Intracranial Dural Metastases and Diagnostic Misunderstandings

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Abstract: Dural metastases are rare intracranial tumors. They are not sufficiently studied and there are still no specific methods are not to detect them. Differential diagnosis is difficult and only the histologic examination allows a sure diagnosis. We reviewed data records from 2016 to 2020 of patients treated for dural metastases. We included only patients with complete anamnestic history, with both known and unknown primitive cancer. Collected data were compared with recent literature. We operated on 16 single dural metastases, also from very unusual cancers. The most common primitive type of cancer, in our series, was lung tumor, in contrast to prostate cancer, recently reported in literature as the most frequent. A retrospective multicenter study is mandatory to assess new epidemiologic evidences.

Keywords: Brain metastasis, Dural metastasis, Differential diagnosis, Meningioma, Metastasis.

1. INTRODUCTION

Brain metastases (BMs) are the most frequent brain tumors in adults and occur in 20-45% of patients with a primary tumor [1]. The most common primary sites for BMs are lung cancer (40-50%), breast cancer (15-25%), and melanoma (5-20%) [2]. The incidence of BMs is increasing with improved cancer survival, aging population, improved awareness of the disease, better diagnostic tests and oncological therapies. Meningeal localization is not frequent with an incidence of 9% [3]. The primary tumors causing dural metastases (DMs): prostate (19.5%), breast (16.5%), lung (11%) and stomach carcinomas (7.5%) [3]. Patients with DMs have a median survival rate of about 6 months [3]. It mainly depends by primary tumor, by the course of the underlying cancer and by the tendency of DMs to recur, particularly at the initial site. The surgical resection is the best procedure if a solitary, circumscribed and accessible lesion is evidenced and systemic disease is controlled. However, these lesions are often misinterpreted and this aspect may delay surgery and could lead to a deleterious impact on patient care. Additionally, although rarely, diagnosis of DMs can precedes the diagnosis of the primitive lesion [4].

In this study we report 16 additional cases of DMs, all characterized by objective difficulties of diagnostic interpretation. We also briefly report other lesions to be differential diagnosed with DMs. Additionally, we compared cases of dural metastasis treated at our Department in the last four years and the discrepancy with last incidence epidemiologic reported data.

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2. MATERIALS AND METHODS

2.1. Patients

We retrospectively reviewed the records of the Unit of Neurosurgery of the University of Messina to identify all patients affected by DMs treated between 2016 and 2020.

Inclusion criteria were: 1) diagnosis of DM based on computed tomography (CT) scans and magnetic resonance imaging (MR) of all patients; 2) histological examination of the metastatic lesion obtained from the tissue taken during the surgical treatment. By reviewing patient records, the following information was also gathered: age, sex, tumor location, and clinical presentation. We collect data for the most common symptoms for intracranial neoplasms: headache, epilepsy, vomiting, motor and language deficits and hypertension syndrome signs. All patients underwent CT scan and MR (Figures **1A** and **1B**, **2A** and **2B**).

The histopathological analyses were performed at the Neuropathology Laboratory of the Department of Human Pathology of the University of Messina. Surgical samples were formalin fixed and paraffin embedded. Four µm consecutive sections were cut from paraffin blocks for histological examination with haematoxylin and eosin stain and immunohistochemistry. Immunostaining was performed with the Bond Polymer Refine Detection kit (Leica Biosystems) in a BOND-MAX system (Leica Biosystems) using mouse monoclonal antibody multi-cytokeratin (clones AE1 and AE3; dilution 1:100).

All patients signed an informed consent with description of surgical procedure and possible

