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**Pictorial Essay: Imaging of Acoustic Neuroma with Brief Literature Review**

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**Pictorial Essay: Imaging of Acoustic Neuroma with Brief Literature Review**

**Abstract**

Acoustic neuroma is the commonest cerebello-pontine angle(CPA) tumour. It is a benign tumour of intracranial segment of the vestibulo-cochlear cranial nerve. In this pictorial essay, we used MRI images to highlight typical imaging features of acoustic neuroma. Our patient was a 51 year old male Cameroonian civil servant with a right CPA mass. This was preceded by right unilateral sensori-neural hearing loss and disequilibrium. MRI has been adjudged to be the most recent and best radiological diagnostic tool in the evaluations of acoustic neuroma

**Key Words:** Acoustic neuroma, Cerebello-pontine angle, MRI, Vestibulo-cochlear

**Introduction**

Acoustic neuroma is a benign tumour of the intracranial segment of the eighth cranial nerve with cerebellar, brainstem and lower cranial symptomatology. These include deafness, tinnitus, and disturbance of the sense of balance (Edwards et al,2006). Acoustic neuroma(AN) is variably called acoustic neurolemmoma, vestibular schwannoma, acoustic schwannoma, Cerebello-pontine angle tumour and Eight nerve tumour (Edwards et al, 2006, Daniels et al, 2000). It accounts for 75-90% of cerebellopontine angle masses (Dahnert, 2007).

Recently, magnetic resonance imaging (MRI) has become an important method for evaluation of ear diseases (Chon et al, 2003). MRI has largely replaced other radiological and clinical investigations in the screening of patients with audio-vestibular symptoms for the presence of acoustic neuroma (Zealley et al, 2003).

**Case Report**

SK, is a 51-year-old male Cameroonian who presented with complaints of unilateral hearing loss of right ear, unsteady gait and tinnitus for

about six months. He is a civil servant and a non-smoker. There was associated headache, but no history of convulsions, trauma or projectile vomiting. No past history of tuberculosis, diabetes mellitus, hypertension, alcoholism or any other chronic illness. Audiometry revealed unilateral deafness. Open surgical intervention was contemplated due to absence of gamma knife radiosurgery in our centre. Patient declined any surgical intervention preferring a trado-medical therapy. Brain MRI studies showed a right CPA TIW isointense, homogenously enhancing mass. This mass which grows superiorly is hyperintense on T2W. A diagnosis of Acoustic neuroma was made.



FIG 1- Axial T1W brain MRI showing an oblong or funnel shaped isointense mass in the right CPA orientated along the axis of IAC



FIG 2-T2W axial MRI showing same as hyperintense with near obliteration of CPA cistern and asymmetry of 4<sup>th</sup> ventricle.



FIG 3- Gd-DTPA Enhanced axial T1W brain MRI showing homogenously enhanced right CPA mass.



FIG 4- Axial T1W MRI showing the extra-canalicular right CPA mass growing in a cranial direction



FIG 5- Enhanced Coronal T1W MRI showing right CPA mass lying in relation to middle cerebellar peduncle.



FIG 6-Enhanced Coronal T1W MRI showing asymmetry of petrous bone due to right CPA mass

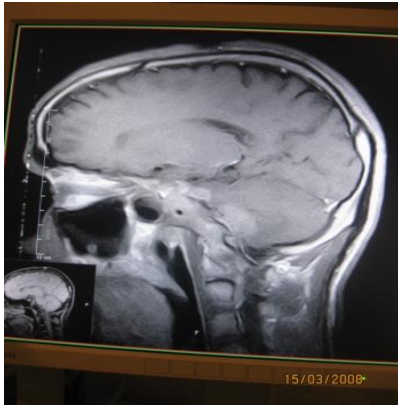


FIG 7- Sagittal contrast T1W MRI showing an enhanced mass compressing the cerebellum



FIG 8-Axial T1W enhanced MRI showing an enhancing right CPA mass effacing ipsilateral cerebellar fissures.



