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RESEARCH ARTICLE

Clinical factors predicting the rate of cognitive decline in a US memory clinic: An electronic health record study

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Abstract

INTRODUCTION: Dementia progression is heterogeneous and predicting who will decline quickly remains an open problem. Most work in this area has focused on volunteer-based cohorts, which are subject to recruitment biases. Instead, we examine predictors of rate of cognitive decline in cognitive assessment scores using electronic health record (EHR) data from a US memory clinic.

METHODS: Data include patients with their first memory clinic visit (baseline) between January 1, 2014 and May 31, 2024. We used a discrete-time model to identify significant predictors of baseline and 6 month change in Mini-Mental State Examination (MMSE) scores (Montreal Cognitive Assessment scores were converted to MMSE equivalents for analysis). Inverse probability weighting was used to account for selection and censoring biases and *p* values were adjusted for multiple comparisons.

RESULTS: The cohort included 9583 patients, of which 7113 had a baseline cognitive assessment. Average MMSE at baseline was 23.2. Variables associated with lower baseline MMSE included female sex, non-White race, Medicaid enrollment, baseline dementia diagnosis, and cholinesterase inhibitor prescription, while higher scores were associated with prior diagnoses of chronic pain or fatigue. Quicker post-baseline decline was associated with older age, dementia diagnoses, and cholinesterase inhibitor prescription, while slower decline was associated with a higher number of total prescriptions, distance from home to clinic, and Social Deprivation Index. Notably, rate of decline was not associated with mild cognitive impairment, other non-dementia cognitive impairment, or any of the comorbidities considered.

DISCUSSION: While several significant predictors were identified, the lack of associations with broad categories of comorbidities and social determinants of health suggest that finer grained predictors may be needed. Additionally, the finding that cholinesterase inhibitor prescriptions predicted quicker decline merits additional

investigation in real-world samples. The model developed in this work may serve as a first step toward an EHR-based progression risk tool.

KEYWORDS
dementia progression, electronic health records, memory care, prediction, time series

Highlights

- In a memory clinic setting, faster decline in Mini-Mental State Examination scores was associated with age, dementia diagnosis, and cholinesterase inhibitor or memantine prescription.
- Slower decline was associated with the patient's total number of prescriptions.
- Neither race nor ethnicity were associated with rate of decline, nor were baseline mild cognitive impairment, other non-dementia cognitive impairment, diabetes, hypertension, obesity, depression, anxiety, chronic pain/fatigue, or hearing loss.

1. INTRODUCTION

Characterizing the presentation, treatment, and progression of patients with dementia in the real world (i.e., beyond study volunteers) is essential for understanding dementia pathogenesis. Many open questions about dementia progression remain. Among the most important remaining open questions is why some patients progress more rapidly than others. While several notable longitudinal studies have contributed greatly to our understanding of dementia progression,[1–5] their scope is limited by their eligibility requirements, the nature of data collection, long time intervals between assessments, and reliance on survey data. Further, these studies often over-represent White and high socioeconomic status patients[6] and may not represent the full diversity of dementia patients both in terms of demographics and medical complexity.[7–11] Studies using data reflecting real patient populations are needed to assess the generalizability of existing study cohorts and identify risk factors they may have missed.

One of the richest and most readily available sources of real-world data (RWD) are electronic health records (EHRs). While EHRs have many of their own sources of bias,[12] they naturally reflect the makeup of real patient populations and allow us to examine existing care patterns in detail, making them an ideal complement to more controlled, volunteer-based longitudinal studies of dementia. Several notable EHR-based dementia studies have been conducted including studies pairing EHRs with data from volunteer-based cohorts,[13] epidemiological studies using centralized EHR repositories,[14] as well as studies using EHRs to develop new algorithms to identify study cohorts,[15,16] detect undiagnosed dementia,[17,18] or predict future dementia.[19–21] A limitation of these studies is their use of incident dementia as the primary measure of cognitive decline, treating cognitive decline as a discrete rather than continuous process.

In this study, we instead adopted the approach of Tschanz et al.[22] to examine predictors of a patient's *rate* of cognitive decline as measured by cognitive assessments using 10 years of EHR data from a US memory clinic. While memory clinic patients do not represent the whole population, they may be more representative than volunteer cohorts. Further, memory clinics capture more detailed information on cognition than is typical in non-specialty clinics. This study can be thought of as a middle ground between population-based and volunteer-only cohorts. We test associations between various demographic and clinical variables with baseline cognitive assessments and change in cognitive assessments over time.

1 | MATERIALS AND METHODS

1.1 | Study setting and population

This study was conducted using EHRs for patients of the Johns Hopkins Memory and Alzheimer's Treatment Center (JHMATC). JHMATC began operating in late 2008 as a multidisciplinary, memory-care specialty program. Over the years, ≈ 20 physicians with backgrounds in psychiatry, neurology, or geriatric medicine have provided care at JHMATC. The study population included patients who had their first JHMATC visit (baseline) between January 1, 2014, and May 31, 2024. The current Epic Systems EHR was not adopted across Johns Hopkins until mid-2013; thus, we included data from 2014 onward. This study was approved by the Johns Hopkins University Institutional Review Board (IRB00269466) and a waiver of consent was granted.