ISSN: 1927-7210 / E-ISSN: 1927-7229/22

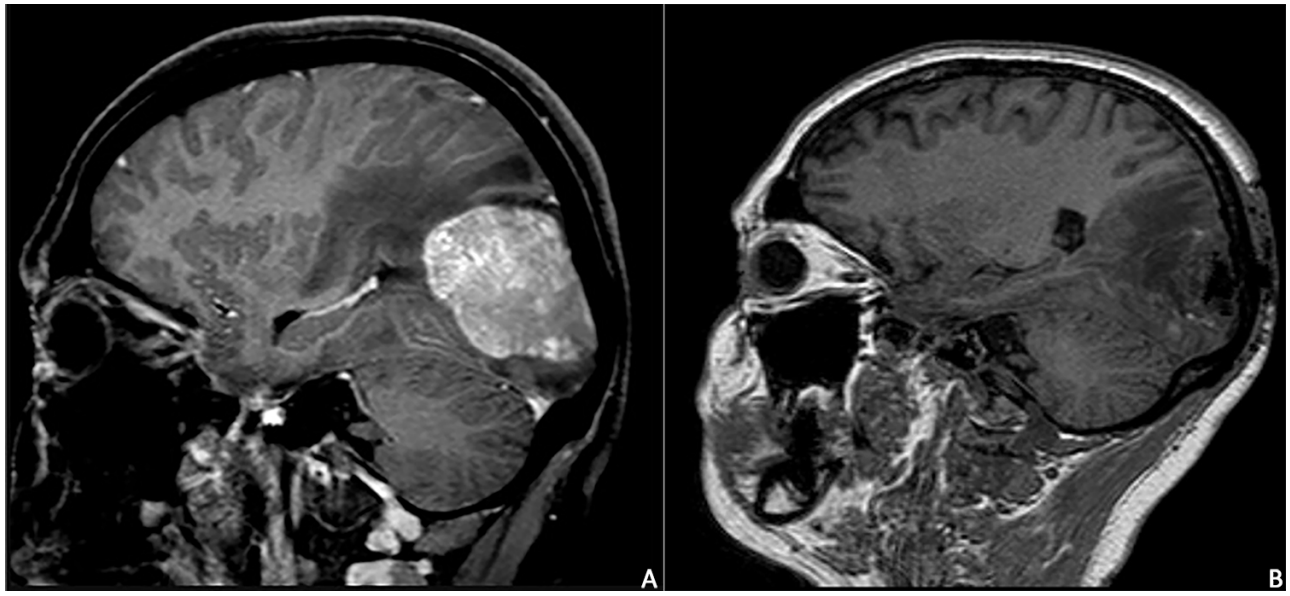

Figure 1: Case nr. 7: dural metastasis from breast adenocarcinoma. **A**: a post-gadolinium T1-weighted MR showed, in sagittal view, in the right parietal lobe, a bulky, extra-axial, dural-based lesion. **B**: Control MR at one month.

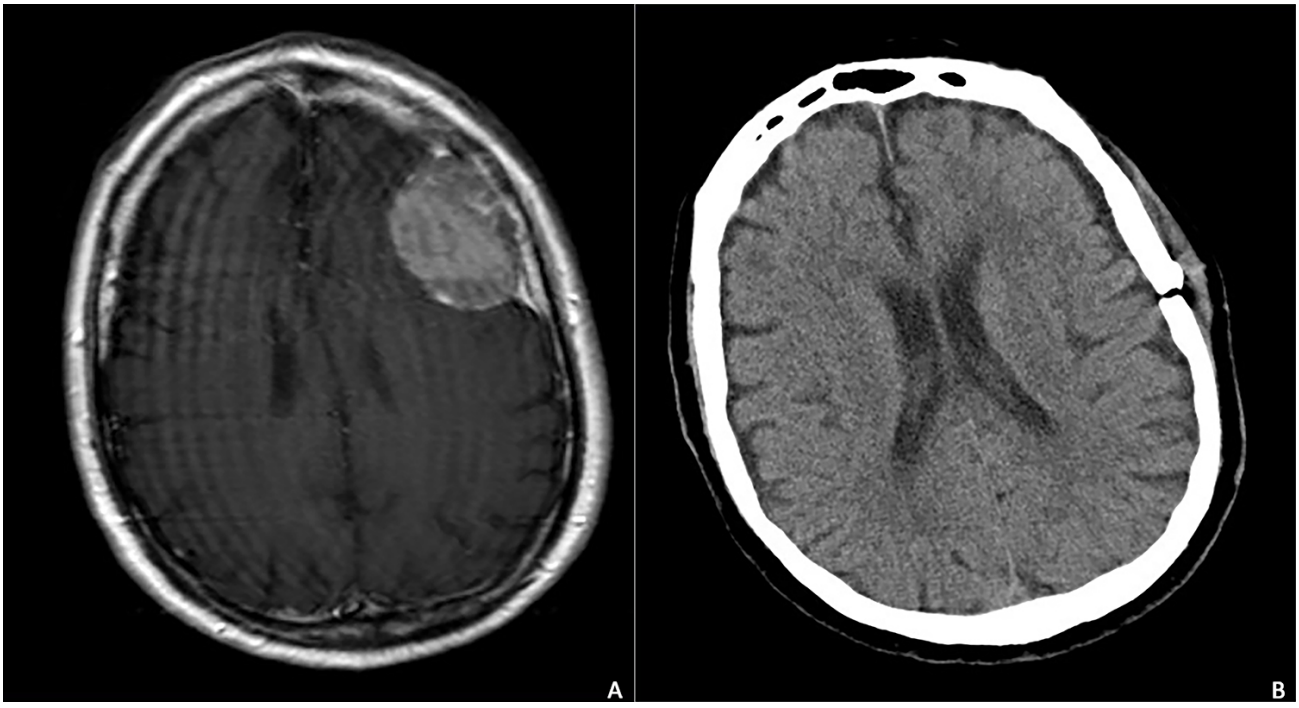

Figure 2: Case nr. 5: dural metastasis from prostate adenocarcinoma. **A**: a post-gadolinium T1-weighted MR showed. In axial view, an extra-axial, dural-based masses in the left frontal convexities with an inhomogeneous enhancement. MR also shows dural enhancement with dural tail sign. **B**: Control CT at 20 days.

complications and for processing data for scientific purposes.

