Parkinsonism Caused by Substantia Nigra Injury Following CO Intoxication: A Quantitative Study by IR Gray Matter Subtraction MR Imaging

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Purpose:

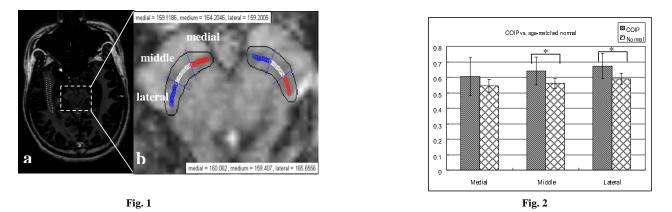
Parkinsonism is a known neurological sequela of carbon monoxide (CO) poisoning. It involves neuron degeneration in the substantia nigra (SN) area where the internal structure is hardly detectable with use of conventional MR sequences [1-2]. MR imaging with inversion-recovery gray matter suppression (IR-GMS) technique has been reported to be sensitive in showing structural changes in pars compacta (PC) of substantia nigra and thus is expected to be useful in deriving information of PC degenerations in carbon-monoxide-induced parkinsonism (COIP) patients. The purpose of this study was designed to evaluate the PC degeneration in COIP patients using GMS sequence with a skeleton-based image segmentation program.

Material and methods:

Eleven patients with acute or delayed COIP (age range, 24-68 years; mean age, 35.5 ± 13.6 years) and eleven age-matched healthy control subjects (age range, 21-70 years; mean age, 34.6 ± 14.9 years) were enrolled in this study. All IR-GMS MR imaging were performed on a 1.5T clinical MR scanner (Vision Plus, Siemens, Erlangen, Germany) with TR/TE/TI=2140/30/420, respectively. An axial image on the basis of uniform white matter signal intensity and intact PC with sufficient contrast between the PC and surrounding structures was chosen for analysis. The IR-GMS images were then transferred to a personal computer for processing. All software programs for postprocessing were developed in house with Matlab programming language. We developed an improved algorithm of using morphology skeleton-based method in the region-of-interest (ROI) analysis. One large area (shown in Fig.1b) containing PC was first manually traced by an experienced neuroradiologist who is experienced in neurological MR imaging over ten years. The morphology skeleton line of this area was then automated extracted with equal distance from the area boundary. The traced SN area was further subdivided into three parts from medial to lateral with regard to equal length of skeleton and the mean intensities for each region were calculated. For purpose of normalization, an additional ROI was placed in the area of normal white matter (Fig. 1a.) to calculate the mean reference signal. All the normalized signals of PC regions relative to the white matter area were compared between two groups with unpaired two-sample t-test.

Results:

Figure 2 shows the mean signal ratios and standard deviations of three segmented PC for these two groups. Since there was no statistical difference between each segment of paired left and right PC in each group, the averaged values of the left and right measurements were used for comparison. The results show significant PC injuries in COIP patients with hyperintensity changes in middle and lateral segments on IR-GMS imaging as compared to the control group (p<0.05). There was also a general trend of increased degeneration in segment approaching to the lateral parts of PC in our preliminary observations (p=0.07, 0.007, 0.004 from medial to the lateral, respectively). Furthermore, the borders of the PC appeared more irregular in COIP patients, especially in the lateral segments. This is consistent with the progression of dopaminergic neuron degeneration.



Discussions and conclusions:

COIP is caused by a progressive degeneration of PC. Although visualization of PC degeneration in dopamine-related diseases has been reported previously, accurate quantification of PC injury in COIP patients has not been previously explored. Noninvasive imaging quantification of PC may provide information about the pathogenetic mechanisms underlying COIP and is potentially of great value for diagnosis and follow-up after hyperbaric oxygen therapy. We employed a novel application with IR-GMS sequence in this study to demonstrate its usefulness in allowing a confident differentiation between COIP and normal control groups. With the morphology skeleton-based segmentation technique, the intrinsic changes of PC for COIP patients and patients without parkinsonism after CO intoxication can be objectively and quantitatively evaluated.

References [1] Hutchinson M, et al. AJNR Am J Neuroradiol 2000; 21:697-701. [2] Minati L, et al. AJNR Am J Neuroradiol 2007; 28:309-313.