



Case Report

Extradural petromastoid calcifying pseudoneoplasm of the neuraxis (CAPNON): Case report and literature review

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ABSTRACT

Background: Calcifying pseudoneoplasm of the neuraxis (CAPNON) is a very rare tumor with fewer than 70 cases reported in the literature. In general, this tumor occurs intracranially either within the brain parenchyma or in an extra-axial location, but it has also been described within the spine as an extra-axial lesion.

Case Description: We describe an unusual case of intracranial-extradural CAPNON involving the mastoid region. This may be only the second such case reported in the literature, as one patient with CAPNON has been reported involving the sinonasal region. Our patient was managed with surgical resection through a translabyrinthine approach with good early result.

Conclusions: We describe an unusual case of extradural CAPNON involving the mastoid bone. It appears that when located extradurally, this tumor may have a predilection for the bony sinuses. This little-known, generally benign entity can mimic more common lesions such as meningiomas, and should be considered in the differential diagnosis of skull base tumors, particularly when associated with heavy calcification.

1. Introduction

Calcifying pseudoneoplasm of the neuraxis (CAPNON) is a very rare tumor with fewer than 70 cases reported in the literature [1–3]. In general, this tumor occurs intracranially either within the brain parenchyma or in an extra-axial location, but it has also been described within the spine as an extra-axial lesion [1,4]. One exceptional case has been reported involving an extradural lesion of the skull base with anterior fossa sino-nasal extension [5]. We describe a second intracranial-extradural case involving the mastoid region. This little-known, generally benign entity can mimic more common lesions such as meningiomas, and should be considered in the differential diagnosis of skull base tumors, particularly when associated with heavy calcification [6,7].

2. Case description

We describe a 39-year-old woman in good health with no significant previous medical history. She presented with a three-week history of

sudden hearing loss in her right ear with associated intrusive right-sided tinnitus. Audiogram demonstrated a mild-moderate asymmetric high frequency sensorineural hearing loss on her right side with a pure tone average of 22 dB. The patient had normal hearing in her left ear. An MRI of her brain (Fig. 1A–D) revealed subtle contrast enhancement of the dura along and just inferior to the right internal auditory canal (IAC).

A CT scan of the temporal bone (Fig. 1E, F) showed a focal circumscribed lucent abnormality with an internal calcified matrix located in the region of the pars nervosa of the jugular foramen on the right side. The patient was initially treated with steroids, which improved her tinnitus, but symptoms recurred immediately upon cessation of the steroids with no change in her asymmetric high frequency hearing loss.

The patient underwent an infracochlear Giddings-Brackmann approach with open biopsy of the lesion, which did not return a diagnosis. She was managed conservatively for 3 months, but sensorineural hearing loss progressed to severe with decline in pure tone average to 55 dB in the affected ear. Follow-up MRI and CT demonstrated progression of the lesion, which now involved the IAC and cochlea, with