1.2 | Outcome

Our primary outcome was the patient's cognitive assessment score at baseline and every 6 months after baseline. Because patient visits do not fall at regular intervals, we used last observation carried forward to estimate cognitive scores at each discrete 6 month time point. We included all Mini-Mental State Examinations (MMSEs) and

Montreal Cognitive Assessments (MoCAs) administered by Johns Hopkins providers and documented in structured fields within the EHR. Additionally, we applied simple regular expressions to extract scores documented in JHMATC clinical notes, but not in structured data. If more than one score was recorded on the same day, then these scores were averaged. All MoCA scores were converted to their MMSE equivalent according to the mapping in Saczynski et al.[23] For brevity, we will refer only to MMSE scores throughout, but all analyses used both MMSE scores and converted MoCA scores.

1.3 | Predictor variables

1.3.1 | Demographics

We included patient age, sex, race, and Hispanic ethnicity from routine intake forms. We categorized race as White, Black, Asian, other, and unknown. The "other" category includes American Indian/Alaskan Native, Pacific Islander, Native Hawaiian, and multiracial patients and was created due to small numbers of patients in these categories.

1.3.2 | Cognitive impairment diagnoses

We further included diagnoses for dementia, mild cognitive impairment (MCI), and other cognitive impairment non-dementia (other CIND). Dementia diagnoses were categorized as Alzheimer's disease (AD) only (G30.XX), vascular only (F01.XX), other, and multiple. The "other" category included frontotemporal dementia (G31.0X), dementia with Lewy bodies (G31.83), and other and unspecified dementias (F02.XX, F03.XX, G23.1, and G31.1). Multiple dementia was defined as the presence of codes for at least two of AD, vascular, frontotemporal, and Lewy body dementia. Indicators for MCI (G31.84) and other CIND (I69.XX, R41.XX, or F09.XX) were positive only if no dementia indicators were positive. We did not include diagnoses for delirium or other cognitive diagnoses made primarily in in-patient settings.

1.3.3 | Comorbidities and medications

For each 6 month time point, we included the total number of chronic diagnoses from the Chronic Conditions Warehouse (July 2023 revision) documented in the preceding 3 years. Additionally, we included individual indicators for 3 year history of established dementia risk factors including hypertension, diabetes mellitus, obesity, depression, anxiety, chronic pain or fatigue, stroke or transient ischemic attack (TIA), and hearing impairment.[24] We included the total number of unique medications prescribed or documented by a Johns Hopkins provider in the prior year. The total number of medications was calculated as the total number of unique RxNorm ingredient codes among all qualifying prescriptions. We include all prescriptions in this period and did not try to distinguish active prescriptions. Additionally, we included an individual indicator for prior prescription of cholinesterase inhibitors or memantine (RxCUIs 135447, 4637, 183379, and 6719).

RESEARCH IN CONTEXT

1. **Systematic review**: We reviewed the literature using traditional sources and identified several studies predicting future Mini-Mental State Examination progression. However, all studies were volunteer based (mainly from Alzheimer's Disease Neuroimaging Initiative), and the large majority focused on imaging data.
2. **Interpretation**: Our work recapitulates findings related to rate or progression in several demographic groups, but challenges findings related to mild cognitive impairment (we found no difference in progression relative to unimpaired patients) and prescription of cholinesterase inhibitors (we found faster progression among prescribed patients).
3. **Future directions**: Additional study is needed to assess the degree to which our findings are affected by confounding and referral bias. This will likely entail more sophisticated modeling, including inverse probability of treatment weighting, and incorporating data from non–memory clinic patients. Simple models of progression may be inappropriate for a highly heterogeneous process, and additional work is needed to develop better models and identify new predictive factors.

1.3.4 | Social determinants of health

Finally, we included social determinants of health (SDoH) that may impact both the patient's health and their likelihood of referral to and follow-up at JHMATC. These SDoH included the Social Deprivation Index (SDI) developed by the Robert Graham Center[25] (0 to 100 with higher indicating more deprivation), enrolment in Medicaid or dual eligibility, distance in kilometers from the patient's home zip code to the JHMATC, and whether and how many Johns Hopkins primary care visits the patient had in the prior year. The SDI combines several measures of SDoH, measured at the census tract level, into a single score ranging from 0 to 100.

