Pathology & Oncology Research (2020) 26:1313–1318
https://doi.org/10.1007/s12253-019-00704-2

ORIGINAL ARTICLE

Spinal Versus Intracranial Meningioma: Aberrant Expression of CD10 and Inhibin with Relation to Clinicopathological Features and Prognosis

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Received: 30 October 2018 / Accepted: 25 July 2019 / Published online: 1 August 2019

Abstract
CD10 and inhibin are used mainly in CNS pathology to distinguish hemangioblastoma from metastatic clear cell renal cell carcinoma. Some meningiomas can mimic both tumors and so we aimed at this study to investigate the expression of both markers in a large number of meningioma cases. One hundred thirty-four meningioma samples were collected, 14 of them were spinal and 120 were intracranial. Manual TMA blocks were constructed using modified mechanical pencil tip method and immunohistochemistry for CD10 and inhibin was done. Intracranial meningioma occurred in significantly younger age than spinal ones. Most of spinal meningiomas were of transitional histology. CD10 was expressed in 14% of cases with significant positivity in spinal rather than intracranial cases. Transitional meningiomas showed the highest positivity for CD10 expression, while the least positive was the meningiotheliomatous type. Inhibin was expressed in 6% of cases with no significant relation to clinicopathological and histological features. There was no significant relationship between the expression of CD10 and inhibin expression in meningiomas. In conclusion, spinal meningiomas differ than intracranial ones in many clinicopathological and biological aspects. Among these differences is CD10 expression being more expressed in spinal meningiomas. However CD10 and inhibin are aberrantly expressed in a proportion of meningiomas, both have no relations to poor prognostic factors but more caution should be exerted during usage of these markers in diagnosis of hemangioblastoma and metastatic RCC. Further studies are suggested for exploring more biological differences between spinal and intracranial meningiomas.

Keywords CD10 · Inhibin · Spinal · Intracranial · Meningioma

Introduction

Meningiomas are common tumors of the CNS originating from the meningeal coverings of the brain and spinal cord and are derived from arachnoidal cap cells [1]. They can arise in any location where meninges exist, as in the middle ear, the paranasal sinuses, the nasal cavity, and also the mediastinum [2]. About 34% of all primary CNS tumors diagnosed in USA are meningiomas, and hence they are the most common primary intracranial neoplasms in adults [3, 4].

Meningiomas are classified according to the latest 2016 WHO classification of CNS tumors into three major groups; WHO grades I (benign), II (intermediate) and III (malignant or anaplastic). There are further 15 histological and cytomorphological subvariants, of which 9 variants are benign, 3 variants are intermediate and 3 variants are malignant. The most common grade I histomorphological variants are meningothelial, fibrous and transitional subtypes. Grade II (atypical) meningiomas are defined by the presence of 4–19 mitotic index /10 high power fields (HPFs) or 3 of the following 5 criteria: high N/C ratio, increased cellularity, sheet-like growth, prominent nucleoli, and spontaneous necrosis. Grade III meningiomas exhibit mitoses of 20 or greater /10 HPFs and have the most aggressive clinical course with tumor recurrences and high mortality [5].

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In addition to histological characterization of meningiomas, important breakthroughs were achieved in determining the molecular and genetic alterations of meningiomas and their relation to the disease behavior. Several tumorigenesis pathways targeting different genes in low versus high grade tumors were explored [6]. Inactivating mutations of NF2 and monosomy 22 are the most common genetic alterations in meningiomas. More recently, next-generation sequencing helped to identify mutations in AKT1, SMO, TRAF7, PIK3CA and KLF4 with relations to tumor location and some histologic types especially secretory meningioma [7, 8]. In addition, other chromosomal and/or genetic alterations are detected in about one third of cases involving several intracellular signaling pathways associated with cell migration, proliferation and apoptosis. Accumulation of these cytogenetic alterations leads to higher tumor grade, more aggressive disease and more tumor recurrence. So, it is crucial to assess tumor cytogenetics together with histopathological and clinical features for tailoring the most appropriate targeted therapy and for optimal follow-up [6].

