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CASE REPORT

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Exploring the uncharted: adenoid cystic carcinoma nestled in temporal bone

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Abstract

Background Adenoid cystic carcinoma is an uncommon malignancy primarily arising from salivary glands. An extremely rare site for adenoid cystic carcinoma is the skull base. We report a case of adenoid cystic carcinoma of skull base who presented with common complaints of pain and right ear discharge. The discussion is made with emphasis on imaging evaluation simulating infective etiology with adjacent skull base osteomyelitis. Careful observation of the imaging findings and further evaluation of the patient revealed the neoplastic nature of the lesion with the final diagnosis being adenoid cystic carcinoma.

Case presentation A 40-year-old female presented to our department with complaints of pain and right ear discharge since 6 months with progressive, extensive facial swelling and facial nerve palsy. The patient had undergone modified radical mastoidectomy thrice, but the details were not available. On imaging, there was a heterogenous extensive lesion extending from scalp till upper cervical region with extensive destruction of skull base and intracranial extension. The possibilities of temporal bone squamous cell carcinoma and extensive skull base osteomyelitis were considered. Further the biopsy of the lesion revealed adenoid cystic carcinoma.

Conclusions Extensive lesions of the skull base can be of infective, neoplastic and inflammatory etiology. Distinguishing between these conditions is crucial, as they have similar imaging characteristics but require different management approaches. The presence of a lesion that displaces or destroys fascial planes, accompanied by solid mass-like enhancement, indicates a higher probability of a neoplastic origin rather than an infectious etiology. With squamous cell carcinoma being the most common neoplasm, adenoid cystic carcinoma of the skull base also needs to be understood due to its propensity for perineural spread and a high likelihood of recurrence.

Keywords Adenoid cystic carcinoma, Temporal bone, Skull base lesions, Perineural spread, Skull base osteomyelitis

Background

Adenoid cystic carcinoma is a malignant neoplasm comprised of epithelial and myoepithelial cells arranged in tubular, cribriform, solid, or mixed architectural shapes. It primarily arises from the submandibular, sublingual, and minor salivary glands [1]. The most frequent way that these malignant neoplasms spread is through local and cerebral infiltration, especially along nerve sheaths [2]. Even though it usually starts in the salivary glands of the head and neck, the ceruminous and lacrimal glands, as well as the excretory glands of the vaginal tract, are additional sites of genesis for this form of cancer.

It is a highly aggressive, slowly developing malignancy that frequently recurs.

Lymphatic dissemination to nearby lymph nodes is infrequent, while hematogenous spread commonly occurs during the progression of the disease [3]. The surgical literature provides a thorough description of the patterns of tumor spread. While magnetic resonance imaging (MR) is favored for defining the extent of cancer

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dissemination along arterial channels, neurological pathways, intra-cranial extension, and extratemporal soft tissue involvement, computed tomography (CT) can show bone erosion in precise detail.

Case presentation

A 40-year-old female presented with complaints of right sided ear pain and discharge since 6 months with right sided hardness of hearing and extensive facial swelling. She also had history of deviation of angle of mouth to left since 6 months and voice change since 3 months.

Patient had undergone modified radical mastoidectomy thrice, specifics of which were not available. Local examination revealed a swelling of 2×2 cm anterior to helix of right ear with blood stained muco-purulent discharge in external auditory canal. Tragal and mastoid tenderness was present. The examination of the left ear, nose, and throat was unremarkable. No palpable cervical lymphadenopathy.

On Cranial nerve examination, deviation of angle of mouth to left, inability to blow cheek and absent forehead crease were observed, implicating right sided UMN type facial nerve palsy. On protrusion of tongue, the tongue was seen deviating to the right indicating right hypoglossal nerve palsy. The rest of the cranial nerve examination was unremarkable.

The patient had a history of voice change and underwent video-assisted laryngoscopy, which showed right vocal cord paralysis with a gap during phonation.

Routine blood tests showed normal WBC count, blood cell morphology, and erythrocyte sedimentation rate. Ear discharge was tested for gram staining and culture sensitivity which showed staphylococcus aureus as isolated organism.

With provisional diagnosis of right post MRM cavity infection and right facial cellulitis, the patient was subjected to contrast-enhanced CT of temporal bone for further valuation.