1.4 | Statistical analysis

To estimate associations between predictors and baseline MMSE, we used a linear regression model. All patients with an MMSE score taken at or in the 6 months before their first visit were included in this analysis. To estimate associations between predictor variables and MMSE progression, we used a linear regression model predicting the patient's MMSE score at each time step (beginning 6 months after baseline) given the patient's predictors and MMSE at the previous time step. Samples were pooled across all time steps and patients were considered censored 6 months after their final recorded assess-

ment. To account for possible ceiling and floor effects, a piecewise linear transformation was applied to the patient's prior score with knots every 5 points (i.e., 5, 10, 15). A similar transformation was applied to the distance to JHMATC with knots at 20 and 40 km. Standard errors were estimated using patient-level cluster bootstrapping (2000 bootstrap samples) to account for correlations between repeated measurements for the same patient. We report raw 95% confidence intervals as well as multiple comparisons corrected p values using the Benjamini–Hochberg procedure. As a secondary analysis, we stratified our progression analysis by cognitive diagnosis (unimpaired, MCI, other CIND, and dementia). Additionally, to test for measurement bias among patients receiving most of their care at Johns Hopkins, we repeated our progression analysis on patients who had a Johns Hopkins primary care visit in the year before baseline. All analyses were conducted in Python using the Scikit-learn package (v 1.5.1).

1.5 | Accounting for missingness and loss to follow-up

Some memory clinic patients may not receive cognitive testing at their first visit or may be lost to follow-up, leading to potential censoring bias. To account for this, we used inverse probability of censoring weighting.[26] We used logistic regression to model the probability of censoring and fit three separate censoring models: one for the probability of having an assessment at baseline, one for the probability of having a second assessment, and one for the probability of becoming censored after subsequent visits. Only the first model was used in the baseline model, while all three were used in the progression model. Specifically, let $\hat{p}_{0i}$ be the estimated probability that the patient i has a baseline measurement and $\hat{p}_{ti}$ be the estimated probability that the patient is uncensored at time t. Then patient i's weight in the baseline MMSE model is $w_{0i} = 1/\hat{p}_{0i}$ and the weight of the patient i at time t in the progression model is $w_{it} = 1/(\hat{p}_{0i}\hat{p}_{ti})$. We included in these models all predictors described above. We evaluated covariate balance after reweighting using absolute standardized difference.[27]

3. RESULTS

1.6 | Cohort description

A total of 9584 patients had their first visit during the study period, at which 7113 had a cognitive assessment. Of these, 2893 had a second assessment at least 6 months after baseline. A comparison of sample characteristics of these three groups is in Table S1 in supporting information. Patients with no baseline MMSE were more likely to be Black, be enrolled in Medicaid, and live farther from JHMATC. They also had a higher total comorbidity burden and had higher rates of diagnosed obesity, depression, anxiety, and chronic pain/fatigue. Among these patients with a baseline cognitive assessment (Table 1), the average age at baseline was 73.4 and 4115 (58%) patients were female. The racial makeup of the cohort comprised 5299 (75%) White patients, 1308 (18%) Black patients, and no more than 3.3% representation in any other racial or ethnic group. The majority of patients (3730; 52%) were diagnosed with dementia at baseline, with AD dementia the most diagnosed subtype (1543; 21.7%), followed by other/unspecified (1490; 20.9%) and vascular (394; 5.5%) dementias. An additional 1524 (21.4%) patients were diagnosed with MCI and 1704 (24.0%) were diagnosed with other CIND. A comparison of sample characteristics for patients who received only MMSE, only MoCA, or both assessments is in Table S2 in supporting information.

1.7 | Baseline cognitive assessments

Among patients with a baseline MMSE, the average score was 22.2 across all patients, 25.3 among patients with no baseline cognitive diagnosis, 25.7 among patients with MCI, 26.0 among patients with other CIND, and 18.8 among patients with a baseline dementia diagnosis. The distribution of baseline scores is in Figure 1A. Associations between the predictor variables and baseline assessment scores are in Table 2. After adjusting for multiple comparisons and censoring (diagnostics for the censoring model are in Figures S1–S3 in supporting information), lower baseline scores were significantly associated with female sex ($\beta = -1.13$ [−1.43 to −0.84] vs. males; $p < 0.001$) and age ($\beta = -0.07$ [−0.09 to −0.06] per year; $p < 0.001$), as well as Black ($\beta = -1.76$ [−2.15 to −1.37]; $p < 0.001$), Asian ($\beta = -1.25$ [−1.99 to −0.52]; $p < 0.001$), other ($\beta = -1.10$ [−1.82 to −0.39]; $p = 0.004$), and unknown race ($\beta = -2.24$ [−3.60 to −0.88]; $p = 0.002$) relative to White patients. Additionally, lower baseline scores were associated with diagnoses of AD ($\beta = -5.38$ [−6.26 to −4.51]; $p < 0.001$), vascular ($\beta = -4.54$ [−5.54 to −3.54]; $p < 0.001$), multiple ($\beta = -5.52$ [−6.59 to −4.45]; $p < 0.001$), and other ($\beta = -5.10$ [−5.96 to −4.25]; $p < 0.001$) dementias compared to unimpaired patients, while no significant associations were found with MCI or other CIND. Higher baseline scores were associated with chronic pain or fatigue ($\beta = 0.70$ [0.32 to 1.08]; $p < 0.001$) while lower baseline scores were associated with prescription of cholinesterase inhibitors or memantine ($\beta = -1.65$ [−2.02 to −1.29]; $p < 0.001$) and Medicaid enrollment ($\beta = -2.68$ [−3.48 to −1.88]; $p < 0.001$).

