Carcinoma Metastasis to a Cerebral Vascular Malformation: Case Report

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Abstract : The association of a metastatic carcinoma with a cerebral vascular malformation is presented. Although metastases into slow growing, vascular intracranial tumors are occasionally reported, this association does not seem to be reported previously. This case supports the hypothesis that distribution of intracranial metastases is closely related to the extent of intracranial blood flow. Hence, lesions with increased vascularity and flow such as vascular malformations are more likely to receive and entrap the metastatic emboli.

Key Words : Cerebral vascular malformation, Metastatic carcinoma.

INTRODUCTION

The association of brain metastases with certain primary intracranial neoplasms has occasionally been reported(1–6,9). Carcinoma metastasis to a cerebral vascular malformation is extremely rare. We report a case of pulmonary carcinoma metastasis within a silent cerebral arteriovenous malformation, recognized during excision of the parietal metastatic nodule.

Case report:

This 41–year–old male patient was examined elsewhere with complaints of a sudden numbness and weakness on the left side of his body followed by a focal motor seizure, a month before admission. Computerized tomography (CT) scanning done elsewhere revealed a right parietal hyperdense nodule with ring enhancement and surrounding edema consistent with a metastatic nodule (Figure 1 a,b). The patient was referred to our department for further evaluation and treatment. Neurological examination at referral showed slight left hemiparesis more pronounced in the upper extremity with hypoesthesia at the same side of the body. Chest x–ray and thorax CT scan revealed a mass at the left pulmonary apex. A CT–guided percutaneous biopsy specimen demonstrated a small–cell carcinoma. Magnetic resonance imaging (MRI) for further evaluation of the intracerebral lesion demonstrated a hyperintense mass with an isointense core, with contrast enhancement and a marked peritumoral edema (Figure 2 a,b). Systemic investigation showed no other metastatic focus than intracerebral lesion so a craniotomy was planned for the right deep parietal nodule.

Operation:

A right sided temporoparietal craniotomy was performed and the tumor was reached through a small posterior parietal cortical incision. The lesion was easily stripped from the surrounding glial tissue and removed in piecemeal fashion with the aid of an operating microscope. As soon as the base of the lesion was excised, profuse bleeding occured from what was presumed to be the feeder of the lesion. After troublesome coagulation of the vessel, multiple vascular bridging channels were identified as the continuation of this artery. The whole lesion, measuring approximately 2 cm. had to be excised by using massive bipolar coagulation and aneurysm clips which required considerable time and energetic blood transfusion. The unexpectedly prolonged operation and brain retraction resulted in brain

