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A case of multiples liver metastases from a grade 2 brain meningioma: a rare entity with comprehensive literature review

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Introduction and importance: Meningiomas are primary benign extra-axial central nervous system neoplasms that originate in meningothelial cells. Extra-neurological metastases are quite rare and occur in 0.1% of cases. The main metastatic sites are usually the lungs, bones, pleura, mediastinum and lymph nodes. Hepatic locations are quite rare and account for ~3% of all extracranial metastases. The dissemination route is still a subject of debate. Suggested routes of dissemination include the venous system, lymph nodes, or even cerebrospinal fluid. The treatment is based on complete surgical excision or on radiosurgery and adjuvant radiotherapy in case of subtotal resection.

Case presentation: The authors present the following clinical case of a 31-year-old healthy male patient with surgical history of meningioma excisions, who presents, 3 years later, evidence of liver masses on tomography and confirmed diagnosis of liver metastases from brain meningioma after biopsy with favorable outcomes after chemotherapy.

Clinical discussion: The overall incidence of extra-neurological metastases of meningiomas remains low. The vast majority of these metastases concern those of high grade, namely WHO grade 2 and 3 as it was reported in our case. Hepatic metastases remain quite rare and account for ~3% of all extracranial locations. Due to these characteristics of having low recurrence and its rare frequency of metastasis to extracranial sites, the authors, through their case, will dig into the literature to dissect this rare entity. **Conclusion:** In the report of liver lesions, the first differential diagnosis in mind should be metastatic lesions, if there is a prior clinical history of primary tumors.

Keywords: brain meningioma, liver metastasis, radiotherapy, surgery

Introduction and importance

Meningiomas are primary central nervous system neoplasms; they originate in meningothelial (arachnoid) cells and represent the most common type of benign extra-axial brain tumor. Traditionally, it is accepted that these neoplasms derive from cells of the arachnoid cap. The most common ones are more likely to arise from arachnoid barrier cells or dural cells^[1]. Meningiomas are divided into three histological grades: benign (WHO Grade 1), atypical (WHO Grade 2) and anaplastic (WHO Grade 3)^[2].

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HIGHLIGHTS

- Meningiomas are primary neoplasms of the central nervous system; they originate in meningothelial cells and represent the most common type of benign extra-axial brain tumor.
- The overall incidence of extra-neurological metastases of meningiomas remains low. The vast majority of these metastases concern those of high grade, namely WHO grade 2 and 3.
- Hepatic metastases remain quite rare and account for ~3% of all extracranial locations. Clinically, these metastatic hepatic meningiomas are often revealed by hypoglycemic episodes.
- Metastatic hepatic meningiomas may present with abdominal pain, nausea, vomiting and massive hepatomegaly.
- The diagnosis is based on the presence of spindle cells arranged in a whorled pattern. By immunostaining studies, meningiomas are usually positive for progesterone receptor and vimentin, while negative for cytokeratin and \$100 proteins.
- For management, the treatment of cerebral meningioma is based on complete surgical excision or on radiosurgery and adjuvant radiotherapy in the case of subtotal resection.

Extra-neurological metastases are quite rare and occur in 0.1% of cases^[3]. The main metastatic sites are usually both lungs (in 60%

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of cases), bones, pleura, mediastinum and finally the lymph $\mathsf{nodes}^{[3]}.$

Hepatic locations are quite rare and account for ~3% of all extracranial metastases. The first case was reported in 1974 by Akagi *et al.*^[4]. The majority of these metastases are discovered at a size not exceeding 10 cm^[2,5]. They can occur simultaneously with the cerebral meningioma^[6,7] or years after surgery for a primary meningioma^[8,9]. The dissemination route is still a subject of debate. Hematogenous way, particularly via the jugular and paravertebral venous systems, represents the most common admitted route^[2,10]. The treatment is based on complete surgical excision or on radiosurgery and adjuvant radiotherapy in case of subtotal resection.

