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J Geriatr Psychiatry Neurol. Author manuscript; available in PMC 2013 June 01.

Published in final edited form as:
J Geriatr Psychiatry Neurol. 2012 June ; 25(2): 107–112. doi:10.1177/0891988712445097.

Differential Reports of Pain and Depression Differentiate Mild Cognitive Impairment From Cognitively Intact Elderly Participants

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Abstract

Background—Positive associations between pain and depression in the general population have been well characterized; however, the interplay between pain, depression, and early cognitive decline, characterized as mild cognitive impairment (MCI), is poorly understood.

Methods—The current study examined the association of self-reported pain complaints (measured by the 36-item Short Form Health Survey) and self-reported depressive symptoms (measured by the 30-item Geriatric Depression Scale) in cognitively intact participants (n = 492) and participants with a clinical diagnosis of MCI (n = 83).

Results—Depressive symptoms and subjective reports of pain were significantly associated in the entire sample ($r = .29$; $P < .0001$). Multiple logistic regression modeling (adjusted for age, education, and APOE4 status as covariates) demonstrated that while depressive symptoms were positively associated with the diagnosis of MCI ($P < .001$), subjective pain reports were negatively associated with MCI ($P < .002$).

Conclusion—While the negative association of subjective pain complaints with MCI might arguably be explained by the development of anosognosia, self-reports of depressive symptoms were actually increased in these participants, suggesting preserved insight into cognitive decline–associated symptoms. It is possible that preferential involvement of limbic circuitry in MCI could explain these findings. Future studies are needed to elucidate the reasons for the dissociation of pain and depressive symptoms in MCI described in the present article.

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Declaration of Conflicting Interests
The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Keywords

aging; pain; depression; mild cognitive impairment

Introduction

The interplay among pain, depression, and mild cognitive impairment (MCI) in older adults is not well understood. Research indicates that pain and depression often co-occur.[1] Older adults reporting higher levels of pain also report higher levels of depressive symptomatology, suggesting a positive correlation between such symptoms.[2,3] Both pain[4] and depression[5,6] have been independently associated with cognitive impairment. While many studies have examined the relationship between pain and depression, pain and cognitive impairment, or depression and cognitive impairment, only limited data are available regarding the interrelationship among all 3 factors.[7] As such, the questions of ascertainment bias and cohort effects represent significant confounds to the understanding of this complex relationship.

Findings of reduced pain reporting in MCI have been described by a number of groups, although the mechanism for reduced pain reporting in MCI has not been elucidated.[2,4,8–10] While it is possible that older adults with MCI may simply have more difficulty reporting their pain, Parmalee, Smith, and Katz demonstrated that cognitively impaired older adults were able to accurately report pain in their cohort.[4] Kunz, Mylius, Schepelmann, and Lautenbacher reported that their participants with MCI appear to experience pain to the same extent as unimpaired participants using objective measures.[11] More recently, Wheeler demonstrated that this may not be the case in all cohorts and further argued that assessing pain in individuals with MCI may require repeating pain detection questions or applying a pain scale developed specifically for MCI participants. [12] Another study demonstrated that older adults with MCI are less likely to be assessed as experiencing pain by health services workers than are their cognitively normal counterparts, calling into question the issue of observer bias.[8] These studies, however, have not examined the potential role of depression in the subjective experience of pain in these cohorts

Depressive symptoms are well documented in early cognitive decline and MCI.[5,6,13–16] Affective symptomatology is well recognized to be part of the neurodegenerative process, even at this early stage. Indeed, depressive symptomatology may precede the development of cognitive decline by a decade or more.[15] Awareness of depressive symptoms in participants with MCI has not been appreciated as a major confound in the multitude of previous studies regarding this association across cohorts. Such studies have not, however, addressed the issue of pain complaints or the interrelationship with such depressive symptoms.

To the best of our knowledge, no research has specifically examined the interplay among self-reported pain, self-reported depressive symptoms, and cognitive status (normal or mildly cognitively impaired). Using data from an ongoing longitudinal study of aging and neurodegenerative disease at the University of Kentucky Alzheimer's Disease Center (UKADC), this study further explores the relationship among cognitive status and self-reports of both pain and depression.

