**Review** paper

Folia Neuropathologica

# Metastases to cranial base meningiomas. Clinical presentations and surgical outcomes. Literature overview

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*Folia Neuropathol 2022; 60 (4): 375-383* *DOI: https://doi.org/10.5114/fn.2022.123500*

**Abstract**

*Tumour-to-meningioma metastasis (TTMM) is an uncommon phenomenon, however repeatedly found in the literature. Meningiomas occur to be the most frequent target of metastatic expansion of systemic cancers. Meningiomas often vary in symptoms and treatment, and this largely depends on the tumour location. Due to their variable locations, they can be classified as convexity meningiomas, which includes falcine and parasagittal tumours, and cranial base, which includes tumours located in the olfactory groove, sphenoid wing, petrous bone and other cranial base locations. The aim of this study was to analyse all data regarding metastases to cranial base meningiomas.*

*We performed a literature search to locate all cases of metastases to cranial base meningiomas in PubMed and Medline databases using the following key words: metastasis to meningioma, meningioma metastasis, and cranial base meningioma. We collected patient and cancer parameters, exact meningioma location and clinical presentations including characteristics which may suggest TTMM.*

*We found 100 articles describing 111 patients of metastasis to cranial base meningioma. Among these articles, 55 cases (49.55%) included metastases to non-skull base meningiomas. In 24 cases (21.62%), the location of meningioma was not precisely described or other data were unavailable, in particular histopathological examination. The most common location of TTMM was sphenoid wing, which was found in 9 patients. The other locations included cerebellopontine angle in 5 patients, and tuberculum sellae in 3 cases. 81.25% cases of TTMM were reported in women, and the most common cancer origins were the breast (28.3%), lung (18.7%), kidney (9.38%) and prostate (9.38%). In two cases the metastatic origin was unclear, and in 15.6% of cases the patients were in remission for more than 1 year. In 78.1% of cases patients presented focal deficits, followed by increased intracranial pressure, and seizures.*

*In almost one-third of cases, TTMM first appeared from a previously unknown cancer. Rapid clinical presentation of cranial nerve palsies may suggest the dual nature of intracranial pathology. The metastasis to cranial base meningioma should be suspected in patients with oncological background, regardless of meningioma parameters or cancer status.*

***Key words:*** *meningioma, carcinoma, tumour to tumour metastases, tumour to meningioma metastasis, meningioma harbouring metastasis, skull base meningiomas.*

## Introduction

Tumour-to-meningioma metastasis (TTMM) is a fairly unusual phenomenon, although the numbers of cases reported in the literature are increasing. Meningiomas occur to be most common intracranial tumours harbouring systemic cancer metastases [28,30]. In order to differentiate tumour-to-tumour metastasis from collision tumours, Campbell *et al.* in 1968 proposed four diagnostic criteria, namely: 1) more than one primary tumour must exist, 2) the host tumour must be a true neoplasm, 3) the metastatic focus must be a true metastasis with established growth in the recipient tumour, not the consequence of contiguous growth or emboli-

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zation of tumour cells, and 4) tumours that have metastasised to the lymphatic system cannot be malignant lymphoreticular tumours. For the purpose of this article, cases of TTMM were identified by criteria of true tumour-to-meningioma metastasis suggested by Pamphlett in 1984, which are as follows: 1) at least partial enclosure of metastasis focus by a rim of benign histologically distinct recipient tumour tissue and 2) the existence of metastasising primary carcinoma must be proven and compatible with a metastasis [7,50].

Meningiomas are common intracranial lesions constituting approximately 37% of central nervous system (CNS) tumours [21,44,48,53]. They are usually benign, indolent and often asymptomatic tumours. However, when symptoms do appear, they are determined by localization of the tumour. Meningiomas can be located in any area of CNS, although most commonly occur as intracranial tumours [27,66]. As intracranial tumours, meningiomas can be classified into skull base meningiomas (SBM) and non-skull base meningiomas (NSBM). We aimed to analyse the literature regarding metastases in SBM. Skull base versus non-skull base meningiomas dichotomy was conducted in accordance with Al-Mefty's definition, [16,44]. Intracranial meningiomas located in areas different than those described below were considered NSBMs (Table I).

