International Journal of Healthcare Sciences ISSN 2348-5728 (Online)
Vol. 4, Issue 2, pp: (1091-1098), Month: October 2016 - March 2017, Available at: www.researchpublish.com

Radiological Evaluation of Primary Brain Tumor Using CT & MRI: Systematic Review

[1]Talal Duhaim M. Alanazi, [2]Fahad Saad Z. Alanazi,
[3]Abdulrazaq Hudhayri A. Alhazeemi, [4]Rawan Mohammed Al Anazi,
[5]Sultana Ali AlShammari, [6]Woroud Mohammed Al Harthi,
[7]Mashael Abdullah Alshugaihi

Abstract: **Primary brain tumors might emerge from various cell types of the brain including glial cells, neurons, neuroglial precursor cells, pinealocytes, the meninges, choroid plexus, pericytes of the vessels, cells of the hypophysis, and lymphocytes. This review aimed to discuss the roles of CT& MRI in neuroimaging for evaluation and diagnosis of primary brain tumors, we intended to demonstrate the features of each of these imaging techniques, and then compare in between the two modalities in different aspects. Computerized and manual searches were performed to identify all relevant data publish through December 2016 looking for only English publications and human subject articles. Search was done through a PubMed search of MEDLINE, EMBASE and the Cochrane Library medical databases was performed. CT and MRI stay the main techniques utilized for the detection of primary brain tumors of the CNS. CT is very helpful in the setting of brand-new neurological indications or signs, with or without a history of malignancy. MRI is extremely delicate for the detection of brain metastases, but currently both MRI and CT are accepted methods of evaluating for brain metastases. But still MRI is a versatile and effective instrument for evaluating patients with main brain tumors. The integration of the MRI into treatment assessment helps in reducing early termination of reliable therapies because of treatment-associated imaging modifications.**

Keywords: **CT& MRI.**

1. INTRODUCTION

Primary brain tumors might emerge from various cell types of the brain including glial cells, neurons, neuroglial precursor cells, pinealocytes, the meninges, choroid plexus, pericytes of the vessels, cells of the hypophysis, and lymphocytes [(1)]. The incidence of primary brain tumors varies between subtypes. The most typical primary brain tumors in grownups are meningiomas and gliomas [(1,2)]. For gliomas, the occurrence is six to 8 in 100,000, with approximately 50% coming from malignant subtypes. Lower-grade gliomas tend to occur in younger patients, whereas higher-grade tumors are more regular in older patients. Gliomas are divided histologically into astrocytomas, oligodendrogliomas, mixed gliomas, ependymal tumors, and tumors of the choroid plexus [(1,2)]. Neuroimaging plays an essential role in the medical diagnosis and treatment of human brain tumors. Structural imaging is most commonly carried out using MRI without and with gadolinium contrast, which is the current standard of care imaging procedure for brain tumors except in cases where MRI is contraindicated [(3,4)]. Standard MRI with contrast forms the basis upon which pre-surgical preparation, post-operative evaluation, pre-radiotherapy planning, and post-treatment evaluations are made. MRI remains a vital tool for the preliminary diagnosis, treatment planning and post-treatment monitoring of brain tumors [(5,6)].

MRI with contrast is generally remarkable to CT for imaging brain tumors, CT stays more commonly available and can provide crucial complementary details [(7,8)]. CT remains the present gold basic imaging modality to detect the presence of acute intracranial hemorrhage, calcifications, and osseous anatomy. In select cases, the info provided by CT might be handy for narrowing the differential diagnosis of a recently identified intracranial mass sore. For example, coarse calcifications may be observed in oligodendrogliomas [(9)], whereas hyperdensity on CT suggests a largely cellular tumor such as lymphoma [(10)]. CT is often acquired prior to MRI for the initial work-up of a presumed intracranial mass lesion, and it is often acquired right away following stereotactic biopsy. Regardless of these situational advantages, limitations of

CT compared with MRI include inferior soft tissue characterization, posterior fossa beam hardening artifact and the use of ionizing radiation [10].

This review aimed to discuss the roles of CT& MRI in neuroimaging for evaluation and diagnosis of primary brain tumors, we intended to demonstrate the features of each of these imaging techniques, and then compare in between the two modalities in different aspects.

