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Case Report

Basal Ganglia Infarction Mimicking Glioblastoma

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Abstract: Modern brain imaging techniques usually allow a very good differential diagnosis of intracerebral lesions, but in some cases the differential diagnosis is difficult. We report the case of a 52 year old male with acute brachiofacial paresis and a hyperintense lesion with mass effect and ring-enhancement in basal ganglia suspiciously to a tumor. The neurosurgeons recommend stereotactical brain biopsy for diagnosis, but the patient recovered in following time gradually and in repeated computertomographic images contrast enhancement disappeared and a hypodense zone in the basal ganglia remains. Our case demonstrates that brain infarctions can mimick glioblastoma in taking cystic appearance and contrast enhancement. Stereotactic biopsy would have been a precipitated invasive procedure in this case.

Key words: basal ganglia infarction, brain infarction, differential diagnosis brain tumor.

Modern brain imaging techniques usually allow a very good differential diagnosis of intracerebral lesions. Even though in some cases it remains difficult to differentiate between malignant brain tumor, abscess and

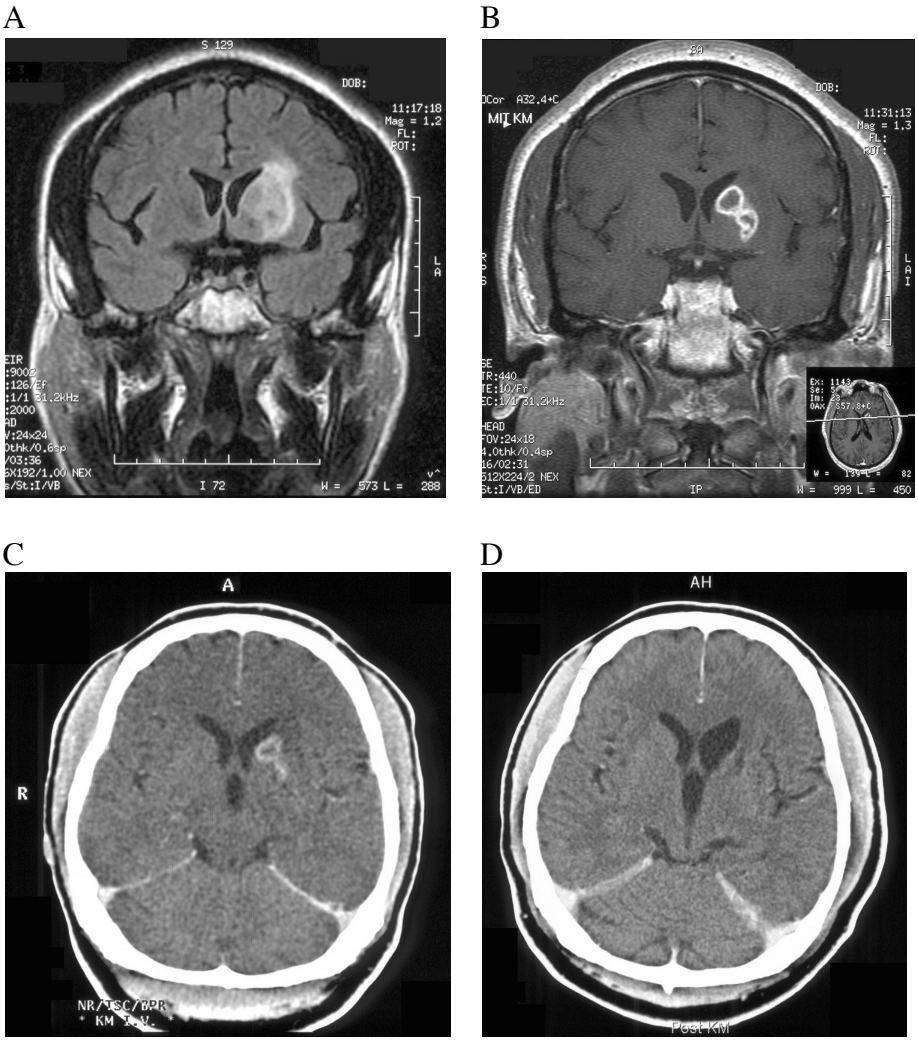

Fig. 1A-B. Initial MR T1-weighted image without (A) and with contrast injection (B). C: CT-scan with contrast injection 8 days later D: CT-scan with contrast injection 4 months later

subacute ischemic infarction. Thus, clinicians should be wary of a possible misdiagnosis.