The patient underwent HRCT scan of temporal bone on a 128-slice Philips CT scanner with slice thickness of 1 mm, mA-400, KV-120. Post-contrast studies were performed after injecting 80 ml of 300 mg I/ml iohexol contrast intravenously. On CECT, there was non-visualization of middle ear ossicles and posterior wall of external auditory canal with exteriorization of mastoid and middle ear cavity indicating post-surgical status. An extensive heterogenous lesion with epicentre in the right petrous temporal bone, exhibiting heterogenous post-contrast enhancement, was seen extending cranially into the right fronto-temporo-parietal scalp, up to right upper cervical anterior and posterior para-vertebral lesion inferiorly, with bulky right para-vertebral muscles (Fig. 1). The lesion is causing erosion of temporal bone (mastoid, petrous and squamous parts), greater wing of sphenoid, parietal and occipital bones on right side with destruction of jugular spine and occipital condyle and hence involving right jugular fossa and hypoglossal canal (Fig. 2). The lesion also seems to involve right external and internal auditory canals, along with right facial canal, causing its destruction. Antero-superiorly, the lesion displays intracranial extension with peri-lesional edema into the right temporal fossa and right temporal lobe through eroded squamous temporal bone. Invasion of the lateral plate was seen, along with extra-dural and dural involvement of right sigmoid and transverse sinuses along with probable involvement of right cerebellum (Fig. 3).

A detailed assessment of the intra-cranial lesion was done using MRI. On MRI, the lesion appeared heterogeneously iso to hyper intense with multiple high signal areas within. Cranially the lesion extended beyond the tectal plate into the middle cranial fossa with invasion of temporal dura and intra-cranial extension for approximately 1 cm. The lesion appeared to involve the inferior temporal gyrus with significant edema in the right temporal lobe. Significant edema and soft tissue thickening was seen involving the right temporal and parietal scalp, with the lesion extending cranially for a length of 6 cm causing loss of normal low signal of outer table, which appeared irregular and extending into diploic space. The lesion also appeared to involve and invade the right lateral sinus, with loss of T2 flow void which could be due to slow flow or thrombosis, with later being more likely due to invasion. The lesion also showed infra-tentorial extension abutting and displacing the right cerebellar dura. There was no evidence of trans-dural extension and cerebellar invasion. Medially, the lesion extended and involved the right jugular fossa and hypoglossal canal with STIR high signal of the right half of the tongue which was suggestive of hypoglossal nerve involvement. No significant fatty infiltration or loss of bulk seen, which was pointing toward acute onset. The lesion extended into the infra-temporal fossa and appears to involve the foramen ovale with loss of bulk involving the right masticator group of muscles with fatty infiltration on T1 indicating involvement of mandibular division of right trigeminal nerve (Fig. 4). Right parotid gland showed diffuse fatty infiltration.

With the above imaging findings, the differential diagnosis of temporal bone squamous cell carcinoma and extensive skull base osteomyelitis were considered.

Under local anesthesia, biopsy was taken from proliferative growth anterior to helix of right ear (specimen A) and also from ulcero-proliferative lesion in right MRM cavity (Specimen B). Both specimens were submitted for histopathological examination. On

