIS MEDIA WITH ACQUIRED CHOLESTEATOMA

It literally means 'skin in the wrong place' . There is growth of squamous epithelium noted in the middle ear and mastoid [33].They usually arise secondary to perforation of the tympanic membranes [35,36] . Pars flaccid cholesteatoma classically arise from the pars flaccid. Erosion of scutum and ossicles are common. Pars tensa cholesteatoma usually occurs in the posterior quarter of tympanic membrane.

The patients present with ear discharge which is often painless and sometimes hearing loss or vertigo. Its management is mainly surgical. [35,36,37,38] .

HRCT is the preferable modality to describe the extent of both the types of cholesteatoma. On HRCT we see soft tissue density I tyampanic cavity involving epitympanum, mesotympanum, hypotympanum and Prussack's space. Associated erosion of ossicles with displacement is seen.

HRCT features of cholesteatoma:

1. Non dependent, soft tissue density mass
2. Hypopneumatization of mastoid
3. Specific site localization
    - Prussack's space
    - Posterior recesses
    - Holotympanic involvement

4. Adjoining bony erosions, chiefly
    - Scutum
    - Ossicles
    - Involvement of facial nerve canal
    - Tegmen
    - Lateral semi-circular canal

PETROUS APEX [26]

It is lined by air cells, therefore they are easily affected when there is concurrent infection in the middle ear cavity & a spectrum of problems including effusion, mucocele, and cholesterol granuloma.

ACUTE PETROSITIS:

Known more commonly as Gradenigo's syndrome comprising of lateral rectus palsy, orbital pain & petrous apictis.

HRCT findings: soft tissue debris within petrous apex air cells, lysis of bony septae and destruction of adjacent bony cortex may also occur. Occasionally it may present only with fluid. [26]

CHOLESTEROL GRANULOMA:

Expansile lesion with erosion in petrous apex.

HRCT findings: heterogenous density lesion in petrous apex. [26]

EFFUSION:

HRCT is necessary for definitive evaluation of the air cells to exclude expansion and destruction.

HRCT findings: Fluid density within the air cells of petrous apex[(26)].

MUCOCELE:

HRCT findings: This cause expansion and remodeling of the petrous apex. It is rare.

Other infectious process that could lead to secondary petrous temporal bone involvement are cholesteatomas and necrotising external otitis. [(26)]

TUMOURS OF THE TEMPORAL BONE

To assess a tumor in the temporal bone region, it is helpful to localize the lesion to one of the following

(a) IAC/CPA

(b) Middle ear

(c) EAC and mastoid

(d) Petrous apex.

(e) Facial nerve

VESTIBULAR SCHWANNOMA

The most common tumor in the IAC/CPA is the vestibular schwannoma, which accounts for 60%–90% of all tumors in this region [(40,41,42).] It is also thought to be the most common intracranial nerve sheath tumor [(43)] . Incidence is highest in the 5th to 7th decades of life [(44)] , although they commonly present in the first 2 decades in thesetting of neurofibromatosis type II, the enphakomatosis associated with bilateral vestibular schwannomas, multiple schwannomas of other cranial nerve origin,

meningiomas and ependymomas in the brain and spine [45] . Common symptoms are hearing loss of sensorineural type, tinnitis, dysequilibrium, &/ or decreased speech discrimination, secondary to pressure by the tumor on the cochlear and vestibular divisions of cranial nerve VIII [46] ; facial nerve manifestations are relatively uncommon [46,47] .

On HRCT images, most vestibular schwannomas are isoattenuating with the cerebellum and are difficult to delineate without contrast material enhancement [41] . However, if the tumor is large and causes expansion of the porusacousticus, this may be readily seen on CT bone window images, with the porus on the affected side asymmetrically wider. Calcification and haemorrhage are rare unless the tumor has been treated [48] . Enhancement is usually avid and homogeneous [49] , but this may be difficult to detect on CT images, especially if the tumor is small.

Secondary changes such as widening of ipsilateral cerebello pontine angle and quadrigeminal cistern, narrowing of contralateral cistern and displacement and compression of 4^{th} ventricle are noted. In addition bony changes such as, difference in canal height of more than 2mm, shortening of posterior wall of canal of more than 3mm, and presence of focal erosion are usually present.

	Acoustic Schwannoma
Location	Centered to IAC
Bone changes	Most enlarging IAC
Shape	Spherical or ovoid, occasionally lobulated, acute bone tumor angle
Density	Most isodense, a few slightly hypodense or hyperdense
CT enhancement	Moderate to marked, often with in homogenous enhancement
Variations	Cytsic changes may be seen in cystic variety of acoustic schwannoma in which only the solid part usually shows enhancement; necrosis may be seen; occasionally blood components, calcifications may be seen in few cases.

TABLE NO 4 – TABLE DESCRIBING CHARACTERISTICS FEATURES OF ACOUSTIC SCHWANNOMA

MENINGIOMAS [(68)]

Meningiomas are common intracranial tumours. 5-10% of them are located at cerebellopontine angles. Enlargement of porus acousticus is more commonluy seen in acoustic neuroma but may also be seen The patients with CP angle meningioma

usually present with sensorineural hearing loss or facial nerve symptoms.Their removal surgically is done mainly to restore hearing. [47] Radiotherapy in cases of involvement of skull base also plays a role. [79]

On comparing with vestibular schwannomas, meningiomas are often eccentric to the porusacousticus,centered at the CPA; when they do extend into the IAC, they seldom expand the porus or the IAC.In the cerebellopointine angle it is a distant second to acoustic schwannoma in incidence. They arise from meningoepithelial arachnoid cells. Its peak incidence is 40 to 60 years, Female: Male - 2: 1 to 4: 1

HRCT: They are sharply circumscribed mass that abuts the dural surface and forms an obtuse angle. Majority of them are hyperdense to brain parenchyma and show strong and uniform enhancement and calcification is seen in 20 to 25% cases with few tumors showing cystic areas. [68]

	Meningioma
Location	Posterior petrous wall most eccentric to IAC
Bone changes	Occasional hyperostosis
Shape	Hemispherical, rarely plaque-like, may herniated, obtuse bone tumor angle
Density	Isodense or mostly slightly hyperdense, some calcified
CT enhancement	Marked and homogeneous
Variations	There may be no bony changes. Sometimes the meningioma may be centered anterior or posterior to the internal acoustic canal.

TABLE NO 5 – TABLE DESCRIBING CHARACTERISTICS FEATURES OF MENINGIOMA

EPIDERMOID

Congenital epidermoid cysts (congenital cholesteatomas) are the third most common mass in the CPA [41,42] ; they consist of stratified squamous epithelial linings surrounding desquamated keratin. They are not true neoplasms and are described in detail under the earlier section, “Inflammatory Lesions.”

On CT images, epidermoids are similar to CSF in attenuation; ifthey are large, smooth remodeling of the adjacent petrous bone may be observed secondary to long-term pressure erosion.

Well defined lucent appearing lobulated masses with attenuation similar to cerebrospinal fluid. Calcification uncommon, occasionally hyperdense due to hemorrhage, high protein content or iron containing pigment.

Age and gender: 20 to 60 years Male = Female

	Epidermoid tumor
Location	Anterolateral or posterolateral to brainstem
Bone changes	Occasional erosion
Shape	Variable with tendency to dumbbell into middle fossa or contralateral CPA
Density	Mostly about CSF density, rarely denser than brain, occasional peripheral calcification
CT enhancement	Non-enhancing
Variations	Calcification may be seen in 10-25% of cases. Sometimes may show minimal enhancement. Bony changes may not be seen sometimes even in chronic cases.

TABLE NO 6 – TABLE DESCRIBING CHARACTERISTICS FEATURES OF EPIDERMOID TUMOUR

ENDOLYMPHATIC SAC TUMOR

Endolymphatic sac tumor is a locally invasive papillary cystadenomatous tumor [50,51,52] . Most cases are sporadic, although a minority may be seen in the setting of Von Hippel Lindau syndrome (7%) [53]. This tumor causes bony destruction around the vestibular aqueduct in the retrolabyrinthine petrous bone.

HRCT Findings: the bone invaded by tumor has a moth-eaten, lytic appearance, with intratumoral bone spicules often seen. [51,52]

MIDDLE EAR

Whenever soft tissue density is noted in middle ear, a vascular structure must be excluded. This includes a persistent stapedial artery, a laterally placed oraberrant carotid artery, a carotid artery aneurysm, and an exposed dehiscent jugular bulb. True neoplasms include paraganglioma (most common); facial nerve lesions extending into the middle ear, such as schwannomas and geniculate region hemangiomas, choristomas,and perineural spread of tumor; meningiomas;adenomatous tumor of the mixedpattern type; and malignancies such as carcinomas and metastases (rare).

Most choristomas in the middle ear consist of salivary gland tissue [54,55] ,and they may be associated with incudo-stapedial or tympanic facial nerve abnormalities [54] .

GLOMUS TUMOURS: [59,69,70]

They are termed according to their location.

1) Glomus tympanicum - glomus formations on cochlear promontory.
2) Glomus jugular tympanicum - involving jugular foramen and middle ear.
3) Glomus vagale - involving nasopharyngeal carotid space

Occur predominantly in women(glomus jugular tumpanicum is 4 times more common in female)

GROWTH PATTERN OF GLOMUS TUMOURS:

These tumors are slow growing and locally infiltrating, growing along planes of least resistance in existing pathways in the temporal bone, and they rarely metastasize. The patients present with deafness, tinnitus and bloody ear discharge. These are highly vascular and therefore enhance avidly. Glomus tympanicum paragangliomas are found against the cochlear promontory and are usually small at presentation since they cause otologic symptoms early on .It is important to inspect the margins of the jugular foramen to exclude a glomus jugulotympanicum; localized lytic or permeative bone destruction is characteristic of a glomus tumor involving the jugular foramen

CT is used to diagnose and stage the disease.

At angiography, enlarged feeding arteries and rapidly draining veins may be seen.

OLDRING AND FISCH CLASSIFICATION [71,72,73]:

A Localized in cleft of middle ear

B Localized in tympanic mastoid

C. Involvement of infralabyrinthine compartments and extending to petrous apex

Cl Destruction of jugular bulb & foramen with limitedly involved vertical carotid canal

C2 Destruction of infra-labyrinthine part with involved vertical carotid canal

C3 Involvement of infra-labyrinthine part and petrous apex & involved horizontal carotid canal.

