



Review

Systemic Tumour Metastasis to Cerebral Meningioma: Literature Review with a Case Report

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Summary

"Tumour-to-tumour metastasis" (TTM) refers to the metastatic deposition from a primary neoplasm to another type of neoplasm within the same patient. While meningiomas have been reported to be the most common intracranial host of malignant metastases, breast and lung carcinomas have been the most frequently reported systemic donor tumours. In the relevant literature, some of the characteristics of meningiomas have been suggested to provide a favourable environment for the metastasis. Furthermore, some factors, such as immunological and hormonal factors, that are thought to make significant contribution to this event prompt research interest. Physiology-based neuroimaging methods, such as perfusion MRI (p-MRI) and proton spectroscopic MRI (proton s-MRI) could be efficient in differentiating TTM characterisation. The current case of this study, a primary breast carcinoma metastasising into two intracranial meningiomas, is presented with a list of pertinent cases reported to date, and literature is reviewed.

Key words: Tumour to tumour, Metastasis, Meningioma, Breast carcinoma

Sistemik Tümörlerin Serebral Menenjiom İçine Metastazı: Alanyazın Taraması ve Olgu Sunumu

Özet

Tümörden tümöre metastaz, (TTM), primer bir neoplazinin aynı hastada görülen diğer bir neoplaziye metastazıdır. Menenjiyomlar, malign metastazlara en sık ev sahipliği yapan intrakranial tümör olarak rapor edilirken, meme ve akciğer karsinomları ise en sık görülen sistemik donör tümörleri olarak bildirilmektedir. İlgili literatürde, menenjiomlara ait bazı özelliklerin metastaz için uygun ortam oluşturduğu belirtilmektedir. Bununla birlikte, tümörün tümöre metastazına önemli katkıda bulunduğu düşünülen immünolojik ve hormonal etkenler gibi bazı etkenler araştırma gereksinimi yaratmaktadır. Perfüzyon MRI (p-MRI) ve proton spektroskopik MRI (proton s-MRI) gibi nöroradyolojik görüntüleme yöntemleri tümörün tümöre metastazının ayırılmasında etkin olabilir. Bu çalışmanın olgusu, primer meme karsinomunun iki intrakranial menenjiyom içine metastazı, güncel bildirilmiş olgu listesi ile sunulmuş ve literatür gözden geçirilmiştir.

Anahtar Kelimeler: Tümörden tümöre, Metastaz, Menenjiom, Meme karsinomu

INTRODUCTION

With the improving medical care facilities and advances in neuroimaging and histopathology, tumour-to-tumour metastasis (TTM) has recently become a well-reported issue despite the limited number of cases reported so far.

Since reported first by Fried in 1930, a case of bronchogenic carcinoma metastatic to a meningioma, TTM has still remained as "a rare, uncommon" occurrence for which many hypotheses have been put forward to explain the mechanism of this phenomenon. As defined by Fried, TTM is "the case of a primary neoplasm metastasis to another primary tumour type within the same person"(1).

Meningiomas have been cited as the most frequent cerebral neoplasm to harbour metastasis from extracranial tumours (2,3). Among the less frequently hosting cerebral neoplasms are acoustic neurinomas, gliomas and pituitary adenomas in the published series (3). The most common extracranial donor neoplasm has been reported to be breast carcinoma and followed by lung carcinoma (2,4,5).

With the current case, the number of reported cases of systemic tumour metastasis to meningiomas has become 133. Reported here is a case of breast lobular carcinoma metastatic to two meningiomas in the left cerebral hemisphere, with a review of literature.

CASE PRESENTATION

A 52-year-old woman underwent a right modified radical mastectomy in 2005 and was diagnosed with multicentric invasive lobular carcinoma of the breast and followed by adjuvant chemotherapy and radiotherapy.

In 2010, in the follow-up, a metastasis was detected in her liver and the cranial

imaging revealed two masses in her left cerebral hemisphere, measuring 1 x 1.5 cm one in the frontal lobe and 3.5 x 2.5 cm one in the parietal lobe, both were consistent with meningiomas (Fig.1). As the cranial lesions were asymptomatic, the patient was treated first by systemic chemotherapy due to the lung and liver metastasis.

