



T.C.
ÜSKÜDAR UNIVERSITY
INSTITUTE OF HEALTH SCIENCES

NEUROSCIENCE DEPARTMENT
NEUROSCIENCE MASTER'S THESIS PROGRAM
MASTER'S DEGREE THESIS

**THE NEUROPSYCHOLOGICAL FEATURES OF DEPRESSION AS
A COMORBIDITY IN PATIENTS WITH DEMENTIA**

Nisa Ülkü Ece ELÇİN

Thesis Advisor
Prof. Dr. Cumhuri TAŞ

İSTANBUL-2024

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ÖZET

DEMANSTA DEPRESYON EK TANISININ NÖROPSİKOLOJİK ÖZELLİKLERİ

Bu tez, demans hastalarında eşlik eden depresyonun bilişsel işlevler ve hastalığın seyri üzerindeki etkilerini nöropsikolojik testler aracılığıyla incelemektedir.

Bu araştırmanın amacı, demans hastalarında depresyonun ek tanısının hastalığın seyri üzerindeki etkilerini nöropsikolojik testlerle incelemektir. İzmir'de demans tanısı (CDR 1) almış 40 hasta üzerinde yapılan çalışmada, depresyon ek tanısı alan 20 kişi ile almayan 20 kişi karşılaştırılmıştır. Kullanılan testler arasında Standardize Mini Mental, Digit Span, Öktem Sözel Bellek Süreçleri Testi, Boston Adlandırma Testi, Stroop Testi, Geriatrik Depresyon Ölçeği ve Cornell Demansta Depresyon Ölçeği yer almaktadır. Betimsel analizler, cinsiyet, yaş, eğitim durumu, medeni durum ve el baskınlığı açısından gruplar arasında istatistiksel olarak anlamlı fark olmadığını göstermiştir. Depresyon ek tanısı olan hastaların bilişsel performanslarının daha düşük olduğu, özellikle Standardize Mini Mental Test ve Geriatrik Depresyon Ölçeği puanlarının anlamlı derecede düşük olduğu bulunmuştur. Öktem Sözel Bellek Süreçleri Testi'nde gruplar arasında fark bulunmazken, Stroop ve Boston Adlandırma Testlerinde depresyon ek tanısı olanların süre ve hata puanları daha yüksek çıkmıştır. Bu sonuçlar, depresyonun demans hastalarının bilişsel işlevlerini olumsuz etkilediğini ve bu nedenle tedavide depresyonun tespiti ve yönetimine yönelik stratejilerin geliştirilmesi gerektiğini vurgulamaktadır.

Anahtar Kelimeler: Demans, depresyon, bilişsel fonksiyonlar, bilişsel testler, nöropsikolojik testler, yaşlılık çağı

ABSTRACT

THE NEUROPSYCHOLOGICAL FEATURES OF DEPRESSION AS A COMORBIDITY IN PATIENTS WITH DEMENTIA

This thesis examines the impact of comorbid depression on cognitive functions and disease progression in patients with dementia using neuropsychological tests.

The study, conducted on 40 patients diagnosed with dementia (CDR 1) in Izmir, compares 20 individuals with comorbid depression to 20 without. The tests used include the Standardized Mini-Mental State Examination, Digit Span, Öktem Verbal Memory Processes Test, Boston Naming Test, Stroop Test, Geriatric Depression Scale, and Cornell Scale for Depression in Dementia. Descriptive analyses revealed no significant differences between the groups in terms of gender, age, education level, marital status, or hand dominance. However, cognitive performance was notably lower in patients with comorbid depression, especially in the Standardized Mini-Mental State Examination and Geriatric Depression Scale scores. While no significant differences were found in the Öktem Verbal Memory Processes Test, patients with comorbid depression scored higher in terms of time and errors in the Stroop and Boston Naming Tests. These findings highlight the detrimental effect of depression on cognitive functions in dementia patients and emphasize the need for developing strategies for the detection and management of depression in dementia treatment.

Keywords: Dementia, depression, cognitive functions, cognitive tests, neuropsychological tests, old age

PREFACE

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Endless THANKS.

Psy. Nisa Ülkü Ece Elçin

August 2024, Istanbul



DECLARATION FORM

I hereby declare that the research titled “Neuropsychological Characteristics of the Additional Diagnosis of Depression in Dementia”, which I have defended as a master's thesis, has been written by me in accordance with scientific ethics and traditions, and that the works I have benefited from consist of those shown in the bibliography, and that they have been used by citing them, and I confirm this with honor. I certify that the findings and results obtained during my thesis study are original and scientifically based, and that this study aims to contribute to academic knowledge.

2024

Nisa  lk  Ece EL İN

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LIST OF SYMBOLS AND ABBREVIATIONS

AD: Alzheimer's Disease

BNT: Boston Naming Test

BPSD: Behavioral and Psychological Symptoms of Dementia

CBT: Cognitive-Behavioral Therapy

CSDD: Cornell Scale for Depression in Dementia

CDR: Clinical Dementia Rating Scale

DSM-IV: The Diagnostic and Statistical Manual of Mental Disorders- Fourth Edition

DSM-V: The Diagnostic and Statistical Manual of Mental Disorders- Fifth Edition

FTD: Frontotemporal Dementia

GDS: Geriatric Depression Scale

ICD-10: International Classification of Diseases

MCI: Mild Cognitive Impairment

MDD: Major Depressive Disorder

NCD: Major Vascular Neurocognitive Disorder

NINDS-AIREN: National Institute of Neurological Disorders and Stroke-Association
Internationale pour la Recherche et l'Enseignement en Neurosciences

PFC: Prefrontal Cortex

PD: Parkinson's Disease

PDD: Parkinson's Disease Dementia

SMMSE: Standardized Mini Mental State Examination

VMPT: Verbal Memory Process Test

VaD: Vascular Dementia

WAIS: Wechsler Adults Intelligence Scale

WMS: Wechsler Memory Scale

1. INTRODUCTION

1.1. Problem State

Aging is a global phenomenon with a worldwide impact. Both in the world and in Turkey, the aging process exhibits similar characteristics. The definition of aging includes beliefs that express the capabilities of an individual in a certain age range and what they should do. In other words, it is the acceptance of the capacities and limitations associated with age (Hooyman & Kiyak, 2018, p. 252). Age at birth or calendar age, also referred to as chronological ageing, is generally used to describe individuals aged 65 years and older (World Health Organization, 1984, p. 8). Looking at the world population today, it is observed that the elderly population is three times higher than it was in the twentieth century (Ari, 2023 pp. 261-278). The population over the age of 65, which currently constitutes 10.2% of the total population in Turkey, is increasing rapidly (TÜİK, 2023). Despite this rapid increase, other age groups are not increasing at the same rate. The increase in the elderly population is an important problem both in Turkey and around the world. This aging process is usually accompanied by various health problems (Bilir & Paksoy Subaşı, 2006). In the aging process, the frequency of chronic diseases generally increases in individuals. Among these, health problems such as hypertension, diabetes, heart diseases, osteoporosis, cancer and cognitive disorders are common. Mental health problems such as depression, anxiety and dementia can be common (Gupta C, Prakash D., 2015). In addition, the aging process can also lead to a weakened immune system, reducing resistance to infections. When these factors come together, the incidence of health risks and diseases increases in older people.

Depression is a common mental health problem in the elderly in addition to physical health problems (Şahin, 2000). Depression poses a significant threat to the well-being of individuals in this age group, making it the most common mental problem. It leads to serious disability and reduced quality of life (Serby and You, 2003). In particular, it can be accepted that depression can sometimes be an early symptom of dementia, especially in older individuals (Göktaş & Özkan, 2006; Serby & You, 2003). Dementia and depression are common mental health problems that deeply affect individuals' cognitive

functions and overall quality of life. It is not unusual for these two conditions to coexist and this is a scenario frequently encountered in clinical practice. Cognitive impairments reported in senile depression may be similar to subcortical dementias and may negatively affect the course of these disorders (Alexopoulos et al., 2000). Overlapping symptoms of dementia and depression may complicate the diagnostic process and this may affect the treatment and care plans of individuals. Considering both the neuropsychological symptoms of dementia and the cognitive effects of depression, the coexistence of these two conditions may have different and significant effects on cognitive functions. Neurophysiologic tests may help to differentiate between depression and dementia (Dykieriek et al., 1998; Newman, 1999). The diagnosis of depression in older adults is often overlooked for various reasons. This overlooking is due to lack of awareness or ignorance of the illness by the patient and family. In addition, symptoms of depression are often attributed to other causes such as physical illness or old age (Tamam and Öner, 2001; Penninx et al., 2000). Although it is a highly treatable condition, depression is not easily recognized. Patients tend to express physical complaints more easily than depressive moods. In addition, the presence of dementia and other physical disorders complicates the diagnostic process (Göktaş and Özkan 2006). Failure to address depression in older adults can lead to detrimental consequences such as isolation from social interactions and decreased overall well-being. In addition, untreated depression places a greater burden on both families and society, causes premature death, increases the risk of suicide, decreases functional abilities and impairs general health (Eker and Noyan 2004). However, by promptly identifying and addressing depression in elderly individuals, their quality of life can be improved, their level of independence in daily activities can be maintained and the risk of premature death can be reduced (Serby and Yu 2003).

The epidemiology of depression in old age is less well studied in contrast to the epidemiology of dementia. It is believed that symptoms of depression are more common among older people, possibly due to the large number of losses experienced at this stage of life. These losses can manifest in a variety of ways, including deteriorating health, reduced organ function, strained relationships and changes in social status. In addition, it is possible that the reality of death, which is getting closer and closer in old age, may further contribute to these losses (Eker E and Noyan 2004).

Older people often find ways to hide their depression and may even deny that they are experiencing any symptoms. This denial is a common occurrence among older people and can prevent doctors from detecting physical or mental health problems in this population. Some older people use phobic defense mechanisms to escape depression. For example, in an attempt to cope with the fear of aging and death, some older people may envision themselves as eternal beings unaffected by the passage of time. Isolation is the general name given to actions taken to protect a certain environment, an object or an individual from external influences. Social isolation, which is considered as one of the types of isolation, is a defense mechanism that individuals use to cope with emotional changes. This type of isolation weakens the individual's connections with other people and leads to isolation, which can increase depression. In older people, somatization is often used to describe depression. Somatization is a process in which mental distress turns into physical symptoms, and in this case, physical complaints become more prominent (Eker & Noyan, 2004).

Mood disorders, including depression, do not originate in a single region of the brain. Mood regulation relies heavily on the prefrontal cortex, amygdala, hippocampus, thalamus, basal ganglia and the connections between these areas. There are two important neural circuits involved in this process. The first is the limbic-thalamic-cortical circuit, which includes the amygdala, the mediodorsal nucleus of the thalamus and the medial and ventrolateral prefrontal cortex. The second is the limbic-striatal-pallidal-thalamic-cortical circuit involving the striatum, ventral pallidum and their respective connections. These circuits play a very important role in the regulation of mood (Soares, & Mann, 1997).

Cognitive impairments associated with neuropsychological disorders associated with depression primarily affect right hemisphere abilities such as visual structuring, non-verbal memory and pre executive functions. These impairments specifically target the prefrontal cortex (PFC), which acts as a hub connecting various regions of the neocortex through cortico-cortico connections. This extensive network allows the integration of multimodal information within a large associative space. In addition, the PFC maintains connections with limbic and subcortical structures, including the cingulate gyrus, hippocampus, basal ganglia and thalamus. The PFC plays a crucial role in prioritizing stimuli, directing attention, modifying cognitive structure, initiating voluntary responses, inhibiting inappropriate responses, working memory, integrating internal representations

of stimuli, determining the sequence of events, generating abstract concepts, and other executive functions. Especially in both depression and schizophrenia, disorders affecting these functions are observed (Yüksel, 2002).

Patients with depression show markedly decreased performance on memory and concentration tests and slowed mental processes. This poor performance on detailed neuropsychological tests was first reported by Cavenar et al. in 1979 and then by Sweet et al. in 1983 (as cited in Veiel, 1997). Patients with depression have an inability to reevaluate and use the material they remember. However, when they are given clues on how to use this material, they are able to fulfill this task. This suggests that patients with depression have difficulty in performing mental operations that would enable them to reuse information stored in memory. In addition, lack of focus in learning tests and a tendency to perseverate in unverified hypotheses are also observed (Johnson & Magaro, 1987; as cited in Yüksel, 2002).

The possibility that cognitive impairments in depressed patients may be due to lack of motivation has also been examined. In general, there is a negative correlation between patient effort and the severity of depression. Regardless of what information is stored in memory, the willingness to report this information may also be an important factor in memory impairment. For example, patients with depression either avoid responding or make more transposition errors. Patients with dementia tend to give irrelevant responses. Patients with depression are more cautious and avoid responding even though they know. This may be a problem with assessing the environment in terms of safety rather than a real impairment. In addition, the hedonic tone of events may also directly affect the recall process of depressed patients. Depressed patients tend to remember negative events more (Johnson & Magaro, 1987; as cited in Yüksel, 2002).

Decreased cognitive functioning and decreased attention are prominent in patients with depression. Individuals with depression have difficulty maintaining motivation for tasks, resulting in below-average performance in various cognitive domains. In particular, their performance on tests is worse compared to their daily functioning. In addition, their overall performance may be further impaired when they receive negative feedback on their test performance. In terms of memory, people with depression differ from people with dementia. Their learning ability remains intact, allowing them to perform well on tasks involving cues or multiple-choice recall. However, their performance on tasks

requiring free recall and recall is compromised by impaired attention and reduced motivation. It is common for people with depression to complain of memory problems. But when assessed using memory tests, there is no consistent pattern of memory impairment seen in dementia. Instead, they have difficulty recalling past events in a fragmented way. While speech may slow down, people with depression usually have no difficulty with word finding or anomia. Naming, word production and verbal fluency remain unaffected (Pfennig et al., 2007).

Impairments in recognition memory, executive functions, psychomotor speed, memory, attention and language functions are emphasized more prominently in elderly depression cases (Purcell et al., 1997). However, a definite evaluation cannot be made because the results of the studies do not show a complete harmony. Especially in terms of short-term memory and psychomotor speed disorders, all studies seem to be compatible. This concordance is more evident in severe cases and is due to the fact that the age, education, diagnosis and intelligence levels of the patient and control groups were not adequately matched (Yüksel, 2002).