D1 < 2cm

D2 > 2cm

D3 Inoperable

Most patients present with otologicconcerns (conductive hearingloss, pulsatile tinnitis, or a retrotympanic mass) [56,57] .Paragangliomas are highly vascular and therefore enhance avidly. Glomus tympanicum paragangliomas are found against the cochlear promontory and are usually small at presentation since they cause otologic symptoms early on [58,59] . It is important to inspect the margins of the jugular foramen to exclude a glomus jugulotympanicum; localized lytic or permeative bone destruction is characteristic of a glomus tumor involving the jugular foramen, andis well seen on CT scans [60] .

MIDDLE EAR ADENOMA

Middle ear adenoma is mixed pattern adenomatous tumor [50] . This is a benign tumor that does not demonstrate bone invasion.

HRCT Findings : Opacity involving middle ear cavity, and this tumor may therefore be difficult to distinguish from otitis media [50] .

EAC AND MASTOID

Tumors in the EAC and mastoid region are often malignant, with squamous cell carcinoma being by far the most common [67] . Patients with EAC squamous cell carcinoma frequently have a long history of chronic ear infections. There is aggressive bone destruction, andthere may be invasion of surrounding soft tissues including intracranial, inframastoid, middle ear, parotid, carotid and temporomandibular joint involvement[61,62,63]. Other malignancies such as basal cell carcinoma, melanoma, lymphoma, myeloma, metastases, chondrosarcoma, and osteosarcoma occur much less frequently.

FIBROUS DYSPLASIA:

It is of two types viz monoostotic & polyostotic. The pathology is abnormal osteoblastic activity of bones. There is abnormal proliferation of fibrous tissue and progressively normal bone gets replaced by this fibrous tissue. It results in expansion of bone and narrowing of vessels, neural foramina. It eventually results in weak bones.

HRCT Findings : Loss of pneumatisation with ground glass opacity noted characteristic of fibrous dysplasia. [67]

PETROUS APEX

True neoplasms in the petrous apex includechondrosarcoma, chordoma, osteosarcoma,and meningioma. Myeloma,lymphoma, and metastases may also occur.

The petrous area may be secondarily involved by regional tumours such astrigeminal schwannoma, jugular paraganglioma,and nasopharyngeal carcinoma.The latter is usually seen along the petrooccipital fissure, superior to the fossa of Rosenmuller. Unlike lesionsin the IAC and middle ear, lesions in the petrous apex usually reach considerablesize before causing symptoms. [67]

CHONDROSARCOMA

Chondrosarcomais the most common primary malignancy to involve the petrous apex.These tumors tend to occur along the petrosphenoidal and petrooccipital synchondroses,off midline. Occasionally,however, chordomas, which arise from notochordal remnants and are typically seen in the midline, may be found off midline as well and may mimic a chondrosarcoma radiologically. Additionally, the chondroid

subtype of chordoma may be difficult to distinguish from the myxoid variant of chondrosarcoma pathologically.

HRCT Findings: Soft tissue density lesion which enhance mildly to moderately, may contain calcifications, and cause surrounding bone destruction [67]

FACIAL NERVE

FACIAL SCHWANNOMA [67]

Facial nerve schwannomas can involve any segment of the nerve and may span multiple segments. The geniculate ganglion is frequently involved. Of note, only a minority of patients initially present with facial palsy; many have no facial nerve symptoms at all. Facial nerve schwannomas in the IAC/CPA typically manifest as sensorineural hearing loss, presumably because the thinly myelinated sensory fibers of cranial nerve VIII are more sensitive to compressive effects by the tumor than are the thickly myelinated motor fibers of the facial nerve. Facial schwannomas in the geniculate region can grow into the middle cranial fossa. Those along the tympanic segment may bulge into the middle ear and compress the ossicular chain, causing conductive hearing loss. Those in the mastoid segment are more likely to present with facial palsy, owing to the surrounding narrow bony canal exerting pressure on the growing tumor. Those in the parotid segment present as painless neck masses.

HRCT Findings - The tumor may be as enhancing hyperdense lesion along the track of facial nerve canal & may cause expansile smooth remodelling of the surrounding bony canal. [67]

HEMANGIOMA [67]

Hemangiomas maybe seen along the intratemporal course of the facial nerve, most often in the region of the geniculate ganglion, followed by the IAC, and are seen least often at the posterior genu. These tumors often grow among bone trabeculae and may form bone; in these cases the term ossifying hemangioma is sometimes used. Recent literature suggests that these lesions are, in fact, venous malformations.

HRCT Findings - These lesions characteristically have an expansile honeycomb appearance and may demonstrate intratumoral bone spicules on CT images. These may be difficult to distinguish from meningiomas with intraosseous involvement.

PERINEURAL SPREAD OF TUMOR

With segmental facial nerve thickening and enhancement, an important malignant process to consider is perineural spreadof tumor. The source of malignancy is usually the parotid gland, for example, adenoid cystic carcinoma or mucoepidermoid carcinoma, ora nearby skin malignancy that secondarily invades or metastasizes to the parotid gland [64,65] . Tumor burden canvary along the course of the nerve, resulting in varying degrees of thickening and enhancement; “skip lesions” may beseen where there are areas of uninvolved nerve between abnormal segments [66] . It is important to note that the original tumor at the primary site could be absent if it has been treated; this does not preclude the diagnostic consideration of active perineural spread detectable at imaging.

OTOSCLEROSIS

The bony labyrinth consists of inner periosteal & outer endosteal, with an interposed endochondral layer [76]. Otosclerosis is development of Haversian bone in

endochondral layer. It involves otodystrophy of otic capsule and is autosomal dominant condition. Its other name is 'otospongiosis' [77,78]. Usually the patients present clinically at 20-40 years age with hearing loss of conductive type usually bilateral (85%).

It has two major categories.

1. **Fenestral type [74,75,76]** :

It involves labyrinth. Other structures to be involved are oval & round window, canal of facial nerve & promontory.

2. **Retrofenestral type [76] :**

Common involvement is cochlea. It occurs in combination with the fenestral type.The normal oval window is a dehiscent along lateral wall in vestibule with its long axis oriented in an A-P direction.

HRCT APPEARANCES:

Demineralized hypodense fenestral otosclerotic focus is visualized in axial HRCT due to antero-posterior orientation of stapes crura & oval window.Even 1 mm sized fenestralotosclerotic foci can be visualised on HRCT

In active otosclerosis, the oval window margins become indistinct, merging with otospongiotic focus of adjacent cochlea which result in "wide window" appearance.In late stages there is thickening of stapes foot plate

The retrofenestral disease is seen as an area of dimeralization within otic capsule. In the remineralized state this may not be seen. [74,75,76]

TRAUMA OF TEMPORAL BONE:

Trauma to the temporal bone usually causes fractures. For the purpose of classification the fractures of the temporal bones are divided into longitudinal and transverse types of fractures. This classification is mainly on basis of the plane of fracture line [83] .There may be also combined complex fractures. The longitudinal fracture plane runs parallel to plane of middle ear. There may be associated injuries like ossicular disruption, otic capsule damage and damage to facial nerve [83,84] .

LONGITUDINAL FRACTURES:

These fractures are fractures where the force or line of impact is along the long axis of the ear cavity in the direction of petrous apex [86,87] . On HRCT the fracture line is seen parallel to this path. There might be involvement of otic capsule. These types of fracture commonly cause injury to the ossicles [88] . Sometimes though rare there might be injury of the facial nerve. [89]

The location of these fractures is in on posterior aspect of the labyrinth. There might be involvement of squamous part or petrous part commonly. First genu of facial nerve is prone to injury. Sometimes an epidural blood collection may be seen due to vascular insult of middle meningeal artery. [86]

TRANSVERSE FRACTURE:

These type of fractures occur when the fracture line or force of impact is in the antero-posterior direction. The line of impact is perpendicular to the direction of long axis of petrous part of temporal bone. It is more commonly seen in injuries of either frontal or occipital regions. Otic capsular or ossicular involvement may or may not be seen. Sometimes facial nerve injury may also be seen [85,89] .

It may be subdivided into medial & lateral types:

This is with respect to the arcuate eminence and both present with sensorineural hearing loss.

- Medial – passes through fundus of internal auditory canal. Injury to cochlear nerve may be associated.
- Lateral - passes through bony labyrinth & may result in sensorineural hearing loss. Injury to stapes footplate may be associated.

MIXED FRACTURES

Many a times the traditional classification fails to classify the fractures and there may be complex fractures which involve both the above mentioned components [(90)] . At this time they may be described as mixed fractures. Depending on the injury to ear ossicles the patient may present with conductive hearing loss and if there is involvement of otic capsule there may be sensorineural hearing loss [(81,82,91)] .

Few other associated injuries with fracture of the temporal bone are:

- Facial nerve injury,
- Perilymphatic fistula,
- Vertigo,
- Cerebrospinal fluid leak,
- Meningitis,
- Acquired cholesteatoma [(92,93)]

OBSERVATIONS AND RESULTS

Total number of patients = 48

Table No. 7 Showing Distribution of Disease in patients

Diseases	No. of Patients	Percentage
Congenital	2	4.2%
Infections	26	54.1%
Trauma	12	25%
Tumors	8	16.7%

Table 8 .Showing Sex Distribution

Sex	No. of Patients	Percentage
Male	28	58.3
Female	20	41.7

Table 9 Showing age and sex distribution of pathologies

	Male	Female
0-10	4	3
11-20	6	3
21-30	6	7
31-40	4	3
41-50	5	1
51-60	0	2
61-70	3	1

Table No. 10 Showing clinical features

Clinical features	No. of Patients	Percentage
Hearing loss	16	32
Ear discharge	39	78
Facial nerve weakness	1	2
Head ache	15	30
Otalgia	6	12
Tinnitus	2	4
Vomiting	14	28
Diplopia	1	2
Altered sensorium	1	2
Fever	14	28
Vertigo	11	22
Swelling	6	12
Post traumatic pain	4	8