Due to these characteristics of having a low recurrence and its rare frequency of metastasis to extracranial sites, we present the following clinical case of a 31-year-old healthy male patient with a surgical history of meningioma excisions, who presents, 3 years later, evidence of a liver mass on tomography and confirmed diagnosis of liver metastases from brain meningioma after biopsy with favorable outcomes after chemotherapy. We will, then, dig into the literature to dissect this rare entity.

This case report has been reported in line with the SCARE 2023 Criteria^[11].

Case presentation

A 31-year-old male patient with no significant personal medical or surgical history, smoker and weaned occasional alcohol consumer was referred to our department of neurosurgery from the emergency room for progressive onset over 3 months of raised intracranial pressure made of throbbing holocranial headaches worsening at night with right parietal side origin. The patient mentioned that this headache was progressively worsening, becoming resistant to simple, non-opioid analgesics and has recently been accompanied by projectile vomiting at the time of peaks with bilateral visual blurring, more marked in his right eye. He also mentioned that his mood had changed noticeably and that he had become very frustrated, irritable and sleep-deprived.

Upon examination, his was awake, alert and well-oriented temporally and spatially. His pupils were intermediate and responded correctly to the light stimulus. There was no motor of sensitive palsy. Cranial nerve assessment was with no abnormalities. Deep fundus examination revealed a grade 1 bilateral papilledema. The rest of his examination was normal. Urgent brain magnetic resonance imaging (MRI) (Fig. 1) showed a right extra-axial parieto-temporo-occipital lesion measuring 89×74×57 mm in diameter in discrete heterogeneous hyposignal on T1-weighted sequence and in discrete hypersignal on T2-weighted sequence. This lesion enhanced moderately after gadolinium-chelate injection and developed areas of necrosis. There were macro-calcifications and edematous reactions all around.

After informed consent from the patient, he underwent incomplete excision of his very hemorrhagic lesion. The postoperative course was uneventful. Histopathological analysis (Fig. 2) evidenced a meningothelial-like tumor cell proliferation with increased cellularity and a predominant perivascular pseudo-papillary growth pattern. Foci of brain invasion were noticed. Focally, the tumor cell proliferation became patternless forming a sheet-like growth pattern. Areas of spontaneous tumor necrosis were also objectified. Other atypical features that is increased mitotic activity; small cell change and macronucleoli were not observed. Immunohistochemical study confirmed the meningothelial nature of tumor cells by showing co-expression of vimentin and epithelial membrane antigen (EMA). Ki-67 index did not outreach 10%. The diagnosis of an atypical papillary grade 2 meningioma was made.

The patient was then referred to the radiotherapy department for further treatment. He consulted us again 6 months later for a rapidly progressive reappearance of his symptoms of raised intracranial pressure with left hemibody heaviness and a hard bony swelling next to his old surgical site. He also reported that a



Figure 1. Axial brain MRI showing a right intra-axial parieto-temporo-occipital lesion measuring $89 \times 74 \times 57$ mm in diameter in discrete heterogeneous hyposignal on T1-weighted sequence [(A) white arrows] and in discrete hypersignal on T2-weighted sequence [(B) black arrows]. Note the moderate enhancement with zone of necrosis [(C) black arrows] after gadolinium-chelate injection.



Figure 2. Histologic section of the tumor specimen showing several diffuse layers with pseudo-papillary aspect and several perivascular pseudo-rosettes. Tumor cells are epitheloid in appearance and fairly abundant eosinophilic cytoplasm. Nuclei are rounded, finely nucleolated (A, B, C). Mitoses rate evaluated at 2/10 (D) with brain tissue invasion and necrosis areas (E). Ki-67 estimated at 5% (F).

