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Deficits in Different Cognitive Domains Predict the Progression of Amnestic Mild Cognitive Impairment: A Literature Review

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Abstract

Introduction: Mild cognitive impairment (MCI) is a heterogeneous syndrome in which older adults show cognitive deficits that do not interfere with daily living. Amnestic mild cognitive impairment (aMCI) is a subtype of MCI where episodic memory is significantly impaired and considered the transition stage between normal aging and Alzheimer's disease (AD). The cognitive profiles of individuals with aMCI may predict various trajectories and inform the risk of AD conversion. Given that cognitive domains beyond memory including language and executive functioning (EF) may contribute to the progression of aMCI, this paper will examine how deficits in these three domains can be used to predict trajectories of the syndrome.

Methods: PUBMED, EMBASE, and CINAHL databases were used to screen for studies to construct this systematic review. A total of 20 studies were reviewed.

Results: Functional changes in memory were observed including the diminished performance in the encoding and recognition phases of episodic memory, associative recall, rapid forgetting, and pattern separation in individuals with aMCI relative to age-matched peers. Overall EF and all three core EF components studied were impaired to similar extents. Dynamic working memory and verbal memory performance was impaired to a greater extent in individuals with multiple-domain aMCI (a more severe subtype of aMCI) relative to single-domain aMCI. Language impairments were associated with AD pathology, including verbal fluency deficits and semantic verbal fluency patterns, all of which were further impaired in individuals with multiple-domain aMCI relative to single-domain aMCI.

Discussion: Structural and functional changes in the medial temporal lobe (MTL) underlie various deficits in memory, EF, and language domains. Research suggests that individuals with single-domain aMCI perform more similarly with healthy controls, while those with multiple-domain aMCI perform more similarly with individuals with AD. This is likely attributed to cognitive domains beyond memory having a drastic impact on aMCI pathogenesis.

Conclusion: Preliminary evidence offers support for a more nuanced use of cognitive profiles to determine future outcomes and take appropriate clinical action earlier for improved prognosis and therapeutic plan development. Earlier formulation of treatment methods could drastically increase the likelihood of slowing or even reversing aMCI pathogenesis.

Keywords: amnestic mild cognitive impairment; mild cognitive impairment; Alzheimer's disease; dementia; memory; language; executive function; cognitive domains

Introduction

Mild cognitive impairment (MCI) is a heterogeneous syndrome in which older adults demonstrate cognitive deficits but preserved functional independence [1]. Individuals with MCI tend to perform worse on cognitive tests relative to healthy individuals of the same age range [1] and are at greater risk for developing dementia such as Alzheimer's disease (AD) compared to their healthy counterparts [2]. MCI has several subtypes classified by which cognitive domains are affected (i.e., memory affected or not) and if it is single-domain or multiple-domain. Single-domain MCI involves deficits in one cognitive domain, such as in either memory, language, or executive function (EF), whereas multiple-domain MCI involves deficits in multiple cognitive domains [2]. Amnestic mild cognitive impairment (aMCI), a subtype in which episodic memory is significantly impaired, requires some acknowledgement due to the importance of its different trajectories of neuropathological progression. Individuals are often clinically cited as having aMCI if (1) individual memory concerns are reported, (2) objective memory impairment for the individual's age relative to peers in the same age range, (3) relatively normal general cognitive function relative to age-matched peers, (4) otherwise normal activities of daily living, and (5) absence of dementia [3]. These individuals may have either isolated

memory impairments (single-domain aMCI) or may have impairments in non-memory cognitive domains as well. As such, aMCI is considered a transition stage between normal aging and AD [2]. Individuals with aMCI have several potential prognostic trajectories including stable, progressive decline to dementia, or slowing decline [4]. Cognitive profiles of these individuals with aMCI could potentially be informative in differentiating trajectories [4]. For example, individuals with a greater severity of aMCI-related deficits could yield an increase in likelihood for AD conversion. A review of the literature would help determine if these trajectories can accurately predict the prognosis and trajectory of these older adults with aMCI.

Although memory has been a primary focus for aMCI, several other domains such as EF and language may be involved. EFs refer to a family of top-down mental processes where reacting purely automatically or relying on instinct or intuition would be ill-advised or detrimental to well-being [5, 6]. The three core EFs include inhibition, working memory, and cognitive flexibility. Typical aMCI impairments are not due to external factors such as major psychiatric illness (i.e. depression, schizophrenia, bipolar illness), neurologic disorders (i.e. stroke, Parkinson's disease), use of acetylcholinesterase inhibitors or medications that significantly alter mood or arousal, depression, substance use, brain injury, or cardiovascular disease. Great disparity exists between how individuals are impacted by aMCI, potentially depending on which domains are affected and the number of domains that are impacted [5]. It is predicted that language and EF domains in conjunction with memory in the context of aMCI will paint a more comprehensive view of cognition, as well as imply certain pathogenic trajectories [6]. How these domains are differentially affected in such a heterogeneous definition of aMCI is currently unclear in the literature. The purpose of this review is to examine the extent to which deficits in different cognitive domains (i.e. memory, EF, language), contribute or predict the progression of aMCI over time.

