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Prevalence of and Factors Associated with Dural Thickness in Patients with Mild Cognitive Impairment and Alzheimer's Disease

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Abstract

Background and Purpose—We performed this study to evaluate the prevalence of and factors associated with dural thickening in patients with mild cognitive impairment and Alzheimer's disease.

Methods—Alzheimer's disease neuroimaging initiative participants with axial FLAIR sequence magnetic resonance imaging (MRI) images were analyzed. Dural thickness was defined by a linear strip of hyperintense tissue signal along the dura mater observed in at least two different images without evidence of leptomeningeal involvement.

Results—Dural thickening was seen in 83 (34%) of 242 persons analyzed (mean age [±SD] 74±7 years: 150 were men) with either mild cognitive impairment or Alzheimer's disease. The mini mental score was not different in persons with (26±0.3) and without (26±0.2) dural thickening (p = 0.6). The proportion of patients with moderate or severe cognitive impairment (defined by mini mental status score) was similar at baseline and at 12-month evaluations. The rates of annual progression according to Alzheimer's disease assessment scale (p = 0.06) and clinical dementia scale (p = 0.001) were higher in persons without dural thickening. The annual rate of volume loss in entorhinal cortex was higher among persons with dural thickening.

Conclusions—We found relatively high prevalence of dural thickening in patients with mild cognitive impairment and Alzheimer's disease.

Keywords

dura; dural thickness; mild cognitive impairment; Alzheimer's disease; FLAIR

Background and Purpose

The dura is composed of elongated and flattened fibroblasts intermingled with extracellular collagen. A specialized layer of fibroblasts is found at the dura-arachnoid junction [1]. The arachnoid layer is composed of larger cells with numerous cell junctions without any extracellular collagen. An interface layer comprised of a complex, tight layer of cells connects the innermost portion of the dura mater and the outermost portion of the arachnoid mater [2]. Presently, there are data that support the presence of meningeal changes, including fibrosis in patients with dementia [3–5]. Mutation in presenilin 1 (PS1) is one of the leading causes of familial Alzheimer's disease and can lead to altered autophagy and degradation among fibroblasts [6,7]. Intense amyloid deposition in the leptomeninges and vessel walls can be observed in familial amyloidosis [8–11]. The drainage of interstitial fluid from the brain passes within dural layers into venous sinuses and appear to be blocked by amyloid-beta in Alzheimer's disease [12].

We performed this study to further evaluate the prevalence of and factors associated with dural changes in patients with mild cognitive impairment and Alzheimer's disease. Specifically, we studied dural thickening that appears as a hypointense area with fine hyperintense edges on T2-weighted images and hyperintense area on coronal T1-weighted images [13–16].

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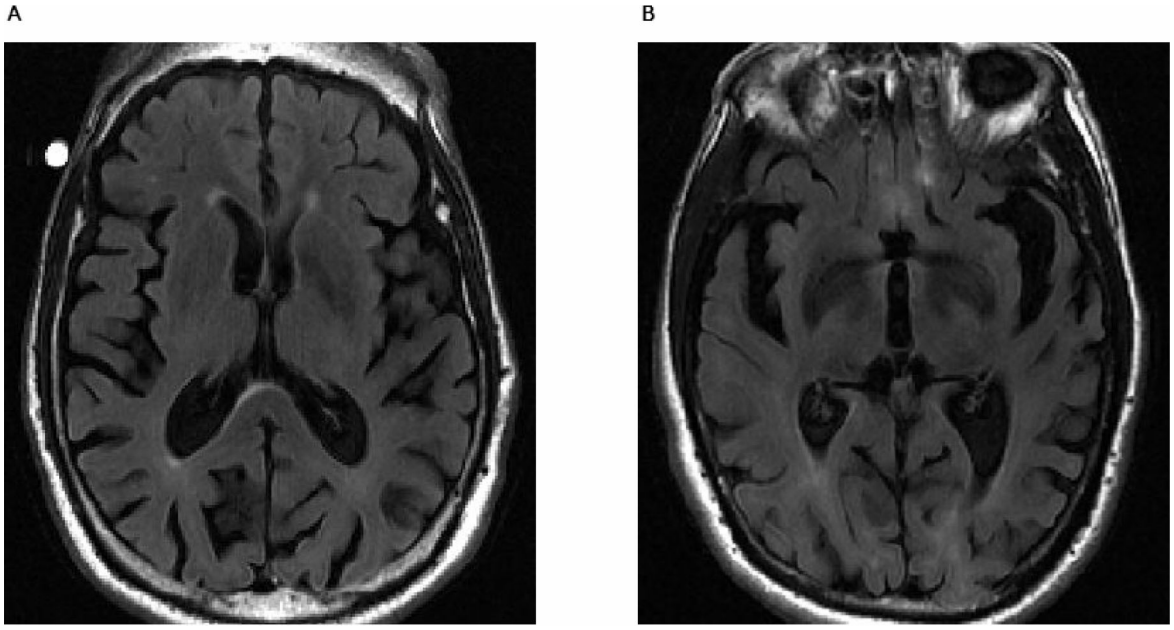