Here, we report the case of a 52 year old male from Ghana with brachiofacial paresis on the right side with acute symptom onset 6 weeks before first presentation in our department. The patient`s history disclosed a mild non-insulin dependent diabetes mellitus type II since 2 years. Other vascular risc factors (hypertonus, hypercholesterinaemia, peripheral artery occlusion disease, coronary heart disease, nicotine abuse) were absent. Magnetic resonance imaging revealed a hyperintense lesion with mass effect resulting in compression of the left ventricle on fluid-attenuated inversion recovery (FLAIR) images (Fig. 1-A). T1-weighted images (T1WI) demonstrated ring-enhancement within the lesion that appeared isointense within its core (1-B). Cerebrospinal fluid, routine blood parameters including antinuclear antibodies, anti-cytoplasmatic antibodies, antiphospholipid-antibodies, protein C, protein S and APC-resistance were normal. HIV-antibodies were absent. Doppler and duplex sonography studies excluded abnormalities of extra- and intracranial arteries. Cranial computed tomography (CCT) eight days later confirmed the preceding findings (Fig 1-C). Therefore, a malignant brain tumor was suspected and a stereotactic biopsy for staging was planned. However, patients symptoms improved slowly without any specific therapy within the next few days. The patient was discharged and control imaging was scheduled. Four months later, the patient presented with minor brachiofacial weakness. Repeated CCT revealed a hypodense area in the left basal ganglia with dilatation of the left ventricle corresponding to a basal ganglia infarction (Fig 1-D). 20 months later the patient had recovered fully.

Discussion

Several case reports have demonstrated that brain tumors may mimick cerebral infarction, abscess and arteriovenous malformation [1-4]. Conversely acute ischemic infarction is primarily diagnosed as malignant brain tumor less often. In one case of suspected brain tumor reported by Koh et al. the contrast enhancement was due to vasculitis induced by a Sjoegren-syndrome [5]. The most striking imaging-related difference of both entities is the regressive character of the infarction but brain tumors can behave regressive, too. In the most cases one find a primary central nervous system lymphoma (PCNSL). Bromberg et al. found in a case series of 12 patients that 50% of regressive intracerebral tumor-like lesions were caused by PCNSL. Furthermore, lesions due to multiple sclerosis or acute disseminated encephalomyelitis may often dissolve spontaneously [6]. In the previously reported case the suspicion of a brain tumor was raised due to a contrast enhanced solid lesion. Our case demonstrates that brain infarctions can mimick a glioblastoma in taking a cystic appearance and having already in the subacute phase contrast enhancement that appears usually 5 to 10 days after infarction during the period of blood-brain-barrier disruption [7-9]. In our case a stereotactic biopsy would have been a premature invasive procedure.

References

1. Henrikson GC, Patel DV (1985) Enhancing cerebral infarction simulating arteriovenous malformation on computed tomography. J Comput Assist Tomogr 9:502-6.
2. Harada S, Niimi M, Murakami K, Nakamura T (2001) Cavernous angioma of the corpus callosum mimicking an astrocytic tumor--case report. Neurol Med Chir (Tokyo) 41:349-51.
3. Sabel M, Neuen-Jacob E, Vogt C, Weber F (2001) Intracerebral neurocysticercosis mimicking glioblastoma multiforme: a rare differential diagnosis in Central Europe. Neuroradiology 43:227-30.
4. Jellema K, van der Meulen MF, Witkamp TD, Taphoorn MJ (2001) Brain tumor or stroke? Ned Tijdschr Geneeskd 145:849-53.
5. Koh MS, Goh KY, Chen C, Howe HS (2002) Cerebral infarct mimicking glioma in Sjogren's syndrome. Hong Kong Med Journal 8:292-4.
6. Bromberg JEC, Siemers MD, Taphoorn MJB (2002) Is a "vanishing tumor" always a lymphoma ? Neurology 59:762-764.
7. Masdeu JC (1983) Enhancing mass on CT: neoplasm or recent infarction? Neurology 33:836-40.
8. Ito U, Tomita H, Kito K, Ueki Y, Inaba Y (1986) CT enhancement after prolonged high-dose contrast infusion in the early stage of cerebral infarction. Stroke 17:424-30.
9. Merten CL, Knitelius HO, Assheuer J, Bergmann-Kurz B, Hedde JP, Bewermeyer H (1999) MRI of acute cerebral infarcts, increased contrast enhancement with continuous infusion of gadolinium. Neuroradiology 41:242-8.

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