1.8 | Progression of cognitive assessments

A random sample of 100 cognitive score trajectories are plotted in Figure 1B and associations between predictor variables and change in MMSE are in Table 3. Coefficients are interpretable as the 6 month change in MMSE. After adjustment, faster average decline was significantly associated with older age ($\beta = -0.01$ [−0.01 to −0.00] per year age; $p < 0.001$), AD ($\beta = -0.82$ [−1.31 to −0.33]; $p = 0.001$), multiple ($\beta = -0.97$ [−1.48 to −0.45]; $p < 0.001$), and other ($\beta = -0.52$ [−1.00 to −0.04]; $p = 0.42$) dementias versus unimpaired, and prescription for cholinesterase inhibitors or memantine ($\beta = -0.14$ [−0.25 to −0.03] vs. no Rx; $p = 0.018$). Slower average decline was observed for patients with unknown race ($\beta = 0.97$ [0.10 to 1.83] vs. White; $p = 0.021$) and a higher number of prescribed medications ($\beta = 0.02$ [0.02 to 0.03] per medication; $p < 0.001$). The relationship between prior MMSE

TABLE 1 Patient characteristics at baseline.

		Baseline diagnosis			
	All	Normal	Other CIND	MCI	Dementia
N	7113	155	1704	1524	3730
Age	73.4 (10.0)	67.0 (12.0)	69.4 (10.9)	72.3 (8.7)	76.0 (9.2)
Female	4115 (57.9%)	90 (58.1%)	999 (58.6%)	815 (53.5%)	2211 (59.3%)
Race					
White	5299 (74.5%)	123 (79.4%)	1316 (77.2%)	1228 (80.6%)	2632 (70.6%)
Black	1308 (18.4%)	15 (9.7%)	248 (14.6%)	198 (13.0%)	847 (22.7%)
Asian	233 (3.3%)	<10	63 (3.7%)	50 (3.3%)	113 (3.0%)
Other	237 (3.3%)	<10	62 (3.6%)	44 (2.9%)	123 (3.3%)
Unknown	53 (0.7%)	<10	15 (0.9%)	<10	27 (0.7%)
Hispanic	138 (1.9%)	<10	37 (2.2%)	21 (1.4%)	79 (2.1%)
SDI score	37.1 (27.9)	37.6 (26.5)	37.4 (27.6)	33.4 (26.1)	38.5 (28.6)
Medicaid	667 (9.4%)	23 (14.8%)	163 (9.6%)	84 (5.5%)	397 (10.6%)
Distance to clinic (km)	94.1 (299.2)	77.3 (172.4)	101.5 (339.6)	100.0 (274.6)	89.1 (293.1)
Years followed	1.2 (1.8)	0.9 (1.7)	1.1 (1.8)	1.5 (2.1)	1.2 (1.7)
Primary care patient	1881 (26.4%)	15 (9.7%)	467 (27.4%)	404 (26.5%)	995 (26.7%)
Baseline dementia	3730 (52.4%)	–	–	–	3730 (100.0%)
AD	1543 (21.7%)	–	–	–	1543 (41.4%)
Vascular	394 (5.5%)	–	–	–	394 (10.6%)
Other	1490 (20.9%)	–	–	–	1490 (39.9%)
Multiple	303 (4.3%)	–	–	–	303 (8.1%)
Baseline CIND	3228 (45.4%)	–	1704 (100.0%)	1524 (100.0%)	–
MCI	1524 (21.4%)	–	0 (0.0%)	1524 (100.0%)	–
Other	1704 (24.0%)	–	1704 (100.0%)	0 (0.0%)	–
Baseline MMSE	22.2 (6.3)	25.3 (5.6)	26.0 (4.2)	25.7 (3.2)	18.8 (6.3)
Total comorbidities	5.4 (4.4)	4.0 (3.5)	5.6 (4.3)	5.4 (4.5)	5.3 (4.5)
Hypertension	4078 (60.3%)	62 (48.4%)	889 (57.3%)	835 (61.3%)	2292 (61.7%)
Diabetes	1449 (21.4%)	26 (20.3%)	309 (19.9%)	324 (23.8%)	790 (21.3%)
Obesity	739 (10.9%)	12 (9.4%)	214 (13.8%)	196 (14.4%)	317 (8.5%)
Hearing	1122 (16.6%)	< 10	281 (18.1%)	275 (20.2%)	559 (15.0%)
Anxiety	1886 (27.9%)	36 (28.1%)	540 (34.8%)	416 (30.5%)	894 (24.1%)
Depression	2411 (35.7%)	63 (49.2%)	675 (43.5%)	514 (37.7%)	1159 (31.2%)
Fatigue	1439 (21.3%)	25 (19.5%)	442 (28.5%)	347 (25.5%)	625 (16.8%)
Stroke	843 (12.5%)	<10	176 (11.3%)	149 (10.9%)	510 (13.7%)
Total medications	8.1 (5.7)	7.7 (5.4)	8.2 (5.9)	7.9 (5.7)	8.1 (5.7)
Cholinesterase inhibitors	2523 (39.4%)	22 (15.7%)	176 (12.3%)	307 (23.0%)	2018 (57.7%)

Note: Discrete variables are reported as "count (percent)" and continuous variables are reported as "average (std)." Counts < 10 are suppressed to protect patient privacy.

