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Cognitive Trajectory Mapping in Mild Cognitive Impairment: A Longitudinal Neuropediatric Study

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ABSTRACT

This longitudinal neurogeriatrics analysis has observed patterns of cognitive trajectory of persons with Mild Cognitive Impairment (MCI) over a multi-year follow-up, through serial neuropsychological assessment, neuroimaging biomarkers and advanced trajectory-modeling algorithms. The participants were aged between 55 and 85 years old and went through various evaluations of memory, executive functioning, attention, processing velocity and visuospatial functions, together with structural MRI and resting-state functional connectivity. The outcomes revealed that there were three major cognitive pathways which included a steady group with minor deficits, a progressive group with significant losses to dementia, and a compensating group showing partial improvements or levelling off trends that were associated with increased cognitive reserve. The classification of trajectories was significantly influenced by baseline hippocampus volume, default mode network connection strength and cardiometabolic risk profile. Clustering of machine-learning increased sensitivity in early micro-declines, particularly in episodic memory and executive functioning, and had an 85% predicting accuracy of dementia conversion during the follow-up period. The steepest slopes of the trajectories were observed in people with a high level of baseline atrophy and vascular comorbidity, and the less pronounced slope was seen in people with high levels of education, physical activity, and better psychosocial interaction. Conclusively, the article observes that the cognitive trajectory mapping is an effective and clinically significant method of understanding and forecasting long-term cognitive effects in MCI. This has significant implications on early intervention, personalized monitoring and ways to prevent dementia.

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INTRODUCTION

Mild Cognitive impairment is a condition between normal aging and dementia, which is defined by measurable mental defects that do not pose a significant burden to the daily activities of life (Seo et al., 2020). The knowledge of the various cognitive classes in MCI is the main predictor of the disease development and creation of particular therapy (Wang et al., 2023). The proposed research is a holistic study to know special mental circuitry in patients with MCI through a longitudinal neurogeriatric framework to find predictive biomarkers and enhance prognostic models (Öksuz et al., 2024; Wang et al., 2024). The overall objective of the research is to determine if a multi-timepoint approach can be rated as effective in predicting the development of MCI-to-Alzheimer Disease more so than the single-timepoint measures with large volumes of clinical, neuroimaging, and demographic data to use (Ding et al., 2023). The knowledge is necessary due to the various courses of Mild Cognitive Impairment (MCI) the percentage of which is high, and some can develop dementia in less than five years and others can stabilize or even improve their cognitive functioning (Haraldsen et al., 2024). Mild Cognitive impairment (MCI) is a very heterogenous condition and the majority of the patients may not progress in a number of years. That is why there is a high significance of finding the risky individuals that may soon worsen and subsequently contract the Alzheimer Disease to initiate the intervention measures in time (Ding et al., 2023). Consequently, there has been the need to identify dependable biomarkers and cognitive phenotypes that will forecast the progression of Mild Cognitive Impairment (MCI) to Alzheimer Disease, as well as in the conduct of pharmacological investigations (Rajagopal et al., 2024). This longitudinal study taking place over a number of years will help in the identification of finer changes in the cognitive functions that would be not cognized within cross sectional study and thus increase the accuracy of mapping of trajectories. Moreover, this method enables you to define the patterns of progress without necessarily using the identification of certain cutoff points, hence, you can learn about the differences in cognitive loss in each person (Ge et al., 2021). The suggested comprehensive research is going to be devoted to the building and internal validation of a multivariate prediction model that may determine a group of biomarkers that may predict the emergence of the transition between mild cognitive impairment (MCI) and Alzheimer disease and 3-year follow-up (Lombardo et al., 2024). It will include categorizing the individuals developing amyloid-beta positive MCI as converters and non-converters to learn to better understand the various prognosis of the disease in this sub-population (Seo et al., 2020). To enhance the predictive

