Editorial

Distant Metastases from Meningiomas — A Myth or Reality?

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EDITORIAL

Meningioma is a common slow-growing benign intracranial neoplasm arising from the arachnoid cap and is composed of neoplastic meningothelial (arachnoidal) cells. Meningiomas account for 14-19% of all primary intracranial neoplasms [1]. Though the majority of meningiomas behave in a benign fashion, malignant behavior of meningiomas is well documented, especially with repeated recurrences. Such malignant behavior is usually limited to local recurrence and/or cranio-axial spread. Extracranial metastases from meningiomas are an exceeding uncommon event and are usually found in <0.1-0.2% of cases, many of whom have advanced disseminated disease, though isolated metastases to the liver and lung have also been reported [1,2]. Rarely, delayed metastases may be seen several decades after treatment of the initial tumor and in the absence of local intracranial recurrence [1].

The World Health Organization (WHO) classifies meningiomas into three types as a risk assessment for the likelihood of recurrence and/or aggressive behavior: a) Benign (grade I) includes meningothelial, fibrous, transitional, psammomatous, and angioblastic; b) Atypical (grade II) includes chordoid, clear cell, and atypical; c) Anaplastic/Malignant (grade III) includes papillary, rhabdoid, and anaplastic. The risk of metastatic spread increases with higher grades. The rare metastases of histologically benign meningiomas typically occur following multiple surgeries for repeated recurrences. The most useful histological predictor for the likelihood of recurrence is tumor grade; however brain invasion and histologic anaplasia, are additional criteria. Benign meningiomas (grade 1) typically have local recurrence rates of 7-20%; atypical (grade 2) meningiomas recur 29-40% while anaplastic meningiomas are 50-78% [3]. Though some authors feel that brain invasion suggests a higher likelihood of recurrence, this remains open to debate as an independent predictive factor [4]. A higher incidence of metastatic spread has been reported in atypical and malignant meningiomas.

Extracranial metastases of meningioma may be detected in the lungs (60%), abdomen and liver (34%), cervical lymph nodes (18%), long bones, pelvis, and skull (11%), pleura (9%), vertebrae (7%), and mediastinum (5%) [1]. Though the primary route of dissemination remains poorly understood, four possible routes of metastases have been proposed. Hematogenous dissemination via the jugular vein may be responsible for metastatic spread, a theory supported by finding metastases in the cervical lymph nodes, cervical soft tissue, parotid gland, thyroid gland, cervical bones, and lung/pleura [5]. Alternatively or additionally, the paravertebral venous plexus may be the primary route of spread, as it connects with the inferior vena cava, accounting for metastases detected in the vertebrae, kidney, peri-renal tissue, and adrenal gland. This is the most likely mechanism as 75% cases have a documented history of prior surgery or venous sinus invasion [2]. Lymphatics may additionally play a role. Finally, the cerebrospinal fluid (CSF) may also be implicated in the spread of meningiomas [5].

Risk factors for the development of metastases from a meningioma include histological criteria such as high cellularity, cellular heterogeneity, high mitotic rate, nuclear pleomorphism, tumor necrosis, and invasion of adjacent blood vessels [4, 5]. Most patients with distant metastases have a history of repeated surgical resection of the primary tumor, suggesting a role for surgical resection in initiating tumor spread [5]. Other factors that may increase the risk of metastatic spread include a previous craniotomy, venous sinus invasion, local recurrence, papillary morphology, and a predisposition to malignancy.

As extracranial metastases from meningiomas are of rare occurrence, a high degree of clinical suspicion is warranted for accurate diagnosis with a special emphasis in patients that have a documented history of multiple surgeries. Thus, it is most likely that the true incidence/prevalence of metastases in meningiomas is largely underreported. In conclusion, therefore, the myth that meningioma is a benign tumor is challenged. The reality is that not only can 'benign' meningiomas become malignant; they can also acquire independent metastatic potential.   In this context, they can leave their abode and travel far and wide, presenting as a diagnostic pitfall for the unwary.

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