Memory Care Monthly

Supporting Healthcare Professionals in Caring for the Aging September / October 2008

NATIONAL MEMORY SCREENING DAY: NOVEMBER 18

November 18 is a National Memory Screening Day. It is a collaborative effort lead by the Alzheimer's Foundation of America (AFA) to promote early detection of Alzheimer's disease and related illnesses, and to encourage appropriate intervention.

AFA collaborates with organizations and healthcare professionals across the U.S. and offer free confidential memory screenings, as well as follow up resources and educational materials to those concerned about memory loss.

For further information, please visit: www.nationalmemoryscreening.org.

FEATURED ARTICLE

ALZHEIMER'S DISEASE: CURRENT ATTITUDES, PERCEPTIONS AND KNOWLEDGE

A new survey revealed disparities between beliefs and behavior in pursuing Alzheimer's screening and diagnosis. The results demonstrate the urgent need for increased education and awareness of disease symptoms and benefits of early diagnosis and treatment.

The online survey of 1,040 U.S. adults over 55 years old was conducted by Harris Interactive and commissioned by the Alzheimer's Disease Screening Discussion Group (ADSDG), a consortium of multi-disciplinary experts in Alzheimer's disease (AD) and senior health.

In the survey, almost 95% agree that they would encourage a loved one to seek early diagnosis upon suspecting signs of AD. However, of 34% who thought their loved one had the disease, only one-quarter prompted that person to take an AD screening and less than 40% encouraged them to seek conversation with doctors. The survey also found that more than 90% could not accurately distinguish early disease symptoms from late disease symptoms or symptoms unrelated to AD although 78% believe that they could notice the signs of AD in themselves or a loved one. Moreover, almost one-third are not aware that there are AD medications currently available and about 85% of those who are aware about the medications do not understand how treatment works.

Alzheimer's Disease Screening Discussion Group (www.seethesigns.com), 2008.

RESEARCH UPDATES

EFFECTS OF AMYLOID-BETA OBSERVED LIVE IN MICE

For the first time, researchers observed what amyloid-beta does to the function and structure of nerve cells in normal adult rats brain.

Dr. Ganesh Shankar from the Brigham & Women's hospital and Harvard Medical School, and his colleagues extracted soluble amyloid-beta protein (Abeta) oligomers directly from the cerebral cortex of patients with Alzheimer's disease (AD), and injected extracts into normal adult rats to see whether Abeta could have acute effects on brain synapses and on behavior.

They found that the oligomers potently inhibited long-term potentiation (LTP), enhanced long-term depression (LTD) and reduced dendritic spine density in a normal rat hippocampus. Soluble Abeta from AD brains also disrupted the memory of a learned behavior in normal rats. These various effects were specifically attributable to Abeta dimers. Mechanistically, metabotropic glutamate receptors were required for the LTD enhancement, and N-methyl D-aspartate receptors were required for the spine loss.

Co-administering antibodies to the Abeta N-terminus prevented the LTP and LTD deficits, while antibodies to the midregion or C-terminus was less effective. Insoluble amyloid plaque cores from AD cortex did not impair LTP unless they were first solubilized to release Abeta dimers, suggesting that plaque cores are largely inactive but sequester Abeta dimers that are synaptotoxic.

Researchers concluded that soluble Abeta oligomers extracted from AD brains potently impair synapse structure and function and that dimers are the smallest synaptotoxic species.

Shankar G, et al. Nature Medicine. 2008. 14: 837 – 42.

PHYSICAL ACTIVITY AND COGNITIVE FUNCTION IN OLDER ADULTS AT RISK FOR AD

A randomized controlled trial of a 24-week physical activity intervention was conducted between 2004 and 2007 to determine whether the intervention can reduce the rate of cognitive decline among older adults at risk for Alzheimer's disease.

A total of 170 people over 50 years old with self-reported memory problems were randomized and 138 participants completed the 18-month assessment. All participants did not meet criteria for dementia at baseline. Participants were randomly allocated to an education and usual care group or to a 24-week home-based program of physical activity. The intervention group improved 0.26 points on the ADAS-Cog at the end of the intervention, while the usual care group deteriorated 1.04 points. The absolute difference on the outcome measure between the intervention and control groups was -1.3 points at the end of the intervention.

At 18 months, the intervention group improved 0.73 points on the ADAS-Cog while the usual care group improved 0.04 points. Word list delayed recall and Clinical Dementia Rating Scale sum of boxes improved modestly whereas cognitive assessments such as word list total recall, digit symbol coding, and verbal fluency did not change significantly.

This study was conducted in metropolitan Perth, Western Australia, and by Dr. Nicola Lautenschlager and his colleagues from Academic Unit for Psychiatry of Old Age, University of Melbourne.

Lautenschlager NT, et al. JAMA 2008; 300(9):1027-37.

WITHIN-PATIENT ACROSS-NEUROPSYCHOLOGICAL TEST VARIABILITY PREDICTS INCIDENT DEMENTIA

Dr. Roee Holtzer and his colleagues from Dept of Neurology, Albert Einstein College of Medicine of Yeshiva University, Bronx, NY, examined whether within-person across-neuropsychological test variability predicts future dementia using data from the Einstein Aging Study (EAS), a population-based longitudinal study of aging and dementia.

A total of 896 from the 1797 EAS enrollees (age 70 or older) were included in this study, and they had follow-up visits every 12 to 18 months between October 1993 and December 2007. Neuropsychological tests included the Free and Cued Selective Reminding Test, the Digit Symbol Substitution subtest of the Wechsler Adult Intelligence Scale Revised (WAIS-R), and the Vocabulary subtest of the WAIS-R. During follow-up (mean [SD], 3.3 [2.4] years), 61 cases of incident dementia were identified, of which 26 were in the highest quartile of within-person across-neuropsychological test variability. Adjusting for gender, education and medical illness, variability was associated with incident dementia. The association persisted even after adjusting for level of performance on individual neuropsychological tests.