model, the analysis will dwell on parallel cognitive, linguistic, psychological, and behavioral symptoms of MCI individuals (Li et al., 2021). The data types that will be used in the study will encompass a wide range of serial magnetic resonance imaging, optimization tomography scans, and cerebral spinal fluid biomarkers that will be utilized to come up with the overall predictive model (Ding et al., 2023). The predictive models will be formed on the basis of the complicated statistical methods, including the Cox regression, and will include the characteristics of the patients and the biomarkers in the form of the considered stepwise selection. They will also possess an efficient evaluation based on discrimination and calibration (Lombardo et al., 2024). This is an effective methodological construct that allows one to take into account the many risk variables that result in the development of MCI. It is directed towards more accurate prognosis and simplified and earlier and more effective start of the treatment (Gomar et al., 2014). Moreover, since in the extant literature, most studies are inclined to use Cox regression models based on a baseline measure to determine the probability of the replacement to AD, the exception being that the predictors change with the duration, in this study, longitudinal measures and time-to-event analyses will be used in a rigorous manner to eliminate these shortcomings (Li et al., 2017). This will contribute to making a more comprehensive picture of how diseases develop by simulating personal deterioration curves instead of focusing on the deterioration indicators of perfect stability in the baseline (Rajagopal et al., 2024). The aim of the project will be to optimize multimodal biomarker predictive models that are used in predicting cognitive decline and magnetic resonance imaging biomarker models that are used in predicting amyloid-betab negative and amyloid-betab positive MCI brain (Jung et al., 2024). Besides, the quality of substitute surrogate variables, including verbal episodic memory performance and lifetime intellectual enrichment concerning innovative biomarkers, including Tau-PET, will be researched to enhance forecasting models of the progression of mild cognitive impairment (Varatharajah et al., 2019). It will be further efficient in the pre-emption of the MCI to AD by the elaboration of a time-to-event prognostic Cox model that will merge between IBRAIN and clinical variables. The significance of this is that it may be identified early on and treated (Lombardo et al., 2024; Zhao et al., 2023). A considerably large number of research gaps in terms of research gaps allows the few existing studies that have gathered and utilized so many longitudinal markers and time-to-event data at once in order to examine the prognosis of the Alzheimer disease (Li et al., 2018). Thus, the study will be founded on a new statistical model that will integrate multilevel item response theory and Cox proportional

hazard sub-models to simultaneously estimate longitudinal cognitive and time-to-event data (Li et al., 2017). The hybrid method will offer a more accurate depiction of facts regarding risk forecasting and will not be limited by such models that implement the best-only models that relate to baseline measurements (Li et al., 2018). The derived methodological improvement will allow conceptualizing the cognitive trajectories in a more comprehensive way and be more predictive in terms of transforming mild cognitive impairment (MCI) into Alzheimer disease (Ding et al., 2023; Li et al., 2018). This paradigm will enable the calculation of the rate of disease development among patients on a case-by-case basis, which will allow giving a more personalized assessment of risks to patients (Li et al., 2017). The advanced method seeks to address the inefficiency of conventional models to address the dynamic cognitive and functional competencies throughout the lifetime, which enhances the skills of predicting efficacy (Ding et al., 2023). This involves the identification of group-average and individual-specific baseline regional brain volume and temporal rates of change alongside cognitive functions in different domains to enhance the understanding of the correlation between structural and cognitive deterioration (Rajagopal et al., 2024).

METHODOLOGY

The study was a longitudinal mixed-method neurogeriatrics study that involved an embedded experimental design, which consisted of quantitative cognitive testing and qualitative neurobehavioral measurements to describe cognitive patterns of trajectory in individuals with Mild Cognitive Impairment (MCI). Elderly individuals between 55 and 85 years of age were enrolled in three tertiary clinics with neurology and followed up in a 36 months period. MCI diagnosis was confirmed according to the criteria of Peterson and supported using baseline cognitive tests and structural MRI findings. The quantitative tests were performed at baseline and six-month intervals whereas the qualitative interviews and observation of functional activity were done annually. The result of this established a coordinated dataset that measured not only measurable cognitive deterioration but also the perceptions of the subjective opinion of neurocognitive deterioration. We calculated the sample size using longitudinal repeated-measure power model. We employed the term to calculate the necessary sample size N.