Figure 1. Axial images on Flair sequence of magnetic resonance imaging.
a. Dural thickness is visualized as curvilinear hyper-intensity surrounding brain parenchyma.
B. Normal dura surrounding brain parenchyma.

Methods

We analyzed clinical and neuroimaging data collected as part of the Alzheimer's disease neuroimaging initiative (ADNI). ADNI is an ongoing, longitudinal, multicenter study designed to develop clinical, imaging, genetic, and biochemical biomarkers for the early detection and tracking of Alzheimer's disease [17]. The ADNI study began in 2004 and included 400 subjects diagnosed with mild cognitive impairment, 200 subjects with early AD and 200 elderly control subjects from 200 normal controls and 400 individuals with mild cognitive impairment recruited at approximately 50 sites in the United States and Canada.

Data used in the preparation of this article were obtained from the Alzheimer's disease neuroimaging initiative (ADNI) database [18]. The goal of ADNI is to recruit 800 adults, aged from 55 to 90, to participate in the research—approximately 200 normal older individuals to be followed for 3 years, 400 people with mild cognitive impairment to be followed for 3 years, and 200 people with early Alzheimer's disease to be followed for 2 years [19]. The presence of cardiovascular risk factors including hypertension, hyperlipidemia, diabetes mellitus, prior stroke, or myocardial infarction is recorded for each participant. Any history of depression or cancer is recorded as well. The mini mental status examination (MMSE), Alzheimer's disease assessment scale, and clinical dementia rating scores at baseline and follow-up visits were determined as part of ADNI.

All ADNI participants with structural magnetic resonance imaging (MRI) images whose MRI scans were analyzed and results available on the ADNI website as of July 2013 (the latest scan was from July 09, 2013) were included. The datasets included standard T1-weighted MR images acquired sagittally using volumetric 3D MPRAGE with 1.25 × 1.25 mm in-plane spatial resolution and 1.2-mm-thick sagittal slices (8°flip angle). Most of the images were obtained using 1.5 T scanners, while a few were obtained using 3 T scanners. Detailed information about MR acquisition and analyses procedures is available in previous publications [20,21]. All participants underwent 1.5 T structural MRI at specified intervals (6 or 12 month) for 2–3 years.

Of the 800 persons with baseline MRI, we selected those who had an axial FLAIR sequence performed. One of the investigators (IL) reviewed all the images and identified those patients who had dural thickening. Dural thickness was defined by a linear strip of hyperintense tissue signal along the dura mater observed in at least two different images without evidence of involvement of the leptomeninges (no abnormal enhancement around the brainstem, within the sylvian fissures, or in the depth

Table 1. Demographic, clinical, and neuroimaging characteristics of patients with mild cognitive impairment or Alzheimer's disease stratified by the presence or the absence of dural thickness