2. METHODOLOGY

Computerized and manual searches were performed to identify all relevant data publish through December 2016 looking for only English publications and human subject articles. Search was done through a PubMed search of MEDLINE, EMBASE and the Cochrane Library medical databases was performed. Articles extracted to the medical subject *brain neoplasms, CNS neoplasms*, Brain tumors, & CT, MRI under subheadings of *diagnosis*, detection, and evaluation along with. Articles that mapped to the medical subject Terms were evaluated to be included in this review. Furthermore, references list of identified articles was manually searched for more relevant trails.

3. RESULTS

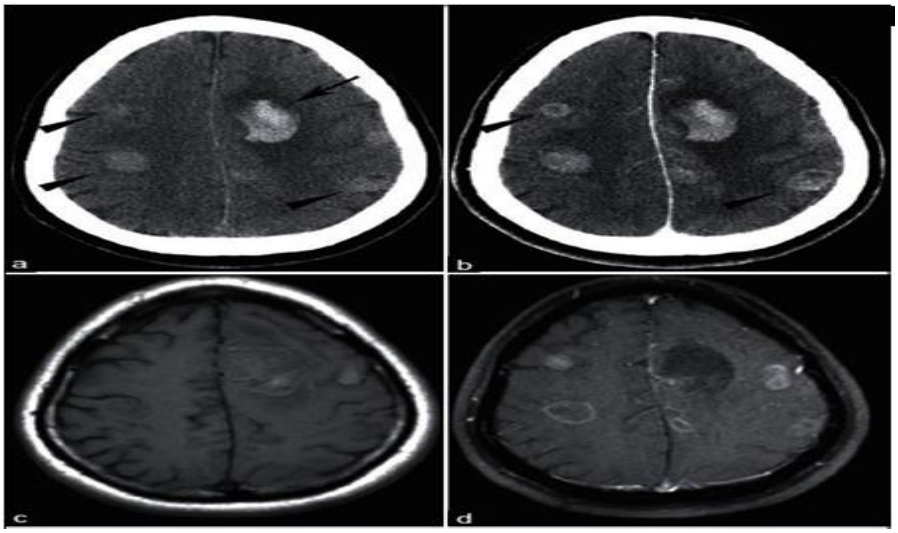
COMPUTED TOMOGRAPHY (CT):

CT may be the very first imaging modality a patient with brain tumor goes through, either in the setting of previously unrecognized malignancy, or with the development of brand-new neurologic findings and a recognized malignancy. CT alone is not delicate enough to screen for cerebral metastases, [11] but findings on CT can recommend the diagnosis. Brain tumor on CT look like solitary or multiple mass lesions with variable surrounding vasogenic edema. In the lack of hemorrhage, metastases may be hypodense, isodense, or hyperdense compared with the brain [12]. Acutely hemorrhagic metastases appear hyperdense to brain tissue (**Figure 1**) [13]. Cancer malignancy metastases tend to be hyperdense to brain on CT even in the lack of hemorrhage [14,15].

Brain metastases normally do not calcify, although there are numerous reports of this in the literature [16]. The presence of calcification may cause the consideration of alternative diagnoses, but metastases need to stay on the differential diagnoses in the appropriate clinical setting [16].

Iodinated contrast improvement is vital to the detection of metastases on CT, and brain metastases may show ring, nodular, or solid enhancement. Several reports in the literature have discovered that more metastases show up on postponed imaging [17,18] and the size of an offered transition may appear to increase on delayed imaging [17,19].

CT may be used to screen for metastases if MRI is contraindicated or unavailable, and CT has been shown to be more sensitive than noncontrast MRI for the detection of cerebral metastases (**Figure 2**) [20]. CT is less sensitive than contrast-enhanced brain MRI, nevertheless, as several studies have actually shown [20, 21,22]. In some earlier research studies, CT was discovered to be equivalent to MRI, which may be related to volume averaging artifact due to thicker pieces used in earlier MRI imaging [23].