few days before admission he developed an acute generalized tonic-clonic seizure of indeterminate duration without postictal state. This seizure was preceded by gustatory changes, hallucinations, vertigo and involuntary jerks of his right upper limb. The neurological examination revealed a left hemiparesis graded as 3/ 5 with brachial predominance, normal deep tendon reflexes and without any facial palsy. Examination of his scalp noted a hard, non-painful swelling with bony consistency without local inflammatory signs indicating his old bone flap extrusion. The patient informed us that he did not want to start his radiotherapy and that he did not follow any treatment during these last postoperative 6 months. A new MRI performed (Fig. 3) showed a large extra-axial solid and cystic right parieto-temporal lesion, well-limited and with polylobed contours measuring 10x9x9 cm in diameter. The fleshy solid component was in hyposignal on T1weighted sequence with intermediate signal on T2-weighted image. This lesion was exteriorized exocranially through the old right parietal bony flap. After gadolinium-chelate injection, the solid component enhances heterogeneously. The entire lesion exerts a mass effect on the ipsilateral parietal cortex, 3rd ventricle, right lateral ventricle and the midline, which is responsible for right subfalcine and uncal herniation. The gradient echo sequence showed intratumoral calcification, and the diffusionweighted image did not reveal any hypersignal.

The patient was re-operated using the same old surgical approach having undergone a complete resection Simpson grade 1. The tumor was firm and elastic in some area, well demarcated, white and egg yolk yellow in color, with a smooth external surface. The patient was then referred to the radiotherapy department after confirming his grade 2 meningioma (Fig. 4). He received a radiation therapy at a dose of 60 Gy in 2-Gy fractions, delivered over 6 weeks. The control MRI performed few months later did not show any tumor remnant. The patient was followed up at our outpatient clinic for almost 3 years and remained clinically asymptomatic under Levetiracetam at dose of 2000 mg daily divided in 4 doses. Later he was admitted to the Department of General and Visceral Surgery for oppressive pain in the epigastrium and the right hypochondrium, which calmed down after finishing every physical activity. This discomfort was associated with abdominal distension and general weakness with progressive unintentional weight loss of up to 11 kg. He also reports having had an episode of nausea and vomiting, postprandial heaviness, and abdominal bloating a month ago; in addition to difficulty eliminating flatus; He had thinner and scarcer stools, there have even been occasions when he went few days without defecating. On examination, he was in fair general condition with stable vital signs and slight pallor. Segmental physical examination revealed palpable hepatomegaly on 4 fingers along the costal margin, non-painful with tympanism in the colonic area and normoactive aerofluidic sounds. Abdominal computed tomography (CT) scan in its portal venous phase or hepatic phase (Fig. 5) revealed an enlarged liver (hepatic arrow = 23 cm) with a right liver riddled with multiple heterogeneous hypodense lesions, the largest of which measured 18 cm. These lesions presented peripheral and annular enhancement. It was associated with a round mesoceliac adenomegaly. The rest of the organs did not show any abnormalities. The patient underwent a CT-guided biopsy of the largest mass. Histological examination (Fig. 6) revealed diffuse tumor proliferation in sheets alternating with large areas of tumor necrosis. Tumor cells were meningothelial in appearance with weakly eosinophilic or clarified cytoplasm with unclear cytoplasmic boundaries. All nuclei were rounded with few pseudo-inclusions. The nuclear atypia was moderate. Mitotic activity has been 1 mitosis per 10 high-power fields.



Figure 3. Axial brain MRI showing a large extra-axial solid and cystic right parieto-temporal lesion, well-limited with polylobed contours. The fleshy solid component is in hyposignal on T1-weighted sequence [(A) white arrows] and in intermediate signal on T2-weighted image [(B) black arrows]. The lesion extends exocranially and enhances heterogeneously after gadolinium-chelate injection [(C) yellow arrows]. The lesion is surrounded by edema on T2-FLAIR sequence [(D) red arrow] and exerts a mass effect responsible for right subfalcine and uncal herniation. The gradient echo sequence showed intratumor calcification [(E) white arrow] and the diffusion-weighted sequence did not show any hypersignal (F).