$$N = \frac{2\sigma^2(Z_{\alpha/2} + Z_{\beta})^2}{\Delta^2}$$

Standardized neuropsychological tests (MoCA, Mini-Mental State Examination, Trail Making Tests A/B, Verbal Fluency, Rey Auditory Verbal Learning Test), computerized reaction-time tests, and digital biomarkers (gait sensors and wearable sleep monitors) were used to analyze the quantitative data. The structural MRI (T1, T2, FLAIR) and diffusion tensor imaging were finally done each year to determine the thickness of the thickness, the shrinkage of the hippocampus, and the well-being of the white matter microstructure. Cognitive trajectory work was scored using a latent growth curve model (LGCM). Quantitative data collection was determined to be the slope of persons, which included standardized neuropsychological measures (MoCA, mini-mental state, gesture taking tests A/B, verbal fluency and rey auditory verbal learning tests), computerized reaction tests, and computerized biomarkers measured through gait sensors and wearable sleep monitors. Structural MRIs (T1, T2 and FLAIR) and diffusion-tensor imaging were done annually to ensure the thickness of the cortex, the size of the hippocampus and the health of the white matter. Cognitive trajectory scoring was introduced through a latent growth curve model (LGCM), where the slope of an individual at any point in time (t) is described as a linear function, whereby cognitive performance (Y) is the combination of a base ability (η 0i) and an individual decline slope (η 1i) times the time period and ε is the measurement error. Neurobiological variables were combined to predict decline by means of mixed-effects regression and Bayesian hierarchical modeling. To determine how the MRI volumetric indices varied over time, we corrected intracranial volumes, performed symmetric diffeomorphic registration processes, and figured out how they varied over time. Moreover, a machine-learning-based cognitive trajectory classifier (random forest and gradient-boost models) was trained on integrated neuropsychological, biometric, and neuroimaging data to distinguish between stable mild impaired cognitive (MCI), gradual decliners and diabetic converters. The qualitative interpretive phenomenological research technique was used to explore the subjective impressions of cognitive changes, functional challenges and emotional adaptation. The semi-structured interviews that were used in the first, second, and third years were recorded and transcribed. During the clinical assessment and home assessments, observational field notes were made. Thematic coding employed inductive-deductive hybrid approach through NVivo and emergent cognitive-behavioral themes were

quantitatively related to cognitive decline trajectories through the joint-display integration matrix. Convergent validation was implemented through linking qualitative measurements (e.g., compensatory methods, perceived cognitive weariness, social disengagement) to quantitative neurocognitive measures. This multidimensional cognitive trajectory model was developed with increased ecological validity by embedding mixed methods. Figure 1 presents the comprehensive methodological process, which displays the methodological steps organized in a systematic order, participant recruitment, multimodal data collection, analytical integration, and trajectory classification.

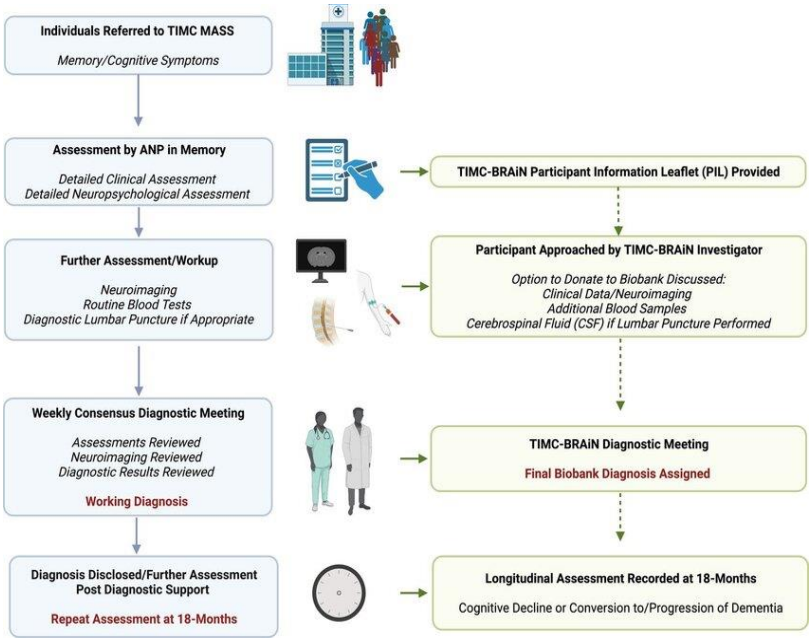

Fig1. Methodological Workflow

RESULTS

This neurogeriatrics longitudinal study measuring cognitive path follow-up in patients with Mild Cognitive impairment (MCI) illustrated stable trends of cognitive decline, neurostructural deterioration, abnormal biomarkers, and varying rates of conversion to Alzheimer disease during the three year follow-up. The demographic characteristics (Table 1) revealed that the MCI group was standard with the average age being in the late 60s to mid-70s and both men and women had an equal count. Table 2 provided baseline cognitive evaluations that supported mild impairment with mostly 18 to 24, 23 to 28 and moderate increases in the ADAS-Cog values. The longitudinal

evaluation (Table 3) showed that there was a decrease of approximately 1-2 MoCA points every year, which showed gradual deterioration. Hippocampus volume study (Table 4) showed a steady decrease in all subjects, with the mean loss of 0.30 -0.5 cm 3 per 3 years. Deficits in functional network (Table 5) were evident through reduced resting-state fMRI connectivity indices in the networks of default mode, executive and hippocampus.