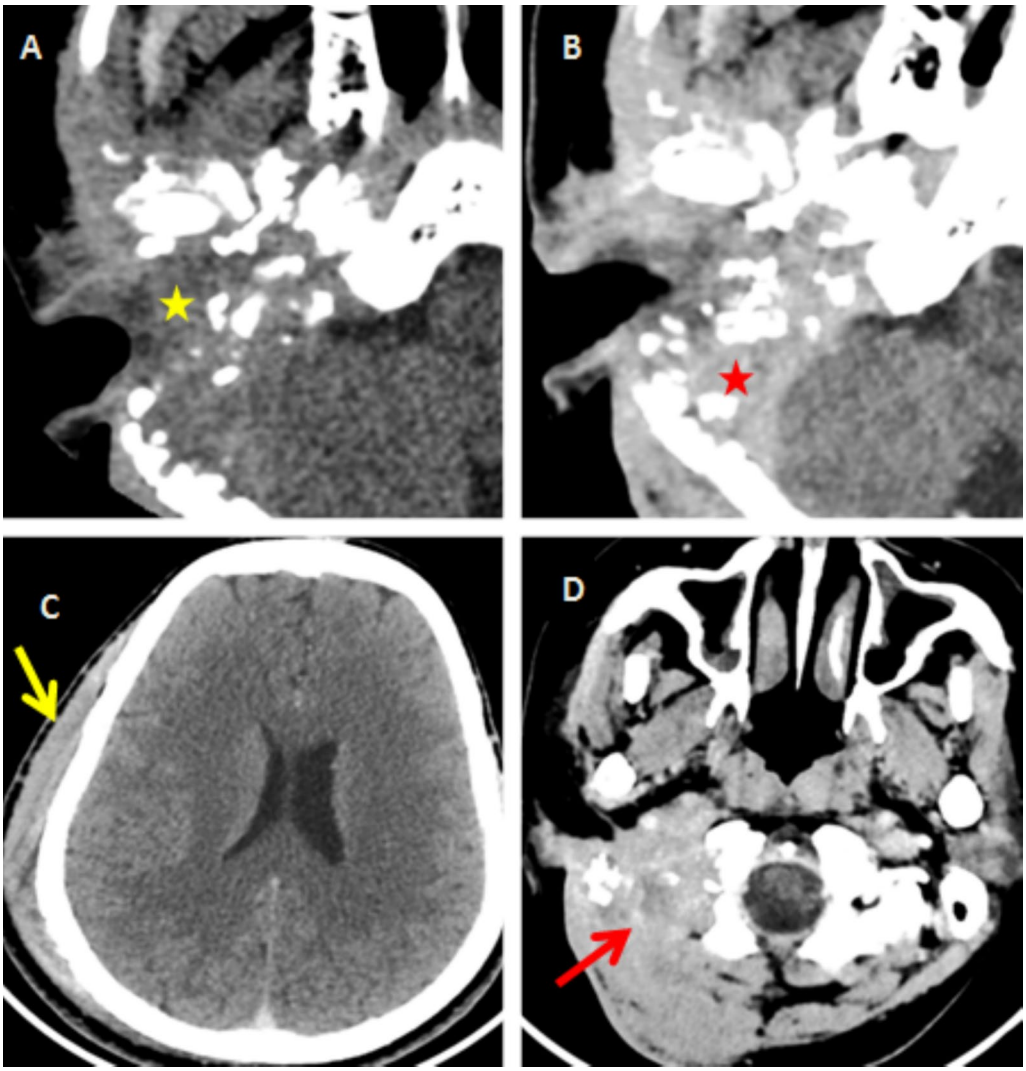

Fig. 1 **A** NCCT in axial section showing a slightly hyperdense lesion (yellow star) with epicentre in right temporal bone. **B**–**D** CECT images depicting mass-like enhancement of the lesion (red star) extending cranially upto the fronto-temporo-parietal scalp (yellow arrow) and inferiorly upto the cervical para-vertebral muscles (red arrow)

histopathological examination, specimen A showed fragments of fibrocollagenous tissue lined by skin with mild basket weave hyperkeratosis, focally flattered rete ridges and attached structures. The underlying dermis showed an ill-defined infiltrative lesion composed of cells arranged in glandular and cribriform pattern (Fig. 5). Specimen B displayed predominantly necrotic tissue admixed with inflammatory exudate with few focal areas of scattered hyperchromatic atypical cells, with final diagnosis given as adenoid cystic carcinoma.

Discussion

Extensive lesions involving the skull base with perineural spread can be of infectious, inflammatory or neoplastic cause. Infectious causes are termed as Skull base osteomyelitis (SBO) which can be central or peripheral type. Neoplastic causes are squamous cell carcinoma (SCC), adenoid cystic carcinoma (ACC), lymphoma, myeloma and metastasis; of which SCC is the most common cause. Recently IgG4 related inflammatory pseudotumor has been the cause for mass-like lesions in various locations.

Most of the time, primary forms of local invasive neoplasms that affect the base of the skull start as a dominant soft-tissue mass or nodule that then spreads to the bone. On cross-sectional imaging, the primary

