Published in final edited form as:

Eur Radiol. 2013 August ; 23(8): 2252–2257. doi:10.1007/s00330-013-2818-x.

Association between linear measurements of corpus callosum and gait in the elderly

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Abstract

Purpose—Segmentation and diffusion-tensor-imaging of the corpus callosum (CC) have been linked to gait impairment. However, such measurements are impracticable in clinical routine. The purpose of this study was to evaluate the association between simple linear measurements of CC thickness with gait.

Methods—Two hundred and seventy-two community-dwelling subjects underwent neurological assessment and brain MRI. Mid-sagittal reformats of T1-weighted images were used to determine CC thickness. The association of measurements with clinical evaluation of gait was assessed by multivariate regression, controlling for numerous clinical and imaging confounders. Differences in CC thickness were, moreover, compared between subgroups with no, moderate or severe impairment of gait.

Results—In univariate analyses, thickness of the genu and body of CC but not the splenium were associated with postural stability ($P<0.01$). Multivariate regression revealed thickness of CC genu as the only imaging variable independently associated with gait ($P=0.01$). Genu thickness was significantly different between subjects with high and low ($P=0.0003$) or high and moderate ($P=0.001$) risk of fall.

Conclusion—Atrophy of the CC genu is an imaging marker of gait impairment in the elderly suggesting higher risk of fall. Simple linear measurements of CC can help in MRI evaluation of patients with gait impairment.

Keywords

Corpus callosum; Gait; Magnetic resonance imaging

Introduction

Gait impairment is common in the elderly affecting up to 15 % of people at the age of 60 and more than 80 % at the age of 85 [1]. As many as 30 % of people age 65 and older fall each year, and such falls are frequently associated with fractures, hospitalisation, loss of independence or death. Moreover, gait impairment in the elderly is a known predictor for future cardiovascular disease and dementia [2]. Determination of clinical as well as imaging correlates of gait is therefore an important public health issue.

Control of gait is a highly complex process and depends on integration of multiple neural systems that include: spinal or brainstem locomotor pattern generators, the basal ganglia, cerebellar modulation and integrated motor planning by the cortex [3]. Gait disorders in the elderly have often been attributed to combinations of cortical atrophy and white matter degeneration and may be attributed to small vessel ischaemic disease or stroke [4, 5]. While regional cortical atrophy is often a challenge to appreciate on imaging studies, white matter degeneration is more easily seen and frequently identified on magnetic resonance imaging (MRI) of the head as scattered fluid attenuated inversion recovery (FLAIR) or T2 hyperintense signal abnormality. Previous MRI studies have linked the degree of apparent white matter hyperintensities (WMH) and stroke-related encephalomalacia to postural instability [4, 6].

The corpus callosum (CC) is the largest interconnecting fibre tract in the brain. Its atrophy may be seen correlated with cortical atrophy, diffuse white matter degeneration and stroke, and thus reflects disruption of gait regulation at multiple levels [7, 8]. Using conventional MRI, regional atrophy of the CC as assessed by computer-assisted segmentation has previously been linked to gait impairment [9]. Likewise, diffusion tensor imaging (DTI) of callosal white matter integrity was found to correlate with gait measures in the elderly [10]. However, these techniques are complex and difficult to incorporate in clinical routine. In a small series of 30 highly selected patients, linear measurement of callosal thickness has been shown to correlate with gait impairment [11]. These findings suggest the possibility of convenient extraction of gait-relevant information from routine brain imaging studies. The purpose of this study was to evaluate the association between simple linear measurements of CC thickness with gait in the elderly.

Methods

Subjects

The study population consisted of 272 community-dwelling participants in the Nutrition, Aging and Memory in Elders (NAME) study. The study design, including the inclusion and exclusion criteria and clinical assessment, have been previously described in detail [12]. Briefly, a subset of 337 subjects from a total of 1,246 subjects recruited from three Aging Services Access Points (ASAPs) underwent detailed psychiatric and neurological as well as successful MRI examination in addition to the routine nutritional, neuropsychological, medical–historical and blood chemistry evaluations. The ASAPs provide services and support for independent living elders who are at least 60 years old and have needs in critical areas such as food or personal care. The Institutional Review Board approved the study, and all participants signed informed consent. Of the 337 subjects with MRI, 272 with available standardised gait scores were included in the study.

MR and image processing

All subjects underwent magnetic resonance imaging (MRI) at 1.5-T (Siemens Symphony, Forchheim, Germany), using an eight-channel head coil. The MRI protocol included coronal 3D magnetisation prepared rapid gradient echo (MPRAGE) sequences with 1×1 mm in-plane resolution (matrix 256×256; FOV 260 mm) and 120–150 slices of 1.5 mm thickness. Imaging parameters were TR 2,190 ms; TE 4.38 ms; TI 1,100 ms; flip angle 15°.