FIG 9- Enhanced axial T1W MRI showing right CPA enhanced ovoid mass orientated along the axis of IAC

## Discussion

Acoustic neuroma (AN) is a non-malignant, soft tissue, slowly-growing tumor involving the Schwann cells of vestibular division of the eighth cranial nerve. (Edwards et al, 2006, Curtin and Hirsch, 2008). Neuroma constitutes 8% of all intracranial tumours (Dahnert, 2007). AN forms 85% of all intracranial neuromas, 60-90% of all CPA tumours and 6 - 10 percent of all intracranial tumors (Edwards et al, 2006, Dahnert, 2007). Its incidence is 1-20 per 100,000 per year (Edwards et al, 2006). AN is a sporadic tumour seen in 35-60 years of life with male:female ratio of 1:2 (Edwards et al, 2006, Dahnert, 2007). It is mainly in individuals aged 50 or more years like our patient (Edwards et al, 2006). Most patients have a unilateral tumor and the tumor arises inside the IAC (Curtin and Hirsch, 2008).

It could equally be seen in type 2 neurofibromatosis but seen in second decade of life (Dahnert, 2007). Such central NF associated AN could present as solitary intracranial schwannoma in 5-25%. (Dahnert, 2007). But bilateral AN allow a presumptive diagnosis of type 2 NF (Curtin and Hirsch, 2008, Dahnert, 2007).

AN can be intra-canalicular or extra-canalicular or partly intra-canalicular/partly extra-canalicular. These regional localisations arose from the fact that AN lesions are thought to originate from or near the glial-Schwann cell junction (Curtin and Hirsch, 2008). This junction point is usually found just inside the internal auditory canal (IAC), but the actual site is variable enough that the AN can develop completely within, completely outside, or partly inside, partly outside the canal (Curtin and Hirsch, 2008). Most commonly, the lesion arises just inside the meatus and then grows out into the CPA cistern like our indexed patient (Curtin and Hirsch, 2008). Such a lesion is said to have both an intracanalicular and an extracanalicular component (Curtin and Hirsch, 2008). AN arises in 80% of cases from the

vestibular portion of 8<sup>th</sup> cranial nerve (around vestibular ganglion of Scarpa) at the glial-schwann cell junction) posterior to cochlear portion, in 15% from the cochlear portion (Dahnert, 2007). It may also arise in CPA cistern at opening of IAC or porus acusticus) with intracanalicular extension in 5% (Dahnert, 2007)

In the extra-canalicular subtype, the Schwann cell-glial junction can actually be outside the meatus of the IAC and the tumor will actually develop within the CPA cistern (Curtin and Hirsch, 2008). The alternative postulation for a totally extracanalicular AN is that a fairly firm or cellular tumor develops in the medial part of the IAC, grows, pressurizes and expand the canal (Curtin and Hirsch, 2008). Eventually, the tumor develops enough leverage against the edge of the canal that the lesion actually pries or lifts itself out of the canal. In doing so, the tumor may actually avulse the nerve rootlets, leaving an "empty canal" (Curtin and Hirsch, 2008).

Histologically, it is an encapsulated tumour composed of proliferating fusiform schwann cells (Dahnert, 2007). The region could be dense and highly cellular (Antoni A) with reticulin and collagen or loose areas with widely separated cells (Antoni B) in a reticulated myxoid matrix (Dahnert, 2007). The latter is commonly associated with degenerative changes, cyst formation, vascular features and lipid-laden foam cells (Dahnert, 2007).

AN present clinically as two distinct types, the unilateral sporadic type (90–95 percent of all ANs) and bilateral hereditary type (Edwards et al,2006). Finally, in the present study, Tumor laterality location is more common on the right side than left, as seen in our indexed patient (Edwards et al,2006).