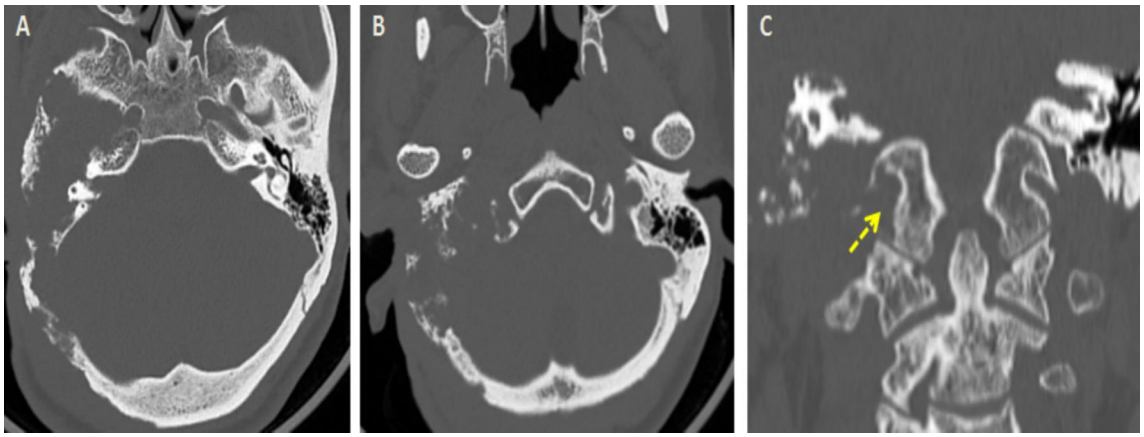

Fig. 2 **A** NCCT in bone window displaying erosion of temporal bone(mastoid, petrous parts) and occipital bone on right side. **B** NCCT in bone window showing destruction of jugular fossa, hypoglossal canal and occipital condyle. (**C**) NCCT in bone window and coronal section depicting loss of normal eagle sign on right side with widening of right hypoglossal canal (yellow dotted arrow)

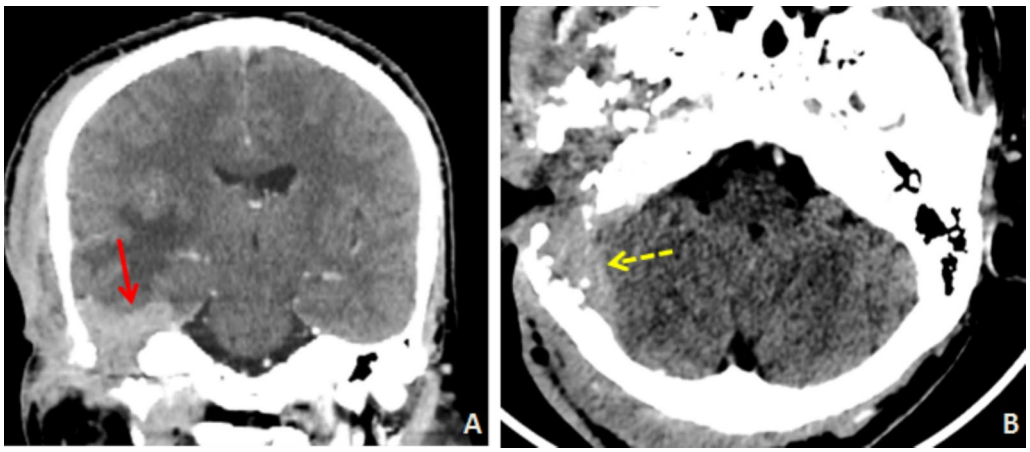

Fig. 3 CECT in coronal (**A**) and axial (**B**) sections displaying intra-cranial extension into right temporal lobe (red solid arrow) with peri-lesional edema, extra-dural and intra-dural involvement with right sigmoid and transverse sinus invasion (yellow dotted line) through eroded lateral plate

lesion appears as a nodule or mass-like enhancement in the extraosseous soft tissues, although the osseous skull base may have significant derangement due to invasion and destruction. Malignant neoplasms, in contrast to SBO, usually replace or displace normal anatomy without maintaining tissue planes. If fascial planes are enhanced and full without being destroyed, this could confirm an SBO diagnosis rather than a tumor [4, 5]. In cases where a lesion involving the base of the skull has a prominent, solid enhancing soft-tissue component that correlates with a clinically apparent mass involving the skin or mucosa, neoplasm is strongly favored over infection [4].

Skull base osteomyelitis (SBO) is a rare, potentially life-threatening infection that presents as a diagnostic challenge clinically and radiologically [6]. It typically manifests in patients with diabetes and recurrent otitis externa. The infection usually extends inferiorly to the compact bone of infra-temporal fossa affecting the neural foramina of lower cranial nerves [7]. Four factors should be looked at when diagnosing SBO: a high index of clinical suspicion, radiologic evidence of infection, multiple biopsies that show no malignancy, and positive microbiologic test results [7].

Imaging findings: Findings of skull base osteomyelitis are non-specific and can mimic malignancy. Computed tomography marks the best modality for evaluation of bone erosion and demineralization. Furthermore, diffuse soft-tissue swelling, obliteration of normal fat planes, involvement of the skull base foramina, and vascular complications can be seen on contrast-enhanced CT scans [8, 9].

