Pathology – Research and Practice xxx (2012) xxx–xxx

Contents lists available at SciVerse ScienceDirect

Pathology – Research and Practice

journal homepage: www.elsevier.com/locate/prp

Review

Meningeal tumors histologically mimicking meningioma

Valeria Barresi[a,*], Maria Caffo[b], Giovanni Branca[a], Rosario Caltabiano[c], Giovanni Tuccari[a]

[a] Department of Human Pathology, Section of Pathological Anatomy, University of Messina, Italy
[b] Department of Neurosciences, Section of Neurosurgery, University of Messina, Italy
[c] Department G.F. Ingrassia, Section of Pathological Anatomy, University of Catania, Italy

ARTICLE INFO

Article history:
Received 31 March 2012
Received in revised form 17 May 2012
Accepted 5 July 2012

Dedicated to Prof. Antonino Ferrara, on the occasion of his 90th birthday.

Keywords:
Meningioma
Mimicker
Mesenchymal
Metastasis
Immunohistochemistry

ABSTRACT

A number of meningeal neoplastic lesions may radiologically and clinically simulate meningioma. In the present paper, we review meningeal non-meningothelial tumors which may also mimic different histotypes of meningioma at the histological examination. Awareness that these lesions exist may facilitate their recognition and correct diagnosis, which is of fundamental importance for prognosis and an appropriate therapeutic approach. Histological and immunohistochemical clues for the differential diagnosis are discussed.

Contents

Introduction

Meningiomas account for approximately 20–30% of all primary intracranial tumors [18]. Though rare, a wide spectrum of dural and leptomeningeal masses may radiologically and histologically mimic meningioma. Due to their infrequency, these lesions may not be considered in the differential diagnosis toward meningioma during intra-operative examination; even more, due to their histological resemblance to different histotypes of meningioma (Table 1), postoperative diagnosis may be challenging as well. This review considers some of the relevant lesions of the meninges that may be mistaken for meningiomas clinically, radiographically and histologically. Awareness that they involve the meninges may facilitate their intraoperative and post-operative recognition which prevents unnecessary additional surgery and allows their correct management. In detail, we discuss the

* Corresponding author at: Dipartimento di Patologia Umana, Policlinico Universitario G. Martino, Pad D, Via Consolare Valeria, 98125 Messina, Italy.
Tel.: +39 902213549.
E-mail address: vbarresi@unime.it (V. Barresi).

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http://dx.doi.org/10.1016/j.prp.2012.07.002

histological and immunohistochemical (Table 2) clues useful for the differential diagnosis between these tumors and meningioma.

Mesenchymal tumors

Hemangiopericytoma (HPC)

Meningeal HPC represents the most frequent primary mesenchymal tumor of leptomeninges [47] and constitutes about 0.4% of all primary central nervous system (CNS) tumors [27]. Its histological similarity to meningioma is attested by its original classification as a variant of angioblastic meningioma [21]. Then, the first report of HPC in the meninges [9] noted that it had characteristics similar to the angioblastic meningioma described by Cushing.

Radiographically, HPC and meningioma show similar patterns of enhancement on computerized tomography (CT) and magnetic resonance imagining (MRI) (Fig. 1a) [17]. However, HPC is less likely to exhibit hyperostosis or intratumoral calcifications compared to meningioma, while it more frequently shows adjacent bone erosion [17]. Histologically, HPC is a highly cellular monomorphous tumor composed of sheets of cells with round or oval hyperchromatic nuclei. A well developed, variably thick-walled, branching staghorn vasculature is the most characteristic feature of HPC (Fig. 1a) [27]; in addition, mitotic figures are easily found in this tumor [27]. Due to its histological aspect and mitotic index, HPC may be mistaken for an atypical meningioma, but in comparison to this tumor, HPC carries a worse prognosis with almost inevitable recurrences, metastases to bones, lungs and liver and a probability of tumor-related death of 61% at 15 years [65]. As HPC may show compact areas with spindle cells, it may also mimic solitary fibrous tumor (SFT). On the whole, the characteristic vascular pattern, the absence of nuclear pseudo-inclusions and of true whorls, together with the increased mitotic activity, may establish the correct diagnosis. Silver impregnation for reticulin staining may be of use in the differential diagnosis versus meningioma; indeed, as a rule, reticulin invests individuals or small clusters of cells in HPC, while a coarse reticulin network between groups of cells is found in meningioma.

Moreover, several immunohistochemical markers may also be of help for the differential diagnosis. Indeed, unlike meningioma, HPC is generally negative for epithelial membrane antigen (EMA) (Fig. 1c); although focal weak immunoreactivity for EMA has been previously reported in HPC (52), this tumor lacks the strong and diffuse labeling for this antigen, which is typical of meningioma. In addition, staining for bcl-2 and CD34 is more frequently patchy in HPC (Fig. 1c and d), in contrast to the diffuse pattern observed in SFT [51]. As vimentin immunostaining is widespread in all three entities, it cannot be considered as a diagnostic aid [52].

Table 1
Mimickers of meningioma according to the histotype.

Histotype of meningioma	Histological mimicker
Meningothelial	Melanocytoma
Fibrous	Solitary fibrous tumor
	Meningeal Leiomyoma
Angiomatous	Hemangioblastoma
Clear cell	Hemangioblastoma
Chordoid	Intradural chordoma
	Hemangioendothelioma
Atypical	Hemangiopericytoma
	Meningeal Leiomyosarcoma
	Metastatic carcinoma
	Meningeal ES-PNET
	Leptomeningeal medulloblastoma
Anaplastic	Metastatic carcinoma
	Leptomeningeal medulloblastoma

Solitary fibrous tumor (SFT)

Initially described as a primary neoplasm of the pleura, solitary fibrous tumor (SFT) has been rarely reported also within the CNS [12,15,63]. While SFT and HPC have been recognized as a unique entity in the WHO classification of soft tissue tumors [24], they are considered separately in the WHO classification of CNS tumors [38]. Indeed, within the CNS, HPC undergoes a more aggressive course in comparison to SFT, although these two entities display overlapping histological and immunohistochemical features, and they have been suggested to belong to the same spectrum of tumors [12]. Primitive CNS HPC needs additional treatments following surgery, as it may develop recurrences and distant metastases [63,65]; thus, its differentiation from SFT is of considerable importance for therapeutic and prognostic purposes [63]. Accordingly, in the WHO classification of CNS tumors, HPC figures as a grade II neoplasia [27].

SFT, HPC and meningioma show overlapping radiological aspects [63]. In addition, both SFT and meningioma present in the same age group and show a strong female predominance [15,63]. The typical histological features of SFT are spindle cells arranged in a pattern-less architecture or in interlacing fascicles, prominent collagenous bands and branching vascular channels with thin walls. Thus, the tumor may radiologically, clinically and histologically mimic fibrous meningioma. However, unlike fibrous meningioma, SFT usually exhibits different histologic patterns throughout the tumor. In addition, whorls, storiform patterns and psamomma bodies are generally absent in SFT, and its spindle cells lack the pseudoinclusions characteristic of meningioma. The absence of

Table 2
Expression of different immunohistochemical markers in meningioma and its mimics.

