What Tactics Should a Surgeon Choose to Treat a Black Extracerebral Tumor? A Case Report of Psammomatous Melanotic Schwannoma of the Meckel Cave and Literature Review

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Key words
- Melanin-containing tumor
- Melanotic schwannoma
- Psammomatous schwannoma
- Tumor of Meckel cave

Abbreviations and Acronyms
CNS: Central nervous system
CT: Computed tomography
MCT: Melanin-containing tumor
MRI: Magnetic resonance imaging
MS: Melanotic schwannoma

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Citation: World Neurosurg. (2018) 116:337-342. https://doi.org/10.1016/j.wneu.2018.04.153

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

BACKGROUND: **Neoplasms located in the Meckel cave account for 0.2%–0.5% of all intracranial tumors. This area is the site of many types of pathologic lesions, most often trigeminal nerve schwannomas and meningiomas. Melanin-containing tumors are rare in this area. These tumor types can be suspected if the magnetic resonance characteristics of a tumor has some differences in comparison with other types of central nervous system neoplasms. In fact, differential diagnosis of melanotic tumors is based mainly on the histopathologic criteria and immunohistochemical profile. This article presents a case report of melanotic schwannoma of the Meckel cave and a literature review of the problem.**

CASE DESCRIPTION: **A 23-year-old man underwent a 2-stage surgery for a dumbbell pigmented mass lesion located in the Meckel cave. No signs of recurrence were seen on follow-up magnetic resonance imaging (MRI) 3.5 years after the operation.**

CONCLUSIONS: **Melanin-containing tumor can be suspected in the presence of radiologic characteristics, such as a hyperintense MRI signal on T1-weighted images and a hypointense signal on T2-weighted images. If a black extracerebral tumor is detected, the main course of surgical treatment is maximal excision despite it possibly being a malignant melanoma and the temptation to perform partial resection because of an unfavorable prognosis. Chemotherapy can be justified in the presence of an aggressive melanotic schwannoma.**

INTRODUCTION

Background

The most frequent lesions located in the Meckel cave are schwannomas of the trigeminal nerve and meningiomas.[1,2] Melanotic meningeal lesions are rare in this area and are difficult to identify. The origin of these tumors from neural crest precursor cells, their transition forms, and the common derivation cause the difficulties in distinguishing melanin-containing tumor (MCT) types.[3] Pigmented primary central nervous system tumors involving the Meckel cave are to be differentiated from other dura-based lesions, such as primary and metastatic melanoma, melanotic schwannoma (MS), and melanocytoma. This report presents a literature review of the problem and a new case of MS of the Meckel cave.

Clinical Case

A 23-year-old man had a 1-year of right-sided face numbness followed by severe morning headache and vomiting. He also suffered from diplopia when looking to the right.

In addition, the man had recurrent acute otitis media 4–5 times per year and had lost hearing in the right ear that year.

His Karnofsky Performance Status score was 80. Neurologic examination revealed hypoesthesia in the I and II divisions of the right trigeminal nerve, dilatation of the right pupil and paresis of the lateral gaze. The patient also developed severe deafness in the right ear and mild static ataxia.

He had no familial history or any component of Carney complex. Examination of the retina and skin; ultrasonography of the eyes; computed tomography (CT) scan of the chest, abdomen, and pelvis; fibrocolonoscopy; and bronchoscopy did not reveal any abnormality.

Neuroimaging. MRI showed a dumbbell mass lesion of 56 × 25 × 33 mm (Figure 1) arising out of the skull base into the right middle cranial fossa. It had a subtentorial extension into the posterior cranial fossa and adjacent cavernous sinus. The cerebellopontine angle was filled, and the patient suffered from severe brain stem compression with a 4-mm midline displacement.