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Methods

Participants

The UKADC longitudinal research program follows a target of 500 participants with normal cognition who undergo annual medical, neurological, and neuropsychological examination; and approximately 250 participants with impaired cognition, largely derived from transitions to MCI and dementia from this normal control cohort. The criteria for enrollment into the normal control cohort are listed in Table 1. In 2005, the UKADC began collecting data according to the protocol established by the National Alzheimer's Coordinating Center Uniform Data Set (UDS; http://www.ncbi.nlm.nih.gov/pubmed/17132964). Data used in this study are from the initial UDS evaluation and include 626 participants (538 with normal cognition and 88 diagnosed with MCI at that evaluation) who had completed both the UDS and a supplemental test battery that included the 36-item Short Form Health Survey (SF-36).[17,18] All participants were evaluated between the years 2005 and 2010. Because cancer pain is qualitatively different from other types of pain and leads to differential psychological and social outcomes, [19] participants reporting active cancer diagnoses (ie, cancer within 2 years of the initial UDS evaluation) were excluded (n = 2 MCI participants and n = 31 cognitively normal participants).

Cognitive Status

All diagnoses were derived by a consensus team consisting of the examining physician, a neuropsychologist, and the research assistant who performed the testing protocol and collected the data. The data available for review included current and longitudinal medical history, neuropsychological testing, magnetic resonance imaging (MRI), and informant interview data. The diagnosis of MCI followed the current consensus guidelines on MCI developed by the Second International Working Group on MCI[20]:

1. A cognitive complaint preferably corroborated by an informant or evidence for longitudinal decline on cognitive test performance.
2. Generally intact global cognition.
3. No or minimal functional impairment (insufficient to meet current diagnostic criteria for the *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition [*DSM-IV*] diagnosis of dementia).
4. Not demented by *DSM-IV* criteria.
5. No identifiable medical or psychiatric cause for cognitive decline including for the purposes of this study, excessive chronic or acute pain syndromes or analgesic use that might interfere with cognitive status. Such participants are diagnosed as "impaired other" at the UKADC in accordance with the NACC guidelines and were not included in the current study.

Self-Report of Depression

Depressive symptoms were reported via the 30-item Geriatric Depression Scale (GDS).[21,22] Designed specifically for use with older adults, the GDS is a well-validated measure for assessing depressive symptomatology in the older adult population.[23] The 30-item scale employs yes–no questions to determine the presence of depressive symptoms that might warrant further psychiatric attention, with a score of 0 to 10 being considered normal and scores above 10 suggesting depression.[21] The GDS has demonstrated usefulness in detecting depressive symptoms among older adults with cognitive impairment, particularly those with mild to moderate impairment,[24] although its sensitivity among those with dementia is

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doubtful.[25,26] Score on the GDS was coded as an ordinal variable with values ranging from 0 to 30 points.

Self-Report of Pain

Pain was reported through the Medical Outcomes Study[27] SF-36. The SF-36 is a multipurpose health survey that has been used in more than 4000 publications and information about its psychometric properties, reliability, validity, and normative data are available.[27,28] The SF-36 includes 2 scales, Physical and Mental Health. The Mental Health scale measures bodily pain with 2 questions about pain magnitude ("Have you experienced bodily pain in the last four weeks?") and pain interference ("Has bodily pain interfered with normal work in the last four weeks?"). The answers to these questions (a 5-level Likert response scale) were converted to a domain score ranging from 0 to 100, where lower scores represent less bodily pain.

Statistical Analysis

Exploratory data analyses included Spearman correlation, the Fisher Exact test, and Satterthwaite *t* test. Logistic regression was used to test the hypothesis that the interaction between self-reported pain and depressive symptoms are significantly associated with cognitive status (normal cognition vs MCI). Analyses were adjusted for age, sex, years of education, and Apolipoprotein E epsilon 4 allele (APOE4) status (at least one 4 present vs absent). Statistical analyses were performed using SAS 9.2.

Results

Data from all 626 participants with clinical diagnoses of cognitively intact or MCI, enrolled in the UKADC longitudinal research cohort, were analyzed. Thirty-three participants with cancer were excluded and an additional 18 participants were excluded because they did not complete the 30-item GDS, leaving 580 in the final analysis.