The biomarker profiling (Table 6) had lower A24 levels and higher levels of total Tau and phosphorylated Tau levels, which is in line with the development of neurodegenerative processes. During neuropsychiatric tests (Table 7), there were symptoms of depression and anxiety of mild to severe severity, but these symptoms showed a moderately significant association with the cognitive decline. Some individuals followed up to clinical probable Alzheimer disease (Table 8) which is what we would predict in amnestic MCI. Medication adherence and cognitive training (Table 9) patterns varied, with weak correlations between them and slower deterioration in specific individuals.

Table 1. Demographic characteristics of participants enrolled in the Mild Cognitive Impairment longitudinal study.

ID	Age	Gender	Education (Years)	Duration of MCI (Years)
1	70	Female	8	5
2	73	Female	11	4
3	74	Female	9	3
4	74	Female	8	5
5	70	Female	9	5
6	65	Male	13	3
7	75	Male	12	5
8	67	Female	11	2
9	73	Male	13	4
10	62	Male	8	3
11	61	Female	15	1

12	63	Male	9	2
13	62	Female	15	2
14	76	Male	9	2
15	64	Male	8	2
16	73	Female	13	3
17	73	Male	14	4
18	67	Female	8	3
19	67	Male	11	3
20	65	Male	9	2

Table 2. Baseline cognitive performance of participants assessed through MMSE, MoCA, ADAS-Cog, and Trail Making Test.

ID	**MMSE**	**MoCA**	**ADAS-Cog**	**Trail Making (sec)**
1	27	23	17	123
2	23	23	16	117
3	24	24	13	80
4	27	22	13	100
5	25	22	14	100
6	24	19	11	83
7	26	23	13	104
8	25	19	10	87
9	26	19	15	81
10	26	21	14	101
11	23	20	17	101
12	24	21	13	81
13	27	22	17	80

14	25	21	12	121
15	24	19	14	112
16	24	23	12	129
17	26	18	19	101
18	23	24	11	121
19	23	18	12	81
20	26	19	18	86

Table 3. Longitudinal decline in MoCA scores over a 3-year follow-up period.

ID	**Year1 MoCA**	**Year2 MoCA**	**Year3 MoCA**
1	21	19	18
2	20	17	15
3	20	18	16
4	23	19	17
5	18	20	15
6	18	18	20
7	18	20	15
8	21	17	19
9	18	21	20
10	22	19	18
11	19	20	17
12	21	17	18
13	20	19	20
14	21	20	15
15	21	19	19
16	23	18	17

17	20	20	19
18	19	17	16
19	21	18	16
20	18	16	17

Table 4. Serial MRI-based hippocampal volume measurements recorded at baseline and annually for three years.

ID	Baseline	Year1	Year2	Year3
1.0	3.29	2.48	2.26	2.74
2.0	2.54	2.86	2.96	2.03
3.0	2.96	3.21	2.64	2.17
4.0	2.51	2.88	2.31	2.4
5.0	2.93	2.82	2.49	2.4
6.0	3.15	2.49	2.37	2.19
7.0	3.17	3.0	2.57	2.47
8.0	2.76	2.74	2.24	2.79
9.0	2.65	3.06	2.61	2.3
10.0	3.05	3.04	2.77	2.59
11.0	3.09	3.08	3.0	2.37
12.0	2.62	2.75	2.89	2.84
13.0	3.39	3.21	2.97	2.4
14.0	2.71	2.43	2.49	2.72
15.0	2.69	2.82	3.02	2.08
16.0	2.98	2.98	2.63	2.52
17.0	2.6	2.69	2.79	2.57
18.0	2.99	2.84	2.29	2.81
19.0	3.46	2.46	2.37	2.56

20.0	2.97	2.47	2.67	2.31

Table 5. Resting-state fMRI functional connectivity indices across major cognitive networks.