Exposures to any loud noise like occupational loud noise, regular non-occupational loud noise, loud noise exposure in leisure setting (particularly when listening to loud music and at work) are all associated with AN (Edwards et al,2006). This risk increased with exposure duration (>6 years' leisure exposure). (Hours et

al,2009) The two types of loud noise exposure with the highest risk of AN were exposure to loud noise from machines, power tools, construction and exposure to loud noise from music, including employment in the music industry (Edwards et al,2006). Some studies of longer term cell phone use have found an increased risk of ipsilateral AN. (Han et al, 2009). In a Swedish INTERPHONE Study of mobile phone use and the risk of AN, the relative risk associated with mobile phone use of at least 10 years' duration was shown to be 1.9 (95 percent CI: 0.9, 4.1). When the analysis was restricted to tumors on the same side of the head as the phone was normally used, the relative risk increased to 3.9 (95 percent CI: 1.6, 9.5) (Edwards et al,2006). In terms of tumorigenesis, it is plausible that AN may arise as a result of cochlear hair cell trauma caused by intense sound exposure. During the cellular repair process, cellular division results in DNA replication errors that may in turn lead to chromosomal changes essential for neoplastic transformation (Edwards et al,2006).

It has been suggested that female hormones may also increase AN risk, although the evidence for this association is suggestive rather than definitive (Edwards et al,2006). There is a tendency toward an increased risk of AN for menopausal women (Edwards et al,2006).

Ionizing radiation exposure is the only well-established exogenous risk factor for AN (Edwards et al,2006). It has been found to increase AN risk among individuals who underwent radiation treatment of tinea capitis during childhood (Edwards et al,2006). Survivors of the atomic bombings in Japan developed an excess of benign and malignant brain tumors of various histologic types, including AN (Edwards et al,2006).

The majority of acoustic neuroma tumors grow slowly (Edwards et al,2006). Many cases have the tumor for several years before a clinical diagnosis is made. Diagnostic delay is the period between the appearance of the first symptom and the time that first medical attention is sought. (Edwards et al,2006). Diagnostic delay has ranged from 2 to 30 years (Edwards et al,2006).

Clinical presentations are long history of slowly progressive unilateral sensorineural hearing loss affecting high frequency sounds more severely (in 95%), tinnitus, vertigo, ataxia. (Edwards et al, 2006, Daniels et al, 2000, Dahnert, 2007, Myrseth et al, 2009, Sorsam et al, 2006). Occipital headache, facial numbness, and double vision have been reported due to facial, abducent and trigeminal nerves dysfunction [(Sorsam et al, 2006)]

Cross-sectional radiological investigatory tools outweigh audiograms and electronystagmography in AN assessment. MRI has become a sensitive, acceptable, and preferred method of evaluating patients with possible ANs. It can even detect inner ear circulatory disorder (Chon et al, 2003, Han et al, 2009). Gadolinium enhanced MRI is the gold standard investigation for the optimal detection of AN (Zeally et al, 2003). It should be remembered that either gadolinium-enhanced MRI or contrast-enhanced CT can demonstrate almost any AN (Curtin and Hirsch, 2008). A negative gadolinium-enhanced MRI examination is considered a reliable indicator that the patient does not have an AN (Curtin and Hirsch, 2008). Non-contrast MRI like high-resolution T2 FSE-MRI has been suggested as an alternative for screening examinations of IAC and CPA for vestibular schwannoma (Daniels et al, 2000, Curtin and Hirsch, 2008).

Imaging of the eighth cranial nerve sheath tumors has progressed from plain radiography to today's MRI (Curtin and Hirsch, 2008). In this evolution, the evaluation has progressed from attempts to show subtle findings that suggested the possibility of a lesion to actual visualization of the smallest of tumors deep within the IAC itself (Curtin and Hirsch, 2008). CT may not visualize the intracanalicular region well, and it is here that MRI establishes its advantage (Curtin and Hirsch, 2008). The soft tissue or standard algorithm in CT gives an improved visualization of the soft tissues, but the contrast of the nerves versus the cerebrospinal fluid (CSF) is still insufficient to allow demonstration of the fine soft tissue elements within the IAC (Curtin and Hirsch, 2008). CT also has a problem of artifact streaking obscuring the region of the porus acusticus and the CPA cistern (Curtin and Hirsch, 2008).

MRI, on the other hand, gives excellent soft tissue visualization but does not show the bony detail nearly as well as CT since cortical bone gives a lack of signal on MRI (Curtin and Hirsch, 2008). Since air is seen as a lack of signal on the MRI scan in normalcy, the observer is not able to differentiate the otic capsule from the air-filled middle ear (Curtin and Hirsch, 2008).

