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Letter to the Editor

Spinal drop metastases of choroid plexus papilloma: a brief report and updated literature review

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Summary
Choroid plexus papillomas (CPPs) are rare central nervous system benign tumours and spinal metastases are even less frequent. Herein, we describe a case of a 51-year-old man with several episodes of loss of consciousness. Brain magnetic resonance imaging (MRI) displayed a fourth ventricle contrast-enhancing lesion that was totally resected, and histopathological findings showed a papillary neoplasm derived from choroid plexus epithelium, with mild pleomorphism and absence of mitotic activity, consistent with CPP. Two months after the first operation, multiple spinal drop metastases at L4, L5 and S1-S2 were revealed through spine MRI. Histological examination of the spinal lesions fulfilled as well the diagnostic criteria for CPP. Radiological and histological findings are presented, and the relevant literature of spinal spreading of CPPs is discussed. This special case, together with the literature review, helps expanding the spectrum of knowledge on benign CPPs, their potential for disease spread and progression and highlights the need to identify biomarkers that correlate with clinical behaviour.

Key words: Choroid plexus papilloma, atypical choroid plexus papilloma, cerebrospinal fluid, metastases, spinal drop, prognosis

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Choroid plexus tumours (CPTs) are extra-axial neoplasms, typically located within the ventricular system where they originate from the neuroepithelial lining of the choroid plexus. Collectively, they comprise a spectrum of three entities with worsening clinical behaviour: choroid plexus papillomas (CPPs), atypical choroid plexus papillomas (aCPPs) and choroid plexus carcinomas (CPCs). According to the 5th Edition of The WHO Classification of Central Nervous System Tumours[1], CPP is a benign neoplasm with very low or absent mitotic activity (WHO grade 1). Although prognosis is generally considered excellent[2,3], even benign CPPs may seed cells into the cerebrospinal fluid (CSF), resulting in drop spinal metastases[4]. The latter event is markedly more common in the malignant counterpart where patients' poor outcome is associated with leptomeningeal seeding, as emerged in a recent case series of CPCs[5]. Although CPPs metastatic potential is considered extremely ra-

re, a growing number of cases of CSF spreading have been described over the last few decades. Herein, we describe spinal drop metastases of CPP occurring upon initial presentation in an adult patient. Clinical, radiological and histological findings are presented, and the relevant literature is discussed.

A 51-year-old man was recently admitted to our Neurosurgical Department after several episodes of loss of consciousness. Brain magnetic resonance imaging (MRI) showed a fourth ventricle contrast-enhancing lesion, and the patient underwent gross total resection (GTR) of the tumour mass. Haematoxylin and eosin (HE) slides revealed a papillary arranged proliferation of cuboidal to columnar monomorphic cells disposed in a single layer and resembling normal choroid plexus. Mild nuclear pleomorphism, absent mitotic activity and lack of additional atypical morphological features were also recorded. Consistently with the morphological features, immunohistochemistry showed positive stains for pancytokeratin AE1/AE3, vimentin, S-100 and focally for CK7. The Ki-67 proliferation index was extremely low (2%). In consideration of the histopathological findings, the tumour was classified as CPP WHO grade 1. After surgery, the patient recovered well and was dismissed without any postoperative neurological deficits. Following the Multidisciplinary Tumour Board discussion of the case, a brain and spine MRI was performed with a staging purpose two months later, revealing three nodular intradural contrast enhancing lesions at the level of the L4, L5 vertebrae and S1-S2 inter-somatic space, respectively. At the preoperative neurological examination, the patient showed no pathological signs or symptoms related to the MRI spinal findings. The patient underwent microsurgery in order to obtain histopathological diagnostic confirmation, with neural decompression through right L4, L5 hemilaminectomy. Histopathological examination of the spinal lesion showed a papillary architecture with slightly increased mitotic activity (1 mitoses/10 HPFs of 0.23 mm2), mild nuclear pleomorphism and focal blurring of the papillary pattern. Immunohistochemical findings corresponded to those of the fourth ventricle lesion. The absence of significant mitotic activity, as well as absence of histological features of anaplasia made the diagnosis consistent with spinal dissemination of the previously known CPP. The patient recovered well, with no postoperative neurological impairment and in consideration of the residual disease, a craniospinal radiation therapy plan was undertaken in order to treat the residual spinal cord lesions. Histopathology and imaging findings of both ventricular and spinal lesions are illustrated in Figure 1.