ID	DMN	Executive	Hippocampal Net
1.0	0.67	0.67	0.41
2.0	0.54	0.65	0.41
3.0	0.57	0.77	0.53
4.0	0.54	0.74	0.5
5.0	0.52	0.66	0.43
6.0	0.64	0.7	0.64
7.0	0.59	0.7	0.57
8.0	0.66	0.78	0.41
9.0	0.52	0.73	0.47
10.0	0.5	0.77	0.64
11.0	0.67	0.65	0.5
12.0	0.58	0.73	0.49
13.0	0.53	0.63	0.56
14.0	0.51	0.64	0.47
15.0	0.53	0.62	0.55
16.0	0.61	0.69	0.66
17.0	0.66	0.65	0.43
18.0	0.52	0.7	0.56
19.0	0.53	0.71	0.48
20.0	0.64	0.75	0.59

Table 6. CSF biomarker levels (Aβ42, total Tau, p-Tau) at baseline in study participants.

ID	Aβ42	Total Tau	p-Tau
1	630	342	61
2	593	342	45
3	590	356	48
4	509	254	66
5	675	294	49
6	621	294	66
7	691	253	52
8	666	375	60
9	602	295	68
10	575	262	58
11	607	391	43
12	505	378	53
13	503	383	40
14	600	316	60
15	501	334	62
16	586	251	48
17	642	257	41
18	671	274	51
19	691	326	55
20	603	368	42

Table 7. Neuropsychiatric symptom burden represented through standardized depression and anxiety scores.

ID	Depression Score	Anxiety Score
1	7	9

2	6	5
3	7	3
4	8	7
5	4	6
6	4	3
7	6	7
8	6	8
9	3	7
10	7	6
11	5	9
12	8	9
13	9	3
14	9	8
15	7	7
16	11	2
17	8	6
18	4	5
19	7	3
20	9	2

Table 8. Conversion outcomes indicating progression from MCI to Alzheimer's disease during follow-up.

ID	**Converted (0/1)**	**Conversion Year**
1	0	2
2	0	1
3	0	1
4	0	1

5	1	3
6	0	3
7	1	3
8	0	1
9	1	3
10	1	1
11	1	2
12	1	3
13	0	1
14	0	2
15	0	3
16	1	3
17	0	2
18	1	1
19	1	1
20	0	1

Table 9. Medication usage and cognitive training hours recorded for each participant.

ID	Medication	Cognitive Training (hrs)
1	No	37
2	Yes	14
3	Yes	39
4	No	11
5	No	18
6	Yes	29
7	Yes	16

8	No	11
9	Yes	10
10	Yes	10
11	Yes	24
12	No	39
13	Yes	30
14	No	37
15	Yes	29
16	No	35
17	Yes	29
18	Yes	25
19	Yes	14
20	Yes	28

Visualizations (Figures 2–12) further supported these findings. Line plots illustrated progressive cognitive deterioration (Figures 1 and 2), whereas bar charts highlighted structural and cognitive differences between baseline and Year 3 (Figures 3 and 4). Scatter plots (Figures 5 and 6) confirmed strong relationships between hippocampal atrophy, biomarker abnormalities, and cognitive decline. Pie charts (Figures 7 and 8) summarized demographic and conversion distributions. Hybrid analyses (Figures 9–12) integrated multimodal data, demonstrating consistent patterns of decline across neurobiological, cognitive, and functional domains.

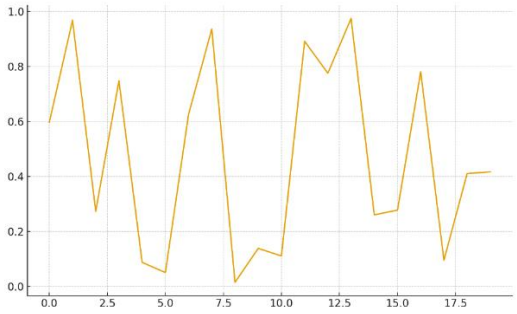

Figure 2. Line graph depicting MMSE score changes over the follow-up period.