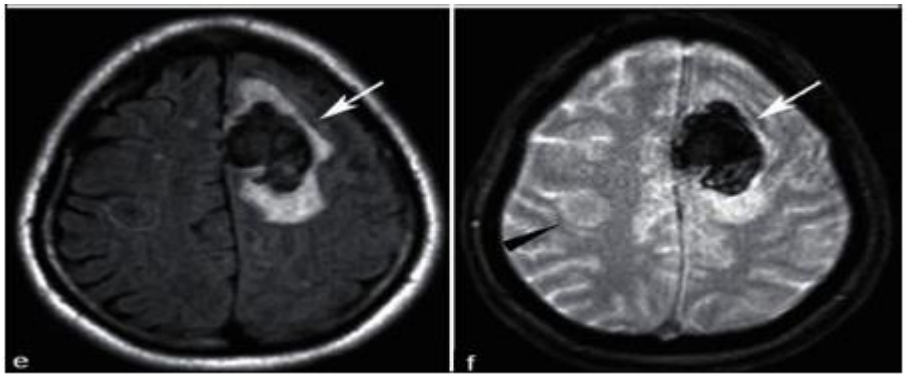


Figure1: 44 year-old found down. (a) NECT shows left frontal hemorrhage (arrow) with additional hyperdense lesions (arrowheads). (b) CECT shows enhancement, better delineating some of the masses (arrowheads). T1-weighted MRI without (c) and with (d) contrast shows multiple enhancing lesions. FLAIR (e) shows vasogenic edema surrounding the hemorrhage (arrow), but little edema associated with other lesions. T2* sequence (f) redemonstrates left frontal hemorrhage (arrow) but no blood within with the other lesions (arrowhead). [20]

CT is recommended on equal footing with MRI for the detection of asymptomatic nonsmall cell lung cancer metastases in the 2007 evidence-based ACCP guidelines, [25] in part because no improvement in survival has actually been reported based on screening with MRI versus CT [26]. If MRI is prepared based upon the NECT, and is readily available in a sensible amount of time, there might be little extra value of giving contrast at the time of CT scanning. If CECT shows multiple brain metastases, there might be little additional value of acquiring brain MRI [27].

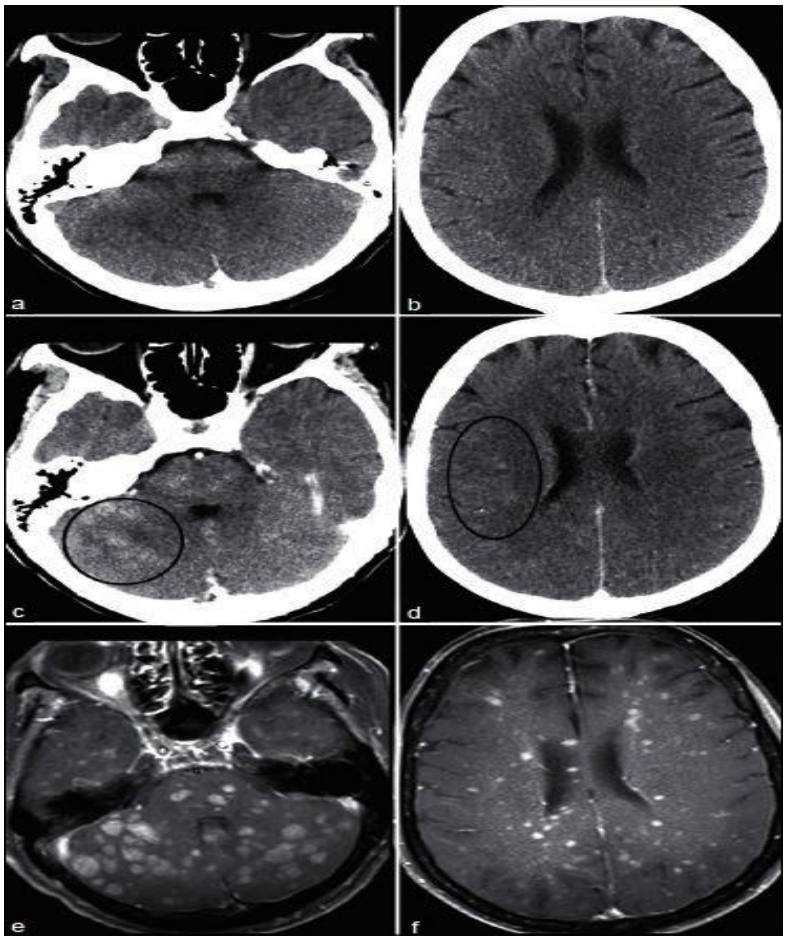
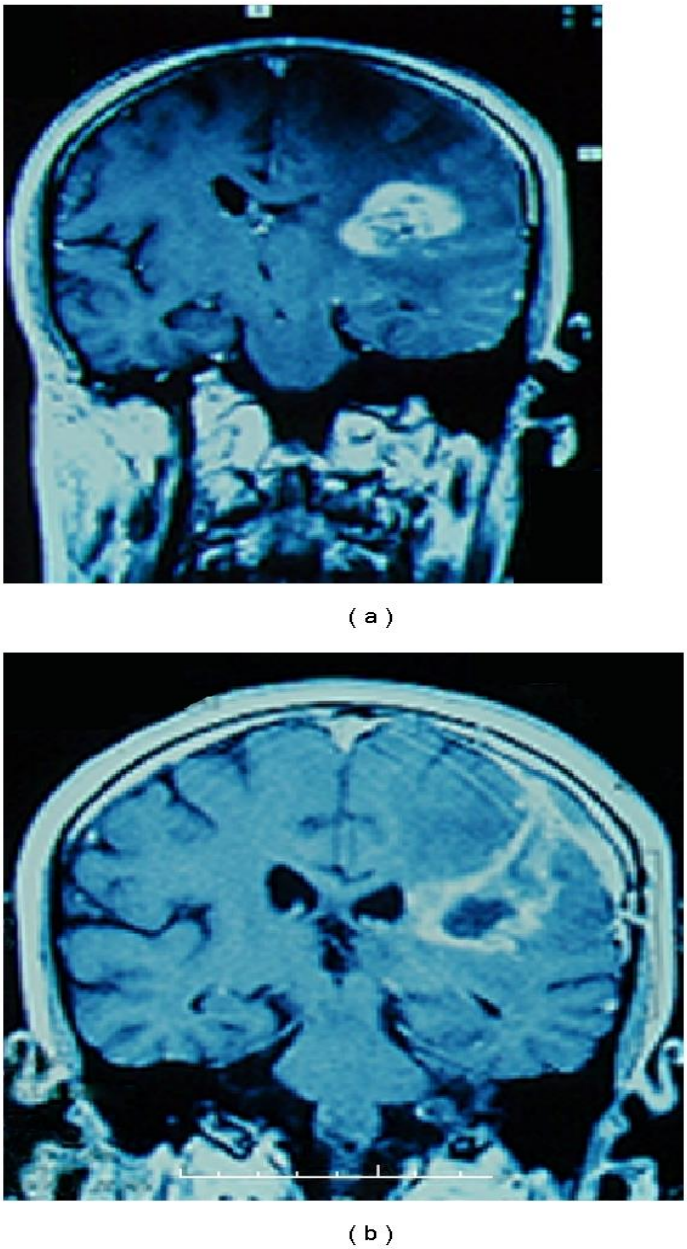