Dementia refers to the deterioration in cognitive functions as a result of damage to the central nervous system of an adult person. This condition involves the gradual loss of mental and social abilities at a level that can affect the person's daily life (Gürvit, 2004). Dementia, which initially manifests with memory problems, may affect other abilities such as attention, language, visual abilities, perception and problem solving over time. Personality changes, behavioral problems and psychiatric symptoms may also occur over time. Definitions of dementia usually include basic features such as progressive mental deterioration, involvement of more than one cognitive function and absence of impaired consciousness (Rossor, 2000). Diagnostic criteria for dementia include features such as cognitive impairments being prominent, affecting activities of daily living, and not being explained by other medical conditions or drug use.

There are significant differences between the diagnosis and categories of dementia in DSM-IV (1994) and DSM-V (2013). In DSM-IV, dementia is classified under the heading “Delirium, Dementia, Amnestic, and Other Cognitive Disorders”. In this system, dementia is defined on the basis of the presence of memory impairment and other cognitive impairments, and specific types of dementia (e.g. Alzheimer's type, Vascular dementia) are given as separate categories. In contrast, in DSM-V, the term dementia was

renamed “Major Neurocognitive Disorder” and defined to encompass a broader spectrum of cognitive impairments. In DSM-V, dementia is included under the heading “Neurocognitive Disorders” and is divided into two main categories: major and minor neurocognitive disorders. Major neurocognitive disorder is diagnosed when there is significant cognitive decline and this decline affects activities of daily living, while minor neurocognitive disorder is defined by milder cognitive decline and does not fully affect activities of daily living. Furthermore, the DSM-V sub-classifies cognitive impairments according to their cause (e.g. Alzheimer's disease, Vascular disease, Traumatic brain injury). These changes allow DSM-V to assess dementia and other cognitive disorders in a more comprehensive and flexible way. While the concept of dementia in DSM-IV was more rigid and specific, DSM-V takes a broader and inclusive approach.

Dementias are usually classified as primary and secondary. Primary dementias are types of dementia caused by neurodegenerative diseases. These diseases cause specific pathological changes in the brain, leading to the loss of neurons and synapses, thus leading to impaired cognitive function. Alzheimer's disease is the most common cause of primary dementia. Secondary dementia, on the other hand, is usually of vascular origin and is usually associated with problems related to blood circulation (Gürvit, 2004).

Dementia and depression are two important psychiatric conditions that are common among the elderly population. Dementia, sometimes referred to as memory impairment, has neuropsychiatric features that are significant enough to cause a decrease in the ability to perform social or occupational activities (Cipriani et al., 2015). Both conditions can significantly affect an individual's quality of life and daily functioning. The most important common feature of dementia and depression in old age is the loss of cognitive functions in both. In both conditions, cognitive functions are affected differently, but in cross-sectional assessment, the clinician needs to take a good anamnesis from the patient and the patient's relatives, a detailed assessment and often neuropsychological evaluation to make a differential diagnosis. Dementia and depression are often interrelated and it is quite common for both conditions to co-exist in an individual. This may indicate a variety of neuropsychological factors that may play an interactive role in both the progression of dementia and the course of depression (Sadavoy, 2004). The neuropsychological features of dementia and depression have attracted the attention of researchers. In particular, impairment of cognitive functions, memory loss, attention deficit and changes in decision-making ability are common features that characterize both conditions. The

effects of depression on dementia may worsen an individual's cognitive abilities, while neuropathological changes caused by dementia may increase the severity of depression.

Episodic and semantic memory are significantly more impaired in dementia than in depression. Subjects with depression do not encode information with the same intensity as non-depressed individuals and perform less well on tests requiring effortful encoding. In addition, the process of forgetting progresses at the same rate among individuals with depression and non-depressed individuals, whereas it progresses faster in dementia patients. The 2000 study included participants with Alzheimer's type dementia with "mild" cognitive impairment, participants with depressive pseudodementia and a control group. Pseudodementia is used to describe a clinical picture that resembles organic dementia, but for which no physical cause can be found, but in which the impairment in cognitive functions improves as a result of treatment of existing depression (Jenike, 1995; Yaluğ et al., 2006). While patients with Alzheimer-type dementia have significant impairment in temporal orientation, visual structuring and visual memory tests, depressive pseudodementia patients have normal results in these tests. These differences are important in differentiating these two conditions. While Alzheimer-type dementia presents a memory profile consistent with cortical dementia, depressive pseudodementia patients exhibit a memory profile similar to subcortical dementias (Crowe and Hoogenraad, 2000).

In a 1997 study by Nahas et al. a total of 320 elderly depressed subjects were examined, including 201 with dementia. Regardless of the presence or absence of dementia, the subjects showed significant improvement with treatment. However, the improvement was less in patients with depression with dementia than in others.

A meta-analysis published by Veile (1997), based on studies published after 1975 with strict methodological criteria, examined neuropsychological deficits in individuals with major depression but otherwise unimpaired. Individuals with major depression have been shown to have a generalized impairment of brain functioning, particularly associated with involvement of the frontal lobes. In tests of attention and concentration, he reported that there was no difference between the depression and control groups in terms of simple attentional functioning, although depression cases tended to be below average. In verbal fluency and visuomotor functioning, the performance of depressed subjects ranged widely compared to the control group. On tests measuring visual motor function,

depressed subjects tended to perform worse than controls. There is a difference between the depression and control groups, to the detriment of moderately depressed subjects. In verbal and non-verbal learning tests, the rate of forgetting is higher in depressed subjects. In mental flexibility tests (Trail B and Stroop), depression cases show significantly lower performance.

Austin et al. (1999) compared the test performance of melancholic and non-melancholic depression cases and found that all depression cases performed poorly on memory tests. This impairment was particularly evident in verbal and visual learning and recall tests. Reaction time was also significantly impaired. However, performance was preserved on tests of number sequences, verbal recognition and psychomotor speed. No significant impairment was found in the Stroop test, digit span test, verbal fluency test, WAIS similarities subtest and WCST. In melancholic cases, prolongation in reaction time and impairment in psychomotor speed tests were more pronounced.

In a 3-year longitudinal study by Berger et al. (1999), individuals who eventually developed symptoms of dementia exhibited a higher prevalence of depression symptoms at the beginning of the study compared to those who did not develop dementia. Moreover, individuals diagnosed with dementia were more likely to have motivation-related symptoms such as apathy, low energy and difficulty concentrating. Conversely, these symptoms were less common in older people without dementia. This finding is consistent with cross-sectional studies showing that apathy-specific symptoms occur at a higher rate in people with dementia than in people without cognitive impairment.

Yaffe et al. (1999) examined the relationship between depression and cognitive impairment in elderly women. Within the scope of the study, depression and cognitive impairment levels of women were evaluated at 4-year intervals. The results showed that cognitive impairment was significantly higher in individuals who were depressed at baseline than in those with few or no depressive symptoms. Cognitive impairment was observed in all participants during the follow-up period. Furthermore, a positive correlation was found between more depressive symptoms and being diagnosed with dementia during the follow-up period.

In a study by Butters et al. (2000), elderly depression cases without dementia were evaluated with attention, concept formation, structuring and memory tests using the Mattis Dementia Rating Scale and Mini Mental State Examination before and after

treatment. Patients were evaluated after 12 weeks of antidepressant treatment (nortriptyline or paroxetine). With treatment, these impairments improved to some extent, but not to normal levels. In particular, memory and executive functions do not reach normal levels. This suggests that late-onset depression may be related to subcortical/frontal lobe circuits. There is an association between late-onset depression, white matter hyperintensities and executive dysfunction.

Jean (2005) conducted a retrospective study spanning 7.5 years while comprehensively examining 44 patients suffering from depression. The findings revealed that 14 of the total participants eventually developed dementia and exhibited notable declines in attention and memory abilities during follow-up. Compared to older individuals without a diagnosis of dementia, those who met the criteria for dementia performed significantly worse. Among 7 people with dementia diagnosed with probable Alzheimer's disease (AD), scores on orientation tasks at the start of the study were particularly low compared with depressed older individuals diagnosed with other types of dementia.

Ganguli et al. (2006) conducted an epidemiologic study focusing on the general population. The study included 1265 participants without dementia who were assessed every two years. The findings revealed that depression played a role in explaining baseline cognitive scores, particularly in the areas of recall memory, visuospatial ability and executive functions. However, these low baseline scores did not affect learning or language compositions. Interestingly, it was only people who were later diagnosed with dementia who showed a decline in cognitive performance over time. This suggests that conducting sequential cognitive assessments may be valuable in establishing a clear diagnosis.

Published in 2011 in *Arch Gen Psychiatry*, the study by Ge Li et al. was conducted with 3410 participants who were at least 65 years old and did not have dementia. This study examined the association between the risk of dementia and baseline depressive symptoms and confirmed that depression at older ages is associated with increased risk of dementia. However, no association was found between depression at an early age and the development of dementia. Depressed mood and perceived performance difficulty were independently associated with dementia. These findings suggest that depression in older age may be an early symptom of dementia.

In 2015, a study by Snowden and colleagues published in the American Journal of Geriatric Psychiatry included 27,776 participants with dementia, mild cognitive impairment and normal cognitive level. The aim of this study was to investigate whether the frequency and severity of depression was higher in individuals with dementia than in those with mild cognitive impairment or normal cognitive level. The findings showed that depression was significantly higher in individuals with mild cognitive impairment or a diagnosis of dementia, both in the present and in future analyses. These results emphasize that efforts to treat older people with dementia should also address comorbid depression.

A review of the literature reveals that both disorders have been the subject of many studies due to the similarity of their clinical symptoms. Depression is a common psychiatric disorder in the elderly and is often associated with dementia. Recognizing this association is of vital importance for the treatment of the elderly, as depression in the elderly may mimic or exacerbate dementia. The aim of this study was to examine the effects of an additional diagnosis of depression on neuropsychological tests in individuals diagnosed with dementia.

1.2. Importance and Purpose of the Research

This research can help us understand how the comorbidity of depression in patients with dementia affects the course of the disease and treatment management. This knowledge may help us to develop more effective diagnostic methods and individualized treatment strategies. Depression can significantly affect the quality of life of people with dementia, and this research can identify the effects of the coexistence of these two conditions on quality of life. Understanding the effects of depression on the progression of dementia may help us to develop more effective strategies for disease management. Furthermore, understanding the societal burden of patients with coexisting dementia and depression is important for health policy making and resource allocation. The relationship between dementia and depression is complex and multifaceted. Neuropsychological research in this area can provide important information to develop more effective intervention strategies at both clinical practice and community level. It may also contribute to better diagnosis and treatment approaches in patients with these conditions.

1.3. Research Hypotheses

H1: Attention and processing speed are lower in dementia patients with comorbid depression compared to dementia patients without comorbid depression.

H2: Verbal learning skills are lower in dementia patients with comorbid depression compared to dementia patients without comorbid depression.

H3: Recall skills are lower in dementia patients with comorbid depression compared to dementia patients without comorbid depression.

H4: Naming skills are lower in dementia patients with comorbid depression compared to dementia patients without comorbid depression.

H5: Attention and concentration efficiency is lower in dementia patients with comorbid depression compared to dementia patients without comorbid depression.

H6: Educational level of dementia patients with comorbid depression is lower than dementia patients without comorbid depression.

1.4. Assumptions of the Research

The assumptions of the study in terms of sampling, methodology and data analysis are as follows:

1. It is assumed that the scales used in the study are valid and reliable scales to measure the research variables.
2. It is assumed that the relatives of the patients participating in the study filled out the scale sincerely and that the answers they gave were valid and reliable answers about themselves and their patients.
3. It is assumed that the answers given by the dementia patients participating in the study during the application are valid and reliable.
4. It is assumed that the sample group of people diagnosed with dementia and their relatives in Izmir province represents the general dementia patient population.
5. It was assumed that external factors (e.g., participants' mood, environmental factors) were minimally influential during data collection.

6. Within the scope of the study, the CDR scale was used to diagnose dementia; for patients diagnosed with depression, the diagnosis was made by physicians referred in accordance with DSM-V diagnostic criteria. It was assumed that the diagnoses made by physicians were reliable and valid.

7. It is assumed that the statistical data analysis methods used to reach the findings on the variables in the study are adequate, valid and reliable methods to determine the findings.

1.5. Limitations of the Research

The most important limitation of the study is that it is limited to the information obtained from people diagnosed with dementia and their relatives in Izmir province. The research data is limited to the information obtained from 40 dementia patients and their relatives. In addition, the study is limited to the sociodemographic data form, Standardized Mini Mental State Examination, Digit Span Test, Boston Naming Test, Öktem Verbal Memory Process Test, Stroop Test, Geriatric Depression Scale and Cornell Scale for Depression administered to the patients and their relatives. However, in our country, where studies on geriatric samples are extremely limited, it is thought that such interdisciplinary studies will be a good model for future research. DSM-V diagnostic criteria were used to diagnose depression in the depression group, and the physician who referred the patient to the study made the diagnosis. Another limitation of the study is that a diagnostic structured interview scale such as the SCID was not used during the diagnosis of depression.

2. GENERAL INFORMATION

2.1. Dementia Syndrome

Dementia is characterized by the impairment of cognitive functions, particularly memory, thinking, orientation, comprehension, calculation, learning capacity, language, and judgment, to the extent that it affects an individual's daily life (Akdemir, Birol, 2011, pp. 839-843; Akça, et al., 2014, pp. 267-274; Şentürk & Küçüküçlü, 2015, pp. 190-194; Göktaş & Özkan, 2006, pp. 30-37). The incidence of dementia significantly increases with age, with prevalence doubling every five years after age 60 (Karaman, 2005, pp. 53-61; Akın, et al., 2001, pp. 33-39). In physically healthy individuals, the prevalence of dementia is approximately 5% between the ages of 65 and 75, with this rate rising to 10% above 75 years of age and reaching 20% above 80 years of age. The risk of dementia doubles every five years between the ages of 65 and 85 (Ineche 1987; Rossor 1991 as cited Aydemir, Kısa, 2001). Among elderly individuals who have been hospitalized for any reason, dementia is detected at a rate approximately six times higher than in the general population, about 30% (Ineche 1987 as cited Aydemir, Kısa, 2001).

Dementia is a syndrome that can result from a series of diseases that progressively destroy nerve cells and damage the brain, leading to cognitive function deterioration beyond what is expected from normal biological aging. Although consciousness is not affected, cognitive impairment is often accompanied or preceded by changes in mood, emotional control, behavior, or motivation (WHO, 2023). According to a 2023 report by the World Health Organization (WHO), there are over 55 million people with dementia worldwide, with more than 60% living in low- and middle-income countries. Approximately 10 million new cases are reported annually.