Tumors	Immunohistochemical markers												
	EMA	CD34	bcl2	SMA	Muscle specific actin	Desmin	CD31	Synaptophysin	CD99	Pankeratin	S100	Brachyury	HMB-45
Meningioma	+++	–	–	–	–	–	–	–	–	–/+	–/+	–	–
HPC	–/+	+	+	–	–	–	–	–	–	–	–	Unknown	–
SFT	–	++	++	–	–	–	–	–	–	–	–	Unknown	–
Smooth muscle tumors	–	–	–	+++	+++	+++	–	–	–	–	–	Unknown	–
Epithelioid Hemangioendothelioma	–	+++	–	–	–	–	+++	–	–	–/+	–	Unknown	–
ES-pPNET	–	–	–	–	–	–	–	+++	+++	–	+++	Unknown	–
Metastatic carcinoma	+++	–	–	–	–	–	–	–	–	+++	–	–	–
Chordoma	+++	–	–	–	–	–	–	–	–	+++	+++	+++	–
Medulloblastoma	–	–	–	–	–	–	–	++	–	–	–	Unknown	–
Melanocytoma	–	–	–	–	–	–	–	–	–	–	+++	Unknown	+++

+++: expression in the majority of cells; ++: expression in about 50% of cells; +: patchy expression; –/+: focal expression in a few cases.

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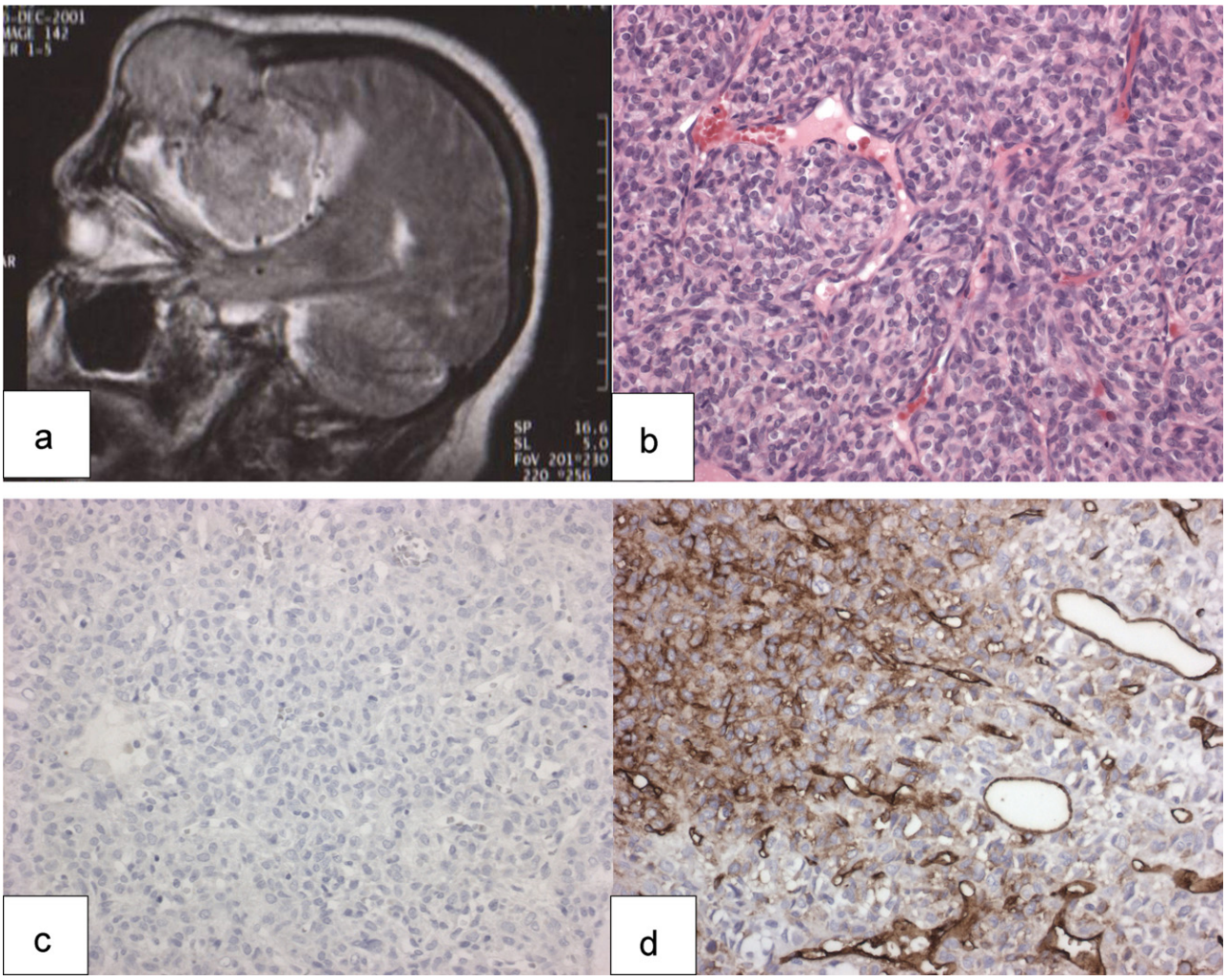

Fig. 1. (a) MNR imaging of HPC. (b) At the histological examination, the tumor is composed of elongated cells with oval nuclei intermingled with a staghorn vasculature (hematoxylin and eosin stain; original magnification, 200×). Immunohistochemistry documents (c) absence of epithelial membrane antigen (EMA) (EMA stain; original magnification, 200×) and (d) a patchy staining for CD34 in the neoplastic cells (CD34 stain; original magnification, 200×).

EMA staining in SFT definitely helps in the differential diagnosis toward meningioma, which instead exhibits a diffuse immunoreactivity for this antigen [15,52]. A capillary pattern and areas with increased cellularity in SFT may suggest HPC, although, as specified above, CD34 and bcl-2 staining may be of use in order to differentiate between these two entities.