Holzer R, et al. JAMA 2008; 300(7):823-30.

PHYSICAL FRAILTY IN ELDERY ASSOCIATED WITH ALZHEIMER'S PATHOLOGY

Researchers from Rush Alzheimer's Disease Center, Ruth University Medical Center, Chicago, IL, studied brain autopsies from 165 cases from the Rush Memory and Aging Project, and examined association between physical frailty in older persons and common age-related brain pathology including Alzheimer's disease (AD), cerebral infarcts and Lewy body disease.

Physical frailty was measured annually in those 165 cases based on grip strength, time to walk 8 feet, body composition and fatigues. Multiple regression analyses were used to examine the relation of postmortem (mean age at death = 88.1 with SD=5.7) neuropathologic findings to frailty proximate to death, controlling for age, gender and education.

The level of AD pathology was associated with frailty proximate to death regardless of a presence of dementia, accounting for 4% variance of physical frailty, while neither cerebral infarcts nor Lewy body disease pathology was associated with frailty.

Buchman AS, et al. Neurology 2008; 71(7): 499-504.

THE IMPORTANCE OF DAYTIME MOVEMENTS IN ELDERY

Dr. Deborah E. Barnes and her colleagues from University of California San Francisco studied the relationship between daytime movements and cognitive functions. 2736 older women (mean age of 83±4) without evidence of dementia participated in the study; 10% were African American.

Daytime movement was assessed using actigraphy, which involved wearing a watch-like device

that objectively quantified accelerometer motion over a mean of 3.0±0.8 days. Cognitive function was measured using the Trail-Making Test, Part B (Trails B) and the Mini-Mental State Examination (MMSE). Cognitive impairment was defined as performing 1.5 standard deviations (SDs) worse than the mean on a given test.

Results show that, after adjustment for age, race, and education, the highest movement quartiles had better mean cognitive test scores (20±0.3 seconds faster on Trails B and 0.3±0.2 points higher on MMSE, both P<.001) than those in the lowest quartile, and were less likely to be cognitively impaired (odds ratio (OR)=0.61, 95% confidence interval (CI)=0.41–0.92 for Trails B; OR=0.68, 95% CI=0.44–1.07 for MMSE). These results were independent of self-reported walking, medical comorbidities, physical function, and other health-related behaviors.

Barnes DE, et al. JAGS 2008; 56(9): 1658-64.

EFFECT OF INFARCTS ON DEMENTIA

A prospective study was conducted by Dr. Juan C. Troncoso from Department of Pathology, Johns Hopkins University, Baltimore to determine the magnitude and mechanism of the effect of brain infarcts on the odds of dementia.

The researchers examined the effects of brain infarcts and Alzheimer's disease (AD) pathology on the risk for dementia in 179 subjects from the Baltimore Longitudinal Study of Aging Autopsy Program. All subjects had longitudinal clinical and cognitive evaluations, and underwent postmortem examination of the brain.

Brain infarcts were common among the study group, and both symptomatic and asymptomatic infarcts conferred a significant increase in the odds of dementia. Risk factors for stroke in the absence of an infarct did not increase the odds of dementia, which was quantitatively related to the number but not the size of hemispheral infarcts; deep subcortical infarcts conferred no increased risk for dementia. The contribution of microscopic infarcts to dementia was significant and equivalent to that of macroscopic infarcts. In subjects with intermediate AD pathology scores, a single macroscopic hemispheral infarct was sufficient to cause dementia. A logistic regression model of the effect of infarcts and AD pathology on dementia indicated that AD pathology alone accounts for 50% of the dementia seen in this cohort, and that hemispheral infarcts alone or in conjunction with AD pathology account for 35%.

Troncoso JC, et al. Ann. Nruol. 2008; 64(2): 168-76.

DEMENTIA DIAGNOSIS AND BREAST CANCER SURVIVORS WHO USE CHEMOTHERAPY

To determine patterns of dementia diagnosis seen after chemotherapy treatment, Dr. Julia E. Heck and her colleagues from Departments of Epidemiology and Health and Behavior Studies, Columbia University, New York, examined the linked Surveillance, Epidemiology, and End Results (SEER)-Medicare database, ICD-9 diagnoses of dementia occurring in the years after breast cancer diagnosis.

From the SEER program, which collects information from population-based tumor registries in seven metropolitan areas (San Francisco and Oakland, Detroit, Atlanta, Seattle, Los Angeles County, San Jose and Monterey Counties, and the greater California area) and eight states (Connecticut, Iowa, New Mexico, Utah, Hawaii, Kentucky, New Jersey, and Louisiana), 18,360 women diagnosed with Stage II, III, or IV breast cancer were identified.

The results show that there were significant differences at baseline between individuals who received and did not receive chemotherapy. In the first few years after breast cancer diagnosis, dementia was more common in women who had not had chemotherapy, probably reflecting group differences at baseline. In the longer term, diagnoses of dementia were more common in women who had chemotherapy treatment (hazard ratio=1.20, 95% confidence interval=1.08–1.33).

This study suggests the possibility of severe cognitive changes associated with chemotherapy, particularly over the long term.

Heck JE, et al. JAGS. 2008; 56(9): 1687-92.

Free Patient Brochure

Make sure your patients know that you now offer memory assessment services. We can send you complimentary copies of our educational patient brochure, "About Memory Loss", in English and Spanish. To request copies, please email us at: customerservice@mccare.com and include the address to where you would like the brochures mailed.

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