Data from the Turkish Statistical Institute (TÜİK) indicate that in 2023, the proportion of the elderly population aged 65 and over in Turkey will exceed 10%, and the prevalence of dementia diseases is expected to continue increasing rapidly. If this trend continues, the number of people with dementia worldwide is projected to reach 135 million by 2050.

The WHO's 2023 report also discusses various risk factors that increase the likelihood of developing dementia. Age is a primary risk factor, with dementia being more common among individuals aged 65 and older. Additionally, cardiovascular risk factors such as hypertension and high blood sugar levels (diabetes) increase the risk of dementia. Excessive weight or obesity, coupled with unhealthy lifestyle habits, negatively impacts brain health, thus increasing the likelihood of dementia. Harmful habits such as smoking and excessive alcohol consumption can also contribute to the development of dementia by directly affecting brain function. A physically inactive lifestyle and social isolation can have negative effects on brain health, further increasing the risk of dementia. Finally, psychiatric conditions such as depression are considered both an independent risk factor for dementia and an accelerating factor for its progression.

Dementia is categorized into three stages: early, middle, and advanced. In the early stage, patients may begin to forget names, have difficulty finding items, and feel the need to make lists to avoid forgetting things. There is also a tendency to forget newly acquired information. At this stage, impairments in recent memory are noticeable, and difficulties with word-finding and disruptions in speech content and fluency occur. Patients can still perform daily activities but face challenges with financial transactions and complex decision-making processes. In the middle stage, memory loss increases, distant memory is affected, and spatial orientation deteriorates, leading to disorientation in unfamiliar places, difficulty with perception, and significant challenges in word-finding. Additionally, psychotic symptoms such as delusions and hallucinations, various behavioral changes, and decreased independence in life skills become apparent. In the advanced stage, patients may not recognize even family members, cannot adjust speech according to time and place, confuse the locations of rooms in their home, and get lost even in familiar places. The person may exhibit aimless wandering, agitation, and repetitive behaviors, leading to complete dependency (Akyar, 2011, pp. 79-88; Özdemir & Özdemir, 2013, pp. 482-486).

2.1.1. Types of dementia

Dementia can be classified into two main categories based on clinical classification: primary and secondary dementia. Primary dementia refers to types of dementia that arise directly from degenerative processes occurring in the brain. In these forms of dementia, irreversible damage occurs in brain cells, leading to a progressive and permanent decline

in cognitive functions. Examples of primary dementia include Alzheimer's disease, Lewy body dementia, and frontotemporal dementia. The primary causes of these dementias are often genetic predisposition, aging, and abnormal accumulation of specific proteins in the brain. Neurodegenerative dementias are age-related, with prevalence estimated at 20% among individuals over 85 years old (Hebert, et al., 2003; Love, 2005).

Secondary dementia, on the other hand, refers to types of dementia that arise as a result of another disease or condition. The underlying causes of secondary dementia are often treatable or manageable. For example, brain trauma, chronic alcoholism, infections (such as HIV or syphilis), metabolic disorders (such as hypothyroidism or vitamin B12 deficiency), or poisoning can lead to secondary dementia. The development of this type of dementia is generally associated with systemic or environmental factors that affect brain function rather than direct pathology in the brain itself (Roman, 2003; Langa et al., 2004).

In summary, primary dementia results from degenerative processes directly within the brain, while secondary dementia arises from other health issues or external factors.

The most common types of dementia are as follows:

2.1.1.1. Alzheimer's disease

Alzheimer's disease (AD), characterized by a gradual decline in cognitive functioning, is one of the most common types of dementia worldwide (Hill et al., 2009). Studies show that the incidence of AD increases with age (Alzheimer's Association, 2013). Although AD has a proportional increase with age, it is not associated with normal aging; rather, it is a result of pathological brain disorders (Küçükgüçlü, et al., 2013)

The neurobiology of Alzheimer's disease is primarily related to the damage and dysfunction of neurons in the brain. The disease typically begins in the entorhinal cortex and progresses to the hippocampus, a region critical for memory. During this process, synaptic dysfunction and neuronal degeneration occur (Öber, A., 2008).

One of the fundamental mechanisms of the disease is the accumulation of beta-amyloid plaques in the brain. These plaques impede communication between neurons, leading to a decline in cognitive functions. Additionally, the reduction in

neurotransmitters such as acetylcholine adversely affects memory and learning processes (Öber, 2008).

Alzheimer's disease also has genetic components; notably, the APOE-e4 gene is thought to play a significant role in the disease's development. Aging is the most significant risk factor for the disease, and genetic predisposition may increase the likelihood of developing Alzheimer's in aging individuals (Durnagözü, 2021; Yılmaz, 2020).

AD is a neurodegenerative process whose exact cause and pathogenesis are not fully understood, especially in the elderly. AD is the most common primary degenerative dementia and is characterized by impairment in cognitive functions such as memory, language, attention, executive and gnostic-praxic functions (Braak et al., 2012; Hannay et al., 4th edn, pp. 133-156). AD usually begins at older ages and its prevalence doubles every five years over the age of 65 (Braak et al., 1997). Early-onset AD (under 60 years of age) is rare, and these patients mostly present to the outpatient clinic with concerns related to work performance. It is known that cognitive performance is worse in the uneducated and undereducated population and even the incidence of AD increases (Lee et al., 2008; Prencipe et al., 1996).

The increase in the elderly population worldwide increases the prevalence of AD. Especially in developed countries, a significant increase in AD cases is observed with the increase in the elderly population. However, genetic factors may also play a role in the development of the disease. Certain variants of the APOE gene may increase the risk of Alzheimer's disease (Vilatela, et al., 2012).

AD is more common in women than in men. However, this gender difference may be due to the fact that women generally live longer in old age (Akpınar, 2009). Finally, the prevalence and impact of AD is a global health problem that seriously affects health systems and economies worldwide (Alicılar, and Çalışkan, 2021). Therefore, the epidemiology of AD poses a significant burden on health policy makers and societies.

The clinical course of Alzheimer's disease usually progresses in three stages (Capezuti, et al., 2011; Işık and Tanrıdağ, 2009). In the first stage, the early stage, symptoms are usually mild and may be difficult to recognize. Symptoms such as memory impairment, attention deficit, mild problem-solving difficulties and language disorders may occur, but activities of daily living can usually be maintained.

In the middle stage, symptoms become more pronounced as the disease progresses. Memory loss increases, language ability deteriorates, complex tasks become difficult and personality changes may occur. Independence in daily activities decreases during this period.

In the advanced stage, the symptoms become serious and dramatic. There is profound memory loss, greatly reduced speech and language skills, difficulties with physical movement and marked personality changes. At this stage, it can become almost impossible to carry out activities of daily living.

The course of AD can vary from person to person and may progress at a different pace for each individual. In general, however, as the disease progresses, symptoms increase and the individual's functionality decreases. This process can take months or years, and treatment and support are important to slow the progression of the disease or manage symptoms.

According to the DSM-V diagnostic criteria for AD, the diagnosis is usually made by observing certain symptoms in the individual. The most common initial symptom is memory impairment. This can often manifest as difficulty in learning new information, difficulty in recalling previous information, or confusion with familiar places and objects. Impairment of at least one cognitive domain other than memory is required. These may include language ability, executive functions, visual perception and spatial skills. The cognitive impairment must be sufficient to affect activities of daily living. For example, there may be a marked decline in performance in work or social activities. Symptoms must be severe enough to significantly impair the individual's ability to function. Other neurological or psychiatric disorders should not explain these symptoms.

Distinctive findings in AD include impaired recent memory, learning difficulties and irregularities in cognitive functioning. Memory problems are usually associated with frontal type amnesia and are more closely related to attention, recall, organization and regulatory factors than to the storage process. Performance is variable and unpredictable. While the frequency of spontaneous speech is reduced, grammatical accuracy is usually preserved and paraphasia is rare. Semantic disturbances are common and comprehension is usually unaffected except for complex sentences. Impaired reading aloud skills may be seen. Late recall tests are the most sensitive in distinguishing early Alzheimer-type dementia from normal aging. Following impairment of semantic abilities, phonological

and syntactic abilities are usually initially preserved. Visual spatial abilities may also be affected early on and patients are often unable to recognize faces. This impairment is due to irregularities in perceptual abilities and memory dysfunction (Pasquier, 1999).

The definitive diagnosis of AD is usually made by a specialist, using a variety of assessment methods, such as neurological examination, cognitive tests and sometimes imaging studies. The combination of these methods helps in the diagnosis of AD.

2.1.1.2. Vascular dementia

Vascular dementia (VaD) accounts for approximately 17-20% of all dementia cases. It is the second most common type of dementia after AD. It may also be referred to as major vascular neurocognitive disorder (NCD) (Rockwood et al., 2000). The neuropathological classification of VaD is based on the idea of classifying lesions in the brain, such as ischemic and hemorrhagic lesions, according to the factors that lead to dementia (Erkinjuntti et al., 1987; Tatemichi, 1990). VaD syndrome is characterized by multiple infarct dementia, strategic single infarct dementia, lacunes due to small vessel disease, senile leukoencephalopathy, multiple lacunar strokes in the basal ganglia and pons, multiple white matter lacunes, Binswanger's disease is classified as hemorrhagic lesions resulting from hypoperfusion, hemorrhagic dementia (cerebral hematomas of vascular origin including amyloid angiopathy, chronic subdural hematoma, SAK) (Chui He et al., 1992; Fields, 1986).

Newman (1999) gives depression in VaD as 21%. It should be kept in mind that these rates are point prevalence.

Conditions such as stroke and cerebral small vessel disease (cSVD) disrupt neurovascular coupling and the integrity of the blood-brain barrier (BBB), leading to inflammation and oxidative stress. These alterations can exacerbate cognitive decline by impairing neuronal health and connectivity (Lecordier et al., 2021; Vinciguerra et al. 2020).

Ischemic events, particularly those affecting small vessels, can result in microinfarcts and white matter changes, which are significant contributors to cognitive deficits seen in VaD. The accumulation of such lesions over time is linked to progressive neurocognitive decline (Kalaria, 2016; Vinciguerra et al. 2020).

Increased neuroinflammatory responses in the brain are associated with vascular injuries. This inflammation can further damage neuronal cells and contribute to cognitive dysfunction (Vinciguerra, et al., 2020).

The imbalance between reactive oxygen species and antioxidants in the brain can lead to neuronal damage, exacerbating cognitive impairment in individuals with VaD (Vinciguerra, et al., 2020).

The true prevalence of VaD is difficult to determine as some patients may have other causes of dementia prior to stroke. Approximately 25% of individuals have onset of dementia after stroke, and the risk of developing the condition within the first five years is nine times higher compared to peers in the same age group (Barba R. et al., 2000; Desmond et al., 2000; Kokmen et al., 1996; Sacco et al., 1994). Community-based studies have found that the prevalence of VaD doubles every five years, particularly among men (Leys, et al, 1998).

Some scholars argue that psychiatric symptoms are significantly higher in VaD cases, especially in moderate and severe cases. Some researchers have also found a correlation between the severity of dementia and the presence of these symptoms. It has been suggested that depression may be a risk factor for VaD cases (Hebert et al., 2000). Compared to AD, VaD cases exhibit significantly higher levels of depression and behavioral symptoms, and the duration of depression tends to be longer in VaD patients (Ballard et al., 2000; Simpson et al., 1999).

Since the clinical picture seen in VaD varies depending on the etiology, the cognitive profiles of patients with VaD do not exhibit a uniform pattern (Bayles, 2013; Lezak, 2004).

Individuals with VaD generally have better verbal learning and memory performance, higher delayed recall performance and lower rates of forgetting, incorrect word recall and confusion. Memory impairments are more often seen as reduced recall, but recognition or cued recognition abilities may remain intact (Rockwood, 2002).

The main impairments observed in VaDs are related to mental processing speed and cognitive functions such as goal setting, task initiation, planning, organizing information, sequencing, and the capacity for abstract thinking. Similarly, attention and executive functions, including goal setting, initiation, planning and organization, are

expected to be affected in cases of subcortical ischemic dementia, subcortical hemorrhages or isolated subcortical strategic infarction. In addition, information processing speed and abstract thinking abilities are also impaired (Sellal, 2004).

Aphasia, which can develop in the left perisylvian region as a result of vascular injury, can impair language expression and formulation, leading to difficulties in communication. People with subcortical ischemic damage may experience a range of symptoms including dysarthria, motor weakness, gait disturbances, executive dysfunction, changes in the tone, pitch and speed of language movements (Bayles K., 2007). VaD is also often characterized by mood swings, depression, changes in personality and fluctuations in emotional states (Rockwood, 2002).

There are many diagnostic criteria for VaD. These criteria are evolving as our knowledge of cerebrovascular diseases and their impact on cognitive function increases. Various clinical criteria are used to diagnose VaD, including DSM-IV, the International Classification of Diseases (ICD-10), and the National Institute of Neurological Disorders and Stroke-Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS-AIREN) (American Psychiatric Association, 1994; McKhann, et al. 1984; Roman, et al. 1983). However, there are some differences between these criteria. For example, DSM-IV and ICD-10 criteria do not require radiological examinations for the diagnosis of VaD and do not take into account white matter lesions detected on brain imaging.

According to the NINDS-AIREN (1993) criteria, certain conditions must be met to diagnose probable VaD. First, the presence of dementia without certain exclusion criteria is required. These exclusion criteria include conditions that interfere with neuropsychological examination, such as impaired consciousness, delirium, psychosis, severe aphasia and sensori-motor changes, and systemic or brain diseases that may explain cognitive changes (e.g. AD). In addition, cerebrovascular disease must be present and there must be an association between dementia and cerebrovascular disease. This association may be dementia onset within 3 months after stroke or sudden deterioration or fluctuation of cognitive functioning.

Clinical features consistent with a diagnosis of VaD include early gait disturbance, unsteadiness or spontaneous falls, urinary symptoms such as frequent urination or urgency to urinate that cannot be explained by urologic causes, pseudobulbar paralysis,

and personality and mood changes. Depression, emotional incontinence, psychomotor retardation or executive dysfunction may also be seen.

The features that make the diagnosis of vascular dementia unlikely are early memory impairment and progressive deterioration in cognitive function without lesions consistent with brain imaging, absence of other focal neurological findings, and absence of cerebrovascular lesions on CT or MRI (NINDS-AIREN, 1993).