The appearance of an AN depends on the internal architecture of the tumor, site of origin along the neural pathway, tumour size and imaging specifics (Curtin and Hirsch, 2008). Wide spectrum of appearance of the AN depends on concentrations of Antoni A and Antoni B histotypes as well as cysts, intratumoral micro-haemorrhages (Curtin and Hirsch, 2008). Degenerative changes appear to be the principal cause of cyst formation and may occur in 5-13% of all AN (Sorsam et al, 2006). A report has even made of a rare case of hemorrhage within a cystic AN appearing on CT and MRI as a solitary cyst with fluid level (Sorsam et al, 2006). Hemorrhage with AN may take the form of a subarachnoid and/or intratumoral bleed and is likely to be related to the rate of growth and tumor vascularity (Sorsam et al, 2006). Cystic component could also represent an arachnoid cyst collocated and formed around a small intra-canalicular AN (Sorsam et al, 2006).

It may be difficult to appreciate AN on CT done without contrast administration because the density differences are insufficient for consistent visualization (Curtin and Hirsch, 2008). Isodense small or hypodense large solid tumour are seen in CT (Dahnert, 2007). Tumour enhancement with small tumours are usually uniformly dense (90% may be missed without CECT) (Dahnert, 2007). Ring enhancement occurs with large tumours (Dahnert, 2007). Some authors have recommended that, at least with CT, the contrast agent be injected 10 to 15 minutes before imaging to allow the contrast agent time to cross the blood-brain barrier into the lesion (Curtin and Hirsch, 2008). Intrathecal contrast or carbon dioxide insufflation is necessary for tumours <5mm (Dahnert, 2007).

On non-contrast MRI, AN are iso/slightly hypointense but brighter than CSF on T1W (Dahnert, 2007, Curtin and Hirsch, 2008). The appearance on a T2-weighted image is variable. with small intracanalicular tumors being consistently darker than CSF (Curtin and Hirsch, 2008). Intra-tumoral cysts may be dark or bright on a T1-weighted image (Curtin and Hirsch, 2008). Bright signal may represent small areas of hemorrhage, but an elevated protein content within a cyst could result in a similar phenomenon (Curtin and Hirsch, 2008)

On contrast MRI, AN shows as a bright white enhancing lesion protruding from the IAC on T1W (Curtin and Hirsch, 2008) Frequently the entire tumor enhances uniformly. If there are cystic regions within the lesion, they may not enhance, (Curtin and Hirsch, 2008) There have been scattered reports of non-enhancing ANs, but if they exist, they are exceedingly rare (Curtin and Hirsch, 2008)

Calcifications are extremely uncommon in AN (Curtin and Hirsch, 2008) In fact, if anything more than minimal calcification is present, an alternative diagnosis, such as meningioma, should be considered (Curtin and Hirsch, 2008). CT is much more likely to show small flecks of calcium if they are present but unlikely to be seen on MRI (Curtin and Hirsch, 2008)

The shape of an AN is determined by its point of origin and size (Curtin and Hirsch, 2008) As the lesion grows within the canal, pressure is exerted on the walls of the canal. AN is contiguous to the bone, which is very dense, and less conspicuous. With MRI using gadolinium, the enhancing tumor is contrasted against the signal void of the cortical bone of the IAC. The sensitivity of MRI in detecting intracanalicular tumors is therefore high. It is the resultant ability of MRI to exclude reliably small intracanalicular tumors that represents the real advantage of MRI over CT (Curtin and Hirsch, 2008)

As a lesion protrudes from the IAC into the CPA cistern, the tumor abuts the CSF and so becomes easily visible on CT and MRI as extracanalicular tumour (Curtin and Hirsch, 2008) .( See Fig 4& 6.) Once the lesion has grown beyond the plane of the porus, CT and MRI are about equal in the ability to detect this lesion

(Curtin and Hirsch, 2008). Even without contrast enhancement, most of these lesions are easily detectable on MRI (Curtin and Hirsch, 2008) (See Fig 1 &2).