## Methods

A literature search was performed in Medline and PubMed databases. We used the terms "metastasis in meningioma", "meningioma metastasis", "cranial base meningioma" and "meningioma harbour cancer" to search published cases. Additional data were identified through a review of references derived from the initial search and we included all articles published up until 2021. Articles in languages other than English, or those with only abstracts available were included when data that were accessible in English included histopathological findings that provided evidence for meningioma harbouring metastasis, and described the location of an intracranial tumour that indicated TTMM in SBM. We included cases in which a systemic cancer was identified, as well as when TTMM was the first evidence of a co-existing neoplasm. Cases of extracranial TTMM were excluded along with metastases in NSBM. Additionally, reports of metastatic meningiomas were also not included. We identified 100 articles reporting 111 cases of metastases to intracranial meningiomas, of which 55 cases described TTMM in NSBM, while in 24 cases, the tumour location was not clearly described or more detailing TTMM features data were unable to be obtained.

**Table I.** Al Mefty classification of skull base meningiomas (SBM)

| |
|---|
| **1. Meningiomas of the anterior cranial base** |
| 1.1. Tuberculum sellae meningiomas |
| 1.2. Olfactory groove meningiomas |
| 1.3. Meningiomas of the orbital roof |
| **2. Meningiomas of the middle cranial base** |
| 2.1. Meningiomas of the lateral and middle sphenoid wing |
| 2.2. Meningiomas of the anterior clinoid |
| 2.3. Meningiomas of the cavernous sinus |
| 2.4. Meningiomas of the optic canal and orbit |
| 2.5. Meningiomas of Meckel's cave |
| 2.6. Cranio-orbital meningiomas |
| 2.7. Meningiomas of the posterior clinoid and upper clivus |
| **3. Meningiomas of the posterior cranial base** |
| 3.1. Clival meningiomas |
| 3.2. Petroclival meningiomas |
| 3.3. Sphenopetroclival meningiomas |
| 3.4. Petrosal meningiomas |
| 3.5. Anterior petrous meningiomas (petrous apex) |
| 3.6. Posterior petrous meningiomas (cerebellopontine angle) |
| 3.7. Jugular foramen meningiomas |
| 3.8. Tentorial meningiomas |
| 3.9. Meningiomas of the temporal bone |
| 3.10. Foramen magnum meningiomas |

## Results

### Patient baseline characteristics

The overall number of cases of metastases in SBM was 32, including 26 females (81.25%) and 6 males (18.75%). Female-to-male ratio was 4.3 : 1, which is more noticeable as opposed to meningiomas in general (2.2 : 1) [48]. The age (mean ± standard deviation [SD]) was 62.81 ±11.47 years. There were no cases reported under 30 years of age. Only one case (3.1%) was reported between 30-40 years of age. Four cases (12.5%) were between 40 and 50 years, and six cases (18.8%) were between 50 and 60 years of age. The majority of reported cases (43.8%) were between 60 and 70 years. Five cases (15.6%) were between 70 and 80 years of age and two cases (6.2%) were over 80 years of age (Fig. 1).