Figure 11 : Showing age distribution

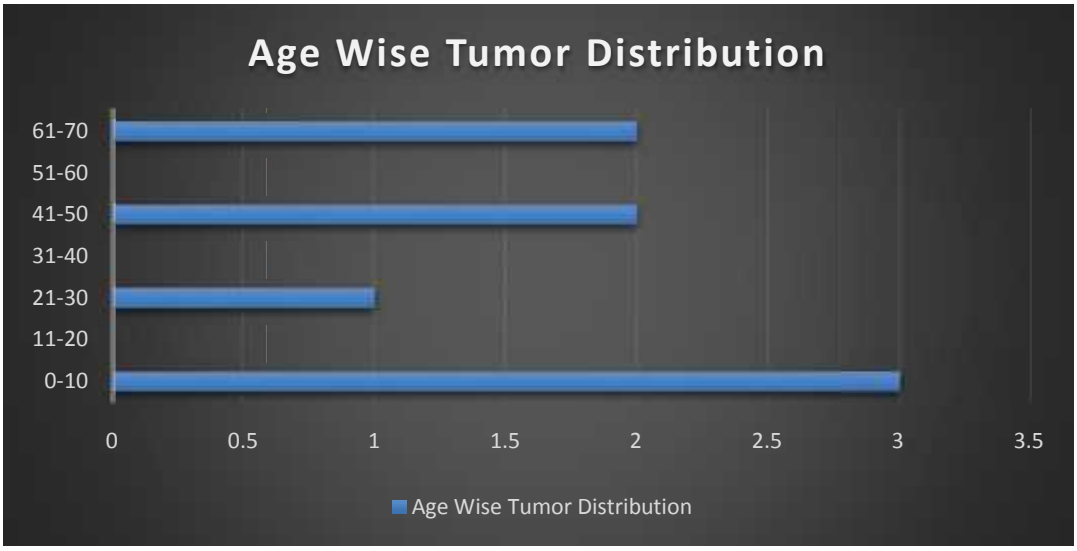


Table no. 11 Showing congenital pathologies

CONGENITAL DISEASE	No. of Patients	Percentage
External ear atresia	1	50%
Cochlear aplasia	1	50%

Table No. 12 Showing distribution of infections:

Distribution of Infection	No. of Patients	Percentage
External malignant otitis	2	8
Cholesteatoma	13	50
Mastoiditis	4	15
Suppurative Otitis Media	5	19
Petrous apicitis	2	8

Table No. 13 Showing side of ear affected

Unilateral/Bilateral	No. of Patients
Left	**11**
Right	**11**
Bilateral	**4**

Table No. 14 **Sex distribution of tumors**

Sex	No. of Patients	Percentage
Male	6	75%
Female	2	25%

Figure 12 : Graph showing Age incidence of tumors

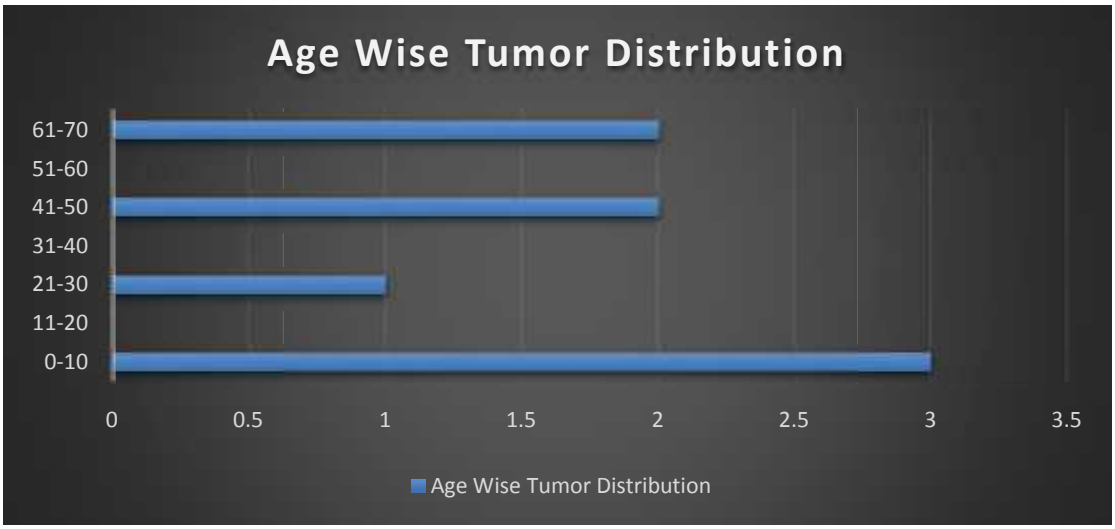


Table No. 15 Showing different tumor affecting temporal bone

Distribution of Neoplasm	No. of Patients	Percentage (%)
Osteochondroma (exostosis)	2	25
Acoustic neuroma	2	25
Meningioma	2	25
Glomus tumour	1	12.5
Rhabdomyosarcoma	1	12.5

Table No 16 Age wise distribution of acoustic neuroma

Age (years)	No. of Cases	Percentage (%)
0-29	1	50
30-65	1	50

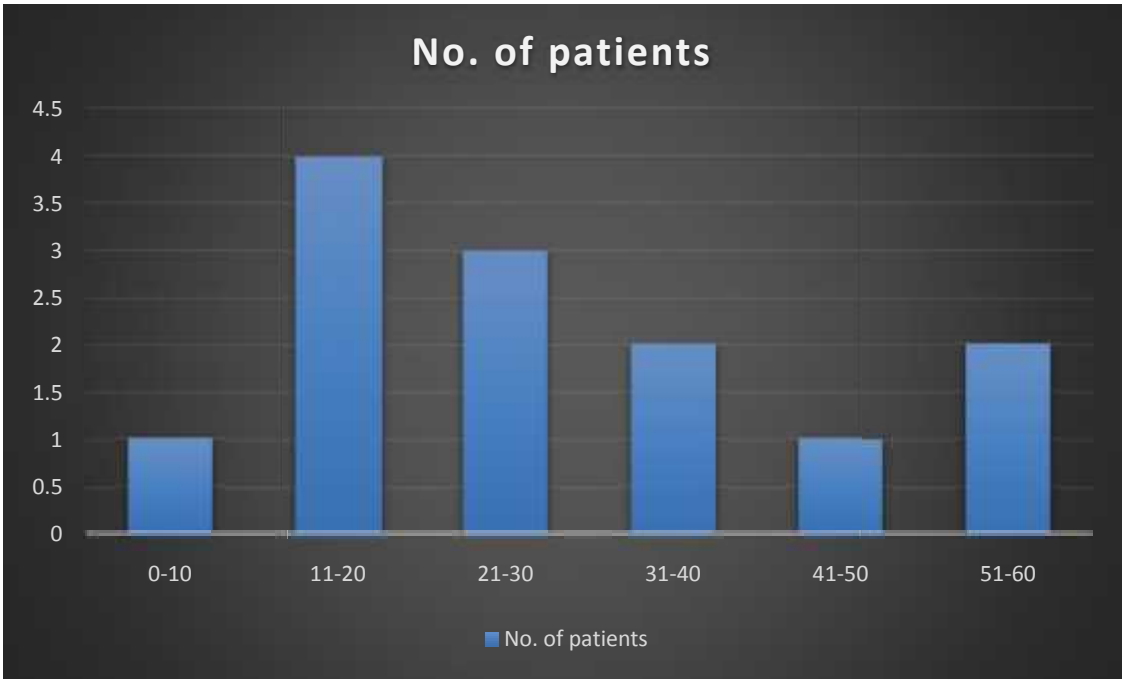


Figure 13 : Graph showing distribution of age group of patients with cholesteatoma

Table No. 17 Intracranial complication of cholesteotoma

Complications	No. of Patients
Meningitis	2
Brain abscess	1
Hydrocephalus	1
Extracranial abscess	2

Table No. 18 Distribution of fracture

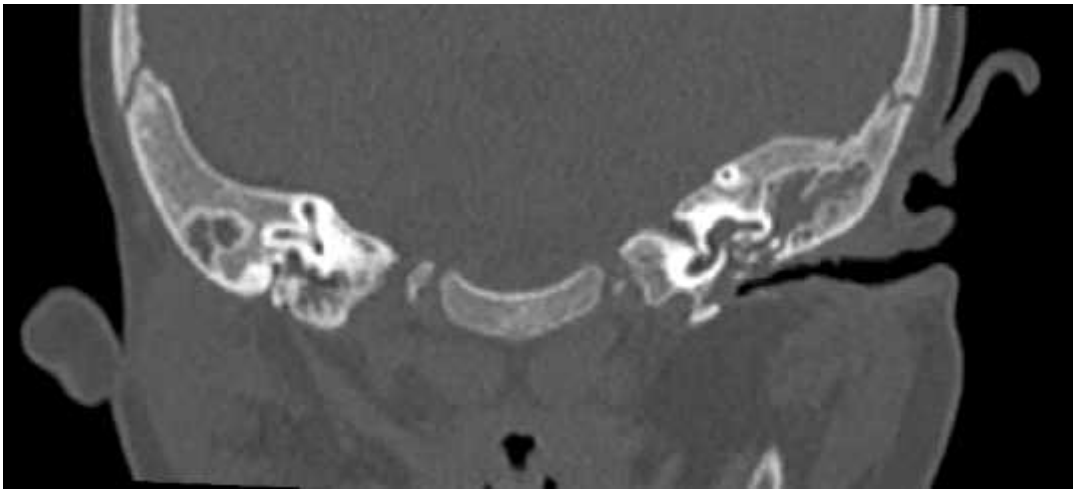
Part of temporal bone involved	No. of cases	Percentage (%)
Squamous Part	4	33
Petrous Part	3	25
Mastoid part	3	25
Tympanic part	2	17

Table No. 19 Showing different types of fracture of the temporal bone

Type of fracture	No. of cases	Percentage (%)
Transverse	5	42
Longitudinal	7	58

IMAGING GALLERY

CASE 1: RIGHT EXTERNAL EAR ATRESIA WITH HYPOPLASTIC RIGHT PINNA.



