CT manifestations of rare intracranial complications in patients with acquired cholesteatoma

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Learning objectives

The aim of this paper was to summarize the intracranial complications in patients with acquired cholesteatoma.
Background

Cholesteatoma is a destructive and expanding mass in the middle ear or mastoid process lined by keratinized squamous epithelium which continues to grow with shedding additional cells into the mass. Thereby, cholesteatoma is histological equivalent to an epidermoid cyst. The reason for its name is high cholesterol content.

Cholesteatomas are divided into two main types: congenital and acquired. The vast majority of acquired cholesteatomas (which make up 98% of all cholesteatomas) develop as a result of chronic middle ear infection and are usually associated with perforation of the tympanic membrane.

Due to its expansive nature, cholesteatoma can cause the destruction of the ossicles with consecutive complications such as labyrinthitis, labyrinthine fistula, perilymphatic fistula, facial nerve dysfunction, inflammatory neuroma of the facial nerve extension through inner ear into internal acustic meatus leading to deafness, cochlear fistula and extension into the petrous apex with petrous apicitis.

Intracranial complications develop due to cholesteatoma spread through the base of the skull with extension into the middle cranial fossa resulting with meningitis, cerebral abscesses with periosteal, temporal lobe, pontine or cerebellar localization, sigmoid sinus thrombophlebitis and multifocal cerebral lesions.

In general, intracranial complications are more likely to appear in acquired cholesteatoma as a result of secondary infection. Erosion into the otic capsule of the lateral semicircular canal is frequently identified where disease spread usually follows an orderly pattern through a route of least resistance via the aditus ad antrum, antrum, and into the mastoid bone proper.
Findings and procedure details

CT examination was performed on Toshiba Aquillion/CXI machine before and after intravenous administration of contrast agent along the axial plane (acquisition parameters: matrix 512, FoV 250, thickness 0.5x64, KV 120, mA 200). Axial slices have been reconstructed parallel to the lateral semicircular canal. Volume acquisitions techniques allowed evaluation of all anatomical structures and pathological features (location and extension of cholesteatoma, erosion of the ossicular chain, integrity of the facial nerve, mastoid air cells, petrosquamosal septum and semicircular canals, changes in the tympanic membrane and intracranial changes). CT reports were correlated with surgical and histopathological findings in all patients.

Cholesteatoma complications are consequences of bone erosion of the pyramid and almost always connected with recurrent infection. In the middle ear complications include: hearing loss (consequence of erosion of ossicles or from suppurative labyrinthitis), labyrinthine fistula, labyrinthitis, petrous apicitis, facial nerve paralysis (acute form due to infection and chronic form as a result of compression from cholesteatoma growing around facial nerve). If there is a dural involvement it always includes bone erosion and destruction of tegmen tympani or tegmen mastoideum and as consequence produce durra involvement that primarily starts as meningitis and it could be seen on postcontrast CT scans as focal enhancement of meninges during active phase. Late interrassial complications include intracranial infections with development of brain abscess (cerebral or cerebellar and it is seen on postcontrast CT scans as a hypodense collection surrounded by enhancing capsule usually followed with perifocal edema in the surrounding brain tissue). If infectious process causes meninges it could be further developed as epidural or subdural abscess. If the process spreads to the nearby venous sinuses, thrombosis could be developed ("delta sign" on postcontrast CT includes perisinus dural enhancement and filling defect of the lateral sinus), the most common is sigmoid sinus thrombophlebitis. Facial nerve palsy could be developed if there is a facial nerve dehiscency, similarly lateral rectus palsy (affection of n. abducens) and sensorineural hearing loss (affection of n. vestibulocochlearis) could be presented, but rarely. Otitic hydrocephalus and intracranial extension of cholesteatoma are also some of the rare intracranial complications.

All patients included in this study had presented with signs and symptoms of non specific chronic otomastoiditis with othorrea, fever and mild form of neurological symptoms. Interestingly, all patients were male neglecting their symptoms for years and were hospitalized due to significant progression of their condition.

CT findings in our series included homogeneous unilateral or bilateral soft tissue mass with bone destruction and propagation into the middle cranial fossa resulting in various intracranial complications - meningitis, cerebral abscesses, sigmoid sinus thrombophlebitis and cerebral lesion with cholesteatoma origin. All patients had
destruction of the tegmen of the pyramid and that was the point of entree for spreading cholestatoma from middle ear into intracranial compartments.

One of the commonest findings in patients with intracranial complications were cerebral abscess formations. One of these patients presented with fully developed brain abscess of the base of temporal lobe with surrounding perifocal edema and local spreading of inflammation through the leptomeninges with postcontrast opacification of the leptomeninges - sign of focal meningitis of the temporal lobe. He underwent surgical intervention with drainage of abscess collection and final diagnosis was hystopathologically confirmed as inflammatory intercranial process. After aggressive antibiotic treatment patient was released from hospital without complications.

The most interesting case was the patient with signs of intracranial extension with multiple homogeneous soft tissue masses intracranially in both middle and posterior cranial fossa without any sign of postcontrast opacification. Those masses were connected, surrounded with perifocal edema and showed compression on surrounding intracranial structures. This was the extremely rare case of multifocal cerebral lesions with cholesteatoma origin. Surgery was performed and pathology report confirmed cholesteatoma origin of intracranial growths. This patient had partial resection of described intracranial masses, but after one week he developed large intracranial hemorrhage and expired.
Images for this section:

**Fig. 1:** Coronal CT before and after i.v. administration of contrast agent demonstrate an intracerebral abscess in left temporal lobe and focal inflammatory meningeal reaction in patient with an acquired cholesteatoma.

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Fig. 2: Axial CECT demonstrates an extension of middle ear cholesteatoma in right cerebelar hemisphere.

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**Fig. 3:** Coronal and sagittal CECT demonstrate multiple intracranial hypoattenuating cerebral and cerebelar lesions cholesteatoma origin.

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Conclusion

CT is the modality of choice for diagnostic evaluation of cholesteatoma and its intracranial complications due to the ability to precisely demonstrate both the anatomy of the temporal bone and potential intracranial consequences of cholesteatoma. MRI is preferable in the postoperative monitoring when CT may be indeterminate since granulation tissue, scarring and recurrent cholesteatoma may all appear similar to.
References