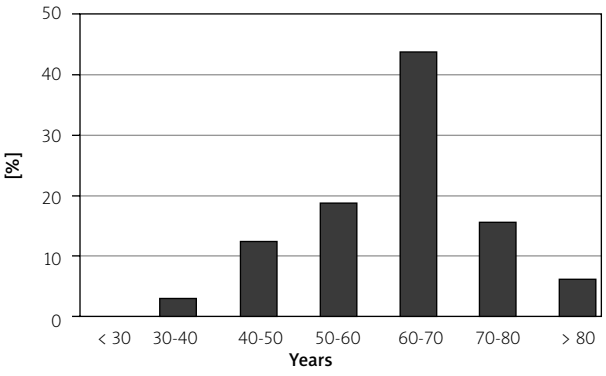


**Fig. 1.** Age distribution among patients affected by the metastasis to cranial base meningioma.

## Origin of metastasis and cancer status

In most cases, the origin of metastatic neoplasms was breast cancer ($n$ = 9, 28.13%) and lung cancer ($n$ = 6, 18.7%). In three cases (9.38%) the primary tumour was renal cell carcinoma (RCC), and prostate cancer. Two cases had no clear metastatic source, although lung and gall bladder carcinoma were suspected as donor tumours. In 9 cases there were several metastatic origins, which were as follows: thyroid follicular carcinoma, rectal neuroendocrine carcinoma, upper gastric body adenocarcinoma, malignant melanoma, olfactory neuroblastoma, hepatic sarcoma, neuroendocrine cancer of the lung, signet ring cell carcinoma from gastro-oesophageal junction and gall bladder adenocarcinoma (Fig. 2).

In almost all cases (96.9%), haematogenic spread of metastases could be presumed. In one case there was metastasis of a skull base cancer, an olfactory neuroblastoma. In nine cases (28.1%) TTMM occurrence was prior to the diagnosis of systemic cancer. In 17 cases (53.1%), a previous cancer was known and treated prior to clinical presentation of an intracranial mass. In five cases, intracranial pathology was the original diagnosis, among them there were two cases (6.2%) of meningioma presence confirmed by histopathological examination, which preceded systemic cancer or TTMM appearance, and in these two cases TTMM was diagnosed in recurrence tumours; both of them were sphenoid wing meningiomas. In six cases (18.7%) TTMM was discovered in the course of post-mortem examination.

In most cases of previously known systemic cancer ($n$ = 15, 88.2%), the time interval from cancer diagnosis to meningioma harbouring metastasis diagnosis had a median of 48 months in length; the longest interval described was 10 years, and the shortest 6 months. In all cases of earlier occurrence of an intracranial tumour, we could determine the time period from original diagnosis to TTMM appearance. The maximum time duration was 13 years, and the minimum – 3 years. The median time interval occurred to be 48 months. In all cases of firstly known meningioma the time between primary diagnosis and metastasis in meningioma manifestation was 36 months.

## Presenting symptoms

For thirty two patients, 30 patients presented neurological symptoms. Most patients developed more than one of the following neurological deficits like visual deficits ($n$ = 11), motor deficit ($n$ = 5), cranial nerves palsy ($n$ = 6), dysphasia/aphasia ($n$ = 5), decreased level of consciousness ($n$ = 4), sensory disturbance ($n$ = 3) and hearing loss ($n$ = 2). 12 patients presented elevated intracranial pressure (ICP) symptoms, e.g. headaches, visual loss; eight of them along with neurological deficits. Only one case included seizures among others symptoms. In accordance with Chen *et al.* and Meling *et al.*, preoperative incidence of neurological deficits is higher in SBM with a lower prevalence of seizures [11,44]. Four patients demonstrated systemic symptoms such as weight loss, fatigue, syncope and others.