Classified into spinal and intracranial meningiomas, studies have shown that spinal meningiomas have some specific clinical and genetic characters. They have a higher incidence in females as compared to intracranial tumors. Most spinal tumors are grade I, mostly of meningothelial, psammomatous and transitional variants. In contrast, about 10% of intracranial meningiomas are of grade II or III. Moreover, many specific patterns of gene expression profiles were detected in spinal as compared to intracranial meningiomas [9]. The clinical course after a surgical resection also differs with variable biologic tendency for invasiveness, and hence the importance of investigating the expression of different biological markers that predict their clinical course [4].

CD10 (synonymous with CALLA, NEP, enkephalinase) is a 90–110-kDa cell surface zinc-dependent metalloprotease that is expressed in various benign and malignant hematopoietic and non- hematopoietic tissues [10]. It is known to decrease receptor binding to peptide substrates and signal transduction, thus regulating their biological activities. It also plays an essential role in carcinogenesis and progression of tumors [11]. Recent studies suggested that CD10 expression in tumor cells has a major role in cell proliferation and apoptosis, while CD10 expression in stromal cells of the tumor contributes to tumor progression [12]. However CD10 expression was reported in many tumors especially hematopoietic and epithelial neoplasms, its role in intracranial tumors is restricted to differentiating metastatic renal cell carcinoma (RCC) from hemangioblastoma, where RCC shows CD10 positivity while hemangioblastoma shows inhibin positivity [13].

Inhibin is a dimeric 32-kDa peptide hormone belonging to the transforming growth factor-β superfamily. It is composed of an α-subunit and a β-subunit linked by disulphide bridges. Two forms of inhibin exist; inhibin-A in which α -subunit is linked to a β -A subunit and inhibin-B in which a -subunit is linked to a β -B subunit [14]. Generally, immunohistochemical (IHC) detection of inhibin is directed towards inhibin-A. Inhibin mRNA has been identified in many normal tissues, as pituitary gland, testis, ovary, placenta, bone marrow, spinal cord, brain, adrenal cortex, pancreas and kidney. Inhibin-A is considered as a sensitive and relatively specific marker for CNS hemangioblastoma and sex cord-stromal tumors of the ovary. However, it is reported also to be expressed in renal cell carcinoma, placental-gestational trophoblastic tumors, granular cell tumors, adrenal cortical tumors, paraganglioma, angiosarcoma, lipoma, liposarcoma, and rhabdomyoma [15].

Many cases of meningiomas, especially intracranial, can mimic hemangioblastoma and metastatic RCC. Expression rates of CD10 and inhibin in these cases, in addition to spinal meningiomas, are not yet clearly clarified. So in this study we aimed to evaluate the expression of inhibin and CD10 in a large number of intracranial and spinal meningioma cases and to evaluate whether their expression is related to clinicopathological findings and recurrence rates (Fig. 1).

Material and Methods

Patients and Samples

Files of all resected meningioma cases received at pathology department, Mansoura, Egypt, were reviewed during the period from 2000 to 2007. Cases with incomplete clinical data were excluded. One hundred thirty-four cases were fulfilling selection criteria, 14 of them were spinal and 120 were intracranial. The patients didn't receive any pre-operative therapy.

Clinical Parameters and Histopathological Evaluation

The clinicopathological data of all 134 cases were reviewed with proper reassessment of all the slides and their relevant details including their location, histological type, atypia, recurrence, age and gender of the patient along with interpretation of any other findings.

Tissue Microarray (TMA) Construction

Tissue microarray blocks were manually constructed using modified mechanical pencil tip method [16], with punching out of multiple 0.8 mm diameter cores from each case. Multiple normal tissue cores were inserted to serve as positive and negative internal controls.

Immunohistochemistry

Sections from TMA blocks were sectioned 4 μm thick. Then, deparaffinized of the sections was done by xylene.