Figure 2: A 67-year-old woman with recurrent ovarian cancer and 3 weeks of progressive difficulty walking. Nonenhanced CT (a and b) was normal. After contrast administration, multiple ill-defined nodules become evident (e.g., circles in c and d). Innumerable enhancing nodules are more conspicuous on contrast enhanced T1-weighted MRI (e and f) [13]

Magnetic Resonance Images (MRI):

MRI is the very best diagnostic technique for identifying CNS metastases [28] (**Figure 3**). Large, singular, lethal metastases can be identical from top-quality astrocytomas. Utilizing conventional MRI, the presentation of an elevated rCBV (relative cerebral blood volume CBV the ratio between the typical surrounding and the pathological location) may recommend a hypervascular sore such as kidney carcinoma or cancer malignancy [29].



(a)

(b)

Figure 3: (a) axial T1-Weighted MRI after injection of Gadolinium, demonstrating an intraparietal lesion with heterogeneous appearance, presenting hypersignal intercalted with necrotic areas. (b) The same lesion in a coronal view. [28]

MRI is extremely conscious pathologic changes of normal parenchyma and has been an essential diagnostic tool in the evaluation of intracranial tumors. MRI allows a precise decision of sore area, level, mass atrophy, subacute, and effect or chronic hemorrhage, and a precise distinction in between a vascular structure and adjacent parenchyma. The typical MR scan for a patient with a brain tumor includes T1/T2-weighted, fluid-attenuated inversion recovery (FLAIR), and postcontrast T1-weighted images (**Figure 4**) [30]. T1-weighted images are most beneficial for portraying anatomic information and show cerebrospinal fluid and most tumors as low signal strength, whereas areas of fat and subacute hemorrhage appear as high signal strength [31].

