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Calcifying pseudoneoplasms of the neuraxis: Report on four cases and review of the literature

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ABSTRACT

Objectives: Calcifying pseudoneoplasms of the neuraxis (CAPNON) are rare lesions occurring anywhere in the central nervous system (CNS). Since their description, only 55 cases have been reported. We present the largest series reviewing their imaging features, histology and potential origins. Patients and methods: four patients with histopathologically verified CAPNON are presented. Subsequently, we review all reports published with respect to study type, number of patients, clinical presentation, anatomical area (intracranial, spinal, or both), radiological features, therapy, histopathologic features, duration of follow-up, complications, and outcome. Moreover, current management of CNS CAPNON are discussed. Autopsy patients were excluded.
Results: Four patients with histopathologically verified diagnosis of CAPNON are presented between 46–73 years-old. Three of them were located in the spinal cord (levels C3, D2, and L2) and one intracranial (left atrium). The spine ones were diagnosed due to radicular pain, paraparesis and numbness in lower limb, the intracranial because of intense headache. The differential diagnosis included cavernous malformation, in the case of the lumbar CAPNON this suspicion put back the surgery six months. All cases were surgically treated with complete resection. No recurrence showed at the 12-month follow-up. A total of retrospective 30 articles were selected: 10 case series (33.33%) and 20 reports of single cases (66.66%). The 30 articles and our additional cases added up to a total of 27 patients with spinal CAPNON and 32 patients with intracranial CAPNON. All patients were treated surgically. A follow-up, conducted in 48 patients, showed no signs of recurrence in 46 of the 48.
Conclusions: Calcifying pseudoneoplasms are rare benign lesions of yet unknown origin. They should be taken into consideration in the differential diagnosis of calcified lesions because an inaccurate diagnosis can result in potentially harmful and unnecessary therapies, as prognosis for these lesions is generally favorable.

1. Introduction

Q2 Calcifying pseudoneoplasms of the neuraxis (CAPNON) are rare, non-neoplastic, calcified lesions of the central nervous system (CNS). Since the original pathologic description by Rhodes and Davis [1], approximately 55 cases have been reported. Little is known about the aetiology, natural history or course of the disease in surgically treated patients. It is important to recognise this entity, as it is a slow-growing lesion with a good prognosis; doing so will allow avoiding further examinations and inappropriate treatment [2,3]. Although the pathogenesis of this lesion is uncertain, it appears to be reactive rather than neoplastic [4,5]. Q3

We present four additional cases of calcifying pseudoneoplasm, the largest series of non-autopsy cases in the literature, reviewing the imaging features, histology, and potential origins.

1.1. Patients and methods

The case histories of four patients with histopathologically verified diagnoses of CAPNON are presented. Moreover, we reviewed all cases of patients with CAPNON published so far. For this, we searched for the abstracts and titles of all articles in MEDLINE (between 1977 and December 2014) with the following keywords: calcifying pseudoneoplasm; calcifying pseudotumour; and

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fibro-osseous lesion. Only studies and reports about patients with CAPNON in English and Spanish were included; and autopsy reports were excluded. From the qualifying articles; the following parameters were collected: study type; number of patients; anatomical area (intracranial; spinal or both); clinical and radiological presentation; therapy; follow-up; incidence and type of complication and outcome. In the review; intracranial and spinal CAPNON were analysed separately. Furthermore; current recommendations for the management of spinal and intracranial CAPNON are discussed.

1.2. Case report 1

A 48-year-old female patient presented with a 1-year history of progressive, worsening headaches. Computed tomography (CT) scans ordered by her neurologist showed a densely calcified mass in the left atrium. Magnetic resonance imaging (MRI) revealed a hypointensity lesion on both T1- and T2-weighted images, with no vasogenic oedema, indicating a cavernoma (Fig. 1). Given the accessibility of lesion and the possibility of bleeding, we performed a complete resection. The lesion was successfully resected via an occipital craniotomy without any complications. Intraoperatively, the lesion was noted to have a significant calcified component. Pathological examination of the mass revealed a predominantly hypocellular basophilic calcified matrix, surrounded by palisading epithelioid cells. A chondroid matrix was present along with abundant fibrovascular stroma and a focal area of osseous metaplasia. Postoperatively, the patient recovered well without any neurological deficits at the 24-month follow-up.