Measurements of the CC were performed on midline sagittal reformats on a clinical PACS workstation. Regional atrophy of the CC was determined by linear thickness measurements of genu, splenium, anterior, mid and posterior body thickness of CC as described by Moretti et al. [11]. Thereby, the principal axis of the CC was initially obtained as the line extending from the most anterior and most posterior margins of the CC in the mid sagittal MPRAGE image. The mid body of the CC was identified and a perpendicular was drawn to the principal axis to intersect the mid body of the CC (Fig. 1a). The maximum thickness of genu and splenium was measured allowing the curved margins of the CC (Fig. 1b). The anterior body thickness of the CC was measured at a point midway between the genu and mid body of CC, and the posterior body thickness of the CC was measured at a point midway between

the splenium and mid body of the CC (Fig. 1b). All measurements of the CC were performed by a board-certified neuroradiologist (2 years of experience). For assessment of inter-observer variability, a second board-certified neuroradiologist re-performed measurements on the first 50 subjects (20 years of experience). Before the study, both readers jointly trained linear measurements of CC thickness on ten routine head MR examinations.

Using conventional T2 and proton density-weighted images, intracranial white matter hyperintensities (WMH) and ventricular volumes were assessed by the quantitative histogram analysis method described by DeCarli et al. [13]. For further analysis, both WMH and ventricular volumes were normalised according to overall intracranial volumes.

Health, psychiatric and neurological assessment

Extensive demographic and laboratory data were collected for each study subject [12]. Participants responded to questions documenting the presence of abnormalities from a list of chronic conditions and health events. Diabetes was defined by the use of anti-diabetic medication or fasting glucose >126 mg/dl. Hypertension was defined by self-report with a systolic pressure >140 mm Hg or diastolic pressure >90 mm Hg, or use of anti-hypertension medication. Arthritis was defined as report of a physician's diagnosis of arthritis.

A board-certified psychiatrist evaluated the subjects and recorded the Hamilton Rating Scale for depression and Mini-Mental Status Examination (MMSE) scores (0–30) for cognitive function [14, 15].

A neurological evaluation was performed by a board-certified neurologist. The neurologist judged whether the subject had clinical evidence of symptomatic stroke(s), peripheral neuropathy or other neurological syndrome. Strokes were classified according to the Trial of Org 10172 in Acute Stroke Treatment criteria [16].

Gait assessment and categorisation of Tinetti scores

Each participant's gait and balance were assessed during neurological examination including a standardised total Tinetti score [17]. The Tinetti Balance and Gait evaluation is designed to assess both balance and gait using a simple “bedside” clinical method that has a maximum total score of 28 (16 points for best balance; 12 points for best gait). The balance examination consists of three broad performance outcomes: 1, sitting and rising (0–5 points); 2, standing and turning (0–9 points); 3, sitting down (0–2 points). Gait is evaluated in the categories of: 1, initiation (0–1 points); 2, step morphology such as length, height, symmetry and continuity (0–6 points); 3, gait trajectory such as path, truncal sway or flexion and walking stance (0–5 points). Based on Tinetti scores, subjects were sub-divided into groups with low (< 25), moderate (19–24) and high (<19) risk of falls [18].

Statistical analysis

Statistical analysis was performed with software (JMP version 6, SAS Institute, Cary, NC; Prism version 4.00, GraphPad Software, San Diego, CA). A *P* value of less than 0.05 indicated statistical significance. Quantitative variables were expressed as means±standard

deviations; categorical variables as frequencies or percentages. The relationship between the total Tinetti score and linear measurements of the genu, splenium and body of the CC was assessed in terms a univariate and multivariate logistic regression, including the following confounders: age, sex, diabetes, arthritis, MMSE score, clinical diagnosis of stroke, depression and neuropathy, as well as normalised volumes of WMH and the ventricles. In case of skewed distribution, logarithmic transformation of x -variables was used ($\log_{10}$). Co-linearity was ruled out by calculating the variance inflation factors.

Genu thickness was further compared between subgroups of subjects with either low, medium and high risk of falls. Similarly, Tinetti scores were compared between the highest, mid and lowest tertiles of genu thickness. These comparisons were performed by one-way ANOVA with Tukey's multiple comparison test. Interobserver reliability was assessed by calculating the two-way random single-measure intraclass correlation coefficient (ICC).

Results

The demographic, clinical and imaging characteristics of study subjects are shown in Table 1. ICCs for linear measurements of the genu, splenium and anterior or posterior aspects of the body of the CC were 0.96, 0.84, 0.86 or 0.73, respectively.