2.2. Results

Sixteen cases were collected. The patients ranged in age from 40 to 80 with a mean age of 65. Nine patients were female (56%) and seven patients were male (44%). We collected the most common sign and symptoms as shown in Table **1**: headache (n=7; 43%), epilepsy (n= 4; 25%), vomit (n= 2; 12,50%), motor deficit (n= 9; 56%), language deficit (n= 6; 37,50%) (Table **1**). All patients underwent brain CT and MR. All patients were undergone to surgical treatment under general anaestesia. Craniotomy was performed in all cases. In all patients, the lesion was single. The

Table 1: Shows the Localizations, Signs and Symptoms of each Patient

Case	Age & Sex	Localization	Headache	Epilepsy	Vomit	Motor deficits	Phasia deficit
1	80y, F	Cerebellar			X		
2	68y, F	Frontal				X	X
3	77y, M	Cerebellar					
4	57y, M	Occipital				X	
5	66y, M	Frontal				X	X
6	40y, F	Frontal	X				
7	43y, F	Occipital	X				
8	59y, F	Frontal		X		X	
9	71y, M	Frontal					X
10	64y, F	Parietal				X	
11	69y, F	Temporal	X			X	X
12	70y, M	Cerebellar	X		X		
13	57y, M	Parietal		X		X	
14	58y, F	Temporo-occipital	X			X	X
15	68y, F	Temporal	X	X			
16	59y, M	Temporo-occipital	X	X		X	X

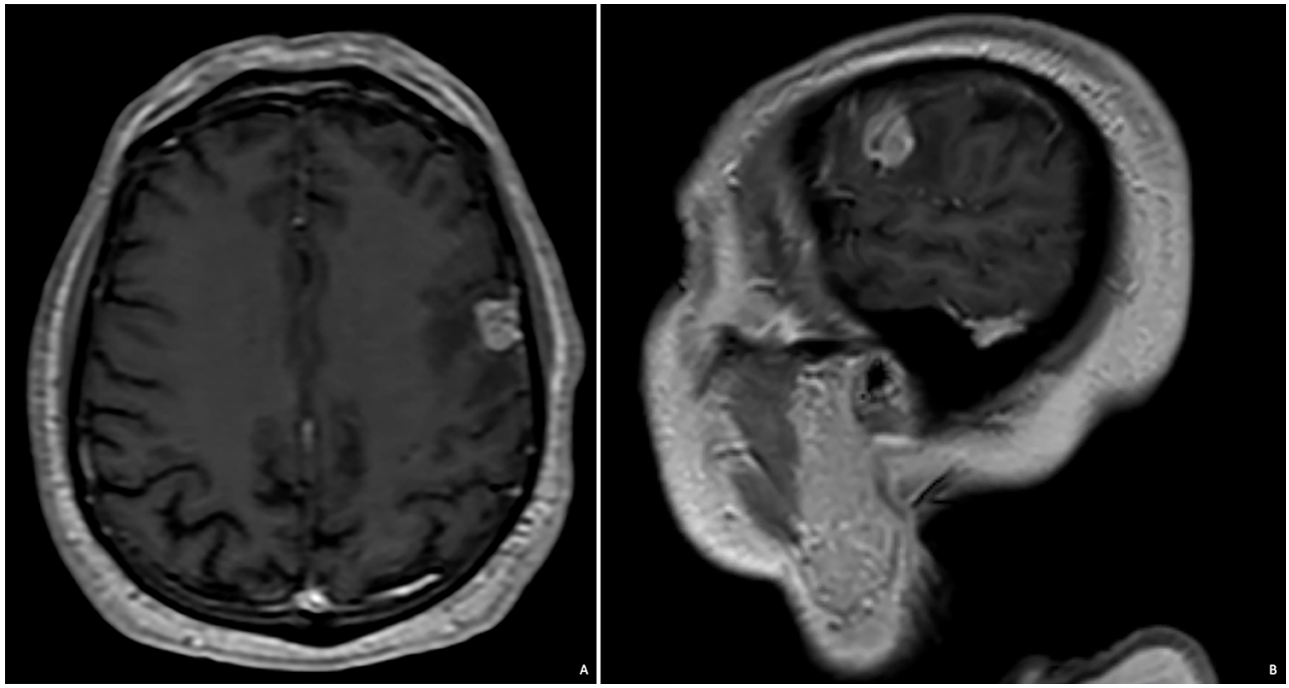

Figure 3: Case nr. 9: left frontal dural metastasis from Merkel cell carcinoma (not known at admission). A post-gadolinium T1-weighted MR showed, in axial view (**A**), and in sagittal (**B**) left frontal lesion.