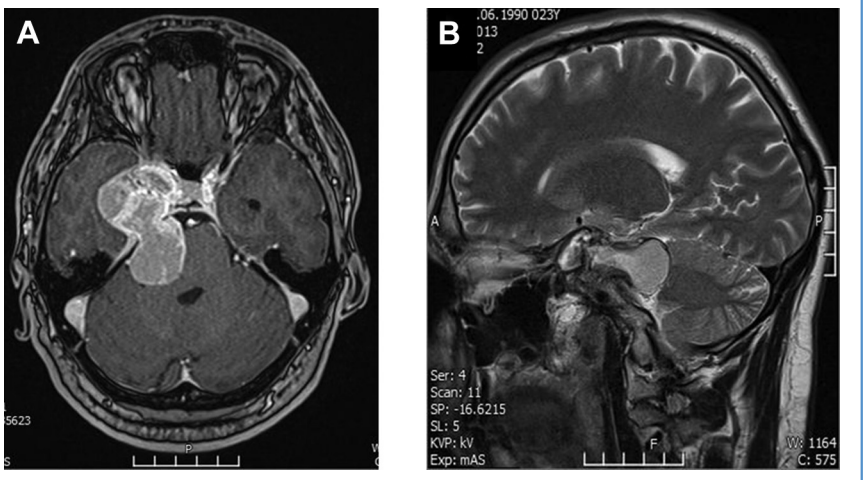

Figure 1. (**A**) Axial T1-weighted magnetic resonance imaging with gadolinium. (**B**) Sagittal T2-weighted magnetic resonance imaging. The lesion has smooth contours and a thin capsule. There is a hyperintense signal on T1- and T2-weighted images relative to the signal intensity of the brain. The homogeneous enhancement on T1-weighted images after the administration of gadolinium can be observed.

Operative Course

The patient underwent 2-stage surgery.

First Operation. The first stage of the procedure included a right infratemporal craniotomy and subtotal tumor resection. The tumor was a black capsule of tightly elastic consistency. It was vascular with moderate bleeding (Figure 2). It extended both inside and outside the dura and involved all branches of the trigeminal nerve. The anterior part of the tumor abutted the posterior aspect of the cavernous sinus it was separated from. The medial part adjacent to the brain stem and clivus was totally resected. The part of the tumor located below the left internal auditory canal was inaccessible. After the operation, the patient developed total ophthalmoplegia and the trigeminal sensory loss became more marked.

Second Operation. The patient underwent retrosigmoid craniotomy and total microsurgical tumor resection and under intraoperative neurophysiologic monitoring. The black tumor with a well-defined gray capsule was located at the apex pyramid displacing the right trigeminal nerve anteriorly and growing into the meninges. The intraoperative electrophysiologic monitoring of the right trigeminal nerve showed no response. The tumor was separated from the lateral side of the pons, facial and trigeminal nerves, the right posterior cerebral and basilar arteries, and the dura of the pyramid and tentorium cerebellum. The evoked potentials of the right facial nerve did not change relative to the preoperative data.

There was no additional deterioration of the neurologic function after the second operation but the total right ophthalmoplegia remained unchanged. Postoperative MRI showed no residual tumor.

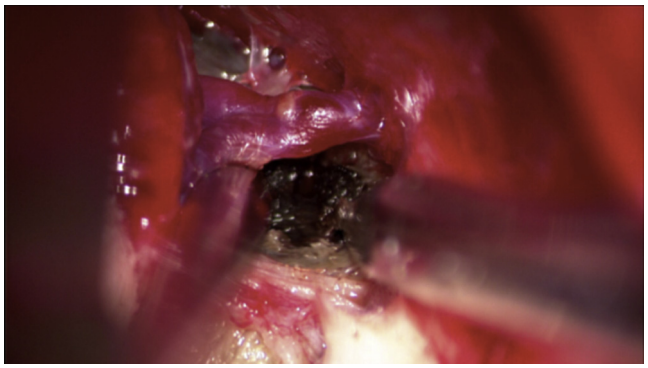

Figure 2. Intraoperative photographs showing heavy pigmented mass.

Pathology

Light microscopy showed extremely large amounts of brown pigmented granules (Figure 3A). The abundance of melanin masked the cells' features and did not permit a detailed assessment of the cells; therefore, the pigment was bleached with 3% hydrogen peroxide. It became evident that the pigment was located mainly intracellularly, and the cells had round or oval shape. The nuclei had no sign of the hyperchromasia and polymorphism typical for malignant neoplasms. Moreover, mitosis was absent (Figure 3B). In a few sections, elongated pigment-free cells forming bundles could be identified (Figure 3C).