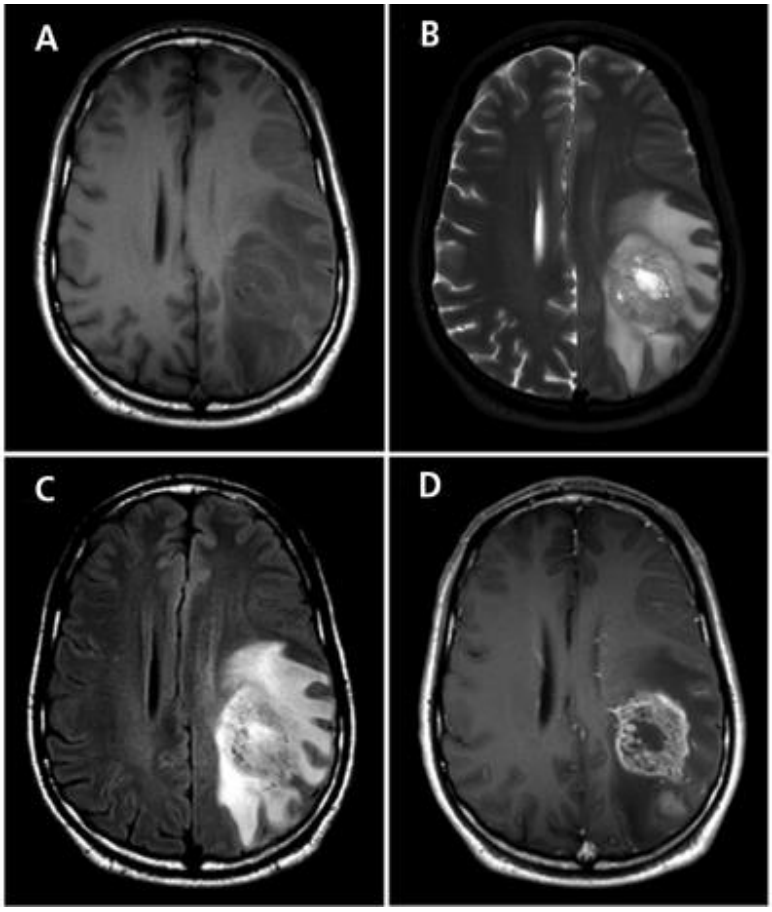


Figure 4: MRI images show a large mass in a patient presenting with hemiparesis on the right side. (A) T1-weighted image shows a hypointensity lesion in the left frontal-parietal region. (B, C) T2-weighted and fluid-attenuated inversion recovery images show a heterogeneous hyperintensity lesion with surrounding edema. (D) T1-weighted image with contrast shows a heterogeneous ring enhancing lesion, a second area of enhancement posterior to the major lesion, and vasogenic edema. Pathology was consistent with glioblastoma. [31]

Structural MRI sequences play a significant role in the assessment of and treatment planning of brain tumors. Basic series carried out using spin-echo methods consist of T2 fluid-attenuated inversion healing (FLAIR), pre-gadolinium T1, and post-gadolinium T1. These sequences are preferably performed in a minimum of 2-orthogonal planes or acquired with a 3-dimensional (3D) series that is reformatted into orthogonal aircrafts (i.e., 3D-T2 FLAIR). High-resolution iso-volumetric sequences such as high-resolution 3D T2 sequences and post-gadolinium T1 spoiled gradient recalled acquisition (SPGR) or similar sequences are usually carried out preoperatively with fiducials in place for use with intraoperative navigational software application [32,33]. Likewise, post-gadolinium T1 SPGR sequences are carried out with a stereotactic head frame in place prior to stereotactic radiosurgery [34,35]. High-resolution 3D T2 * gradient echo series such as vulnerability weighted imaging (SWI) are also routinely performed. These susceptibility sensitive sequences are extremely conscious blood products and calcification and might be practical to depict post-radiotherapy micro-hemorrhages [36,37,38].

The primary functions of structural MRI in preliminary brain tumor assessment consists of figuring out the place of the sore (i.e., extra-axial vs. intra-axial), developing the specific location within the brain for treatment/biopsy planning, evaluating mass effect on the brain, ventricular system, and vasculature, and together with physiologic MRI series recommending a possible diagnosis. Extra-axial tumors such as meningiomas, schwannomas, and skull base tumors can normally however not always be separated from intra-axial tumors. The differential diagnosis for intra-axial tumors depends upon patient age and the presence of another primary malignancy [39,40].

- ***MRI versus CT in primary brain tumors evaluation:***

The favored modality for detecting brain lesions, whether they originate from main brain cancer such as glioblastoma or whether they are metastatic sores originating from other primary sites, is MRI with contrast administration [41,42].

Regardless of the accuracy of MRI, CT imaging is often used when MRI is either not ideal or not available for the private case [43,44,45]. The argument has been made that the schedule, performance, and cost-effectiveness of CT imaging make it the technique of option in detecting brain lesions [45]. Pearl et al. suggested that non-contrast CT must be the preliminary examination to find any hemorrhage or calcification, followed by a double-contrast CT to identify lesions [45]. Nevertheless, MRI has the advantage of producing multi-planar images with greater contrast [44]. Images produced by T2 weighted MRI are likewise more conscious smaller sores [43]. Srikanth et al. reported a correlation between the morphologic and histologic functions of tumors shown on both MRI and CT imaging [44].