1.3. Case report 2

A 51-year-old female patient with previous sacroiliitis presented with more than three months' history of lower back pain radiating to both legs. MRI showed a hypointensity lesion on both T1- and T2-weighted images, with no vasogenic oedema in the posterior epidural space at L2. A CT scan was performed and it was confirmed as a calcified lesion. After a laminectomy of L2, a gross total resection was done. The mass was firm and adherent to the dura mater. Histologically, epitheloid cells in a granuloma-like pattern, fibrocellular stroma with spindled fibroblastic cells, and calcified materials were found. These pathological findings confirmed a CAPNON. Postoperatively, back pain and radicular pain were resolved. A follow-up MRI at 12 months after surgical resection confirmed no recurrence, and symptoms continued to be almost completely resolved at that time.

1.4. Case report 3

A 46-year-old female patient presented with a one-year history of posterior neck pain. CT scans of the cervical spine showed a prominent central-to-right side calcified intraosseous mass with a distinctly increased density at the body of C3. Laminectomy of C3 and total removal of the mass were performed. After mass resection, fixation was performed from C2 to C4. Pathological findings indicated a typical chondromyxoid matrix in a nodular pattern with palisading spindles and epithelioid and scattered psammoma bodies, all of them consistent with a CAPNON lesion. Symptoms resolved after surgical resection, and there was no recurrence at 27-month follow-up.

1.5. Case report 4

A 73-year-old male patient with six-month history of progressive paraparesis consulted the department of neurology. A cervicodorsal MRI scan was performed revealing a well-circumscribed intradural extramedullary hypointensity lesion on both T1- and T2-weighted images in the posterior part of T2. In the subsequently performed CT scan, we confirmed the existence of calcium in the lesion. Because of the progressive nature of the symptoms, we performed a T2 laminectomy with a total resection of the lesion. Histopathological examination revealed a chondroid matrix with abundant fibrovascular stroma and a focal area of osseous metaplasia, which confirmed the existence of a CAPNON. Postoperatively, the motor weakness regressed completely and there was no recurrence at 12-month follow-up.

2. Results of literature review

We identified 364 possibly relevant publications. After application of the inclusion criteria, a total of 30 articles were selected: 10 case series (33.33%) and 20 reports of single cases (66.66%). The 30 articles and our additional cases added up to a total of 27 patients with spinal CAPNON and 32 patients with intracranial CAPNON. All patients were treated surgically. A follow-up, conducted in 48 patients, showed no signs of recurrence in 46 of the 48.

Intracranial CAPNONs have been documented in the literature in 32 cases, including those in this paper (Table 1). The ages of the affected individuals show a wide range (from 6-to 67-years-old), and there were 19 male (59.38%) and 13 female patients (40.63%). Twenty cases (62.50%) were located supratentorially, and of these, 8 cases (25%) affected the temporal lobe. The CAPNONs of 12 patients (37.50%) were located in the posterior fossa.

The mode of presentation included epileptic seizures in 9 (28.12%) headache in 9 (28.12%), and cranial nerve affection in 5 (15.62%). Further symptoms reported included vomiting, papilloedema, pituitary dysfunction and developmental delay, dizziness, tinnitus, hallucinosis, decreased hearing, limb paresis, cranial nerve affection, and jugular foramen syndrome. Complete resection of the lesion was possible in 22 of the 32 cases (68.75%). Incomplete resection was performed in ten cases (31.25%). Revision surgery was necessary in one patient with near complete removal of the lesion in the left cerebellopontine angle. The follow-up period was reported in the cases of 22 patients (68.75%), with a median of 57.9 months (range: 2–360 months). At the last follow-up, 21 patients (95.45%) were recurrence-free. The condition was stabilised in the cases of 13 patients who underwent complete resections and in the cases of 8 who underwent incomplete resections, however, one incomplete resection led to disease recurrence and subsequent debulking surgery three years after the initial surgery.