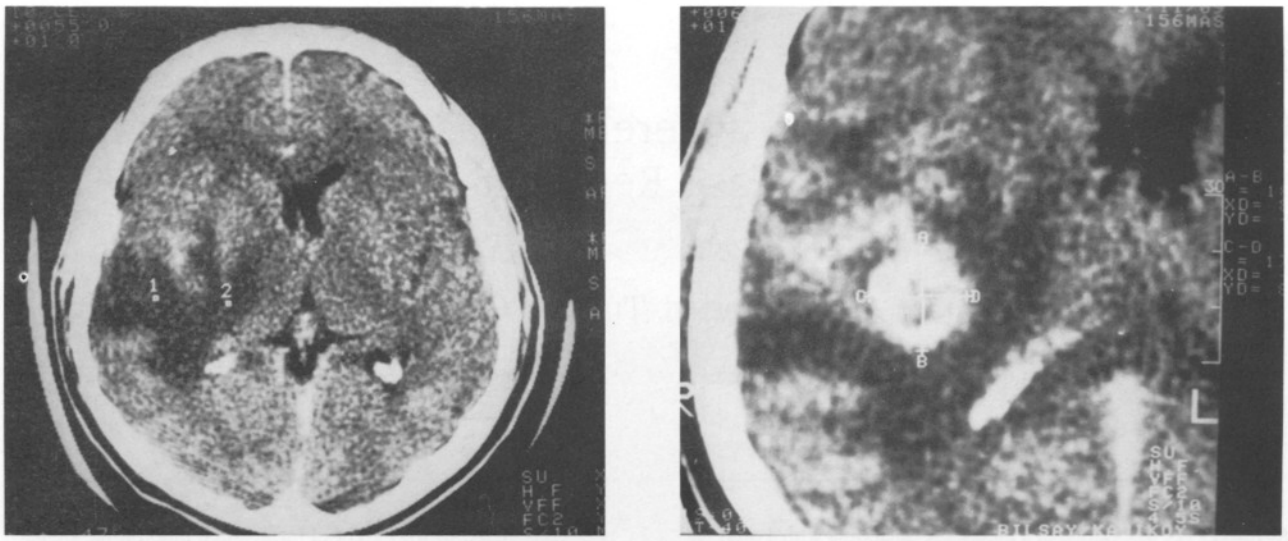

Fig. 1 : a) Pre–contrast CT scan reveals a right parieto–temporal focal edema with a midline shift. b) Post–contrast CT shows a right parietal round lesion with hypodense core and ring enhancement within the edematous area.

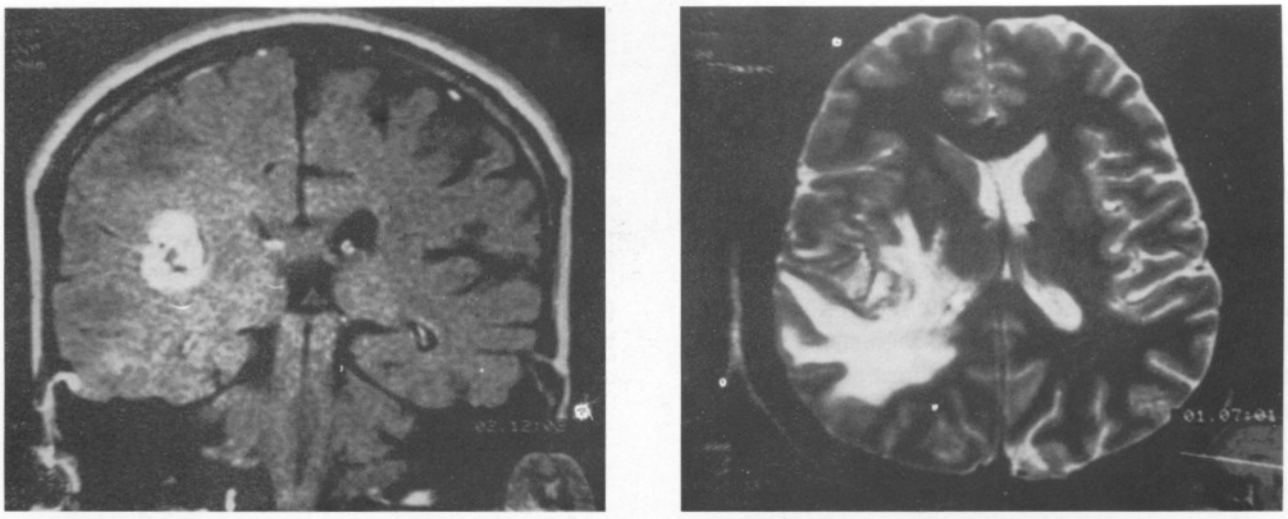

Fig. 2 : a) T1 weighted sagittal MR image obtained after iv. gadolinium injection showing the parieto–temporal hyperintense lesion with a hypointense core. b) T2 weighted axial image demonstrates the peritumoral edema and the obstruction of the right lateral ventricle.

swelling and a duraplasty had to be performed during closure. The patient recovered from the anaesthesia with worsened hemiparesis and a left homonymous hemianopia. Early postoperative CT scan revealed temporoparietal edema with no mass or enhancing lesion. After early rehabilitation therapy the patient was referred to the cardiothoracic surgery for primary tumor excision.

Pathological examination of the operative specimen was consistent with indifferentiated small cell carcinoma metastasis with an arteriovenous malformation (Figure 3 a,b).