2.1.1.3. Frontotemporal dementia

Frontotemporal dementia (FTD) constitutes the third largest group of neurodegenerative diseases and occurs in an estimated 5% to 7% of the population. Both AD and FTD are primary causes of dementia (Benussi, et al., 2017; Gürvit, 2004). Although AD and FTD are clinically well defined by diagnostic criteria, they are not homogeneous disorders. FTD is a clinical picture characterized by marked changes in personality and social relationships, and cognitive impairment is mostly characterized by impairment of the executive functions of the frontal lobe (Lichter, 1999; Varma, et al., 1999).

From a neurobiological perspective, the pathogenesis of frontotemporal dementia (FTD) is associated with neuronal degeneration in the frontal and temporal lobes. This process results from the interaction between genetic factors and environmental influences. Certain subtypes of FTD are linked to specific genetic mutations, which can affect the progression of the disease (Ulugut, 2021).

The neurobiological characteristics of FTD are critically important in the diagnosis and treatment of the disease. Neuronal loss can lead to asymmetric atrophy in specific regions of the brain, contributing to the diversity of clinical symptoms (Erkol, Savrun, 2002; Gündoğdu, Kotan, 2017).

Subtypes of FTD include behavioral variant FTD, progressive arrested aphasia, semantic dementia and FTD-ALS (Galton, et al. 2000; Gürvit, 2004; Laforce, Rabinovici, 2011).

Although it constitutes 12.5-16.5% of degenerative dementias, studies on the epidemiology of FTD are limited (Kaye, et al., 2010). In postmortem studies, the prevalence rate among all dementias is 3-10% (Sjögren, Andersen, 2005)

Among the few prevalence studies, the Zuid, Netherlands study found a prevalence of 3.6 per 100,000 population aged 50-59 years, 9.4 per 60-69 years and 3.8 per 70-79 years (Rosso, et al. 2003).

Gender distribution is similar in FTD. FTD is also known as the youngest onset type of dementia. The onset is between the ages of 45-65 and the average life expectancy is 6-9 years (Arvanitakis, 2010; Sjögren, Andersen, 2005).

Personality change and impaired social behavior are the main features at the onset of FTD and throughout the course of the disease. Instrumental functions related to perception, spatial abilities, praxis and memory are intact or relatively well preserved. Based on the initial presentation, some authorities divide FTD into three clinical sub-syndromes: 1) frontal variant of frontotemporal dementia, 2) semantic dementia, 3) progressive non-fluent aphasia. In the frontal variant, the main symptoms include progressive personality change with disinhibition, loss of empathy, changes in eating habits, ritualistic or stereotypic behavior and apathy (Hodges, et al. 2004). Semantic dementia is characterized by progressive loss of knowledge of the meaning of people, objects, events and words. Progressive non-fluent aphasia is characterized by impairments in phonological and syntactic elements of language and may progress to corticobasilar degeneration (Spar, Rue, 2007).

FTD usually meets the criteria for FH and cannot be differentiated by standardized testing. However, personality and behavioral changes are prominent in the clinical picture of FTD, which is an important factor in differentiating them. FTD shows marked neuropsychological impairments and memory problems are often consistent with frontal-type amnesia. Memory deficits are more closely related to attention, recall, organization and regulatory factors than to storage. Performance is often variable and unpredictable. Spontaneous speech may be reduced but speech grammar is usually correct and paraphasia is rare. Semantic disturbances are common and comprehension is usually unaffected except for complex sentences. Reading aloud skills are usually preserved. Verbal fluency may be impaired early (Pasquier, 1999).

2.1.1.4. Parkinson's disease dementia

Although Parkinson's Disease (PD) is primarily characterized by motor symptoms, non-motor symptoms can occur even before the onset of the disease. The

increasing availability of treatment options for PD and the prolongation of life expectancy have led to an increase in the occurrence and prevalence of dementia, which has important implications for both mortality and morbidity. Even if mild impairments in cognitive function can be detected early in PD, significant cognitive impairments usually occur in the later stages of the disease and with advancing age (Hanagasi Ha, et al., 2017). Parkinson's patients have problems with organizing goal-directed behaviors, executive functions, decision-making, learning, visuospatial ability and memory (Friedman and Miyake, 2017).

Severe dementia is common in 25-30% of individuals with PD and affects various cognitive functions such as executive functions, attention, visuospatial abilities and memory. In the early stages of PD diagnosis, 10-38% of patients have mild cognitive impairment (as cited in Çelik, et al., 2023). In the early stages of diagnosis, 10%-38% of PD patients have mild cognitive impairment. Approximately 14-28% of people with Parkinson's develop dementia within five years of diagnosis (Aarsland et al., 2021). In addition to cognitive impairments, individuals with PD may also experience apathy, stress, anxiety and depression, all of which have a detrimental impact on their quality of life and functional abilities (Goldman and Sieg, 2020).

PDD is a slowly progressive dementia with an insidious onset. In the early stages, executive dysfunction, impairment in attention and visual-spatial functions, mild episodic memory impairment and word-finding difficulties are observed. Basic language functions are usually preserved. Apathy, hallucinations and delusions are common among behavioral symptoms. Increased daytime sleepiness and reversal of day-night rhythm are also common. In advanced stages, speech is impaired, the patient is unable to maintain speech, and incontinence and bed or wheelchair dependency occur. The most common neuropsychiatric symptoms include depression, hallucinations, apathy, anxiety and delusions (Aarsland, 2007; Noe, et al., 2004; Norman, et al., 2002; Petrova, et al., 2012).

PDD is marked by the degeneration of dopaminergic neurons, particularly in the nigrostriatal pathway, alongside the accumulation of α -synuclein protein in Lewy bodies. This pathological hallmark is complemented by the presence of neurofibrillary tangles and amyloid plaques, which are also associated with Alzheimer's disease (AD). Up to 50% of individuals with PDD may exhibit sufficient AD-type pathology to warrant a dual diagnosis, indicating a significant overlap between these neurodegenerative processes (Gratwicke et al. 2015; Irwin et al. 2023).

The cognitive decline in PDD is influenced by the loss of multiple neurotransmitter systems, including dopamine, noradrenaline, serotonin, and acetylcholine. The interplay between these systems is crucial, as dopaminergic deficits alone do not account for the full spectrum of cognitive impairments. Genetic factors, such as variations in the α -synuclein gene and mutations in the parkin gene, also play a role in the risk and progression of PDD, further complicating the neurobiological landscape (Halliday et al. 2014; Irwin et al., 2023).

Recent research has underscored that Parkinson's disease dementia (PDD) is not merely a consequence of motor dysfunction but involves distinct cognitive deficits arising from the disruption of multiple neural networks. For instance, the fronto-striatal networks, which are critical for executive functions and cognitive control; the mesocortical and corticopetal cholinergic networks, which play a role in attention and memory processes; and the medial temporal and noradrenergic networks, which are associated with memory and emotional regulation. The interaction and dysfunction of these networks contribute to the cognitive impairments observed in PDD, including deficits in attention, memory, and visual perception, as well as the emergence of hallucinations and cognitive fluctuations (Gratwicke et al., 2015; Halliday et al., 2014; Morelli & Pinna, 2023).

Clinical history, examination and the presence of symptoms of parkinsonism occurring at least one year before the diagnosis of dementia are key factors in the diagnosis of PDD. It is also important to exclude other possible causes. Diagnostic imaging, neuropsychological tests and laboratory results do not provide specific diagnostic information. Diagnostic criteria for PDD were published in 2007 by a working group of the Movement Disorders Society (Emre, et al. 2007).

As outlined by the Movement Disorders Society, the first step in diagnosing a patient with Parkinson's disease involves adhering to the criteria set out by the “Queen Square Brain Bank”. It is necessary to consider historical data and conduct detailed clinical and psychological examinations in order to identify the dementia syndrome, which has a gradual and insidious onset. This syndrome requires the presence of cognitive impairment in multiple domains, a noticeable decline in the individual's previous cognitive functioning, and the severity of these impairments must significantly affect the person's ability to participate in daily social, occupational or self-care activities.

When the associated clinical features are examined, cognitive features encompass various aspects such as attention, executive functions, visuospatial functions, memory and language. Attention disorder is characterized by a decrease in both spontaneous and focused attention, as well as below-average performance on attention tests. In addition, attention levels may vary throughout the day or from one day to the next. Executive dysfunction is manifested by difficulties with initiating tasks, planning, conceptualizing, identifying rules, adapting to rule changes and guarding against potential risks. Mental speed is also affected, causing a slowdown known as bradyphrenia. Visual-spatial dysfunction can be observed due to difficulties with visual-spatial orientation, visual perception and construction tasks. Memory impairment becomes evident in tests that require free recall of recent events or learning new information. However, memory recall tends to improve with cueing, and recognition abilities are generally better than free recall. Language functions are usually preserved, although there may be difficulties with word retrieval and understanding complex sentences.

Behavioral symptoms include apathy, hallucinations and delusions. Apathy is characterized by a decrease in spontaneity, motivation, interest and voluntary actions. There may be depressive features and anxiety changes in personality and mood. Visual hallucinations are common and often presented as vivid images of individuals, animals or objects. Delusions tend to be paranoid in nature; they include beliefs about deception or the existence of another individual. Excessive daytime sleepiness may also occur.

The diagnosis of PDD can be challenging when there are additional abnormalities that could potentially contribute to cognitive impairment, such as the identification of significant vascular disease through imaging. Another factor complicating diagnosis is the lack of information regarding the time of onset of motor and cognitive symptoms.

To accurately diagnose PDD, it is crucial to exclude other conditions with similar features, such as acute confusion, systemic diseases or abnormalities, drug intoxication, major depression according to DSM-IV or meeting the criteria for “Probable Vascular Dementia” according to the NINDS-ARIEN guidelines.

2.1.2. Emotional and mood changes in dementia syndromes and depression

Throughout the course of dementia, patients may exhibit not only cognitive impairments but also significant changes in emotional, thought, and personality

dimensions. Dementia is characterized by alterations in emotional state, thought processes, and personality traits, which are collectively referred to as "Behavioral and Psychological Symptoms of Dementia" (BPSD) by the International Psychogeriatric Association (Finkel et al., 1996). These symptoms are widespread and represent a significant clinical target for intervention (Katona et al., 2007). Despite their prevalence, BPSD are not typically included in the diagnostic criteria for dementia.

As dementia progresses, emotional and mood changes, including depression, frequently emerge. Depression is a common symptom in dementia and can complicate both the diagnosis and treatment of the disease. These mood changes can adversely affect the patient's quality of life and potentially accelerate the progression of dementia. Depression in individuals with dementia often manifests through symptoms such as sadness, loss of interest, energy depletion, feelings of guilt, and hopelessness. Additionally, anxiety, irritability, and agitation are frequently observed emotional responses that are considered part of the mood alterations in dementia (Finkel et al., 1996).

The relationship between depression and dementia is complex; depression may sometimes precede the onset of dementia, while in other cases, it may develop as a consequence of the disease. This intricate relationship necessitates accurate recognition and management to ensure appropriate treatment. Untreated depression can exacerbate cognitive symptoms of dementia and negatively impact the patient's overall health. Therefore, early diagnosis and treatment of depression in dementia patients can positively influence the disease's trajectory and improve the patients' quality of life (Prado-Jean et al., 2010; Robert et al., 2009).

Apathy is another condition frequently observed in dementia patients, leading to both cognitive and emotional impairments. In apathy, there is a marked reduction in emotional responses and indifference, whereas depression is characterized by intense sadness, hopelessness, and low self-esteem. Moreover, apathy is associated with a lack of motivation and interest, while depression encompasses emotional and physical fatigue along with these symptoms.

2.1.2.1 Definition and epidemiology of depression

Depression is the most prevalent mood disorder among psychological disorders. It affects up to 43% of patients with dementia and is associated with an increasing number of neuropsychiatric symptoms, including agitation, anxiety, and irritability (Prado-Jean et al., 2010). Epidemiological data indicate that approximately 13% of women and 8% of men experience depression in any given year. In Turkey, the prevalence of depressive episodes is reported to be twice as high in women compared to men, with rates of 2.3% in men, 5.4% in women, and an overall prevalence of 4% (Psychopharmacology Scientific Study Unit).

Depression is characterized by a prolonged decrease in self-esteem, inability to derive pleasure from activities and life, hopelessness, and lack of motivation, which can persist for days, months, or even years. Risk factors for developing depression include medical illnesses, low socioeconomic status, divorce, unemployment, a history of loss, childhood trauma, and living alone (Kafes, 2021).

According to DSM-5, several disorders fall under the category of depressive disorders, including disruptive mood dysregulation disorder, major depressive disorder, persistent depressive disorder, premenstrual dysphoric disorder, substance/medication-induced depressive disorder, and depressive disorder due to another medical condition. Commonly referred to as depression in everyday language is Major Depressive Disorder (MDD).

For a diagnosis of Major Depressive Disorder according to DSM-5, an individual must exhibit at least five of the following symptoms over a period of at least two weeks: a depressed mood (nearly every day, most of the day), or a noticeable decrease in interest or pleasure in activities once enjoyed. Other symptoms include significant weight loss or gain (without dieting), disturbances in sleep patterns (insomnia or excessive sleeping), psychomotor agitation or retardation (restlessness or slowed movements), persistent fatigue or loss of energy, feelings of worthlessness or excessive guilt, and difficulties with thinking, concentration, or decision-making. Additionally, the presence of thoughts of death or suicide, suicide attempts, or plans is also considered. At least one of these symptoms must be either depressed mood or loss of interest, and these symptoms must lead to significant impairment in social, occupational, or other important areas of functioning.

2.1.2.2. Neurobiology of depression

The neurobiology of depression is primarily associated with neurotransmitter imbalances, changes in brain structures, and disruptions in hormone levels. Reductions in serotonin, norepinephrine, and dopamine levels are observed in connection with depressive symptoms, while decreases in hippocampal volume and dysfunctions in the prefrontal cortex are considered neurobiological markers of depression. Additionally, increased cortisol levels support the relationship between stress and depression. Genetic and epigenetic factors also play a significant role; genetic predisposition to depression and environmental influences can affect risk factors by altering gene expression.

The limbic system plays a crucial role in the pathophysiology of depression, with research focusing particularly on the hippocampus, a key component of the limbic system. Stress-induced hippocampal apoptosis and decreased neurogenesis, along with the reversal of these adverse changes with antidepressant treatment, suggest that neuroplasticity or neuroplastic changes might contribute to the development of depression (Slattery et al., 2004 as cited Uzbay, 2004).