postoperative course was regular for each patient. A postoperative MR study was performed at each patient at follow-up control. All cases, at neuroradiological examination seemed meningioma. In 2 cases (12,5%) the primitive cancer was unknown before the histological analysis (Figures **3A** and **3B**).

The primitive cancer was lung carcinoma (n=7; 43,75%), breast carcinoma (n=4; 25%), melanoma (n=2; 12,5%), thyroid and prostate and skin (n=1 each; 6,25% each). The immunohistochemical features of each lesion are shown in Table **2** (Figures **4A** and **4B**).

Table 2: Shows the Immunohistochemical Features and Primary Tumor of each Patient

Case Number	Immunohistochemistry	Primitive
1	CK7: ++, CA125: +, TTF1: -, Ck20: -, ER: -, PGR: -	Ovarian adenocarcinoma
2	Melan-A: ++, S100: ++, HMB45: ++, CK: +, Ki-67: 30%	Melanoma
3	CK5: -, CK6: -, CK7: -, CK20: ++, TTF1: ++, Ki-67: 30%	Lung adenocarcinoma
4	CK7: +, CK20: +++, TTF1: -	Lung adenocarcinoma (not known at admission)
5	CK: ++, Racemase: ++, PSA: -, Ki-67: 30%	Prostate adenocarcinoma
6	CKAE1-AE3: ++, HER2: +++ ER: -, AR: -, PGR: -, Ki-67: 30%	Breast adenocarcinoma
7	HER2: +++, AR: 10%, ER: -, PGR: -, Ki-67: 30%	Breast adenocarcinoma
8	CK7: +, TTF1: +, p63: -, CK5/6: -, Ki-67: 40%	Lung adenocarcinoma
9	CKAE1-AE3: ++, CK7: +, CK20: ++, Synaptophysin: ++, Chromogranin A: ++, CD56: ++, NSE: ++, CD99: ++, INI-1: ++, TTF1: -, S100: -, HMB45: -	Merkel cell carcinoma (not known at admission)
10	ER: 100%, PGR: 1%, Ki-67: 50%	Breast adenocarcinoma
11	CKAE1-AE3: +, CK7: +, EMA: +, TTF1: -, GFAP: -, CK20: -, Vimentin: -	Lung adenocarcinoma
12	CKAE1-AE3: +, CK20: +, CDX2: +, TTF1: -, CK7: -	Gastric adenocarcinoma
13	Melan-A: +, HMB45: +, CKAE1-AE3: -, Ki-67: 20%	Melanoma
14	CK7: +, TTF1: +, Napsin A: +, GFAP: -, p40: -, CK20: -, CD56: -, MIB-1: >50%	Lung adenocarcinoma
15	CKAE1-AE3: ++, HER2: ++ ER: -, PGR: -, Ki-67: 35%	Breast adenocarcinoma
16	CKAE1-AE3: +, CK7: +, CK19: +, Napsin A: +, TTF1: -, Vimentin: -, CK20:-	Lung adenocarcinoma

CK: Cytokeratine; CA125: Cancer antigen 125; TTF1: Thyroid transcription factor-1; *ER:* Estrogen receptor; *PGR: P*rogesterone receptors; Melan-A: Melanoma antigen; HMB: Human melanoma black; PSA: Prostate-specific antigen; HER2: human epidermal growth factor receptor 2; AR: *A*ndrogen receptor; p63: tumor protein 63; CD56: Neural cell adhesion molecule; NSE: Neuron-specific enolase; CD99: Cluster of differentiation 99; INI-1: Integrase interactor 1; EMA: Epithelial membrane antigen; GFAP: Glial fibrillary acidic protein; CDX2: Homeobox protein; p40: tumor protein 40.