Participants with MCI were significantly older than those with normal cognition (80.0 vs 74.3 years, $P < .0001$), less likely to be female (53.0% vs 68.9%, $P < .0038$), reported more depressive symptoms (4.7 vs 3.2 average score on GDS, $P = .0099$), and reported less bodily pain (22.7 vs 28.9 average SF-36 pain score, $P = .019$). The MCI participants examined in the present study were relatively homogeneous, with 92.1% exhibiting a prominent amnestic deficit (67.9% had memory domain-specific deficits) and only 7.9% lacking an amnestic component. Table 2 contains demographic and clinical data on the research participants analyzed.

Pain and depression were positively correlated (Spearman $r = .29$, $P < .0001$) across all participants.

The associations between self-reported pain, self-reported depression, and cognitive status were studied in a multiple logistic regression model, controlling for age, sex, education, and APOE4. The results from this model are shown in Table 3. Because the interaction between pain and depression was not significant ($P = .25$), it was removed from the model. Pain and depression were each independently, albeit inversely, associated with the diagnosis of MCI. Specifically, each point increase on the GDS was associated with a 14% increase in odds of an MCI diagnosis (ie, odds ratio [OR] = 1.13; 95% confidence interval [CI] = 1.06–1.21), while higher levels of self-reported pain were associated with lower odds of MCI (OR = 0.98; 95% CI = 0.97–0.99).

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Discussion

Self-report of pain was positively associated with higher levels of depressive symptomatology in the overall sample. However, those with MCI reported more depressive symptoms, yet fewer pain symptoms than the cognitively intact control group. This finding calls into question speculation on whether a potential underreporting of pain in MCI is related to anosognosia, a simple lack of awareness of these symptoms. Recent research has indicated that persons with MCI may experience early signs of anosognosia,[29,30] although other studies have reported evidence of intact awareness of cognitive deficits in MCI.[31,32] If anosognosia and memory loss played a role in the reduced reporting of pain complaints in the present study, it is unclear why the present participants would be so keenly aware of their depressive symptoms.

A key issue central to this consideration is the potential heterogenity of MCI and the well-recognized prevalence of nonamnestic forms of MCI that may be less prone to anosognosia. [31,32] While such debate is valid in many samples with high prevalence of nonamnestic deficits, the current sample is enriched in amnestic forms of MCI (92%), representing a relatively homogeneous population of amnestic MCI participants. Removing the nonamnestic MCI participants from the analysis did not alter the statistical significance of the findings (data not shown). While anosognosia has been considered to be a sequelae of amnestic deficits, this contention has been challenged by recent work, suggesting that anosognosia is invariant in cognitive decline, irrespective of domain-specific deficits.[33] Thus, it is possible that the underreporting of pain in the present MCI participants could be due, at least partially, to nonamnestic cognitive domain involvement. The fact that anosognosia in this study was stimuli dependent (pain vs depression), rather than universal across stimuli, strongly argues for a selective deficit in nociceptive processing pathways rather than a general deficit in anosognosia.

The potential involvement of central nociceptive systems by early degenerative disease pathology in MCI needs to be considered as a plausible explanation for the lack of association between MCI, depression, and pain complaints in this study. Nociceptive inputs from the periphery rely on corticolimbic integration in the anterior cingulate to produce the pain experience that is directly linked to the hippocampus and other components of the Papez circuitry.[34,35] These components of the central nociceptive pathways have all been implicated in the early pathogenesis of AD. Understanding such circuitry is critical for the formation of testable hypotheses that might explain the current data regarding the dissociation of subjective pain complaints with MCI and depression. Further work examining specific pathological involvement of the corticolimbic circuitry is needed to better understand the dissociation of conscious perception of pain and depression seen in the present study.

As one of the first studies to examine the association of self-reported pain and depression with cognitive status among older adults, this study is not without limitations. Most participants were caucasian, highly educated, and of advanced age, and so the results may not generalize to disparate populations. Detailed data on many causes of chronic pain syndromes such as fibromyalgia, osteoarthritis, degenerative spinal disease and so on are not part of the routine acquisition of data from this cohort precluding analysis or adjustment for such variables in the data analysis. In addition, data on subjective experience of pain were derived from 2 items on a brief survey measure rather than on more informative gradient measures of pain experience. The use of 2 items to assess pain in this sample is an adequate approximation of the typical clinical experience, given the limited time physicians often have to assess their older adult patients, further supporting the need for practitioners to modify their pain assessment techniques among depressed older adults with MCI.