Spinal drop lesions of CPPs are considered a highly uncommon event. We searched the PubMed and Embase databases for previous reports of CPPs with disseminated spinal drop lesions. Tumour spinal metastases through CSF pathway have been occasionally described; 24 such cases have been reported in the literature[4,6-26]. The median age at diagnosis of the collected series is 38 years (range: 7-74), the median time from the primary tumour to spinal metastases is 3 years (range: 0-19 years), including 9 of 24 with spinal disease upon initial presentation. Therefore, although the spinal spread is considered a rare event in the natural history of the disease, the number of cases described in the literature appears substantial, raising the doubt that spinal spread is actually an underestimated event, and that the natural biology of this tumour is still poorly understood. The full list of previously published cases with detailed clinicopathological features is summarized in Table I. The increased mitotic activity represents the only atypical histological feature independently associated with recurrence, based on a retrospective series of CPP patients[27], and is therefore the main criteria for grading according to the 5th Edition of The WHO Classification of Central Nervous System Tumours[1]. Although the CPTs spectrum of entities is histologically defined, median expression percentages of both the Ki-67 proliferation marker and the p53 tumour suppressor protein raise across the three histological subtypes (from CPP to APP and then CPC)[28], suggesting an ordinal categorisation of increasingly severe CPTs based on immunohistochemical findings. Normal choroid plexus shows a Ki-67 proliferation index of nearly zero, while mean values of 1.3-4.5%, 5.8-9.1%, and 13.4-20.3% have been reported in CPPs, aCPPs and CPCs respectively[28-30]. Prognostic correlations have also been described for various biomarkers, such as S100, transthyretin, and CD44[29], but they did not find satisfactory confirmation as helpful tools in grading CPTs in individual cases to be included as markers of grading. Therefore, a better knowledge of molecular factors involved in CPT biology is needed to develop prognostic biomarkers that are able to identify patients at recurrence risk. Chromosomal imbalances, such as duplications and deletions, have been recognised in CPPs[31]. Chromosomes 7, 12, 15, 17, and 18 were found duplicated in CPP[31]. However, the total number of specific chromosomal gains or losses does not seem to impact on overall survival in CPPs patients[32]. Furthermore, frequent hyperdiploidy in CPPs and aCPPs were detected and the two entities appear very similar in their cytogenetic profiles[32], in contrast to CPC that markedly differs from the CPP/aCPP group, typically showing abundant chromosomal losses[32]. Methylation profiling of choroid plexus tumours provided useful prognostic information in addition to histopathology[33], revealing

