

# Cerebral Metastasis: Case Series Cerebrum as A Safe Haven

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## Abstract

Cerebral metastasis may be a common manifestation of systemic diseases. Cerebral metastasis are reported in 15-25 percent of patients with Brain tumors. Parenchymal blood flow is an important regulator of metastasis and most of the metastatic lesions are found in the cerebrum .most brain metastasis arise by hematogenous dissemination followed by dessimination through CSF.

**Index terms**— metastasis, cerebral, cerebellum, MRI, disseminate, microinvasion.

## 1 Introduction

eninges are the layers of connective tissue covering the brain and spinal cord The meninges consist of three membranous layers. Duramater: outer most, Arcahnoid: middle layer: Piamater: inner most. Duramater is the thickest and toughest membrane covering the brain and consists of two layers. Endosteal layer: outer. Serves as internal periosteum, (endocranium). Meningeal layer: inner: provides the protective membrane to brain. These two layers are fused to each other except where venous sinuses are enclosed between them.the cerebral metastatic lesions can be defin Supratentorial and infratentorial. The supratentoprial usually cause seizures, cognitive defects and headaches. The infratentorial lesions usually cause ataxia, Diplopia, dysarthria or brain herniation.

## 2 II. Text a) Case 1

A 45 year old male presented in emergency Department with gradual onset headache ,. There was no past history of hypertension, diabetes, Sinusitis, pyrexia. The patient was able to move her extremities, Cranial nerves were normal on examination and Brain Stem Reflexes were normal. CT showed metastatic lesions in Occipital lobe

## 3 Symptom

Percentage of Patients / 30 patients

## 4 Epidemiology

Brain Metastasis are common tumors of Brain. 1 Any tumor can spread to the brain. But the most common tumors spreading to the brain have been noticed to be lung,breast , melanoma, renal cell carcinoma, lymphoma, leukemias,thyroid, colorectal, from unknown sources. 2,3, ??, ??, ??, ??, ?? The high rates of pulmonary disease spreading to Brain has been well documented. The tumor reaches the brain via hematogeneous route. The tumor cells circulate in blood, to the left side of the heart and are carried by way of the carotid vessels to the circle of Willis from where they metastasize to different parts depending upon the dynamics of blood flow. The zones where the blood flow is considerably reduced along with narrowing of vessel diameter causes aggregation of tumor cells in these areas and represents an embolic source of disease. The parts of brain effected are the cerebrum, cerebellum and the brain stem.

In addition to the blood flow the soil seed theory of a cancer proliferation from the site of lodgement of malignant cells away from a primary source where the tumor cells grow and disseminate is important in case of cerebral metastasis and here the mitotic activity of cells is very important for rates of growth. The big and

large metastasis are due to rapid cell growth in contrast to smaller metastatic lesions. The micromechanisms involved follow a cascade of intravasation, dissemination, extravasation and colonization [10,11]. In a survey conducted from multiple hospitals from the state it was seen that most of the cases of metastasis were from lungs, others from breast in case of females and from thyroid. A large number of cases were from other cancers.

The patients usually present with headache, seizures, vomiting, alterations of mental status, visual alterations. However patients can be asymptomatic as well. Headache is the most common symptom in case of cerebral metastasis. In adults the lung cancer followed by breast cancer and melanoma are the most common sources of primary tumors. Interestingly in many cases the intracranial involvement may be the first presentation. The rates of dissemination of the tumors in pediatric age group are significantly lower than that found in adults. The main problem with the cerebral metastasis is that the tumour size is not of much significance as even small lesions can cause considerable neurological sequelae. Sometimes the patients can also present with a haemorrhage inside the metastasis. This may produce a sudden headache, coma or a severe focal neurodeficit. Metastasis from melanomas, thyroid cancers and choriocarcinomas are particularly prone for hemorrhage. Usually in about one third of cases, patients with metastasis are diagnosed on routine investigations.

IV.

## 5 Radiology

At present MRI and CT remain to be the most cost effective and non invasive techniques for detection of cerebral metastasis. Contrast enhancement can add finer details in the form of disruption of Blood Brain Barrier. [12] Typically the metastasis are well demarcated from the surrounding parenchyma. There may be peritumoral edema as well. The radiographic features differ and there is a lot of variability among these tumors. The metastasis may be solitary or multiple. Mostly the metastasis are multiple.

On Pre contrast imaging CT Scanning may show iso dense or hypodense lesions. There may be vasogenic edema. Following administration of contrast media, the enhancement may be nodular, punctate or ring enhancing. The main site of primary is the lung, breast, melanoma, renal cell carcinoma. [13] The metastasis should be differentiated from primary brain tumors, cerebral abscesses, stroke, radiation necrosis, granulomatous brain lesions, demyelination and infarcts. The clinical implication in general is that the prognosis in a patient with cerebral metastasis is generally poor. Over the past few years whole brain radiotherapy has been considered as a standard treatment. Stereotactic radiotherapy has been introduced lately and is proving to be effective. Surgery in the form of resection is used for a selective group of patients.

V.

## 6 Conclusion

Brain metastasis are common following solid cancers. A look out for cerebral metastasis should be essentially carried out as the impact on survival remains serious. Early diagnosis and aggressive therapy can be beneficial for the patient. Neuroimaging Clin N Am 1999;9:651-69.

## 7 Volume XIV Issue III Version



Figure 1:

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Figure 2: Figure 1 and 2 :

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Figure 3: I



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