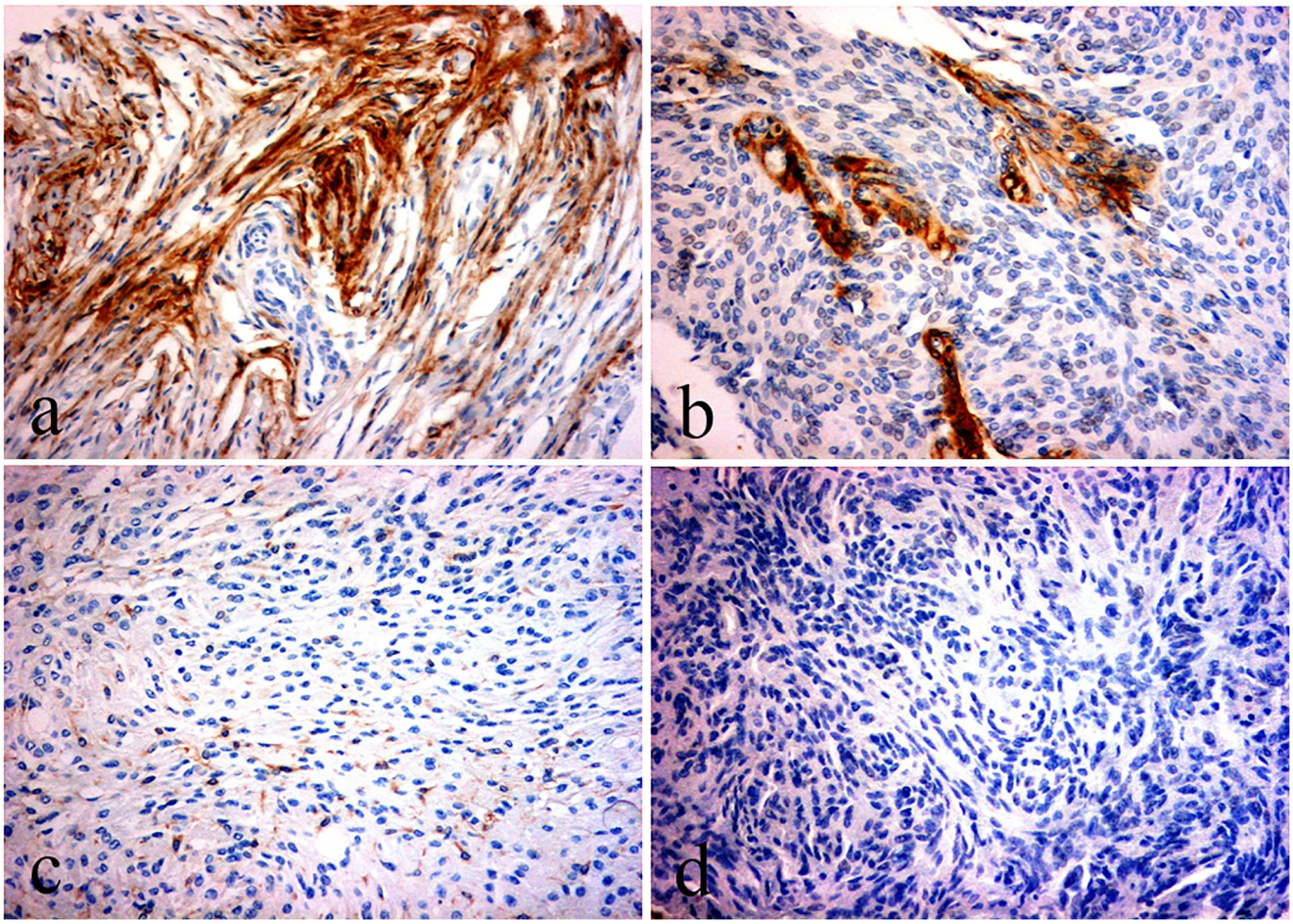

Fig. 1 **a** Strong CD10 cytoplasmic staining in a case of fibroblastic intracranial meningioma (**b**) Strong focal CD10 cytoplasmic staining in a case of transitional spinal meningioma (**c**) Faint inhibin cytoplasmic staining in a case of meningotheliomatous intracranial meningioma (**d**) Negative inhibin staining in a case of transitional spinal meningioma (×200)

