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# "The Silk Road" via subarachnoid cisterns. Cerebrospinal fluid dissemination of meningiomas

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## ABSTRACT
Meningiomas are generally slow-growing extra-axial benign tumours and in rare cases they can metastasize both neural and extra-neural. Intracranial meningiomas with leptomeningeal dissemination are extremely rare and the exact pathogenesis still remains unknown. The aim of this review is to analyse the pathways of intracranial and spinal metastatic spread of intracranial meningiomas and to discuss their particular clinical and pathological features. We highlight the fact that there is a possibility of leptomeningeal dissemination, even if cerebrospinal fluid cytology is negative, in patients with a medical history of a resected meningioma. We identified three possible ways of dissemination: haematogenous, through the CSF, or during surgery. From a histopathological point of view, the more malignant the meningioma, the more likely its leptomeningeal dissemination.

## INTRODUCTION
Meningiomas are generally benign intracranial tumours and represent approximately 30% of all primary central nervous system tumours, with an incidence that has increased in recent years (1, 2, 3, 4, 5). Usually occurring on the surface of the brain as they originate in the arachnoid cells, meningiomas are generally slow-growing extra-axial benign tumours. In very rare cases, especially when the tumours become malignant, meningiomas can metastasize both in neural and extra-neural sites (6).

Only 0.1% of meningiomas are thought to metastasize (7), and usually these cases were atypical and anaplastic meningiomas (8, 9, 10), also known for their tendency to relapse after surgery (2, 11, 12, 13). Ather Enam et al. report an overall metastatic risk of 5% for atypical meningiomas and of 30% for anaplastic meningiomas (8), although other authors consider these percentages to be overestimated (11, 14, 15).

Meningiomas can metastasize in various organs (lung, liver, spleen, adrenal gland, thyroid, parotid gland) or bones, skin and deep soft tissue (16), but also in the intracranial or spinal space through cerebrospinal fluid (CSF) pathways. According to the existing literature, metastatic dissemination of meningiomas through CSF is a rare presentation (17, 18, 19, 20, 21) and, since 1950, only 45 cases have been reported (22). Nevertheless, different other authors consider that the CSF dissemination risk of meningiomas is an uncommon event, as it may occur in 4% of metastasizing meningiomas (17, 23).

However, the first historical description of leptomeningeal metastasis was made by Oliver in 1837, followed by Eberth in 1870 (24, 25) Also, the first description of carcinoma cells in the CSF was realized by Dufour (26) and, in 1912, Berman was the first who used the term "meningeal carcinomatosis" (27). However, Kalm, in 1950, was the first who published a case of a malignant tentorium tumour, i.e. an anaplastic meningioma that metastasized in the medulla oblongata and in the leptomeningeal space (28).