In the histologic samples, the hyalinized connective tissue that also had focal accumulations of brown pigment and both transverse and longitudinal sections of the small nerve trunks with severe degenerative changes of the axons were often detected. Clusters of psammomatous bodies were seen in one of the samples (Figure 3D).

Melanocytic markers such as Melan A, tyrosinase, HMB-45 (Figure 3E) were expressed. An immunohistochemical study showed the expression of protein S-100 in all the structures except for the connective tissue and vascular walls (Figure 3F). The proliferative activity (Ki-67) did not exceed 3.4% (Figure 3G, H).

Expression of MGMT (O^6-methylguanine DNA methyltransferase) gene allowed estimating the intracellular condition more distinctly than methylation of MGMT gene promoter. The level of expression of the *MGMT* gene was indicated with real-time polymerase chain reaction as moderate (1.4). *BRAF* mutation was not revealed. The final diagnosis for this tumor was a psammomatous melanotic schwannoma.

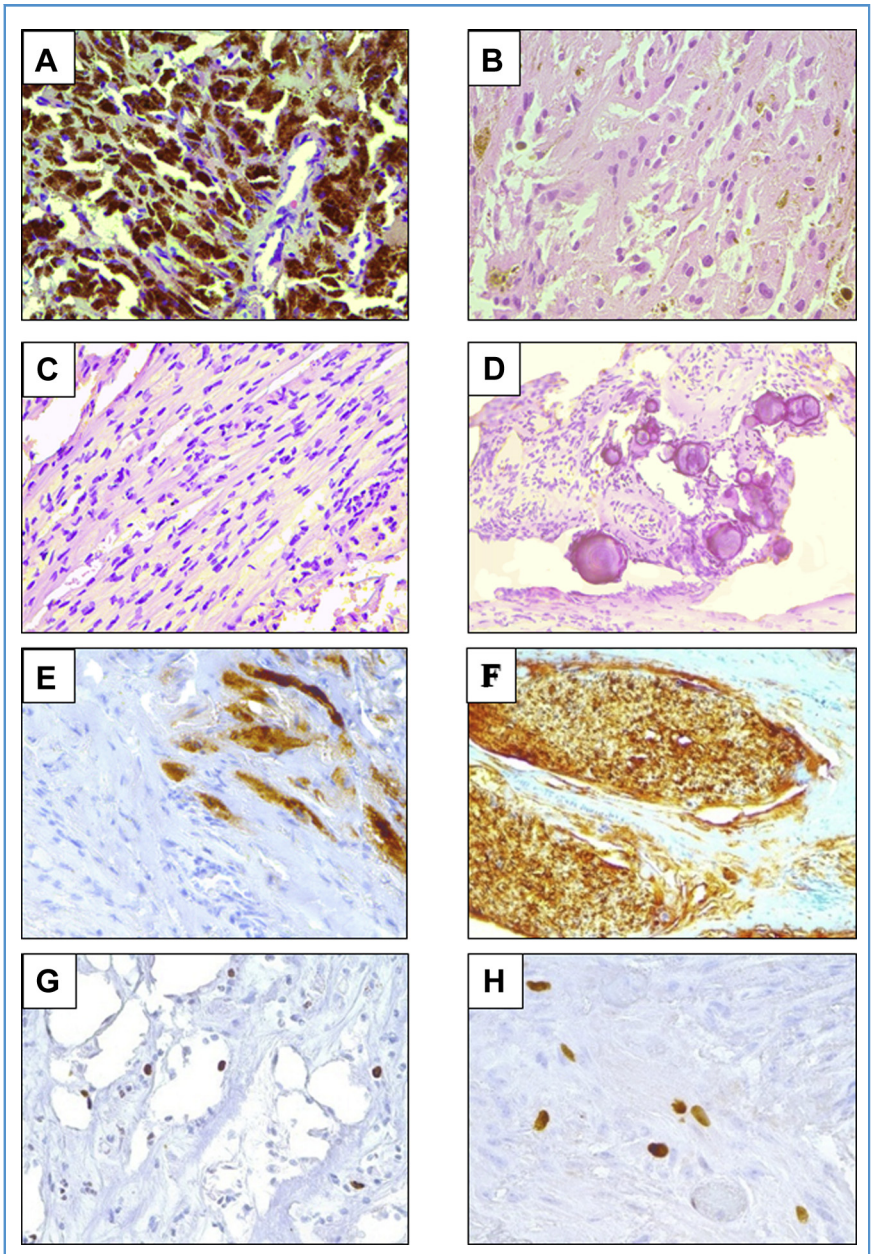

Figure 3. (**A**) Microscopic view of the tumor showing the abundance of the pigment (hematoxylin and eosin [H&E], original magnification ×400). (**B**) The plexiform pattern of the tumor after removal of pigment (H&E, original magnification ×400). (**C**) A cellular tumor composed of spindle-shaped cells arranged in fascicles (H&E, original magnification ×400). (**D**) Low-power view of the tumor showing collections of psammoma bodies (H&E, original magnification ×200). (**E**) Immunohistochemical stains were positive for HMB-45 (original magnification ×400). (**F**) Immunohistochemical examination revealed the tumor cells to be stained positive for the S-100 protein (original magnification ×200). (**G**) Immunohistochemical examination showed nuclear expression of Ki-67 in the nonpigmented area (original magnification ×400). (**H**) Nuclear expression of Ki-67 in cells previously contained melanin (original magnification ×400).

