The Application of DWI and ADC Map in Cerebral Infarction

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Introduction

Rapid assessment of acute ischemia is critical for the management of acute stroke patiens who may benefit from thrombolytics or neuroprotective therapeutics. Differentiation of acute from chronic stroke, which may all appear hyperintense on T2- Weighted Imaging(T2WI), is essential in determining the management of patiens. This is essential because a single new ischemic event may treated differently from multiple new ischemic events, but differentiation between acute and chronic infarction cannot be easily determined from CT or T2WI. Diffusion-weighted imaging(DWI) has been shown to be useful in the early assessment of clinical stroke[1,2]. The apparent diffusion coefficient(ADC) of acutely ischemic brain tissue is initially below normal and increase to above normal after a few days. For time points between the acute and chronic stage, however, there are controversial studies regarding the exact time course of the ADC[3,4]. In this paper we studied the roles of isotropic DWI and ADC maps in diagnosing early cerebral infarctions, monitoring the development of cerebral infarctions and defining the time course of the rADC in infarct lesions.

Materials and Methods

86 work-ups of 70 patients with cerebral infarction (8 hyperacute, 13 acute, 32 subacute, 11 steady, 22 chronic) were imaged with both conventional MRI and single-shot echo-planar isotropic DWI(b=0,1000s/mm2;TR/TE/Fov/Matrix/Thickness/Space=10000ms/ minimum/36cm18cm/128128/7mm/2mm). All measurements were carried out using 1.5 T GE Signa EchoSpeed clinical MR scannar. EPI diffusion images were processed to generate isotropic ADC maps. 14 patients with early infarction had re-MRI and their infarct sizes as measure on second T2WI were compared with their initial sizes on DWI and the relationship of the infarct lesion with the clinical manifestation was analysed. The average ADC, mean rADC(rADC=ADC stroke/ADC contralateral100%) and the ADC from center to periphery of the lesions were calculated.

Results

8 hyperacute cerebral ischemic regions were revealed at DWIs and ADC maps , but T2WIs were not. Hyperacute and acute infarcts appeared as areas of hyperintensity on DWI, and their average ADC was significantly depressed compared with homologous contralateral tissue(0.6980.104 verus 0.9900.66110-3mm2/s, p0.001). RADC in hyperacute and acute stage was minimum, and increased progressively as time pass and appeared as 'pseudonormal' values approximately 8 to 14 days, thereafter rADC became greater than normal in chronic stage(figure 1).

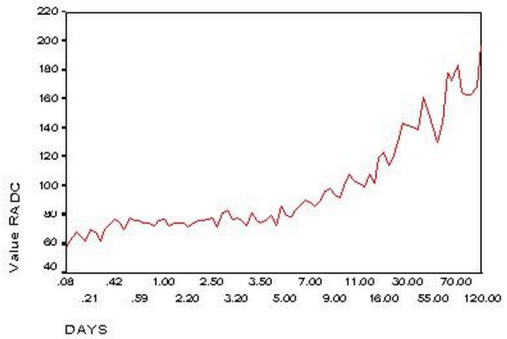

Figure 1. rADC plotted against time from stroke onset in days for all cases.

There was positive correlation between rADC and time (r=0.928, p0.001). ADC values in 21 hyperacute, acute and 7 subacute lesions had gradient sign(figure 2).

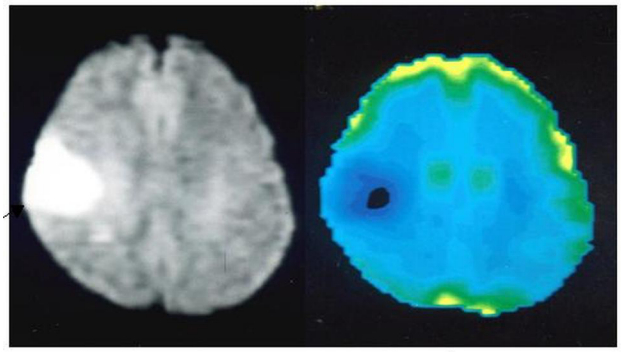

Figure 2. (a) DWI shows early ischemic lesion(arrow) of the right hemisphere 24 hours afrer the onse of symptom. (b) The corresponding ADC map confirms that ADC reveals gradient sign,the ADC is lower in the lesion core than in peripheral part.

In the 14 cases which had re-MRI, the infarct lesion became smaller than its initial lesion in DWI in 7 cases and their clinical manifestation was better; the lesion became greater in 4 cases, and their clinical manifestation became worse; and the lesion of the remainder 3 cases remainded unchanged, whose clinical manifestation was unchanged.

Discussion and Conclusion

Isotropic DWI and ADC map have greater sensitivity for acute, especially hyperacute cerebral infarction than T2WI, can diagnose hyperacute and acute stage of cerebral infarction rapidly, accurately. ADC map reveals a lesion core and periphery with distinct ADC values, this variability has been attributed to the heterogeneity of stroke evolution[5]. DWI and rADC can reflect cellular status and hence may predict ischemia outcome. RADC have specific evolving rule on ADC map, ADC elevation indicating some residual tissue demage[6], combined with DWI and T2WI can differentiate different stages of infarction, and may portend a time when stroke may be treated more effectively and with individually tailored therapy, and evaluate the efficacy of treatments by dynamic observation.

References

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