Most large ANs have both an intracanalicular and an extracanalicular component, but this is not always the case (Curtin and Hirsch, 2008) . The typical appearance of the mass growing into the CPA cistern is the mass tapering toward the porus (Curtin and Hirsch, 2008) . The round or funnel-shaped mass is centred on long axis of IAC and angle made as the tumor meets the posterior surface of the petrous bone is acute rather than obtuse (Dahnert, 2007, Curtin and Hirsch, 2008) . (See Fig 1,4,9) These findings indicate that the site of origin is the IAC, and therefore the most likely diagnosis is AN (Curtin and Hirsch, 2008)

Initially as the tumor grows into the CPA cistern, the advancing margin will be round until it encounters the brain stem. AN pushes into great horizontal fissure of the cerebellum as the level of the IAC is close to this fissure (Curtin and Hirsch, 2008). The tumor may deform or flatten against the middle cerebellar peduncle giving the appearance of a fairly rounded tumor with a flattened edge along the posteromedial aspect. (Curtin and Hirsch, 2008) (See Fig 5,6,7) Widening/obliteration of ipsilateral CPA cistern also occurs (Dahnert, 2007) (See Fig 5)

Further growth can push the pons and brain stem toward the contralateral side with attendant fourth ventricular asymmetry, deformity and compression leading to obstructive hydrocephalus (Curtin and Hirsch, 2008 Dahnert, 2007) .(See Fig 2&3). Larger ANs can occasionally be associated with edema in the adjacent brain (Curtin and Hirsch, 2008)

Extracanalicular tumor can also extend superiorly or inferiorly (Curtin and Hirsch, 2008) . This can easily be shown on coronal or axial MRI (Curtin and Hirsch, 2008). Superiorly the tumor can approach the fifth cranial nerve or may place the edge of AN at the tentorial incisura (Curtin and Hirsch, 2008)

If the tumor has a component of growth in the caudal direction, the tumor is seen passing along the medulla and medial to the jugular foramen (Curtin and Hirsch, 2008). AN is thus differentiated from tumour arising from jugular foramen as it does not obliterate the CSF signal (MRI) or density (CT) in the pars nervosa of the jugular foramen, nor does it erode the margins of the foramen (Curtin and Hirsch, 2008)

Inferomedially directed growth follows the course of the seventh and eighth cranial nerves toward the root exit zone at the pontomedullary junction (Curtin and Hirsch, 2008). The foramen of Luschka can be covered by the tumor (Curtin and Hirsch, 2008). A small protrusion of choroid often passes through the foramen of Luschka into the lower CPA cistern (Curtin and Hirsch, 2008). If this small bit of choroid is incorporated into the tumor, an arachnoid cyst can be formed in conjunction with AN (Curtin and Hirsch, 2008). Alternatively a portion of the CPA cistern can become isolated from the rest of the cisternal circulation, and a cyst or CSF collection can form posterolateral to the main part of the tumor (Curtin and Hirsch, 2008). Cyst formation in tumour could be central necrosis in 15% of large tumours or coexistent extramural arachnoid cyst adjacent to tumour (Dahnert,2007)

Other imaging techniques like plain radiography and angiography remain useful tools in certain clinical settings. AN vascular supply is from external carotid artery branches (Dahnert, 2007). Angiography will show early hypervascular tumour with tumour blush (Dahnert,2007). Elevation and posterior displacement of Anterior inferior cerebellar artery(AICA) can be seen on basal views and large tumours can also elevate superior cerebellar artery (Dahnert,2007). Basilar artery can be displaced contralaterally or anteriorly/posteriorly(Dahnert, 2007). Petrosal vein can be compressed and posterior-laterally displaced (Dahnert, 2007). Choroid point of posterior inferior cerebellar artery can be displaced(Dahnert, 2007)

In conventional radiography, a difference in canal height of >2mm is abnormal and

indicates schwannoma in 93% (Dahnert, 2007). Flaring of porus acousticus, expansion and erosion of IAC are also seen (Dahnert, 2007). This is more obvious with CT showing IAC enlargement /erosion in 70-90% of cases (Dahnert, 2007).