Postoperative Course. Considering the aggressive behavior of melanotic schwannomas in 10%–15% of cases and the moderate level of the patient's MGMT gene expression, temozolomide could be used and might possibly be effective; therefore, the patient received a temozolomide chemotherapy course. MRI was performed 3.5 years after surgery and demonstrated no sign of recurrence (Figure 4A, B). The next examination was 4 years after the operation, and some improvement in the right ophthalmoplegia was noted.

DISCUSSION

To our knowledge, 43 cases of MCT of the Meckel cave have been described in the English-language literature. In these publications, the most common MCT located in the Meckel cave were melanomas (19 cases), followed by melanocytomas (17 cases), and less often MSs (7cases) (Table 1).

Epidemiology and Localization of MS

Fewer than 200 cases of MS have been described in the English-language literature and less than 1% of central nervous system (CNS) schwannomas are melanotic; the frequency and extent of this tumor in the CNS are unknown.[10] MS is observed with equal frequency in both sexes, predominantly in young and middle age; the mean age is 38–41 years.[9,11]

In 1932, Millar for the first time described MS of the thoracic sympathetic ganglion. Currently, the cases of various locations have been described for MS including skin, bone, visceral organ, sinuses, the adrenal glands, uterus, bronchus, pancreas, orbit, choroid, intracranial, and intramedullary MS.[9] The most frequent location of MS in the CNS is the spinal nerves (46%).[9,12] Among intracranial locations, MSs of the trigeminal nerve, VIII nerve, cerebellum, and posterior fossa have been diagnosed.[9]

According to the publications, MSs have multiple locations such as bronchus and middle cranial fossa diagnosed at the same time and the lumbar spine and trigeminal nerve diagnosed in the period of 30 months.[5,6]

MS has had the shortest duration of symptoms before diagnosis compared with other melanin-containing tumors of the Meckel cave (6 months). There have been 2 cases of multiple locations aside from the Meckel cave when the masses were located in the bronchus and lumbar spine L5\S1.

Types of MS

The two types of MS are psammomatous and nonpsammomatous. Psammomatous MS arises from the nerves of the visceral organs, intestines, and cranial nerves, whereas nonpsammomatous MS is often sporadic and found in the spinal nerves and paraspinal ganglia.[13]