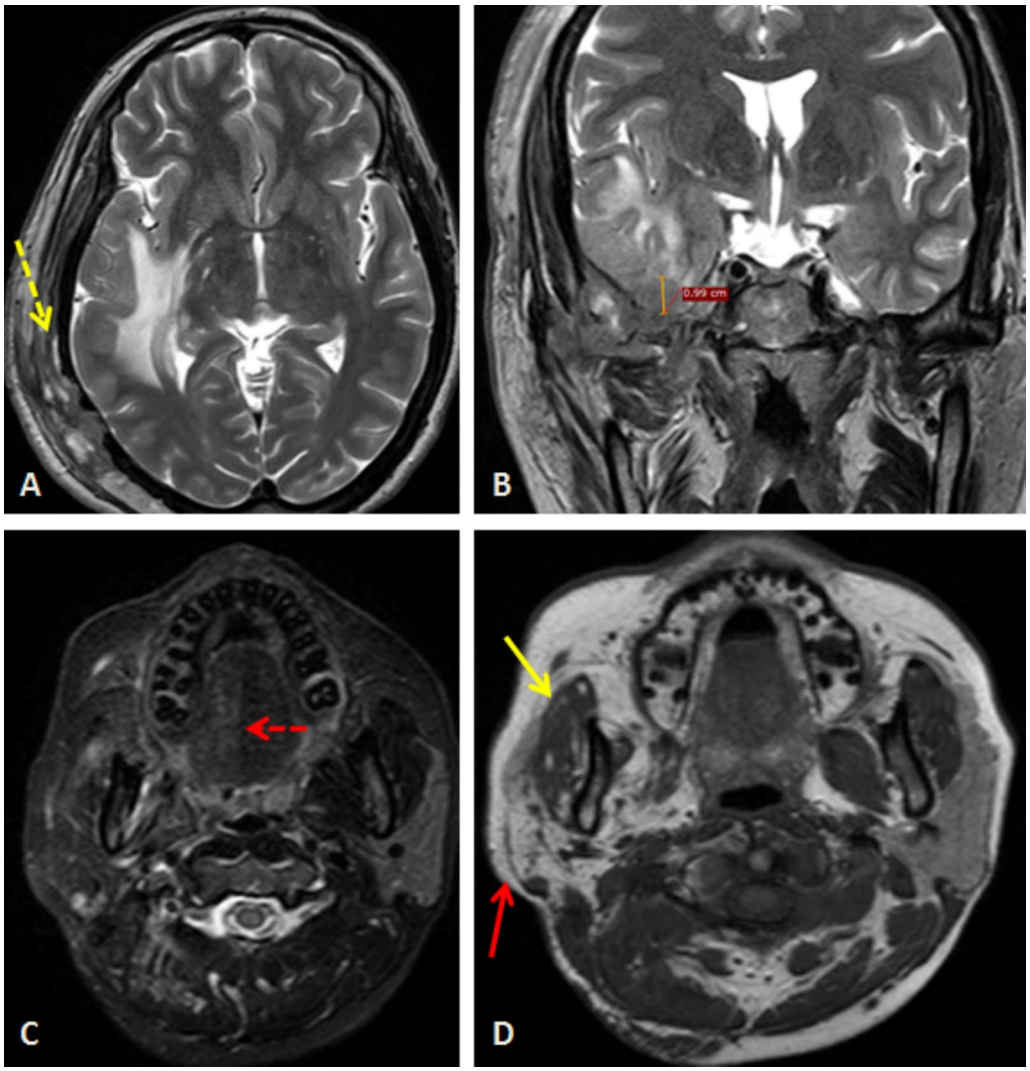

Fig. 4 T2 weighted images in axial (**A**) and coronal (**B**) sections showing loss of normal low signal of outer table (yellow dotted line) with adjacent soft tissue thickening of scalp and intra-cranial extension of lesion approximately for 1 cm. STIR and T1 weighted images in axial sections displaying high signal of right half of tongue (red dotted arrow) suggestive of hypoglossal nerve involvement and loss of bulk involving masticator group of muscles with fatty infiltration (yellow solid line)along with diffuse fatty infiltration of right parotid gland (red solid arrow). Technique: Philips Insta 1.5 T MRI—T2WI axial and coronal: TR-4843 ms, TE-100 ms, matrix 324 × 260 and slice thickness—3 mm. T1WI axial: TR-522 ms, TE-18 ms, matrix 380 × 258 and slice thickness—3 mm. STIRcoronal and sagittal: TR-3762 ms, TE-80 ms, TI-150 ms,matrix 240 × 168 and slice thickness—3 mm

To assess the anatomic location and extent of the infection, MRI is helpful. An enhancing soft-tissue mass with an infiltrating pattern can be seen on MRI in patients with SBO, along with bone marrow infiltration (shown by hyperintensity and contrast enhancement at T2-weighted MRI and hypointensity at T1-weighted MRI), effacement of fat planes, intra-cranial and perineural extension, and the involvement of lateral structures (temporomandibular joint, parotid gland). With high signal intensity on diffusion-weighted MRI and low signal intensity on apparent diffusion coefficient (ADC) maps, the inflammatory process typically shows restricted diffusion [9–11].