Spinal CAPNONs have been reported in 27 cases: fifteen male (55.55%) and twelve female patients (44.45%) with a mean age of 53.96 years (range: 12–90 years) were available for analysis (Table 2). The most common location of the spinal CAPNONs reported was the cervical spine (9 cases, 33.33%). The spinal CAPNONs were located epidurally in 22 cases (81.48%), intradurally in 4 cases (14.81%), and intraosseously in 2 cases (7.41%). The clinical presentation included diffuse neck or back pain (or both) in 13 cases (48.15%) and muscular paresis in 7 cases (25.92%). Complete excision of the lesion was performed in 13 cases (48.15%); in one patient (3.70%), a single level laminectomy for an epidural CAPNON was performed, but the authors do not specify the degree of resection. The median follow-up for those cases in which one was reported (16 of the 27 cases, 59.25%) was 38.43 months (range: 2–112 months). While 15 of those16 patients were reported recurrence-free (93.75%), one patient with an incomplete excision showed disease recurrence with local progression at the 24-month follow-up.

3. Discussion

CAPNONs are rare lesions: only 59 cases have been reported, and this is the longest series of cases in the literature. Six

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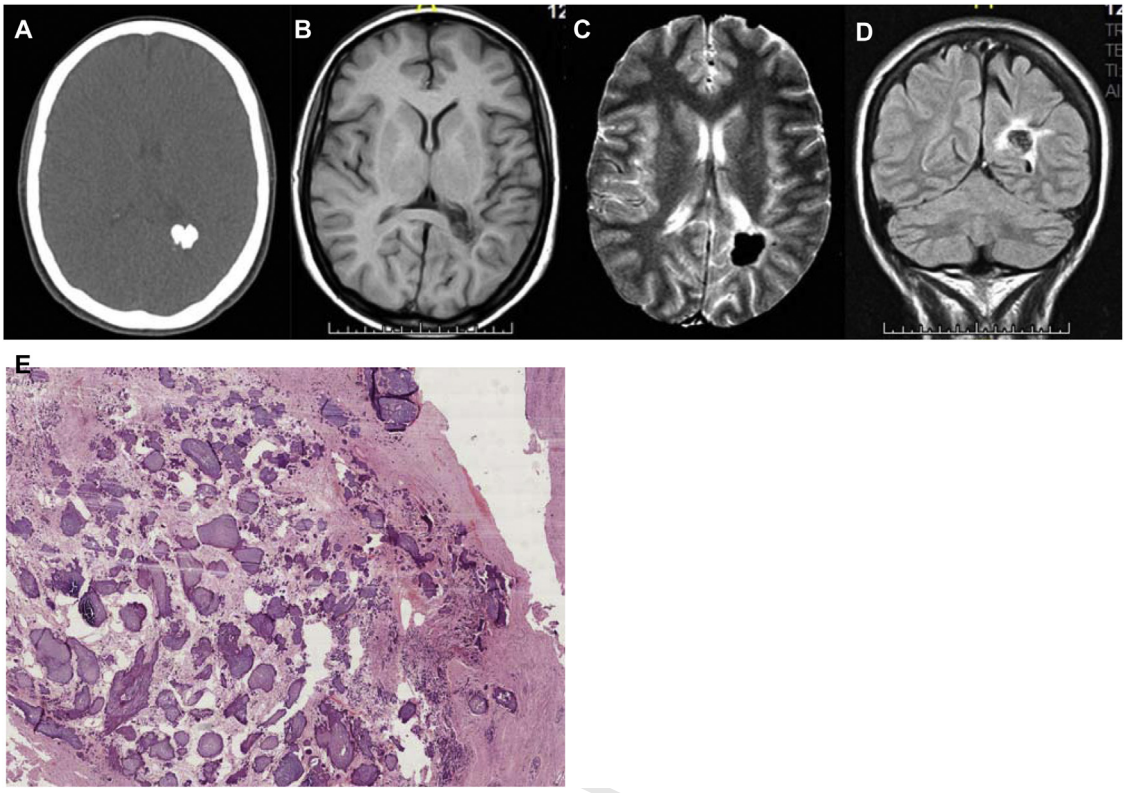

Fig. 1. Imaging studies of the patient presented in case 1. (A) A CT scan brain window image shows a calcified hyperdense lesion in the left atrium. (B) Axial view in T1. (C) Axial view T2-weighted imaging (WI) revealing a hypointense and homogeneous lesion. (D) Coronal view in T1-Flair showing a lack of significant surrounding edema. (E) Amorphous calcifying masses with osseous metaplasia and fibrovascular stroma from the lesion obtained during surgery performed on the patient presented in case 1 (haematoxylin-eosin, original magnification ×100).