Rehydration was then performed using graded alcohols followed by antigen retrieval using heat and retrieval solution pH 7.6 then washing in distilled water and phosphate-buffered saline. Blockage of endogenous peroxidase activity was done by incubation of the sections for about 30 min with 3% hydrogen peroxide. Primary antibodies against monoclonal rabbit anti-human CD10 Ab (Clone SP67, Cat.# 790–4506, ready-to-use for IHC, Ventana) and monoclonal rabbit anti-human α inhibin Ab (Clone MRQ-63, Cat.# 760–6081, predilute ready-to-use for IHC, Ventana) were then incubated with the sections in citrate buffer solution and left for 60 min at room temperature. ImmunoPure Ultra-Sensitive ABC Peroxidase was added, with diaminobenzidine as chromogen. Sections were then rinsed in distilled water and counterstained with haematoxylin followed by rehydration in ascending alcohols and xylene then covered with coverslips.

Evaluation of IHC

Independent semi-quantitaive assessment of each case for CD10 and inhibin expressions was carried out by two of the authors (Foda AA and Rafi S). Staining of each tissue core on the TMA was interpreted as either positive or negative as previously described [17]; tumors in which the tumor cells showed either complete negativity of immunostaining or showed faint staining in <5% of tumor cells were scored as negative. If at least 5% of the tumor cells were unequivocally stained, the tissue core was interpreted as positive. Cases were excluded from the analysis if the tissue core was lost in processing or if there was no recognizable tumor in the core.

Statistical Analysis

Data were analyzed using SPSS version 24.0 for Windows (SPSS Inc., IBM, Chicago, Illinois). Significant differences of CD10 and inhibin expressions in relation to clinicopathological and histological parameters were tested using $\chi 2$ (Chi-square) test. A 2-tailed $P \leq 0.05$ was considered significant in all tests.

Results

A total of 134 meningioma specimens were included in the current study. The clinicopathological and histological features of the tested meningioma cases are listed in Table 1.

Majority of the samples belonged to females (103 cases; 77%) while 31 cases (23%) were from males. Intracranial meningiomas consisted of 90% of the samples (120 cases) while spinal meningiomas consisted of 10% of the samples (14 cases). Age range at presentation for meningioma was 6 to 80 years (mean, 47 years) and SD ± 15.18. The most frequent age of presentation was 50 years in females, and 52 years in males. Statistically significant difference ($p = 0.005$) between meningioma and age of presentation was found, with 82.5% of intracranial cases presented in ages <60 years. However no statistical significance ($p = 0.873$) was found between gender and the occurrence of meningioma (Table 1).

Intracranial meningiomas included in the current study presented mostly at the skull base including sphenoidal wing and olfactory groove (49 cases; 41%) followed by parito-occipital (26 cases; 22%) and frontal (24 cases; 20%) region. The least common presentation was in the temporal region (21 cases; 18%) (Table 1).

Table 1 Clinicopathological and histological features of Intracranial and Spinal meningiomas

