ELSEVIER

Brain & Development xxx (2017) xxx–xxx

BRAIN & DEVELOPMENT
Official Journal of the Japanese Society of Child Neurology

www.elsevier.com/locate/braindev

Original article

Use of high *b* value diffusion-weighted magnetic resonance imaging in acute encephalopathy/encephalitis during childhood

Yoshiko Tsubouchi [a,1,*], Shinji Itamura [a,1], Yoshiaki Saito [a], Eijiro Yamashita [b], Yuki Shinohara [b], Tetsuya Okazaki [a], Koyo Ohno [a], Yoko Nishimura [a], Masayoshi Oguri [a], Yoshihiro Maegaki [a]

[a] *Division of Child Neurology, Department of Brain and Neurosciences, Faculty of Medicine, Tottori University, Yonago, Japan*
[b] *Division of Radiology, Department of Pathophysiological and Therapeutic Science, Faculty of Medicine, Yonago, Japan*

Received 20 February 2017; received in revised form 30 May 2017; accepted 21 July 2017

Abstract

Aim: To determine the use of high *b* value diffusion-weighted imaging (DWI) in the diagnosis and assessment of acute febrile encephalopathy/encephalitis in childhood.

Subjects and methods: We enrolled 22 children, for whom we examined DWI with $b = 1000$ s/mm^2, DWI with $b = 3000$ s/mm^2, and apparent diffusion coefficient (ADC) map with $b = 1000$ during the acute phase of febrile encephalopathy/encephalitis. Clinical diagnoses included acute encephalopathy with biphasic seizures and late reduced diffusion (AESD; $n = 6$), clinically mild encephalopathy/encephalitis with a reversible splenial lesion (MERS; $n = 6$), and herpes simplex virus encephalitis (HSE; $n = 3$), unclassified acute encephalopathy/acute encephalitis ($n = 2$); acute encephalitis with refractory, repetitive partial seizures (AERRPS; $n = 1$); other encephalopathy ($n = 1$); infarction ($n = 1$); head injury ($n = 1$); or mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes ($n = 1$). The diagnostic quality of brain lesions was compared between $b = 1000$ and $b = 3000$ DWI images by visual inspection. In addition, we attempted a quantitative assessment using apparent diffusion coefficient (ADC) value and an index of signal intensity (SI) ratio, defined as the mean SI at the affected lesion divided by the mean SI at the pons.

Results: High intensity lesions were either visible only on $b = 3000$ DWI (n = 5; 2 AESD, 1 MERS, 1 HSE, and 1 unclassifiable encephalopathy) or more effectively identified on $b = 3000$ DWI than on $b = 1000$ DWI (n = 17). The outcome of the former five subjects was favorable, without motor or intellectual sequelae. The mean SI ratio of $b = 3000$ was significantly greater than that of $b = 1000$ in AESD and MERS subgroups as well as in all 22 subjects. Mean ADC values were lower in the AESD and MERS than that in the HSE subgroups.

Conclusion: We concluded that $b = 3000$ DWI was superior to $b = 1000$ DWI in detecting abnormal lesions in acute encephalopathy/encephalitis during childhood.

Keywords: High *b* value; Diffusion-weighted imaging; Apparent diffusion coefficient; Acute encephalopathy; Childhood

1. Introduction

Diffusion-weighted imaging (DWI) visualizes the movement of water molecules and is superior to classical magnetic resonance imaging (MRI) for the visualization

* Corresponding author at: Department of Pediatrics, National Hospital Organization Yonago Medical Center, Kuzumo 4-17-1, Yonago 683-0006, Japan. Fax: +81 859 34 1580.
E-mail address: tsubouchi-yoshiko@yonagomc.jp (Y. Tsubouchi).
[1] These authors equally contributed to this article.

http://dx.doi.org/10.1016/j.braindev.2017.07.012
0387-7604/

Table 1
Findings on diffusion weighted images and clinical/neuroimaging outcome of each patient.