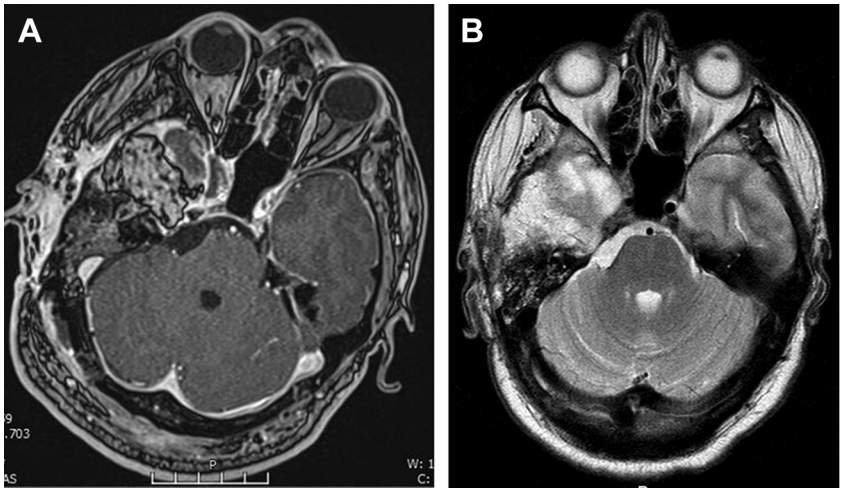

Figure 4. (**A**) Axial T1-weighted magnetic resonance imaging after 2-stage total tumor resection. (**B**) Axial T2-weighted magnetic resonance imaging (3 years and 6 months after surgery) showed no evidence of tumor recurrence.

Identifying psammomas bodies can help to diagnose Carney complex type 1 because psammomatous MSs are included as a diagnostic criterion for Carney complex.[13,14] However, 2 cases of primary nonpsammomatous MSs were described in patients with CC[13]; therefore, it can be suggested that the absence of psammomas bodies or MS cranial location do not exclude this diagnosis.

MRI and CT Characteristics of MS

Typically, MCTs appear in MRI as isointense or hyperintense well-circumscribed masses on T1-weighted images and as hypointense on T2-weighted images because of the inherent paramagnetic effect of melanin.[15,16] These MRI characteristics help to differentiate melanotic tumors from nonmelanotic lesions. The more melanin, the shorter T1 and T2 become.[17] It is noted that different types of MCT cannot be distinguished with CT or MRI.[17] A dumbbell-shaped form of the lesion in the Meckel cave is not a significant sign to distinguish the type of tumor because MS, melanocytoma, and melanoma may have this form depending on the location, especially if they have seeding from Gasserian ganglion to cerebellopontine angle, as seen in our patient.

View on Surgery

Intraoperatively, MSs have often been described as unencapsulated circumscribed lesions associated with a nerve.[13] In our patient, the thin tumor capsule was seen on MRI and revealed at craniotomy when the neoplasm had a tight connection with all branches of trigeminal nerve.

Peculiarities of Histology

Criteria such as the nature and condition of tumor vessel walls, the presence or absence of connective tissue, a variable amount of melanin, fat, psammoma bodies or reticulin fibers are nonspecific for differential diagnosis. However, it has been described that psammoma bodies and fat are not seen in melanocytoma cells.[18] The clinicopathologic features of MS with and without psammoma bodies were identical.[9]

In addition, the cells of schwannoma, melanocytoma, and melanoma may look the same from the spindle to rounded (epithelioid) or polygonal shapes.[19-22] Massive deposits of pigment sometimes can completely clog histologic features and cause difficulties for cytologic analysis of a tumor; therefore, bleaching melanin with 3% hydrogen peroxide solution or potassium permanganate is recommended. The basal lamina is rarely found in an ultrastructural study of malignant melanoma; this is often a feature of MS.[13,21]

It has been reported that the MIB-1/Ki-67 labeling index may help to predict an aggressive clinical behavior of MCT.[9] However, many authors emphasize that there are no reliable histologic criteria that clearly indicate malignancy of MS, and histologically malignant MS have a benign clinical course. As a result, the tumor behavior, such as regrowth and metastasizing, may be unpredictable.[18,23]

Electron microscopy can help to distinguish melanocytes and neurocytes, which have different ultrastructural signs (e.g., collagen IV type, the distance between collagen fibers), but potentially MCT can contain different cells.[9] The differential diagnoses of MS, melanocytoma, and melanoma is based on histopathologic criteria and the immunohistochemical profile; however, in some cases, difficulties in estimating the nature of the tumor may persist between MS and melanocytoma.[24,25]