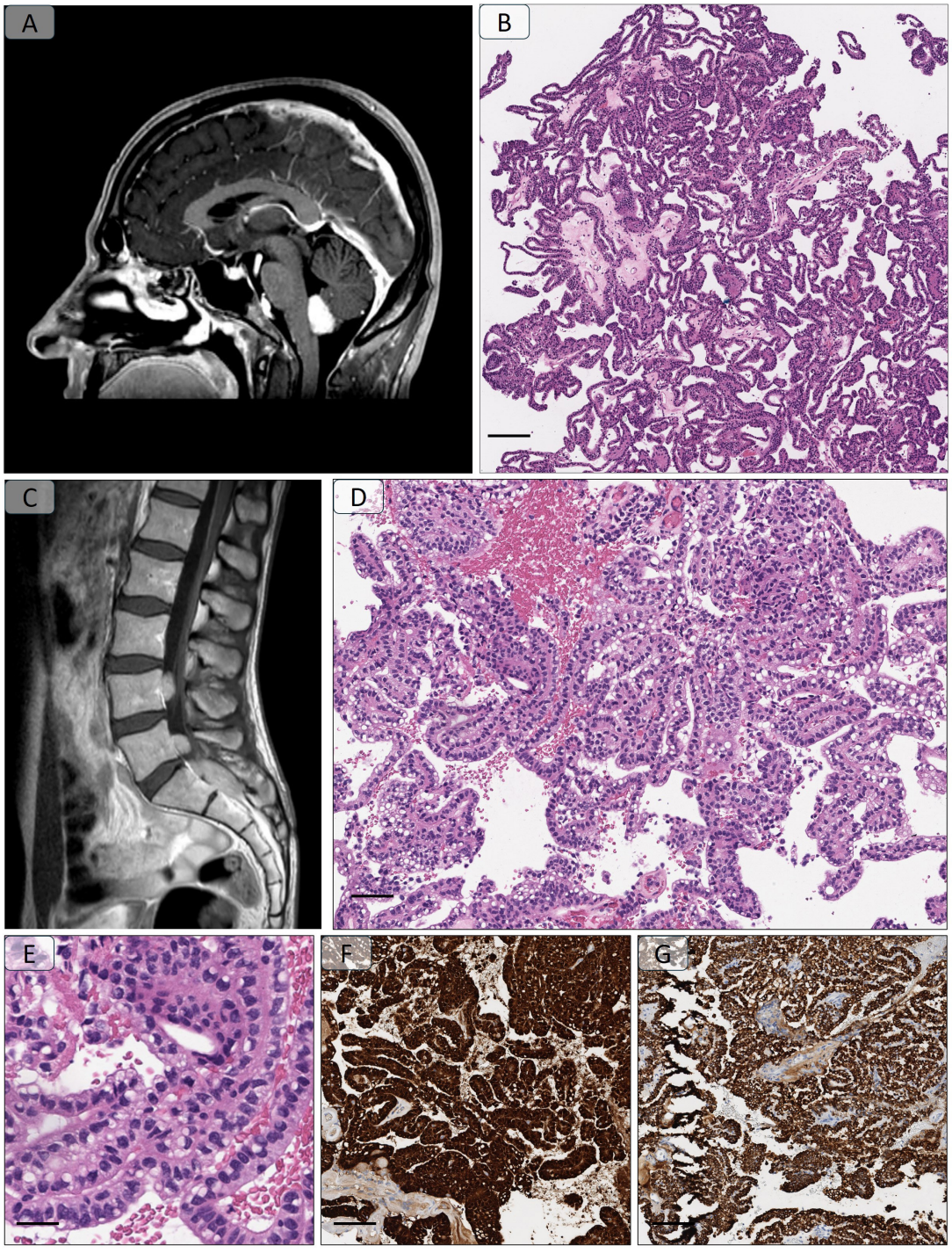

Figure 1. Imaging and histopathological finding of the fourth ventricle and spinal lesion. (A) Sagittal contrast-enhanced T1-weighted image showing lesion obliterating the fourth ventricle. (B) Haematoxylin and eosin slide of the fourth ventricle lesion, showing well differentiated fibrovascular fronds with cells showing round to ovoid, monomorphic nuclei, and absent mitotic activity [magnification 100x, scale bar 200 µm]. (C) Sagittal contrast-enhanced T1-weighted image showing enhancing lesions at L4, L5 and S1-S2. (D - E) Histopathological findings of the spinal lesions: haematoxylin and eosin slide showing a papillary arranged proliferation of cells with pleomorphic nuclei and focal blurring of the papillary pattern [D: magnification 200x, scale bar 100 µm; E: magnification 400x, scale bar 50 µm]. (F) S-100 positive stain [(magnification 200x, scale bar 100 µm]. (G) AE1/AE3 pancytokeratin positive stain [magnification 200x, scale bar 100 µm].

Table I. Full summary of clinicopathological features of reported choroid plexus papilloma cases with disseminated spinal drop lesions.

