Materials and Methods

ASD and healthy normal subjects were recruited from a child and adolescent psychiatric clinical groups, well-defined community sources and schools in Koyang area in South Korea. We enrolled thirty four male subjects (17 ASD: mean age 11.2 ± 2.06 years old, 17 healthy control: mean age 10.18 ± 2.04 years old). We used SRS (Social Responsiveness Scale), ASSQ (Autism Spectrum Screening Questionnaire), ADI-R-K (Autism Diagnosis Interview-Revised-Korean version), and ADOS-K (Autism Diagnostic Observation Schedule-Korean version) to screening and diagnosis all ASD subjects. We performed MRI scans using a 1.5T clinical MRI scanner (Avanto 1.5T, Siemens, Erlangen, Germany) with a gradient strength of 40 mT/m. 12-channel head matrix coil was used for study. Diffusion weighted images were acquired using a spin-echo based single-shot echo-planar diffusion sequence. The specific MR imaging parameters used were as follows: TR = 6500 ms; TE = 86 ms; number of diffusion gradient directions = 30; b value = 900 s/mm²; number of excitation = 2; GRAPPA factor = 2. The in-plane resolution was 1.8 mm, and the slice thickness was 3 mm without gap. Preprocessing of MR images and calculate diffusion indicies map such as FA images were performed using FSL (FMRIB Software Library -www.fmrib.ox.ac.uk/fsl). For SPG brain analysis we generated a WM (white matter) only FA (or MD) image. This study was approved by the institutional review board for human subjects at the Myong-Ji Hospital affiliated with Kwandong University College of Medicine in Kyunggi and Gachon Neuroscience Research Institute in Incheon, where all subjects were scanned, in South Korea.

Results

As shown in Table 1, the ASD group and healthy control group were group-matched on age, IQ, and handedness. In Table 2 shows the results of individual ROI based analysis, ASD subjects showed significantly reduced FA values in corpus callosum (CC), both inferior longitudinal fasciculus (IFL) adjacent to facial fuisiform area (FFA), left uncinate fasciculus (overlapped with inferior fronto-occipital fasciculus) and both anterior thalamic radiation compared to the control subjects. We did VBM style group analysis of FA value and the results are displayed in Fig.1(a) and (b). The activated area represent that ASD group have significantly (p<0.05) lower FA value than healthy control. In CC (a) and left IFL (b) we found significant difference of FA value number between two groups. To confirm these VBM analysis results we did individual ROI data based independent t-test in CC and IFL regions. In both regions the differences are significant (Fig. 1c-d). We can find one more information in Fig.1(d), the difference between two groups are much significant at the left side. And compare with left and right hemisphere FA value in each group, healthy control group shows big difference however ASD group shows not much. So we calculated laterality (absolute value of (left-right)/(left+right)) in IFL and compare this value between two groups. The comparison result of laterality is shown in Fig.2.

Conclusions

Our preliminary findings which showed significant reduction of FA in white matter structure related social cognition in ASD subjects compared control subjects support previous findings that social brain structure may be disrupted in ASD. These findings will help on understanding of more advanced neurobiological basis underlying the social deficits in ASD. Nevertheless, we will still need to focus on evaluating the association between each symptom shown in ASD individuals and brain abnormalities in the future study.

References