The results of univariate analyses among subject demographic, clinical and imaging characteristics, and Tinetti gait scores are shown in Table 2. Gait scores demonstrated a significant association with age, MMSE score, stroke, neuropathy, WMH volume, ventricular volume, CC genu and anterior, mid or posterior portion of the CC. There was no association between gait scores and other characteristics such as sex, arthritis, or depression. Likewise, there was no correlation between CC splenium and gait scores.

In a multivariate logistic regression considering the effects of genu, anterior, mid or posterior body and splenium thickness, as well as normalised WMH and ventricular volume, age, sex, diabetes, arthritis, scores in MMSE, diagnosis of stroke, depression and neuropathy, the only factors with independent impact on gait were linear measurement of the CC genu, stroke and neuropathy (Table 3).

Differences in genu thickness among subjects with low (>25), moderate (19–24) and high (<19) risk of fall as determined by the Tinetti scores are shown in Fig. 2. Genu thickness was significantly different between those with low risk of fall compared with high or moderate risk of falls.

Similarly, Tinetti gait scores were significantly different among highest, middle and lowest tertiles of the CC genu thickness with most pronounced effect seen when highest tertiles of genu was compared with the lowest tertile (Fig. 3).

Discussion

The results of this cross-sectional prospective, population-based study of the elderly show that atrophy of the genu of CC significantly correlates with clinical quantification of gait. According to a comprehensive multivariate regression, this effect is independent of other

known imaging correlates of gait, most notably WMH and ventricular volume. While stroke and neuropathy are other important clinical markers of gait impairment, genu thickness remains an independent imaging marker in a multivariate model. Genu thickness is also significantly different between subgroups of patients that were clinically classified using previously described cut-offs of Tinetti scores into low, moderate and high risk of fall. Interestingly, regional thicknesses of the splenium and body of the CC were not independently associated with abnormality of gait.

While control of gait is a multifactorial and highly integrated activity, studies from acallosal mice and split brain patients suggest a central role for the CC in neural circuits of locomotion [19–21]. Hence, its atrophy may be seen as a measureable endpoint of malfunction at various higher levels of gait control. Our finding of independent association between regional callosal atrophy and gait disorder is in line with results from previous MRI studies on motor dys-function and structural abnormalities of the CC. In a large series of 596 elderly subjects with WMH on MRI, Ryberg et al. [9] found a significant correlation between regional decrease in CC area (determined by computer-assisted segmentation) and subjective gait difficulty, objectively graded motor performance and walking speed. Using DTI technique to determine white matter integrity in the CC of 173 non-selected elderly, Bhadelia et al. [10] could demonstrate significant correlation between white matter tract disruption in the callosal genu and quantitative measurements of gait. In our study, a multivariate regression model was used to account for a large number of possible confounders. Most notably, the impact of genu thickness was proved independent of WMH, ventricular size, MMSE, history of stroke and neuropathy. This is important as all of these conditions have previously been associated with gait abnormality [6, 7, 22, 23]. Altogether, our data and previous findings by Ryberg et al. and Bhadelia et al. imply that the pathophysiology reflected in regional callosal atrophy may be fairly specific for gait disorders rather than merely reflecting the ageing brain.

In this context, it is interesting to note that only atrophy of the genu and not the body or splenium was independently correlated with gait disorder. Again, this finding supports previous observations and is consistent with the presumptive existence of a gait control centre involving frontal lobes and anterior intercortical white matter connections. In fact, walking disorders are well documented in patients with frontal lobe pathology. Likewise, elderly patients with manifest gait impairment often find it difficult to perform multiple frontal lobe-based tasks, especially walking and talking, likely due to poor functional reserve in the frontal lobe cortex [24]. From autopsy studies, it is well known that connections between the left and right frontal lobes, especially the pre-frontal and anterior frontal cortices, are dominant in the genu, whereas body and splenium primarily contain connecting fibres from somatosensory or auditory and temporal, parietal and occipital cortices [25]. Our data support the argument that disruption of the bi-frontal connections causes compromise of normal walking [26].

The finding of independent correlation between gait and simple linear measurement of the callosal genu has important implications for interpretations of MRI scans in subjects with gait impairment. Compared with computer-assisted segmentation of the callosal area and determination of anisotropy from DTI, which both require offline analysis, straightforward

measurement of genu thickness can be performed on a clinical PACS workstation, and can be included easily in everyday routine. Ultimately, our data support previous results by Moretti et al. [11], who likewise assessed atrophy of the CC in terms of its thickness. However, while observations in that study were based on only 30 selected patients with known gait disorder, our findings confirm a simple clinico-anatomic correlation in a larger and non-selected population-based cohort. Although linear measurement of the genu cannot replace a thorough clinical evaluation, it is a readily available tool in the routine analysis of MRI brain studies to bring attention to patients who may be at risk for falling.