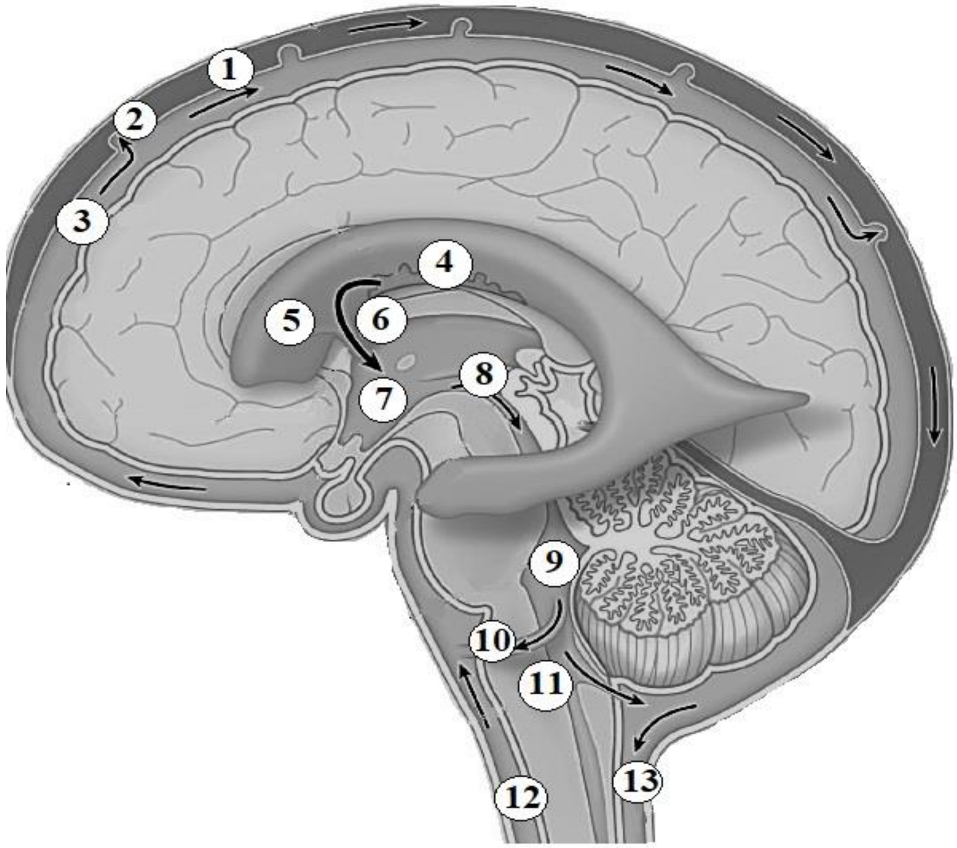


FIGURE 1. Cerebrospinal fluid circulation: (1) superior sagittal sinus, (2) arachnoid granulation, (3) convexity subarachnoid space, (4) choroid plexus (site of CSF production), (5) lateral ventricle, (6) interventricular foramen (Monro), (7) third ventricle, (8) cerebral aqueduct (Sylvius), (9) fourth ventricle, (10) lateral aperture (foramen of Luschka), (11) median aperture (foramen of Magendie), (12) ventral subarachnoid space, (13) dorsal subarachnoid space. (Adapted after Ropper AH et al., Adams and Victor's Principles of Neurology, 11th Edition)

## PATHWAYS OF METASTATIC SPREAD IN MENINGIOMAS

Meningioma cells gain access to the subarachnoid space in several ways: haematogenous pathway, through the CSF, or during surgery. More frequently, the spread is haematogenous via the venous system and tumour cells gain access to the CSF through the dural sinuses or epidural plexus, especially in meningiomas that invade dural sinuses (29, 30). A second route of dissemination is by CSF with the tumour spreading throughout the neuroaxis (Figure 1).

In a review, Rawat et al. highlighted the fact that approximately 75% of patients with meningiomatous metastasis regardless of the route they followed have had previous operation for the primary tumour (31). Also, it was hypothesized that tumour cells gain access to the vascular channels and meningeal surface by seeding during surgery (32). Although this theory has earlier been rejected (33, 34), now it is accepted that surgical manipulation may release tumour cells into the CSF (30, 31).

Metastatic spread during or after surgery exists theoretically, even though this theory is unlikely. But the incidence of leptomeningeal dissemination after meningiomas surgery is low and there are also cases with leptomeningeal dissemination through CSF without surgical intervention (35). Regarding malignant transformation of meningiomas after surgical resection, there is also the theoretical one (22). Koenig et al. reported that at the site of surgical injury, the growth factors can promote malignant changes inside the meningeal tumour (36) and this event was observed by Morantz and Shain in an experimental rat model (37).

## Intracranial and spinal cord dissemination through CSF pathways

There are variable sites of meningioma metastasis through CSF pathways. The metastasis occurred due to leptomeningeal seeding from the neighboring meningioma, and the spinal canal is the second most common site (17) (Table 1).

In literature, spinal metastasizing meningiomas are rarely reported (17, 38, 39, 40, 41). In spinal intradural dissemination, the tumour cells have a tendency to accumulate more frequently at dorsal nerve root level, especially in the medullary cone and cauda equine, probably due to directional active flowing of the CSF throughout the neuroaxis, and also to the effect of gravity (42, 43). In a large study on 200 consecutive meningiomas, of WHO grade I, which were followed prospectively during a median time of 8.5 years, Chamberlain and Glantz reported that 4 patients (2%) were diagnosed with spinal metastasis (21). Vries et al. also reported CSF or drop metastasis in 5% of non-benign meningiomas (44).

Meningioma metastasis may be simultaneous, both in the intracranial space and in the spinal cord (17, 45). In 1992, Akimura et al. reported a malignant meningioma metastasizing through the CSF pathways, both in the cerebellopontine angle cistern and in the thoracic spinal cord. The primary tumour was a parasagittal malignant meningioma two-times operated, the second time for recurrence. At the first surgery, the frontal horn of the lateral ventricle was opened because the meningioma infiltrated into the deep frontal brain. The authors concluded that this artificial communication between the meningioma cavity and the CSF pathways enhanced the probability for tumour metastasis into cerebellopontine angle cistern and thoracic spinal cord (17).

## Neoplastic meningitis

It is well known that neoplastic meningitis is more common with solid carcinoma such as lung, breast and gastrointestinal cancer (46), but literature also reported few malignant meningioma cases with CSF dissemination into the brain ventricles (47). There were only eight cases (47, 48) with intracranial or intraspinal malignant meningioma arising from low-grade meningiomas, which disseminated throughout the CSF (47). In neoplastic meningitis, CSF dissemination of malignant meningioma cells may cause a variety of neurological symptoms such as disturbances of multiple cranial nerves, hydrocephalus, cerebellar dysfunction and multiple spinal nerve roots or cauda equine symptoms (49).