Abbreviations: AD, Alzheimer's disease; CIND, cognitive impairment non-dementia; MCI, mild cognitive impairment; MMSE, Mini-Mental State Examination; SDI, Social Deprivation Index.

and predicted MMSE is shown in Figure S4 in supporting information. Results from the progression model stratified by cognitive diagnosis are in Table S3 in supporting information and results when restricted to patients with prior Johns Hopkins primary care visits are in Table S4 in supporting information.

2 | DISCUSSION

In this paper, we used EHR data from a US memory clinic to identify predictors of baseline cognitive assessment scores and changes in these scores over time. In both cases, we found several significant

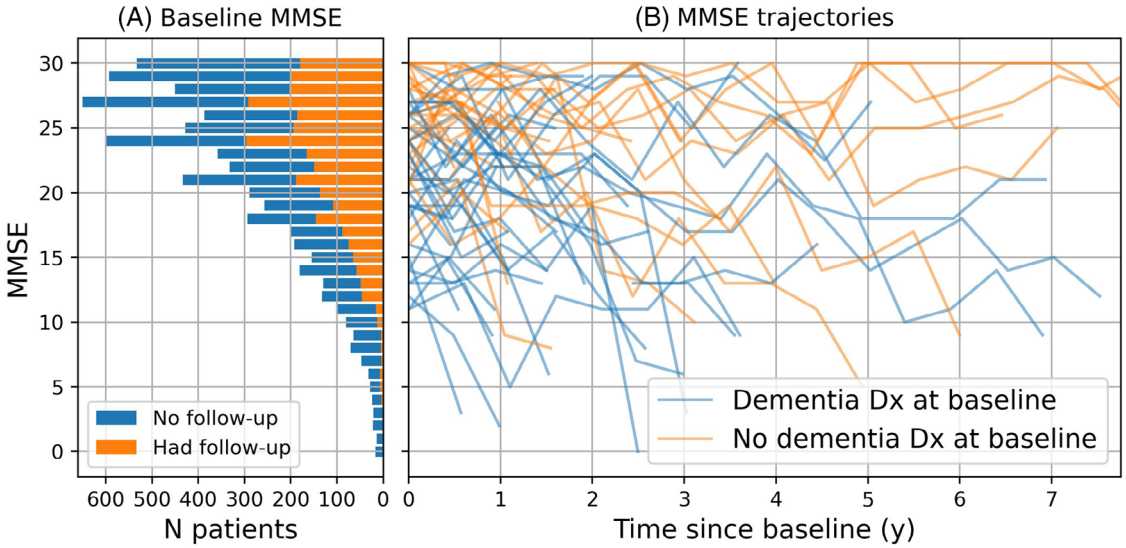

FIGURE 1 Baseline MMSE distribution and sample trajectories. A, Histogram of baseline MMSE measurements (including MoCA measurements mapped to equivalent MMSE values). The blue and orange portions of each bar represent patients with and without follow-up measurements, respectively. B, Random sample of 100 MMSE trajectories along with the average trajectory for patients with baseline diagnoses of dementia, MCI, other CIND, and unimpaired (i.e., no diagnosis). Individual trajectories are colored blue if the patient had a diagnosis of dementia at or before baseline and orange otherwise. Both scores and follow-up times were randomly perturbed to protect privacy. Average trajectories were calculated using a 6 month sliding window. CIND, cognitive impairment non-dementia; MCI, mild cognitive impairment; MMSE, Mini-Mental State Examination, MoCA, Montreal Cognitive Assessment.

associations including both obvious and less obvious. This work represents a first attempt at broader precision medicine efforts using RWD to understand why some patients progress more quickly than others, identify distinct groups of patients who progress differently, and predict decline before it happens. Several models for predicting change in cognitive assessment scores have been developed, though almost exclusively on publicly available datasets from the Alzheimer's Disease Neuroimaging Initiative[28–38] and other volunteer-based studies.[1,39] Our goal in this work was to present an initial progression model developed on EHR data using only readily available measurements and, to our knowledge, this is the first such model.

