Amide proton transfer (APT) imaging of brain infection in children

hong zhang^{1,2}, Xuna Zhao³, Jinyuan Zhou², and Yun Peng⁴

¹Imaging Center, Beijing Children's Hospital, Capital Medical University, Beijing, Beijing, China, ²Neurosection, Division of MR Research, Department of Radiology, Johns Hopkins University, Baltimore, MD, United States, ³Peking University, Beijing, China, ⁴Beijing Children's Hospital, Capital Medical University, Beijing, Beijing, China

TARGET AUDIENCE: Neurologists and radiologists who pay attention to diagnosis of brain infection. **PURPOSE**

APT imaging is a molecular MRI technique that provides contrast based on endogenous mobile proteins and peptides, such as those in the cytoplasm. Previous studies¹ on human brain tumors have indicated that APT imaging characterizes high-grade gliomas as hyperintense lesions, since such tumors over-express proteins and peptides, compared to the normal brain. Brain infection may be caused by infectious agents including bacteria, viruses, and granulomatous agents. The study was performed with the aim of characterizing infectious lesions of different aetiology using protein-based APT imaging.

METHODS

Children with brain infection [one with tuberculous abscess (TA), one with pyogenic abscess (PA); and three with viral encephalitis (VA)] that were diagnosed on the basis of laboratory, clinical, and radiologic findings were recruited in this study. MRI data was acquired using a Philips 3T MRI scanner, including multiple MRI scans, T_1 -weighted, T_2 -weighted, isotropic apparent diffusion constant (ADC), Gd- T_1 w, and APT-weighted. Gd T_1 w was the last sequence acquired. APTw MR imaging was based on the single-slice, single-shot TSE (saturation time = 800 ms; saturation power = 2 μ T). The APT effect was quantified using an MT-ratio asymmetry

analysis at the offset of 3.5 ppm: $MTR_{asym}(3.5ppm)$, and displayed using a window of -4% to 4%. The $Gd-T_1w$ image was used as the reference of ROI analysis.

RESULTS AND DISCUSSION

Fig. 1 shows examples of the APTw and standard MR images for patients with TA, PA, and VE. Both TA and PA demonstrated clear gadolinium enhancement. The APTw signal was high in the gadolinium-enhancing rim of the lesion, compared to peripheral edema and contralateral normal-appearing brain tissue. This hyperintense rim on APTw MRI may be due to the inflammatory cellular infiltrate and granulomas², leading increased content of cellular proteins and peptides¹. Most non-enhancing areas on T₁w may be liquifactive necrosis of the lesion, showing APTw iso-intensity. The portion inside the center of the lesion showing an APTw hyperintensity may be due to a large amount of neutrophils and proteins, which are released in the necrotic cavity³. For VE, T₂w showed a symmetric hyperintense lesion in the basal ganglia. The lesion shows no enhancement on Gd-T₁w and iso-intensity on APTw, mainly be associated which may vasogenic/interstitial collection of fluid⁴.

Table 1 summarizes the results for two patients with abscess. APT image intensity is higher in Gd-enhancing wall of the abscess than in non-Gd enhancing core, peripheral edema and contralateral normal-appearing brain tissue. Thus, APT-MR imaging may help better distinguish the heterogeneous portions of infectious lesions.

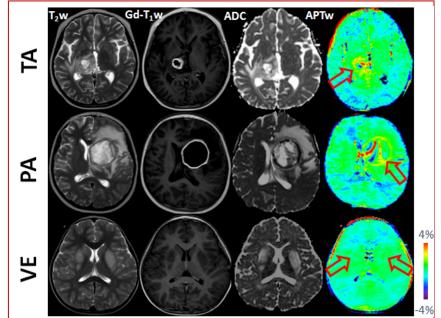


Fig. 1. Standard and APTw MRI features of brain infection.

Table 1. MTR_{asym}(3.5ppm) in several areas for two patients (mean \pm sd).

	CE	NE	Peripheral edema	CNAB
TA	2.30±0.07	1.27±0.06	0.58±0.07	0.37±0.03
PA	2.27±0.17	0.85±0.10	0.91±0.02	0.45±0.03

CE: Gd-contrast enhancing wall, NE: non-Gd enhancing core, CNAWN: contralateral normal appear-appearing brain tissue.

CONCLUSION

These initial data show that APT-MR imaging is an important technique for the detection and characterization of infectious lesions of different aetiology. APTw MRI is based on endogenous contrast agents, and thus, no contrast agent injection is required. This is particularly significant for pediatric cases, where intravenous access is often problematic.

REFERENCES

(1) Zhou J et al. JMRI 2013;38:1119-1128. (2) Rakesh K et al. Clinical Radiology 2001;56:656-663. (3) Rakesh K et al. AJNR 2001;22:1503-1509. (4) Yilmaz K. et al. Neuroradiology 2006;48:875-880.