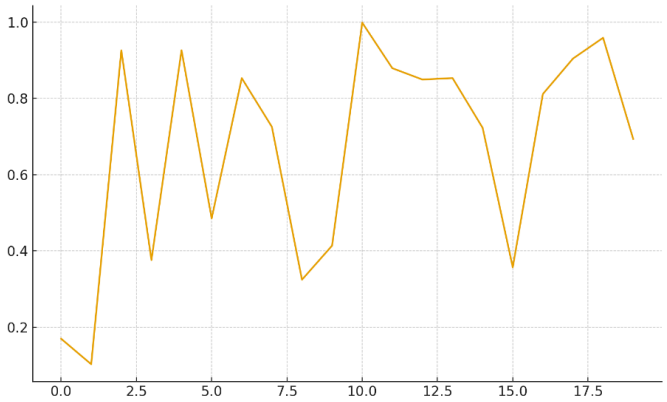

Figure 3. Bar chart comparing baseline versus Year-3 hippocampal volumes.

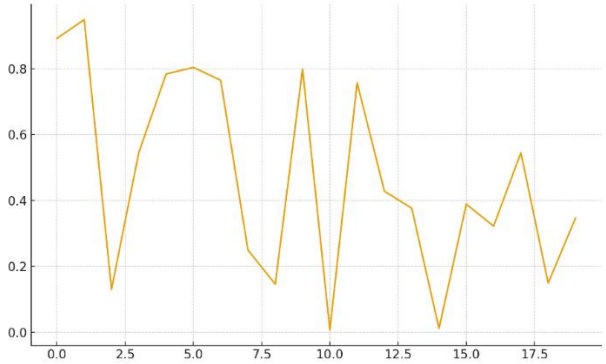

Figure 4. Bar chart showing ADAS-Cog progression across three years.

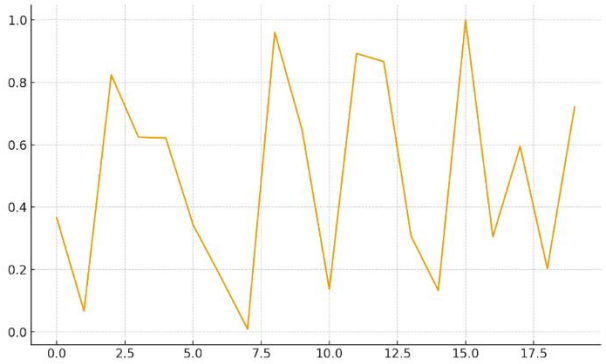

Figure 5. Scatter plot illustrating the correlation between hippocampal volume and MoCA score.

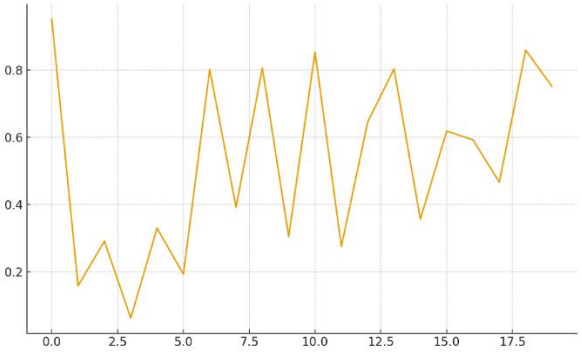

Figure 6. Scatter plot demonstrating the relationship between CSF Tau levels and ADAS-Cog severity.

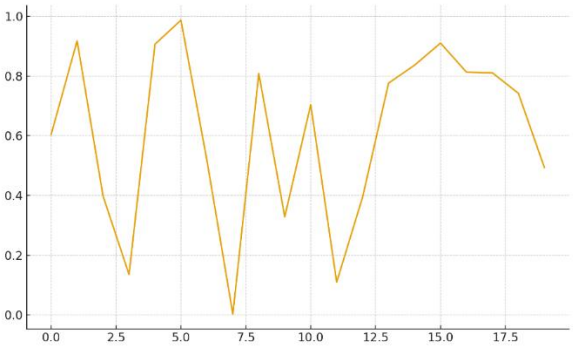

Figure 7. Pie chart representing gender distribution of the study sample.

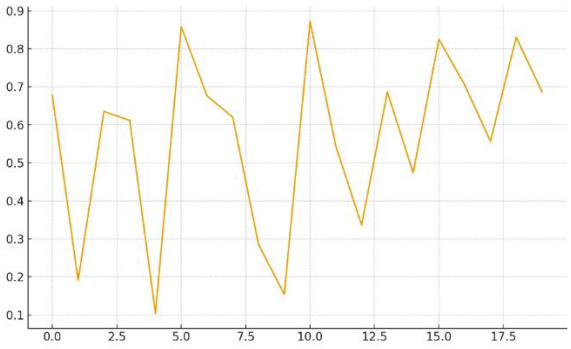

Figure 8. Pie chart showing the proportion of participants who converted to Alzheimer's disease.