The basic and only schwannoma marker, the S100 protein, can be expressed in other lesions composed of spindle-shaped melanocytic cells, including melanoma or even scar tissue with regenerative nerve process.[26]

Melanocytic markers (Melan A, HMB-45, MITF, tyrosinase) usually stain melanocytes and do not give any evidence for an accurate diagnosis of schwannomas.[19,22]

It has been reported that the expression of laminin and collagen type IV can be used for the differential diagnosis between melanoma and MS, because these immunohistochemical stains are positive only in MS and are not expressed in malignant melanoma.[9,11]

Immunohistochemical study shows that Leu-7 and vimentin are highly expressed in most MS cells, and focal GRB2-related adaptor protein may be observed. However, vimentin can also be indicated in common tumor cells.[9,11,20] Melanocytomas are negative for Leu-7 and in this way are differentiated from MS.[27]

Clinical Course of MS

It is considered that the most cases of MS are benign, with good prognosis; however, the authors have reported that malignant MSs are 10%–15% and have poor long-term prognosis.[11,12,28] The authors stressed that recurrences of MS can occur, even after 5 years of observations or without histologic signs of tumor malignancy, and MSs need to be monitored with serial MRI for regrowth.[18,28,29] Metastases of MS have been seen in 9%-44%, not only

Table 1. Reported cases of Melanotic Schwannomas of the Meckel Cave

References	Age (years)	Sex	Duration of symptoms (months)	Tumor Location	Histopathology	Extent of Surgery	Adjuvant Treatment	Follow-up
Quencer et al., 1979[4]	22	Female	3	Gasserian ganglion	ND	Total excision	None	No recurrence at 14 months
Beck and Menezes, 1987[1]	12	Male	12	Meckel cave	Malignant schwannoma	ND	FRT	Recurrence at 2 years, FRT, no recurrence during subsequent 9 years
Rowlands et al., 1987[5]	27	Male	5	Middle cranial fossa and metastasis to the bronchus	Malignant schwannoma	Biopsy	FRT	Recurrence at 14 months
Buhl et al., 2004[6]	28	Male	4 days	Trigeminal nerve	MIB-1 staining 1%–2%	ND	None	30 months later surgery for metastatic spinal lesion at L5-S1
Carrasco et al., 2006[7]	34	Female	2	Trigeminal nerve	No signs of malignancy	Incomplete excision	None	Recurrence at 3 months
Nenashev et al., 2012[8]	27	Female	6	Trigeminal nerve	MIB-1 staining <1%	Subtotal excision	GKR	No recurrence at 6 months
Torres-Mora et al., 2014[9]	19	Female	ND	Trigeminal nerve	ND	ND	ND	No recurrence at 49 months
This clinical case (2014)	23	Male	12	Gasserian ganglion	Psammomatous melanotic schwannoma	Total excision	Temozolomide	No recurrence at 42 months

ND, no data; FRT, fractionated radiotherapy; GKR, Gamma Knife radiosurgery.

within the CNS but also as distant metastases.[9,28] Torres-Mora et al.[9] analyzed 40 cases of MS and raised the issue to recognize MS as a malignant tumor; they also revised histologic signs of malignancy of these neoplasms and proposed their reclassification as "malignant melanotic schwannian tumors."[9] Only increased mitotic activity has been suggested as a prognostic factor of MS metastasis.[9]

According to the literature, in cases of MS of the Meckel cave, if there have been no histological criteria of malignancy, the recurrence has been seen after incomplete excision of the tumor over a short period. The optimal treatment for MS is gross total surgical resection when possible and radiation therapy for cases of partial excision.[27,28] Although the efficacy of radiotherapy and chemotherapy is not proved for intracranial MS, considering the aggressive potential of these tumors for malignant transformation and recurrence, radiotherapy and chemotherapy are recommended for complete resection of the tumor.[6,12]

A recent report has shown that in cases of incomplete tumor removal gamma-knife surgery is an effective adjuvant treatment, as what makes MSs the most suitable target for such a treatment is their extra-axiality.[8] Because the poorly demarcated tumor margins in our patient did not allow for radiosurgery, he underwent chemotherapy considering his moderate level of *MGMT* gene expression.