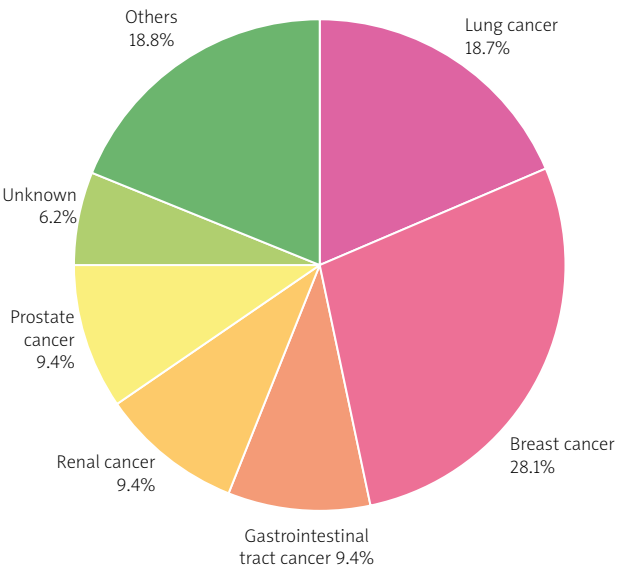


**Fig. 2.** Origins of metastatic tumours found in cranial base meningiomas.

## Characteristics of operated meningiomas

Twenty six patients (81.25%) underwent surgical resection of an intracranial mass. Among them the most common operated tumours were sphenoid wing meningiomas ($n$ = 9, 34.6%), followed by cerebellopontine angle meningiomas ($n$ = 3, 11.5%). In seven cases (26.9%), the location was specified as the floor of the anterior fossa/anterior skull base mass without a more sufficient description. In the remaining 7 cases, there were seven differing meningioma locations (Fig. 3). In comparison to meningiomas in general, the most common SBM locations are the anterior cerebral fossa and sphenoid wing meningiomas [33,44]. The clinical characteristics in patients with TTMM are presented in Table II. This table includes detailed information concerning individual patients on sex, age at presentation, tumour-to-meningioma location, cancer status, clinical outcome with follow-up as well the histopathology of the tumours.

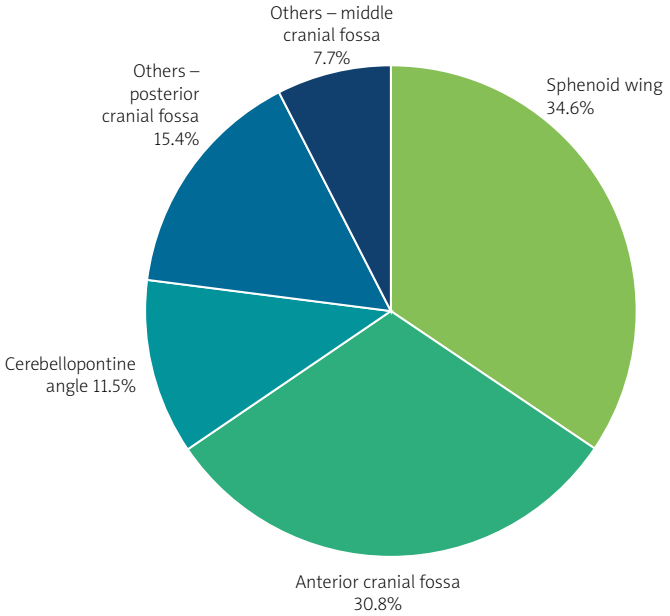


**Fig. 3.** Locations of cranial base meningiomas invaded by metastatic tumours.

Median patient age at surgery was 61.5 years. In comparison, in meningiomas in general, one study stated median age at surgery with 54.2 years [44].

The extent of resection (EOR) was established from the surgery descriptions in 12 cases. Gross-total resection (GTR) was reported in 6 cases. Among them, in only two cases EOR was described in accordance with Simpson resection grades. The other six cases reported a tumour resection defined as subtotal ($n$ = 4) or partial ($n$ = 2). Two reports did not include any description of the extent of tumour resection.

Fifteen case reports included follow-up times, with a mean of 10.4 months. The majority of known cases of operated tumours reported follow-up time over six months ($n$ = 8, 53.3%) including 6 cases with over 1 year in remission. Nonetheless, none of the above case reports described follow-up protocols, thus a maximum follow-up time length for patients who did not develop clinical nor radiological signs of recurrence/progression was not presented.

## Histopathological findings

In all 32 cases, histopathological examination findings were described and led to final diagnoses of TTMM. In 6 cases, TTMM was diagnosed during autopsy. Histological grading for meningiomas was based on the 2016 World Health Organization (WHO) classification [42]. In 14 cases we could ascertain a histological grade. Nine of them reported WHO G I and five of them described meningiomas subtypes as meningothelial ($n$ = 3) and transitional ($n$ = 2); both of them were established as WHO G I in accordance with the WHO classification. According to Meling *et al.*, SBMs have a lower risk of higher histological WHO grades than NSBMs [44]. As an example we have presented the typical histopathology of TTMM in Figures 4 and 5.

## Discussion

In this study, all cases of metastases to SBMs published up until 2021 were investigated to identify differences between TTMM and SBM group in general.

Tumour-to-meningioma metastasis appeared to be less frequent in SBMs than NSBMs. In several studies, in general NSBMs were more common than SBMs [3,9,35,36,40,44].