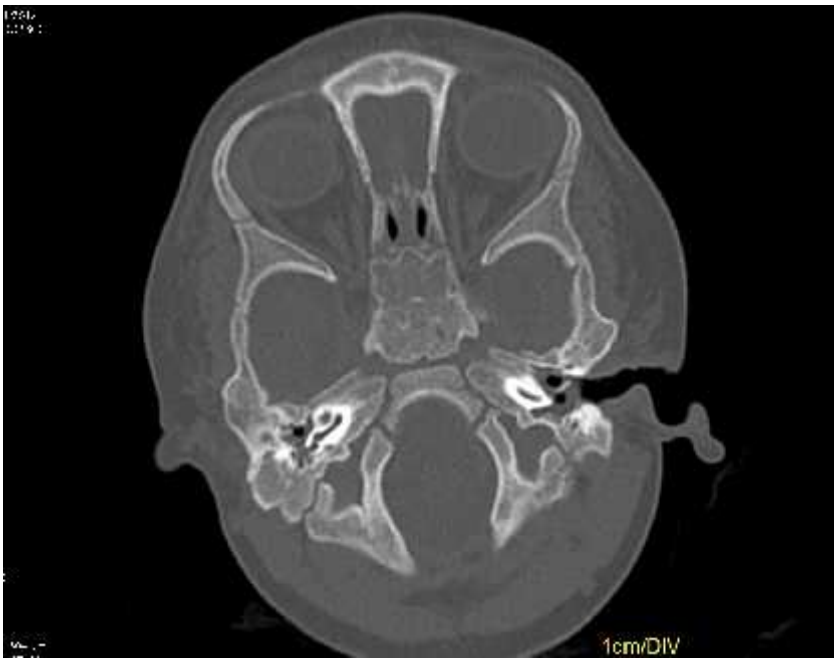


Figure 14,15 - HRCT Axial and coronal reformatted image shows hypoplastic right pinna. There is aplasia of right external auditory canal. Middle ear and inner ear appeared well formed. There was soft tissue density noted within the middle ear cavity bilaterally suggestive otitis media.

CASE 2: MALIGNANT OTITIS EXTERNA

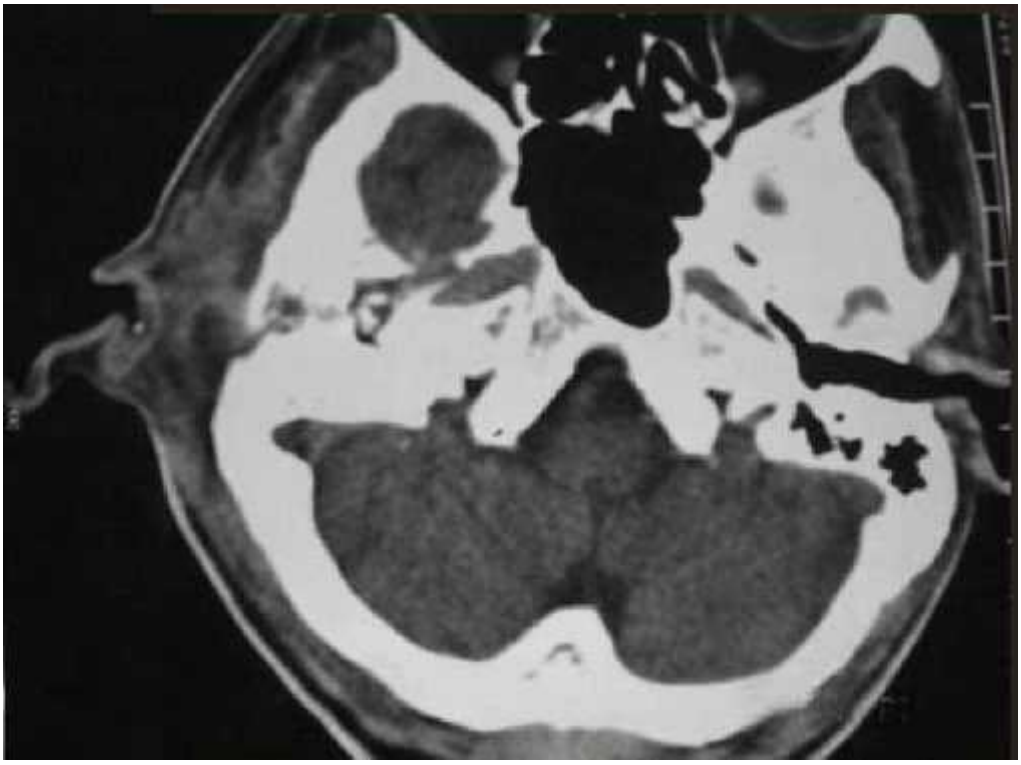
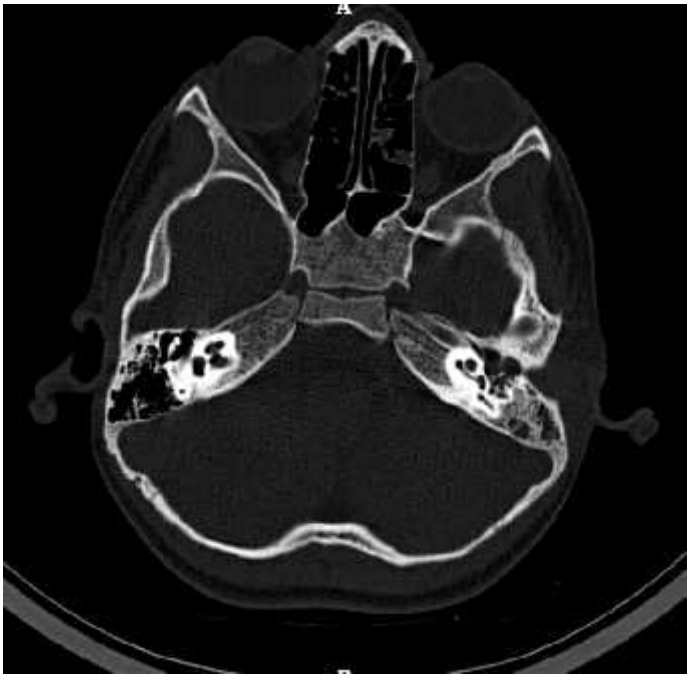


Figure 16 - HRCT Axial brain window shows enhancing soft tissue density mass involving the right external auditory canal.

CASE 3: CHOLESTEATOMA



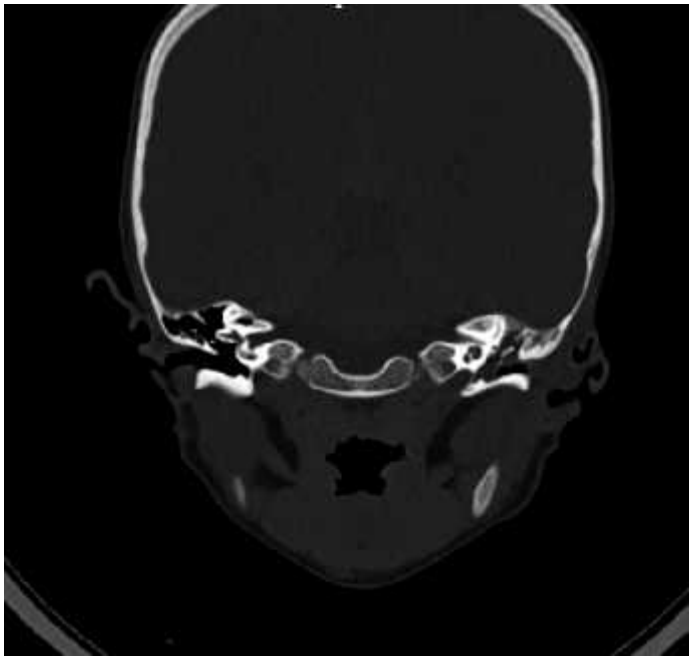


Figure 17,18 – HRCT Axial and coronal reformatted bone window images showing evidence of soft tissue density noted within the left middle ear cavity involving the aditus, antrum and Prussac's space. There was also destruction of scutum, incus, stapes and part of posterior aspect of head of malleus noted. Mild erosion and sclerosis with soft tissue density within is also noted within the mastoid cavity.

CASE 4: CHOLESTEATOMA WITH INTRACRANIAL COMPLICATION OF LEFT CEREBELLAR ABSCESS.

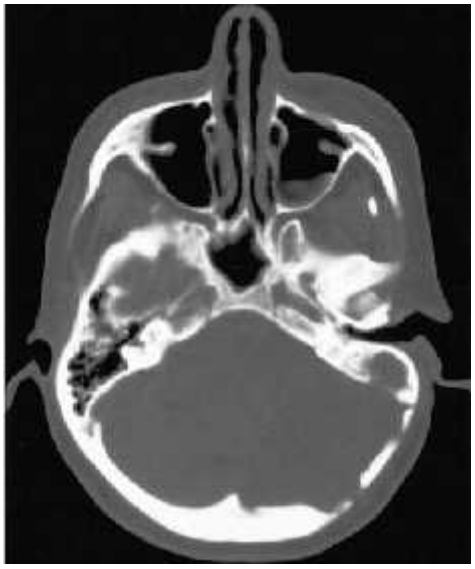


Figure 19 - Axial bone window images showing evidence of soft tissue density noted within the left middle ear cavity with erosion of petrous part of left temporal bone and erosion and scalloping of occipital bone on left side.

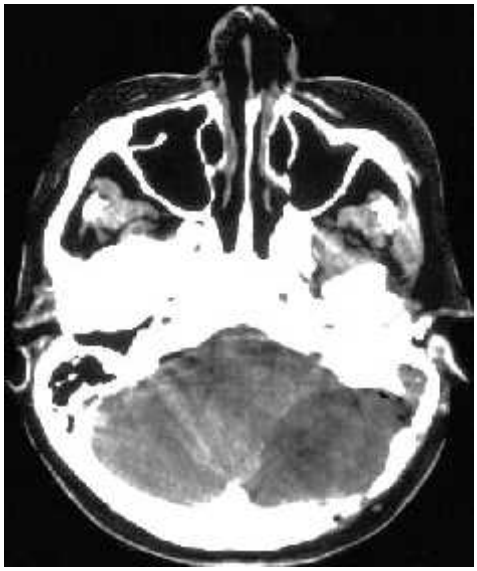


Figure 20 - HRCT Axial Brain window shows evidence of ill defined - hypodense area noted in left cerebellar region.