Patient	Age (months)	Sex	Diagnosis	Abnormal intensity lesion	Day of illness MRI examined	Findings on DWI	Follow-up months	Follow-up MRI	Sequela
1	20	F	AESD	lt. frontal subcortical white matter lesion	day 5	3000 only	15	no atrophy	none
2	13	F	AESD	rt. frontal subcortical white matter lesion	day 15	3000 only	9	no atrophy	none
3	48	M	AESD	lt. frontal and temporal subcortical white matter lesion	day 5	3000>1000	72	atrophy	rt. hemiparesis
4	23	F	AESD	bil. frontal and temporal subcortical white matter lesion	day 4	3000>1000	43	no atrophy	none
5	32	F	AESD	rt. frontal cortical and subcortical white matter lesions	day 5	3000>1000	4	no atrophy	none
6	16	M	AESD	bil. frontal subcortical white matter lesion	day 6	3000>1000	25	no atrophy	epilepsy, psychomotor retardation
7	53	F	MERS	splenium of corpus callosum	day 4	3000 only	1	no atrophy	none
8	115	M	MERS	splenium of corpus callosum	day 3	3000>1000	3	no atrophy	none
9	6	M	MERS	splenium of corpus callosum	day 2	3000>1000	17	no atrophy	none
10	23	F	MERS	rt. genu of corpus callosum	day 4	3000>1000	1	no atrophy	none
11	73	M	MERS	splenium of corpus callosum	day 3	3000>1000	12	no atrophy	none
12	36	M	MERS	splenium of corpus callosum, bil semioval center	day 1	3000>1000	1	no atrophy	none
13	43	F	unclassifiable acute encephalopthy	rt. frontal subcortical white matter lesion	day 3	3000 only	6	no atrophy	none
14	114	F	unclassifiable acute encephalopthy	bil. pulvinar	day 3	3000>1000	12	no atrophy	none
15	20 days	M	HSE	rt. frontal subcortical white matter lesion	day 4	3000 only	9	no atrophy	epilepsy
16	36	F	HSE	lt. lateral medial temporal cortex and subcortical white matter lesion	day 26	3000>1000	5	atrophy	none
17	25 days	M	HSE	bil. medial temporal lobe、insular cortex and orbitofrontal cortex	day 4	3000>1000	16	atrophy	speech delay
18	101	F	AERRPS	splenium of corpus callosum	day 3	3000>1000	26	atrophy	epilepsy, emotioal disorder
19	1	F	Other encephalitis	bil. periventricular lesion	day 2	3000>1000	33	no atrophy	none
20	5	M	Infarction	rt. caudate nucleus, putamen, thalamus and splenium of corpus callosum	day 2	3000>1000	7	atrophy	lt. hemiplasia, lr. facial palsy
21	15	M	Head injury	subcortical lesion of lt. hemicerebrum	day 5	3000>1000	37	atrophy	none
22	126	M	MELAS	lt. medulla oblongata	day 3	3000>1000	34	no atrophy	none

AESD: acute encephalopathy with biphasic seizures and late reduced diffusion, MERS: clinically mild encephalitis/encephalopathy with a reversible splenial lesion, AERRPS: acute encephalitis with refractory, repetitive partial seizures, MELAS: mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes, 3000: abnormalities are detected only on b = 3000 images, 3000 > 1000: b = 3000 images are superior to b = 1000 images.

of abnormal lesions, including brain infarct, tumor, and posterior reversible encephalopathy syndrome. This modality has also been applied to neurological disorders during childhood, which has facilitated the diagnosis and assessment of hypoxic–ischemic encephalopathy/acute febrile encephalopathies [1]. DWI has contributed to the establishment of acute encephalopathy with biphasic seizures and late reduced diffusion (AESD) and mild encephalopathy with reversible splenial lesion (MERS) by detecting white matter and callosal lesions, respectively [2,3]. Additionally, DWI has proven to be useful in the diagnosis and assessment of neonatal herpes simplex encephalitis [4]. Apparent diffusion coefficient (ADC) demonstrated the degree of restricted movement of water molecules. It is not subject to the T2 shine-through effect and represents the exact water diffusivity, which helps to identify whether high intensity on DWI represents a true restriction of diffusion. In addition, ADC values decreased and increased in intracellular and extracellular edema, respectively, and are useful in the determination of lesion pathophysiology.

Clinically, MRI scanners with a static magnetic field up to 1.5 T have been typically used to date. This has limited *b* values to 1000–1500 s/mm^2 in routine brain DWI [5]. With the recent advent of more powerful gradient hardware along with a magnetostatic intensity of 3 T, it is possible to generate greater *b* values and thereby obtain higher diffusion sensitivity [5]. The use

Please cite this article in press as: Tsubouchi Y et al. Use of high *b* value diffusion-weighted magnetic resonance imaging in acute encephalopathy/encephalitis during childhood. Brain Dev (2017), http://dx.doi.org/10.1016/j.braindev.2017.07.012