On immunohistochemistry, there was significant positivity for EMA and vimentin contrasting with negativity for keratin and AE1/AE3. All these features were in favor of a secondary location of his meningioma.

The patient was then referred again to the radiotherapy department for further management. He received six courses of chemotherapy based on bevacizumab at the rate of one course every 15 days for a period of 3 months with favorable and







Figure 5. Axial (A) and coronal (B) abdominal CT scan in portal venous phase revealed a significant hepatomegaly (hepatic arrow = 23 cm) with a right liver riddled with multiple heterogeneous hypodense lesions, the largest of which measured 18 cm [(A, B) black arrows]. These lesions presented peripheral and annular enhancement.

satisfactory outcomes at the first visit to our outpatient clinic. He started to gain weight and he mentioned a clear reduction in complaints while waiting to finish the rest of his treatment.

Clinical discussion

Meningiomas are the most common primary intracranial tumor (15–20%) and the most prevalent extraparenchymal tumor in the supratentorial compartment in adults. Approximately 10–15% of all meningiomas are located in the spinal cord and constitute up to 75% of all intradural tumors at this level. In about 10% of cases, meningiomas can be multiple. The incidence of meningiomas increases with age and most are diagnosed in adults, with a significant increase after 65 years of age. There is a predominance in female sex (female/male ratio > 2/1) and in the Afro-descendant race^[12]. Ninety-eight and six tenths of diagnosed meningiomas (grade 2) and much more rare are malignant or anaplastic meningiomas (grade 3)^[2,13]. Our patient had a tumor with the histopathological characteristics of grade 3 meningioma.

The overall incidence of extra-neurological metastases of meningiomas remains low. The vast majority of these metastases concern those of high grade, namely WHO grade 2 and 3 as it was reported in our case. The first case of extracranial localization of malignant meningiomas was reported in 1886 by Power^[14]. In one of the largest 18-year studies of cranial meningiomas by Enam et al.^[15], only seven of 396 were classified as high grade, with a metastatic rate of 43%. Among the 389 remaining lowgrade meningiomas, the metastasis rate was 0.76%. In another study carried out in 2019 by Dalle et al.[16], of 1193 patients, only 28 presented extracranial metastases, of which 1 patient was grade 1, 16 patients were grade 2 and 11 patients were grade 3. Reaffirming that the incidence of metastatic disease largely depends on the grade of the primary tumor and then grade 3 meningioma are most likely to spread far away as we reported in our patient.

In a systematic review of the literature on distant metastases of meningiomas ranging from 1990 to 2012 performed by Surov *et al.*^[17], 115 cases with 164 metastatic lesions were identified. The primary tumor was grade 1 in 33.9%, grade 2 in 20.9% and



Figure 6. Histologic section of the liver tumor biopsy revealing diffuse tumor proliferation alternating with large areas of tumor necrosis. Tumor cells are meningothelial in appearance with weakly eosinophilic or clarified cytoplasm and unclear cytoplasmic boundaries. All nuclei look rounded with few pseudo-inclusions with moderate nuclear atypia.

grade 3 in 40% of cases (in 5.2% of cases, the grade was not indicated). This emphasizes the theory of the fairly high metastatic power of high-grade meningiomas. In the same review, and all metastases combined, 93% of meningiomas were resected even before the appearance of the metastasis as it was seen in our report where there was a gap of 3 years between both episodes. In 6.1% of cases, the metastasis and the primary tumor were identified simultaneously and only in 0.9%, the metastasis was highlighted before the discovery of the primary tumor^[17].