Controversies regarding the grading and adjuvant treatment of MS remain unresolved; therefore, it is mandatory to revise and detect the histologic malignancy features, the points of prognosis, and adjuvant therapy to improve the treatment outcomes [9,25].

Currently, the main course of action in the surgical treatment of a black extracerebral tumor is maximal excision, despite it possibly being malignant melanoma. In addition, there can be a temptation to perform a partial resection because of an unfavorable prognosis.

CONCLUSION

Intracranial MSs are rare neoplasms. Radiologic characteristics of the mass in the Meckel cave, such as hyperintense

MRI signal on T1-weighted imaging and a hypointense signal on T2-weighted imaging, can help us to detect the presence of MCT in a preoperative period. The differential diagnosis of pigmented tumors involving the Meckel cave includes primary and metastatic melanoma, MS, and melanocytoma. We strongly believe that a black neoplasm revealed during surgery has to be resected completely despite it possibly being an aggressive melanoma. The final diagnosis between different types of melanotic tumor is based on histopathologic criteria and the immunohistochemical profile, but it can be challenging. Complete surgical resections of MCT of the Meckel cave are associated with better outcomes. It should be born in mind that MS shows clinical aggressive behavior and may undergo malignant transformation, thereby requiring long-term observation to reveal the tumor's regrowth. Gamma-knife surgery and radiotherapy can be used in cases of incomplete tumor resection. In our opinion, chemotherapy can be justified in the presence of histologic malignancy in the specimen. Determining the most successful treatment paradigm for MS requires more cases with long-term follow-up.