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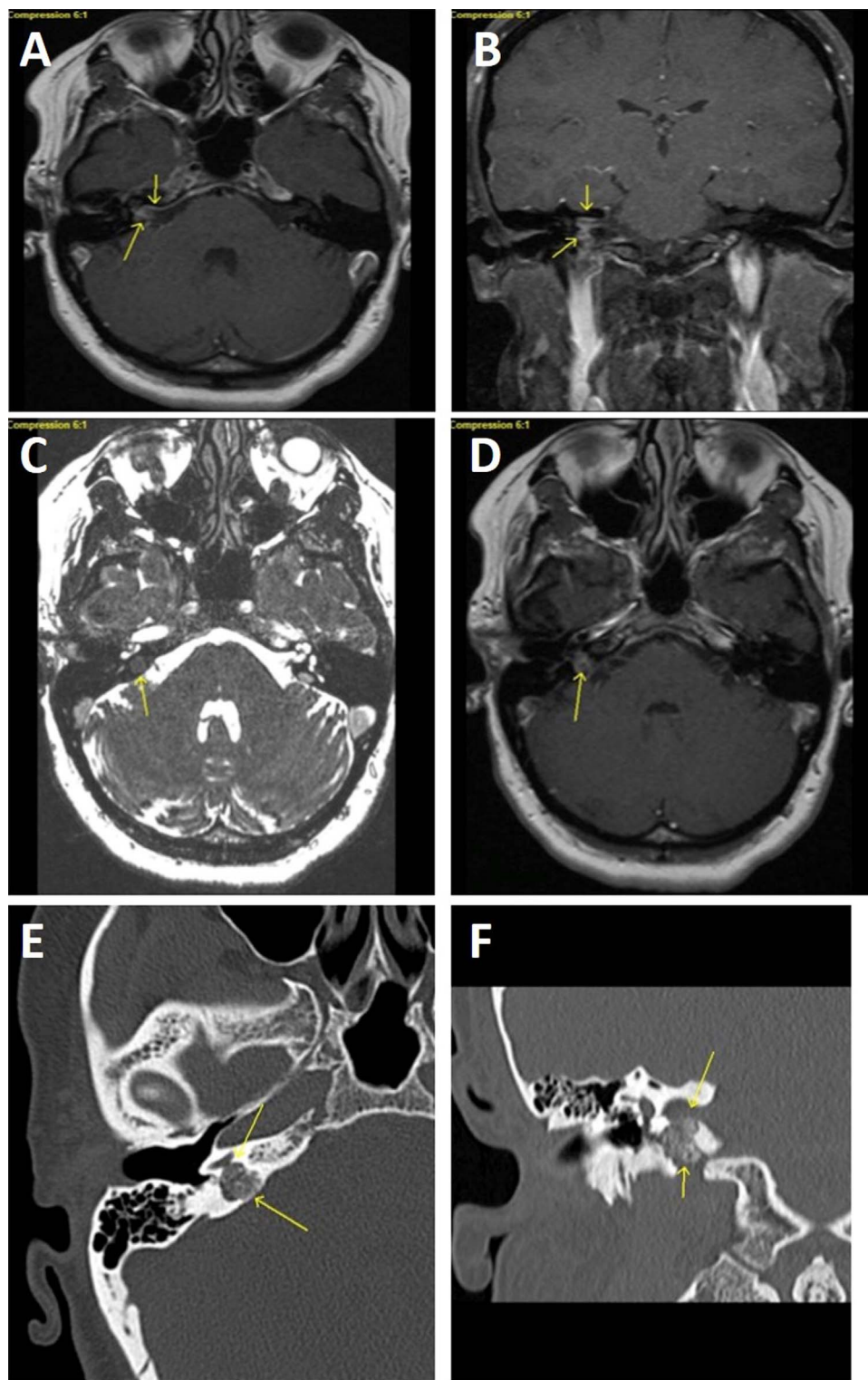


Fig. 1. A: Axial T1 post-contrast image demonstrates linear enhancement along the walls of the right internal auditory canal (arrows). B: Coronal T1 post-contrast image demonstrates linear enhancement along the walls of the right internal auditory canal contiguous with an enhancing lesion just inferiorly in the right petrous apex (arrows). C: Axial FIESTA T2-weighted sequence demonstrates a faintly hyperintense lesion in the right petrous apex adjacent to the basal turn of the cochlea (arrow). D: Axial T1 post-contrast image demonstrates enhancement within the lesion (arrow). E: Axial temporal bone CT image demonstrates a circumscribed lucent lesion with calcified internal matrix in the right petrous apex adjacent to the basal turn of the cochlea (arrow). F: Coronal temporal bone CT image demonstrates the lucent lesion with internal calcified matrix extending from the inferior aspect of the right internal auditory canal down to the pars nervosa of the right jugular bulb (arrows).

new erosion of the carotid canal in the petrous apex. She was offered a translabyrinthine approach for resection based on the location of the tumor inferior to the IAC and superior to the jugular bulb.

2.1. Intraoperative management

At the time of the resection, an extradural lesion involving the mastoid bone was encountered above the jugular bulb and was removed up to the inferior border of the IAC (Fig. 2). The involved bone

was moth-eaten in appearance, gray-white, and softer than normal bone. Postoperatively, the patient’s symptoms of imbalance and vertigo completely resolved; postoperative CT and MRI showed complete surgical resection of the lesion with no residual enhancing tissue; the dural sinuses were patent (Fig. 3). She experienced an expected loss of hearing on the right side due to resection of the labyrinth for exposure of the lesion. She has been fitted with a contralateral routing of sound (CROS) hearing aid.



Fig. 2. Intraoperative photograph of the CAPNON tumor.