There are similarities between patient characteristics in TTMM in SBMs and in meningiomas generally: female preponderance and mean age of 63 years in meningiomas harbouring metastasis vs. 66 years in meningiomas [11]. Furthermore, we found the most common donor tumours, being breast and lung carcinoma, are also the ones that were presented with overall intra-meningiomas metastasis [65]. There is a strong association between meningioma and breast cancer, described for the first time by Schoenberg *et al.* in 1975 [63]. Both entities appear more commonly in the 5$^{th}$ and 6$^{th}$ decades of life and both tumours display accelerated growth during pregnancy [21,60,64]. Evidence suggests contribution of sex hormones in meningioma biology, which includes expression of estrogenic and progesterone receptors in meningiomas, as well as protective effects of breastfeeding [13,21,67].

In TTMM presented symptoms were as follows: focal neurological signs, symptoms of elevated ICP, systemic cancer signs and seizures. Pre-operative seizures are less common in SBMs, which are more likely to be associated with neurological deficits. In comparison to overall TTMM cases seizures were more frequent than increased ICP [11,44,63].

In fourteen (53.8%) of 26 operated tumours, the histological grading was determined as WHO grade I meningiomas. In the remaining 12 cases, histological grades of meningiomas were not described. In most cases of SBMs histological WHO grade I was stated with 95.2% and occur to be more common than in NSBMs [6]. In TTMM in general WHO grade I appeared in over 90% of cases [62].

Postoperative oncological treatment depended on the type of malignant tumour, type of spread and of course general and oncological state of a patient. Some patients died before starting any oncological treatment, radiotherapy or chemotherapy [12,17,22,28,49,59].

In most presented cases, over 95% of metastatic tumour tissue was found in benign meningiomas WHO grade I, some in grade II. It is very important to point