	Spinal No. (%)	Intracranial No. (%)	*P* value
Age (y)			
- < 60	99 (82.5%)	07 (50%)	**0.005***
- ≥ 60	21 (17.5%)	07 (50%)	
Gender			
- Male	28 (23.3%)	03 (21.4%)	0.873
- Female	92 (76.7%)	11 (78.6%)	
Histological Type			
- Meningotheliomatous	48 (40.0%)	0 (0.0%)	**0.006***
- Transitional	53 (44.2%)	11 (78.6%)	
- Fibroblastic	10 (08.3%)	0 (0.0%)	
- Psammomatous	09 (7.5%)	03 (21.4%)	
Atypia			
- Benign	114 (95.0%)	14 (100%)	0.392
- Atypical features	06 (5.0%)	0 (0.0%)	
Recurrence			
- Negative	109 (90.8%)	14 (100%)	0.237
- Positive	11(9.2%)	0 (0.0%)	
CD 10 expression			
- Negative	84 (85.7%)	08 (61.5%)	**0.030***
- Aberrant positive	14 (14.3%)	05 (38.5%)	
Inhibin expression			
- Negative	101 (93.5%)	13 (92.9%)	0.925
- Aberrant positive	07 (6.5%)	01 (7.1%)	

*$P \leq 0.05$ is significant

There was a significant difference in the histologic type between intracranial and spinal meningiomas ($p = 0.006$). The most common presenting histologic type among spinal meningiomas was transitional (78.6% of the cases) followed by psammomatous type (21% of the cases). There was no meningotheliomatous or fibroblastic type of spinal meningiomas, while meningotheliomatous type accounted for 40.0% of tested intracranial meningiomas (Table 1).

Ninety-five percent of intracranial and 100% of spinal meningiomas was benign, with only 6 cases of intracranial meningiomas having atypical features (5%). No statistically significant difference was found between intracranial and spinal meningiomas regarding presence of atypia ($p = 0.392$). Similarly, there was no statistical significant relationship between the location of the tumor and its recurrence ($p = 0.237$). There was no recurrence in all spinal tumors, while recurrence was found in 9% of intracranial cases (Table 1).

CD10 was expressed in 19 (about 14%) of studied meningioma cases. There was a statistical significance ($p = 0.030$) between location of meningioma and CD10 expression. Almost 38.5% of spinal tumors were aberrantly positive for CD10 expression, while only 14.3% of the intracranial tumors showed aberrant CD10 positivity (Table 1). There was statistically significant difference ($p = 0.020$) between CD10 expression and the histologic type of meninigioma. Almost all types of tumors were positive for CD10 expression, transitional meningiomas showed the highest positivity for CD10 expression (74% of the cases), with the least positive was the meningiotheliomatous type (about 5% of the cases. However, no statistical significance of age ($p = 0.299$), gender ($p = 0.341$), location ($p = 0.178$), atypia ($p = 0.298$) and recurrence ($p = 0.800$) was found with the CD10 expression.

Inhibin was expressed in 8 (about 6%) of studied meningioma cases. There was no statistically significant difference in inhibin expression between spinal and intracranial meningiomas ($p = 0.925$) (Table 1). Similarly, no statistical significance was found between inhibin expression and any of the studied clinicopathological and histological parameters (data not shown). There was no statistically significant relationship ($p = 0.284$) between the expression of CD10 and inhibin expression in meningiomas; only 2 cases (10.5%) showed co-expression of both markers.

Discussion

Meningioma is one of the most common primary intracranial neoplasms. There have been recent advances in the understanding of the genetic factors involved in meningioma pathogenesis. Meningiomas share some biologic features with malignant tumors as frequent intratumoral heterogeneity, natural tendency for invasion, in addition to the correlation between clinical behavior and biologic profile. Therefore,

identification of tumor biological markers is crucial in for tumor therapy as well as an important way to differentiate meningioma from other tumors [4].

CD10 is a zinc-dependent, cell membrane metallopeptidase that is expressed in many normal tissues as in small and large intestinal surface epithelium, myoepithelial cells of breast ducts and lobules, kidney glomerular and proximal convoluted tubular epithelium, endometrial stromal cells, prostatic ductal epithelial cells and lung alveolar cells [18]. CD10 is also expressed in many tumor tissues. It is a common diagnostic marker for the sub classification of acute leukaemias and non-Hodgkin lymphomas [19]. Overexpression of CD10 has been detected in almost all well and moderately differentiated colorectal adenocarcinomas [20], in 100% metastatic clear cell RCC [21, 22] thus helping helping in differentiating it from hemangioblastoma of the CNS [13]. On the other hand, CD10 is down regulated in other tumors as lung and prostatic cancers [23].