REFERENCES

1. Beck DW, Menezes AH. Lesions in Meckel's cave: variable presentation and pathology. *J Neurosurg*. 1987;67:684-689.
2. de Benedittis G, Bernasconi V, Ettorre G. Tumours of the fifth cranial nerve. *Acta Neurochir (Wien)*. 1977;38:37-64.
3. Carbonell AL, Boya J, García-Mauriño JE. Presence of melanin in normal human Schwann cells. *Histol Histopathol*. 1992;7:329-332.
4. Quencer RM, Stokes NA, Wolfe D, Page LK. Melanotic nerve sheath tumor of the gasserian ganglion and trigeminal nerve. *AJR Am J Roentgenol*. 1979;133:142-144.
5. Rowlands D, Edwards C, Collins F. Malignant melanotic schwannoma of the bronchus. *J Clin Pathol*. 1987;40:1449-1455.
6. Buhl R, Barth H, Hugo HH, Mautner VF, Mehdorn HM. Intracranial and spinal melanotic schwannoma in the same patient. *J Neurooncol*. 2004;68:249-254.
7. Carrasco CA, Rojas-Salazar D, Chiorino R, Venega JC, Wohllk N. Melanotic nonpsammomatous trigeminal schwannoma as the first manifestation of Carney complex: case report. *Neurosurgery*. 2006;59:E1334-E1335.
8. Nenashev EA, Rotin DA, Stepanian MA, Kadasheva AB, Cherekaev VA. Differential diagnosis between melanotic schwannoma of gasserian ganglion and metastatic melanoma of middle cranial fossa. *Zh Vopr Neirokhir Im N N Burdenko*. 2012;76:58-64 [in Russia].
9. Torres-Mora J, Dry S, Li X, Binder S, Amin M, Folpe AL. Malignant melanotic schwannian tumor: a clinicopathologic, immunohistochemical, and gene expression profiling study of 40 cases, with a proposal for the reclassification of "melanotic schwannoma.". *Am J Surg Pathol*. 2014;38:94-105.
10. Murali R, Field AS, McKenzie PR, McLeod DJ, Stretch JR, Thompson JF, et al. Melanotic schwannoma mimicking metastatic pigmented melanoma: a pitfall in cytological diagnosis. *Pathology*. 2010;42:287-289.
11. Zhang HY, Yang GH, Chen HJ, Wei B, Ke Q, Guo H, et al. Clinicopathological, immunohistochemical, and ultrastructural study of 13 cases of melanotic schwannoma. *Chin Med J (Engl)*. 2005;118:1451-1461.
12. Liessi G, Barbazza R, Sartori F, Sabbadin P, Scapinello A. CT and MR imaging of melanocytic schwannomas: report of three cases. *Eur J Radiol*. 1990;11:138-142.
13. Louis DN, Ohgaki H, Wiestler OD, Cavenee WK. *WHO Classification of tumors of the central nervous system. 4th edition*. Lyon, France: IARC Press; 2007.
14. Stratakis CA, Kirschner LS, Carney JA. Clinical and molecular features of the Carney complex: diagnostic criteria and recommendations for patient evaluation. *J Clin Endocrinol Metab*. 2001;86:4041-4046.
15. Smith AB, Rushing EJ, Smirniotopoulos JG. Pigmented lesions of the central nervous system: radiologic-pathologic correlation. *Radiographics*. 2009;29:1503-1524.
16. Liubinas SV, Maartens N, Drummond KJ. Primary melanocytic neoplasms of the central nervous system. *J Clin Neurosci*. 2010;17:1227-1232.
17. Chen CJ, Hsu YI, Ho YS, Hsu YH, Wang LJ, Wong YC. Intracranial meningeal melanocytoma: CT and MRI. *Neuroradiology*. 1997;39:811-814.
18. Carney JA. Psammomatous melanotic schwannoma: a distinctive, heritable tumor with special associations, including cardiac myxoma and the Cushing syndrome. *Am J Surg Pathol*. 1990;14:206-222.
19. Yeh I, Argenyi Z, Vemula SS, Furmanczyk PS, Bouffard D, McCalmont TH. Plexiform melanocytic schwannoma: a mimic of melanoma. *J Cutan Pathol*. 2012;39:521-525.
20. Gardiman M, Altavilla G, Marchioro L, Boscolo L, Alessio L, Piazza M. Meningeal melanocytoma: a rare lesion of the central nervous system. *Tumori*. 1996;82:494-496.
21. Kurtkaya-Yapıcıer O, Scheithauer B, Woodruff JM. The pathobiologic spectrum of schwannomas. *Histol Histopathol*. 2003;18:925-934.
22. Arvanitis LD. Melanotic schwannoma: a case with strong CD34 expression, with histogenetic implications. *Pathol Res Pract*. 2010;206:716-719.
23. Watson JC, Stratakis CA, Bryant-Greenwood PK, Koch CA, Kirschner LS, Nguyen T, et al. Neurosurgical implications of Carney complex. *J Neurosurg*. 2000;92:413-418.
24. Chang PC, Fischbein NJ, McCalmont TH, Kashani-Sabet M, Zettersten EM, Liu AY, et al. Perineural spread of malignant melanoma of the head and neck: clinical and imaging features. *AJNR Am J Neuroradiol*. 2004;25:5-11.
25. Küsters-Vandevelde HV, van Engen-van Grunsven IA, Küsters B, van Dijk MR, Groenen PJ, Wesseling P, et al. Improved discrimination of melanotic schwannoma from melanocytic lesions by combined morphological and GNAQ mutational analysis. *Acta Neuropathol*. 2010;120:755-764.
26. Ren Y, Xiao A, Wu X, Zhang Y. Meningeal melanocytoma of the middle cranial fossa (the Meckel's cave). *Neurol India*. 2015;63:260-261.
27. Wang H, Zhang S, Wu C, Zhang Z, Qin T. Melanocytomas of the central nervous system: a clinicopathological and molecular study. *Eur J Clin Invest*. 2013;43:809-815.
28. Goasguen O, Boucher E, Pouit B, Soulard R, Le Charpentier M, Pernot P. Melanotic schwannoma, a tumor with a unpredictable prognosis: case report and review of literature [in French] *Neurochirurgie*. 2003;49:31-38.
29. Ditz C, Brunswig K, Meyer C, Reusche E, Nowak G, Tronnier V. Intracranial melanotic schwannoma: a case report of recurrence with extra- and intradural manifestation two decades after initial diagnosis and treatment. *Cent Eur Neurosurg*. 2011;72:211-214.

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Received 15 November 2017; accepted 20 April 2018

Citation: World Neurosurg. (2018) 116:337-342.
https://doi.org/10.1016/j.wneu.2018.04.153

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1878-8750/$ - see front matter