Contrast-enhanced cranial magnetic resonance imaging (MRI) performed due to the seizure complaints of the patient in 2012 demonstrated two different mass patterns, one in left frontal lobe and another in left parietal lobe, having peripheral oedemas (Fig.2). Hypointense areas in both mass patterns were identified. The patient underwent a frontoparietal craniotomy as the two masses detected were consistent with meningioma.

Surgical observation of the tumours agreed with the preoperative diagnosis of meningioma. In post-surgical course, the patient had temporary aphasia accompanied with mild right-sided hemiparesis, and both complications were disappeared in the follow-up course.

The histopathological diagnoses of the two intracranial tumours were as follows; the tumour in the frontal lobe was a metastasis of breast carcinoma on the basis of transitional meningioma and the second was a lobular carcinoma metastasis in parietal lobe (Fig.3).

The patient was treated with radiotherapy after the removal of the intracranial meningiomas.

From 18 February to 8 March 2013, 30 gray (Gy) in 10 fractions was delivered to the whole brain and a boost dose of 15 Gy in 5 fractions to the tumour bed with helical tomotherapy, which was very well tolerated, was delivered.

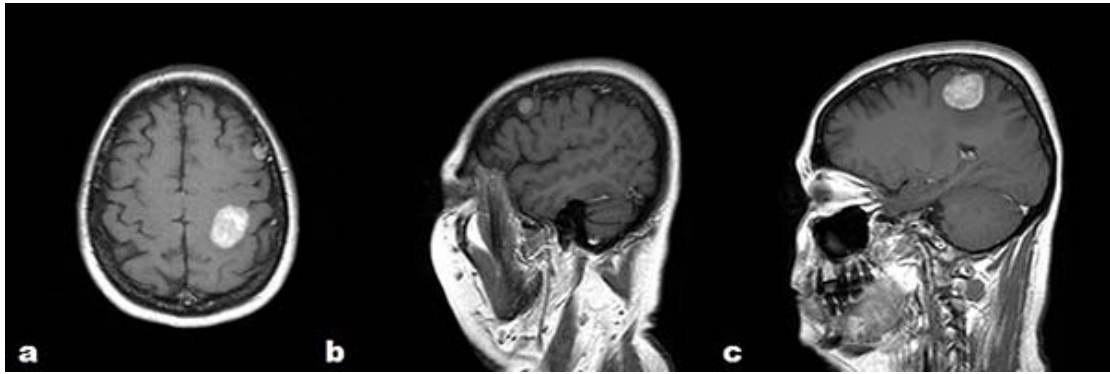


Fig.1. Axial (a), sagittal(b,c) gadolinium-enhanced T1-weighted MRI scans showing two extra axial hyperintense masses.

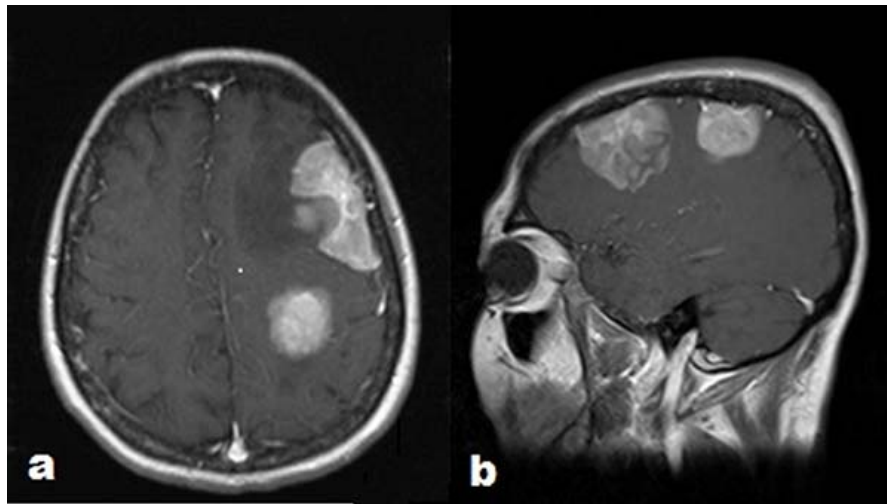


Fig.2. Axial (a), sagittal(b) gadolinium-enhanced T1-weighted MRI scans showing two larger extra axial hyperintense masses with peritumoural oedema and additionally, hypointense area on frontal hyperintense lesion.