Neuroplasticity refers to the structural and functional changes in neurons and synapses in the brain in response to environmental factors. These changes can lead to the development of various diseases, such as depression, under stress, but they also play a critical role in the development of essential central functions and the reversal of disorders (Uzbay, 2004).

Vyas and colleagues (2002) demonstrated that chronic stress causes dendritic damage not only in the hippocampus but also in the amygdala. This data suggests that the amygdala might also be a neuroanatomical region contributing to the development of depression.

One of the most valid hypotheses related to depression is the monoamine hypothesis, proposed in the mid-1960s. This hypothesis suggests that depression is associated with deficiencies in neurotransmitters. While the monoamine hypothesis aims to explain the role of neurotransmitter deficiencies in the biological etiology of depression, the mismatch between the increase in neurotransmitter levels and the timing of clinical response to antidepressant treatments has necessitated the development of new hypotheses (Stahl, 2000).

Research has shown that the role of neurotransmitters in depression etiology cannot be fully explained by the monoamine hypothesis alone; molecular events regulating receptor activity and gene expression are also crucial. According to the receptor hypothesis, deficiencies in neurotransmitters or inherited receptor abnormalities can lead to upregulation of postsynaptic receptors, which may contribute to the development of depression. Pathology in receptors can stem from monoamine neurotransmitter defects, receptor abnormalities, or issues related to signal transduction (Stahl, 2000).

The majority of noradrenergic neurons in the brain are located in the locus coeruleus. Activation of this system leads to attention, fear, and panic reactions. Norepinephrine (NE) deficiency plays a role in the development of depression because norepinephrine is vital for cognitive functions, mood, and behavior. NE deficiency is associated with reduced interest, concentration issues, and depressive symptoms. Furthermore, reduced sensitivity of beta-adrenergic receptors is an important factor in depression treatment (Kaplan & Sadock, 2003).

Serotonin pathways are involved in sleep regulation, behavior patterns, appetite, and hormonal regulation. Serotonin deficiency is considered a cause of depression. Serotonin receptors have different roles: 5-HT_{1A} provides anxiolytic effects, while 5-HT_{2A} is responsible for movement control and obsessions. Decreased serotonin reuptake and reduced serotonin transporter levels are linked to depressive symptoms (Schatzberg & Nemeroff, 1998; Yüksel, 2003).

Dopaminergic pathways are part of the extrapyramidal system and are involved in movement and motivation. The mesolimbic pathway is associated with pleasure and psychosis, while the mesocortical pathway is responsible for cognitive symptoms in schizophrenia. Decreased dopamine activity can lead to depressive symptoms, suggesting that dopamine-enhancing drugs may alleviate depressive symptoms (Schatzberg & Nemeroff, 1998; Stahl, 2000).

While the monoamine hypothesis posits that depression results from neurotransmitter deficiencies, this hypothesis has been expanded by the gene expression theory in recent years. According to this theory, instead of neurotransmitter loss, there is a molecular-level disorder. Specifically, decreased expression of neurotrophic factors such as Brain-Derived Neurotrophic Factor (BDNF) under stress leads to neuronal

atrophy and apoptosis, which can result in depression and treatment resistance. Antidepressants are thought to reverse these effects (Stahl, 2000).

Neuroactive peptides such as GABA, vasopressin, and endogenous opioids are also thought to play roles in the pathophysiology of depression. Neurotoxic effects of glutamate and NMDA receptors are associated with chronic stress. Furthermore, hormones and peptide hormones are linked with depression, and endocrine disorders are frequently associated with psychiatric symptoms. Hormones can function similarly to neurotransmitters or affect neuronal functions through hormonal release (Ceylan & Oral, 2001).

Neuroendocrine abnormalities associated with depression include alterations in the adrenal, thyroid, growth hormone, and prolactin axes. Hyperactivity or hypoactivity in these axes can be part of depression, though their role in etiology remains debated (Eşel & Sofuoğlu, 2001).

The role of neuroimmunological events in the etiology of depression is under investigation. Major depressive disorder has been associated with increased serum concentrations of proinflammatory cytokines such as IL-1 and IL-6, as well as acute-phase reactants like C-reactive protein and haptoglobin. Stress and CRH have been shown to induce proinflammatory cytokines, but the precise source of immune activation in major depressive disorder remains unclear. High rates of major depression can be observed during interferon-alpha treatment and may respond to antidepressant therapy. Depressive patients also show high levels of hypercortisolism, infection, allergy, and cancer. However, significant changes in total lymphocyte count and B cells are not evident, though abnormalities in T4/T8 ratios are observed. Depressive patients show reduced lymphocyte response to mitogens and increased plasma prostaglandin E levels. These findings suggest a possible relationship between depression severity and immunity (Ceylan & Oral, 2001; Doksat & Savrun, 2002).

Depression has also been examined in relation to nitric oxide levels, selenium, zinc, and folic acid. Zinc and selenium deficiencies have been found to be related to depression in major depressive disorder patients, with selenium supplementation showing positive effects on mood (Suzuki et al., 2001). Additionally, individuals with depression often have low folic acid and vitamin B12 levels, with low folate levels being associated with treatment resistance (Fava et al., 1997). The relationship between the adipocyte

hormone leptin and depression is also being investigated, though findings remain inconclusive. Some studies suggest that low leptin levels may be associated with depression and that increasing leptin levels might alleviate depressive symptoms. However, the exact mechanisms of these relationships are still unclear and require further research (Auwerx & Steals, 1998).

Geriatric depression is characterized by age-related brain changes, cognitive impairments, and social factors. Brain atrophy and white matter lesions in elderly individuals can increase the risk of depression, while changes in serotonin and norepinephrine levels also play a significant role. Additionally, elderly individuals may experience difficulties in cortisol regulation, and social isolation and physical health issues can impact geriatric depression. Understanding these factors is critical for elucidating the neurobiological foundations of depression in older adults.

2.1.2.3. Geriatric depression and pseudodementia

In the aging population, depression can manifest with distinct symptoms compared to the general adult population. Geriatric depression, a subtype of depression occurring in older individuals, primarily affects those aged 65 and above, though it can also impact others with age-related health issues. This condition is often associated with health problems, loneliness, social isolation, loss, and grief resulting from life changes. It is crucial to carefully consider specific aspects of the assessment process when evaluating elderly patients to avoid overlooking depression. During the examination, a detailed history should be obtained not only from the patient but also from family members or others familiar with the patient. Evaluating the patient's recent functionality is especially important. Additionally, a thorough inquiry into previous psychiatric diagnoses, family history of mental illness, personality, chronic physical problems, the development of symptoms, and psychiatric and non-psychiatric treatments is essential (Ceylan & Göka, 1998).

Geriatric depression often presents with somatic symptoms in addition to typical depressive symptoms. These may include loss of appetite, sleep disturbances, fatigue, lack of energy, and chronic pain. Cognitive dysfunctions such as memory issues and concentration difficulties can also manifest as signs of depression in older adults. Geriatric depression can severely affect the quality of life and negatively impact physical

health. Furthermore, untreated depression in the elderly can complicate the management of other health issues and shorten life expectancy.

Diagnosis of geriatric depression typically requires a comprehensive evaluation and is usually conducted by a psychiatric or geriatric specialist. Treatment often involves a combination of psychotherapy, pharmacotherapy, and social support. Effective management of geriatric depression can enhance quality of life and improve overall health.

Depression not only affects mental health but can also exacerbate pre-existing medical conditions, leading to higher morbidity and mortality rates. Individuals with depression are at risk of cognitive decline and even dementia. Thus, elderly adults who develop depression are at increased risk of developing dementia in the near future. A history of lifelong depression also elevates the risk of developing Alzheimer's disease. Numerous studies have highlighted the association between stress, depression, and chronic diseases (Alexopoulos, 2005; Almeida, 2014).

Prevalence studies of geriatric depression show varying results across different elderly populations, ranging from 1% to 60% depending on the group structure, study objectives, and methodologies (Alexopoulos, 2000; Blazer, 1994; Wu & Anthony, 2000). Depression prevalence among the elderly typically ranges from 15% to 20% (Türkseven et al., 2020), with depressive symptoms observed in 10% to 15% of older adults (Baldwin, 2008). Both prevalence and incidence of depression increase with age, doubling after 70-85 years and affecting 1-4% of the elderly population, with women being twice as likely to be affected (Alexopoulos, 2005).

Elderly individuals with dementia are found to develop depression at higher rates compared to the general population, with the prevalence of depression in dementia patients around 50%. The clinical course of depression in the elderly can differ from that in younger adults. In some older individuals, depression may be more pronounced, while in others, symptoms may be milder or accompany other health issues. Depression in the elderly is often associated with somatic symptoms, with psychiatric symptoms being less pronounced (Jenike, 1995). Additionally, depression in older adults frequently coexists with underlying medical conditions or medication side effects, complicating accurate diagnosis.

The clinical course of depression in the elderly often tends toward chronicity. If untreated, symptoms can worsen over time, significantly impacting quality of life and potentially shortening life expectancy. Thus, early recognition and effective management of depression in older adults are crucial.

There are no specific diagnostic criteria for depression in the elderly; however, clinical evaluation can lead to a diagnosis (Yuvakgil et al., 2023). Diagnosing depression in older adults can be more complex than in earlier stages of life due to various factors (NIH, 1992). Diagnostic criteria, generally outlined in DSM-5 or ICD-10, include depressive mood or loss of interest lasting at least two weeks, affecting daily activities; changes in sleep, appetite, energy, concentration, or self-care; impairment in quality of life or social functioning; and exclusion of symptoms caused by dementia or other medical conditions. The diagnostic process may vary based on the patient's symptoms, age, medical history, and other factors, necessitating consultation with a specialist.

It is also important to note that depression in elderly individuals can sometimes be mistaken for pseudodementia. Pseudodementia refers to cognitive decline mimicking dementia but resulting from depression. Accurate diagnosis and appropriate treatment are crucial in such cases. Pseudodementia is characterized by cognitive impairments similar to dementia but with reversible cognitive function improvements following depression treatment (Jenike, 1995). Studies assessing cognitive function in depressed elderly patients have revealed declines in various cognitive domains, leading to the use of the term pseudodementia (Spar, 2007; Wells, 1979). When cognitive impairments such as memory deficits are present, clinicians should consider pseudodementia or depression in dementia patients (Tamam & Öner, 2001).

Pseudodementia in elderly patients often occurs as part of severe depressive disorders, accompanied by retardation, delusions consistent with depressive mood, hopelessness, helplessness, and anxiety. Cognitive impairment in pseudodementia is typically milder compared to true dementia. Sometimes, the reversibility of cognitive decline is only apparent after the treatment of depressive episodes, making differentiation from dementia challenging. Pseudodementia patients often exhibit impairments in attention, memory, conceptualization, and mental processing speed. Recent research has shown that patients with pseudodementia, despite full recovery from depression, have a high likelihood of developing dementia. Specifically, patients who continue to have some cognitive impairments after depression treatment may be in the early stages of dementia.

Studies indicate that elderly patients with pseudodementia often develop irreversible dementia at a high rate during follow-up, with an average annual incidence rate of 20%, higher than in the general elderly population. Therefore, pseudodementia observed in depressed elderly patients is considered a potential indicator of irreversible dementia (Alexopoulos, 2000; Forsell et al., 1994). Hence, careful evaluation and monitoring of these patients are emphasized to differentiate underlying and treatable neurological disorders during follow-up.

Dementia patients are found to develop depression at higher rates compared to the general population, with the prevalence of depression among dementia patients approximately 50%. The similarities between depressive symptoms and those of dementia-related diseases, particularly pseudodementia, pose significant challenges for differential diagnosis (Bekaroğlu, 1995). Clinical features that distinguish depressive pseudodementia from progressive dementia are important (Crowe & Hoogenraad, 2000). A history of depression can be a risk factor for depressive pseudodementia. Recent reports indicate that many patients with pronounced pseudodementia later experience their first depressive episodes (Alexopoulos, Young, & Meyers, 1993), with decreased recovery rates and persistent symptoms of true dementia (Kral & Emery, 1989; Reding, Haycor, & Blass, 1985). Increased research on depression in later life has suggested that pseudodementia might reflect the development of actual dementia. Current understanding indicates that a significant portion of late-onset depression may be associated with or facilitate the development of dementia. The frequent co-occurrence of dementia and depression underscores the importance of thorough differential diagnosis for both conditions, as early diagnosis and treatment are critical. Early detection of dementia allows for the use of cognitive function-enhancing medications such as cholinesterase inhibitors and memantine, which can improve quality of life and provide opportunities for future planning (Weiner & Lipton, 2009).

2.1.2.4. Comorbidity of dementia and depression

A significant proportion of patients with dementia suffer from depression. Depression can worsen the course of dementia and reduce the overall quality of life of patients. Studies show that 20-50% of dementia patients have symptoms of depression (Cankurtaran, Arıoğlu; Okudur et al. 2018).

Studies indicate that a substantial number of patients with late-life depression have comorbid dementia. For instance, one study found that approximately 21% of patients hospitalized due to major depressive disorder (MDD) had dementia, with a meta-analysis reporting a prevalence of 15.9% to 24% in various studies (Brauch et al.2019; Ochi et al.2022).

The presence of depression in individuals with dementia can lead to more severe cognitive impairment and complicate the clinical picture, making it challenging to differentiate between symptoms of depression and dementia itself (Brauch et al.2019; Ochi et al.2022).

Treatment of comorbid depression plays a critical role in the management of dementia. Methods such as antidepressant treatment and psychotherapy can alleviate symptoms of depression and improve the general condition of patients with dementia (Okudur et al. 2018).

While antidepressants are commonly used to treat depression, randomized controlled trials have shown limited effectiveness in improving depressive symptoms in patients with comorbid dementia. Additionally, these medications may increase the risk of side effects, necessitating careful consideration before use (Brauch et al.2019).

Psychological therapies, such as cognitive-behavioral therapy (CBT), have shown promise in managing depression in dementia patients. Structured interventions, including sleep hygiene programs, exercise, arts therapies, and music therapy, may also provide benefits, although evidence is still emerging (Brauch et al.2019).

Integrated care approaches that involve collaboration among healthcare providers, caregivers, and mental health professionals are crucial for effectively managing the complexities of patients with both dementia and depression (Kverno, Velez, 2018).

Monitoring cognitive functions in patients with comorbidity of dementia and depression requires a multidisciplinary approach. A combination of methods such as neuropsychological tests, clinical assessments, laboratory tests and neuroimaging are effective for monitoring the cognitive status of these patients and optimising treatment processes. In this process, a customised assessment plan should be created according to the individual needs of the patients.