Methods

This systematic review was constructed using information from three databases: PUBMED, EMBASE, and CINAHL. Keywords such as "mild cognitive impairment" OR "amnestic mild cognitive impairment" AND ("prediction/progression," "prognosis," "outcome") AND "executive function," OR "memory," OR "language" were used systematically. Exclusion criteria included preprints (i.e., from BioxRIV, MedxRIV, PsychAr) and articles not in English. Inclusion criteria included studies with a participant sample mean age range of at least 60 years, screening criteria for MCI diagnosis (i.e., Peterson's criteria)), and those that included an outcome measure in at least one cognitive domain in their study. Studies published in peer-reviewed journals after the year 2000 were preferred. This methodology yielded 653 articles in total, but for brevity the first 200 articles yielded by search terms were screened. After screening, 20 were included in the review. Primary research papers and quantitative data were preferred.

Results

Memory

In one study [7], three memory paradigms involving encoding and recognition tasks were performed during functional magnetic resonance imaging (fMRI). Participants were shown (1) common outdoor pictures, (2) pairing of faces and occupations, and (3) unrelated word pairs of objects and locations. Each paradigm consisted of six periods of encoding, control and recognition. Reaction time and accuracy of responses were recorded. Individuals with aMCI generally spent more time and performed less accurately than those without. Results of neuropsychological testing demonstrated poorer memory performance. Specifically, delayed recall and poorer learning efficiency yielded no differences in working memory but were demonstrated in individuals with aMCI. Compared to controls, individuals with aMCI showed decreased activations in the right medial temporal lobe (MTL) including the hippocampus, parahippocampal gyrus, fusiform gyrus, and left middle temporal gyrus during encoding. Decreased activation in the right parahippocampal gyrus was observed during recognition as well [7]. Therefore, individuals with aMCI show functional physiological changes (particularly in the left hippocampus) that translate to functional deficits in the encoding and recognition processes of episodic memory.

It was observed that item and associative memory performance in individuals with aMCI could be used for detection and predicting AD trajectories. Another study examined object-location recall and symbol-symbol recall in this population [8]. Item recall was impaired in individuals with aMCI compared to healthy controls. Associative recall in individuals with aMCI was impaired to an even greater extent than item recall compared to healthy controls ($p < 0.01$). The best discriminative factor between the two groups was associative recall, where sensitivity and specificity for detection were 75% and 90% respectively for symbol-symbol recall and were 85% and 97% respectively for object-location recall [8]. Associative memory is particularly sensitive to early cognition changes due to heavy dependencies on the MTL as observed with episodic memory. This was expected by the study because MTL structures are often the earliest affected in aMCI [7]. Consistent with aforementioned findings, evidence of rapid forgetting and compromised pattern separation (i.e., the specificity of episodic memory) was observed in individuals with aMCI, and these deficits were comparable to those with AD [9].

One study examined recognition memory performance among mild AD, aMCI, and healthy control groups. Participants saw everyday images presented and were instructed to categorize pictures as "old," "similar," or

"new" with differing lags between encoding and retrieval. Healthy controls had higher behavioral pattern separation (BPS) scores than individuals with AD but were comparable to those with aMCI. Individuals with aMCI also displayed lower BPS scores as the lag increased with more intervening distractor objects [9]. The longer the delay, the more individuals with aMCI mirrored individuals with AD in pattern separation performance. The study noted that, similar to past literature, episodic memory was observed to be compromised in individuals with aMCI

Executive Function

Overall impairment of core EF in individuals with aMCI appears to be of similar extent regardless of prior apparent clinical dysfunction [10]. In one study, individuals with aMCI were compared to healthy controls, and the sample of people with aMCI were further divided into EF-intact and EF-deficit groups according to neuropsychological assessments. All three core components of EF — working memory, inhibition, and task-switching — were studied based on the theoretical model of EF [6]. EFs were tested using a 2-back task and keep-track task for working memory, stop-signal task and Stroop task for inhibition, and the more odd-shifting task for task-switching [10]. Overall EF was significantly impaired in those with aMCI with moderate effect size, and the four tasks favored individuals without aMCI ($p < 0.01$). There was no significant difference in Stroop performance between the two groups. However, the EF-intact aMCI group exhibited significantly impaired ($p < 0.01$) performance in all tasks compared to the control group. Moreover, the overall EF and all three core EF components studied were significantly impaired in both EF-intact and EF-deficit aMCI groups [10]. The three core EF components were impaired to the same extent between the four tasks among the EF-intact and EF-deficit aMCI groups ($p < 0.01$).