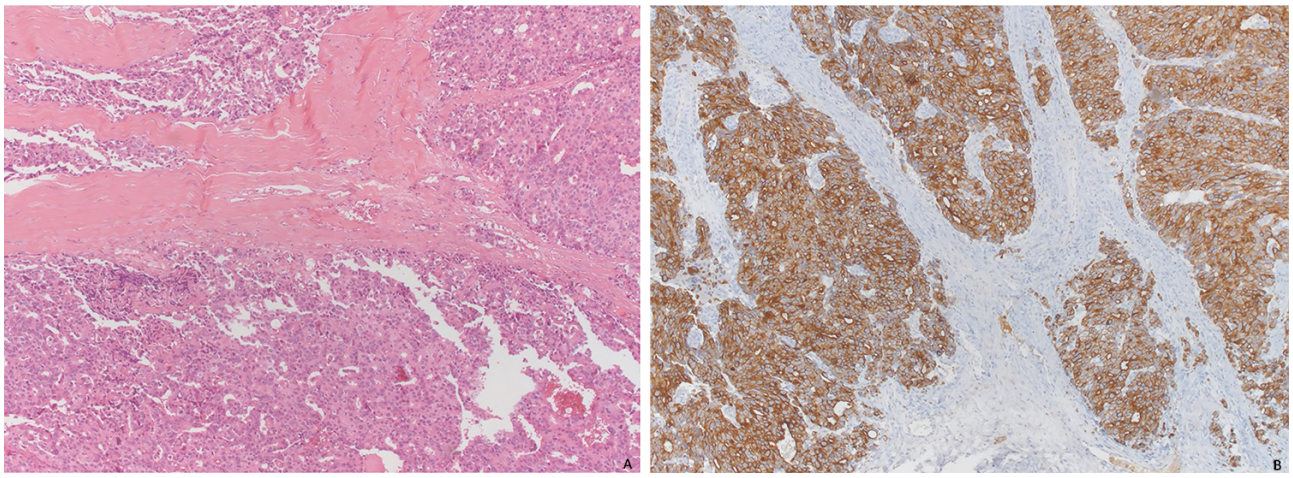

Figure 4: Case nr. 5: left frontal convexities dural metastasis from prostate adenocarcinoma. Immunoprofile: CK: ++, Racemase: ++, PSA: -, Ki-67: 30%. **A**: cribriform pattern and neoplastic cells with prominent nucleoli. **B**: Diffuse and strong staining for cytokeratins.

At one-month follow-up, all patients showed a further, progressive improvement of the clinical condition. Postoperative neuroradiological studies confirmed the absence of persisting lesion and no appearance of new metastases. The patients were then referred to the Oncology Department to follow treatments for specific cancer.

3. DISCUSSION

Many lesions, both neoplastic and non-neoplastic can affect the dura mater. These lesions may present neuroradiological findings similar to those of DMs, thus being able to cause errors in diagnostic interpretation. Below we will briefly describe the most frequently encountered injuries.

3.1. Neoplastic Lesions

Meningioma is an extra-axial tumor arising from arachnoid cap cells. If multifocal lesions involve meninges, they can be suggestive of metastatic lesions, but if a solitary solid tumor is present in a patient with no previous history of primary cancer, this latter aspect can cause diagnostic difficulties. Cytokeratins are markers of epithelial differentiation and they can be identify at immunohistochemistry. Really, metastatic carcinoma has diffuse and strong staining for cytokeratins, while meningioma has focal staining or no staining at all [5]. The dura tail sign, the linear enhancement of thickened dura mater adjacent to an extra-axial mass seen on T1-post-contrast images, is observed frequently in DMs. In addition, both metastases and meningiomas, show similar appearance on T1 and T2 – weighted images with a similar homogeneous contrast enhancement. Although the MR pattern in meningiomas and DMs is very similar, they both present different characteristics in perfusion-weighted imaging (PWI). Meningioma shows very high rCBV ratios compared to dural values, whose specific values are typically between 6 and 9 with some differences on the basis of histological type of meningiomas. However, not all DMs show a low perfusion; it depends on the primary cancer: Merkel carcinoma, kidney carcinoma or metastases from melanoma due to their elevated rCBV values are indistinguishable from meningiomas. 123I-MIBG scintigraphy can help to distinguish DMs from meningiomas. There is an accumulation of the tracer in DMs, particularly in carcinoid. In meningiomas, magnetic resonance spectroscopy (MRS) reveals high choline and alanine peaks and low NAA, no lipid or lactate peak [6]. Meningiomas have also higher alanine/creatinine ratios and this allows distinguishing them from other intracranial tumors [7].

Intracranial hemangiopericytomas are neoplasms arising from fibroblasts. On CT, hemangiopericytomas are heterogeneous, hyperdense, dural-based lesions, not associated with calcifications or hyperostosis, and they typically show a vivid heterogeneous enhancement, more heterogeneous with increasing grade. The erosion of adjacent bone is a common feature seen in more than half of hemangiopericytoma cases [8]. On MR, hemangiopericytomas are heterogeneous, predominantly isointense masses on T1WI and T2WI sequences. They can seem DMs, but hemangiopericytoma shows prominent internal vessel voids especially in T2. Approximately one-third of hemangiopericytomas exhibit a narrow base of dural attachment, two-thirds show broad based attachment with a dural tail sign.

