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A retrospective chart review of course of hypertension in Alzheimer's disease patients

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Introduction: Higher blood pressure (BP) levels have been associated with dementia, particularly for Alzheimer's disease. Therefore, a significant proportion of patients with AD are on antihypertensive medications. However, during the course of AD, BP levels decline due to a number of factors, including degeneration of brain stem autonomic neurons. Such changes appear to reduce and possibly eliminate need for antihypertensive drugs. Continued maintenance on antihypertensive medications in these situations may predispose patients to falls, fractures and episodes of confusion.
Objective: To determine the relationship between the progressions of Alzheimer's disease (AD) and pre-existing hypertension.
Methods: We retrospectively reviewed the charts of all patients diagnosed of possible or probable Alzheimer's disease using National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's disease and Related Disorders Association (NINCDS-ADRDA) criteria from our own private clinical practice who had antihypertensive medications discontinued subsequent to being diagnosed.
Results: There were 16 patients with probable or possible AD who demonstrated a significant decrease in their BP requiring discontinuation of their antihypertensive drugs as their condition progressed. None of these patients experienced an increase in BP requiring reinstitution of their anti-hypertensives.
Conclusions: From a review of the literature and based on our small exploratory study, clinicians may do well to periodically reassess need for antihypertensive medications in patients with Alzheimer's disease.

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Transcranial magnetic stimulation of deep brain regions in Alzheimer's disease

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at frequency 10Hz and intensity 10% above the motor threshold. Efficacy measured by changes of ADAScog and MMSE, showed a trend to clinical improvement. 2 patients reported significant clinical improvements. No significant adverse events were recorded. These preliminary data support continuation of the study.

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Isolated bulbar paralysis in a patient with medullar tau pathology: a case report

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Objective: We report a 77-year-old female patient who presented with a 6 year history of dyspnoea, stridor and dysphagia. Other than that her neurological status was normal. Contrast radiographical examination of swallowing showed retention of the contrast and weak peristaltic activity of the oesophagus. Extensive diagnostics including EMG, micro-EMG, ANCA were normal. MRI of the head showed atrophy of the temporoparietal region. Paralysis of both vocal folds was found and from time to time she needed mechanical ventilation. Tracheotomy was performed. Gradually dysphagia worsened and she became cachectic. In December 2007 the patient was admitted to the Intensive Care Unit of our Neurological Department because of another sudden respiratory failure. On admission she was mechanically ventilated, awake, cooperative, afebrile. CT scan of the head was interpreted as normal. Chest X-ray and a positive aspirate were diagnostic of pneumonia. After antibiotic treatment her clinical status improved. She was breathing unaided, talking and walking. The following day she unexpectedly died in her sleep. Histopathological examination disclosed extensive degeneration of the medulla by tau pathology. The majority of symptoms and signs could be explained by the medullary tau pathology.
Conclusion: This is the first case report of a patient with bulbar symptoms and signs, which could be explained by prominent tau pathology of the medulla. Whether medullary tau pathology in this case was a rare aberrant progression of Alzheimer's disease or a new presentation of tauopathy concomitant with subclinical Alzheimer's disease should be elucidated by additional studies.