

MAGNETIC RESONANCE IMAGING OF CNS PARASITIC INFECTIONS

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Introduction

In the past, parasitic infections were relatively rare in industrialized nations, although endemic in underdeveloped countries. With the increase of mobility and the pandemic spread of AIDS, this situation changed radically.

The major protozoal infections that can involve the CNS, presents similar symptoms with focal neurological signs, headache and in many cases epilepsy. Also the CNS and its covering show relatively nonspecific patterns of response to physiochemical irritants and microbiologic agents.

In the CNS the neuroectodermal structures (nerve cells and glia), and the mesodermal structures (vessels and connective tissue) are closely interwoven. Inflammatory disease elicit a predominantly mesenchymal response of the vascular connective tissue, which is most pronounced around the capillaries. The absorptive granulation of the abscess capsule originates from the mesenchymal tissue and not from the glia. The inflammatory changes in the brain initiates profound physiochemical changes which can affect the MR-signal of the protons 1.

The high soft tissue contrast of MR-Imaging especially T2 weighted images makes this method superior to CT in detection and extent of inflammatory brain lesions. In addition using Gd-DTPA disruption of the blood-brain barrier can be proved. The purpose of the present study was to demonstrate the diagnostic value of MR-imaging in the detection and characterization of parasitic CNS infections.

Methods

MR-Imaging was performed on a 1.5 Tesla system (Magnetom, Siemens, Erlangen, Germany) using a circular polarized head coil (FOV=25cm).

The 4mm sagittal T1-weighted SE (TR=520msec; TE=15msec; AC=2) and 5mm coronal and axial PD/T2-weighted SE (TR=2400msec; TE=15/90; Ac=1) sequences were used. In addition, 4mm T1-weighted coronal and/or axial SE images were obtained before and after intravenous administration of 0.1 mmol/kg Gd-DTPA (Schering, Berlin, Germany). Intensive care patients were monitored during the MR-examination by electrocardiography, continuous

noninvasive blood pressure, transcutaneous pulsoximetry and capnography.

Patients

The cases and data presented were taken from 115 examinations of 80 patients with presumed or proven inflammatory brain disease. In 70 of this cases both unenhanced and contrast-enhanced MR studies were carried out.

All diagnosis were proven by laboratory test or biopsy.

Neurocysticercosis

Cysticercosis occurs after ingestion of the ova of the tapeworm *Tenia solium*. The larve penetrate the wall of the intestine and were dispersed by the circulation. In the cranial cavity the disease can be subdivide in a parenchymal form, where the lesions are in the brain tissue, a meningeal and a ventricular form. These different forms can be present in any combination 2.

The parenchymal lesions detected by MR can be diffus low density with little or no enhancement, focal with rounded, central enhancement, or cystic lesions with enhancement of the borders. In some cases the scolex with a nodular structure and a particularly intense border can be seen. There is a tendency towards puntiform calcification which is indirectly visible by MR but more so with CT 3.

A frequent complication of neurocysticercosis is usually caused by inflammatory occlusion of the arteries at the base of the brain secondary to cysticercotic arachnoiditis. In most cases, the involved vessels are of smale diameter and the neurological picture is limited to a lacunar syndrom secondary to a small cerebral infarct.

Case report 1:

Indian citizen, 27 years old, waiter.

Came to our attention after repeated episodes of a sensitivity deficit of the right hemisoma and minor walking disorders.

MRT shows contrast-enhanced lesions at the left semioval paramedian center, and in the frontal, parietal and insular subarachnoid space (Fig. 1).

Cerebral toxoplasmosis

Cerebral toxoplasmosis is one of the most frequent opportunistic infections in patients with the cellular defects mediated by HIV. One of the most important factors for successful treatment is early diagnosis.

The detection of multiple contrast-enhancing nodular lesions on MR-imaging favors but does not make the diagnosis of toxoplasmosis.

Next laboratory tests many clinician opt for an empiric trial of pyramethamine/sulfadiazine. In patients who faild to respond a brain biopsy is often indicated 6.

Case report 2:

Austrian citizen, 32 years old. HIV positive. Come to our notice after tonic-clonic generalized episode.

Neurologic symptoms: Diffus organic brain syndrome with mild right side hemiparesis. MR-Imaging shows a lesion on the left precentral region with large edema (Fig 2).

Case report 3:

Ghanian citizen, HIV positive. Came to our notice after repeated episodes of frontal-nucal cephalgia without neurological signal. The anamnesis refer to an episode of GM.

MRT: 5mm intraparenchymal lesion with a weak signal in T2 in correspondence with the precentral right gyrus which does not modify in T1. In T1 right frontal lesion 20x15mm and a small lesion on the splenium with high signal intensity. This finding is due to a haemorrhagic lesion containing methaemoglobin (Fig 3a,b).