In our indexed patient, though histology was not done because of patient abscondment but we were not reluctant in hazarding our diagnosis. Since the MRI appearance of an AN is usually characteristic enough that the diagnosis can be made with confidence, and the possibility that the lesion is something else is remote (Curtin and Hirsch, 2008)

MRI with gadolinium may detect small acoustic neuromas incidentally before significant symptoms have developed (Telian, 1994). Treatment protocol is influenced by tumor size, hearing level, life expectancy, individual's aversion to unilateral hearing loss and facial paralysis (Telian, 1994).. The most critical variable appears to be the probability that the tumor will remain stable in size (Telian, 1994). Unless life expectancy is short, surgery at the time of diagnosis is appropriate, assuming that growth of the tumor is anticipated (Telian, 1994). Smaller tumours can now be detected with MRI leading to early surgical interventions with improved rates of hearing preservations (Zeally et al,2003). Surgical option are open microsurgery (using a sub-occipital, trans-temporal or trans-labyrinthine) and gamma knife radiosurgery (GKRS) (Sorsam et al, 2006). There are better facial nerve and hearing outcomes from GKRS than from open surgery for small- and medium-sized AN (Myrseth et al,2009 and Geoganov et al, 2009). Cystic tumors represent a particular threat to patients and should only be treated conservatively with caution (Martin et al,2009)

MRI has become the investigation of choice to follow up patients after AN resection. (Brors et al, 2003). Differentiation of residual tumor from scar tissue in the IAC after resection requires close, long-term follow-up. While nodular and progressive enhancements in the internal auditory canal indicate residual tumor after AN resection, linear enhancement has



been found to be a common finding (Brors et al, 2003)

In terms of prognosis, increased tumour stage and volume worsen facial nerve function after surgery (Gerganov et al, 2009), Concomitantly, larger extra-meatal tumour diameters in three dimensions (sagittal, coronal and axial) lead to worse function (Gerganov et al, 2009), Anterior and/or caudal tumour extension had more significant correlation than posterior and/or cranial extension (Gerganov et al, 2009), Polycyclic AN has the worst prognosis, followed by the tumours with oval shape (Gerganov et al, 2009), . Patients with headache as an initial symptom, gait instability and/or pre-operative poor facial nerve function had significantly worse immediate facial nerve outcome (Gerganov et al, 2009), . The extent of intra-meatal tumour growth as well as different angles, lengths and diameters of the internal auditory channel showed no significant correlation with facial nerve outcome (Gerganov et al, 2009), .

The doubling time of AN is 2years (Dahnert,2007). Follow-up recommendation is an initial MRI scan at 6 months, then annually for 2 years.(Martin et al,2009). A further scan 2 years later will identify any patient with indolent tumors (Martin et al,2009). Thereafter, follow-up should be lifelong every 5 years (Martin et al,2009)

Rarer types of AN originate from otic labyrinth instead of usual IAC (Curtin and Hirsch, 2008) This shows on CT as enhancement within the confines of the labyrinth with some bony expansion or erosion (Curtin and Hirsch, 2008) . Differential diagnosis of AN include other tumors that occur in the region of the IAC and the CPA cistern and any cause of sudden loss of deafness. Examples are Cystic neurilemmoma of CPA cistern probably arising from glossopharyngeal nerve. Others are arachnoid cyst, meningioma, colloid cyst..Causes of deafness include inner ear lesions, intra-axial lesions (infarctions, multiple sclerosis, mesial temporal lobe sclerosis), Viral labyrinthitis (measles, mumps), vertebrobasilar circulation disorder of the inner ear, rupture of the cochlear

membrane, syphilis, and allergy (Dahnert, 2007,Curtin and Hirsch, 2008). Circulation disorders of the inner ear are presumed to be a major cause of sudden deafness. The inner ear artery, which supplies blood to the inner ear, is provided through the anterior inferior cerebellar artery, the basal artery, and the posteroinferior cerebellar artery with the rostral vertebral artery (Hours et al.2009) . In many cases, the decrease in the blood supply to the inner ear artery is caused by a decrease in the blood supply to the VBS or by vascular obstruction of the inner ear artery itself (Chon et al, 2003).

### **Conclusions**

We have used the MRI images of a typical case of acoustic neuroma to reiterate their salient radiological features which otherwise would have gone unnoticed without cross-sectional imaging.

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