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The strengths of this study lie in the large sample size of cognitively, medically, and functionally well-characterized participants. The MCI participants were derived from the normal control cohort rather than a separate source allowing direct comparisons of pain and depression in relation to the independent variable of cognitive status and reducing the confounds of comorbid conditions that might differentially affect pain or depressive symptoms in the participants studied.

In summary, this report is the first to demonstrate a dissociation of subjective pain complaints and depression in early, pre-dementia cognitive impairment (MCI). This finding has direct implications for clinical care practices when dealing with such patients. Care should be exercised in evaluating pain in participants with MCI, recognizing that traditional methods such as self-report may be unreliable. Close attention should be paid to the development of comorbid depression in such disorders. Shifting the focus of pharmacological intervention away from treatment of chronic pain disorders, given the realization that perception of such disorders is minimized in this disease state and that many of the current treatment paradigms for pain management can further interfere with cognitive function, may be the most appropriate therapeutic course at this stage in the disease process. Focusing instead on enhancing cognitive status and treating the even mild symptoms of depression may prove far more effective in improving quality of life for those with MCI.

Acknowledgments

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This study was approved by the University of Kentucky IRB and supported by NIH/NIA 1 P30 AG028383.

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Table 1

Criteria for Enrollment into the Cognitively Intact Control Cohort

Inclusion Criteria	Exclusion Criteria
Minimum age 70	History of substance abuse (including alcohol)
Cognitively and neurologically normal	Major head injury
Family history of AD or dementia is preferred	Major psychiatric illness
Agree to brain donation at death to the UKADC	Medical illnesses that are nonstable, impairing, and/or have an effect on the Central nervous system
Live within a 2-hour drive of Lexington	Chronic infectious disease (HIV)
Must have a designated informant for structured interview (e.g., Clinical Dementia Rating Scale (CDR), Neuropsychiatric Inventory (NPI), Functional Assessment Questionnaire (FAQ))	Transient ischemic attack (TIA), stroke, or other significant cerebrovascular disease (subarachnoid hemorrhage, subdural hematoma, amyloid amgiopathy, ect)
Willing to undergo annual cognitive testing, physical, and neurological examination	History of encephalitis, meningitis, or epilepsy

Abbreviations: CNS, central nervous system; AD, Alzheimer disease; UKADC, University of Kentucky Alzheimer's Disease Center.

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Table 2

Characteristics of the Sample by Cognitive Diagnosis[a]

Variable	Total Sample (N = 575)	Normal Cognition (n = 492)	MCI (n = 83)	*P* Value
Age, years; mean (SD)	75.1 (7.7)	74.3 (7.4)	80.0 (7.7)	< .0001
Female gender, n (percent)	383 (66.6)	339 (68.9)	44 (53.0)	.0038
White race, n (percent)	532 (92.5)	453 (92.1)	79 (95.2)	.50
Education, years; mean (SD)	16.0 (2.8)	15.9 (2.8)	16.0 (2.8)	.73
APOE 4, at least 1 copy; n (percent)[b]	158 (29.3)	136 (29.3)	22 (29.3)	1.00
Geriatric Depression Scale, mean (SD)	3.4 (3.6)	3.2 (3.3)	4.7 (5.0)	.0099
SF-36 Bodily Pain, mean (SD)	28.0 (23.0)	28.9 (23.2)	22.7 (21.5)	.019

Abbreviations: MCI, mild cognitive impairment; SF-36, the 36-item Short Form Health Survey; SD, standard deviation.

[a]Means were compared using Satterthwaite *t* test, and proportions were compared using Fisher Exact test.

[b]Data on APOE 4 status were missing for 8 MCI participants and 28 normal participants.

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Table 3

Unadjusted and Adjusted Odds of Having the Diagnosis of MCI for Single Unit Increases in Pain and Depression[a]

Variable	Point Estimate	95% Confidence Interval	*P* Value
Pain			
Unadjusted	0.987	0.977–0.998	.0254
Adjusted	0.98	0.97–0.99	.0015
Depression			
Unadjusted	1.10	1.04–1.16	.0008
Adjusted	1.14	1.07–1.22	.0002

Abbreviation: MCI, mild cognitive impairment.

[a]Adjusted odds control for the effects of age, sex, education, APOE4, and pain or depression. Unadjusted odds are derived from models with no other predictors.