2.2. Histology

Cytological tumor evaluation revealed extensive irregular mineralized material with compressed stroma staining for hematoxylin and eosin. Macrophage marker CD68 shows scant staining of stroma and

nonspecific reactivity in the mineralized material. Cytokeratin immune staining was negative. Immune stain for epithelial membrane antigen, which is seen in the majority of meningiomas and in epithelial proliferations, was also negative (Fig. 4).

3. Discussion

CAPNON was first described by Rhodes and Davis [1,6]. Since then, fewer than 70 cases have been reported in the literature [1–3,6]. Our case may be only the second description of this tumor occurring in an extradural location involving the bone of the skull base. A single case reported by Fletcher et al. described bone infiltration and dehiscence of the left ethmoid sinus and the lamina papyracea of the cribriform plate [5]. The lesion in our case also involved the skull base, was located extradurally involving the right petro-mastoid region, and was associated with infiltration and dehiscence of the mastoid sinus. Bone erosion extended to and involved the IAC.

In general, CAPNON tumors are fibrous-osseous lesions occurring in men and women of all ages; however, fifty percent of cases have been reported in patients between 40 and 60 years old, with a slight male predominance. Similarly, these lesions are more common in the brain than the spine. The etiology of CAPNON tumors is uncertain and

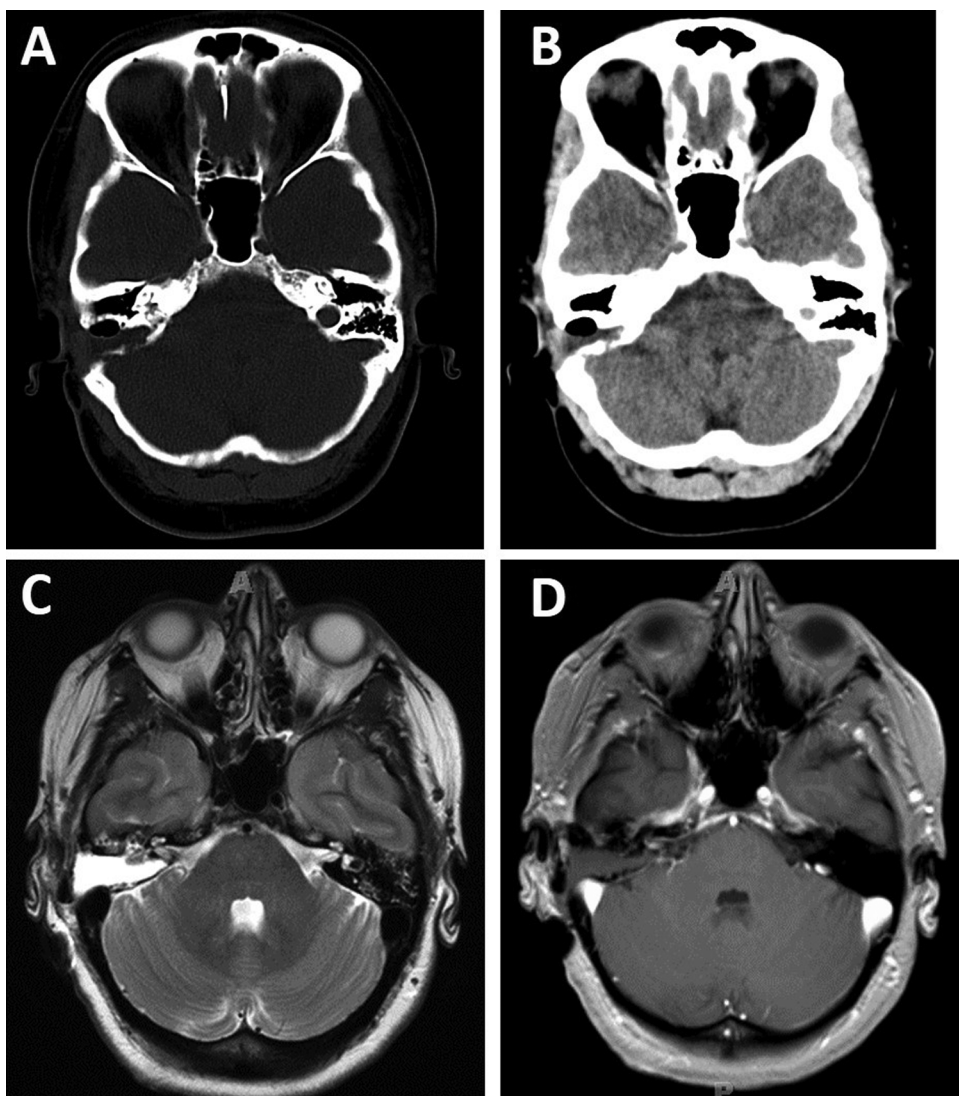


Fig. 3. A and B: Postoperative CT showing changes of mastoidectomy with expected post-operative air-fluid level in the mastoidectomy cavity. There was complete surgical resection of the lesion. C: T2 weighted MRI showing postoperative changes of mastoidectomy with fluid in the resection cavity. D: Post-operative CE-T1 weighted MRI shows no residual enhancing tissue. The dural sinuses are patent.