Low-grade solitary fibrous tumors (SFTs) do not invade or occlude nearby venous sinuses, in contrast to high-grade. Solid area of tumor demonstrates restricted diffusion on DWI [9]. Lipid, lactate and high myo-inositol peaks (the latter is absent in DMs) are observed on MRS examination in both low-grade and high grade STF [8]. On perfusion imaging, SFTs are hyperperfused with rCBV values of 7 to 7.5 [10]. This can help differentiate SFTs from metastases.

Gliosarcoma is a rare variant of IDH-wildtype glioblastoma. Two types of radiological appearance are visible in gliosarcoma: deep parenchymal lesions and peripherical located lesions. The latter with dural attachment could mimic a DM [11]. On CT scan, gliosarcoma is a defined, round or lobulated, hyperdense solid mass with homogeneous contrast enhancement and peritumoral oedema. In T1WI sequences of MR, lesions are generally heterogeneous and hypointense mass with surrounding isointense regions. The hyperintensity is caused by intra-lesional haemorrhage. On T2WI sequences, lesions have heterogeneous signal due to haemorrhagic and necrotic components. Half of tumors are homogenous, well demarcated and demonstrate strong homogenous enhancement: a quarter of all lesions has dural tail sign [11]. The remaining are heterogeneous and only a little percentage of cases has the peripheral ring-enhancement characteristic of glioblastoma. On MRS, solid enhancing components of the tumor shows lactate peaks, along with increased choline, low NAA and low creatine values.

Central nervous system lymphoma represent the 0,6% of all intracranial tumors [12]. It is classified as primary, in absence of systemic disease, or secondary, with extranodal feature of systemic lymphoma. The frequency is variable, depending on the histological subtype, up to 0,5% in Hodgkin lymphoma and about 27% in non-Hodgkin lymphomas [13]. To correctly diagnose a lymphoma, corticosteroids must be

suppressed. Consequently, a second MR, one week later the interruption, would show an increasing in dimension of lymphoma and this could be helpful to differential diagnosis.

3.2. Non-Neoplastic Lesions

Despite tuberculosis has considered for many years a pathology most prominent in developing countries, the incidence of tuberculosis is rising elsewhere, due to immigrations and spreading of HIV [14]. Although the infection is typically confined to the respiratory system, can progress to multisystem disease, particularly in immunocompromised patients. CNS tuberculosis accounts for approximately 1% [15]. Being a great mimicker radiologically simulates numerous diseases [16]. Affected areas are isodense to hyperdense on CT [15]. On MR, lesions appear isointense on T1WI and T2WI with strong uniform enhancement with greater amount of perilesional vasogenic oedema. Tuberculomas arise anywhere in the brain, but mostly parenchymal tuberculomas have a close relation to the dura, sometimes appearing to have a dural attachment. On MRS, lesions demonstrate decreased NAA:Cr and NAA:choline, with lipid-lactate peaks also being elevated in 86% due to areas of necrosis [17]. Diffusion characteristics are variable and can be similar to metastases. Perfusion techniques show tuberculous lesions have rCBV values that are similar to or lower than most metastases [18].

Neurosarcoidosis occurs in approximately 5% of patients with sarcoidosis [19]. Neuroradiological findings of neurosarcoidosis include pachymeningeal/ dural masses, leptomeningeal involvement, enhancing brain parenchymal lesion, and cranial nerve involvement. A dural-based mass is one of the least common manifestations of neurosarcoidosis. Lesions typically homogeneously enhance on contrast-enhanced T1-weighted images.

3.3. Dural Metastases

Dural involvement is most commonly due for direct extension of adjacent metastatic skull lesions. This way is evidenced especially with lung, cervical, some prostatic and breast carcinomas and Ewing sarcoma. Tsukada *et al*. hypothesized a bone-dura spreading, such as a dissemination of tumor cells from vertebral bodies into the dura mater, through a retrograde reflux of tumor cells into veins and venous plexi [20]. In absence of skull invasion, the haematogenous spreading of cancer cells to dura is the most common way. Mainly the spreading occurs by arterial circulation. Sgouros and Walsh described an unusual case of tentorial metastasis in association with occipital scalp metastases [21]. The authors hypothesized that tumor cells follow the route of the external carotid circulation. Other ways of dissemination are the extension through the lymphatic circulation.