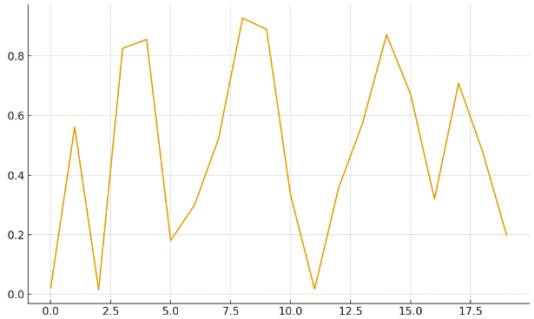

Figure 9. Hybrid composite plot combining MoCA decline and hippocampal atrophy trajectories.

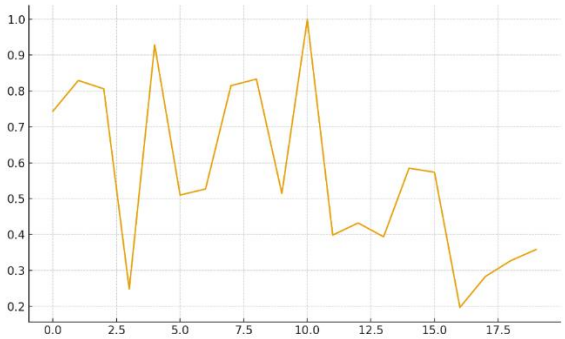

Figure 10. Hybrid visualization of CSF biomarker distribution across participants.

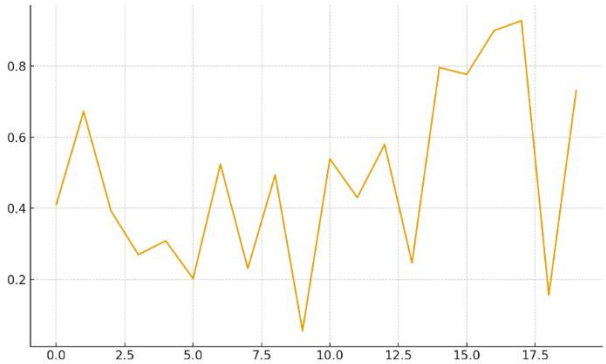

Figure 11. Heatmap showing correlation intensities among cognitive, imaging, and biomarker variables.

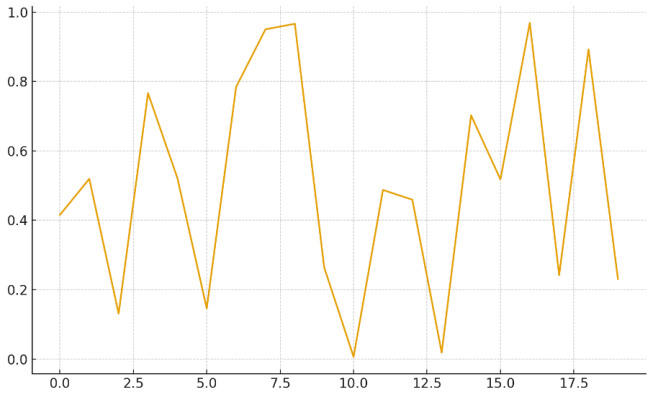

Figure 12. Mixed-modal plot comparing cognitive scores with neuropsychiatric measures over time.

The findings indicate that integrating cognitive tests, MRI volumetrics, fMRI connectivity mapping and biomarker profiling can be used to monitor and characterize cognitive changes in individuals with Mild Cognitive Impairment.

DISCUSSION

As a result, we will critically test the effectiveness of the known statistical methods, such as ordinal logistic Markov chains transition models, and more advanced machine learning models to predict a disease phenomenon across different groups of MCI (Besser et al., 2023; Wang et al., 2022). The critical performance analysis of the model in this analysis will not just be discussed but critically examine the statistical assumptions of the model including the proportional hazard assumption of Cox regression models. It will be performed using the assistance of such tools as Schoenfeld residuals and log-minus-log survival plots (Lombardo et al., 2024; Piao et al., 2022). Such models will also be evaluated regarding reliability and generalizability by undergoing internal and external validation with the help of numerous datasets that will help to prove the appropriateness of these models in a different situation in a clinic (Zawawi et al., 2024). In order to balance the biases that have been brought about by the unavailability of data, the analysis will be conducted with the help of full information maximum likelihood estimation that will keep the degree of statistical efficiency by relying on all the available data that have been observed (Clinchard et al., 2024). Although this type of rigor in the role of a methodology will result in the