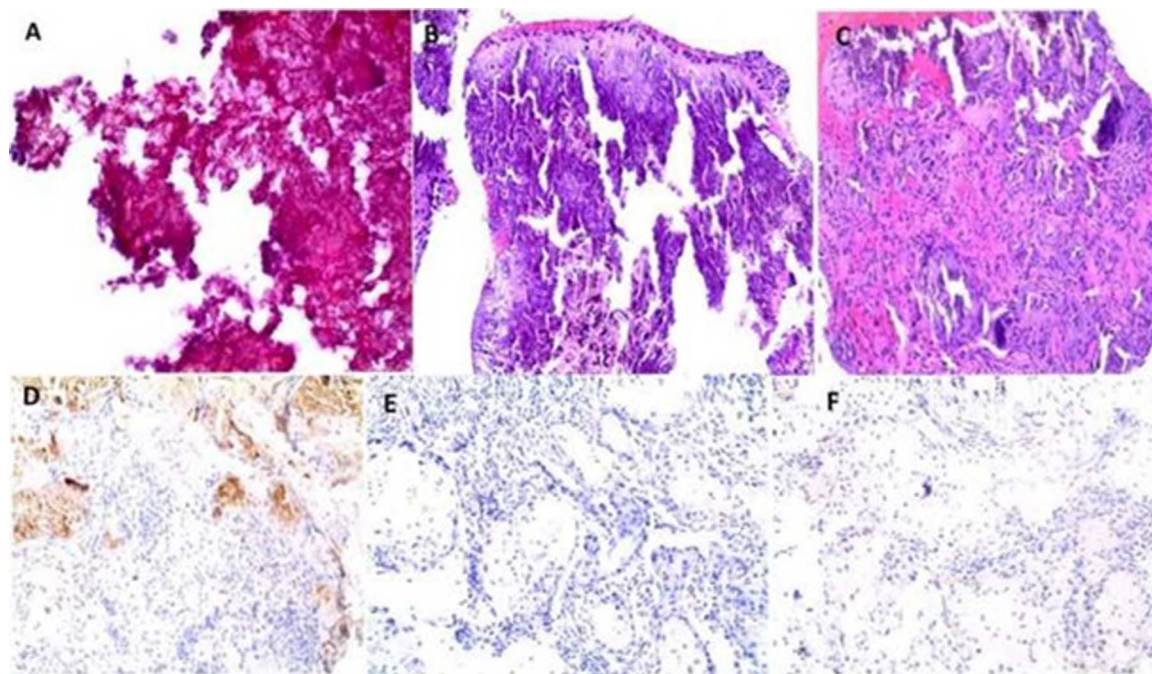


Fig. 4. A: Hematoxylin and eosin cytopology preparation demonstrating irregular, mineralized material, 200× magnification; B: Hematoxylin and eosin histological, 200× magnification, showing compressed stroma; C: Hematoxylin and eosin staining, 200× magnification, demonstrating stroma adjacent to mineralized material; D: CD68 immunohistochemical stain. Macrophage marker CD68 showing scanty staining of stroma and nonspecific reactivity in mineralized material; E: Cytokeratin cocktail, demonstrating negative immunostain proliferation; F: Histological section showing stroma adjacent to mineralized material. Negative immunostain for epithelial membrane antigen, seen in the majority of meningiomas and epithelial proliferations.

possibly various; it might represent a reactive fibro-osseous proliferation or a neoplasm of unclear cellular origin with self-limited growth potential [1]. A review of the literature reveals that, when these lesions occur intracranially, there is a relatively higher incidence of intra-axial lesions with a predilection for the dura at the base of the skull (see Table 1) [1,4,8]. Out of 64 patients with reported data, 27 were female (42.2%), and the average patient age was 45 years (range: 6–74). Common symptoms were seizure (14/64, 21.9%) back, neck, or hip/leg pain (14 patients, 21.9%), and headache (4, 6.2%). Forty-one patients (64.1%) had intracranial lesions, of which 34 were intra-axial (82.9%); 4 were in the craniovertebral junction and 17 were in the spine (26.6%). One lesion from our review was found in the skull base (1.6%) and our lesion was found in the mastoid region (1.6%).