3. METHOD

In this section, the research model, population and sample, data collection tools, data collection process, analysis of the data obtained are emphasized.

3.1. Research Model

The study was designed as a cross-sectional correlational study. It will find a place in the literature as an analysis of a study in which the whole population is examined in the same pool in a naturalistic design and the correlation of depression severity, dementia severity (CDR 1) and neuropsychological differences are analyzed, rather than examining patients with depression and dementia and patients with dementia without depression separately. Depression was diagnosed according to DSM-V diagnostic criteria.

3.2. Population and Sample

The population of the study was defined as patients diagnosed with dementia and their relatives living in Izmir province.

The research sample consists of 40 people diagnosed with dementia (CDR 1), consisting of 20 patients with comorbid dementia and depression and 20 patients with dementia only. Participants will be selected on a voluntary basis. Informed Consent Form was used for the patients and their relatives and consent was obtained from the participants.

The methodological approach we employed for categorizing the patients included in our research group was meticulously designed. Participants were divided into two main groups based on clinical evaluations conducted by neurology and geriatrics specialists: those diagnosed with major depressive disorder (MDD) and comorbid dementia, and those diagnosed with dementia only. The diagnosis of MDD was made according to the DSM-V criteria, while the diagnosis of dementia was determined using the Clinical Dementia Rating (CDR) scale. All dementia patients in our study were assessed as CDR 1.

Throughout the study, the specialists were unaware of which participants would undergo which tests; therefore, a blind approach was adopted during data collection and test administration. This method ensured that the evaluations made by the specialists were impartial and unbiased.

The entire data collection and evaluation processes were conducted by the author of this thesis, N. E. E., to maintain the objectivity and consistency of the study. The same tests were administered to both groups, and the selection and administration of these tests were carried out with great care. This ensured the consistency and validity of comparisons between the groups.

In the analysis of the obtained data, the demographic and clinical characteristics of both groups were examined in detail. These examinations are crucial for the validity and reliability of the research findings.

3.3. Data Collection Tools

Scales were applied to the individuals with dementia and their relatives who participated in the study. Firstly, the “Sociodemographic Data Form” was asked to the individual diagnosed with dementia and the missing and incorrect information was confirmed by the relatives. Then, the relatives were removed from the room and “Standardized Mini Mental State Examination”, “Digit Span Test”, “Boston Naming Test”, “Öktem Verbal Memory Process Test” and “Stroop Test” were applied to the individuals diagnosed with dementia. Then, the “Geriatric Depression Scale” was completed with the patient and immediately afterwards, the “Cornell Scale for Depression” was completed by interviewing only the patient's relatives. The data obtained from the patient's relatives and individuals diagnosed with dementia were combined and analyzed on the relationship between dementia and depression and statistical information was obtained. During the application, the participants were informed about the purpose of the study and the principle of confidentiality and approval was obtained by signing the Informed Voluntary Consent Form.

3.3.1. Sociodemographic data form

The purpose of use is to contribute to the surveys applied and to obtain detailed information about individuals. Questions were created to reinforce the questionnaires and certain forms and to complete the desired data. Information such as age, gender, education, marital status, hand dominance, etc. can be accessed in this way.

3.3.2. Standardized mini mental state examination (SMMSE)

The original Mini Mental Test first published by Folstein et al. (1975) and the standardized version and administration instructions created by Molloy and Standish (1997) were translated into Turkish by Güngen et al.

MMT is used in clinical practice for the diagnosis of mild dementia. It assesses cognitive functions globally. It consists of orientation, recording memory, recall, language, attention and calculation subscales. MMT consists of 11 items and these questions are categorized under 5 main headings: 11 items are evaluated out of 30 points in total. The administration time of the test varies between 5-10 minutes. However, the test is not timed.

In the evaluation out of 30 points, 24 or above indicates a normal cognition state. Below (19-23) is considered mild, (10-18) is considered moderate.

3.3.3. Digit span test

The number sequence test is a subtest of both the Wechsler Adult Intelligence Scale (WAIS) and the Wechsler Memory Scales (WMS). In this test, subjects are read a series of numbers and asked to tell the experimenter either in the order in which they were told from the beginning (forward) or in the reverse order from the end to the beginning (backward). In the test, the forward range of numbers indicates the efficiency and capacity of attention and concentration. The backward digit span is an executive task that depends specifically on working memory. The Digit Span subtest can be scored as a single summary value (this is the age-specific score that contributes to the summary scores on Wechsler tests) or can be scored separately for forward and backward performance. Forward and backward performance can be reported as sub-scores (the number of correct items of each type) or interval scores (the maximum number of digits correctly produced by subjects, either forward or backward).

3.3.4. Boston naming test (BNT)

It was originally developed by Kaplan, Goodglass and Weintraub (1983). It is the best known neuropsychological tool for assessing language skills including object naming and word retrieval.

BNT is used to assess children, adults and elderly individuals with different clinical pathologies such as communication disorders, aphasia, dementias or brain lesions.

The longest version, consisting of 60 items, is the BNT-60. The administration time varies between approximately 10-20 minutes. The 60 different stimulus cards are presented in a spiral booklet with black and white drawings of increasing difficulty. Each

item is given 20 seconds to name. If the item cannot be named, a semantic or phonemic clue is given. The minimum score is 0 and the maximum score is 60.

The reliability of the test in terms of Cronbach's alpha coefficient was 0.90 for the Spontaneous Naming score and 0.82 for the **KA+SNA** score.

3.3.5. Öktem verbal memory process test (VMPT)

The test, which assesses the ability to learn verbal material and to recall and recognize learned material from memory storage, is widely used to evaluate neurological and psychiatric patients. It is an important verbal memory test used in the differential diagnosis of dementias. In the Criterion-Related Validity Study, the correlative relationships between the Öktem-VMPT and Wechsler Memory Scale (WMS) subtests were determined by Pearson correlation analysis.

In the reliability analyses of the Öktem-VMPT, the correlative relationships between Öktem-VMPT A and B and lists A and C were examined by Pearson correlation analysis using the Equivalent Form Reliability method. According to the Pearson correlation analysis for the equivalence between lists A and B, statistically significant correlations were found at the level of $r=0.81$, $p<.01$ in the Total Learning dimension; $r=0.70$, $p<.01$ in the Highest Learning dimension; $r=0.33$, $p<.01$ in the USB Spontaneous Recall dimension; and $r=0.88$, $p<.01$ in the USB Total Recall dimension. In the validity analyses of Öktem-VMPT, list A was used since the equivalence of lists A-B and A-C was shown. The correlative relationships between Öktem-VMPT List A and WMS dimensions were determined by Pearson correlation analysis. According to the findings, statistically significant positive correlative relationships were found at $p<0.05$ and $p<0.01$ levels between the Öktem-VMPT A list Immediate Memory, Total Learning, Highest Learning, USB Spontaneous Recall and USB Total Recall dimensions and all of the WMS dimensions (Tanör, 2011).

3.3.6. Stroop test

The Stroop Test is a neuropsychological test that reflects frontal region activity. (Karakas, et al., 1999) This test measures the ability to resist interference and the ability to suppress the habitual response and give the desired response.

Karakaş and his colleagues (Karakaş and Başar, 1993, Karakaş et al., 1996) were involved in the introduction of the Stroop Test into the culture of our country. These

studies on the Stroop Test were organized within the scope of the standardization of a neuropsychological test battery. The battery in question, which was created to address the relationship between the scores obtained from neuropsychological tests and cognitive electrophysiology parameters, is the “Neuropsychological Test for Cognitive Potentials (NTCP) Battery”. Since the standardization project was supported by the Basic Sciences Research Group of TÜBİTAK, the Turkish form of the Stroop Test was named the Stroop Test TBAG Form. The adaptation of the Stroop Test TBAG Form within the scope of the NTCP Battery has been completed.

3.3.7. Geriatric depression scale (GDS)

It is a self-report scale developed by Yesavage et al. (1983) consisting of 30 self-report questions, each of which can be answered “yes” or “no”. Each response in favor of depression is evaluated as one point.

Turkish validity and reliability study were conducted by Ertan (1996) (test-retest consistency, $r=0.77$; internal consistency $\alpha=0.92$).

3.3.8. Cornell scale for depression in dementia (CSDD)

It is a scale developed by Alexopoulos et al. (1988) to specifically assess signs and symptoms of depression in patients with dementia. It is administered separately by first interviewing the caregiver and then the patient face to face and then scoring the clinician's opinion on the scale. It consists of 19 items collected in 5 subgroups. The 5 subgroups include items assessing mood-related symptoms, behavioral disorder, physical symptoms, cyclical functions and thought disorder.

3.4. Data Collection

Participants were informed about the purpose of the study before the application and approval was obtained by signing the “Informed Voluntary Consent Form”. Data collection was carried out between January and May 2024 and the number of participants included in the evaluation was 40 patients with dementia and their relatives. Tasks that could not be performed in the scales were not included in the evaluation.

At the beginning of the application, the patient and his/her relatives were taken into the room. While filling out the sociodemography form, information about the patient's age, gender, marital status, marital status, educational status, hand dominance, history of chronic diseases, history of surgery, history of psychiatry, neurological

diagnoses, alcohol-smoking history, and social life were obtained. The patient's relative was then removed from the room. The standardized mini mental state examination (SMMSE) was started with the instruction “I would like to ask you some questions and show you some problems to solve, please try to give the best answer you can” as given in the application instructions. Questions measuring place and time orientation were asked and the order was followed. The recording memory section was started and the patients were asked to repeat three words. In the attention and calculation section, patients were asked to count backwards from 'to'. When it came to the recall section, the patients were expected to recall the three words we said, regardless of the order. In the language section, they were first shown a pen and a watch and were expected to know what they were. They were given 10 seconds to answer. Immediately afterwards, the written sentence in the mini mental test was read and the patient was expected to repeat it completely and accurately within 10 seconds. Then, the patient was asked to apply the 3-digit operation in the mini mental test. The patient was asked to read and apply the text written on the paper. Finally, the patient was given application paper, pencil and eraser and asked to copy two intertwined pentagons whose shapes were shown. 1-minute time was given. Sociodemographic form and Mini mental test took approximately 15 minutes.

As soon as the Standardized Mini Mental State Examination was finished, the number sequence test was started immediately. It starts with the advanced digit span. Patients are informed that they will be told the number sequences and that they should repeat the number sequences when they finish counting. The digit they can advance is noted (maximum 8). When the forward number sequence is finished, the backward number sequence is started. Patients are told that they will read the number sequences again, but this time they have to say the numbers from the end to the beginning. An example is given with a number sequence that is not on the list (3 digits). Patients who had difficulty in receiving the instruction were given an example with a 2-digit number and continued by experimenting with a 2-digit number. The number of digits they can advance is noted (maximum 7). While the forward number sequence measures simple attention and concentration skills, the backward number sequence measures complex attention and concentration skills. This test takes approximately 5 minutes.

After the Digit Span test was completed, the Öktem Verbal Memory list was started. As it is written in the Öktem-VMPT Handbook, if the patient's forward digit span is below 4 in the Digit Span test, it is recommended to work with 10 words, as this patient

cannot be expected to cope with 15 words, and among patients with a forward digit span above 4, there may be those who cannot work with 15 words, it is necessary to reduce to 10 words. Digit Span started with 15 words with patients whose Digit Span Advanced Number Range exceeded 4. When the sample was examined, it was seen that these patients could not cope with 15 words and were suitable for working with 10 words, and 10 words were used to keep the data constant (List A). It was informed that ten repetitions would be done with ten words. They were informed that the aim was to reach the highest number of words, so they should repeat the words they said before in each round. Fifteen words were said for ten rounds. Each round they were given time to count what they remembered. The words they remembered incorrectly were noted on the side of the page. The recall and recognition aspects were done by returning to the test afterwards, so the next practice was started before the recall and recognition questions were asked. This practice, which did not include recall and recognition, took approximately 10 minutes.

After the completion of ten rounds of Öktem Verbal Memory process, the Boston Naming Test was administered. When the long form of the test (60 items) was used, the test was started at Item 30 for non-aphasic adults in accordance with the administration instructions. The test was continued forward unless the participant failed before Item 38. If the participant failed, the test was returned to Item 29 and worked backwards until 8 consecutive items were correctly named without semantic or phonemic cueing. Items before this new starting point were considered to have been named without cueing. This test took approximately 15 minutes.

When the Boston Naming Test was finished, the Geriatric Depression Scale was administered. Consisting of 30 questions, this scale was read to the patients one by one and they were asked to answer yes or no. This test took approximately 5 minutes.

The Öktem Verbal Memory test was returned for the recall and recognition stages, which are the last part of the Öktem Verbal Memory test. For the recall phase, the patients were asked to count ten words and whether they could remember any of these words. Those they could count were filled in the form and those they remembered incorrectly were noted on the side of the paper. For the recognition phase, they were told that there was a list and the words on the list were read. Similar to the previous stage, the words he/she could count were filled in the form and the words he/she recognized incorrectly were noted. This phase lasted approximately 5 minutes.

After the last part of the Öktem Verbal Memory Test was completed, the Stroop Test was started. Before the participant was given the first card, which was the color names written in black, he was told that he should read it quickly and accurately by following the order like reading a book. After the first card was finished, the participant was given the card with the color names written in color and was again told to read it quickly and accurately by following the order like reading a book. After the application, another card with colored shapes was given. The participants were asked to say the names of the colors in order, quickly and accurately. After this card was finished, a card with words without color names written in color was given. The participants were asked to say the ink color in which the words were written. Finally, the participants were again given a card with color names written in color. As in the previous card, they were asked to say the ink color in which the words were written. At each stage, the participant's performance was monitored by timing and recording their errors and corrections. For each card, the elapsed time and the number of errors and corrections were noted. This application took approximately 5 minutes.

Immediately after the tests applied to the patient were finished, the patient was taken to the waiting room and the interview with the patient's relative was started. Through the interviews with the patient's relatives, the Cornell Scale for Depression in Dementia was completed. This scale takes approximately 5 minutes to complete.

The administration of all tests took approximately 65 minutes.

3.5. Statistical Analysis of the Data

Statistical Package for Social Sciences (SPSS) 26.0 software was used for statistical analysis of the research data.

