Assessment of the Neurologic Effects of Intracranial Gadolinium Deposition Using a Large Population Based Cohort

Wednesday 3:00-3:10 PM | SSM16-01 | Room: N226

PURPOSE

The neurotoxic potential of intracranial gadolinium (Gd) deposition following intravenous administration of gadolinium based contrast agents (GBCAs) is undefined. In the current study, we used the world's largest prospective population-based cohort on aging to study the effects of Gd exposure on neurologic and neurocognitive function.

METHOD AND MATERIALS

The Mayo Clinic Study of Aging (MCSA) cohort was enumerated from the Rochester Epidemiology Project in 2004 to study the incidence and natural history of cognitive impairment and dementia. All participants underwent extensive longitudinal clinical (neurologic evaluation, neuropsychological testing) assessment at baseline and 15-month follow-up intervals. Neurologic and neurocognitive scores were compared using standard multivariate methods between MCSA patients with no history of prior Gd exposure and those who underwent prior Gd-enhanced MRI. Progression from normal cognitive status to mild-cognitive impairment and dementia was assessed using multistate Markov model analysis.

RESULTS

Among 4261 cognitively normal study participants aged 50-90 (mean age (SD): 71.9 yrs (10.7), mean study participation (SD): 3.7 yrs (3.0)), 1092 (25.6%) received one or more GBCA doses (median: 2 doses, range: 1-28 doses) unrelated to their participation in the MCSA. Median time since first Gd exposure was 5.6 years (IQR=2.2-9.3 years). After adjusting for age, sex, education level, baseline neurocognitive performance, Charlson comorbidity index, and ApoE4 status, GBCA exposure was not a significant predictor of cognitive decline (changes in clinical dementia rating (p=.48), Blessed dementia scale (p=.68), or mental status exam score (p=.55)), diminished neuropsychological performance (p=.13), or diminished motor performance (Unified Parkinson's Disease Rating Scale (p=.43)). No dose-related effects were observed among these metrics (p=.89-.20). Finally, Gd exposure was not an independent risk factor in the rate of cognitive decline from normal cognitive status to dementia in this cohort (p=.91).

CONCLUSION

GBCA administration was not associated with worse overall neurologic or neurocognitive performance nor does it significantly affect the natural progression of cognitive decline in a large population-based cohort.

CLINICAL RELEVANCE/APPLICATION

Despite evidence of Gd accumulation following intravenous GBCA administration, Gd exposure is not associated with adverse neurologic outcomes.