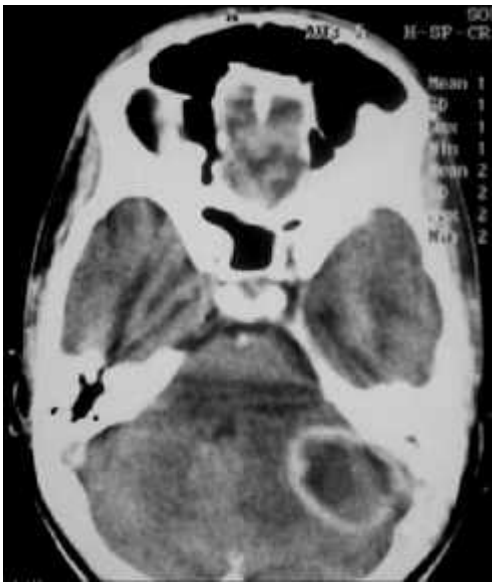


Figure 21 -CECT Shows densely ring enhancing area in left cerebellum suggestive of left cerebellar abscess developed as a complication of Cholesteatoma.

CASE 5: PETROUS APICITIS

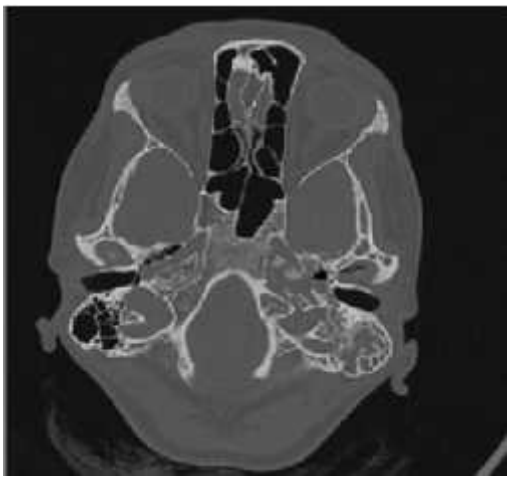


Figure 22 - HRCT axial image bone window shows erosion and widening of left petrous apex (arrow) and part of left clivus.

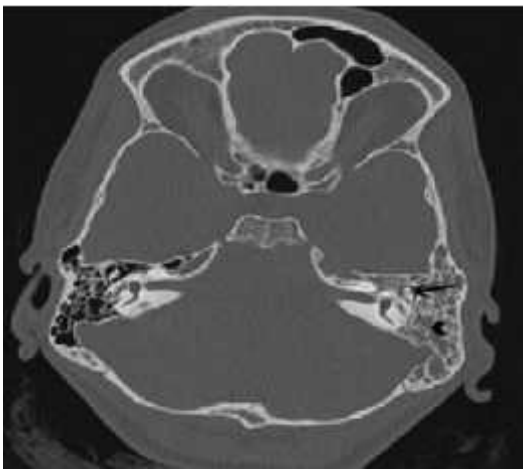


Figure 23 - HRCT axial image shows soft tissue density noted within the left middle ear cavity (arrow) and mastoid cavity (arrow head) suggestive of otomastoiditis.

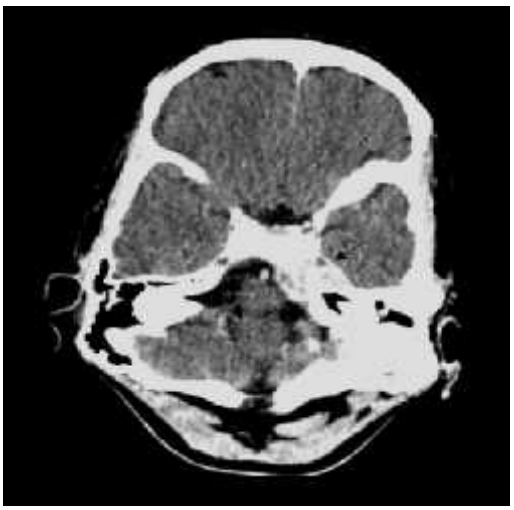


Figure 24 - HRCT axial post contrast study shows soft tissue density enhancing collection in left petrosal apex, mastoid and middle ear (arrow head) with extra-axial biconvex enhancing epidural abscesses (arrow) in left temporal region left prepontine cistern extending to left CP angle cistern.

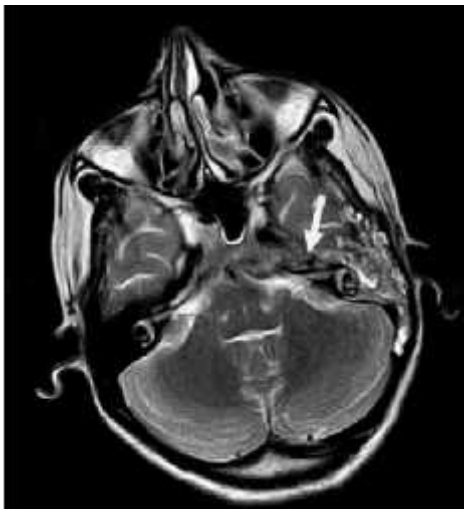
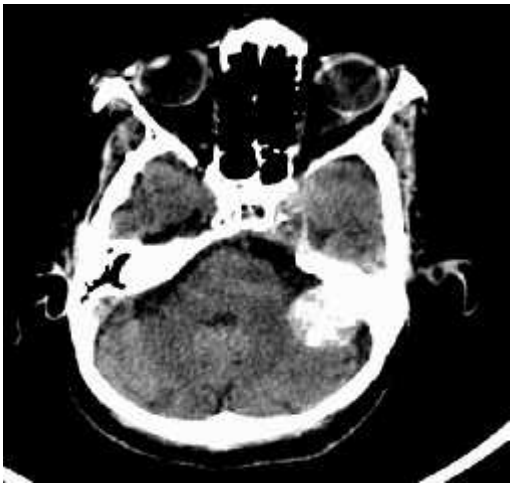


Figure 25- MRI-AXIAL T2 weighted image shows left otomastoiditis with iso to hyper intense collection in petrous part of left temporal bone, left prepontine cistern and middle ear (arrow).

CASE 6: MENINGIOMA



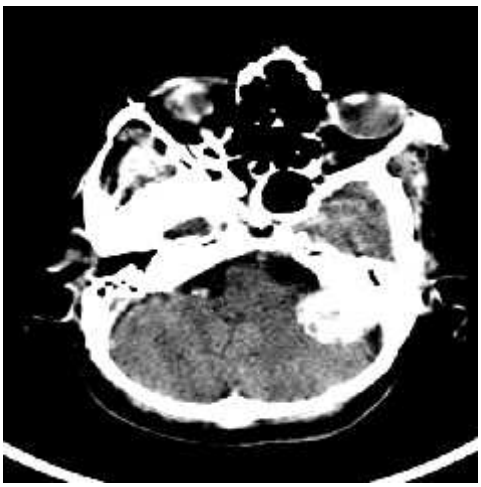


Figure 26,27 - HRCT AXIAL Brain window shows evidence of well defined mass lesion broad based towards dura in the region of left cerebellopontine angle with calcifications within and enhancement on post contrast study.

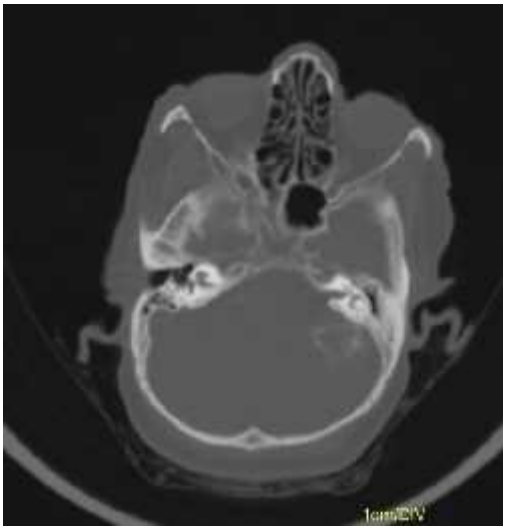


Figure 28- HRCT Axial bone window shows evidence of mass lesion near left cerebellopontine angle with few calcific foci within.

CASE 7: ACOUSTIC NEUROMA

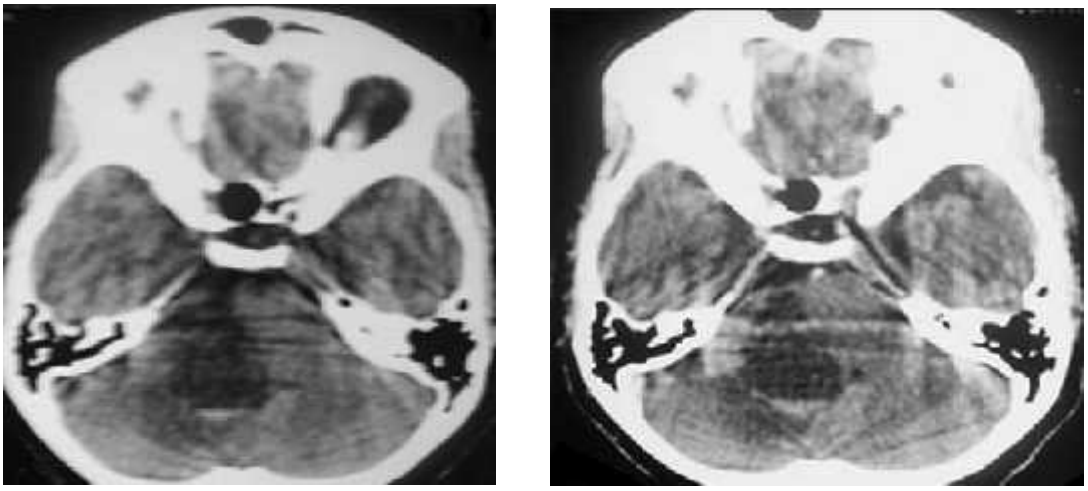


Figure 29,30 - HRCT axial brain window shows well defined mass lesion with few few hypodense cystic components noted in the region of right cerebellopontine angle causing widening of porus acousticus and showing heterogenous enhancement of solid components on post contrast study.