Smooth muscle tumors (leiomyoma and leiomyosarcoma)

Primary dural-based mesenchymal smooth muscle tumors have been rarely described within the CNS [3,10,33,43]. They mainly arise in males, in association with human immunodeficiency virus-1 infection and in immunocompromised individuals [10,33,43]. Meningeal metastatization from peripheral leiomyosarcoma is also a rare event [39]. Meningeal smooth muscle tumors present as solitary, extra-axial masses with dural adhesion, radiologically suggesting meningioma [3,33]. In addition, also the histological aspect of these tumors may simulate meningioma; in particular, as it is composed of spindle cells, leiomyoma may simulate fibrous meningioma, while meningeal leiomyosarcoma, which tends to be histologically low-grade and is characterized by scattered mitoses, may be erroneously classified as atypical meningioma. The histological characteristics of primitive meningeal smooth muscle tumors showing cigar-shaped nuclei in the absence of nuclear pseudoinclusions may help to define the correct diagnosis. Finally, the immunohistochemical profile with expression of at least one of the smooth muscle markers, such as smooth muscle actin (SMA), muscle-specific actin, or desmin, in the absence of staining for EMA, has been considered diagnostic for these neoplasias [33,34].

Epithelioid hemangioendothelioma

Epithelioid hemangioendothelioma (EH) was first described as a rare vascular tumor of soft tissues [69], with a biological behavior intermediate between malignant angiosarcoma and benign angioma [35]. CNS involvement is rare, with 37 cases reported in the literature [42,51,70]. Differently from soft tissues EH, intracranial cases affect a notable number of individuals at pediatric age [35]. Intracranial EH may present as a meningeal extra-axial mass showing a cystic aspect and contrast enhancement at MRI [51], which may simulate meningioma. Histologically, its most common aspect displays cells arranged in cords or branching pattern and embedded in variable amounts of myxoid or fibrohyaline stroma [35]. Due to the presence of cords and myxoid stroma, EH may be reminiscent of chordoid meningioma. Nonetheless, differently from the latter, EH is negative for EMA, and expresses endothelial markers CD31

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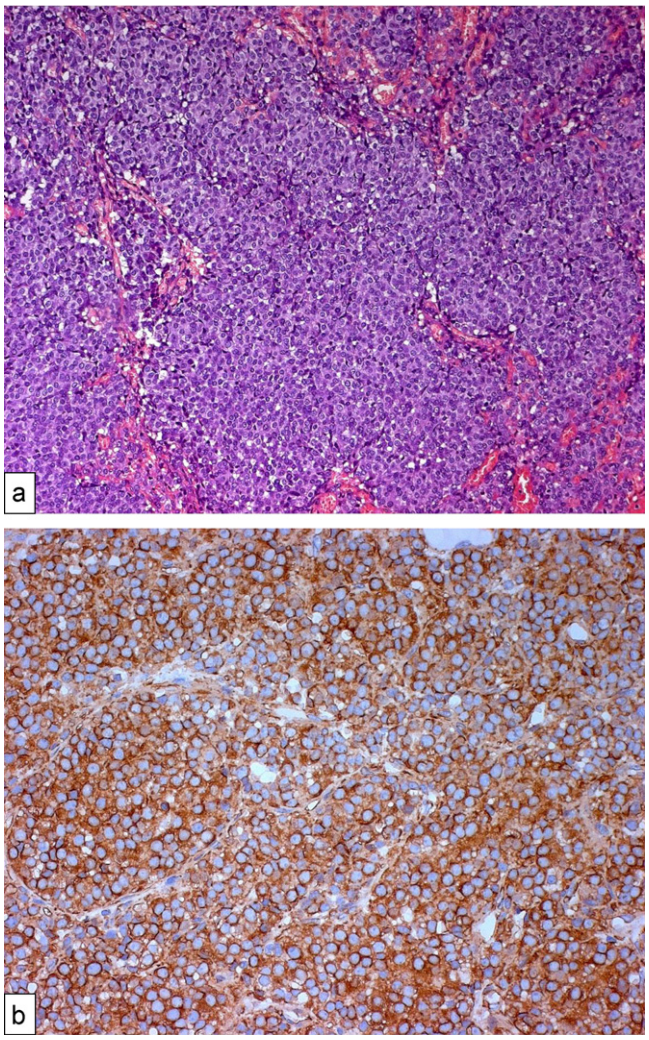

Fig. 2. (a) Histological aspect of meningeal pPNET-ES with nests or lobules of neoplastic uniform round cells (hematoxylin and eosin stain; original magnification, 100×). (b) Diffuse, strong membrane expression of CD99 in the same cells (CD99 stain; original magnification 200×).

and CD34 [35,51]. Distinction between the two entities may have a prognostic significance as, though rarely, metastatic dissemination has been reported in EH [51].

Peripheral primitive neuroectodermal tumor-Ewing sarcoma (pPNET-ES)

pPNET-ES is a small round cell tumor mainly involving soft tissues and bones [24] but rarely arising as a primary dural-based neoplasm [4,5,23,50], radiologically mimicking meningioma [23]. Genetically, pPNET-ES is characterized by a specific chromosomal translocation resulting in the oncogenic activation of the EWS gene. The most frequent chromosomal translocation, encountered in 90% of pPNET-ES, is t(11,22) (q24;q12), while about 10% of pPNET-ES show the unusual translocation t(21;22) (q22;q12) [4]. pPNET-ES is histologically composed of sheets of monotonous small cells with round nuclei and finely dispersed chromatin and inconspicuous nucleoli [5] (Fig. 2a). Due to its histological features, it may simulate meningioma [4] or central PNET (cPNET) [50]. Distinction from these entities is of crucial importance from therapeutic and prognostic viewpoints. Indeed, pPNET-ES carries a more favorable prognosis compared to cPNET, due to its sharp margins and broad implantation to the dura which allow radical surgical resection [4]; in addition, differentiation from atypical meningioma is fundamental as pPNET needs to be submitted to additional combined chemotherapy and radiotherapy in order to prevent local recurrences [4]. Thus, despite its rarity, primitive meningeal pPNET-ES has to be kept in mind in the differential diagnosis of meningeal tumors.

Periodic Acid Shiff (PAS) staining highlights thin rims of diastase-sensitive, glycogen-rich cyroplasm in the neoplastic cells of pPNET-ES. Also, immunohistochemistry is helpful in the differential diagnosis toward meningioma, as pPNET shows strong and diffuse membrane immunoreactivity for CD99 (Fig. 2b), at least focal staining for neuronal markers such as synaptophysin and neuron-specific enolase (NSE), and lack of EMA labeling [49]. Nevertheless, other tumors, such as cPNET or medulloblastoma, may show a similar immunohistochemical profile [49]; thus, the diagnosis of pPNET-ES needs a genetical confirmation through real time polymerase quantitative reaction (RT-PCR) or fluorescence in situ hybridization (FISH) in order to confirm the presence of the translocations mentioned afore [49].