Of these lesions, 34 were completely resected (53.1%), 9 were partially resected (14.1%), 5 were treated medically (7.8%), and 16 were not treated (25%). Complications were rare; apart from autopsy discoveries and unreported outcomes (27 patients, 42.2%), 31 patients had completely resolved symptoms (31/37, 83.8%), while 6 had ongoing symptoms (16.2%). All other symptom, location, treatment, and outcome data are summarized in Table 2.

It seems that the slow-growing, non-neoplastic, non-metastatic, bony-infiltrative characteristics of this tumor can be attributed to its chondromyxoid-like matrix features. Within the matrix, there is a strong fibrous stroma cell structure composed of palisading spindle and epithelioid cells, which is one of CAPNON's histologically distinctive features. Other histological characteristics of CAPNON consist of: fibrous stroma, calcification, osseous metaplasia, scattered psammoma bodies, and foreign body type reaction with giant cells. These histological features are occasionally similar to more common lesions such as meningioma, vestibular schwannoma, chordoma, and chondrosarcoma, among others [4,7].

Radiologically, CAPNON appears as a solid calcified lesion with

mixed low signal intensity on CT scan and uniform hypointensity on T1 and T2 weighted imaging MRI sequences. MRI hypointensity has been described in almost 80% of cases on T1-weighted sequences, and in 90% on T2-weighted images. There are rare reports of isointensity on both T1 and T2 [4,7,8].

From the surgical point of view, gross total resection remains the treatment of choice for this lesion. If complete resection is difficult or dangerous, subtotal resection should be attempted to obtain histopathological diagnosis. Notably, Kwan et al. reported a single case of thoracic CAPNON which resolved both radiographically and symptomatically after treatment with oral non-steroidal anti-inflammatory agents [9]; therefore, despite the failure of steroids in our case, they can be useful as an initial treatment attempt. Currently, there is little evidence of the efficacy of radiation or chemotherapy for treating CAPNON.

The majority of reported cases have a benign course after complete or subtotal surgical excision, although recurrence is possible following partial resection. For example, Bertoni et al. described a case in which a tumor, following partial removal, recurred with multiple areas of bony involvement at the level of the cerebellopontine angle region, jugular foramen, vertebral canal, and oropharynx [7]. Our case suggests that, despite the commonality of sinus lesions, CAPNON can also be found in the mastoid region, and especially if the IAC is involved, a translabyrinthine approach can be used safely and with good outcomes.

4. Conclusions

We describe a rare case of CAPNON involving the extradural mastoid region. This little known, generally benign entity can mimic common skull base lesions and should be considered in the differential diagnosis, particularly when associated with heavy calcification.

Table 1
Patient and tumor characteristics, management, and outcomes based on a literature review of reported cases of CAPNON.