	Patients with dural thickness	Patients without dural thickness	
Overall numbers (%)	83	159	
Age (mean±SD) in years	74±8	74±7	0.1
Gender			
Men	44(53.0)	106(66.6)	
Women	39(47.0)	53(33.4)	0.05
Race			
White	78(94.0)	148(93.1)	
Other	5(6.0)	11(6.9)	0.9
Comorbids			
Depression	9(10.8)	10(6.3)	0.2
Hypertension	11(13.2)	17(10.7)	0.5
Diabetes mellitus	8(9.6)	10(6.3)	0.4
Hyperlipidemia	11(13.2)	19(12.0)	0.8
Atrial fibrillation	3(3.6)	15(9.4)	0.1
Cigarette smoking	12(14.5)	16(10.1)	0.4
APO E4 gene			
No copies	38(45.8)	59(37.1)	
Heterozygous	33(39.8)	80(50.3)	
Homozygous	12(14.6)	20(12.6)	0.3
Cognitive scales – mean ± SE			
Mini-mental state examination			
Mini-mental state examination (Baseline)	26±0.3	26±0.2	0.6
Mild impairment	81(97.6)	156(98.1)	
Moderate impairment	2(2.4)	3(1.9)	0.9
Mini-mental state examination (12 month)	24±0.5	24±0.3	0.8
Mild impairment	65(78.3)	133(83.6)	
Moderate impairment	18(21.7)	25(15.8)	
Severe impairment	0(0)	1(0.6)	0.4
Mini-mental state examination (Annual % decrease)	5±1.5	5±1.1	0.8
Alzheimer disease assessment scale			
Alzheimer disease assessment scale (Baseline)	14±0.7	13±0.5	0.5
Normal	19(22.9)	34(21.8)	
Possible cognitive deficit	13(15.6)	33(20.7)	
Definitive cognitive deficit	51(61.5)	92(57.8)	0.6
Alzheimer disease assessment scale (12 month)	15±0.9	16±0.6	0.8
Normal	22(26.5)	31(19.6)	
Possible cognitive deficit	7(8.5)	33(20.7)	
Definitive cognitive deficit	54(65.0)	95(59.7)	0.04
Alzheimer disease assessment scale (annual % Increase)	19±7.4	30±6.5	0.06
Clinical dementia rating - sum of boxes range			
Clinical dementia rating - sum of boxes range (Baseline)	2.6±0.2	2.5±0.1	0.5
Normal to mild dementia	83(100)	159(100)	
Clinical dementia rating - sum of boxes range(12 month)	3±0.3	3±0.2	0.5
Normal to mild dementia	81(97.6)	151(95.0)	0.5
Moderate dementia	2(2.4)	8(5.0)	
Clinical dementia rating - sum of box range (Annual % increase)	31±9.6	44±9.6	0.001
Brain Volumes (mean±SE)			
Whole brain (Baseline)	9,84,055±12,765	9,95,845±9914	0.5
Whole brain (12 month)	9,70,102 ±12574	9,83,778±10027	0.3
Whole brain (Annual % decrease)	1.4±0.2	1.2±0.1	0.8
Hippocampus (Baseline)	6,046±136	6,202±97	0.9
Hippocampus (12 month)	5,854±136	5,985±99	0.9
Hippocampus (Annual % decrease)	3.2±0.4	3.6±0.3	0.5
Ventricles (Baseline)	45,637±2,666	45,289±2,014	0.6
Ventricles (12 month)	49,630±2,773	48,630±2,120	0.6
Ventricles (Annual % decrease)	(−10)±1.1	(−8.1)±0.6	0.05
Middle temporal (Baseline)	17,986±315	18,267±259	0.2
Middle temporal (12 month)	17,462±317	17,715±273	0.08
Middle temporal (Annual % decrease)	3.0±0.4	3.2±0.4	0.6
Fusiform (Baseline)	15,683±300	16,048±210	0.7
Fusiform (12 month)	15,323±301	15,610±207	0.6
Fusiform (Annual % decrease)	3.0±0.5	2.6±0.4	0.9
Entorhinal (Baseline)	3,155±90	3,130±60	0.4
Entorhinal (12 month)	3,020±94	3,054±63	0.5
Entorhinal (Annual % decrease)	3.6±1.7	2.1±1.0	0.03

of cerebral sulci). A second review (NU) reviewed all the images and either concurred or disagreed with the first reviewer's decision. For persons with MRI that resulted in disagreement, the matter was resolved by mutual discussion.

Each scan was analyzed for regional volumetric analysis. Briefly, images were first preprocessed by alignment to the AC-PC plane and removal of extra-cranial material. Brain tissue was segmented into grey matter, white matter, and cerebrospinal fluid, using brain tissue seg-

mentation. After high-dimensional image warping to a standardized brain atlas (template), regional volumetric maps, termed RAVENS maps were used to quantify the regional distribution of gray matter, white matter, and cerebrospinal fluid. The RAVENS approach [10,11] uses a highly conforming high-dimensional image warping algorithm and tissue-preserving transformations that captures finer structural details and preserves the amount of grey matter, white matter, and cerebrospinal fluid tissue present. A high dimension pattern classification approach identifies a minimal set of regions as follows: hippocampus, inferior lateral ventricle, middle temporal lobe, inferior temporal lobe, fusiform lobe, and entorhinal lobe. Total brain, intracranial, and ventricular volumes are quantified as well.

Statistical Analysis

Univariate analyses were performed comparing continuous variables with analysis of variance (ANOVA) and categorical variables using chi-square test between patients with and without dural thickening.