Metastatic carcinoma

Intracranial metastases constitute a significant cause of mortality and morbidity in cancer patients [44]. Brain parenchyma is the most common site involved by tumor metastases, but dural compartment may also be affected [44]. Dural metastases develop in about 9–10% of all patients with systemic cancer [54,61]. Breast, prostate and lung cancers represent the most common sources [34,44,58], although dural metastases from renal cell carcinoma, thyroid carcinoma, carcinoids, thymic carcinoma, squamous cell cancer or adenocarcinoma of unknown primary origin have been reported [44]. Due to their rarity and presentation as single masses with dural enhancement, dural metastases may radiologically be misinterpreted as meningiomas; radiological diagnosis may be even more challenging in the presence of the "dural-tail" sign, traditionally regarded as a typical feature of meningioma [44] (Fig. 3a). In addition, the pre-surgical differential diagnosis between metastatic carcinoma and meningioma may be particularly difficult in women with primary breast cancer, who are known to be also at increased risk of meningioma [22]. The histological examination of the tumor usually reveals its nature. In particular, cribriform architecture and prominent nucleoli favor a diagnosis of metastatic prostatic cancer, whereby ductal growth with desmoplasia is a diagnostic feature of breast cancer [58]. Nonetheless, lobular growth of metastatic carcinoma may mimic whorls or a syncytial pattern, raising the suspicion of meningioma [32]. In addition, the differential diagnosis toward atypical or malignant meningioma may be particularly challenging in the case of undifferentiated metastatic carcinoma. However, distinction of these entities is extremely important due to their markedly different treatment and prognosis. Although meningiomas have been traditionally considered negative for cytokeratins [28], some authors found the expression of these intermediate filaments in malignant meningiomas [30,37], questioning the value of immunohistochemistry against cytokeratins for the differential diagnosis versus metastatic carcinoma. In order to test the diagnostic value of cytokeratin expression for the differentiation of these lesions, we evaluated the immunohistochemical expression of wide-spectrum cytokeratins (clone AE1-AE3; Dako Cytomation, Glostrup, Denmark; working dilution, 1:50; micro-wave pre-treatment) in a cohort composed of 8 atypical and 4 anaplastic formalin-fixed and paraffin embedded meningiomas resected from patients with no evidence or clinical history of systemic cancer (*unpublished data*). Cytokeratins staining was evidenced in 6 cases, and, specifically, in 3 atypical and 3 anaplastic meningiomas. Nevertheless, in all the positive cases

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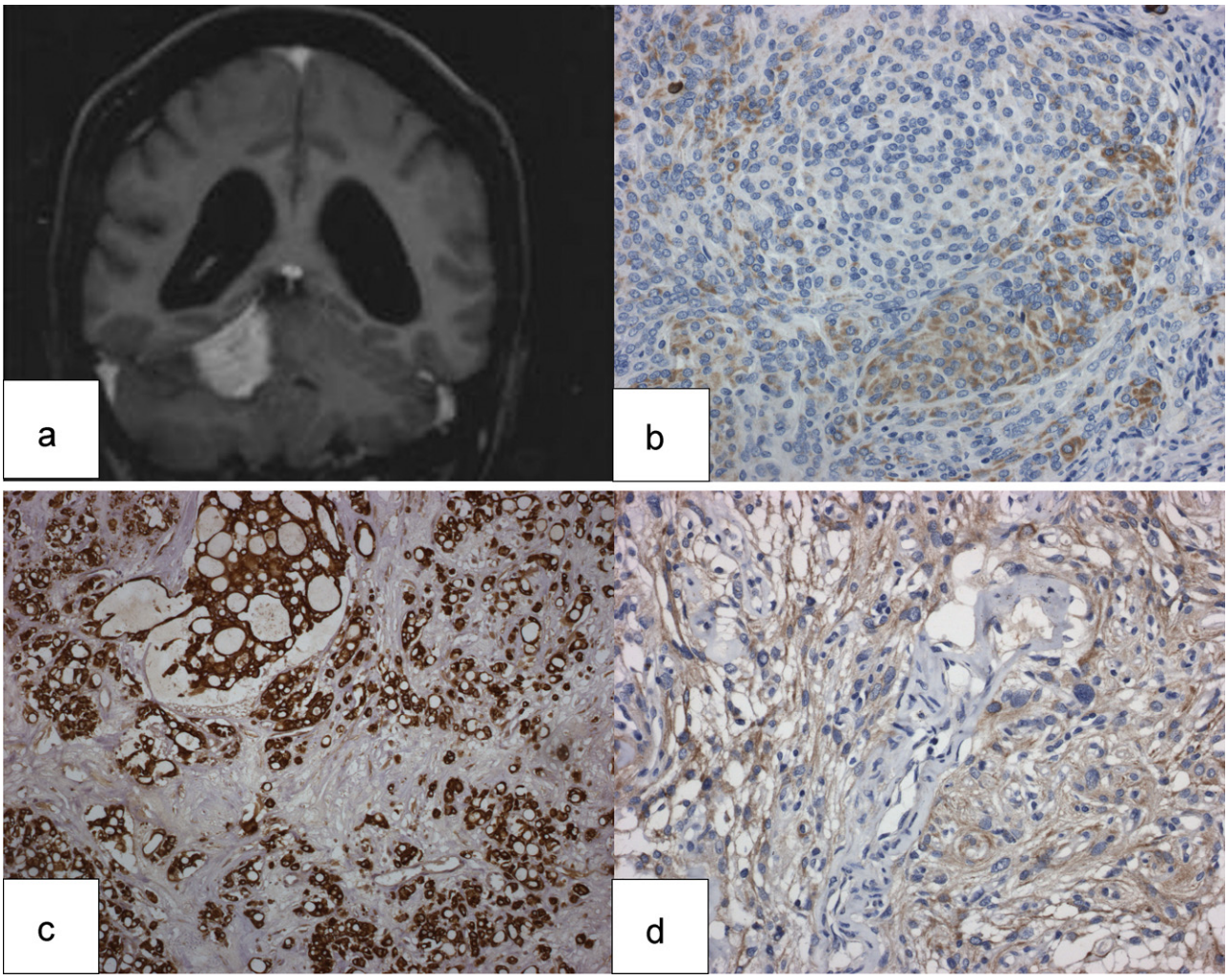

Fig. 3. (a) NMR imaging of a meningeal metastasis from a breast carcinoma, showing a well demarcated extra-axial mass with contrast enhancement and dural tail sign. (b) Wide-spectrum cytokeratins staining in atypical meningioma (pankeratin stain; original magnification, 200×), and in dural metastasis from carcinoma (c) (wide-spectrum cytokeratin stain, original magnification, 100×). (d) Keratin-18 positive staining in the neoplastic cells of an atypical meningioma (cytokeratin-18 stain; original magnification, 200×).

staining was focal and involved a small percentage (<20%) of neoplastic cells (Fig. 3b), in contrast to the diffuse and strong positivity commonly found in metastatic carcinoma (Fig. 3c). Thus, we believe that a diffuse and strong staining for wide spectrum cytokeratins argues against a diagnosis of anaplastic or atypical meningioma and that it may be helpful in order to differentiate these tumors from metastatic carcinoma. Besides, cytokeratin expression has been shown to remain relatively stable as carcinomas evolve and metastasize [66]. Among the other cytokeratins, cytokeratin-18 has been reported to be the most frequently expressed in meningioma (Fig. 3d) [41]; thus, it may not be very useful for the differential diagnosis between meningioma and metastatic carcinoma. On the other hand, antibodies against cytokeratins-7, -8 and -19 seem to be more useful in this context, since they only rarely label a significant number of meningioma cells, whereas they are very often positive in different carcinomas, especially adenocarcinomas [41]. Positivity for cytokeratin-20 strongly supports the diagnosis of carcinoma, as this cytokeratin is not expressed by meningioma [41].

