Aims and objectives

GENERAL ASPECTS:

Brain metastases are the most common intracranial tumors in adults, affecting 8.3 to 14.3 per 100,000 people and 15-40% of patients with cancer, [1,2] and represent one of the most frequent neurologic complications of systemic cancer as a major cause of morbidity and mortality [10, 11]. Brain metastases are asymptomatic up to 60-75% of the time[2].

80% of brain metastases can be accounted for by five primary tumors [3];19.9% of lung cancers, 6.9% of melanomas, 6.5% of renal cancers, 5.1% of breast cancers and 1.8% of colorectal cancers[4]. Symptoms may include headache, seizure, syncope, focal neurological deficit, or papilledema.[1, 2, 5]

Imaging plays an important role in the diagnosis of central nervous system (CNS) metastasis and provide initial confirmation of previously unsuspected malignancy in patients with neurologic symptoms. Imaging may confirm metastatic disease in the setting of known systemic malignancy, and may be used to stage and restage CNS involvement during the course of treatment.

Computed tomography (CT) and magnetic resonance imaging (MRI) are the key imaging modalities used in the diagnosis of brain metastases, however magnetic resonance imaging (MRI) is more sensitive than computed tomography (CT) for detection and characterization of brain metastases [12,13,14,15].

Metastatic disease can involve different compartments of the CNS. Most commonly, metastatic disease affects the skull and/or brain parenchyma. Metastases can also involve the leptomeninges and pachymeninges [6,16,17,18]. Parenchymal blood flow is an important determinant of the distribution of metastases. 80% of metastases localize to the cerebral hemispheres, 15% localize to the cerebellum and 3% localize to the basal ganglia. Often these tumors can be found at the gray/white matter junction [19].

Typically metastases are relatively well demarcated from the surrounding parenchyma and usually there is a zone of peritumoral edema out of proportion with the tumor size. There is a great deal of variability in the appearance of these tumors, however some generalizations can be made. It should also be noted that although we tend to think of cerebral metastases as being multiple, approximately 50% are seemingly solitary at diagnosis and in a minority of cases no known or identifiable malignancy is present [4,20].

In T1 typically are iso to hypointense, if it’s hemorrhagic, may have intrinsic high signal. Melanoma metastases are hyperintense due to the paramagnetic properties of melanin. Enhancement pattern can be uniform, punctate, or ring-enhancing, but it is usually intense. Delayed sequences may show additional lesions, therefore contrast-enhance MR is the current standard for small metastases detection. In T2 are typically
hyperintense, and in FLAIR are typically hyperintense with hyperintense peritumoral edema.

However, although current imaging methods like magnetic resonance (MR) have favored the early diagnosis of metastases, there is still the difficulty to give it’s histological variety.

**AIMS AND OBJECTIVES:**

In this study we analyzed brain magnetic images and try to detect if there are specific characteristics of brain metastases with carcinoma as histological type, and compare with the general characteristics about brain metastases written in the literature, using the basic MR sequences.
Methods and materials

We retrospectively analyzed 42 MR brain images with histopathological diagnosis of cerebral carcinoma metastases. All aspects of this retrospective study were approved by the institutional review board at our institution.

The MR studies were performed in a 1.5T GE equipment on T1, perfusion, T1 with gadolinium, T2, FLAIR, and DWI sequences. We made a synoptic table and categorize the number of the lesions as unique when presented only one lesion, or multiple with two or more lesions. The location was classified in lobes; frontal, parietal, temporal and occipital, and if the lesions were in one lobe or more than one; The aspect was filed as cystic if the intensity was similar to the CSF, solid if the intensity was different to the CSF, or mixed if it had a part cyst and a part solid; the signal intensity was divided in hypointense, isointense or hyperintense to the gray matter in the different sequences. For the edema size we choose the axial FLAIR sequence and select the bigger lesion to do the measure of the bigger diameter of the lesion. After that we select the image with the bigger diameter of edema, made a measure from the external border of the lesion to the distal border of the edema, next to the union with de normal parenchyma, and calculate the percentage of the edema size in comparison with the lesion size.

All the information was written into a database and we used the IBM SPSS Statistics 22.0 program to do the analysis.
Fig. 1: Axial FLAIR image show a solid corticosubcortical lesion, isointense to gray matter, with peritumoral edema. We measure the size of the lesion (Fig 1A) and the edema size (Fig 2B) and compare each other.

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Fig. 2

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Results

42 patients were studied with basics MR sequences: 73.80% were unique and 26.20% were multiple; 83.33% had a corticosubcortical location distributed 33.33% in the frontal lobe, 26.20% in the parietal, 14.28% in the temporal, 11.90% in the occipital lobe and 14.28% occupy two lobes.

61.90% of the lesions were solid, 28.57% were cystic and 9.52% were mixed.

The 62% of the lesions presented edema smaller than the largest diameter of the lesion.

The most frequent intensity was isointense in T1, T2 and FLAIR with diffusion restriction in the 58.33%. The 12% of the patients had necrosis in connection with primary breast and kidney tumor. The 40.48% had hemorrhage in connection with primary liver tumor.

With gadolinium the 70% had an intense ring enhancement.
Fig. 3: Axial FFE show a hypointense border of hemosiderin (Fig 3) with an adjacent hyperintensity of diffusion restriction (Fig 4), a necrotic center, peripheral enhancement with gadolinium (Fig 5) and peripheral hyperperfusion (pink roi Fig 6 y 7).

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Fig. 5: Axial T1 with gadolinium observing moderate enhancement, predominantly peripheral and heterogeneous due to the presence of a necrotic center and peritumoral edema.

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Fig. 4: Axial diffusion sequence with diffusion restriction predominantly peripheral.

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Fig. 6: Perfusion sequence showing a large area of decreased left temporal perfusion with a circular area of increased perfusion.

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Fig. 7: Perfusion curve showing increased perfusion in the periphery of the lesion seen as pink curve.

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Conclusion

In this study we analyze the imaging characteristics of brain metastases with carcinoma as histological type, by standard MRI sequences with gadolinium application. The MRI was able to demonstrate all aspects analyzed. We could observe that only 16.67% of the lesion doesn't have a cortico-subcortical location, specially those which has multiple injuries having at least one lesion cortico-subcortical.

We note that although we tend to think of cerebral metastases as being multiple, the most were unique in 73.80%.

33.33% of the lesions were frontal and 26.20% were parietal probably because they have a better blood irrigation and this help for the grow of the lesions.

And the 58.33% had diffusion restriction and hyperperfusion for the hypercellularity of the metastases.

Unlike what is known about metastasis, we found that Brain Metastases for carcinoma has less edema than the size of the lesion.

In conclusion, when we have a unique lesion, frontal or parietal, corticosubcorftical, with the size of the edema less or equal than the size of the lesion, peripheral hyperperfusion and diffusion restriction, we have to think in metastases of a primary carcinoma as a possible diagnosis. However, we can extend this study with other sequences as spectroscopy, SWAN, DTI and ADC for a complete imaging study.
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