Author/Year	Age/Sex	Symptoms	Location	Radiological features	Management	Outcome
Miller (1922)[10]	–	–	brain	–	–	–
Swartenbroeckx (1962)[11]	–	–	brain	–	–	–
Tiberin and Beller (1963)[12]	40/F	seizure	left temporal area	Skull radiographs demonstrated 7 calcified intracranial bodies	Surgically excised	Significant improvement at 1 year
Case 2	33/F	seizure, chronic discharge from right ear	right posterior temporal region	Radiographs of the skull showed a dense, sharply defined shadow in the right posterior temporal region	Surgically excised	No further seizures
Case 3	42/M	seizure, frontal headache	left temporal region	A radiograph of the skull showed defined ovoid shadow, the size of a plum, in the left temporal region	Further treatment refused, anticonvulsive medication recommended	–
Case 4	45/M	seizure	left, posteriotemporal region	Radiograms of the skull revealed a large calcified body in the left posteriotemporal region	Medically with Luminal Sodium and Mesantoin	Patient did not return for follow-up
Kwan et al. (1976) [9]	48/M	radiculopathy	spine	–	Indomethacin	Complete resolution at 16 weeks
Averback (1977)[13]	30/M	seizure	left frontal region	Opaque mass on SXR	Surgical	Improved
Rhodes and Davis (1978)[6]	27/F	bilateral headaches	right frontal lobe	Calcification was noted overlying the right orbital roof on skull radiograph films.	Surgical	Improved, no recurrence
Case 2	55/F	autopsy finding	brain, dura	–	–	–
Case 3	60/M	autopsy finding	cerebellum	–	–	–
Case 4	74/F	autopsy finding	brain, dura	–	–	–
Case 5	46/M	autopsy finding	4th ventricle	–	–	–
Case 6	6/2/M	autopsy finding	pineal meninges	–	–	–
Case 7	83/M	autopsy finding	brain, dura	–	–	–
Jun and Burdick (1984)[14]	55/M	dizziness, headache, vomiting	corpus callosum	Radiograph and CT showed a calcific mass, 3 cm in	right frontoparietal craniotomy	No recurrence, neurologically intact
Maruki et al. (1984) [15]	43/F	seizure	right temporal lobe	dumb-bell shaped calculus measuring 17 × 10 mm found in right temporal lobe	Surgical excision	No recurrence, no further symptoms
Hashimoto et al. (1986)[16]	29/M	seizure	right temporal lobe	Radiograph and CT demonstrated a calcified mass in the right temporal lobe	Medical initially, followed by anterior temporal lobectomy, excision	–
Nitta et al. (1987) [17]	28/F	vertigo, nausea	cerebellum	CT showed spotty high-density area at the right dentate nucleus of the cerebellum	Surgical	–
Garen et al. (1989) [18]	44/M	facial pain	Meckel's cave	CT scan showed a 1-cm calcified mass impinging on the right trigeminal ganglion	Surgical excision	Complete relief of pain.
Bertoni et al. (1990) [7]	31/M	hoarseness, JF syndrome, hoarseness	GPA, JF, vertebral canal, oropharynx	–	Intralesional excision, debulking procedure	Recurrence at 3 yrs, died of cerebrovascular accident at 13 yrs
Case 2	50/M	neck and occipital area pain	FM	–	3 yrs later for recurrence	No evidence of disease at follow-up
Case 3	48/M	XI CN palsy	cerebellar tonsil	–	Debulking procedure	No evidence of disease at follow-up
Case 4	23/M	back pain	spine, T10	–	Wide excision	Lost to follow-up
Case 5	58/M	back pain	spine, C2–C3	–	Marginal excision	No evidence of disease at follow-up
Case 6	32/M	seizure	frontal	–	Marginal excision	No evidence of disease at follow-up
Case 7	45/F	autopsy finding	skull base	–	Wide excision	No evidence of disease at follow-up
Case 8	58/M	hoarseness, hearing loss	JF	–	–	–
Case 9	12/M	neck pain	spine, C6	–	Intralesional excision	Lost to follow-up
Case 10	32/M	back pain	spine, L4–L5	–	Curettage	No evidence of disease at follow-up
Case 11	33/F	back pain	spine, T9	–	Intralesional excision	Lost to follow-up
Case 12	68/F	hip pain	spine, L4–L5	–	Marginal excision	Degenerative joint disease in lumbar region.

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Table 1 (continued)