Hepatic metastases remain quite rare and account for approximately 3% of all extracranial locations. The majority of these metastases are discovered at a size not exceeding 10 cm^[2,5]. In our patient, the largest lesion was 18 cm. The main route of the distant spread of meningiomas is poorly understood. Suggested routes of dissemination include the venous system, lymph nodes, or cerebrospinal fluid. Hematogenous way, particularly via the jugular and paravertebral venous systems, represents the most common route $^{[2,10]}$. In this context, dissemination occurs via the vertebral venous system. This system connects the cerebral veins, those of the spinal canal and the vertebral column to the thoracic and abdominal wall^[17,18]. The jugular vein itself can transmit tumor cells to the cervical organs, lungs, liver and other organs^[19,20]. Moir et al.^[7] reported an unusual mechanism of hepatic metastatic spread of an intracranial meningioma via a ventriculoperitoneal shunt.

Clinically, these metastatic hepatic meningiomas are often revealed by several hypoglycemic episodes^[21]. This is most likely linked to the significant replacement of healthy liver parenchyma by neoplastic cells. This will result in the depletion of glycogen stores as well as excessive use of glucose by neoplastic tissues^[21]. Metastatic hepatic meningiomas may present with abdominal pain, nausea, vomiting^[7], and massive hepatomegaly^[22]. Our patient reported oppressive pain in his epigastrium and right hypochondrium associated with abdominal distension and general weakness with progressive weight loss. He also reported an episode of nausea and vomiting in addition to difficulty eliminating flatus.

From a histological point of view, the diagnosis is based on the presence of spindle-shaped cells arranged in whorled pattern. By immunostaining studies, meningiomas are usually positive for progesterone receptor and vimentin, while negative for cytokeratin and S100 proteins^[23]. EMA and vimentin are useful in distinguishing between the different types of meningiomatous lesions and speeding up their diagnosis^[24]. It is advisable to perform genetic and molecular studies to correlate the findings with other types of tumors. In our patient, there was diffuse positivity for both EMA and vimentin and negativity for the GFAP, CD34 and keratin. His Ki-67 was estimated at 5%. Barrett *et al.*^[25] showed that meningiomas expressing high Ki-67 and having active mitosis were predisposed to recurrence postoperatively.

Generally, it is accepted that extracranial metastases of meningiomas are more common with anaplastic (30%) and atypical (5%) cases^[15]. However, this rate seems overestimated since 2016 the histopathological diagnostic criteria for high-grade meningiomas have been updated. Additionally, in the past, reported cases of "metastatic angioblastic meningioma" were not true metastases, as they are now diagnosed as hemangiopericytomas^[26,27].

For management, the treatment of cerebral meningioma is based on complete surgical excision or on radiosurgery and adjuvant radiotherapy in case of subtotal resection^[5,17]. However, Kessler *et al.*^[3] studied the records of 168 patients between 1993 and 2014 and showed that there was no significant difference in progression-free survival whether or not radiotherapy was administered postoperatively. In the same subject, Keric *et al.*^[28] examined, in 2020, the medical records of all patients operated on for histologically diagnosed primary atypical meningioma over a 10-year period to evaluate progression-free survival and prognostic factors. Their conclusion was that radiotherapy following surgery did not improve progression-free survival. Furthermore, the most important prognostic factors were the quality of the surgery and the age of the patients.

Regarding hepatic metastatic locations, there is no wellcodified treatment^[29], but their surgical resection has led to a good prognosis and good survival^[7]. If the liver metastasis remains asymptomatic, it may be monitored radiologically^[9]. Rampurwala et al.^[9] monitored a case of metastatic meningioma to the liver for 5 years with stability of the patient's condition. Recently, new research has proven the usefulness of (68Ga) DOTATATE PET/CT since meningiomas express somatostatin receptor, which normal tissues lack^[30]. Chemoembolization of the liver mass appears to be the ideal solution for patients suffering from hypoglycemia^[21]. This technique may be applied to symptomatic tumors with large liver mass. Chemotherapy showed limited or no benefit in meningioma. However, recent guidelines published by the National Comprehensive Cancer Network suggest that only four molecules including sunitinib, bevacizumab, bevacizumab and everolimus, and somatostatin analogs may be used in the treatment of recurrent meningiomas^[31]. In 2014, Kaley et al.^[32] reviewed 47 publications analyzing the effectiveness of several molecules (temozolomide, bevacizumab, irinotecan, IFN- α , hydroxyurea, octreotide analogs, gefitinib, imatinib, erlotinib, mifepristone, megestrol acetate). They reported poor progression-free survival at six months for WHO grade II and III lesions in case of systemic chemotherapy. Our patient received 6 courses of chemotherapy based on bevacizumab for a period of 3 months with favorable and satisfactory outcomes at our outpatient clinic. He started to gain weight and he mentioned a clear reduction in complaints while waiting to finish the second half of his courses.