Brainstem damage by CSF dissemination of malignant cell is rare, literature reporting a few cases with central hyperventilation, Wallenberg syndrome and diplopia, facial nerve palsy and unsteadiness of gait secondary of CSF dissemination of malignant tumour cells (20, 47, 50).

It is important to mention, in terms of diagnosis, that only 54% of all cases with leptomeningeal meningiomatous dissemination revealed malignant cells in the CSF on initial lumbar puncture, and only 8% of these cases remained negative, even after repeated examinations (51). The reason for this low specificity of the lumbar puncture remains unclear, although Fujimaki et al. speculate the fact that malignant cells adhere rather than float freely in the CSF (47). Although identification of malignant cells by CSF cytology has been considered the diagnostic gold standard, these paradigms have changed nowadays due to limited sensitivity of cytology.

### Histopathology of CSF-disseminated meningiomas

Even though the pathogenesis of CSF - disseminated meningiomas is not completely understood, over time researchers have issued various theories. In this regard, Engelhard proposed three different pathways of dissemination: (1) tumour cells could be "shed" away directly into the CSF due to direct contact between an anaplastic meningioma and CSF pathways; (2) tumour cells might invade the leptomeningeal space during its progression; 3) the tumour cells might be inoculated within the CSF at the time of the surgery (52).

Other authors, such as Russel and Rubinstein, considered that tumour friability may play an important role in meningioma dissemination within CSF (42). However, considering that meningiomas arise from arachnoid cells and are naturally exposed to CSF during their growth, it is difficult to explain the scarcity of meningioma dissemination through CSF (22) as Chamberlain and Glantz reported that only 4% of the meningiomas could present leptomeningeal dissemination and positive CSF cytology at the time of the diagnosis (21).

More theoretically, the risk of intraventricular meningiomas to metastasize through CSF pathways should be high, but literature reports only nine cases of intraventricular meningiomas (22). As an explanation, Miller and Ramsden considered that the dynamics of the CSF pathways might prevent fragment formation and deposition of tumour cells (22, 53). However, in a review of 45 cases of meningioma with leptomeningeal dissemination through CSF, Park et al. noticed that the period of time needed for leptomeningeal dissemination in cases with intraventricular meningiomas is the shortest when compared to other intracranial and spinal meningiomas (22).

All histological subtypes of meningiomas can metastasize (Table 1), even benign meningiomas (45, 54, 55, 56, 57, 58), but meningeal tumours with clearly malignant features have a higher metastatic rate (59). Metastatic meningiomas are associated more frequently with aggressive meningiomas (WHO grade 2, and WHO grade 3), with a range of occurrence of 10-25% (21), i.e. the more malignant the meningioma, the more likely its leptomeningeal dissemination (9, 17, 41, 60).

According to literature, from a histological point of view, several factors are predictive of meningioma metastasis, including high cellularity, nuclear pleomorphism (Figure 2), high mitosis rate, tumour necrosis, and invasion of blood vessels (7, 8). On the other hand, other authors consider that the metastasizing behaviour of these tumours is not correlated with their histological features (30, 31).

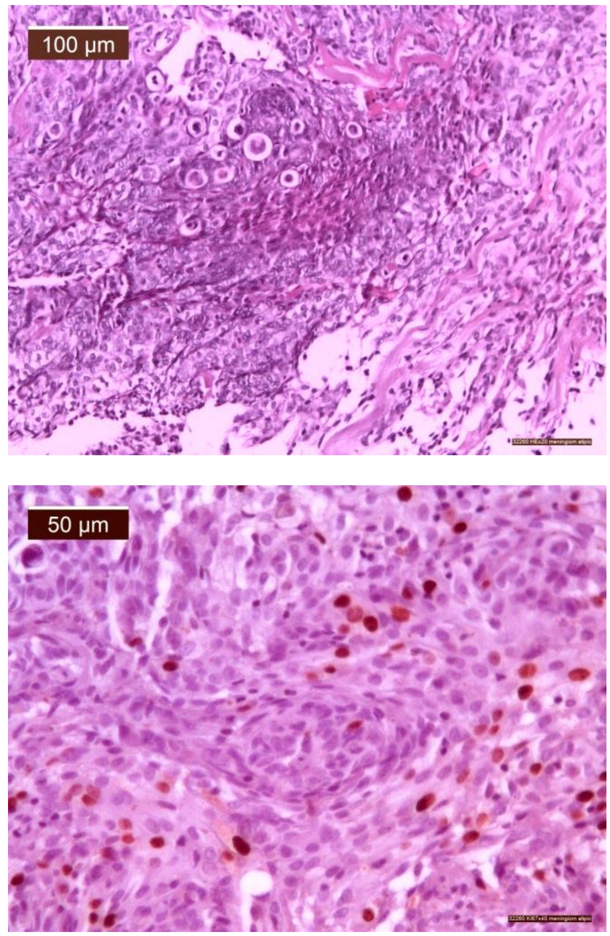


**Figure 2.** Microphotographs of atypical meningioma, WHO grade II. A. A tumour made up of meningothelial cells arranged in a sheet-like pattern, with increased cellularity, pleomorphism, and areas with small cells having high Nucleus/Cytoplasm ratio (HE staining, x20); B. Ki67 labelling index has high values and indicates a malignant neoplasia (Anti-Ki67 antibody immunohistochemical staining, X40).