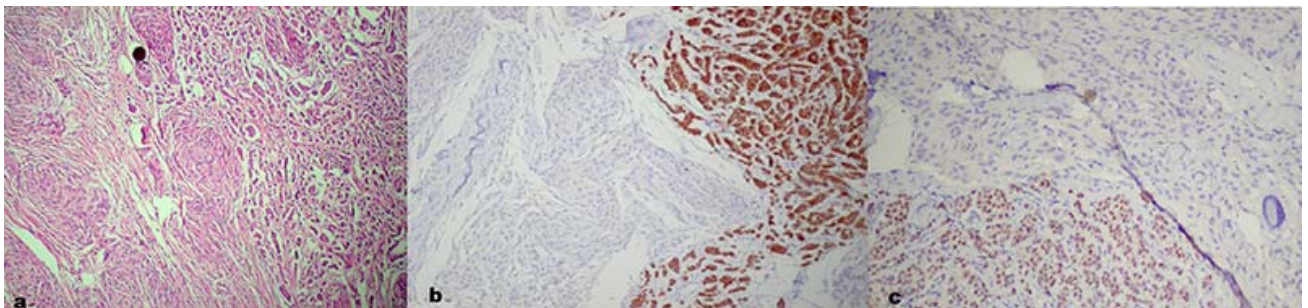


Fig.3. (a) typical meningioma areas on the left and carcinoma areas, both in group and individual cell forms. HE*4. (b) Carcinoma areas positive with milk fat globulin. DAB*4. (c) estrogen-positive cells in carcinoma areas. DAB*4.

DISCUSSION

Metastasis of a tumour tissue to another primary tumour has been reported to be as an infrequent event in almost every article published on this issue. Due to the rarity of this occurrence, it took four decades to establish some criteria of diagnosis of TTM after the first definition was suggested by Fried in 1930.

On the basis of previously published cases, the criteria for the histopathological diagnosis of TTM were first proposed by Campbell et al. (6). In order to identify an event as a TTM case; (i) more than one primary tumour must exist within the same patient; (ii) the recipient tumour must be a true neoplasm; (iii) the metastatic neoplasm should be a true metastasis with established growth within the recipient tumour, not the result of contiguous growth from an adjacent tumour, or embolisation of tumour cells; and (iv) tumours that have metastasized to the lymphatic system

where lymphoticular malignant tumours already exist are excluded.

According to Chambers et al. in order for the diagnosis of 'true' TTM; (i) the metastatic focus must at least be partially enclosed by a rim of histologically distinct tumour tissue; and (ii) the existence of the metastasising primary tumour must be proven and be compatible with metastasis (7).

The patient of the present case fulfils these criteria.

However, collision tumours, the two separate neoplasms in the same patient, are found adjacent to each other in the same organ (8). The case- collision tumour of meningioma and malignant astrocytoma- presented by Khalatbari et al. could be an example for collision entity. Despite being classified as collision tumour cases in the pertinent literature, Breast Case 58 and Renal Case 6 in Table 1 are in fact consistent with TTM characteristics stated before (9).

Table 1: Cases of extracranial tumours metastasis to meningiomas in literature

Donor Tumour	Sex/ age	Subtype of meningioma	Case #	Reference
Breast	F/72	ND	1	Bernstein, 1933 (32)
	F/45	Psammomatous	2	Lapresle et al., 1952 (33)
	F/59	ND	3	Helpap, 1965 (34)
	F/42	ND	4	Anlyan et al., 1970 (35)
	F/74	Psammomatous	5	Theologides and Lee, 1972 (36)
	F/72	Psammomatous (Spine)	6	Hockley, 1975 (37)
	F/68	Transitional	7	Di Bonito and Bianchi, 1978 (38)
	F/66	Psammomatous	8	Chambers et al., 1980 (7)
	F/53	Meningothelial	9	Nunnery et al., 1980 (39)
	F/53	Endotheliomatous	10	Lodrini and Savoiaro, 1981 (40)
	F/23	Meningothelial	11	Joglekar et al., 1981 (41)
	F/ 56	Meningothelial	12	Barz, 1983 (43)
	F/ 68	Psammomatous	13	Schmitt,1984 (3)
	F/ 64	Meningothelial	14	Doron and Gruszkiewicz, 1987 (21)