Through our case, we emphasize the importance of thorough and repeated reevaluation of patients with a history of cerebral meningioma, particularly those with WHO grade II or III. This reevaluation aims for the fairly early discovery and management of any distant metastasis, even if these metastases are rare, and this to improve the long-term prognosis of patients.

Conclusion

In conclusion, in the report of liver lesions, the first differential diagnosis in mind should be metastatic lesions, if there is a prior clinical history of primary tumors. In the case presented, there was a previous diagnosis of high-grade meningioma, and, according to the literature, metastases from this type of meningiomas remain rare and occur more frequently after several recurrences of the primary tumor. As a result and due

to this rarity, there are currently no guidelines regarding the treatment or classification of this entity. Additional studies must take place in order to better understand the pathophysiology of this disease and to better target its treatment.

Ethical approval

Not applicable.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Patient perspective

During hospitalization and at the discharge, the patient has given the opportunity to share their perspectives and he was satisfied with the care.

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References

- Kalamarides M, Stemmer-Rachamimov AO, Niwa-Kawakita M, et al. Identification of a progenitor cell of origin capable of generating diverse meningioma histological subtypes. Oncogene 2011;30: 2333–44.
- [2] Costea CF, Cucu AI, Bogdănici CM, *et al.* The myth of Prometheus in metastatic meningioma to the liver: from craniotomy to hepatectomy. Rom J Morphol Embryol 2021;62:351–9.
- [3] Kessler RA, Garzon-Muvdi T, Yang W, et al. Metastatic atypical and anaplastic meningioma: a case series and review of the literature. World Neurosurg 2017;101:47–56.
- [4] Akagi T, Iwata K, Utsunomiya T, et al. An autopsy case of meningioma with extracranial remote metastases. Acta Pathol Jpn 1974;24: 667–71.
- [5] Goldsmith BJ, Wara WM, Wilson CB, et al. Postoperative irradiation for subtoally resected meningiomas. A retrospective analysis of 140 patients treated from 1967 to 1990. J Neurosurg 1994;80:195–201.
- [6] Ku JK, Vasquez JC, Hoyt DB, et al. Metastatic meningioma to the liver successfully treated with a wedge resection and radiation therapy: report of a case. Surg Today 2005;35:82–5.
- [7] Moir JA, Haugk B, French JJ. Hepatic metastasis via a ventriculo-peritoneal shunt from an intracranial meningioma: case report and review of the literature. Case Rep Gastroenterol 2010;4:267–72.
- [8] Garcia-Conde M, Roldan-Delgado H, Martel-Barth-Hansen D, et al. Anaplastic transformation of an atypical intraventricular meningioma with metastases to the liver: case report. Neurocirugia (Astur) 2009;20: 541–9.
- [9] Rampurwala M, Pazooki M, Schauer P. Delayed hepatic metastasis from a benign fibroblastic meningioma thirty-one years after surgical resection of the intracranial tumor. J Clin Oncol 2011;29:e214–5.
- [10] Sheng B, Liu YLC. Liver metastasis from typical meningioma. World Neurosurg 2021;145:334–7.