**Table II.** Clinical characteristics of patients with tumour-to-meningioma metastasis (TTMM)

| Authors and year of publication | Number of individuals | Sex | Age | Meningioma location | Cancer status | Clinical outcome | Follow-up | Metastatic histopathology | Meningioma histopathology |
|---|---|---|---|---|---|---|---|---|---|
| Honma *et al*. [30] 1989 | 1 | F | 74 | Posterior fossa CPA | Known | Death | 3 months | Stomach adenocarcinoma | Transitional meningioma |
| Arnold *et al*. [2] 1995 | 1 | F | 71 | Orbit | Known | Death | 1 year | Lung cancer | Optic nerve sheath meningioma |
| Bhojwani *et al*. [5] 2016 | 1 | F | 65 | Middle fossa Meckel's cavity | Known | Good | N/A | Rectal carcinoid | Meningioma subtype N/A |
| Chaturvedi *et al*. [10] 2010 | 1 | F | 45 | Parasellar | Known | Good | 1 month | Thyroid ca | Transitional meningioma |
| Chou *et al*. [12] 1992 | 1 | F | 50 | Petrous apex | Known | Good | N/A | Breast cancer | Meningioma subtype N/A |
| Dadlani *et al*. [14] 2013 | 1 | F | 60 | Tuberculum sellae | Known | N/A | N/A | Breast cancer | Psammomatous meningioma |
| Neville *et al*. [47] 2016 | 1 | M | 68 | Sphenoid wing | Unknown | Good | 2 months | Prostatic cancer | Transitional meningioma |
| Eren *et al*. [20] 2019 | 1 | M | 72 | Sphenoid wing | Unknown | Death | 3 weeks | Gallbladder carcinoma | Meningothelial meningioma |
| Farrag *et al*. [22] 2018 | 1 | F | 57 | Sphenoid wing | Unknown | Good | 3 years | Breast cancer | Transitional meningioma |
| Hamperl *et al*. [28] 2015 | 1 | F | 69 | Sphenoid wing | Known | Good | 2 weeks | Non-small cell carcinoma | Endothelial meningioma |
| How *et al*. [32] 2015 | 1 | F | 66 | Sphenoid wing | Unknown | Death | 4 months | Stomach adenocarcinoma | Meningothelial meningioma |
| Hope and Symon [31] 1978 | 1 | F | 61 | Sphenoid wing | Known | Death | 24 hours | Large cell adenocarcinoma | Meningioma subtype N/A |
| Dimou *et al*. [17] 2011 | 1 | M | 62 | Sphenoid wing | Known | Good | N/A | Olfactory neuroblastoma | Meningioma subtype N/A |
| Savoiardo and Lodrini [59] 1980 | 1 | F | 53 | Planum sphenoidale | Known | Good | 1 year | Breast cancer | Endothelial meningioma |
| Breadmore *et al*. [6] 1994 | 1 | F | 82 | Convexity | Known | Poor | N/A | Renal clear cell cancer | Anaplastic meningioma |
| Klotz *et al*. [37] 2018 | 1 | F | 33 | Parasellar | Known | Death | 1 week | Breast cancer | Meningothelial meningioma |
| Lanotte *et al*. [38] 2009 | 1 | F | 64 | Sphenoid wing | Unknown | Death | 20 months | Breast cancer | Meningothelial meningioma |
| | 1 | M | 61 | Foramen magnum | Known | Good | > 6 months | Renal clear cell cancer | Meningothelial meningioma |
| Lim *et al*. [39] 2013 | 1 | M | 68 | Parasellar | Known | Good | 18 months | Prostatic cancer | Meningothelial meningioma |
| Lodrini and Savoiardo [41] 1981 | 1 | F | 53 | Planum sphenoidale | Known | Good | 1 year | Breast cancer | Endothelial meningioma |
| | 1 | M | 59 | Parasagittal | Unknown | Death | 2 months | Pancreatic carcinoma | Fibroblastic meningioma |
| Pal *et al*. [49] 2010 | 1 | F | 53 | Sphenoid wing | Unknown | Good | N/A | Melanoma malignum | Fibroblastic meningioma |
| Peison and Feigin [51] 1961 | 1 | F | 74 | Parasellar | Unknown | Death | Autopsy | Carcinoma N/A subtype | Meningothelial meningioma |
| Roy *et al*. [57] 2019 | 1 | F | 95 | CPA | Unknown | Death | Autopsy | Primary hepatic angiosarcoma | Meningothelial meningioma |
| Fernandez *et al*. [23] 2020 | 1 | F | 56 | Sphenoid wing | Known | Death | 3.5 years | Breast cancer | Meningioma subtype N/A |
| Weems *et al*. [66] 1977 | 1 | F | 68 | Sphenoid wing | Known | Death | 40 days | Lung cancer | Meningioma subtype N/A |

**Table II.** Cont.

| Authors and year of publication | Number of individuals | Sex | Age | Meningioma location | Cancer status | Clinical outcome | Follow-up | Metastatic histopathology | Meningioma histopathology |
|---|---|---|---|---|---|---|---|---|---|
| Mansour *et al.* [46] 2020 | 1 | F | 62 | CPA | Unknown | Death | 6 months | Lung cancer | Meningioma subtype N/A |
| | 1 | F | 64 | Petroclival | Known | Death | 6 months | Lung cancer | Meningioma subtype N/A |
| Döring [18] 1975 | 1 | M | 73 | Posterior fossa CPA | Known | Death | 10 days | Prostatic cancer | Endothelial meningioma |
| Han *et al.* [29] 2000 | 1 | F | 67 | Tentorium | Known | Death | 3 months | Renal clear cell cancer | Meningioma subtype N/A |
| Best [4] 1963 | 1 | M | 48 | Sphenoid wing | Known | Death | 6 weeks | Lung cancer | Meningioma subtype N/A |
| Sayegh *et al.* [61] 2014 | 1 | F | 50 | Planum sphenoidale | Known | Good | > 6 months | Breast cancer | Meningioma subtype N/A |
| Rzehak *et al.* [58] 2021 | 1 | F | 66 | Sphenoid wing | Unknown | Death | 4 weeks COVID+ | Neuroendocrine cancer | Meningothelial meningioma |