Table 1
Q5 Intracranial CAPNON.

Patient no.	Author & year	Age (years)	Sex	Presentation	Localisation	Treatment	Follow-up (months)	Recurrence
1	Present case	48	F	Headache	Left atrium	GT	18	No
2	Kerr, 2011	56	M	Headache	Right cerebellopontine angle	ST	6	No
3	Rhodes and Davis, 1978	27	F	Headache	Right frontal lobe	ST	84	No
4	Jun and Burdick, 1984	55	M	Dizziness and vomiting	Corpus callosum	GT	N/A	No
5	Garen, 1989	44	M	Facial pain	Trigeminal ganglion region	GT	N/A	No
6	Bertoni, 1990	31	M	Jugular foramen syndrome	Left jugular foramen	ST	156	Yes
7	Bertoni, 1990	48	M	Right xi paralysis	Right cerebellar tonsil	GT	228	No
8	Bertoni, 1990	32	M	Seizures	Frontal lobe	GT	360	No
9	Bertoni, 1990	58	M	Decreased hearing	Jugular foramen	ST	N/A	N/a
10	Tsugu, 1999	22	F	Seizures	Right parietal lobe	GT	96	No
11	Shrier, 1999	32	F	Incidental	Left temporal lobe	GT	12	No
12	Qian, 1999	33	F	Developmental delay	Left temporal lobe	GT	31	No
13	Qian, 1999	47	F	Seizures	Parasagittal frontal	GT	72	No
14	Qian, 1999	49	M	Tetraparesis	Clivus	GT	90	No
15	Tatke, 2001	6	M	Seizures	Left temporal medial region	ST	6	No
16	Aiken, 2009	16	M	Incidental	Right temporal horn	GT	N/A	No
17	Aiken, 2009	35	M	Seizures	Right temporal lobe	GT	N/A	N/a
18	Aiken, 2009	49	F	Seizures	Left hippocampus	GT	N/A	N/a
19	Aiken, 2009	59	M	Left arm numbness	Right parietal lobe	GT	N/A	N/a
20	Montibeller, 2009	67	F	Seizures	Right inferior colliculus	GT	18	N/a
21	Mohapatra, 2010	48	M	Seizures	Right temporabasal region	GT	N/A	No
22	Hodges, 2011	36	M	Headache and tinnitus	Left cerebellopontine angle	ST	7	No
23	Stienen, 2011	46	M	Seizures	Right parietal lobe	ST	10	No
24	Stienen, 2011	56	F	Hallucinosis	Left frontoparietal lobe	ST	22	No
25	Muccio, 2012	55	F	Hypoesthesia	Cervicomedullary junction	GT	14	No
26	Nonaka, 2012	56	M	Ear infection	Right temporal lobe	GT	N/A	N/a
27	Nonaka, 2012	35	M	Headache	Left occipital condyle	ST	6	No
28	Grabowski, 2013	49	F	Headache	Pineal region	GT	21	No
29	Fatih, 2014	59	F	Headache	Cerebellomedullary cistern	GT	N/A	N/a
30	Wisniewski, 2015	29	M	Headache	Foramen magnum	GT	2	No
31	Tan, 2016	45	M	Headache and CN VI palsy	Superior medullary velum	GT	3	No
32	Alshareef, 2016	59	F	Gait instability	Cervicomedullary junction	ST	12	No

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Table 2
Q6 Spinal CAPNON.