Intradural chordoma

Chordomas are uncommon bone tumors that arise from embryonic remnants of the notochord; their most frequent localization is the sacrococcygeal region, followed by the spheno-occipital and the vertebral regions. Because of their tendency to invade and destroy the involved bone, chordomas are considered malignant tumors with local aggressiveness. They are mainly extra-dural tumors that may secondarily invade the dura; however, cases of intradural chordomas without bone involvement have been rarely described [6,11,60]. Prognosis of intradural chordomas seems to be better than that of their extra-dural counterparts, as their sharply circumscribed margins and the absence of bony invasion make them amenable to complete excision. Although only data on mid-term follow-up are available, neither recurrences after total surgical excision nor re-growth following subtotal removal have been reported [11]. The radiological aspect of intradural chordoma, showing a well-circumscribed, contrast-enhanced, extra-axial mass without bone invasion, may suggest meningioma (Fig. 4a) [6]. Due to its intradural location and microscopic aspect, with eosinophilic epithelioid cells arranged in cords within a mucoid matrix, intradural chordoma may be mistaken for chordoid meningioma (Fig. 4b). Chordoma was originally described as one of the unique "triple positive" EMA/S100/keratins neoplasia in bone and soft tissue pathology [1,19,46]. In particular, diffuse immunostaining for wide spectrum cytokeratins, cytokeratin-8, cytokeratin-19 and cytokeratin-18 was demonstrated in chordoma, while labeling for cytokeratin-7 was present in sporadic cases [46]. Thus, a panel including EMA, cytokeratins and S100 may be helpful

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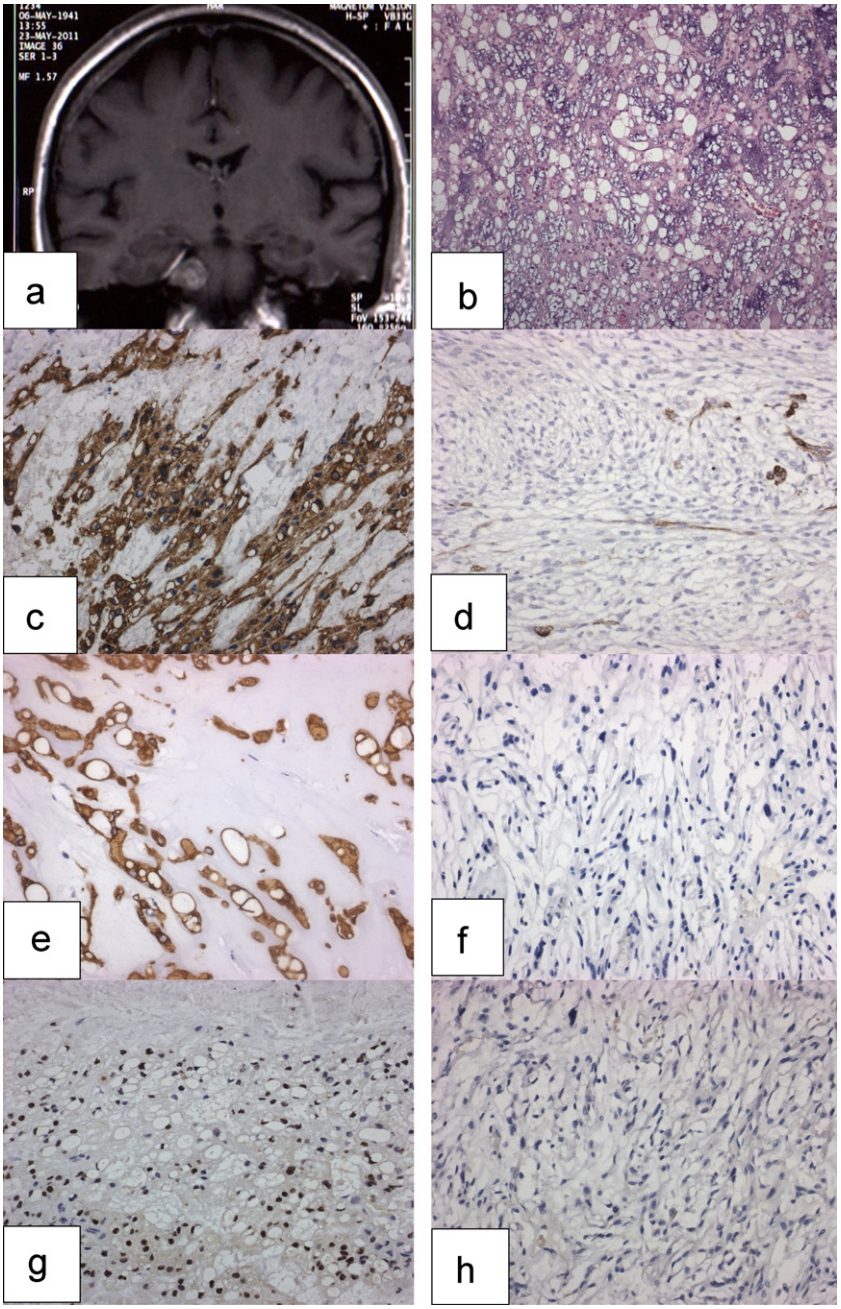

Fig. 4. (a) NMR imaging of an intradural chordoma, showing a well demarcated extra-axial mass with contrast enhancement and dural tail sign. (b) Histological aspect of intradural chordoma, with cords of eosinophilic cells in a mucoid background (hematoxylin and eosin stain; original magnification, 100×). Wide spectrum cytokeratin, S100 and brachyury stain in intradural chordoma (c: wide spectrum cytokeratin stain; original magnification, 100×; e: S100 stain; original magnification, 200×; g: brachyury stain, original magnification, 200×) and in chordoid meningioma (d: wide spectrum cytokeratin stain; original magnification, 200×; f: S100 stain; original magnification, 200×; h: brachyury stain, original magnification, 200×).

for the differential diagnosis between chordoma and chordoid meningioma. Indeed, although the expression of wide spectrum cytokeratins and that of S100 was reported in chordoid meningiomas [57], only focal and weak staining for these antigens was found in the positive cases [57], in contrast to the strong and diffuse staining expected in chordoma [6,46]. D2-40, which is expressed in chordoid meningioma but not in chordoma, was indicated as an additional potential marker useful to distinguish between these lesions [29]. Finally, the immunohistochemical detection of nuclear brachyury, the transcription factor protein product of a T-box gene which regulates the formation of the mesoderm and notochord in humans [53,55], has been demonstrated to be a sensitive and specific marker for chordoma in the differential diagnosis toward other neoplasias showing a chordoid appearance [6,31,57,64].