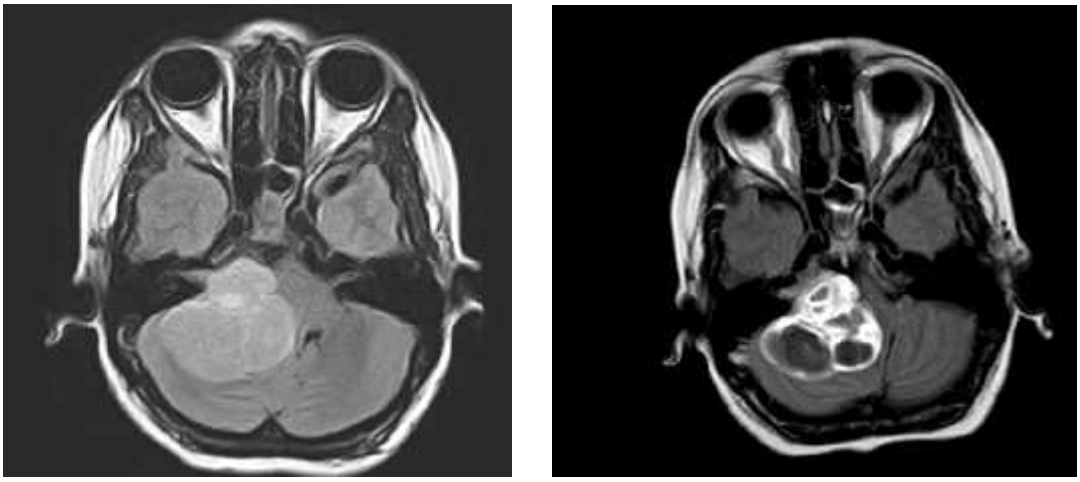


Figure 31,32 - On MRI it appears heterogenous signal intensity on T2 FLAIR with few cystic commponents image in right cerebellopontine angle. On Post Contrast T1 axial study it is showing heterogenous enhancement of intra and extra canalicular segments and trumpeted internal auditory canal suggestive of acoustic neuroma.

CASE 8: LONGITUDINAL FRACTURES

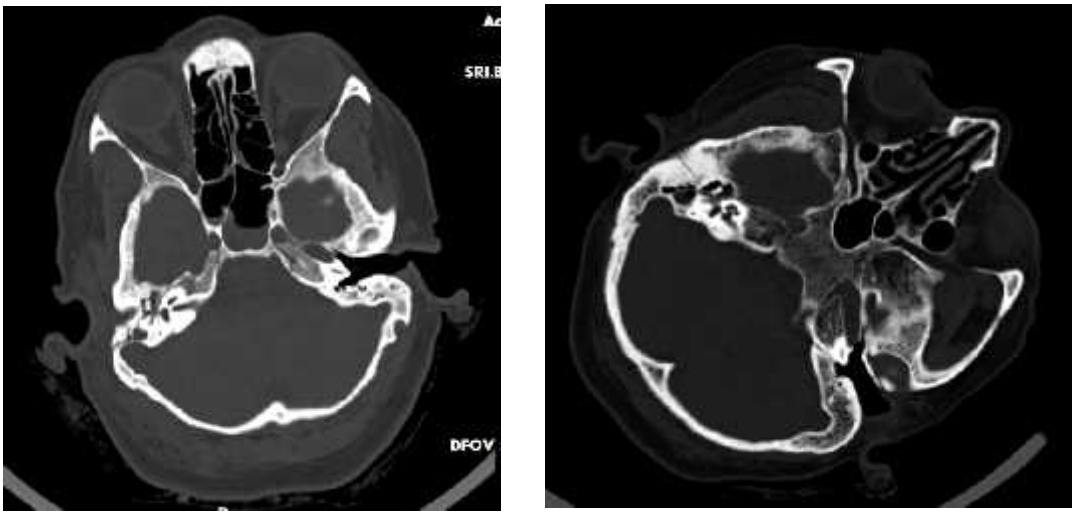


Figure 33,34 - 2 cases in CT axial bone window showing fracture line involving petrous and mastoid parts of right temporal bone with the fracture line being parallel to that of petrous ridge suggestive of longitudinal fractures. There was blood density collection noted within the middle and external ear. Ossicular chain appears intact in both cases.

DISCUSSION:

Computed Tomography is acquiring an increasingly important role in the radiographic assessment of temporal bone. Radiographic assessment of temporal bone is difficult owing to complicated anatomical structure of middle and inner ear.

This study is undertaken to develop a systemic method for evaluation of temporal bone as there are a variety of other imaging modalities. The lowest radiation dose to the lens, visualization of small bony structures, technical factors, case of patient positioning, interpretation of the images and economical factors were all considered.

HRCT has the advantage of excellent topographic visualization, devoid of artifacts from superimposition of structures. It helps in accurate assessment of pathology prior to surgical exploration regarding location, extent and complication of the disease.

CONGENITAL:

The external auditory canal, middle ear, and bulk of the ossicular chain develop from the first branchial groove, first and second branchial arches, and first pharyngeal pouch. Embryologic development of these structures is complex and rarely two anomalies are identical. Development of the inner ear structures occurs independently of external ear structures, and concomitant involvement is unusual. The study conducted by Joel D. Swartz & Eric N. Faerber et al. in 1985 included 21 cases of congenital abnormalities of ear.[15] They found 11 cases to be having congenital abnormalities of external ear, 5 to be having congenital abnormalities of middle and inner ear each. They studied patients with computed tomography only, because it was

believed that the bony and soft-tissue detail achieved is superior to that with conventional multidirectional tomography.

In our study we found only 2 cases of congenital malformations, one being external ear atresia which was bilateral and other case was of cochlear aplasia. Comparing the two studies:

INFECTION:

Patient with infection form the largest proportion of cases studied. The age range was from 2 years to 70 years, 26 cases of infections were found out of total 48 cases and out of which cholesteatoma were 13, 5 were suppurative otitis media, mastoiditis 4, petrous apicitis 2 and 2 were malignant otitis externa.

Study by GAS Lloyd et al in 30 patients with CT showed infection as the 3rd most common cause of temporal bone lesion. 1st and 2nd were tumor and temporal bone trauma respectively [(72)] . This variation could be due to the increasing number of complications associated with the infections because of the late presentation of the disease in our study which could be attributed to the low socio economic strata and illiteracy of the patients.

Mohammad F. Maffee et al(1988) studied cholesteatoma in 48 patients with Computed Tomography preoperatively [(92)] . Operative reports of these patients were correlated with CT findings in all the patients. The hallmark of cholesteatoma on CT scans are a soft tissue mass in attic and mastoid antrum associated with smooth bony expansion, scalloping of the mastoid, erosion of lateral wall of attic and erosion of scutum and ossicles. Comparing the imaging changes in the attic with findings at

operation they found agreement between the radiographic interpretation and surgical findings in 90% of the cases.

The incidence of cholesteatoma could not be estimated in the general population. In a study of the general population in Iowa, Horker and Koontz (1977) reported the overall incidence of cholesteatoma to be 6 per 1,00,000 it is however estimated that 15-25% of all cases of chronic suppurative otitis media are associated with cholesteatoma.

In our study maximum cases are seen in the 2nd and 3rd decade. 54% of the cases in this were in the age group of 11-30.

Out of 26 patients in the present study 17 belonged to low socio economic groups. This is accordance with studies and a well acknowledged fact. Poor nutrition and poor hygiene coupled with illiteracy perhaps plays a major role as most patients were found to be illiterate and ignorant about ear disease. Most patients sought medical advice very late.

The common presenting symptoms were otorrhea and otalgia. The discharge was scanty, foul smelling and purulent. Most patients presented with chronic ear discharge.

Increase ear discharge, persistent ear ache, fever, post auricular swelling and facial weakness heralded complications of cholesteatoma. The presence of vomiting, headache, drowsiness and altered sensorium indicated to more sinister threat of lurking intracranial complications. Bilateral cholesteatoma are rare.

Although western reports indicate low rate of complications as low as 1-2 %, they are common in our series in fact 6 out of 13 patients had complications. The high incidence in these cases could be because most of the cases presented late and because of illiteracy and late referral to the institute because of poor availability of medical facility at the district level.

Cholesteatoma in children and adolescent is said to be more aggressive. This is validated by the high incidence of complication in the first two decades of life and further substantiated by the fact that very extensive disease at the time of surgery is more frequent in children than in adults and also by higher rates of recidivism in children.

Limitations of the use of CT in evaluation of chronic middle ear disease:

CT scans of chronically draining ears demonstrated abnormal soft tissue densities in the middle ear or mastoid. However, if this soft tissue mass was not associated with bone erosion, it was not possible to discern whether or not cholesteatoma was present.

Infrequently the soft tissue masses were proved to be granulation tissue or mucosal hypertrophy. Of greater predictive value in the diagnosis of cholesteatoma was the presence of abnormal soft tissue densities with bony erosion.

Tympanic membrane thickening and perforations were difficult to assess on HRCT and better seen on otoscopy.

NEOPLASM:

They constitute 16.7 % of our study which is not correlated with the study of

GAS Lloyd et al (1980) which claimed neoplasms to be the most frequent lesions [72] . Age group of these patients in our series varied from 5years to 45 years with female preponderance.

Acoustic neuroma: Out of 8 neoplastic lesions that were scanned 2 (42.8%) were diagnosed as acoustic neuromas. Right CP angle predominance was noted in our study. All cases were hypodense to isodence to the surrounding brain with dense enhancement on contrast administration and depicted internal auditory canal erosion. Taylor S [68] in his study had reported bony erosion on CT in upto 87% of the cases. This difference can be because we encountered all large size acoustic neuromas. Acoustic neuroma was the most common internal auditory canal and/or CP angle lesion in a study by P Wolf (1987) [8] and GAS Lloyd (1980). [72]

Meningioma: [68]

Its incidence in our study was same as that compared to acoustic neuroma i.e. accounting for 2 out of 8 cases of neoplasms. On CT they appeared as sharply circumscribed mass that abuts the dural surface and forms an obtuse angle. They were hyperdense to brain parenchyma showing strong and uniform enhancement with positive dural tail sign.

Rhabdomyosarcoma: Rhabdomyosarcoma (RMS) is a malignant tumour of mesenchymal origin thought to arise from cells committed to a skeletal muscle lineage. The disease has a peak age presentation at 2 to 5 years and again at 15 to 19 years, and there is a slight male predominance. Three main histopathologic types of rhabdomyosarcoma have been described (1) embryonal, which accounts for 75% of tumours and is most common in younger children; (2) alveolar (20%), which has the worst prognosis and is seen in older children; and (3) pleomorphic, which accounts

for 5% of the cases. The embryonal and alveolar subtypes of RMS have been found to have distinct genetic alterations that may play a role in the pathogenesis of these tumours. Head and neck disease is further divided into, three categories based on the site of occurrence; (1) Orbital, which carries the best prognosis; (2) parameseningeal, which has the poorest prognosis; and (3) other sites.