### Conclusion

Intracranial meningiomas with CSF dissemination are extremely rare and, despite reports from literature, their pathogenesis remains unknown. In the case of a patient with a resected meningioma, the possibility of CSF dissemination of tumour cells should be borne in mind, even if CSF cytology is negative. Also, a spinal MRI should be performed, especially when spinal signs and symptoms are present.

**TABLE 1.** Literature review of meningioma cases with CSF- dissemination

| Year of publication, Author | Primary site of meningioma | WHO Grade | Intracranial metastasis | Spinal metastasis | Time to CSF disseminated disease |
|---|---|---|---|---|---|
| 2013, Tsuda et al. (43) | T10-11 intradural | I →II | - | + | 12 years |
| 2011, Wu et al. (61) | Convexity | III | + | - | 1.7 years |
| 2011, Kim et al. (62) | Posterior fossa | III | + | - | 0 |
| 2011, Peng et al. (63) | Medial temporal | III | + | - | 2 years |
| 2009, Kuroda et al. (64) | Skull base | I→II→III | + | + | 6.3 years |
| 2009, Eom et al. (6) | Lateral ventricle | II→III | - | + | 1.3 years |
| 2008, Erkutlu et al. (65) | Posterior fossa | III | - | + | 2.7 years |
| 2008, Santhosh et al. (65) | Convexity | III | + | + | 9 months |
| 2007, Shintaku et al. (67) | Lateral ventricle | I→III | + | - | 4.3 years |
| 2006, Chuang et al. (68) | Convexity | III | - | + | 3 months |
| 2005, Cramer et al. (60) | C1-C3 intradural | II | - | + | 1.4 years |
| 2005, Al-Habib et al. (69) | Not mentioned | III | + | + | 2 months |
| 2005, Chamberlain and Glantz (21) | Not mentioned, 8 cases | I | + (8 cases) | + (6 cases) | - |
| 2005, Wakabayashi et al. (70) | Frontal convexity | III | + | - | 13 years |
| 2005, Koenig et al. (36) | Temporal lobe | III | - | + | 1 month |
| 2005, Darwish et al. (71) | Lateral ventricle | II→III | + | + | 7 months |
| 2002, Ramakrishnamurthy et al. (35) | Lateral ventricle | I | + | - | 4 years |
| 2001, Conrad et al. (72) | Convexity | I→II→III | + | + | 6.4 years |
| 2000, Meinsma-VdTuin et al. (73) | C2-C4 intradural | III | + | + | 6 months |
| 2000, Lee et al. (74) | Convexity | III | + | + | 9 years |
| 1998, Lee and Landy (41) | Convexity<br>Skull base<br>Convexity | III<br>I<br>III | +<br>+<br>- | +<br>+<br>+ | 1.5 years<br>3.8 years<br>8.5 years |

| | | | | | |
|---|---|---|---|---|---|
| 1995, Peh and Fan (39) | Lateral ventricle | III | + | + | 5 years |
| 1993, Greenberg et al. (75) | Lateral ventricle | III | + | + | 2 months |
| 1992, Satoh et al. (18) | Skull base | I | + | + | 0 |
| 1992, Akimura et al. (17) | Convexity | III | + | + | 1.8 years |
| 1989, Kamiya et al. (76) | Lateral ventricle | III | - | + | 6 months |
| 1987, Strenger et al. (77) | Third ventricle | III | + | - | 1.5 months |
| 1985, Kleinschmidt-DeMasters and Avakian (20) | Lateral ventricle | III | + | + | 1.7 years |
| 1975, Ludwin and Conley (78) | Convexity | III | + | + | 10 months |
| 1972, Miller and Ramsden (53) | Convexity | III | + | - | - |
| 1971, Riley et al. (22, 78) | Convexity | I→III | - | + | - |
| 1970, Shuanghoti et al. (22, 78) | Convexity | III | - | + | - |
| 1963, Russell et al. (22, 78) | Skull base | I | + | - | - |
| 1960, Hoffman et al. (22, 78) | Convexity | I→III | + | + | - |
| 1954, Winkelman (22, 78) | Skull base | I | + | - | - |
| 1950, Kalm (22, 78) | Posterior fossa | III | + | - | - |

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