Author/Year	Age/Sex	Symptoms	Location	Radiological features	Management	Outcome
Case 13	20/F	incidental finding	spine, C2–C3	–	Curettage	Lost to follow-up
Case 14	56/F	back pain	spine, L4–L5	–	Curettage	Lost to follow-up
Smith et al. (1994) [19]	48/M	hip pain, leg pain	spine, L2–L3	MRI of the spine showed a 7-to-8 mm hypointense lesion in the posterior subarachnoid space at the L2–L3. Radiograph showed a 10-mm calcific density in spinal canal	Surgical excision	–
Tokunaga et al. (1995) [20]	72/F	tinnitus, ataxia	cerebellum, posterior fossa	CT scan revealed a dense bony mass in the posterior calcified lesion in the left superior temporal gyrus with mild mass effect.	Surgical excision	Improvement of symptoms
Qian et al. (1999) [21]	33/F	developmental delay, pituitary dysfunction	temporal	MRI and CT identified a sharply demarcated, calcified mass in the left superior temporal gyrus with mild mass effect.	Surgical excision- left frontal craniotomy	Patient remained well, no recurrence
Case 2	49/M	LE stiffness	upper cervical-clivus	–	Surgical excision	No recurrence
Case 3	59/M	neck pain, shuffling gait	spine, C1–C2	Imaging showed a large extraaxial mass at C1–C2	Surgical excision	No recurrence
Case 4	47/F	seizure	frontal	CT and MRI scans demonstrated an intraaxial calcified mass in parasagittal frontal region	Surgical excision	No recurrence
Shrier et al. (1999) [22]	32/F	incidental finding, pituitary dysfunction	temporal	CT showed an 8-mm calcified mass in the left superior temporal gyrus with mild mass effect.	Surgical excision	No recurrence
Case 2	59/M	neck pain, shuffling gait	FM	Cervical MRI for investigation of cervical spondylosis showed a 2-cm mass giving low signal on T1- and T2-weighted images in the left anterolateral part of the foramen magnum.	Surgical excision	No recurrence, symptom relief
Tsugu et al. (1999) [23]	22/F	seizure	parietal	Skull radiography revealed a round, calcified mass lesion approximately 15 mm in diameter located in the right parietal region.	Surgical excision- right parietal craniotomy	No recurrence, symptom relief
Chang et al. (2000) [24]	60/M	neck pain	spine, C2	Radiographs found a calcifying lesion. A sagittal T1-weighted MRI showed masses with slightly low signal intensity in the body, dens, and laminae of the axis, with multiple foci with dark signal intensity	Laminectomy of the axis and occipitocervical fusion rod system. One month later: curettage of the body and dens of the axis and autogenous iliac bone graft	Neck pain relieved, Local recurrence of the lesion in the axis with progression of preexisting lesion in the facet joints at 24 months.
Mayr et al. (2000) [25]	58/M	LE jerkiness, back pain	spine, T10–T12	MRI revealed a posterior lesion at T10–T12 with low signal attenuation on T1- and T2- weighted images.	A T11–12 laminectomy was performed. Then, intralesional debulking and wall resection was performed	Symptoms relieved, no progression of small, residual mass
Case 2	63/M	gait dysfunction	spine, C3–C4	MRI demonstrated a dorsal lesion at the C3–4 disc space causing cervical stenosis with low signal intensity on short and long repetition-time images. A CT scan revealed a large extradural defect left laterally and posteriorly at C3–4.	Laminectomies were performed from C-2 to C-5. Fibrous tissue in epidural space was filled was resected.	Improvement of symptoms, no recurrence
Albu et al. (2001) [26]	53/F	HA, visual loss	frontal-parietal	CT of the brain revealed multiple circumscribed foci of mineralization over the left frontal and parietal, and the right central brain parenchyma.	–	–
Tatke et al. (2001) [27]	6/M	seizure	temporal	CT showed a hyperdense enhancing irregular mass in the left medial temporal region.	Partial surgical resection.	Symptoms improved, no recurrence.
Ghosal et al. (2007) [28]	26/F	seizure	lateral ventricle	MRI revealed a nodular calcified lesion in the right temporal area.	Right temporal craniotomy with removal of the calcified mass.	No recurrence.
Park et al. (2008) [29]	59/F	neck pain, radiculopathy	spine, C7-T1	MRI showed a C7-T1 extradural mass with an isointense signal to the spinal cord on T1- and T2-weighted sequences.	Laminectomy with gross total resection.	–

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Table 1 (continued)