The prognosis of patients with BM is poor; the definition of subgroups in relation to well-recognized prognostic factors is essential for the choice of the therapeutic strategy tailored to each patient. Although many patients with BM die because of extracranial disease progression, a significant amount suffer from the local tumor progression in the CNS [22]. In suspicion of BM the procedure should include a complete chest-abdomen CT with contrast and / or PET-CT to detect the presence of a neoplasia outside the CNS. In case of negative diagnostic investigations, the diagnosis of nature should be made by surgical excision or stereotaxic biopsy. The most appropriate treatment for BM is based on interdisciplinary evaluation and can be individualized based on primary tumor and on the extent and control of the systemic disease. Surgery, stereotactic radiosurgery, radiotherapy and chemotherapy must be integrated as therapeutic options, depending on the number, site and size of secondary brain lesions. According to the RTOG classification, the prognosis for patients with BM varies between 2.3 and 7.1 months in relation to the RPA class (recursive partitioning analysis class) in which they are identified [23]. However, there is no mention of DMs. DMs should be considered as a non-encephalic extra-axial lesion. Although the diagnostic work-up should be vital for patients, no guidelines on the management algorithm are reported in the literature. DMs are sometimes indistinguishable from other lesions using conventional MR. The symptoms are superimposable and the complications are similar. To differentiate a DM from a meningioma or from another mimicking primary tumor can lead to a different planning and to the most appropriate treatment.

We reviewed the most recent literature and compared the result of our series with the last epidemiological data. Our series is quite representative and accurate: DMs from prostatic carcinoma are not even the most common as reported in literature with an incidence of 6,25%. DMs from breast cancer had also a low incidence. In our series, lung cancer represents the most common neoplasm causing DMs. Primary tumor was unknown in two patients. None of our patients appeared to have cranial bone lesions.

Computed tomography and magnetic resonance are methods of choice to study DMs. CT scan with bone windows can delineate not only the metastatic lesion, but also bone involvement. On the other hand, MR gives a better contrast resolution, avoids bony artifacts and thanks to its multiplanarity leads to better delineate dural metastases and their connection with bone structures. DMs appear with several patterns: usually they look like a localized thickening of the dura-mater that sometimes could spread along the dura layer; sometimes, a nodular pattern is also evident. The alteration of blood-brain barrier causes an intense and homogeneous contrast enhancement. On CT images, DMs appear hyperdense and calcified. Soft tissue metastases invade and cause destruction of the adjacent bone, a prostate cancer causes osteoblastic metastases with hyperostosis, similar to meningiomas [24]. In MR lesions are usually isointense or hypointense on T1-weighted MR images and variable on T2 weighted images**.** MR provides also differential diagnosis between pachymeningeal and leptomeningeal involvement: in the first case, the dural enhancement involves the inner table of the skull and does not delineate the gyral circonvolutions, in contrast to leptomeningeal enhancement. Dural metastases shows a reduced perfusion with relative cerebral blood volume (rCBV) values less than 2 [25]. The exceptions are renal carcinoma, melanoma and Merkel cell neuroendocrine skin carcinoma metastases that show high perfusion due to their hypervascularization. In our case 16 (left temporo-occipital dural metastasis from lung adenocarcinoma) in the perfusion study, we found a significant increase in rCBV and KTrans (Figure **5**). This finding appears compatible with a neoformation characterized by a high neo-angiogenesis component. Melanoma metastases are typically hyperintense on T1WI. In MR spectroscopy there is an increased choline/creatine ratio, a prominent lipid peak, an occasional lactate peak and the absence of N-acetylaspartate (NAA) peak [26].

About 20% of DMs are clinically silent and they are an incidental finding during other medical investigations, follow-up examinations or autopsies. In the remaining cases, DMs have different clinical presentations, typically they behave as parenchymal