In order to test their diagnostic value in the differential diagnosis between chordoid meningioma and chordoma, we analyzed the immunohistochemical expression of S100 (Dako Cytomation, Glostrup, Denmark; working dilution: 1:400; pronase pre-treatment), wide spectrum cytokeratins (clone AE1-AE3; Dako Cytomation, Glostrup, Denmark; working dilution, 1:50; micro-wave pre-treatment), cytokeratin 7 (clone OV-TL 12/30; Dako Cytomation, Glostrup, Denmark; working dilution: 1:100; micro-wave pre-treatment), cytokeratin-8 (clone 35BH11; Abcam, Cambridge, United Kingdom; working dilution: 1:100; micro-wave

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pre-treatment), cytokeratin-18 (clone DC 10; Dako Cytomation, Glostrup, Denmark; working dilution: 1:100; micro-wave pre-treatment), cytokeratin-19 (clone RCK 108; Dako Cytomation, Glostrup, Denmark; working dilution: 1:100; micro-wave pre-treatment), D2-40 (Dako Cytomation, Glostrup, Denmark; working dilution: 1:100; micro-wave pre-treatment) and brachyury (clone N-19; Santa Cruz Biotechnology, Heidelberg, Germany; working dilution: 1:50; micro-wave pre-treatment) in a cohort including 10 formalin-fixed and paraffin-embedded chordoid meningiomas, as well as 10 chordomas (*unpublished data*).

We found strong and diffuse staining for S-100, wide spectrum cytokeratins, cytokeratin-8, cytokeratin-18, cytokeratin-19 and brachyury in all the chordomas analyzed (Fig. 4c–g). On the other hand, in line with the findings reported by Sangoi et al. [57], only a focal positivity for wide-spectrum cytokeratins and S100 was evidenced in one out of the 10 chordoid meningiomas analyzed (Fig. 4d and f), while no staining for cytokeratins -8 and -19 and for brachyury was found in all of the chordoid meningiomas (Fig. 4h). Staining for cytokeratin-18 was present and widespread in all of the chordomas and in 4/10 chordoid meningiomas, while cytokeratin-7 and D2-40 antibodies failed to stain all the cases. Thus, in the presence of an intradural mass showing a chordoid appearance, we believe that diffuse and strong staining for wide spectrum cytokeratins and S100 argues against the diagnosis of chordoid meningioma and rather suggests chordoma, while cytokeratin-18 is not of use for the differential diagnosis between chordoma and chordoid meningiomas, as it stains both the lesions. Brachyury may be used as an additional marker for chordoma in doubtful cases. In our opinion, differently from that reported by other authors [29], D2-40 staining is not useful in the differential diagnosis between these lesions.

Leptomeningeal medulloblastoma

According to the WHO classification of tumors of the CNS, medulloblastoma is defined as a malignant, invasive embryonal tumor of the cerebellum with preferential manifestation in children and with a tendency to metastasize via the cerebrospinal fluid pathways [26]. It may rarely arise in adults, mostly in the third and fourth decade [26]. While most childhood medulloblastomas arise in the vermis, a hemispheric location is more common in adults, and its close proximity to the dura may give extra-axial imaging characteristics simulating meningioma [8,25]. In addition, hemispheric medulloblastoma may radiologically mimic meningioma in the presence of dural tail sign [25].

Rare cases of primary leptomeningeal medulloblastomas have been reported [40,56]. In all the cases, MRI revealed diffuse leptomeningeal enhancement, with no evidence of a solid intraparenchymal mass [40,56], which makes it challenging to suspect the presence of a tumor with only radiological examination. We have recently observed the first case of primary meningeal medulloblastoma presenting as a solid mass. The tumor was resected from a 36 year-old male patient referred to our hospital for symptoms consisting of vertigo of one month duration. The MRI showed a lobulated mass in the posterior fossa with inhomogeneous contrast enhancement and dural tail sign, in the absence of cerebellar involvement (Fig. 5a). The mass was resected under suspicion of an atypical or anaplastic meningioma. At surgery, the tumor was located beneath the arachnoid and it was implanted on the tentorium, in the absence of any adhesion to the cerebellar parenchyma, which allowed total removal. At intraoperative examination with frozen sections, overtly atypical cells showing frequent mitoses were recognized in a desmoplastic stroma; however, the extra-axial location of the mass was misleading and a diagnosis of atypical meningioma was made. The surgical specimen was formalin-fixed and paraffin-embedded for histological examination with permanent sections. At the microscopic evaluation with hematoxylin and eosin stain, the tumor was composed of blue cells, showing nuclei with moderate variation in size and shape and arranged in large patternless areas with dense background desmoplasia (Fig. 5b), although some nodular areas were also recognized. The presence of nodules and the desmoplastic stroma, together with the high mitotic index, raised the suspicion of a desmoplastic medulloblastoma. Then the diagnosis was confirmed through immunohistochemistry, which demonstrated absence of staining for EMA (Fig. 5c) (Clone E29; Dako Cytomation, Glostrup, Denmark; working dilution, 1:100; microwave pre-treatment), wide spectrum cytokeratins (clone AE1-AE3; Dako Cytomation, Glostrup, Denmark; working dilution, 1:50; micro-wave pre-treatment), CD99 (clone 12E7; Dako Cytomation, Glostrup, Denmark; working dilution, 1:50), and positive labeling for synaptophysin (Fig. 5d) (clone SY38; Dako Cytomation, Glostrup, Denmark; working dilution, 1:100) and Ki-67 (clone MIB-1; Dako Cytomation, Glostrup, Denmark; working dilution, 1:100; micro-wave pre-treatment) with a labeling index of 50%. Also, reticulin stain highlighted the presence of desmoplasia throughout the tumor, as well as the presence of small differentiating nodules consistent with nodular/desmoplastic medulloblastoma. Following the diagnosis, the patient was submitted to specific chemotherapy and radiotherapy treatments; he was alive with no evidence of disease after a six-month follow up.

Melanocytic tumors

According to the WHO 2007 classification, melanotic neoplasms of the central nervous system (CNS) that arise from leptomeningeal melanocytes include diffuse melanocytosis, melanomatosis, melanocytoma, and malignant melanoma [14].