F/ 57	Meningothelial	15	Zon et al., 1989 (42)
F/ 53	ND	16	Fabaron et al., 1990 (44)
F/ 64	ND	17	Bucciero et al., 1992 (45)
F/ 50	Meningothelial	18	Chou et al.,1992 (46)
F/ 60	ND	19	Völker and Thierauf, 1993 (47)
F/ 85	Psammomatous	20	Di Bonito et al., 1993 (48)
F/ 73	Transitional	21	Di Bonito et al., 1993 (48)
F/ 85	Transitional	22	Di Bonito et al., 1993 (48)
F/ 82	Psammomatous	23	Di Bonito et al., 1993 (48)
F/ 85	Meningothelial	24	Di Bonito et al., 1993 (48)
F/ 74	Psammomatous	25	Di Bonito et al., 1993 (48)
F/ 84	Transitional	26	Di Bonito et al., 1993 (48)
F/ 56	Psammomatous	27	Di Bonito et al., 1993 (48)
F/ 95	Psammomatous	28	Di Bonito et al., 1993 (48)
F/ 66	Psammomatous	29	Di Bonito et al., 1993 (48)
F/ 62	Transitional	30	Di Bonito et al., 1993 (48)
F/ 52	Psammomatous	31	Di Bonito et al., 1993 (48)
F/ 65	Fibroblastic	32	Cervoni et al., 1994 (19)
F/ 62	ND	33	Fornelli et al., 1995 (49)
F/ 47	Transitional	34	Salvati and Cervoni, 1996 (50)
F/ 54	Psammomatous	35	Salvati and Cervoni, 1996 (50)
F/ 53	Transitional	36	Salvati and Cervoni, 1996 (50)
F/ 48	Psammomatous	37	Salvati and Cervoni, 1996 (50)
F/ 55	Meningothelial	38	Salvati and Cervoni, 1996 (50)
F/ 49	Transitional	39	Salvati and Cervoni, 1996 (50)
F/ 61	Meningothelial	40	Salvati and Cervoni, 1996 (50)
F/ 58	Transitional	41	Salvati and Cervoni, 1996 (50)
F/ 50	Transitional	42	Salvati and Cervoni, 1996 (50)
F/ 79	Transitional	43	Lee et al., 1998 (51)
F/ 51	ND	44	Elmaci et al., 2001 (20)
F/ ND	ND	45	Maiuri et al., 2002 (52)
F/ ND	ND	46	Maiuri et al., 2002 (52)
F/ ND	ND	47	Maiuri et al., 2002 (52)
F/ 49	ND	48	Watanabe et al., 2002 (17)
F/ 40	Psammomatous	49	Lieu et al., 2003 (53)
F/ 60	Meningothelial	50	Lieu et al., 2003 (53)
F/ 42	Meningothelial	51	Lieu et al., 2003 (53)
F/ 46	Meningothelial	52	Lieu et al., 2003 (53)
F/ 54	Fibroblastic	53	Baratelli et al., 2004 (54)
F/ 56	Psammomatous	54	Aghi et al., 2005 (4)