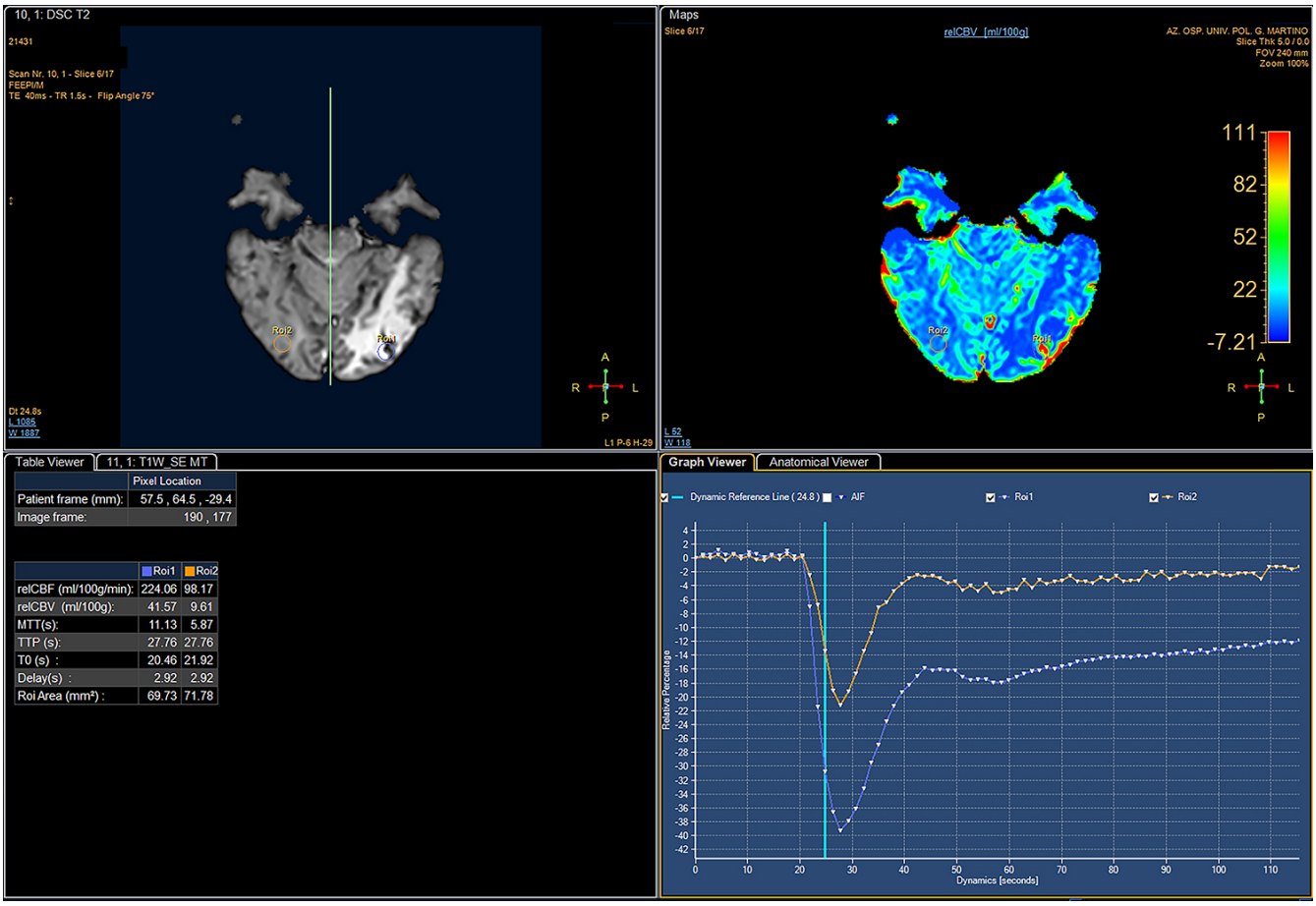

Figure 5: Case nr. 16: left temporo-occipital dural metastasis from lung adenocarcinoma. The perfusion and permeability study highlights a significant increase in rCBV and KTrans of the lesion at the left temporo-occipital site.

metastases causing compression or invasion of the underlying brain leading to an increasing of the intracranial pressure (23,5%), deficit (20%), coma (10%), seizure (9%), cranial neuropathy (10%), headache (7%), confusion (4,5%). In our series pattern of symptoms is quite similar to other kind of cerebral tumor, and it is different from the aforementioned reported series.

The surgical resection is the best procedure if there is a solitary, circumscribed and accessible lesion and the systemic disease is controlled. Some surgeons recommend resection even if there is a progression of the systemic disease and the metastases cause severe symptoms too. If DM is not well accessible, wide spreading or low patient's life expectancy, radiation therapy is indicated. Intrathecal chemotherapy has no indication in DMs. Systemic chemotherapy is usually associated with surgery and/or radiotherapy.

4. CONCLUSIONS

Neoplastic lesions of various histology and different organs can, although infrequently, cause dural metastases. Although they are uncommon, DMs can be mistaken for meningiomas and many other tumors or inflammatory or infective disease. Due to the uncertain diagnosis for the lack of specific diagnostic methods, despite primitive cancer was known in 14 out 16 patients, meningioma was proposed in all cases as the most possible diagnosis.

We recommend that any patient with primitive tumor known should be investigated with appropriate counseling and diagnostic workup. Despite the small size of our sample, our data showed a different prevalence of primary tumors. A retrospective multicenter study to update epidemiologic literature on such rare tumor is mandatory.

CONFLICT OF INTEREST

The authors declare no competing interests.

FUNDING

None.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by Medical Ethics Committee of University of Messina. Written informed consent was obtained from each patient. All applicable consents for publication were acquired.

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Received on 02-09-2022 Accepted on 05-10-2022 Published on 14-10-2022

https://doi.org/10.30683/1927-7229.2022.11.08