Meningeal melanocytoma is a rare neoplasm and accounts for 0.06–0.1% of brain tumors [36]. It is a solitary, benign, low grade tumor, occurring in all ages ranging from 9 to 73 years, but most frequently in the fifth decade with a slight female predominance [13]. On RMI, melanocytoma appears as a well circumscribed, hyperintense, extra-axial mass arising with homogeneous contrast enhancement, mimicking meningioma [14]. Histologically, the spindle or oval tumor cells show a nested and whirling pattern, resembling meningioma. The distinguishing feature of melanocytoma is the presence of heavily pigmented cells, which account for the red-brown, black, color observed macroscopically [14]. Indeed, melanocytomas were once referred to as melanotic meningioma [13]. Following the ultrastructural demonstration that pigmented cells are immature premelanosomes and mature melanosomes, lacking the characteristics of meningothelial cells, namely desmosomes, interdigitating cytoplasmic processes and intracytoplasmic fibrils, the term melanotic or pigmented meningioma was abandoned [13,36]. A further characteristic feature of melanocytoma is the presence of grooves in the nuclei of the neoplastic cells, which may be of help in case of amelanotic cases. Then, melanocytoma typically should not have nuclear pleomorphism, atypia or macronucleoli, and contain no more than occasional mitotic figures, or exhibit necrosis [14]. The final diagnosis may be obtained through immunochemistry and in challenging cases through electron microscopy. Meningeal melanocytomas are strongly immunoreactive for S-100 protein, HMB-45, and vimentin and are nonreactive for epithelial membrane antigen and glial fibrillar acid proteins [13,45]. Although meningeal melanocytoma is commonly regarded as a low grade neoplasm [14], similarly to meningothelial meningioma, the differentiation of these two entities is of crucial importance. Indeed, cases of malignant transformation of partially removed melanocytoma to melanoma have

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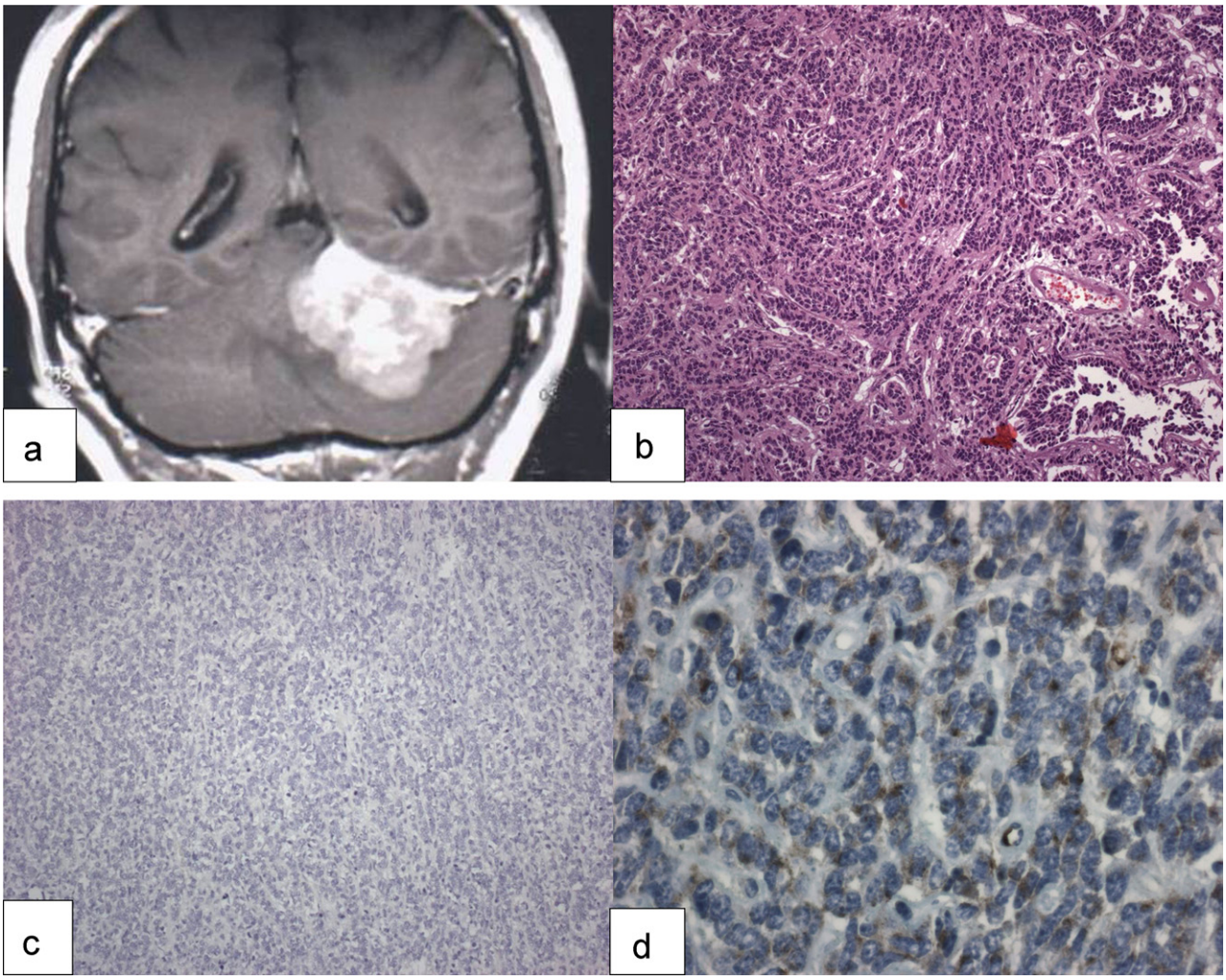

Fig. 5. (a) NMR imaging of leptomeningeal medulloblastoma showing a lobulated mass, with inhomogeneous contrast enhancement and dural tail sign. (b) Histological aspect of the tumor with cords of neoplastic cells in a desmoplastic stroma (hematoxylin and eosin stain; original magnification, 100×). (c) Absence of EMA staining (EMA stain; original magnification, 100×) and (d) dot-like positivity for synaptophysin (synaptophysin stain; original magnification, 200×) in the tumor cells of meningeal medulloblastoma.

been reported [67,68], showing that the biological behavior of this neoplasm is uncertain and that physicians should be cautioned that a meningeal melanocytoma may represent a precursor of more aggressive lesions.

Hemangioblastoma

Hemangioblastoma (HBL) is a grade I CNS neoplasm, which accounts for 1.5–2.5% of all intracranial tumors [16] and which is included among the meningeal tumors in the WHO 2007 Classification [2]. The posterior fossa, and especially the cerebellum, represents the most frequent site for HBL, but this tumor may also arise in the cerebral hemispheres, medulla and spinal cord. In addition, supratentorial leptomeningeal hemangioblastomas have been reported [59,62]. HBL may occur as a sporadic entity, due to sporadic mutations of the Von Hippel-Lindau (vHL) gene, or as a component of the Von Hippel-Lindau (VHL) syndrome. The latter is an autosomal dominant syndrome, characterized by germline mutations of the vHL gene and by the predisposition to develop HBL in the CNS and clear cell renal cell carcinomas (CRCC) in the kidney [20].