Performance in dynamic working memory may also be informative in the progression to dementia such as AD. In another study, individuals with single-domain aMCI were compared to controls through working memory tests via the self-ordered pointing task (SOPT) and an n-back task of varying intervals. Evidence was found that individuals with aMCI made more errors and required longer times to develop an organizational strategy in the SOPT [11]. Individuals with single-domain aMCI did not differ significantly from healthy controls on the 1-back or 2-back tests but showed poorer discrimination than controls on the 3-back test ($p < 0.01$).

The level of EF among individuals with aMCI was found to influence verbal memory and predict the thickness of prefrontal and posterior cingulate areas [12]. In another study by Chang and colleagues [12], individuals with aMCI were divided into high (HEF) versus low EF groups (LEF), based on EF tests such as the Trail Making Test part A and B [13]. It was found that both HEF and LEF aMCI groups were significantly impaired on all memory measures relative to controls ($p < 0.01$) [12]. The aMCI HEF group demonstrated better verbal performance than the LEF group. However, neither aMCI group differed in terms of medial temporal morphometric measures [12]. The aMCI LEF group showed significant thinning in the dorsolateral prefrontal and posterior cingulate cortices bilaterally compared to aMCI HEF and healthy controls. Additionally, thickness in numerous regions of the frontal cortex and the bilateral posterior cingulate was significantly associated with memory performance in aMCI [12], demonstrating a structural pattern between individuals with aMCI that have greater EF impairment and verbal memory ability.

Language

In one study, healthy controls were compared to individuals with aMCI on generation of words starting with the letter "F" (phonetic fluency) and belonging to the category of *animals* (semantic fluency) [14]. Three measures were used: the number of words generated, measures of clustering (generation of contiguous words from within the same subcategory), and switching (shifting from one subcategory to another). Clustering was found to be compromised with temporal-lobe dysfunction, while switching was compromised with frontal-lobe dysfunction. There was also a progressive advantage of controls>aMCI>AD in semantic relative to phonemic fluency. Fluency patterns in the aMCI group also reflected degradation of semantic networks, demonstrating that the initial neuropathology may extend beyond early changes in hippocampal regions. This is further categorized by degradation of semantic relative to phonemic fluency performance when comparing those with aMCI to controls [14]. The study stated that semantic fluency is especially vulnerable in aMCI because it relies more on the association between exemplars of subordinate categories than does phonemic fluency. Early neuropathological changes in AD may thus extend beyond the hippocampus to include cortical areas supporting semantic memory. Patterns of disproportionately impaired semantic relative to phonemic fluency were found to be specific to AD, and potentially to aMCI [1].

Further research suggests similar semantic verbal fluency patterns in aMCI and AD, which may inform future trajectories. One study tested semantic and phonemic verbal fluency in AD, aMCI, non-amnestic MCI, and healthy control groups [1]. Semantic fluency was assessed by asking individuals to generate as many animals as possible within 60 seconds. The Controlled Oral Word Association Test [15] was used to assess phonemic fluency by asking individuals to generate as many words as possible beginning with the letters "F," "A," and "S", with 60 seconds per letter. Individuals with aMCI demonstrated disproportionately greater deficits in semantic versus phonemic fluency compared to controls. Verbal fluency patterns in the aMCI group were comparable to the AD

group [1]. When examining the relative difference between semantic versus phonemic fluency, this measure did not provide additional utility for predicting progression from aMCI to AD [1]. Relatively poorer performance on semantic verbal fluency tasks are suggested to be affected by disproportionately greater neurodegenerative changes in the temporal lobe, especially earlier in the course of the syndrome [16].

In another study, verbal fluency was studied between MCI subtypes (i.e., single- and multiple-domain aMCI, and non-amnestic MCI (naMCI) [17]. All participants were then administered the Delis-Kaplan Executive Functioning System letter and category verbal fluency subtests [18], which were then analyzed across two 30-second intervals for the total words produced, cluster size, and switching. Individuals with single-domain aMCI performed comparably with controls on each measure. Individuals with multiple-domain aMCI showed performance deficits in both total words and switching production compared with controls on both tasks. The naMCI group produced fewer words and switches on the letter fluency subtest. Each group produced more words and switches during the first 30 seconds except for the naMCI group whose switching on letter fluency did not decrease as the task progressed. Thus, verbal fluency performance decreases as domains other than memory become impaired in MCI. The greatest impairment in verbal fluency were from individuals with multiple-domain MCI and naMCI. This is consistent with findings that compromised EF contributes most to reduced switching ability [17].