	F/ 59	Meningothelial	55	Caroli et al., 2006 (5)
	F/ 53	Fibrous	56	Caroli et al., 2006 (5)
	F/ 65	Fibrous	57	Caroli et al., 2006 (5)
	F/ 56	ND	58	Jun et al., 2006 (28)
	F/ 72	Meningothelial	59	Seçkin et al, 2006 (55)
	F/ 47	Transitional	60	Miyagi et al., 2007 (56)
	F/ 64	Meningothelial	61	Lanotte et al., 2009 (11)
	F/ 63	Metaplastic	62	Lin et al., 2009 (18)
	F/ ND	ND (Spine)	63	Pablo et al. 2009 (57)
	F/ 60	Psammomatous	64	Dadlani et al., 2013 (58)
	F/ 69	Fibrous (Spine)	65	Okada, 2014 (59)
	F/ 52	Transitional	66	Current case, 2015
Lung	F/ 57	Meningothelial	1	Fried, 1930 (2)
	M/ 71	Meningothelial	2	Osterberg, 1957 (60)
	M/ 48	Meningothelial	3	Best, 1963 (61)
	M/ 39	Meningothelial	4	Wilson et al., 1967 (62)
	M/ 64	Psammomatous	5	Wolintz and Mastri, 1970 (63)
	M/ 75	ND	6	Zoos, 1970 (64)
	F/ 69	Transitional	7	Gyori, 1976 (65)
	F/ 68	Meningothelial	8	Weems and Garcia, 1977 (66)
	F/ 61	Meningothelial	9	Hope and Symon, 1978 (67)
	M/ 72	Meningothelial	10	Chambers et al., 1980 (7)
	M/ 59	Fibromatous	11	Lodrini and Savoiaro, 1981 (40)
	F/ 65	Fibroblastic	12	Smith et al., 1981 (22)
	M/ 52	Meningothelial	13	Jomin et al., 1982 (68)
	F/ 66	Meningothelial	14	Barz, 1983 (43)
	M/ 62	Meningothelial	15	Barz, 1983 (43)
	M/ 60	Angiomatous	16	Schmitt, 1984 (3)
	M/ 79	Angioblastic	17	Pamphlett, 1984 (69)
	M/ 69	Transitional	18	Conzen et al., 1986 (70)
	M/ 71	Meningothelial	19	Arnold et al., 1995 (71)
	F/ 62	Transitional	20	Gardiman et al., 1996 (72)
	M/ 52	Transitional	21	Bhargava et al., 1999 (16)
	F/ 48	Secretory	22	Cserni et al., 2002 (73)
	ND/ ND	ND	23	Maiuri et al., 2002 (52)
	F/ 69	Microcystic	24	Takei and Powell, 2009 (74)
	ND/ ND	ND	25	Duprez, 2009 (75)
	F/ 71	Fibrous	26	Kim et al., 2013 (76)
	F/ 74	Meningothelial	27	Chatani et al., 2014 (77)
	M/ 57	Meningothelial	28	Glass et al., 2013 (78)

Renal	F/ 45	Psammomatous	1	Stortebecker, 1951 (79)
	M/ 51	ND	2	Osterberg, 1957 (60)
	F/ 82	ND	3	Breadmore et al., 1994 (80)
	F/ 67	Meningothelial	4	Han et al., 2000 (81)
	F/ 70	Lipomatous	5	Kimiwada et al., 2004 (82)
	M/ 67	Meningothelial	6	Chahlavi et al., 2005 (83)
	M/ 61	Meningothelial	7	Lanotte et al., 2009 (11)
	M/ 72	Meningothelial	8	Gutierrez Morales et al., 2009 (84)
	M/ 61	Meningothelial	9	Tsunoo et al., 2010 (85)
	M/ 70	Atypical (Grade II)	10	Iżycka-Świeszewska et al., 2013 (26)
	F/ 74	ND	11	Ventura, 2013 (86)
	M/ 54	ND	12	Carr et al., 2014 (87)
Prostate	M/ 75	Meningothelial	1	Döring, 1975 (88)
	M/ 67	Angiomatous	2	Chambers et al., 1980 (7)
	M/ 55	ND	3	Bernstein et al., 1983 (89)
	M/ 78	Atypical	4	Cluroe, 2006 (90)
	M/ 70	Meningothelial	5	Pugsley et al., 2009 (91)
	M/ 72	Atypical	6	Mitchell et al., 2011 (92)
	M/ 58	ND	7	Moody et al., 2012 (29)
	M/ 57	ND	8	Moody et al., 2012 (29)
Malignant Melanoma (MM)	M/ 63	ND	1	Wong et al., 1999 (13)
	F/ 51	ND	2	Shariff et al., 2009 (15)
	F/75	Fibroblastic	3	Takei and Powell, 2009 (74)
	ND/ ND	ND	4	Pal et al., 2010 (93)
Cervix uteri Genitourinary Ovary	F/42	ND	1	Wu et al., 1977 (94)
	F/55	ND	2	Ho, 1980 (95)
	ND/ ND	ND	3	Maiuri et al., 2002 (52)
Thyroid gland	F/ 45	Transitional	1	Chaturvedi et al, 2010 (96)
Parotid gland	M/ 68	Microcytic	1	Van Zandijcke and Casselmann, 1996 (97)
	M/ 68	Microcytic	2	Ebner et al., 2011 (25)
Hematopoietic System Multiple Myeloma	M/ 54	Fibroblastic	1	Widdel et al., 2010 (14)
Lymphoma	F/ 45	Fibroblastic	2	Widdel et al., 2010 (14)