Despite the advanced level of sensitivity of MRI, 11% of patients with a brain lesion are given a false-positive diagnosis, based exclusively on MRI, of either primary or metastatic cancer [46,47]. More recently, techniques such as perfusion MRI have been used to separate primary gliomas and brain metastasis. Frequently, medical diagnosis of brain sores need to be confirmed by pathology and histology examination, utilizing excisional biopsy of the tumors [41].

The appearance of lesions on MRI or CT imaging will not constantly cause a specific medical diagnosis. After examination of multiple images, diagnosis depends upon many other factors. If the sore is indicative of cancer, radiologists and oncologists must determine whether the lesion is a primary brain tumors, such as glioblastoma, or metastasis from a non-localized site somewhere else in the body. Approximately 80% of brain transition cases are identified in patients who currently have a recognized primary site of cancer. This metachronous discussion makes the differential medical diagnosis less challenging. In cases of concurrent presentation, patients are identified with a primary cancer around the very same time that their brain transition is discovered. Before a main site is found, it can be very unclear whether brain lesions are metastatic or not. For up to 15% of patients detected with brain metastasis, the primary cancer site will remain unknown regardless of thorough investigation [43]. When brain metastasis is believed [48], positron-emission tomography imaging is a good tool for spotting an unknown main.

MRI, have actually made the differential diagnosis in between malignant and non-cancerous sores a lot easier. Prior to making a diagnosis of cancer, radiologists look for several functions distinct to lesions. However, the appearance of primary brain tumors and of brain metastases is frequently rather comparable, making it difficult to compare them, specifically when considering just a single image. On MRI slices, metastatic lesions typically appear as little, well-defined, ring-enhancing sores surrounded by edema [43,45]. They might also show central necrosis or hemorrhage, or both [45]. Gliomas are likewise discovered to have actually main necrosis surrounded by a ring of contrast enhancement; they likewise typically present with edema having mass result [41]. A recent research study comparing the look of brain lesions, including those triggered by non-cancerous disease, discovered that 40% of ring-enhancing lesions on MRI slices were triggered by gliomas which 30% were connected with brain transition [49]. Lesions caused by metastasis and gliomas were likewise found to have comparable rates of hypo-intense verge on T2 weighted MRI and to have comparable rates of heterogeneous centres or central necrosis [49].

4. CONCLUSION

CT and MRI stay the main techniques utilized for the detection of primary brain tumors of the CNS. CT is very helpful in the setting of brand-new neurological indications or signs, with or without a history of malignancy. MRI is extremely delicate for the detection of brain metastases, but currently both MRI and CT are accepted methods of evaluating for brain metastases. But still MRI is a versatile and effective instrument for evaluating patients with main brain tumors. The integration of the MRI into treatment assessment helps in reducing early termination of reliable therapies because of treatment-associated imaging modifications. Advanced aspects of MRI, including spectroscopy, perfusion, and diffusion, provide vibrant details related to brain tumor reaction and development.

REFERENCES

[1] Kleihues P, Soylemezoglu F, Schauble B, Scheithauer BW, Burger PC. Histopathology, classification, and grading of gliomas. Glia 15: 211–221, 1995.

[2] Louis DN, Holland EC, Cairncross JG. Glioma classification: a molecular reappraisal. Am J Pathol 159: 779–786, 2001.

[3] Wen PY, Macdonald DR, Reardon DA, et al. Updated response assessment criteria for high-grade gliomas: response assessment in neuro-oncology working group. J Clin Oncol. 2010;28:1963–1972.

[4] Macdonald DR, Cascino TL, Schold SC, Jr, Cairncross JG. Response criteria for phase II studies of supratentorial malignant glioma. J Clin Oncol. 1990;8:1277–1280.

[5] van den Bent MJ, Wefel JS, Schiff D, et al. Response assessment in neurooncology (a report of the RANO group): assessment of outcome in trials of diffuse low-grade gliomas. Lancet Oncol. 2011;12:583–593.

[6] Vogelbaum MA, Jost S, Aghi MK, et al. Application of novel response/progression measures for surgically delivered therapies for gliomas: Response Assessment in Neuro-Oncology (RANO) Working Group. Neurosurgery. 2012;70:234–243. discussion 243-234.