Descriptive statistics such as mean, standard deviation and median of the Standardized Mini Mental State Examination, Digit Span Test, Öktem Verbal Memory Process Test, Geriatric Depression Scale, Cornell Scale for Depression in Dementia, Stroop Test and Boston Naming Test scores of the participants with and without additional diagnosis of depression were given.

Standardized Mini Mental State Examination, Digit Span Test, Öktem Verbal Memory Process Test, Geriatric Depression Scale, Cornell Scale for Depression in Dementia, Stroop Test and Boston Naming Test scores of dementia patients with and without additional diagnosis of depression included in the study were examined by

Kolmogorov-Smirnov test and Shapiro-Wilk test and it was determined that they did not show normal distribution. In this context, nonparametric tests were used in the study. Mann-Whitney U test was used to compare the Standardized Mini Mental State Examination, Digit Span Test, Öktem Verbal Memory Process Test, Geriatric Depression Scale, Cornell Scale for Depression in Dementia, Stroop Test and Boston Naming Test scores of dementia patients with and without additional diagnosis of depression.



4. FINDINGS

4.1. Descriptive Analysis

Table 1: Comparison of socio-demographic characteristics of dementia patients with and without comorbid depression

	Dementia		Dementia+ Depression		Total		X ²	P
	N	%	N	%	N	%		
Gender								
Female	11	55	7	35	18	45	1,616	0,204
Male	9	45	13	65	22	55		
Age	72,95±8,83		70,70±8,48		71,82±8,62		0,787	0,431
Educational Background								
Primary School	12	60	14	70	26	65	1,354	0,716
Secondary School	4	20	2	10	6	15		
High School	2	10	1	5	3	7,5		
Undergraduate	2	10	3	15	5	12,5		
Marital Status								
Married	15	75	15	75	30	75	0,000	1,000
Widowed-Divorced	5	25	5	25	10	25		
Dominant Hand						0	2,105	0,147
Right	20	100	18	90	38	95		
Left	0	0	2	10	2	5		

Table 1. shows the results of the chi-square test for the comparison of the socio-demographic characteristics of dementia patients with and without additional diagnosis of depression.

According to Table 1, 55% of dementia patients without additional diagnosis of depression were female, 45% were male, the mean age was 72.95±8.83, 60.0% were primary school graduates, 40.0% were secondary school graduates, 75% were married and 100.0% were right-handed dominant.

It was determined that 35% of dementia patients with depression comorbidity were female, 65% were male, the mean age was 70.70 ± 8.48 , 70.0% were primary school graduates, 10.0% were secondary school graduates, 75% were married and 90.0% were right-handed dominant.

It was determined that there was no statistically significant difference between dementia patients with and without additional diagnosis of depression according to their socio-demographic characteristics ($p > 0.05$).

4.2. Examination of the Relationship Between Additional Diagnosis of Depression in Dementia and Scales

Table 2: Comparison of standardized mini mental state examination scores of dementia patients with and without comorbid depression

	DIAGNOSIS	n	$\bar{x}$	S	M	SO	ST	Z	P
Std. Mini Mental State Test	Dementia	2	17,5	3,3	18,5	25,6	512,0	-	0,006
		0	0	3	0	0	0		
	Dementia+ Depression	2	13,2	5,0	12,0	15,4	308,0		
		0	0	5	0	0	0		

* $p < 0,05$

Table 2 shows the results of the Mann-Whitney U test for the comparison of the Standardized Mini Mental Test scores of dementia patients with and without additional diagnosis of depression included in the study.

It was determined that there were statistically significant differences between the Standardized Mini Mental Test scores of the dementia patients included in the study according to the additional diagnosis of depression ($p < 0.05$). It was determined that the Standardized Mini Mental Test scores of dementia patients with additional diagnosis of depression were higher.

Table 3: Comparison of number sequence test scores of dementia patients with and without comorbid depression

	DIAGNOSIS	n	$\bar{x}$	S	M	SO	ST	Z	P
Digit	Dementia	2	4,8	0,9	5,0	28,15	563,00		
Span		0	5	3	0				0,000
Test	Dementia+	2	3,2	1,0	3,0	12,85	257,00	-4,284	*
(Forward)	Depression	0	0	6	0				
Digit	Dementia	2	2,4	1,0	3,0	24,98	499,50		
Span		0	5	0	0			-2,571	0,010
Test	Dementia+	2	1,4	1,2	2,0	16,03	320,50		*
(Backward)	Depression	0	5	8	0				

* $p < 0,05$

Table 3. shows the results of the Mann-Whitney U test for the comparison of the Digit Span Test scores of dementia patients with and without additional diagnosis of depression who were included in the study.

According to Table 3, it was determined that there was a statistically significant difference between the Number Sequence Test (forward) scores of dementia patients with and without additional diagnosis of depression ($p < 0.05$). It was determined that the Digit Span Test (forward) scores of dementia patients with additional diagnosis of depression were lower.

It was determined that there was a statistically significant difference between the Digit Span Test (backward) scores of dementia patients with and without additional diagnosis of depression included in the study ($p < 0.05$). The Digit Span Test (backward) scores of dementia patients with additional diagnosis of depression included in the study were lower.

Table 4: Comparison of ökten verbal memory process test scores of dementia patients with and without comorbid depression

	DIAGNOSIS	n	$\bar{x}$	s	M	SO	ST	Z	P
Immediate Memory	Dementia	4	2,07	1,14	2,00	13,4	138,0	-	0,72
	Dementia+Depression	1	1,91	1,04	2,00	12,4	137,0	0,347	9
Learning	Dementia	18	31,72	10,68	30,00	14,11	254,00	-	0,17
	Dementia+Depression	13	36,92	10,62	37,00	18,62	242,00	1,363	3
Recall	Dementia	3	1,33	1,15	2,00	3,00	9,00	-	0,16
	Dementia+Depression	5	3,20	1,64	4,00	5,40	27,00	1,383	7
Recognition	Dementia	9	6,00	2,69	6,00	6,39	57,50	-	0,17
	Dementia+Depression	5	7,80	1,64	8,00	9,50	47,50	1,354	6
High Learning	Dementia	12	2,00	1,13	2,00	8,50	102,00	-	0,24
	Dementia+Depression	6	3,00	1,90	3,00	11,50	69,00	1,169	2
High Recall	Dementia	4	1,75	1,71	1,50	3,75	15,00	-	0,71
	Dementia+Depression	3	2,00	1,00	2,00	4,33	13,00	0,360	9
High Recognition	Dementia	9	5,11	2,80	5,00	6,56	59,00	-	0,92
	Dementia+Depression	3	4,67	4,04	4,00	6,33	19,00	0,094	5
Inconsistency	Dementia	17	3,47	2,00	4,00	13,53	230,00	-	0,15
	Dementia+Depression	13	4,54	1,71	5,00	18,08	235,00	1,417	6
Perseveration	Dementia	8	3,13	2,17	2,00	5,56	44,50	-	0,18
	Dementia+Depression	4	4,75	2,63	5,00	8,38	33,50	1,333	2
Highest Learning	Dementia	18	4,61	1,38	4,00	14,42	259,50	-	0,22
	Dementia+Depression	13	5,23	1,36	5,00	18,19	236,50	1,224	1

Table 4 shows the results of the Mann-Whitney U test for the comparison of the Öktem Verbal Memory Process Test scores of dementia patients with and without additional diagnosis of depression.

When Table 4. is examined, it was determined that there were no statistically significant differences between the Öktem Verbal Memory Process Test scores of dementia patients with and without additional diagnosis of depression included in the study ($p>0.05$).

Table 5: Comparison of geriatric depression scale scores of dementia patients with and without comorbid depression

	DIAGNOSIS	n	$\bar{x}$	s	M	SO	ST	Z	P
Geriatric Depression Scale	Dementia	20	11,8	7,8	8,00	14,6	292,5	-3,183	0,001*
	Dementia+	20	19,5	5,3	19,0	26,3	527,5		
	Depression	0	5	5	0	8	0		

* $p<0,05$

Table 5 shows the results of Mann-Whitney U test for the comparison of Geriatric Depression Scale scores of dementia patients with and without additional diagnosis of depression, and it was determined that the difference between Geriatric Depression Scale scores of dementia patients with and without additional diagnosis of depression was statistically significant ($p<0.05$). It was determined that Geriatric Depression Scale scores of dementia patients with additional diagnosis of depression were higher than those without additional diagnosis of depression.

Table 6: Comparison of cornell scale for depression in dementia scores of dementia patients with and without comorbid depression

	DIAGNOSIS	N	$\bar{x}$	s	M	SO	ST	Z	P
Cornell Scale for Depression in Dementia	Dementia	20	14,8	4,2	14,0	14,6	293,5	-3,15	0,002*
	Dementia+	20	20,7	6,9	22,0	26,3	526,5		
	Depression	0	0	7	0	3	0		

* $p<0,05$

Table 6. shows the results of the Mann-Whitney U test for the comparison of the Cornell Scale for Depression in Dementia scores of dementia patients with and without additional diagnosis of depression.

According to Table 6, there are statistically significant differences between the Cornell Scale for Depression in Dementia scores of dementia patients with and without additional diagnosis of depression ($p < 0.05$). It was determined that the Cornell Scale for Depression in Dementia scores of dementia patients with additional diagnosis of depression were higher than those without additional diagnosis of depression.

Table 7: Comparison of stroop test scores of dementia patients with and without comorbid depression

	DIAGNOSIS	n	$\bar{x}$	s	M	SO	ST	Z	P
Stroop I	Dementia	20	24,7	15,2	21,0	14,9	298,0	-3,033	0,002*
Duration	Dementia+Depression	20	55,4	45,7	37,5	26,1	522,0		
Stroop I	Dementia	20	0,10	0,31	0,00	17,8	356,0	-2,001	0,045*
Error	Dementia+Depression	20	1,20	2,61	0,00	23,2	464,0		
Stroop I	Dementia	20	0,05	0,22	0,00	19,0	380,0	-1,416	0,157
Correction	Dementia+Depression	20	0,20	0,41	0,00	22,0	440,0		
Stroop II	Dementia	20	30,2	18,8	25,5	15,4	309,0	-2,561	0,010*
Duration	Dementia+Depression	19	54,3	32,8	49,0	24,7	471,0		
Stroop II	Dementia	20	2,35	3,38	1,00	18,6	373,5	-0,769	0,442
Error	Dementia+Depression	19	3,16	3,82	2,00	21,3	406,5		
Stroop II	Dementia	20	0,55	1,00	0,00	18,6	373,0	-0,874	0,382
Correction	Dementia+Depression	19	0,68	0,89	0,00	21,4	407,0		

Table 7: Comparison of stroop test scores of dementia patients with and without comorbid depression (Continue)

Stroop III	Dementia	20	36,25	15,84	32,50	12,60	252,00	-	0,000
Duration	Dementia+Depression	18	67,72	25,53	64,00	27,17	489,00	4,037	*
Stroop III	Dementia	20	0,95	1,61	0,00	14,50	290,00	-	0,002
Error	Dementia+Depression	18	4,17	3,68	3,00	25,06	451,00	3,068	*
Stroop III	Dementia	20	0,90	1,02	0,50	19,33	386,50	-	0,913
Correction	Dementia+Depression	18	1,28	2,30	1,00	19,69	354,50	0,109	
Stroop IV	Dementia	20	64,80	22,02	62,50	13,70	274,00	-	0,001
Duration	Dementia+Depression	17	99,35	38,66	93,00	25,24	429,00	3,231	*
Stroop IV	Dementia	20	5,20	6,00	3,50	14,00	280,00	-	0,002
Error	Dementia+Depression	17	13,88	7,66	15,00	24,88	423,00	3,066	*
Stroop IV	Dementia	20	1,90	1,41	2,00	21,25	425,00	-	0,159
Correction	Dementia+Depression	17	1,59	2,32	1,00	16,35	278,00	1,408	
Stroop V	Dementia	20	79,20	28,92	75,00	18,63	372,50	-	0,819
Duration	Dementia+Depression	17	86,18	42,70	76,00	19,44	330,50	0,229	
Stroop V	Dementia	20	10,05	5,83	9,50	14,98	299,50	-	0,014
Error	Dementia+Depression	17	16,59	7,43	21,00	23,74	403,50	2,458	*
Stroop V	Dementia	20	2,60	1,85	2,00	22,90	458,00	-	0,015
Correction	Dementia+Depression	17	1,41	2,00	1,00	14,41	245,00	2,426	*

* $p < 0,05$

Table 7 shows the Mann-Whitney U test results for the comparison of Stroop Test scores of dementia patients with and without additional diagnosis of depression.

It was determined that there were statistically significant differences between Stroop Test I (duration) and Stroop Test I (error) scores of dementia patients with and without additional diagnosis of depression ($p < 0.05$). Stroop Test I (duration) and Stroop Test I (error) scores of dementia patients with additional diagnosis of depression were higher than those without additional diagnosis of depression.

It was determined that there were statistically significant differences between the Stroop Test II (duration) scores of dementia patients with and without additional diagnosis of depression ($p < 0.05$). Stroop Test II (duration) of dementia patients with additional diagnosis of depression was found to be higher than those without additional diagnosis of depression.

There were statistically significant differences between Stroop Test III (duration) and Stroop Test III (error) scores of dementia patients with and without additional diagnosis of depression ($p < 0.05$). Stroop Test III (duration) and Stroop Test III (error) scores of dementia patients with comorbid depression were higher than those without comorbid depression.

There were statistically significant differences between Stroop Test IV (duration) and Stroop Test IV (error) scores of dementia patients with and without additional diagnosis of depression ($p < 0.05$). Stroop Test IV (duration) and Stroop Test IV (error) scores of dementia patients with additional diagnosis of depression in the study were higher than those without additional diagnosis of depression.

There were statistically significant differences between Stroop Test V (error) and Stroop Test V (correction) scores of dementia patients with and without additional diagnosis of depression ($p < 0.05$). Stroop Test V (error) scores of dementia patients with additional diagnosis of depression were higher and Stroop Test V (correction) scores were lower than those without additional diagnosis of depression.