DISCUSSION

Brain metastases from various primary sources represent a major cause of morbidity and mortality in cancer patients. Parenchymal metastases are found in nearly 20% of patients dying of cancer (7–10,12). This high predilection for the brain parenchyma especially by certain types of cancer is closely related to their tendency to spread via the blood stream. Circulating cells from tumors of the lung or tumors that

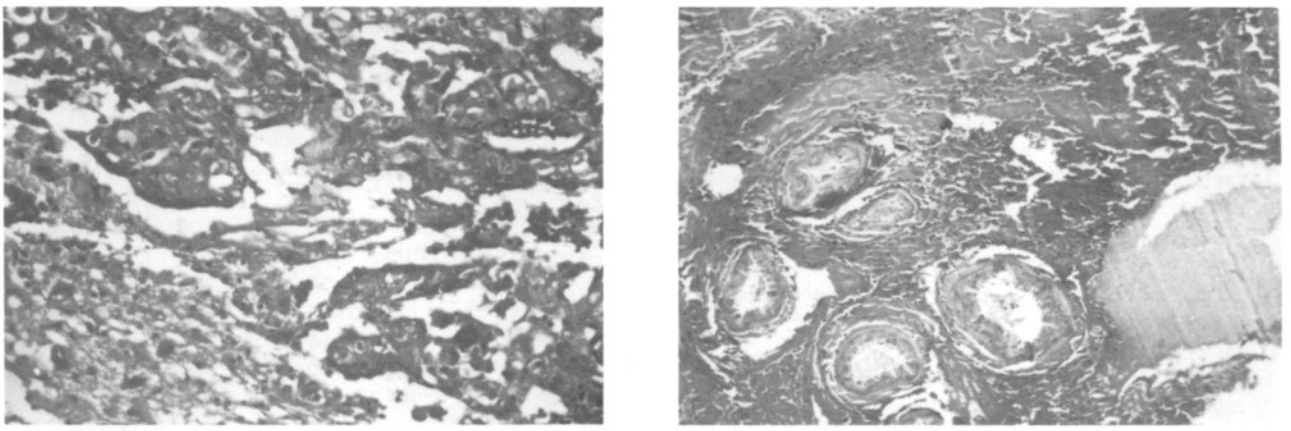

Fig. 3 : a) Histopathological examination of the lesion reveals atypical, round cells consistent with a small cell carcinoma metastasis from the lung(H & E, X110. b) Second specimen obtained adjacent and below the first lesion is proved to be an arteriovenous malformation with abnormal and primitive vascular channels of various diameters (H & E, X110).

frequently metastase to the lung have a very high incidence of entering the cranial arterial circulation. Theoretically once the tumor cells enter the intracranial bloodstream they are most often trapped at the acute arterial narrowing sites, the cortical–white matter junction. Their distribution within the cranial cavity is closely related to the region of blood flow and relative brain volume(7,12). In most cases, a preference due to middle cerebral artery territory is noted for the above mentioned reasons(12). One exception to this theory is the cerebellar metastasis, which occurs more frequently than might be suggested by relative brain weight and blood supply(11). The probable explanation for this discrepancy is that the lesions of this region present themselves with earlier and pronounced signs and symptoms compared to supratentorial involvement(12). Further evidence supporting the role of blood flow in intracranial metastatic spread is the rare but consistently reported involvement of certain primary intracranial tumors by deposits of metastatic carcinoma(5,6,12). Meningiomas and acoustic neuromas are reported to be the most common tumors that are invaded by metastatic tumor cells(1–6,8,9).The preponderance of these tumors over gliomas suggests that a rich blood supply and slow growth is a major determinant in blood–borne metastatic deposits(5,8,9). The same mechanism is proposed for neoplasm to neoplasm metastasis elsewhere in the body. Renal carcinoma, adenomas of the thyroid and adrenal cortex constitute the majority of host tumors due to their excellent blood supply and delicate stroma permitting expansile growth(4,5,7). Although this should apply for arteriovenous malformations as well, the association of a metastatic neoplasm with a supratentorial vascular malformation has not been reported previously. The only histopathologically verified case in the literature is that of Greene et al.(6), where metastatic cell deposits were detected within a cerebellar vascular malformation, which had been operated for intracerebellar hematoma following a stroke. Contrary to this case, no evidence of recent spontaneous hemorrhage or episodes of previous bleeding that would require an effort to delineate the vascularity of the lesion was seen in our patient. The association of two distinct lesions complicated the surgery due to unexpected profuse bleeding through abnormal vessels resistant to bipolar coagulation.

The presented case and previously reported cases in the literature support the hypothesis that intracranial metastatic spread is highly related to the region of intracranial blood flow, hence increasing tumor emboli entrapment by highly vascular primary lesions such as vascular tumors and malformations.

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