Radiologically, HBL presents as a gadolinium enhancing extra-axial mass with or without associated cyst (Fig. 6a). According to the WHO Classification of tumors of CNS, HBL is defined as a slow-growing, highly vascular tumor, histologically composed of stromal cells and small blood vessels (Fig. 6b) [2]. The most characteristic feature of the stromal cells of HBL is the presence of numerous lipid-containing vacuoles which give them a clear-cell appearance. Due to its high vascularity, this tumor may mimic angiomatous meningioma, which is characterized by a predominance of blood vessels over that of tumor cells [53]; on the other hand, the stromal cells of HBL may also simulate the neoplastic cells of clear cell meningioma at the histological examination [48]. Supratentorial meningeal HBL also needs to be differentiated from meningioma [59,62]. The distinction of angiomatous meningioma from HBL is irrelevant from the therapeutic viewpoint, as both are benign tumors, but in the case of confirmed diagnosis of HBL, further analyses are warranted in order to exclude VHL disease. The differential diagnosis of HBL toward clear cell meningioma may be more significant, as this variant of meningioma is associated with adverse prognosis and increased risk of recurrence [53]. Immunohistochemistry may be helpful in the differential diagnosis of these lesions, as HBL is negative for EMA, which stains instead

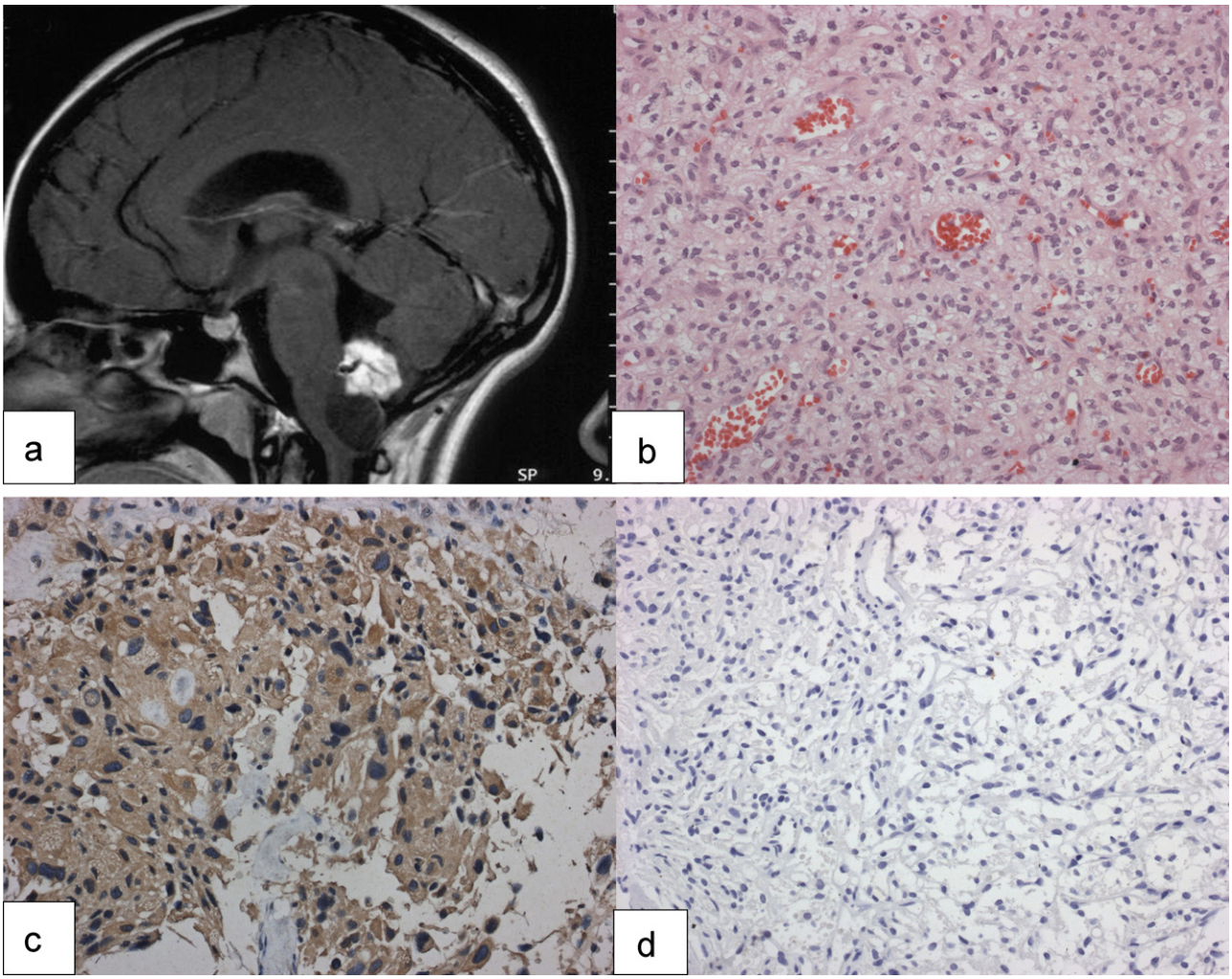

Fig. 6. (a) NMR imaging of a solid hemangioblastoma, showing a well demarcated hyperdense mass. (b) Histological aspect of hemangioblastoma, showing small blood vessels and clear stromal cells (hematoxylin and eosin stain; original magnification, 100×). (c) Brachyury stain in hemangioblastoma (original magnification, 200×) and in (d) angiomatous meningioma (original magnification, 200×).

the neoplastic cells of meningioma. In addition, we have recently shown that brachyury is a marker of the stromal cells of HBL, useful in the distinction of the latter from meningioma (Fig. 6c and d) [7].

Conclusions

A number of neoplastic meningeal lesions may radiologically mimic meningioma and even show the dural tail sign characteristic of this neoplasia. The clinical features of some of these mimickers, in terms of age and sex distribution, may be further misleading, supporting a pre-surgical diagnosis of meningioma. Thus, the histological examination of these lesions is of fundamental importance in order to confirm or rebut the diagnosis of meningioma. Nonetheless, some of the mimickers are also reminiscent of the different histotypes of meningioma histologically. Awareness of their existence may facilitate their recognition, which is of fundamental importance from the prognostic viewpoint and for an appropriate therapeutic approach. Indeed, if a correct diagnosis is not established, oncologic therapy may be delayed under the mistaken impression of meningioma, causing deleterious effects on patient care. Careful examination of the histological aspect of the tumor, as well as immunohistochemistry, may be helpful in order to achieve the correct diagnosis.

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