*CPA – cerebello-pontine angle, N/A – not applicable*

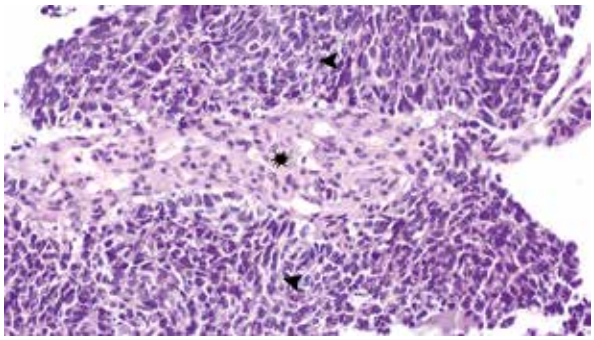

**Fig. 4.** Metastasis of neuroendocrine carcinoma (^) and meningioma cells with intranuclear inclusions surrounded by marginated chromatin (*), HE.

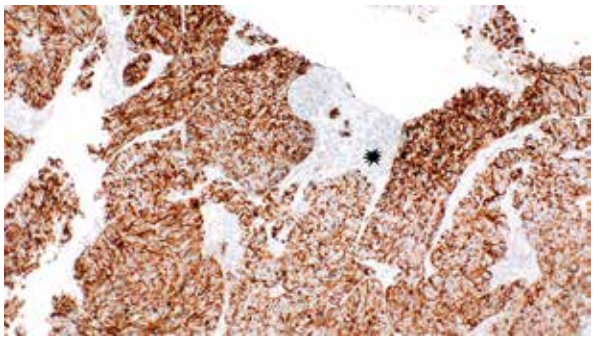

**Fig. 5.** Neuroendocrine carcinoma metastasis immunopositive (^) and meningioma negative reaction for CK AE1/3 (*).

out that prognosis for life in this very special group of patients depends on the spread of the primary malignant tumour. That is why the overall results of treatment were bad or even fatal within first weeks or months after surgery or just after diagnosis was established. In 2 cases, the patient's neurological and general health status deteriorated very rapidly and diagnosis was based on autopsy only [51,57].

Several pathophysiological theories were presented to explain mechanisms of TTMM development. Some characteristics of meningiomas found them as favourable environment to metastasis cell proliferation. Rich tumour vascularization permit haematogenic spread of metastasis [8,25,60]. Indolent growth of meningiomas increases exposure to donor tumour cells and low metabolic rate ensures non-competitive environment for metastasis cell growth. Additionally, high lipid and collagen content provide fertile environment for metastatic cell proliferation [8,19,25,60]. Cell adhesion molecules, particularly E-cadherin, are also correlated to TTMM occurrence. According to Aghi *et al.*, meningiomas harbouring metastasis had a higher rate of

E-cadherin expression in comparison to meningiomas in general, which indicates why meningiomas may be susceptible to metastatic spread [1].

## Conclusions

Although tumour-to-meningioma metastasis is an infrequent phenomenon and was first reported by Fried in 1930 [24], it should be taken in consideration as plausible diagnosis in patients with known systemic cancer, who developed new-onset focal neurological symptoms, especially women in the 5$^{th}$ or 6$^{th}$ decade, with known breast or lung cancer; in newly diagnosed meningiomas with rapid tumour size progression or early symptom occurrence. It is very important to point out that prognosis for life in this very special group of patients depends on malignancy of TTMM. That is why the overall results of treatment were bad or even fatal within first weeks or months after surgery or just after diagnosis in some reported cases [51,57].

Pre-operative TTMM diagnosis with conventional neuroimaging techniques is challenging and in most cases inconclusive, due to lack of characteristic features of these lesions. Some researchers suggested physiology-based neuroimaging techniques such as magnetic resonance spectroscopy (MRS) and perfusion magnetic resonance imaging (pMRI). MRS can provide metabolic ratios that separate meningiomas from other neoplasms, while pMRI is used to distinguish tissue types, due to haemodynamic differences in microvasculature [8,34,52].

It is unclear whether management of TTMM should be different than that for meningiomas in general. Some studies have suggested the necessity of a modified approach, specifically *en bloc* tumour removal, to prevent intra-surgical systemic spread of cancer cells [60]. More data such as histological WHO grades of meningiomas, a follow-up time longer than one year and overall survival are needed to examine more detailed and unified management of this phenomenon.

## Disclosure

The authors report no conflict of interest.

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