Number	Authors & year	Age (years)	Sex	Presentation	Location	Treatment	Follow-up (months)	Recurrence
1	Present cases	51	F	Back pain	L2ie	GT	39	No
2		46	F	Neck pain	C3io	GT	27	No
3		73	M	Paraparesis	D2ie	GT	12	No
4	Bertoni, 1990	50	M	Neck pain	FMe	ST	42	No
5	Bertoni, 1990	23	M	Back pain	Th10e	ST	N/A	No
6	Bertoni, 1990	58	M	Paresthesias	C2e	ST	112	No
7	Bertoni, 1990	12	M	Neck pain	C6e	ST	39	No
8	Bertoni, 1990	32	M	Back pain	L4e	ST	83	No
9	Bertoni, 1990	33	F	Back pain	Th9e	ST	N/A	No
10	Bertoni, 1990	68	F	Sciatica	L4e	ST	16	No
11	Bertoni, 1990	20	F	Incidental	C2e	ST	N/A	No
12	Bertoni, 1990	56	F	Back pain	L4e	ST	N/A	No
13	Smith, 1994	48	M	Sciatica	L2e	GT	N/A	No
14	Shrier, 1999	59	M	Tetraparesis	FMe	GT	24	No
15	Qian et al., 1999	59	M	Tetraparesis	C1e	GT	46	No
16	Chang, 2000	60	M	Neck pain	C2io	ST	24	Yes
17	Mayr, 2000	58	M	Back pain	Th10e	ST	48	No
18	Mayr, 2000	63	M	Tetraparesis	C3e	ST	60	No
19	Liccardo, 2003	40	M	Thoracic pain	Th8e	GT	36	No
20	Park, 2008	59	F	Radiculopathy	C7e	GT	N/A	No
21	Tong, 2010	67	F	Neurogenic claudicaction	L4e	Laminectomy	N/A	No
22	Ozdemir, 2011	53	M	Monoparesis MII	FMid	GT	N/A	No
23	Muccio, 2012	57	M	Paraparesis	T10-11e	GT	2	N/a
24	Song, 2015	77	F	Back pain	T12e	GT	5	No
25	Song, 2015	67	F	Radiculopathy	L2-3e	GT	N/a	N/a
26	Song, 2015	78	F	Back pain	L1e	GT	N/a	N/a
27	Singh, 2016	90	F	Lower extremity weakness	C7-D1id	ST	N/a	N/a

FM—foramen magnum; Th—thoracic spinal segment; C—cervical spinal segment; L—lumbar spinal segment; N/A—not available; e—epidural; ie—intradural extramedullary; id—intradural; io—intraosseous; GT—Gross total resection; ST—subtotal resection.
ST—subtotal remove; GT—Gross total remove; N/A—not available.

additional autopsy cases have been described by Rhodes and Davis [1]. There do not seem to be more strongly associated with any particular age or location—in fact, the age range was from 6 to 90 years old; however, a male predominance (1.92:1) was noted [3]. Traditionally most reported intracranial lesions have been extra-axial [6–8], but several intra-axial cases have also been reported and the present literature review revealed a relatively higher incidence of intra-axial lesions (66.3% vs. 33.6%). It may be worthy of note that most of the lesions in the spine were in the epidural space (81.48%). The lesions seem to be slow growing and presenting symptoms tend to be related to local compression or irritation of adjacent tissues. In spinal CAPNONs, the predominant cause of presentation was pain (in 48.15% of the patients). In contrast, seizures with headache (18.15%) were the most common symptom of intracranial CAPNON. In three cases (spinal: one, intracranial: two) CAPNONs were incidental findings. The underlying cause of CAPNON remains unknown, but complete surgical resection seems to be curative [7,9].

CT images of CAPNON typically show solid attenuated calcifications, and MR imaging often shows a well-defined lesion that is uniformly hypointense on both T1-and T2-weighted images without surrounding oedema [7]. None of our cases demonstrated solid enhancement. However, an extra-axial mass presented by Shrier et al. [10] in the foramen magnum did show more solid enhancement, mimicking meningioma. None of our lesions showed significant surrounding oedema [7]. Further investigation would be needed to identify the "typical" enhancement or pattern of oedema in these rare lesions.