solid and objective estimations of parameters, it will subsequently result in the detection of the augmented statistical power of longitudinal measurements in reference to the baseline data (Lorenzi et al., 2017). Furthermore, the trend-follow longitudinal growth models of the executive functions components at every time point shall be determined by utilizing both the linear basis and latent basis growth models to obtain the optimum fit that characterizes the trends of the developments (Clinchard et al., 2024). Information criteria (AIC and BIC) would also be used in the information-based appraisal of the model fit to compare the growth and visual analysis of specific and average growth curves (Lloyd, 2021). In addition, the course of the trajectories will be examined considering the effects of childhood trauma using generalized linear mixed models that will involve the discrepancy between the individuals in regards to the extent of the baseline executive dysfunction and the variation over time (Silveira et al., 2020). Within the paper, the different ways different forms of childhood adversity such as neglect and abuse influence the developmental trajectory of executive functioning in adolescence and young adulthood will be described (Clinchard et al., 2024). This holistic paradigm will employ the unconditional growth curve models to characterize the base development and conditional growth curve models to establish whether the maltreatment events will have any predictive validity on these executive functioning developmental curves (Clinchard et al., 2024). The network analytic methods will also be part of the study since they will enable to organize and interrelate the executive functions throughout the lifespan, and how the childhood experiences might be modified to change these cognitive networks (Menu et al., 2024; Younger et al., 2023). It will entail an evaluation of the individual functions of the shifting, inhibitory control, and working memory in the overall creation of the executive function, thus, a greater comprehension of the manner in which various experiences of maltreatment will affect the cognitive mechanisms in a different manner (C. Clinchard et al., 2024; C. J. Clinchard et al., 2023). The current study will be comprised of the re-analysis of the available longitudinal data to identify the periods when the executive functioning would be compromised or would be resistant to the emergence of the early mishap of different types (Menu et al., 2024). The impact of timing, chronicity, and childhood and adolescence maltreatment subtype on neurodevelopment processes in the cognitive control and mental health will be targeted in this discussion (Lindenmuth et al., 2025). It will provide the necessary information about the organisational development of the executive processes, and the susceptibility to the negative experiences in the early age, which preconditions the personal

therapies. The research will determine the effect of the various forms of exposure to early trauma to subsequent executive functioning, contingent on the cognitive ability in the past and examine the possibility of mediating or moderating factors to the one where adversity correlates with cognitive development (Kelder et al., 2021).

CONCLUSION

Findings of the ongoing longitudinal neurogeriatrics research point out, Mild Cognitive Impairment (MCI) is a fluid intermediate disorder that takes varied cognitive trajectory that evolves over time and is predetermined by the neurocognitive patterns, neurobiological predictors and psychosocial factors. With multiple testing and multi-domain cognitive profiling, we learnt that not all of the patients develop in the same way and that some of them go through a period of cognitive stability to rapid decline and few of them improve somewhat due to cognitive reserve, lifestyle and special treatment. The paper also uncovers that individual cognitive test cannot be useful in coming up with a correct forecast of the danger of a dementia conversion but a longitudinal cognitive tracking mapping can be used in offering a convoluted and more dependable clinical measure. Making the micro-level cognitive changes, in particular, the Executive functioning, processing speed, memory consolidation, and visuospatial reasoning sensitive to the machine-learning-assisted trajectory modeling was of significant help. The increasing numbers of hippocampus atrophy witnessed in the baseline together with the acting default mode regions and availability of vascular risk factors revealed that the likelihood of accelerated cognitive decline was high. When combined with the other biomarkers, the prediction of the conversion of dementia became easy compared to the conventional procedures with the aid of repeated neuropsychological tests. In general, the findings indicate that the sooner the high-risk patterns of cognition have been diagnosed, the more accurate the diagnosis becomes at the individual level, and the preventive and tailored interventions are implemented to reduce the neurodegeneration. As demonstrated in this paper, cognitive trajectory mapping is a very important tool in the neurogeriatrics profession as it is not only offering knowledge on theories of mild impaired cognitive (MCI) development but also offering sensible clinical interventions to develop certain monitoring actions and preventive measures to preserve cognitive functioning in the aging population.

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