When examining baseline cognitive assessments, the largest associations were with baseline dementia diagnoses. AD and multiple dementias had the largest such associations—both ≈ 5.4 and 5.5 MMSE points lower than unimpaired patients—while patients with vascular and other dementias scored 4.5 and 5.1 points lower than unimpaired patients, respectively. Additionally, in our progression model, all dementia types except vascular ($\beta = -0.43$ [−0.94 to 0.08]) were predictive of faster rates of decline with multiple dementia associated with the fastest decline at ≈ 1 point per 6 months faster decline than undiagnosed patients. While the relationship between prior and predicted MMSE scores was largely linear—indicating that the average unimpaired patient is largely stable—there was evidence of a floor effect, with patients having prior MMSE scores < 5 predicted to have a slight increase in MMSE (see Figure S2). In contrast, MCI and other CIND diagnoses were significantly associated with neither baseline MMSE scores nor rate of decline. This runs contrary to previous results from volunteer-based study cohorts finding differences in rate of change in MMSE.[40,41] This discrepancy may reflect a referral bias wherein patients are unlikely to be seen at the memory clinic without some concern regarding cognitive impairment. Similarly, while loss to follow-up was accounted for through weighting, it is possible that residual dropout bias remains. Finally, there may be differences in the operationalization of MCI between clinical and research settings, though additional study is needed to test this hypothesis. Regardless of the cause, this finding should serve as a caution for those using EHR data from memory clinics and indicates that additional work is needed to understand referral practices when using such data.

We found several patient demographic variables that were associated with lower baseline MMSE, including female sex, older age, and non-White racial groups. These associations are largely consistent with the literature showing higher dementia rates among these groups.[42–44] However, it is worth noting that, while baseline dementia diagnoses were higher in all of these groups (Table 1), the model included baseline diagnoses, meaning that these demographics were associated with baseline MMSE scores within diagnosis groups. For example, in the baseline model Black patients had 1.8 point lower MMSE scores compared to White patients *after* accounting for baseline dementia diagnosis. It is unclear from these results how much of this gap in baseline cognitive assessment scores is due to patients receiving memory care later in their disease course[45,46] or due to well-established racial disparities in common cognitive assessments,[47–49] both of which are supported by existing literature. In contrast, age was the *only* demographic variable predictive of faster decline (Table 3), while other and unknown racial groups were associated with slower decline. The lack of associations with other demographics, especially Black race and Hispanic ethnicity, is consistent with literature findings that, despite cross-sectional disparities in MMSE scores, minoritized groups,

TABLE 2 Associations between predictors and baseline MMSE.

	Unweighted		Inverse probability of censoring weighted	
	Coefficient [95% CI]	*p* value*	Coefficient [95% CI]	*p* value*
Female	−1.27 [-1.52 to -1.02]	<0.001	−1.13 [-1.43 to -0.84]	<0.001
Age	−0.08 [-0.09 to -0.06]	<0.001	−0.07 [-0.09 to -0.06]	<0.001
Race				
White	Ref.		Ref.	
Black	−2.00 [-2.35 to -1.65]	<0.001	−1.76 [-2.15 to -1.37]	<0.001
Asian	−1.75 [-2.45 to -1.05]	<0.001	−1.25 [-1.99 to -0.52]	<0.001
Other	−1.59 [-2.31 to -0.88]	<0.001	−1.10 [-1.82 to -0.39]	0.004
Unknown	−2.69 [-4.19 to -1.19]	<0.001	−2.24 [-3.60 to -0.88]	0.002
Hispanic	−0.36 [-1.26 to 0.55]	1.000	−0.42 [-1.29 to 0.46]	1.000
Baseline diagnosis				
Unimpaired	Ref.		Ref.	
Alzheimer's disease	−5.34 [-6.27 to -4.42]	<0.001	−5.38 [-6.26 to -4.51]	<0.001
Vascular dementia	−4.45 [-5.49 to -3.41]	<0.001	−4.54 [-5.54 to -3.54]	<0.001
Multiple dementia	−5.40 [-6.51 to -4.29]	<0.001	−5.52 [-6.59 to -4.45]	<0.001
Other dementia	−5.04 [-5.95 to -4.12]	<0.001	−5.10 [-5.96 to -4.25]	<0.001
MCI	0.53 [-0.34 to 1.40]	0.619	0.38 [-0.41 to 1.16]	1.000
Other CIND	0.56 [-0.30 to 1.43]	0.465	0.42 [-0.35 to 1.20]	0.652
Total comorbidities	0.09 [0.04 to 0.14]	<0.001	0.07 [0.00 to 0.13]	0.082
Hypertension	−0.12 [-0.40 to 0.17]	1.000	−0.07 [-0.40 to 0.25]	1.000
Diabetes	−0.10 [-0.41 to 0.21]	1.000	−0.01 [-0.35 to 0.33]	1.000
Obesity	0.17 [-0.23 to 0.56]	1.000	0.36 [-0.07 to 0.79]	0.210
Hearing	0.37 [0.02 to 0.71]	0.070	0.38 [0.04 to 0.73]	0.052
Anxiety	0.03 [-0.27 to 0.33]	1.000	0.14 [-0.20 to 0.48]	1.000
Depression	0.03 [-0.27 to 0.32]	1.000	0.18 [-0.18 to 0.53]	0.970
Chronic pain/fatigue	0.76 [0.43 to 1.08]	<0.001	0.70 [0.32 to 1.08]	<0.001
Stroke/TIA	−0.05 [-0.45 to 0.34]	1.000	0.05 [-0.36 to 0.46]	1.000
Total medications	0.02 [-0.01 to 0.04]	0.262	0.02 [-0.00 to 0.05]	0.185
Cholinesterase inhibitor	−1.42 [-1.74 to -1.10]	<0.001	−1.65 [-2.02 to -1.29]	<0.001
Distance to clinic (km)	0.02 [-0.01 to 0.06]	0.553	0.00 [-0.03 to 0.04]	1.000
Distance to clinic (>20 km)	−0.02 [-0.08 to 0.03]	1.000	−0.00 [-0.06 to 0.06]	1.000
Distance to clinic (>40 km)	0.00 [-0.02 to 0.02]	1.000	−0.00 [-0.03 to 0.02]	1.000
Medicaid	−1.87 [-2.34 to -1.40]	<0.001	−2.68 [-3.48 to -1.88]	<0.001
SDI score	−0.00 [-0.01 to 0.00]	0.325	−0.00 [-0.01 to 0.00]	0.730
Primary care patient	−0.10 [-0.47 to 0.27]	1.000	0.04 [-0.34 to 0.42]	1.000
No. primary care visits (1 year)	−0.02 [-0.05 to 0.02]	1.000	−0.02 [-0.05 to 0.02]	0.832