Author	Year	Age	Sex	PT site	WHO grade (PT)	Surgery (PT)	Spinal MT site	WHO grade (MT)	Surgery (MT)	Time to spinal MT	Adjuvant therapy	FU (time from MT)	Ref.
Masuzawa et al.	1981	7	M	4th ventricle, CPA	1	STR	C6-C7	1	STR	4 years	None	NA	[6]
Domingues et al	1991	35	F	Foramen magnum	1	STR	L3-L4	1	None	Synchronous	None	NA	[7]
Shakespeare et al.	1997	27	F	4th ventricle	1	STR	Multiple	1	None	3 years	CHT	AWD (13 months)	[8]
Leblanc et al.	1998	19	F	4th ventricle	1	GTR	Multiple	1	STR	Synchronous	None	AWD (3 years)	[9]
Jagielski et al.	2001	50	M	3rd, 4th ventricles	1	STR	Multiple	1	STR	3 years	RT	DOD (1 year)	[10]
Heese et al.	2002	38	M	4th ventricle	1	GTR	L1-L2	1	None	Synchronous	None	AWD (8 months)	[11]
McEvoy et al.	2002	51	M	4th ventricle	1	GTR	S1-S2	1	STR	5 years	None	NA	[12]
Doglietto et al.	2005	16	M	Cisterna magna, CPA	1	GTR	L5-S1	1	None	Synchronous	None	NA	[13]
Yu et al.	2006	49	M	4th ventricle	1	GTR	C3-C4, T7	1	STR	19 years	CHT	NA	[14]
McCall et al.	2006	30	F	4th ventricle	1	GTR	C1-C7	1	STR	8 years	CHT	NED (17 months)	[15]
McCall et al.	2006	22	F	4th ventricle	1	GTR	Multiple	1	None	5 years	CHT	AWD (4 years)	[15]
Uff et al.	2006	32	M	4th ventricle	1	GTR	C7-T1	1	STR	3 years	RT	DOD (23 months)	[16]
Kaptanoglu et al.	2007	51	F	4th ventricle	1	GTR	L4-S1	1	STR	7 years	None	AWD (1 year)	[17]
Jinhu et al.	2007	32	F	4th ventricle	1	GTR	T8-T9	1	GTR	1 month	RT	NED (3 months)	[18]
Ahn et al.	2007	42	F	4th ventricle, CPA	1	GTR	L3-S1	1	STR	6 years	CHT	NA	[19]
Akil et al.	2008	74	M	4th ventricle	1	GTR	Multiple	1	None	Synchronous	None	DOD (2 weeks)	[20]
Palmer et al.	2010	74	M	4th ventricle	1	GTR	T5-T8	2	STR	13 years	RT	NA	[4]
Stuivenvolt et al.	2012	50	F	4th ventricle	1	GTR	T9, S1-S2	2	STR	6 years	RT	AWD (15 months)	[21]
Serifoglu et al.	2016	55	M	4th ventricle, cerebellum	1	STR	Multiple	1	STR	Synchronous	CHT + RT	AWD (1 year)	[22]
Morshed et al.	2017	14	M	4th ventricle	1	GTR	S1-S2	1	GTR	Synchronous	None	NED (9 months)	[23]
Ochoa-Cacique	2020	48	M	4th ventricle	1	GTR	L3-L4	1	STR	7 years	RT	NA	[24]
Karthigeyan et al.	2021	38	F	CPA	1	GTR	C2-C6	1	GTR	6 years	RT	AWD (14 months)	[25]
Johnson et al.	2022	40	F	4th ventricle	1	GTR	T8, L2	1	STR	Synchronous	None	AWD (4.5 years)	[26]
Johnson et al.	2022	11	F	4th ventricle	1	GTR	Multiple	1	None	3 years	None	AWD (5 years)	[26]
Present case	2024	51	M	4th ventricle	1	GTR	L4-L5, S1-S2	1	STR	Synchronous	RT	AWD (6 months)	

PT, primary tumour; MT, metastatic tumour; FU, follow-up; WHO, World Health Organization; CPA, cerebellopontine angle; GTR, gross-total resection; STR, sub-total resection; CHT, chemotherapy; RT, radiotherapy; AWD, alive with disease; DOD, dead of disease; NED, not evidence of disease; NA, not available.

3 distinct subgroups: pediatric low-risk choroid plexus tumours (cluster 1), adult low-risk choroid plexus tumours (cluster 2), and pediatric high-risk choroid plexus tumours (cluster 3). The authors highlighted both the opportunities and pitfalls of methylation profiling for CPTs tumours. In their study, a subset of CPPs

and aCPPs that carried an increased risk of recurrence and clustered with CPCs was identified. Multivariate regression models revealed only WHO grade as an independent predictor of overall survival, and only WHO grade and *TP53* status were independent predictors of progression-free survival[33]. Clinical management of CPP patients represents a critical point. The independent prognostic clinical variables for adult patients with CPT include age[34], tumour size[34], extent of resection[34], radio- and chemotherapy[34,35]. Since disseminated CPPs are uncommon, there are no standardised treatment protocols. Many patients receive targeted radiotherapy to the spinal lesions and reports of various chemotherapy regimens are described[21,36]. Furthermore, clinical benefit of bevacizumab has been reported[37]. Some patients remain stable without any adjuvant treatment, making the evaluation of stable disease a demanding task. In conclusion, spinal drop lesions of CPPs are a rare but underestimated event so that a spine MRI may be recommended. Larger case cohorts with complete molecular profiling could further help to clarify pathogenesis and prognosis of CPPs with such unusual clinical behaviour and could represent a useful tool for patient risk stratification and therapeutic management.

Conflicts of interest statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Authors' contributions

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Ethical consideration

The research was conducted ethically, with all study procedures being performed in accordance with the requirements of the World Medical Association's Declaration of Helsinki. Written informed consent was obtained from each participant/patient for study participation and data publication.

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