Discussion

The MTL is often the earliest structure impacted in aMCI progression [7], and structural and functional changes in this region may underlie various memory deficits from the aforementioned studies. For example, decline in episodic memory can be mapped through decreased activation in the right MTL, which shows functional changes in individuals with aMCI in terms of encoding memory. Potential functional recognition deficits could allude to the recognition phase being impacted negatively as well; a finding further supported by associative memory being particularly sensitive early in aMCI progression [19]. Overall EF ability and each individual EF component studied were significantly impaired in individuals with aMCI, consistent with meta-analyses [19,20]. No significant differences were observed in the extent of impairment of each of the three EF components among individuals with aMCI, suggesting that the three core EF components are impaired to similar extents. EF deficits in aMCI were found to influence verbal memory and predict the thickness of prefrontal and posterior cingulate areas. Much research was also found supporting verbal fluency patterns associated with aMCI are indicative of an AD-type pathology, where early neuropathological changes displayed disproportionately impaired semantic relative to phonemic fluency [20]. Similar semantic verbal fluency patterns in aMCI and AD are present, which can be used to determine trajectories and the extent of conversion. However, there is much disparity between single and multiple-domain aMCI in verbal fluency ability [20]. The research suggests individuals with single-domain aMCI perform comparably with healthy controls across fluency tasks, while those with multiple-domain aMCI performed worse, suggesting that deficits in memory, language, and EF contribute and predict the progression of aMCI to dementia, in particular to AD. As follows, semantic verbal fluency patterns should be focused on determining future trajectories of individuals with aMCI or conversion to AD.

Overall, the literature is mostly homogenous. The studies came to similar conclusions that cognitive domains beyond memory in individuals with aMCI have a substantial impact in predicting trajectories of aMCI. All three of memory, EF and language appear to have some predictive value in predicting progression of aMCI to dementias such as AD. However, these cognitive domains seem to vary in predictive value. Memory in particular is very sensitive in the early stages of aMCI due to MTL changes, particularly in episodic memory and associative memory. Since the MTL is impacted early in the neuropathogenic progression of aMCI, the extent of changes in the MTL used in conjunction with clinical observances such as rapid forgetting and compromised pattern separation could be used to predict the speed of progression and the likelihood of conversion to AD or other dementias. Decreased activations of MTL including the hippocampus, parahippocampal gyrus, fusiform gyrus, and left middle temporal gyrus during encoding are particularly noticeable in many screening tests that involve neuroimaging. Individuals with aMCI could be screened for changes in thickness of the MTL as that may be indicative of future episodic memory and associative memory declines [19]. Semantic fluency could also be focused on for determining future trajectories of aMCI or conversion to AD. Relatively poorer performance on semantic verbal fluency tasks are suggested to be caused by disproportionate neurodegenerative changes in the temporal lobe, like how the memory domain is impacted.

The literature defined aMCI similarly, with most studies employing Peterson's criteria in screening. An aMCI diagnosis is useful because MCI is a vaguely defined syndrome with a heterogenous population. Screening for literature that focused on aMCI subtypes created a more homogeneous pool of studies that captured this construct and allowed for greater empirical support. Sample sizes of literature studied were strong, and results accounted for potential biases or confounding variables such as level of education. Testing for cognitive domains has been largely standardized over the past couple decades and the screening requirement to only use recent literature ensured that testing methods in studies were not outdated. A potential limitation

of the present paper is the small size of literature analyzed for the sake of brevity. A greater examination of the literature would likely yield more information pertaining to MCI progression and may offer more counter-arguments to acknowledge. Further examination of neuroimaging methods such as electroencephalography may bolster the predictive value of these measures.

Conclusions

In conclusion, this review summarizes how deficits in the memory, EF, and language cognitive domains can predict the trajectory and progression of aMCI to dementia such as AD. The results offer preliminary evidence in a more nuanced examination of cognitive profiles of aMCI. Furthermore, neuroimaging screening in individuals with aMCI may help determine future outcomes. Earlier screening could potentially allow for better prognosis and for therapeutic plans to be administered earlier in the progression of the disease to increase the chance of slowing progression or possibly reversing aMCI pathogenesis. These prevention efforts could lead to greatly improved quality of life for many individuals with this diagnosis.

List of Abbreviations Used

MCI: mild cognitive impairment
aMCI: amnestic mild cognitive impairment
naMCI: non-amnestic mild cognitive impairment
AD: Alzheimer's disease
EF: executive function
MTL: medial temporal lobe
fMRI: functional magnetic resonance imaging
BPS: behavioral pattern separation
SOPT: self-ordered pointing task
HEF: high executive function
LEF: low executive function

Conflicts of Interest

The author, Redwan Haque, declare that they have no conflict of interests.

Ethics Approval and/or Participant Consent

The study did not require ethics approval and/or participant consent because the study took the form of a literature review.

Authors' Contributions

RH: Conceptualized the design of the work; the collection, analysis and interpretation of the data; drafted the manuscript; gave final approval of the version to be published

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