[7] Ricci PE. Imaging of adult brain tumors. Neuroimaging Clin N Am. 1999;9:651–669.

[8] Hutter A, Schwetye KE, Bierhals AJ, McKinstry RC. Brain neoplasms: epidemiology, diagnosis, and prospects for cost-effective imaging. Neuroimaging Clin N Am. 2003;13:237–250.

[9] Lee YY, Van Tassel P. Intracranial oligodendrogliomas: imaging findings in 35 untreated cases. AJR Am J Roentgenol. 1989;152:361–369.

[10] Lee YY, Bruner JM, Van Tassel P, Libshitz HI. Primary central nervous system lymphoma: CT and pathologic correlation. AJR Am J Roentgenol. 1986;147:747–752.

[11] Grossman ZD. Cost-effective diagnostic imaging: The clinician's guide. 4th ed. Philadelphia, Pa: Mosby; 2006.

[12] Potts DG, Abbott GF, von Sneidern JV. National Cancer Institute study evaluation of computed tomography in the diagnosis of intracranial neoplasms. III. Metastatic tumors. Radiology. 1980;136:657–64.

[13] Fink KR, Fink JR. Imaging of brain metastases. Surgical Neurology International. 2013;4(Suppl 4):S209-S219. doi:10.4103/2152-7806.111298.

[14] Ginaldi S, Wallace S, Shalen P, Luna M, Handel S. Cranial computed tomography of malignant melanoma. AJR Am J Roentgenol. 1981;136:145–9.

[15] McGann GM, Platts A. Computed tomography of cranial metastatic malignant melanoma: Features, early detection and unusual cases. Br J Radiol. 1991;64:310–3.

[16] Nakase H, Sakaki T, Fujita T, Tsunoda S, Nakamura M, Imai T, et al. Multiple calcified metastatic brain tumor--case report. Neurol Med Chir (Tokyo) 1991;31:787–91.

[17] Blatt DR, Friedman WA, Agee OF. Delayed computed tomography contrast enhancement patterns in biopsy proven cases. Neurosurgery. 1993;32:560–9.

[18] Shalen PR, Hayman LA, Wallace S, Handel SF. Protocol for delayed contrast enhancement in computed tomography of cerebral neoplasia. Radiology. 1981;139:397–402.

[19] Sidhu KP, Cooper P, Ramani R, Schwartz M, Franssen E, Davey P. Delineation of brain metastases on CT images for planning radiosurgery: Concerns regarding accuracy. Br J Radiol. 2004;77:39–42.

[20] Sze G, Shin J, Krol G, Johnson C, Liu D, Deck MD. Intraparenchymal brain metastases: MR imaging versus contrast-enhanced CT. Radiology. 1988;168:187–94.

[21] Davis PC, Hudgins PA, Peterman SB, Hoffman JC. Diagnosis of cerebral metastases: Double-dose delayed CT vs contrast-enhanced MR imaging. AJNR Am J Neuroradiol. 1991;12:293–300.

[22] Krüger S, Mottaghy FM, Buck AK, Maschke S, Kley H, Frechen D, et al. Brain metastasis in lung cancer.Comparison of cerebral MRI and 18F-FDG-PET/CT for diagnosis in the initial staging. Nuklearmedizin. 2011;50:101–6.

[23] Schellinger PD, Meinck HM, Thron A. Diagnostic accuracy of MRI compared to CCT in patients with brain metastases. J Neurooncol. 1999;44:275–81.

[24] Akeson P, Larsson EM, Kristoffersen DT, Jonsson E, Holtås S. Brain metastases-comparison of gadodiamide injection-enhanced MR imaging at standard and high dose, contrast-enhanced CT and non-contrast-enhanced MR imaging. Acta Radiol. 1995;36:300–6.

[25] Silvestri GA, Gould MK, Margolis ML, Tanoue LT, McCrory D, Toloza E, et al. Noninvasive staging of non-small cell lung cancer: ACCP evidenced-based clinical practice guidelines (2nd edition) Chest. 2007;132(3 Suppl):178S–201.

[26] Yokoi K, Kamiya N, Matsuguma H, Machida S, Hirose T, Mori K, et al. Detection of brain metastasis in potentially operable non-small cell lung cancer: A comparison of CT and MRI. Chest. 1999;115:714–9.

[27] Soffietti R, Cornu P, Delattre JY, Grant R, Graus F, Grisold W, et al. EFNS Guidelines on diagnosis and treatment of brain metastases: Report of an EFNS Task Force. Eur J Neurol. 2006;13:674–81.