High index of clinical suspicion—Laboratory findings with an increased erythrocyte sedimentation rate, elevated C-reactive protein levels, and leukocytosis help raise the index of clinical suspicion [12].

Radiologic evidence of infection—The most frequent finding is a poorly defined enhancing mass in the posterior wall of the nasopharynx, with otomastoiditis, bone erosion, and enhancement. Adjacent soft-tissue enhancement is usually observed, without architectural distortion

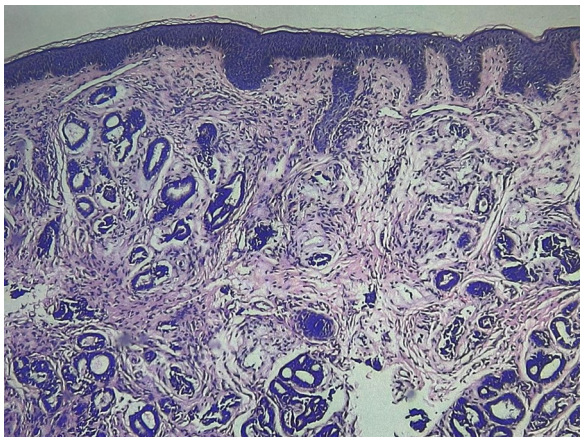

Fig. 5 Histopathology image showing fragments of fibrocollagenous tissue lined by skin with mild basket weave hyperkeratosis, focally flattened rete ridges and attached adnexal structures. The underlying dermis shows an ill-defined infiltrative lesion composed of tumor cells arranged in glandular and cribriform pattern

and with preservation of the smooth nasopharyngeal mucosa.

Multiple biopsies that show no malignancy—Repeated biopsies of the process will rule out malignancy and reveal non-specific inflammatory changes as well as acute or subacute inflammatory granulation tissue [8, 10].

Positive microbiologic test results—It is not always possible to find microorganisms in biopsied soft tissue or bone; however, microbiologic isolation of a causative pathogen enables diagnosis confirmation [10, 13].

Inflammatory pseudotumor is an uncommon disorder with an unknown cause that occurs more frequently in the orbits or lungs than in the skull base. When the lesion occurs in the skull base, it may impact the cranial nerves, akin to SBO. The main finding on CT is an enhancing soft-tissue mass that invades the surrounding structures. The tumor has low signal intensity on T1 and T2 weighted images because of its fibrous nature, and it shows uniform signal enhancement after gadolinium injection. Biopsy serves as the typical diagnostic method, uncovering a chronic inflammatory infiltration containing lymphoid components, encompassing both T and B cells [14–17].

Our examination revealed a lesion affecting the base of the skull and the right ear, causing significant damage to surrounding soft tissues and erosion of nearby bones, resulting in intra-cranial extension and multiple cranial nerve palsies. The lesion showed a mass-like enhancement, disrupting fascial planes with normal levels of white blood cells and ESR, indicating a neoplastic rather than infectious cause. Tissue analysis confirmed the presence of neoplastic nature of lesion, i.e. adenoid cystic carcinoma.

Differential diagnosis of extensive skull base lesions with perineural spread, along with imaging characteristics are outlined in Table 1. Adenoid cystic carcinoma is a malignant neoplasm comprised of epithelial and myoepithelial cells arranged in tubular, cribriform, solid, or combined architectural forms. It primarily arises from the submandibular, sublingual, and minor salivary glands [1]. It is the fourth most frequent malignant salivary gland tumor, accounting for 10% of all such tumors, and a relatively uncommon neoplasm, making up about 1% of all malignant tumors in the head and neck region [18, 19]. It is more common in the fifth and sixth decades of life, with a slightly higher incidence in females than in males [20, 21]. Half of these tumors develop in glandular regions outside of the major salivary glands, mainly in the hard palate. However, they also originate in the tongue and other regions where minor salivary glands are present [18, 22].

Uncommon sites encompass the external auditory canal, nasopharynx, lacrimal glands, breast, vulva, esophagus, cervix, and Cowper glands [23].

It is a slow growing cancer but exhibits high invasiveness and has a notable recurrence rate. While lymphatic spread to nearby lymph nodes is uncommon, hematogenous dissemination frequently occurs throughout the course of the disease [3].

The occurrence rate of carcinoma in the external auditory canal (EAC) is estimated to range from one to six cases per one million individuals [24]. Histologically, the majority of these tumors are squamous cell carcinomas, with the remainder being mucoepidermoid and adenoid cystic carcinomas.