In our study we got only one case of rhabdomyosarcoma which was a 20 year old girl and imaging findings were characteristic.

Imaging shows a soft tissue mass, often with bony destruction. Bony destruction is typically lytic and destructive, but bony remodeling can also be seen. The tumour is usually heterogeneous, maybe hemorrhagic or necrotic, and has relatively well circumscribed borders. The tumours enhance following the administration of contrast for CT. Treatment involves surgery, radiation therapy and chemotherapy.

TRAUMA:

Betsy A. Holland & Michael Brant-Zawadzki [(9)] reviewed computed tomographic (CT) findings in 18 patients with temporal bone trauma. Eight patients suffered longitudinal fractures of the petrous bone, which were associated with ossicular dislocation in two patients. Transverse fractures were detected in six patients, with a contralateral mastoid fracture in one patient. In four patients, the fractures were restricted to the mastoid region. Of the 14 patients in whom adequate neurologic evaluation was available, seven had a permanent facial nerve or hearing deficit while five suffered at least a transient neurologic deficit related to the temporal bone trauma. Routine head CT demonstrated only eight of 19 petrous bone injuries. Clues to such injury included opacification of the mastoid air cells, sphenoid sinus,

external canal and middle ear air space, and local pneumocephalus. In our study we found 12 cases of fracture of temporal bone.5 of the fractures of temporal bone were longitudinal type and 7 were of transverse type. 2 of the longitudinal fractures were associated with facial nerve injury and 4 were associated with ossicular disruption. 2 of the transverse fractures were associated with facial nerve injury and 3 were associated with ossicular disruption.

CONCLUSION

HRCT outweighs the conventional modalities of investigations and provides higher spatial resolution and better soft tissue contrast.

For the assessment of middle-ear infections, a close clinical correlation is essential to evaluate the nature of middle-ear soft tissue masses as cholesteatoma is mimicked by many other middle-ear pathologies. In these cases, HRCT:

1. Is far advantageous in assessing the complications of infection.
2. Lays down an anatomical roadmap for the surgeon preoperatively.
3. Predicts certain normal variants of surgical significance preoperatively.
4. Identifies the hidden areas of the middle-ear, namely the posterior recesses.

HRCT Scan Plays An Important Role:

1. To evaluate congenital abnormalities and their extent with the compartment of ear involved.
2. To visualize the extent of traumatic injuries of temporal bone with associated ossicular disruption or facial nerve injury helping in planning of management.
3. To comment regarding the extent of surgery, and the general overall condition of the postoperative temporal bone including the internal auditory canal.
4. The residual/recurrent disease can be assessed.
5. Status of the inner ear can be established.
6. The facial nerve anatomy can be clearly depicted. The relationship of the facial nerve to any surgical change or cholesteatoma tissue can be studied.
7. The status of the ear ossicles or prosthesis employed by the surgeon can be seen. A neoplastic disease of the middle ear is best staged with HRCT. HRCT is

not diagnostic of the pathological condition, hence the nature of the neoplastic process needs to be evaluated by a post-contrast scan.

The major functions of HRCT in the valuation of tumours of the temporal bone are summarized as follows:

1. When tumours present in the middle ear, HRCT serves to differentiate tumour from vascular anomalies and to determine the extent of deep involvement, often obviating the need for angiography.
2. Where tumours present by tinnitus or cranial nerve deficit without mass in the middle ear, HRCT serves to differentiate tumour from other benign and malignant lesions. When a lesion is large or appears atypical, angiography is of complementary value. Otherwise, unless embolisation is contemplated, angiography is not always necessary.
3. By precisely defining intratympanic, mastoid, jugular wall, infralabyrinthine and petrous apical involvement as well as posterior, middle and infratemporal fossa extension. HRCT provides essential information for planning the surgical approach.

SUMMARY

- Out of the four common pathologies of temporal bone we studied, infections were predominant with 54.1 %, followed by trauma (25%), neoplasms (16.7%) and congenital abnormalities (4.2%).
- In case of infections common presenting symptoms were otorrhea, otalgia, hearing loss and headache.
- There was overall male preponderance (58.3%) in our study.
- Most common tumours were acoustic neuroma and meningioma with female preponderance noted.
- Excellent diagnostic accuracy was noted with imaging findings in HRCT temporal bone.
- HRCT is ideal for evaluation of Temporal Bone lesion.
- LIMITATIONS OF OUR STUDY:

  - Radiation exposure to subjects during the study, especially in pediatric age group.
  - The number of cases and period of study was limited.
  - Facial nerve pathologies like facial schwannoma were difficult to differentiate from other vestibular schwannomas. Perineural spread of tumour through facial nerve would be better delineated on MRI as HRCT was less sensitive in tracing the coarse of nerve roots.

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ANNEXURE – I

B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR – 586103
INSTITUTIONAL ETHICAL COMMITTEE

No/58/2015
20/11/15

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 17-11-2015 at 03 pm scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title "Role of high resolution computed tomography in evaluation of pathologies of temporal bone"

Name of P.G. Student : Dr Vaishnav parth avinash
Dept of Radiodiagnosis

Name of Guide/Co-investigator : Dr Bhuthan. N. Lakkkar
Prof & HOD.

DR.TEJASWINI VALLABHA
CHAIRMAN
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:
1)Copy of Synopsis/Research Project
2)Copy of informed consent form.
3)Any other relevant documents.

ANNEXURE II

PROFORMA

Name:

Age:

Sex:

COMPLAINTS:

- Ear discharge
- Loss of hearing / hard of hearing
- Otalgia
- Vertigo
- Post auricular swelling
- Fever
- Headache
- Facial weakness
- Foul odour
- Hearing loss
- Tinnitus
- Vomitting

- Other complaints

ANY SIGNIFICANT FAMILY/ PERSONAL HISTORY :

IMAGING MODALITY

Computed Tomography

Description of lesion

Number of lesion

Site : Single / Multiple

Size :

Shape:

Base : Broad / narrow

Margins :

Homogenous / Heterogeneous

CALCIFICATION

- Type :
- Area :
- Density:

HAEMORRHAGE

After contrast administration

Contrast enhancement

- Intensity
    - Precontrast enhancement
    - Post contrast attenuatin
    - Mild/moderate/intense
- Pattern of enhancement
- How much enhancement

CYSTIC AREAS

Necrosis

Adjacent brain

- Buckling
- Oedema

SHIFT OF MIDLINE STRUCTURES

Ventricles

Cisterns

Generalized Brain Oedema

FRACTURES

Longitudinal

Parts of Temporal Bone Involved

Transverse

Parts of Temporal Bone Involved

Skull Underlying the Lesion

Diameter of the Internal Auditory Canal

Any Other Mass

ANNEXURE III

SAMPLE INFORMED CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYPUR – 586103, KARNATAKA

TITLE OF THE PROJECT:	**ROLE OF HIGH RESOLUTION COMPUTED TOMOGRAPHY IN EVALUATION OF PATHOLOGIES OF TEMPORAL BONE**
PRINCIPAL INVESTEGATOR:	DR. VAISHNAV PARTH AVINASH DEPARTMENT OF RADIO DIAGNOSIS *Email: vaishnavparth007@gmail.com*
PG GUIDE:	DR. BHUSHAN N. LAKHKAR PROFESSOR AND HOD DEPARTMENT OF RADIO-DIAGNOSIS SHRI B.M. PATIL Medical College & Research Centre, Sholapur Road, VIJAYPUR - 586103

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to study normal variations, congenital anomalies and infective pathologies of the temporal bone along with their complications on HRCT.

I understand that I will undergo detailed history and clinical examination and investigations.

PROCEDURE:

I/my ward have been explained that, I/my ward will be subjected to 32 slices CT screening of temporal bone region.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved and I may experience mild pain during the above mentioned procedures.

BENEFITS:

I understand that my participation in this study will help in determining role of high resolution computed tomography in evaluation of pathologies of temporal bone.

CONFIDENTIALITY:

I/my ward understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file

and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr.Vaishnav Parth Avinash is available to answer my questions or concerns. I/my ward understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me and that a copy of this consent form will be given to me for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I/my ward understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I/my ward also understand that Dr. Vaishnav Parth Avinash will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

(Signature of Patient/Guardian) Dr. Bhushan Lakhkar Dr. Vaishnav Parth A

(If the patient is conscious (Guide) (Investigator)

well oriented and fully aware)

KEY TO MASTER CHART

HRCT	:	High Resolution Computed Tomography
0	:	Absent
1	:	Present
M	:	Male
F	:	Female
EE	:	External Ear
ME	:	Middle Ear
IE	:	Inner Ear
CP	:	Cerebello Pontine
SOM	:	Suppurative Otitis Media