In the current study, we attempted to identify if CD10 is aberrantly expressed in meningiomas and whether it has relations to clinicopathological features and prognosis. Majority of the samples belonged to females that yet illustrated the female predominance of the meningiomal tumefaction. We found that CD10 was aberrantly expressed in about 14% of studied meningioma cases. This is in concordance with the results of a previous study that tried to differentiate between clear cell meningioma and metastatic clear cell RCC [24]. They reported that 5/18 clear cell meningiomas (27.8%) showed positive staining. However we did not have clear cell meningioma in our case, we found a near percent because we had included a large number of cases in our study. Most cases that express CD10 in the current study were of the transitional histologic type. So, regardless the histology, CD10 can be expressed in meningiomas which can be misleading in differentiation it from metastatic RCC. This can be added to the dilemma of differentiating hemangioblastoma and metastatic intracranial RCC. CD10 was even more significantly expressed in spinal rather than intracranial meningiomas in our study. However, it was not associated with any differences in age and gender of the patients as well as poor prognostic factors as atypia and recurrence rate. Therefore we suggest further molecular and functional studies to explore the role of CD10 specifically in the pathogenesis of spinal meningioma.

Another marker that is involved in the diagnostic dilemma is inhibin. Just like CD10, over-expression of inhibin-alpha was reported in many tumors as RCC, placental-gestational trophoblastic tumors, granular cell tumors, adrenal cortical tumors, paraganglioma, angiosarcoma, lipoma, liposarcoma, and rhabdomyoma [25], but its most important role in intracranial tumors is to distinguish hemangioblastoma from metastatic clear cell RCC [13].

Few previous studies had explored inhibin expression in meningioma and revealed contradictory results. Camille et al. study reported that inhibin was exclusively expressed by hemangioblastomas with no expression of inhibin in meningiomas [17]. In contrast, Gurses et al. [25] provided more significant data with 14 out of 20 meningiomas (70%) expressing inhibin! In our study, out of the 134 cases examined only 8 specimens (6%) showed inhibin alpha expression. We believe that increasing the number of studied cases can lead to the actual expression rate. That is why we found some inhibin expression (6%) with neither total negativity nor extensive positivity! Moreover, in contrast to CD10, most of these cases were intracranial rather than spinal. Furthermore, inhibin expression was not associated with any of the histological or clinicopathological features of meningioma cases including atypia and recurrence rate. In contrast, Gurses et al. [25] reported that among the 14 aberrantly inhibin-positive meningiomas in their study, all WHO grade I meningiomas were positive whereas only 4 grade III (anaplastic) meningiomas showed immunoreactivity. Thus, they concluded that inhibin positivity decreases with anaplasia. Again, their findings can be explained by the fact that their study included only a small number of cases. Finally, although CD10 and inhibin were expressed in some cases of meningioma, our study revealed that both are unrelated to each other.

To conclude, spinal meningiomas differ than intracranial ones in many clinicopathological and biological aspects. Among these differences is CD10 expression, being more expressed in spinal meningiomas. CD10 and inhibin are aberrantly expressed in a proportion of meningiomas and both have no relation to poor prognostic factors. Nevertheless, caution should be exerted during the use of these markers in the diagnosis of hemangioblastomas and metastatic RCCs. Also, further studies are suggested to explore and seek more biological differences between spinal and intracranial meningiomas.

Acknowledgements The authors thank Dr. Abdelhadi Shebl for his help to the team of this research.

Author Contributions All authors have contributed significantly and are in agreement with the content of the manuscript.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval The current study protocol was approved by the ethics committee of the Faculty of Medicine, Mansoura University, Egypt, and of the Batterjee Medical College for Sciences and Technology, Jeddah, Saudi Arabia.

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