Abbreviations: CI, confidence interval; CIND, cognitive impairment non-dementia; MMSE, Mini-Mental State Examination; SDI, Social Deprivation Index; TIA, transient ischemic attack.

*All *p* values have been adjusted for multiple comparisons using the Benjamini–Hochberg procedure.

especially Black patients, progress at the same rate as non-Hispanic White patients.[47–49] The replication of these results in real-world clinical data reinforces the need to understand why gaps in cognitive assessments and delays in referral, assessment, and diagnosis may arise.

Finally, among comorbidities, only chronic pain and fatigue was significantly associated with baseline MMSE and no comorbidities were associated with rate of progression. As above, it is important to note that these models included cognitive diagnoses. While these comorbidities are well established as risk factors for dementia, these results

TABLE 3 Associations between predictors and MMSE change over time.

	Unweighted		Inverse probability of censoring weighted	
	Coefficient [95% CI]	*p* value*	Coefficient [95% CI]	*p* value*
Female	−0.11 [-0.19 to -0.03]	0.006	−0.06 [-0.14 to 0.03]	0.461
Age	−0.01 [-0.01 to -0.00]	0.002	−0.01 [-0.01 to -0.00]	<0.001
Race				
White	Ref.			
Black	0.08 [-0.02 to 0.18]	0.221	0.10 [-0.02 to 0.22]	0.143
Asian	0.08 [-0.11 to 0.28]	1.000	0.18 [-0.07 to 0.44]	0.319
Other	0.22 [-0.00 to 0.44]	0.080	0.27 [-0.00 to 0.54]	0.067
Unknown	0.85 [0.02 to 1.68]	0.062	0.97 [0.10 to 1.83]	0.035
Hispanic	0.07 [-0.23 to 0.37]	1.000	0.09 [-0.20 to 0.38]	1.000
Baseline diagnosis				
Unimpaired	Ref.		Ref.	
Alzheimer's disease	−0.70 [-1.17 to -0.22]	0.005	−0.82 [-1.31 to -0.33]	0.001
Vascular dementia	−0.35 [-0.84 to 0.15]	0.334	−0.43 [-0.94 to 0.08]	0.159
Multiple dementia	−0.81 [-1.31 to -0.31]	0.002	−0.97 [-1.48 to -0.45]	<0.001
Other dementia	−0.42 [-0.90 to 0.05]	0.135	−0.52 [-1.00 to -0.04]	0.042
MCI	0.13 [-0.33 to 0.60]	1.000	−0.01 [-0.46 to 0.43]	1.000
Other CIND	0.24 [-0.22 to 0.70]	0.900	0.10 [-0.33 to 0.53]	1.000
Total comorbidities	0.01 [-0.01 to 0.03]	0.402	0.01 [-0.01 to 0.03]	1.000
Hypertension	−0.03 [-0.11 to 0.06]	1.000	−0.03 [-0.12 to 0.07]	1.000
Diabetes	0.09 [-0.01 to 0.18]	0.124	0.09 [-0.02 to 0.19]	0.177
Obesity	0.09 [-0.03 to 0.20]	0.299	0.10 [-0.03 to 0.23]	0.209
Hearing	0.04 [-0.07 to 0.14]	1.000	0.05 [-0.07 to 0.17]	1.000
Anxiety	−0.11 [-0.20 to -0.02]	0.020	−0.09 [-0.20 to 0.01]	0.126
Depression	0.02 [-0.07 to 0.11]	1.000	0.03 [-0.07 to 0.14]	1.000
Chronic pain/fatigue	−0.04 [-0.15 to 0.06]	1.000	−0.03 [-0.16 to 0.10]	1.000
Stroke/TIA	−0.08 [-0.21 to 0.05]	0.555	−0.09 [-0.23 to 0.05]	0.611
Total medications	0.02 [0.02 to 0.03]	<0.001	0.02 [0.02 to 0.03]	<0.001
Cholinesterase inhibitor	−0.12 [-0.21 to -0.02]	0.019	−0.14 [-0.25 to -0.03]	0.018
Distance to clinic (km)	0.01 [-0.00 to 0.02]	0.732	0.01 [-0.00 to 0.02]	0.270
Distance to clinic (>20 km)	−0.01 [-0.02 to 0.01]	1.000	−0.01 [-0.03 to 0.01]	0.496
Distance to clinic (>40 km)	0.00 [-0.00 to 0.01]	1.000	0.00 [-0.00 to 0.01]	1.000
Medicaid	−0.01 [-0.16 to 0.13]	1.000	−0.03 [-0.21 to 0.15]	1.000
SDI score	0.00 [-0.00 to 0.00]	0.190	0.00 [-0.00 to 0.00]	0.424
Primary care patient	−0.12 [-0.24 to -0.00]	0.055	−0.12 [-0.25 to 0.02]	0.135
No. primary care visits (1 year)	0.01 [-0.00 to 0.02]	0.803	0.00 [-0.01 to 0.02]	1.000