SR. NO	NAME	DATE	AGE	SEX	IP NO.	HRCT NO.	DIAGNOSIS	PATHOLOGIES															
								CONGINITAL CONDITIONS			INFLAMMATORY CONDITIONS						NEOPLASMS				TRAUMA	
								EE INVOLVEMENT	ME INVOLVEMENT	IE INVOLVEMENT	OTITIS EXTERNA	INFLAMMATORY CONDITION OF ME			MASTOIDITIS	PETROUS APICITIS	EE TUMOUR	ME TUMOUR	IE/ CP ANGLE TUMOUR	BONY TUMOUR	LONGITUDINAL FRACTURE	TRANSVERSE FRACTURE
												OTITIS MEDIA	CHOLESTEATOMA	INTRACRANIAL EXTENSION								
1.	BORAMMA YARAGAL	23.09.2015	60	F	38141	5028	PETROUS APICITIS	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**
2.	MAHANTAYYA MATPATI	30.10.2015	40	M	398297	6717	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
3.	BASAVARAJ	29.11.2015	17	M	435944	9349	COCHLEAR APLASIA	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
4.	GURANNA	25.11.2015	55	M	431672	7277	TRANSVERSE	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**
5.	NEELAMMA	25.11.2015	60	F	431619	7273	LONGITUDINAL	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**
6.	MABUBEE	20.11.2015	45	F	425221	7163	TRANSVERSE	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**
7.	BASAPPA N.C.	30.11.2015	41	M	436622	7362	LONGITUDINAL	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**
8.	VEERAPPA CHAKRASALLI	02.12.2015	43	M	440124	7444	SOM	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
9.	RENUKA DORANALLI	03.12.2015	44	F	441937	7476	LONGITUDINAL	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**
10.	RAVI SAJAN	04.12.2015	25	M	443844	7496	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
11.	RATNAWWA	07.12.2015	55	F	446493	7544	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**
12.	MYBOOBA WALIKAR	07.12.2015	49	F	446881	7563	LONGITUDINAL	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
13.	SADASHIV LALSANGI	08.12.2015	45	M	448174	7589	MASTOIDITIS	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
14.	NORRJAAN GHATI	15.12.2015	20	F	457137	7739	RHABDOMYOSARCOMA	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**
15.	SAHERABANU	22.12.2015	52	F	465873	7885	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
16.	SALEEM BIRADAR	31.12.2015	17	M	477626	8094	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
17.	MALAPPA	08.01.2016	73	M	684	199	GLOMUS TUMOUR	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**
18.	SUMANGALA	09.01.2016	35	F	12014	237	TRANSVERSE	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**
19.	BHIRENDRA	14.01.2016	38	M	16272	346	EXTERNAL MALIGNANT OTITIS	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
20.	GOURAWWA	04.02.2016	70	F	2025	808	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
21.	TASLEEM	29.02.2016	19	F	79446	1309	LONGITUDINAL	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**
22.	NIRMALA	20.02.2016	30	F	67501	1138	TRANSVERSE	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**
23.	RAJU CHAVAN	29.03.2016	26	M	115137	1854	LONGITUDINAL	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**
24.	BHIMAPPA	29.03.2016	15	M	115395	1864	MASTOIDITIS	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
25.	YAMANAWWA	05.04.2016	80	F	124139	2039	MENINGIOMA	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**
26.	SUJATA TAMBAKE	30.05.2016	15	F	191772	3610	SOM	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**

27.	SUSHANT	02.05.2016	16	M	156319	2529	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
28.	SWAPNIL PATIL	16.06.2016	7	M	64111	3924	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
29.	BASAVARAJ M. MIURADI	16.06.2016	14	M	20016	3932	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
30.	GURAPPA	23.06.2016	22	M	20436	4088	MASTOIDITIS	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
31.	LALBEE	12.07.2016	82	F	243077	4415	LONGITUDINAL	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**
32.	GANAPATI	13.07.2016	65	M	22846	2989	OSTEOCHONDROMA (EXOSTOSIS) EXTERNAL EAR	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**
33.	NANDINI	27.07.2016	8	F	23742	4694	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
34.	KASTURI	20.07.2016	35	F	253105	4663	MENINGIOMA	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**
35.	RAVI JADHAV	02.08.2016	16	M	270066	4792	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
36.	BASAVARAJ	22.11.2016	45	M	398720	529	OSTEOCHONDROMA (EXOSTOSIS) EXTERNAL EAR	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**
37.	MALLIKARJUN	11.12.2016	40	M	40669	757	MASTOIDITIS	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
38.	IMAMSAB	17.12.2016	6	M	434770	822	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
39.	BEBI BIRADAR	07.01.2017	31	F	7846	93	CHOLESTEATOMA	**0**	**0**	**0**	**0**	**1**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
40.	S M KASHI	07.02.2017	37	M	46036	341	LEFT ACOUSTIC NEUROMA	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**
41.	MALASHREE	25.02.2017	21	F	68806	777	SOM	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
42.	SAKANDAR	17.03.2017	22	M	8542	1056	TRANSVERSE	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**
43.	ARADYA	22.03.2017	10 MONTHS	F	100130	1153	EXTERNAL EAR ATRESIA	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
44.	BHAGYA	04.04.2017	17	F	10401	12312	PETROUS APICITIS	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**
45.	SUPROT	24.04.2017	2	M	138520	1603	EXTERNAL MALIGNANT OTITIS	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
46.	DHARMANNA	25.04.2017	21	M	139600	1613	SOM	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
47.	PRIYANKA	07.05.2017	16	F	152451	1123	LEFT ACOUSTIC NEUROMA	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**
48.	BHAGYASHREE	16.05.2017	25	F	15276	2082	SOM	**0**	**0**	**0**	**0**	**1**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**

“ROLE OF HIGH RESOLUTION COMPUTED TOMOGRAPHY IN EVALUATION OF PATHOLOGIES OF TEMPORAL BONE”

By

Dr. VAISHNAV PARTH AVINASH

Dissertation submitted to the

B.L.D.E. UNIVERSITY VIJAYPUR, KARNATAKA

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

In

RADIO-DIAGNOSIS

Under the guidance of

Dr. BHUSHAN N. LAKHKAR M.D.

PROFESSOR and HOD

DEPARTMENT OF RADIO-DIAGNOSIS

B.L.D.E.U’S SHRI B. M.PATIL MEDICAL COLLEGE

HOSPITAL & RESEARCH CENTRE, VIJAYPUR

KARNATAKA

2017

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYPUR

DECLARATION BY THE CANDIDATE

I, **Dr. VAISHNAV PARTH AVINASH,** hereby declare that this dissertation entitled **"ROLE OF HIGH RESOLUTION COMPUTED TOMOGRAPHY IN EVALUATION OF PATHOLOGIES OF TEMPORAL BONE"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. BHUSHAN N. LAKHKAR** Professor and HOD, Department of Radiodiagnosis, B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Vijaypur.

Date:

Place: Vijaypur

Dr. VAISHNAV PARTH AVINASH
Post Graduate Student,
Department of Radiodiagnosis,
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Vijaypur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYPUR

CERTIFICATE BY THE GUIDE

This to certify that the dissertation entitled **"ROLE OF HIGH RESOLUTION COMPUTED TOMOGRAPHY IN EVALUATION OF PATHOLOGIES OF TEMPORAL BONE"** is a bonafide research work done by **Dr. VAISHNAV PARTH AVINASH**, under my overall supervision and guidance, in partial fulfilment of the requirements for the degree of M. D. in Radiodiagnosis.

Date:

Place: Vijaypur

Dr. BHUSHAN N. LAKHKAR M.D.
Professor and HOD
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Hospital & Research Centre, Vijaypur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYPUR

ENDORSEMENT BY THE HEAD OF DEPARTMENT

This to certify that the dissertation entitled **"ROLE OF HIGH RESOLUTION COMPUTED TOMOGRAPHY IN EVALUATION OF PATHOLOGIES OF TEMPORAL BONE"** is a bonafide research work done by **Dr.VAISHNAV PARTH AVINASH** under the guidance of **Dr. BHUSHAN N LAKHKAR** Professor,& Head of Department of Radiodiagnosis at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Vijaypur.

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Place: Vijaypur

Dr. BHUSHAN N. LAKHKAR M.D.
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ENDORSEMENT BY THE PRINCIPAL

This to certify that the dissertation entitled **"ROLE OF HIGH RESOLUTION COMPUTED TOMOGRAPHY IN EVALUATION OF PATHOLOGIES OF TEMPORAL BONE"** is a bonafide research work done by **Dr. VAISHNAV PARTH AVINASH** under the guidance of **Dr. BHUSHAN N LAKHKAR** Professor and HOD, Department of Radiodiagnosis at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Vijaypur.

Date**:**

Place: Vijaypur.

Dr. S. P. GUGGARIGOUDAR
Principal, B.L.D.E.U's
Shri B. M. Patil Medical College,
Hospital & Research Centre, Vijaypur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYPUR

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DECLARATION BY THE CANDIDATE

I hereby declare that the B.L.D.E. UNIVERSITY, VIJAYPUR, Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purposes.

Date:

Place: Vijaypur

Dr. VAISHNAV PARTH AVINASH
Post Graduate Student,
Department of Radiodiagnosis,
B.L.D.E.U's Shri B. M. Patil Medical College, Hospital & Research Centre,
Vijaypur.

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Date:

Place: Vijayapur. ***Dr. Vaishnav Parth Avinash***

ABSTRACT

BACKGROUND & OBJECTIVES

Pathology of the ear is one of the most common reasons of visiting an otolaryngologist, with inflammatory conditions being predominant. The evaluation and diagnosis of complex lesions of the temporal bone is challenging task both for the radiologist as well as otolaryngologist. Earlier, clinical examination was used along with X- rays for the diagnosis. However with increasing prevalence of infective pathologies of ear, this approach proved inadequate.

Complicated anatomical structure of middle and inner ear makes radiographic assessment of temporal bone difficult. CT has the advantage of producing images with higher contrast and a better spatial resolution. High resolution CT (HRCT) images are obtained with thin sections and special bony algorithm for high details. HRCT, a modification of routine CT, provides a direct visual window into the temporal bone providing minute structural details. It is an excellent tool for evaluation of the middle ear diseases and adjacent bone and has the advantage of being devoid of artifacts from superimposition of structures. HRCT also helps in accurate assessment of pathology prior to surgical exploration regarding location, extent and complication of the disease.

This study is undertaken to study congenital anomalies, infective, trauma and and neoplastic pathologies of the temporal bone along with their complications on HRCT.

AIMS & OBJECTIVES OF THE STUDY:

1. To study the extent of middle ear infections and their complications.

2. To study the congenital anomalies of the ear according to compartment involvement.
3. To evaluate changes in temporal bone due to trauma.
4. To characterize neoplasms and assess their extent of involvement in temporal bone.

SOURCE OF DATA:

Data for the study is collected from the patients attending/referred to the Radiology department of B.L.D.E.U's Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur who fulfill the inclusion criteria.

METHOD OF COLLECTION OF DATA:

The study was done on patients, who visited the Department of Radio Diagnosis during the period from OCTOBER 2015 to MAY 2017. Consent taken for each case.

RESULT: In our study series of 48 cases, we got 26 cases of infective etiology, 12 cases of traumatic etiology, 8 cases of neoplasms and 2 cases of congenital anomalies. Majority of the temporal bone pathologies included infections (72%). Among infective conditions, cholestetoma was commonest with more preponderance in young age. Trauma accounted for 14% followed by neoplasms (10%) and congenital anomalies (6%).

INTERPRETATION: HRCT is a revolutionary imaging modality that helps in evaluating the normal anatomical structures, normal variants, distribution features, localization and assessing the extent of various pathologies affecting the temporal bone.

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