Case report 4:

Austrian citizen, 36 years old, unemployed, HIV positive. No focal neurological signals.

MRT: an inhomogenous oval modification in corrispondence with the left basal ganglia of ca. 45x30x30mm with mass effect on the left anterior horn. In T2 there is a slight hyperintense effect.

Cerebral malaria

Cerebral malaria is the most common complication in severe Plasmodium falciparum malaria with a mortality rate of 20-50. Its exact pathogenesis is still unknow. However, it is generally accepted that blockage of cerebral capillaries by parasite-infected erythrocytes, in combination with marked glucose use and lactate production by the parasites, leads to diffuse tissue hypoxia and organ dysfunction. An other neurophatological aspect of malaria is characterized by vasculopathy with an increase in permeability of the endotelial cells with edema and eventual perivascular demyelination. Neurologic abnormalities observed in cerebral malaria frequently include disturbances of consciousness, acute organic brain syndrome and epileptic seizures. Focal neurologic signs are unusual in the acute phase of cerebral malaria. Recently vascular complications related to cerebral malaria were detected by MR-imaging and reported by Millan et al.

Case report 5:

Dutch citizen, 27 years old, airline steward. Three weeks after a return temp from Gambia, was observed in coma with spontaneous vertical nystagmus. CT and CSF normal. From a blood sample we observed that 60% of erythrocytes were with trophozoits.

On MR-examination we observed at level of the pons a triangular shaped

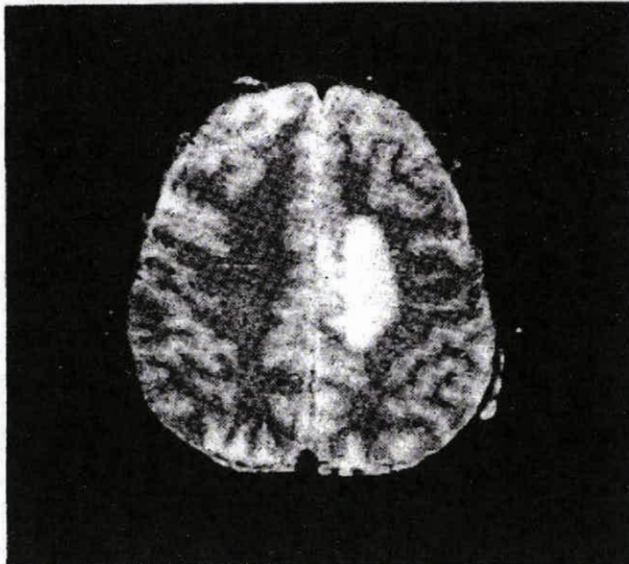
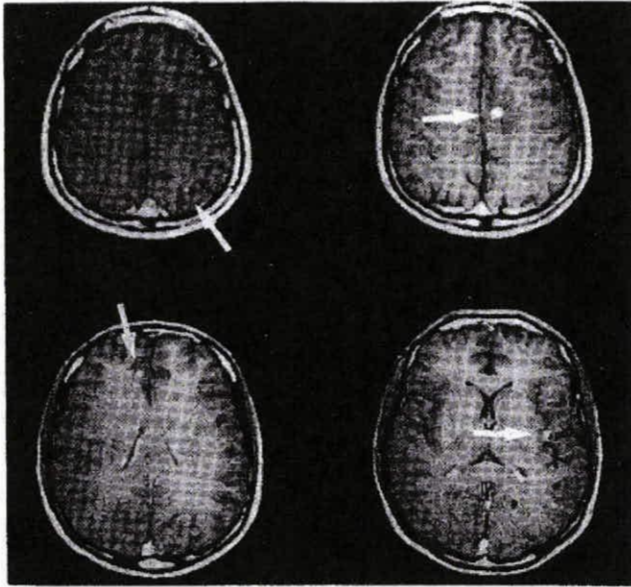


Fig 1: Axial T1-weighted images after application of Gd-DTPA. Shows multiple ring-enhancing lesions at gray-white junction (Arrows).

Fig 2: Axial T2-weighted image shows a large lesion with high signal intensity on the left precentral region.