Adenoid cystic carcinoma of external auditory canal seems to originate from the ceruminous glands, sweat glands, or ectopic tissue of salivary glands [25]. In certain instances, the tumor might have originated in the adjacent parotid salivary gland and subsequently spread into the ear canal [26].

Early detection of these tumors is crucial because delaying diagnosis raises the likelihood of distant metastasis [27]. ACC patients are known to have a predisposition for perineural invasion and a propensity for local recurrence [18].

Conclusions

Extensive lesions of the skull base can be of infective, neoplastic and inflammatory etiology. Distinguishing between these conditions is crucial, as they have similar imaging characteristics but require different management approaches.

Table 1 Differential diagnosis for Extensive skull base lesions with perineural spread

Pathology	Adenoid cystic carcinoma	Squamous cell carcinoma	Skull base osteomyelitis	Inflammatory pseudotumor
Age	5th–6th decade	60–69 years	Elderly with diabetes or immune-compromised state	Middle age
Sex	More in females	Male	–	Slight male predilection
CT	Primary lesion presents on cross-sectional imaging as nodular or mass-like enhancement Typically displace or replace normal anatomy without preservation of tissue planes Erosive mass with irregular borders, Bone destruction and invasion of adjacent	Primary lesion presents on cross-sectional imaging as nodular or mass-like enhancement Typically displace or replace normal anatomy without preservation of tissue planes Well-defined mass with smooth margins and Perineural spread along cranial nerves	Enhancement and fullness without destruction of fascial planes Bony destruction and sclerosis with moth-eaten appearance on CT	Soft tissue infiltrating mass with enhancement More common in orbits or lungs than skull base
MRI	T1—isointense to muscle T2—↑↑ signal T1c+—marked enhancement T1 and T1c+—low signal capsule Present	T1—isointense to muscle T2—↑ signal T1c+—enhancement T1 and T1c+—low signal capsule absent	T1—hypo to isointense to muscle T2—↑ signal T1c+—heterogenous enhancement	T1—low signal T2—low signal T1c+—homogenous enhancement

The presence of a lesion that displaces or destroys fascial planes, accompanied by solid mass-like enhancement, indicates a higher probability of a neoplastic origin rather than an infectious etiology. With squamous cell carcinoma being the most common neoplasm, adenoid cystic carcinoma of the skull base also needs to be understood due to its propensity for perineural spread and a high likelihood of recurrence.

Abbreviations

MRI	Magnetic resonance imaging
CT	Computed tomography
SBO	Skull base osteomyelitis
SCC	Squamous cell carcinoma
ACC	Adenoid cystic carcinoma
CECT	Contrast enhanced computed tomography
MRM	Modified radical mastoidectomy

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Author contributions
Dr KM = identification of the imaging findings in CT and diagnosed the condition followed by manuscript preparation. Dr GM = Data collection including image retrieval, histopathological slides and equally participated in manuscript preparation. Dr EP = gave expert opinion on the imaging features, discussed differentials and contributed in manuscript preparation. Dr UA = gave expert opinion on the imaging features, discussed differentials and contributed in manuscript preparation. Dr JV and Dr BM = Helped in refining the manuscript and identified the findings in MRI. All the authors have participated sufficiently in contributing to the content of 'exploring the uncharted: adenoid cystic carcinoma nestled in temporal bone' and have read & approved the manuscript.

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Declarations

Ethical approval and consent to participate
Ethical approval is not obtained, since it is a retrospective case report. Informed written consent had been obtained from the participant for participation and publication of the same as a case report.

Consent for publication
Informed written consent had been obtained from the participant for publication of the same as a case report.

Competing interests
The authors declare no competing interests.

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References

1. Azumi N, Battifora H (1987) The cellular composition of adenoid cystic carcinoma. An immunohistochemical study. Cancer 60(7):1589–1598
2. Michaels L (1987) Adenoid cystic carcinoma. In: Michaels L (ed) Ear, nose and throat histopathology. Springer-Verlag, Heidelberg, pp 183–185
3. Huang M, Ma D, Sun K, Yu G, Guo C, Gao F (1997) Factors influencing survival rate in adenoid cystic carcinoma of the salivary glands. Int J Oral Maxillofac Surg 26(6):435–439
4. Jain N, Jasper A, Vanjare HA, Mannam P, Mani SE (2020) The role of imaging in skull base osteomyelitis—reviewed. Clin Imaging 1(67):62–67
5. Lesser FD, Derbyshire SG, Lewis-Jones H (2015) Can computed tomography and magnetic resonance imaging differentiate between malignant pathology and osteomyelitis in the central skull base? J Laryngol Otol 129(9):852–859
6. Chapman PR, Choudhary G, Singhal A (2021) Skull base osteomyelitis: a comprehensive imaging review. Am J Neuroradiol 42(3):404–413
7. Álvarez Jáñez F, Barriga LQ, Iñigo TR, Roldán LF (2021) Diagnosis of skull base osteomyelitis. Radiographics 41(1):156–174
8. Johnson AK, Batra PS (2014) Central skull base osteomyelitis: an emerging clinical entity. Laryngoscope 124(5):1083–1087
9. Van Kroonenburgh AM, Van der Meer WL, Bothof RJ, Van Tilburg M, Van Tongeren J, Postma AA (2018) Advanced imaging techniques in skull base osteomyelitis due to malignant otitis externa. Curr Radiol Rep 6:1–4
10. Mejzlik J, Cerny M, Zeinerova L, Dedkova J, Kopriva J, Zadrobilek K, Adamkov J, Chrobok V, Pellantova V (2019) The routes of infection spread