- [11] Sohrabi C, Mathew G, Maria N, et al. The SCARE 2023 guideline: updating consensus Surgical CAse REport (SCARE) guidelines. Int J Surg Lond Engl 2023;109:1136.
- [12] Casas I, Báez A, Banfi N, et al. Meningiomas en neurooncología. Neurol Argentina 2016;8:210–26.
- [13] Thomas RZ, Dalal I. Extracranial metastases of anaplastic meningioma. BJR Case Rep 2017;3:20150092.
- [14] D P. Fibrosarcoma of the dura mater. Trans Pathol Soc Lond 1886;37:12–4.
 [15] Enam SA, Abdulrauf S, Mehta B, *et al.* Metastasis in meningioma. Acta Neurochir 1996:138:1172–8.
- [16] Ore CLD, Magill ST, Yen AJ, et al. Meningioma metastases: incidence and proposed screening paradigm. J Neurosurg JNS 2019;132: 1447-55.
- [17] Surov A, Gottschling S, Bolz J, et al. Distant metastases in meningioma: an underestimated problem. J Neurooncol 2013;112:323–7.
- [18] Figueroa BE, Quint DJ, McKeever PE, et al. Extracranial metastatic meningioma. Br J Radiol 1999;72:513–6.
- [19] Som PM, Sacher M, Strenger SW, et al. "Benign" metastasizing meningiomas. AJNR Am J Neuroradiol 1987;8:127–30.
- [20] Zaghouani H, Yahyaoui S, Chabchoub I, et al. Vertebral metastases from intracranial meningioma. Acta Radiol Short Rep 2014;3:20479 81613494199.
- [21] Ferguson JM, Flinn J. Intracranial meningioma with hepatic metastases and hypoglycaemia treated by selective hepatic arterial chemo-embolization. Australas Radiol 1995;39:97–9.
- [22] Jenkinson ML, Watson AJ, Boyle S. Hepatic metastasis from intracranial meningioma. Postgrad Med J 1987;63:199–200.
- [23] Commins DL, Atkinson RD, Burnett M. Review of meningioma histopathology. Neurosurg Focus 2007;23:E3.
- [24] Abdelzaher EAD. Expression of mesothelioma-related markers in meningiomas: an immunohistochemical study. Biomed Res Int 2014; 2014:968794.
- [25] Barrett OC, Hackney JR, McDonald AM, et al. Pathologic predictors of local recurrence in atypical meningiomas following gross total resection. Int J Radiat Oncol Biol Phys 2019;103:453–9.
- [26] Kabus D, Sidhu GS, Wieczorek RLCH. Metastatic meningioma. Hemangiopericytoma or angioblastic meningioma? Am J Surg Pathol 1993;17:1144–50.
- [27] Louis DN, Ohgaki H, Wiestler OD, et al. The 2007 WHO Classification of Tumours of the Central Nervous System. Acta Neuropathol 2007;114: 97–109.

- [28] Keric N, Kalasauskas D, Freyschlag CF, et al. Impact of postoperative radiotherapy on recurrence of primary intracranial atypical meningiomas. J Neurooncol 2020;146:347–55.
- [29] Beutler BD, Nguyen ET, Parker RA, et al. Metastatic meningioma: Case report of a WHO grade I meningioma with liver metastases and review of the literature. Radiol Case Rep 2019;15:110–6.
- [30] Kowalski ES, Khairnar R, Gryaznov AA, et al. 68Ga-DOTATATE PET-CT as a tool for radiation planning and evaluating treatment

responses in the clinical management of meningiomas. Radiat Oncol 2021;16:151.

- [31] Nabors LB, Portnow J, Ahluwalia M, et al. Central Nervous System Cancers, Version 3.2020, NCCN Clinical Practice Guidelines in Oncology. J Natl Compr Canc Netw 2020;18:1537–70.
- [32] Kaley T, Barani I, Chamberlain M, et al. Historical benchmarks for medical therapy trials in surgery- and radiation-refractory meningioma: a RANO review. Neuro Oncol 2014;16:829–40.