Results

We analyzed a total of 242 persons (mean age [±SD] 74±7 years: 150 were men) with either mild cognitive impairment or Alzheimer's disease. Dural thickening on MRI was seen in 83 (34%) of 242 persons. There was a trend toward higher proportion of women among persons with dural thickening compared with those without dural thickening. The proportion of various cardiovascular risk factors and depression were similar between the two groups. There were no differences in the APO E4 gene polymorphism between persons with and without dural thickening.

The mini mental score was not different in persons with (26±0.3) and without (26±0.2) dural thickening (p = 0.6). The proportion of patients with moderate or severe cognitive impairment (defined by mini mental status) was similar at baseline at 12-month evaluations. There was also no difference in the rate of annual decrease in mini mental status between the two groups. There was no difference in the proportion of patients with possible or definite cognitive deficits on Alzheimer's disease assessment scale at baseline (64 of 83 vs. 125 of 159, p = 0.6). The rates of annual progression according to Alzheimer's disease assessment scale (p = 0.06) and clinical dementia scale (p = 0.001) were higher in persons without dural thickening. The annual rate of volume loss in the entorhinal cortex was higher among persons with dural thickening.

Discussion

Dural thickening on MRI was observed in one-third of the persons with mild cognitive impairment or Alzheimer's disease. There was higher proportion of women in persons who had dural thickening. There were no prominent differences between persons with and without dural thickening in regards to cardiovascular risk factors, APE e-4 genotype, depression, and cognitive deficits at baseline. At 12 months, the rate of annual progression of cognitive deficits was lower according to Alzheimer's disease assessment scale and clinical dementia scale among persons with dural thickening. Because of the close proximity of inner layer dura mater and outer arachnoid layers[2], we cannot differentiate on the basis of MRI whether the changes involved the dura mater or arachnoid layers or both. Previous studies have observed changes in the dura and arachnoid layers in association with various diseases that result in cognitive decline. In a study of frontal leptomeningeal and brain biopsies in 27 patients with normal pressure hydrocephalus, degenerative cerebral changes, most often Alzheimer (6 cases) or vascular changes (7 cases) were detected in 14 biopsies. Arachnoid fibrosis was found in nine of the 18 biopsies containing arachnoid tissue. A tendency toward higher improvement rates after shunt placement was noted in the subgroups presenting with arachnoid fibrosis [3]. In another report, the same group reported that meningeal fibrosis involving the arachnoid tissue with was found in 12 of 25 biopsies derived from patients with Alzheimer's disease (10 cases, vascular dementia (10 cases), and others (n = 5)[5]. No correlation with disease severity was noted.

Localized or diffuse thickening of the dura mater may be seen in hypertrophic pachymeningitis that may be associated with rheumatoid arthritis, syphilis, Wegener's granulomatosis, tuberculosis, and cancer [22–26]. An idiopathic variant of pachymenningitis has been reported which presents with headaches, ataxia, and cranial nerve palsies [22–24,27]. Biopsies of dura demonstrate infiltrates of small mature lymphocytes, plasma cells, and epithelioid histiocytes, but no evidence of neoplasia, vasculitis, or infectious agents. Corticosteroid therapy results in improvement of clinical symptoms. Infrequently, necrotizing vasculitis of the small arteries located in the dura and the cortical surface or dural venous thrombosis may be seen [22,28]. Changes in dural thickness have been reported in conjunction with changes in intracranial pressure [29,30].

Dural thickening appears as hypointense area with fine hyperintense edges on T2-weighted images and hyperin-

tense area on coronal T1-weighted image [13,15,16,14]. Dural thickening appears hyperintense on FLAIR MR imaging and provides the best discrimination from CSF [31,32]. Contrast-enhanced MRI is also another method to identify dural thickening secondary to neoplastic dural invasion [33]. Contrast enhancement may be seen in the presence of inflammatory or proliferative components [34,35]. We did not have contrast-enhanced images available for our review in the current study population. Previous comparison between fluid-attenuated inversion-recovery (FLAIR) and contrast-enhanced T1-weighted images have shown similar results for identifying thickening of the dura mater associated with meningiomas [31], diffuse pachymeningeal thickening with intracranial hypotension [32], and leptomeningeal metastases [36]. Ahmadi et al.[37,38] and Wilms et al. [39] found that neoplastic infiltration of the dura resulted in loss of enhancement in dura on MRI based on validation by histopathological assessment. The thickened-enhanced portion of the dura represented reactive changes. Therefore, the dural thickening may be underestimated with contrast enhancement in the absence of inflammatory changes.

In conclusion, we found a relatively high prevalence of dural thickening in patients with mild cognitive impairment and Alzheimer's disease. Future studies would have to determine the underlying etiology for and whether there are any clinical correlates of such dural thickening.

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