in central skull-base osteomyelitis and the diagnostic role of CT and MRI scans. BMC Med Imaging 19:1–1
11. Goh JP, Karandikar A, Loke SC, Tan TY (2017) Skull base osteomyelitis secondary to malignant otitis externa mimicking advanced nasopharyngeal cancer: MR imaging features at initial presentation. Am J Otolaryngol 38(4):466–471
12. Khan M, Quadri S, Kazmi A, Kwatra V, Ramachandran A, Gustin A, Farooqui M, Suriya S, Zafar A (2018) A comprehensive review of skull base osteomyelitis: diagnostic and therapeutic challenges among various presentations. Asian J Neurosurg 13(04):959–970
13. Blyth CC, Gomes L, Sorrell TC, Da Cruz M, Sud A, Chen SA (2011) Skull-base osteomyelitis: fungal versus bacterial infection. Clin Microbiol Infect 17(2):306–311
14. Gasparotti R, Zanetti D, Bolzoni A, Gamba P, Morassi ML, Ungari M (2003) Inflammatory myofibroblastic tumor of the temporal bone. Am J Neuroradiol 24(10):2092–2096
15. Han MH, Chi JG, Kim MS, Chang KH, Kim KH, Yeon KM, Han MC (1996) Fibrosing inflammatory pseudotumors involving the skull base: MR and CT manifestations with histopathologic comparison. Am J Neuroradiol 17(3):515–521
16. Park SB, Lee JH, Weon YC (2009) Imaging findings of head and neck inflammatory pseudotumor. Am J Roentgenol 193(4):1180–1186
17. Patnana M, Sevrukov AB, Elsayes KM, Viswanathan C, Lubner M, Menias CO (2012) Inflammatory pseudotumor: the great mimicker. Am J Roentgenol 198(3):W217–W227
18. Spiro RH, Huvos AG, Strong EW (1974) Adenoid cystic carcinoma of salivary origin: a clinicopathologic study of 242 cases. Am J Surg 128(4):512–520
19. Dillon PM, Chakraborty S, Moskaluk CA, Joshi PJ, Thomas CY (2016) Adenoid cystic carcinoma: a review of recent advances, molecular targets, and clinical trials. Head Neck 38(4):620–627
20. Li N, Xu L, Zhao H, El-Naggar AK, Sturgis EM (2012) A comparison of the demographics, clinical features, and survival of patients with adenoid cystic carcinoma of major and minor salivary glands versus less common sites within the Surveillance, Epidemiology, and End Results registry. Cancer 118(16):3945–3953
21. Jaso J, Malhotra R (2011) Adenoid cystic carcinoma. Arch Pathol Lab Med 135(4):511–515
22. Berdal P, Besche AD, Mylius E (1970) Cylindroma of salivary glands a report of 80 cases. Acta Otolaryngol 69(sup263):170–173
23. Zainor S, Mamat H, Saad SM, Yunus MR (2013) Adenoid cystic carcinoma of external auditory canal: a case report. Egypt. J. Ear Nose Throat Allied Sci 14(1):41–44
24. Barrs DM (2001) Temporal bone carcinoma. Otolaryngol Clin North Am 34:1197–1218
25. Fliss DM, Kraus M, Tovi F (1990) Adenoid cystic carcinoma of the external auditory canal. Ear Nose Throat J 69(9):635–638
26. Szanto PA, Luna MA, Tortoledo ME, White RA (1984) Histologic grading of adenoid cystic carcinoma of the salivary glands. Cancer 54(6):1062–1069
27. Dong F, Gidley PW, Ho T, Luna MA, Ginsberg LE, Sturgis EM (2008) Adenoid cystic carcinoma of the external auditory canal. Laryngoscope 118(9):1591–1596

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