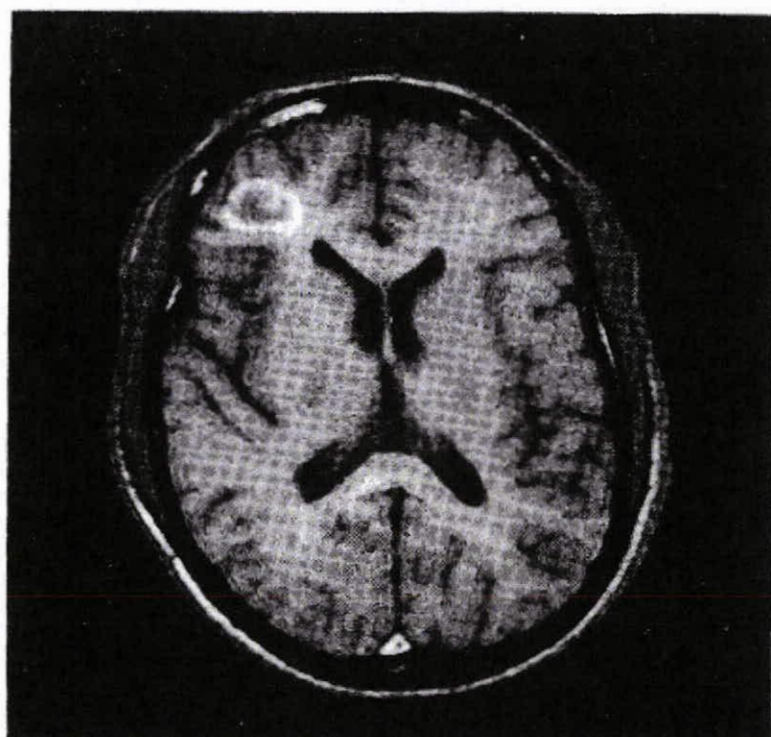


Fig 3a,b: Axial T1-weighted images after pre-and post application of contrast material shows a unchanged hemmorrhagic lesion on the right frontal lobe and a lesion on the splenium corpori callosi more pronounced after contrast application (Arrow).

References

- HUR W.J., LOTZ J.W., HEWLET R.H.: *Inflammatory diseases of the central nervous system. Magnetic Resonance Imaging of central Nervous Diseases*, Huk W.J., Gademann J., Friedmann G. Springer Verlag Berlin, Heidelberg 1990.
- MONTEIRO L., ALMEIDA PINTO J., STOCKER A., SAMPAIO SILVA M.: *Active neurocysticercosis, parenchymal and extraparenchymal: a study of 38 patients*, J. Neurol. 241, 15-21, 1993.
- CREASY J.L., ALARCON J.J.: *Magnetic resonance imaging of neurocysticercosis*, Top. Magn. Reson. Imaging 6, 59-68, 1994.
- ALARCON F., HIDALGO F., MONCAJO J., VINAN I., DUENAS G.: *Cerebral cysticercosis and stroke*, Stroke, 23, 224-8, 1992.
- DEL BRUTTO O.H.: *Cysticercosis and cerebrovascular disease: a review*, J. Neurol. Neurosurg. Psychiatry, 55, 252-4, 1992.
- ROBERT T. SCHOOLERY: *Encephalitis in neurological intensive care*, 3rd edition. Allan H. Ropper. Chapt 22, 411-435, 1993 Sarwar M., Falkoff G., Naseen M.: *Radiological techniques in the diagnosis of CNS infection*. Neurological Clinics, 44, 41-68, 1986.
- AIKAWA M., ISEKI M., BARNWELL J.W., TAYLOR D., OO M.M., HOWARD R.J.: *The pathology of human cerebral malaria*, Am. J. Trop. Med. Hyg. 43, 30-37, 1990.
- PHILIP P., NASNTEL S., BENNY W.B.: *Exchange transfusions as an adjunct to the treatment of severe falciparum malaria, Case report and review*. Rev. Infect. Dis. 12, 1100-1108, 1990.
- WARREL D.A.: *Cerebral malaria*, Schweiz. Med. Wochenschr., 122, 879-86, 1992.
- KAMPFL A.W., BIRBAMER G.G., PFAUSLER B.E., HARING H.H., SCHMUTZ-HARD E.: *Isolated pontine lesion in algida cerebral malaria: clinical features, management, and magnetic resonance imaging findings*, Am. J. Trop. Med. Hyg. 48, 818-822, 1993.
- MILLAN J.M., SAN MILLAN J.M., MUNOZ M., NAVAS E., LOPE-VELEZ R.: *CNS complication in acute malaria: MRI findings*, Am. J. Neuroradiol., 14, 493-4, 1993.
- MILLER L.H., GODD M.F., MILON G.: *Malaria pathogenesis*, Science 264, 1878-83, 1994.
- SHUPER A., LEVITSKY H.I., CORNBLATH D.R.: *Early invasive CNS aspergillosis*, Neuroradiology, 33, 183-185, 1991.
- COX J., MURTAGH F.R. WILFONG A., BRENNER J.: *Cerebral aspergillosis: MR imaging and histopathologic correlation*, A.J.N.R. 13, 1489-1492, 1992.
- ASHDOWN B C., TIEN R.D., FELSBERG G.J.: *Aspergillosis of the brain and paranasal sinuses in immunocompromised patients: CT and MRI imaging findings*, A.J.N.R. 162, 155-159, 1994.