Age-related changes of susceptibility and phase changes at subependymal nodes in infants and children with tuberous sclerosis.

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Introduction

Subependymal nodules (SENs) in tuberous sclerosis are a kind of hamartomatous lesion; those are seen at the periphery of the lateral ventricles [1]. The signal intensities of SEN are variable depending on calcification or the contrast to the surrounding white matter. In neonates, SENs are usually hypersignal on T1-weighted imaging and hyposignal on T2-weighted imaging, sometimes mimicking hemorrhage.

Susceptibility-weighted imaging (SWI) is a recent technique to enhance the susceptibility objects using the phase data [2]. Recently, SWI phase image has been shown to be well-visualization method for calcification [3]. The purpose of this study was to assess the age-related changes of SWI and phase image at SENs in infants and children with tuberous sclerosis.

Subjects and Methods

Images of 20 children (age range, 0 to 12 years; mean, 4.6 years) with tuberous sclerosis, who showed SENs, were included in this study. SWI and filtered phase images were retrospectively assessed. The finding of SWI and phase image at the SENs were assessed by two experienced radiologist with a consensus, using the three-point scale; 1, no signal loss (no phase shift); 2, slight signal loss (slight phase shift); 3, prominent signal loss (prominent phase shift). When multiple subependymal nodules were appeared in children, score at the largest subependymal nodule was considered as a representative one. The age of children with and without signal loss on SWI, the age of children with and without the phase shift on phase image at the SENs was compared.

Results

The median age of score 0 and 2 on SWI was 0 and 9 years, respectively (Figure 1). There were no children with score 1 on SWI. The age of children showing no signal loss at the SENs was significantly lower than those showing signal loss (median age, 0 vs 9 years, p<0.001). The median age of score 0,1 and 2 on phase image was 0,1 and 9, respectively (Figure 2). The age of children showing no phase shift at the SENs was significantly lower than those showing phase shift (median age, 0 vs 7 years, p=0.001). Representative images are shown in Figures 3,4.





Figure 1.

Graph shows the relationship between the score on SWI at the subependumal nodule and age. The age of children showing no signal loss at the subependymal nodules was significantly lower than those showing signal loss.



Figure 3.

Images of an 8-day-old neonate with tuberous sclerosis. A: T1-weighted image, B: T2-weighted image, C: SWI, D: phase image Subependymal nodules are shown as hypersignal on T1-weighted image (Fig. A), and hyposignal on T2-weighted image (Fig. B). No signal loss on SWI and no phase changes on phase image are seen at the subependymal nodules.

Graph shows the relationship between the score on phase image at the subependymal nodule and age. The age of children showing no phase shift at subependymal nodules was significantly lower than those showing phase shift





Images of a 2-year-old child with tuberous sclerosis. A: T1-weighted image, B: T2-weighted image, C: SWI, D: phase image Subependymal nodule is shown as slight hypersignal on T1-weighted image (Fig. A), and hyposignal on T2-weighted image (Fig. B). Signal loss on SWI and phase shift on phase image are seen at the subependymal nodule.

Discussion and Conclusions

We found no signal loss and phase shift at the SENs in neonates. It seems that there is no calcification at the SENs in neonates with tuberous sclerosis. Therefore, SWI is useful to discriminate these hamartomatous lesions from hemorrhage, especially at neonatal periods. In this study, the occurrence of the phase shift at the SENs was identified around 1-year-old, while signal loss on SWI appeared after 2-year-old. The manifestation of the phase shift at the SENs seems to precede that of signal loss on SWI. These findings are useful for correct interpretation of the MR images of children with tuberous sclerosis.

In conclusions, age-related changes of susceptibility were identified at the SENs in infants and children with tuberous sclerosis.

References

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