The radiological differential diagnosis of these intracranial and intraspinal calcifying lesions can be broad. The primary differential consideration for extra-axial CAPNON at the skull base is meningioma, but some authors have also included chordoma, chondrosarcoma, and vestibular schwannoma. However, the uniform T2 hypointensity without enhancement would be unusual for chordoma, chondrosarcoma, or vestibular schwannoma. Important differential considerations for intra-axial calcified masses include calcifying neoplasms such as ganglioglioma and oligodendroglioma, vascular lesions such as cavernous malformation, and infections such as tuberculosis [9]. Intraventricular masses can also raise the possibility of choroid plexus tumours, meningioma, or ependymal tumours. Among intraspinal lesions, herniated disc fragments, synovial cysts, neurinomas, and psammomatous meningiomas should be primarily considered. In addition, epidural abscesses and old calcified hematomas should also be taken into account [11]. The differential diagnosis can, however, be narrowed on the basis of MR imaging features.

The "classic" histopathological features in CAPNON include a distinctive set of common elements: (1) a typical chondromyxoid matrix in a nodular pattern; (2) palisading spindles to epithelioid cells; (3) variable amounts of fibrous stroma; (4) calcification, osseous metaplasia, and scattered psammoma bodies; and (5) foreign-body reaction with giant cells. The presence of each component is highly variable, and some examples may not show all of the elements [6]. Immunostaining of the surrounding palisading to epitheloid cells reveals reactivity to vimentin and epithelial membrane antigen (EMA), and spindle cells in the fibrous matrix have been found to have immunoreactivity to osteocalcin [7,12]. It has been suggested, but not proven, that CAPNONs may develop as a healing response to an array of inciting factors, which could account for the variations in histopathological features. The causal factors are not yet understood, but response to possible trauma, infection, or inflammation has been proposed [5]. The tissue of origin most likely includes the arachnoid mater or fibroblasts in the choroid plexus stroma, but this has not been conclusively proven either [1].

For the histopathological workup, chordomas, chondroblastomas, chondrosarcomas, and infectious granulomatous diseases are the main histological differential diagnoses. The lobular pattern and the chondromatous appearance are shared elements in CAPNON and chordomas; however, the lack of ribbon-like cells (arranged in an abundant extracellular matrix) and absence of a vacuolated pattern of the cytoplasm make chordomas unlikely. Chondrosarcomas and chondroblastomas may be

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considered because of the cartilaginous aura and because of the calcifications and the epitheloid cells. They can be distinguished by a lack of evidence of nodular or confluent granulomatous configuration. Moreover, the absence of cellular elements in proliferation emphasises the benign nature of CAPNON. Lastly, the absence of lymphocytes and Langhans giant cells rules out tuberculoma and bacterial etiopathogenesis [3,9].

Once the diagnosis of a symptomatic or growing CAPNON is suspected on the basis of clinical presentation and imaging studies, a complete surgical resection should be attempted, if technically feasible, because in two cases of our review recurrence and local progression of the preexisting lesions occurred following partial resection, complicating the postoperative course [13,14]. Calcifying pseudoneoplasms thus bear the potential for slow but progressive growth. Still, there are not yet any recommendations concerning adjuvant therapy or the frequency and length of the radiological follow-up. The closest differential diagnoses are chondroid tumours, both benign and malignant. It is therefore important to recognize this entity, which appears to be a benign non tumour lesion, to avoid inappropriate therapy like the aggressive surgical treatment we would plan for a chordoma: in the case of a CAPNON we can leave a small capsule behind to avoid major injury of cranial nerves or vascular structures because the lack of oncological objective removes the need to be radical. On the basis of the reported slow progression of residual tumour cells, we would propose a follow-up programme analogous to that of patients with a low-grade meningioma [3].

Calcifying pseudoneoplasms are rare benign lesions of the CNS that can occur as either extra-axial or intra-axial masses. Although the cause and pathogenesis are unclear, the histopathological appearance is distinctive along a spectrum, and the imaging features can suggest the diagnosis. These should be taken into consideration in the differential diagnosis of calcified lesions because an inaccurate diagnosis can result in potentially harmful and unnecessary therapies [8,15], as prognosis for these lesions is generally favourable.

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