A potential limitation of our analysis lies with the inclusion criteria of the NAME study. Since, only subjects living independently at home were enrolled, our findings may be subject to selection bias towards healthier elderly, hence reducing the strength of the observed effects. Nevertheless our results support the premise that a routine study can provide an indicator of the risk of falling even in this relatively healthy subject population.

In conclusion, data from this large population-based cohort of elderly demonstrate a significant clinico-anatomical association between simple linear measurement of the callosal genu and clinical assessment of gait. The correlation is independent of various important confounders and confirms a central role of the genu in the fronto-subcortical centre of gait control. While the assessment of elderly patients with gait impairment remains a complex inter-specialty challenge, our findings provide a simple tool for gait evaluation to the neuroradiologist reading MR scans in a wide variety of patients.

Acknowledgments

This work was supported by a grant from National Institute on Aging (NIA: AG21790-01).

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Key Points

- Regional atrophy of the corpus callosum reflects disruption of gait regulation
- Genu thickness on cranial MRI is an independent marker of gait impairment
- Findings help in the MRI evaluation of patients with gait impairment

Fig. 1.

Mid sagittal MPRAGE images showing linear measurements of corpus callosum (CC) thickness. Standardisation of measurements was achieved by extending a line from the most anterior to the most posterior margin of the CC and obtaining a perpendicular to this principal axis, intersecting the mid body of the CC (**a**). Thickness of genu and splenium was assessed along the principal axis, allowing for curved margins of CC (**b**). The anterior or posterior body thickness of the CC was measured at points midway between genu or splenium and mid body of the CC (**b**)

Fig. 2.

Genu thickness in different subgroups of patients with fall risk determined clinically using Tinetti scores. Linear measurements of the genu of CC were significantly different between patients with low and moderate ($P=0.0012$), and low and high risk of falls ($P=0.0003$)

Fig. 3.

Total Tinetti scores at lowest, middle and highest tertial of genu thickness. Gaits scores were significantly different between the lowest and middle ($P=0.037$), lowest and highest ($P=0.0006$) or middle and highest tertile ($P=0.046$)

Table 1

Demographic, clinical and imaging characteristics of subjects

Age, years	72.7±7.9
Women, <i>n</i> (%)	199 (73.1)
Hypertension, <i>n</i> (%)	223 (81.9)
Diabetes, <i>n</i> (%)	84 (30.8)
Arthritis, <i>n</i> (%)	202 (74.2)
MMSE, score 0-30	25.5±3.2
Depression diagnosis by psychiatrist, <i>n</i> (%)	92 (33.8)
Stroke diagnosis by neurologist, <i>n</i> (%)	47 (17.2)
Neuropathy diagnosis by neurologist, <i>n</i> (%)	101 (37.1)
Tinetti score (0-28)	24.2±4.8
Low risk of fall (≥ 25), <i>n</i> (%)	66 (24)
Medium risk of fall (19-24), <i>n</i> (%)	135 (50)
High risk of fall (<19), <i>n</i> (%)	71 (26)
Ventricular volume corrected for ICV	0.035±0.01
WMH volume corrected for ICV	0.0032±0.0032
CC thickness, cm	
Genu	0.83±0.2
Splenum	1.3±0.2
Anterior body CC	0.38±0.1
Mid body CC	0.43±0.1
Posterior body CC	0.36±0.1
CC genu thickness tertiles, cm	
Highest	1.10±0.09
Middle	0.83±0.07
Lowest	0.58±0.09

Data are presented as mean±standard deviation for continuous or ordinal variables and as number of subjects affected (percent) for categorical variables

MMSE Mini Mental Status Examination, *ICV* intracranial volume and *WMH* white matter hyperintensities

Table 2

Results of univariate regression of clinical and imaging characteristics against Tinetti Scale of gait

Variable	<i>P</i> value
Age	0.0005
Sex	0.20
Diabetes	0.63
Arthritis	0.40
MMSE score	0.005
Depression	0.52
Stroke	0.0001
Neuropathy	0.0025
Log WMH	<0.0001
Ventricular volume	<0.0001
CC genu	<0.0001
CC splenium	0.09
CC anterior	0.003
CC mid	0.003
CC posterior	0.009

CC corpus callosum, *WMH* white matter hyperintensities, *MMSE* Mini Mental Status Examination

Table 3

Results of multivariate regression of clinical and imaging characteristics against Tinetti Scale of gait

Variable	<i>P</i> value
Age	0.71
Sex	0.44
Diabetes	0.20
Arthritis	0.96
MMSE score	0.21
Depression	0.19
Stroke	0.0003
Neuropathy	0.0008
Log WMH	0.06
Ventricular volume	0.07
CC genu	0.01
CC splenium	0.79
CC anterior	0.60
CC mid	0.65
CC posterior	0.55

CC corpus callosum, *WMH* white matter hyperintensities, *MMSE* Mini Mental Status Examination