		Atypical meningioma	3	Sonet et al., 2001 (98)
Colon	F/ 76	Fibrous	1	Benedetto et al., 2007 (10)
Colorectal	M/ 57	Meningothelial	2	Iżycka-Świeszewska et al., 2013 (26)
	M/ 77	ND	3	Moody et al., 2012 (29)
Stomach	ND/ ND	ND	1	Honma et al. 1989 (99)
Pituitary Carcinoma	M/ 67	Meningothelial	1	Zhou et al., 2013 (100)
Gallbladder	F/ 74	Meningothelial- partly fibroblastic	1	Peison and Feign, 1961 (101)

F: female; M: male; ND: either 'non-defined' or 'not available'.

Compared to other intracranial neoplasms, meningiomas have been regarded as the most intracranial tumours to harbour metastatic tumour cells, followed by haemangioblastoma, astrocytoma, pituitary adenoma, schwannoma, oligodendroglioma and ependymoma and therefore reported more frequently as a result of their propensity to the deposition of metastasis from extracranial tumours (4,5,10).

Meningiomas are generally slow growing benign tumours (WHO grade 1) arising from leptomeninges; atypical meningiomas (WHO grade 2) and anaplastic meningiomas (WHO grade 3). Although the reason for TTM favouring specific intracranial tumours is still obscure, there are some metabolic and immunologic properties of meningiomas which seem to make them a favourable environment for metastatic seeding and growing (2,11-13). These characteristics could be listed as follows: (i) their being slow growing neoplasms and the concomitant slow metabolic rate within the cranial venous system leading a long-term exposure to

metastatic tumour cells (14); (ii) their high collagen and lipid content which may provide adequate nutrients for the metastatic tumour; (iii) their rich vascularity network that may act as a vascular filter and increase the possibility of receiving malignant cells and to form a metastasis. However, only the presence of rich vascularisation, as in the case of anaplastic primary intracranial tumours such as glioblastomas, does not imply a deposition of metastasis from a systemic cancer. Since metastases there have rarely been reported in the relevant literature, other factors must be considered to play a prominent role in metastatic occurrence (2,11,13,15).

A meticulous review of the literature has revealed 133 published cases of TTM of extracranial tumours to meningiomas (Table 1). Compared to the number of rare cases of metastasis from tumours of the kidney, prostate, skin or genitourinary, the preponderance of the breast and lung tumours metastasising to intracranial meningiomas has proved that tumours

originating from the breast and lung are the most common donor neoplasms (2,4,5,11) and constitutes 70% of the TTM to meningioma cases reported to date. Metastasis into intracranial meningiomas has most frequently been detected in the breast cancer cases in women, and it is the lung cancer cases in men (16,17).

According to the epidemiological studies carried out so far, there is a high frequency of the simultaneous occurrence of breast cancer and meningiomas (18,20) and there are some factors which might elucidate the associations between them. First, when the ages of the patients in reported series is considered, it is clear that both types of tumours occur predominantly in women in the fifth to seventh decades of their lives and moreover, meningiomas tend to grow during pregnancy. This finding suggests a possible hormonal relationship between breast tumours and meningiomas and their simultaneous occurrence in the same patient (5,11,18-20). In a large proportion of meningiomas published in TTM case reports, the presence of some specific receptors at the cellular level has called attention and receptors for sex hormones have been identified (21,22). Furthermore, receptor expressions of progesterone (PgR), estrogen (ER) and androgen (AR) are thought to be inversely related to tumour grade- WHO grade 1 meningiomas are more apt to have these receptors than atypical/ anaplastic histotype (23,24).