Abbreviations: CI, confidence interval; CIND, cognitive impairment non-dementia; MCI, mild cognitive impairment; SDI, Social Deprivation Index; TIA, transient ischemic attack.

*All *p* values have been adjusted for multiple comparisons using the Benjamini–Hochberg procedure.

suggest that they may not have predictive value for heterogeneity within diagnosis groups and thus may not be useful for identifying diagnostic subtypes beyond those that already exist. The slightly higher scores among chronic pain/fatigue patients may reflect a referral bias, though additional investigation is needed to confirm this. On the other hand, we did find significant associations with both included medication variables. First, we found that prescription for cholinesterase inhibitors or memantine was associated with 1.7 point lower baseline MMSE score. This is expected and reflects increased prescribing among sicker patients, and the fact that clinicians are unlikely to

prescribe these medications without evidence of cognitive and/or functional impairment. However, we also found that prescription of cholinesterase inhibitors or memantine at baseline was associated with faster post-baseline decline. As this study was not designed to estimate the causal effects of these medications, this may easily be caused by residual confounding. However, the progression model included several major confounders (age, sex, diagnoses, and baseline MMSE), so this finding warrants additional investigation. Additionally, we found that the total number of medications at baseline was associated with *slower* MMSE progression. Because of the lack of associations with comorbidities, it is hard to attribute this to increased medical complexity. Instead, it may be possible that this association reflects increased care-seeking behavior or fewer barriers to care, but further study is required to confirm this.

The use of EHR data has several limitations. Medication and diagnosis data reflect only orders and diagnoses made by Johns Hopkins providers or self-reported by the patient and do not reflect medical care by providers unaffiliated with Johns Hopkins. Further, diagnosis codes not only reflect the provider's assessment of the patient's health, but also the value to the patient of making a particular diagnosis. There is also substantial selection, measurement, and censoring bias in EHR data. In this study, we used inverse probability weighting to account for selection bias, but the possibility of residual bias remains. The JHMATC patient population reflects a population of patients that (1) have received a referral or sought out memory care and (2) are sufficiently unburdened by barriers to health care that they are able to access care. Further, as seen in Table S2, the choice of who receives cognitive assessments and which assessment is administered is not random and reflects the perceived level of impairment. In our data, MoCA assessments were more common among patients with lower levels of impairment owing to MoCA's improved sensitivity among such patients.[50] Such cohorts may be inappropriate for epidemiological questions but are still valuable for developing prognostic models and risk prediction tools, defining novel dementia phenotypes, and assessing the impact of treatments in specialty care populations, so long as we acknowledge and account for these biases. Finally, we used area deprivation and Medicaid enrollment as proxies for socioeconomic status when adjusting for censoring; however, these proxies may not accurately represent individual socioeconomic status.

3 | CONCLUSION

In this study, we used real-world clinical data to examine predictors of cognitive decline after an initial memory care visit. Our findings were largely consistent with existing literature suggesting that there are several groups who access memory care later in their disease course, but that, once in care, these groups largely decline at similar rates. Future efforts will focus on the inclusion of a larger set of predictors, improving our understanding of referral biases in this data, and developing models that better capture heterogeneity in rates of decline. Of particular interest are patients who appear stable for a period but then decline rapidly, which is not well captured by existing models.

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CONFLICT OF INTEREST STATEMENT

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DATA AVAILABILITY STATEMENT

The data used in this study comprise electronic health records which have not been made publicly available to protect patient privacy. These data can be made available under appropriate data use agreements. Please contact RA for additional details.

CONSENT STATEMENT

Because the study constituted secondary use of existing records with no new measurements or interventions, and owing to the number of patients included in the study, a waiver of consent was granted by the Johns Hopkins University IRB (Nos. IRB00228485 and IRB00269466).

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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