Author/Year	Age/Sex	Symptoms	Location	Radiological features	Management	Outcome
Rodriguez et al. (2008)[30]	67/F	incidental finding	cerebellum	CT showed an incidental mass lesion in the right cerebellar hemisphere. CT and MRI showed the mass to be cystic, solid, and $2.9 \times 2.0 \times 1.9$ cm.	Surgical resection in right posterior fossa craniectomy.	Patient recovered well.
Aiken et al. (2009) [4]	16/F	incidental finding	temporal, parietal	MR imaging scans demonstrated markedly hypointense signal intensity on both T1- and T2-weighted images, with no vasogenic edema.	Complete surgical resection.	–
Case 2	35/M	seizure	temporal	“	Complete surgical resection.	–
Case 3	49/F	seizure	hippocampus	“	Complete surgical resection.	–
Case 4	59/M	UE numbness	parietal	“	Complete surgical resection.	–
Montibeller et al. (2009)[31]	67/F	dizziness	inferior colliculus	Lesion was isointense on T1-weighted imaging, hypointense on T2-weighted imaging, and homogeneously enhanced with contrast.	Surgical resection via the infratentorial supracerebellar approach	Symptoms resolved, no recurrence.
Mohapatra et al. (2010)[2]	48/M	seizure	temporal	Lesion radiologically mimicked an oligodendroglioma	Complete surgical resection	–
Tong et al. (2010) [32]	67/F	back pain, inability to walk	spine, L4–L5	CT of the lumbosacral spine showed spinal canal stenosis and multiple calcified foci around the left L4–L5 facet joint.	–	–
Hodges et al. (2011) [33]	34/M	HA, dizziness	CPA	CT scans showed a 3.3×3.5 cm, densely calcified posterior fossa mass. MRI revealed enhancing mass measuring $4.3 \times 2.9 \times 2.9$ cm, with the margin tip from the left occipital condyle.	A transcondylar craniotomy approach was used to access and resect the lesion.	Symptoms improved, no recurrence.
Ozdemir et al. (2011) [34]	53/M	facial pain	FM	MRI revealed a calcific mass at the left side of the spinal cord at the level of the foramen magnum.	Median suboccipital craniectomy and total tumor resection.	No additional neurological deficit postoperatively
Kwan et al. (2012) [9]	48/M	left T9 radiculopathy	thoracic spine	calci-fying pseudotumor	Medical- Indomethacin, 25 mg 3 times daily for 8 weeks	Pain relieved at day 3, mass resolved completely at 16 weeks
Stienen et al. (2013) [8]	46/M	partial seizures involving upper left arm	Right parietal lobe	MRI revealed a lesion of the right	Medically with antiepileptic drugs (levetiracetam and lamotrigine), subsequently a near complete lesionectomy was performed; intraoperatively	No new deficits, seizure-free under antiepileptic treatment with levetiracetam and lamotrigine at the 10 months
Case 2	55/F	progressive hallucinosis and behavioral disorders	Left frontal parietal lobe	CT revealed a left hemispheric frontoparietal On MRI rim contrast enhancement and perilesional oedema.	Surgical- subtotal resection of the	Neurological deficits after surgery, completely regressed at 10 and 22 months
Fletcher et al. (2012) [5]	19/M	Nasal discharge, frontal headache with retro-orbital pain, lacrimal drainage, vision loss	anterior skull base, ethmoid sinus, frontal lobe	CT scans and MRI revealed a heterogeneous, 4.5 cm \times 4.8 cm mass	Endoscopic, transthemoidal resection of the lesion	No problems post-surgically, no recurrence
Zerehpoo et al. (2017)[3]	25/M	None, incidental finding	temporal lobe	CT scan revealed a 3 cm calcified ovaloid mass in the left medial temporal region. T1 and T2 MRI showed a hypointense mass heterogeneous enhancement	Mass resection surgery	Uneventful postoperative period, patient well 3 years later
Present Case	39/W	Hearing loss, tinnitus	mastoid region	Contrast enhancement of dura inferior to internal auditory canal on MRI, lucent abnormality on CT	Surgical resection	Expected hearing loss

Table 2
Summary of Symptoms, Locations, Treatments, and Outcomes from CAPNON Literature Search.

Symptoms, n (%)	Location	Treatment	Outcome
Seizure	14 (21.9%) Intracranial, intra-axial	34 (53.1%) Surgical resection	34 (53.1%) Resolved
No symptoms: incidental or autopsy findings	8 (12.5%) Intracranial, extra-axial	7 (10.9%) Partial surgical resection	9 (14.1%) Remaining symptoms
Back pain	6 (9.3%) Spine, intradural	14 (21.9%) Medical	5 (7.8%) Significant remaining symptoms
Neck pain	6 (9.3%) Spine, extradural	3 (4.7%) No treatment, not specified, or Other	16 (25%) Death
Hip or leg pain	2 (3.1%) CVJ	4 (6.2%)	Patient lost to follow-up
Headache	4 (6.2%) Skull base	1 (1.6%)	Not specified
Nausea and vomiting 2 (3.1%)	2 (3.1%) Mastoid region	1 (1.6%)	6 (9.3%)
Dizziness	3 (4.7%)		21 (32.8%)
Shuffling gait	2 (3.1%)		
Hearing loss	3 (4.7%)		
Pituitary dysfunction	2 (3.1%)		
Radiculopathy	3 (4.7%)		
JF syndrome	3 (4.7%)		

Conflicts of Interest

None.

Patient Consent

Patient consent was obtained for the publication of this manuscript.

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