[28] Chiarion-Sileni V, Murr R, Pigozzo J. Brain metastases from malignant melanoma. *Oncology*. 2001;13:170–185.

[29] Kremer S, Grand S, Berger F, et al. Dynamic contrast-enhanced MRI: differentiating melanoma and renal carcinoma metastases from high-grade astrocytomas and other metastases. *Neuroradiology*. 2003;45(1):44–49.

[30] Leung D, Han X, Mikkelsen T, Nabors LB. Role of MRI in primary brain tumor evaluation. J Natl Compr Canc Netw. 2014 Nov;12(11):1561-8.

[31] Woodruff WW, Jr., Djang WT, McLendon RE, Heinz ER, Voorhees DR. Intracerebral malignant melanoma: high-field-strength MR imaging. Radiology. 1987;165(1):209–213.

[32] Gumprecht HK, Widenka DC, Lumenta CB. BrainLab VectorVision Neuronavigation System: technology and clinical experiences in 131 cases. Neurosurgery. 1999;44:97–104. discussion 104-5.

[33] Elhawary H, Liu H, Patel P, et al. Intraoperative real-time querying of white matter tracts during frameless stereotactic neuronavigation. Neurosurgery. 2011;68:506–516. discussion 516.

[34] Zhang B, MacFadden D, Damyanovich AZ, et al. Development of a geometrically accurate imaging protocol at 3 Tesla MRI for stereotactic radiosurgery treatment planning. Phys Med Biol. 2010;55:6601–6615.

[35] Saconn PA, Shaw EG, Chan MD, et al. Use of 3.0-T MRI for stereotactic radiosurgery planning for treatment of brain metastases: a single-institution retrospective review. Int J Radiat Oncol Biol Phys. 2010;78:1142–1146.

[36] Haacke EM. Susceptibility weighted imaging (SWI) Z Med Phys. 2006;16:237.

[37] Haacke EM, Mittal S, Wu Z, Neelavalli J, Cheng YC. Susceptibility-weighted imaging: technical aspects and clinical applications, part 1. AJNR Am J Neuroradiol. 2009;30:19–30.

[38] Bian W, Hess CP, Chang SM, Nelson SJ, Lupo JM. Susceptibility-weighted MR imaging of radiation therapy-induced cerebral microbleeds in patients with glioma: a comparison between 3T and 7T. Neuroradiology. 2014;56:91–96.

[39] Borja MJ, Plaza MJ, Altman N, Saigal G. Conventional and advanced MRI features of pediatric intracranial tumors: supratentorial tumors. AJR Am J Roentgenol. 2013;200:W483–W503.

[40] Sze G, Milano E, Johnson C, Heier L. Detection of brain metastases: comparison of contrast-enhanced MR with unenhanced MR and enhanced CT. AJNR Am J Neuroradiol. 1990;11:785–791.

[41] Behin A, Hoang–Xuan K, Carpentier AF, Delattre JY. Primary brain tumours in adults. Lancet. 2003;361:323–31.

[42] Subramanian A, Harris A, Piggott K, Shieff C, Bradford R. Metastasis to and from the central nervous system the "relatively protected site. Lancet Oncol. 2002;3:498–507.

[43] Tosoni A, Ermani M, Brandes AA. The pathogenesis and treatment of brain metastases: a comprehensive review. Crit Rev Oncol Hematol. 2004;52:199–215.

[44] Srikanth SG, Jayakumar PN, Chandrashekar HS. ct features of intracranial metastases of unknown primaries. Neurol India. 2002;50:282–5.

[45] Pearl ML, Talgat G, Valea FA, Chalas E. Psychiatric symptoms due to brain metastases. Med Update Psychiatr. 1998;3:91–4.

[46] Posner JB. Management of brain metastases. Rev Neurol. 1992;148:477–87.

[47] Patchell RA, Tibbs PA, Walsh JW, et al. A randomized trial of surgery in the treatment of single metastases to the brain. N Engl J Med. 1990;322:494–500.

[48] Jeong HJ, Chung JK, Kim YK, et al. Usefulness of whole-body 18F-fdg pet in patients with suspected metastatic brain tumors. J Nucl Med. 2002;43:1432–7.

[49] Schwartz KM, Erickson BJ, Lucchinetti C. Pattern of T2 hypointensity associated with ring-enhancing brain lesions can help to differentiate pathology. Neuroradiology. 2006;48:143–9.