Immunohistochemical examination of the surgical specimen demonstrated estrogen receptors (ER) in the carcinoma areas of both meningiomas in the current case.

In order to highlight the exact mechanisms responsible for TTM phenomenon, recent studies- especially the ones on implantation of extracranial tumours into intracerebral meningiomas- have also focused on the cell-to-cell interaction and the roles of the expression of certain surface adhesion molecules, in particular E-cadherin (E-cad) (4,17). As a cell surface glycoprotein, E-cad is thought to

play a prominent role in calcium-dependant cell-to-cell adhesion and therefore, promote implantation of breast cancer metastasis into meningioma (4,17). The presence of E-cad in both metastatic tumour and meningiomas has been encountered in several cases of TTM (4,11,17,25,26).

Furthermore, a combination of immunological and molecular factors within both metastatic donor neoplasm and meningioma has been postulated to have a role in the predisposition for a benign intracranial tumour to metastatic implantation (5,15). However, as for many issues about the TTM phenomenon, immunological and molecular factors and their effects require further extensive research.

Central nervous system TTM can be quite challenging to recognise in most cases due to the fact that they often remain asymptomatic (16,27). Some cause significant oedema, requiring rapid surgical resection. Our patient was asymptomatic at the time of diagnosis. When no information as to prior or concomitant malignancy is provided before surgery, the condition could be incidentally identified on surgery or autopsy and the incidentally found metastatic lesions within meningioma might be identified only on histopathological examination of a resected specimen (16).

The preoperative diagnosis of TTM based on routine radiological imaging techniques, such as computerised tomography (CT) and magnetic resonance imaging (MRI), is quite difficult to make. Moreover, the existence of metastasis within a meningioma cannot be eliminated reliably only with CT or MRI. On the other hand, physiology-based neuroimaging techniques, such as perfusion MRI (p-MRI) and proton spectroscopic MRI

(proton s-MRI) have been proposed as useful diagnostic tools in noninvasively differentiating tumour histology (11,17,28,29).

P-MRI depends on hemodynamic differences in microvasculature to make a distinction between peculiar tissue types preoperatively (29). Perfusion changes in meningiomas, highly vascular tumours, could point out a metastasis within (28). Proton s-MRI has been reported to be useful in distinction of malignancy within meningioma (30). Compared to MRI alone, it can provide more data concerning tissue characterisation (18).

An increase in the lactate/creatinine ratio is an indicator to determine the degree of malignancy of lesions. In other words, when deposition of malignant cells to a meningioma occurs, there is an increase in lactate/creatinine ratio (31). Moreover, increases in lipid/creatinine and alanine/creatinine ratios could differentiate metastasis and meningiomas from other intracranial tumours, respectively (29).

Also, in a breast cancer metastasis to an intracranial meningioma case, a high choline/creatinine ratio and lactate/lipid peak were found on proton s-MRI, suggesting malignancy in the meningioma (17). When a patient with a primary cancer history is radiologically diagnosed with an atypical meningioma, proton s-MRI should be performed to gather valuable data before surgery.

It is of grave importance to inform the pathologist as to the surgical observation of the lesions and the metastatic foci, and if there is any, to assist the histopathological examination and avoid any possible misdiagnosis.

CONCLUSION

With the improvements in diagnostic techniques and the increasing life expectancy, the rare phenomenon TTM has recently become a well-reported issue of rising interest. However, metastatic meningiomas could remain asymptomatic during/ after the treatment of primary systemic cancer, the presence of it could be taken no notice of until an abrupt deterioration in the patient's general/

neurological condition, -if previously known- rapid tumour growth and atypical imaging characteristics occur.

Misdiagnosis of TTM may occur due to the numerous meningioma variants, very small metastatic deposit or histological mimicry between metastatic tumour and meningioma (27). For this reason, in a diagnosed case of breast carcinoma, sharing information as to primary cancer history of the patient and the radiological atypical characteristics of meningioma is also crucially important for accurate diagnosis of TTM and even for the administration of postoperative adjuvant therapy.

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