Table 8: Comparison of boston naming test scores of dementia patients with and without comorbid depression

	DIAGNOSIS	n	$\bar{x}$	s	M	SO	ST	Z	P
BNT	Dementia	20	30,8	9,40	28,0	25,6	513,5	-2,806	0,005*
(Total)	Dementia+Depression	20	20,2	12,4	19,0	15,3	306,5		
BNT	Dementia	20	1,30	1,63	1,00	23,2	465,5	-1,677	0,094
(Semantic)	Dementia+Depression	20	0,55	1,05	0,00	17,7	354,5		
BNT	Dementia	20	2,50	3,19	1,00	21,4	429,5	-0,544	0,586
(Phonemic)	Dementia+Depression	20	1,65	1,87	1,00	19,5	390,5		
BNT	Dementia	20	27,0	6,98	25,5	25,7	515,5	-2,858	0,004*
(Spontaneous)	Dementia+Depression	20	18,0	12,0	18,0	15,2	304,5		

* $p < 0,05$

Table 8 shows the results of the Mann-Whitney U test used to compare the Boston Naming Test scores of dementia patients with and without additional diagnosis of depression.

According to Table 8, it was determined that there was a statistically significant difference between the total and spontaneous sub-dimension scores of the Boston Naming Test of dementia patients with and without additional diagnosis of depression ($p < 0.05$). The scores of dementia patients with additional diagnosis of depression on the total and spontaneous sub-dimension of the Boston Naming Test were found to be lower than those without additional diagnosis of depression.

There was no statistically significant difference between the scores obtained from the semantic and phonemic sub-dimensions of the Boston Naming Test by dementia patients with and without additional diagnosis of depression ($p > 0.05$).

Table 9: Correlations between the cornell scale for depression in dementia and the geriatric depression scale

		Cornell Scale for Depression in Dementia		
		Dementia	Dem+Dep	Total
Geriatric Depression Scale	rho	0,380	0,409	0,494
	p	0,048	0,043	0,001
	N	20	20	40

There is a statistically significant positive correlation between the Cornell Scale for Depression in Dementia and the Geriatric Depression Scale scores in dementia patients ($p < 0.05$).

A statistically significant positive correlation is also observed between the scores on the Cornell Scale for Depression in Dementia and the Geriatric Depression Scale in patients with both dementia and depression ($p < 0.05$).

Furthermore, for both patient groups, the Cornell Scale for Depression in Dementia and the Geriatric Depression Scale scores exhibit a statistically significant positive correlation ($p < 0.05$).

5. ARGUMENT, RESULT AND RECOMMENDATIONS

In the study, age, marital status, hand dominance, standardized mini mental state test results, digit sequence test results, Öktem verbal memory process test results, boston naming test results, stroop test results, geriatric depression scale and cornell scale for depression in dementia scores of patients with and without additional diagnosis of depression in dementia patients were evaluated. This part of the study consists of two sub-sections. The first section includes the comparison and evaluation of the data obtained regarding the additional diagnosis of depression in dementia with the literature. In the second part, recommendations for further research are given.

5.1. Argument and Result

In our country, studies examining patients with comorbid depression in dementia are extremely limited. In the present study, using tests/scales whose standardization, validity and reliability studies have been conducted for the culture of our country, elderly individuals with additional diagnosis of depression in dementia and those with dementia but without depression were examined in terms of their cognitive abilities with a rigorous subject selection. In this study, the effects of comorbid depression on neuropsychological functions in patients with dementia were investigated. The findings revealed that there were significant differences between the groups with dementia and no depression and the groups with dementia and depression.

As a result of the study, no significant difference was found between the groups in terms of variables such as gender, age, educational status and marital status. This finding allows the other results of the study to reflect the differences in neuropsychological functions (Table 1).

The SMMST is a test used to assess general cognitive functions. This test measures attention, memory, language and visual-spatial skills. In our study, SMMST scores of dementia patients with comorbid depression (Table 2) were significantly lower than those of patients with dementia alone ($p<0.05$). This suggests that depression causes an additional impairment on cognitive functions.

The Digit Span Test is a test that assesses working memory and executive functions. It consists of two parts: forward and backward number sequence. The forward number sequence measures short-term memory capacity, while the backward number sequence assesses the ability to manipulate and process information. In both the forward and backward number sequence tests (Table 3), the scores of the dementia group with an additional diagnosis of depression were significantly lower ($p < 0.05$). These results reveal the negative effects of depression on working memory and executive functions. In 1979, Cavenar et al. and then Sweet et al. in 1983 reported a significant decline in memory and concentration tests in patients with depression (as cited in Veiel, 1997). In these studies, the participants studied in the sample were patients with major depression and the dementia dimension was not included in these comparisons. In addition, the study by Cavenar et al. is based on examinations with adult but non-geriatric patients. In the meta-analysis conducted by Veiel (1997), it was reported that there was no difference between the depression and control groups in terms of simple attention functions in attention and concentration tests, and it was noted that depression cases tended to be below average. In this study, in which simple attention functions were also examined in dementia patients with comorbid depression and dementia patients without comorbid depression, the scores of the dementia group with comorbid depression were found to be significantly lower.

Öktem Verbal Memory Process Test is a comprehensive memory test that assesses different memory processes such as immediate memory, learning, recall and recognition. Significant differences were observed between the groups in various sub-sections of the Öktem Verbal Memory Process test (immediate memory, learning, recall, recognition, etc.). Especially in the recall and recognition sections, dementia patients with comorbid depression performed worse (Table 4). In addition, this test is one of the tests in which dementia patients without additional diagnosis of depression have as much difficulty as patients with additional diagnosis of depression.

Tests such as the Öktem verbal memory skills test are used to assess memory abilities. However, diseases such as dementia and depression affect short and long-term memory, recall and the ability to process information. These illnesses can also impair attention and concentration. To successfully complete the tests, it is necessary to listen carefully to instructions, process and remember information. However, dementia and depression can reduce patients' ability to pay attention and focus, which can negatively affect test performance. In addition, these patients often experience anxiety and stress,

which can negatively affect their performance during testing. Depression can reduce a person's participation in tests and lower their motivation. It is difficult for people with depression and dementia to do well on such memory skills tests and test results are often affected. In this study, both patients with comorbid depression and patients with dementia without comorbid depression found it difficult to cooperate. Therefore, the most missing value in the data set is in this test.

In 1987, Johnson and Magaro reviewed the literature on memory processes in mood disorders such as depression and mania and observed a lack of focus in learning tests. When the possibility that cognitive impairments in depressed patients may be due to lack of motivation was investigated, a negative correlation was found between the severity of depression and the patient's effort. Regardless of what information is stored in memory, the desire to express information may also be an important component of memory impairment. For example, patients with depression often avoid responding or make more transposition errors, whereas patients with dementia tend to give irrelevant responses. Patients with depression may be more cautious and avoid responding even though they know. This may be a problem with assessing the environment for safety rather than an actual disorder (as cited in Yüksel, N., 2002).

When Table 4 is examined, even if it is not statistically significant, it is possible to see that patients with dementia participated more than patients with depression comorbid dementia in the immediate memory stage, patients with dementia participated more than patients with depression comorbid dementia in the learning score calculation, and patients with dementia participated more than patients with depression comorbid dementia in the recognition stage. In the recall stage, dementia patients with comorbid depression participated more than dementia patients with comorbid depression. When we look at false learning, false recall and false recognition, we may be observing the tendency to give irrelevant answers in dementia patients as stated in Yüksel's (2002) study.

When we look at the Geriatric Depression Scale (Table 5) and the Cornell Scale for Depression in Dementia (Table 6), the group of dementia patients with an additional diagnosis of depression scored significantly higher on both scales than the dementia only group ($p < 0.05$). This result shows that depression symptoms can be assessed by both patients with dementia and their relatives.

The Stroop Test measures attention, inhibitory control and processing speed. The stages of the test (I, II, III, IV and V) are evaluated according to duration and number of errors. In all stages of the Stroop Test, the group with an additional diagnosis of depression in dementia showed significantly worse results (Table 7). This suggests that depression impairs the attention and inhibitory control abilities of patients with dementia. A 1997 study by Veiel noted that people with depression performed very poorly on the Stroop test, a test of mental flexibility. The findings of this study are correct. Again, Austin et al. In 1999, Austin et al. compared the test performance of cases of non-melancholic depression and melancholic depression and found no significant impairment on the Stroop test. Melancholic depression is a type of major depression and can be more severe and more severe than other types. This suggests that the impact of depression on cognitive functioning may vary regardless of the type or severity of depression.

BNT is a test used to assess language functions and object naming ability. The test results (Table 8) show that the total and spontaneous naming scores of the group with an additional diagnosis of depression were significantly lower than the group without a diagnosis of depression ($p < 0.05$). This finding reveals that depression causes an additional impairment in language functions in patients with dementia.

The findings suggest that depression further impairs cognitive functions in patients with dementia. This suggests that depression affects both emotional and cognitive functions and reduces patients' overall quality of life. This study shows that the additional diagnosis of depression causes greater impairment in cognitive functioning in patients with dementia. Depression impairs the ability of dementia patients to maintain activities of daily living and reduces their overall quality of life.

The neuropsychological explanation for why patients with comorbid depression and dementia exhibit worse cognitive functioning compared to those with dementia alone can be attributed to several interrelated factors. Depression directly impacts cognitive functions such as attention, memory, executive functions, and learning, leading to increased cognitive impairment in individuals already affected by dementia. The decreased motivation and energy levels associated with depression make it challenging for patients to engage effectively in cognitive tasks, further exacerbating their performance deficits. Emotional and psychological stress induced by depression can also impair cognitive abilities by reducing attention, memory, and problem-solving skills.

Additionally, depression complicates cognitive processing, making it harder for individuals to process and recall information, thus deepening existing cognitive deficits from dementia. The complexity of treating both conditions simultaneously can hinder effective management and exacerbate cognitive decline. Moreover, depression can amplify the fluctuations in cognitive function associated with dementia, resulting in more erratic cognitive performance. Psychiatrically, symptoms like anxiety, low self-esteem, and hopelessness associated with depression further deteriorate cognitive function. Collectively, these factors highlight the critical need for integrated management of both depression and dementia to mitigate their combined impact on cognitive performance.

The cognitive impairments observed in patients with comorbid dementia and depression can be attributed to various neurobiological mechanisms affecting brain function and neurotransmitter systems. Depression often leads to reduced activity in the prefrontal cortex and frontal lobes, which are crucial for high-level cognitive processes such as attention, executive functions, and decision-making. This reduction in activity can exacerbate cognitive deficits in dementia patients. Additionally, depression adversely impacts the hippocampus, a region vital for memory formation and retrieval, further impairing cognitive abilities. Neurotransmitter imbalances associated with depression, including reductions in serotonin and noradrenaline, can exacerbate cognitive dysfunction by affecting mood regulation, attention, and arousal. Dopamine dysregulation, on the other hand, may contribute to decreased motivation and engagement. Moreover, depression can disrupt synaptic communication and functional connectivity between brain regions, particularly within networks involved in executive functions and cognitive control. Chronic stress and increased inflammation associated with depression can also have detrimental effects on brain function, intensifying cognitive impairments. These neurobiological changes contribute to a compounded effect, where depression exacerbates the cognitive decline seen in dementia, leading to significantly impaired cognitive performance.

In this thesis study, there are various difficulties encountered while applying cognitive tests to patients with dementia and depression. These challenges can affect both the applicability of the tests and the accuracy of the data obtained.

The biggest challenge when conducting cognitive tests with dementia patients is their memory and attention problems. These patients may have difficulty understanding

and following instructions. Communication difficulties make it difficult for patients to understand and answer questions correctly, which can affect the reliability of test results. Furthermore, fluctuations in the cognitive abilities of patients with dementia can cause tests to give inconsistent results on different days. Patients' lack of motivation and fatigue can prolong the testing process, which can lead to loss of attention.

The main challenges when administering cognitive tests with depressed patients are lack of motivation and low energy levels. Depression can negatively affect patients' interest and concentration, making it difficult for them to put enough effort into the tests. When accompanied by anxiety and depression, patients' performance can be further impaired, resulting in variable test results. Furthermore, the emotional state of patients with depression can make them feel uncomfortable during the testing process and find it difficult to cooperate.

To overcome these difficulties, it is important to provide flexibility during the administration of the tests, allowing patients enough time and providing as comfortable an environment as possible. It may also be helpful to take breaks or break the tests into shorter sessions when necessary, taking into account the emotional and cognitive state of the patients. These approaches can improve the accuracy of the tests and patient engagement.

In conclusion, this study highlights that an additional diagnosis of depression leads to more severe cognitive impairment in individuals with dementia than in those without depression. This finding highlights the important role of depression in exacerbating the effects of dementia and underscores the need for special attention and care for this category of individuals. These findings make it clear that a comprehensive approach to the management of both dementia and depression is needed and emphasize the importance of further research in this area.

5.2. Recommendations

If the symptoms of depression in patients with dementia are carefully monitored and treated, in other words, if depression can be managed effectively, it can mitigate the effects of cognitive decline and improve the quality of life of patients. Educating patients and their relatives about the symptoms of depression can facilitate early diagnosis and

treatment. Psychoeducation programs can educate patients and their caregivers about the signs of depression and lead to the provision of appropriate help.

In future studies, should be conducted with larger sample sizes and participants from different regions. Additionally, longitudinal studies can be conducted to examine the long-term effects of depression on dementia. Further investigation of the interaction between dementia and depression and the effects of different treatment modalities may yield important results. In particular, larger clinical trials should be conducted to evaluate the effectiveness of pharmacological and psychological treatments. The efficacy of both pharmacologic and psychosocial treatments should be determined with larger studies in patients with concomitant dementia and depression. A particular focus could be on the efficacy of antidepressants and psychotherapies such as cognitive behavioral therapy. In addition, depression needs to be further investigated in relation to various types of dementia. More importantly, longitudinal studies are needed to determine the impact of depression on the development of dementia. Neuroimaging approaches can be used to reveal the impact of depression on both structural and functional aspects of the brain in patients with dementia. Similarly, in-depth biomarker studies can reveal the biological signals present in both conditions: depression and dementia; moreover, it can shed light on potential determinants of treatment response in these patients.

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APPENDIX

APPENDIX 1. Approval from Ethics Committee

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T.C. ÜSKÜDAR ÜNİVERSİTESİ GİRİŞİMSEL OLMAYAN ARAŞTIRMALAR ETİK KURULU BAŞKANLIĞI	
SAYI: 61351342/ OCAK 2024-36	31/01/2024
Sayın; Prof. Dr. Cumhuri TAŞ (Nisa Ülkü Ece ELÇİN)	
Üsküdar Üniversitesi Girişimsel Olmayan Araştırmalar Etik Kurulunun 29/01/2024 tarihinde yapılan 01 No.lu toplantısında “Demans Hastalarında Depresyon Ek Tanısının Nöropsikolojik Özellikleri” adlı araştırma projenizin etik açıdan uygun olduğuna karar verilmiştir.	
Bilgilerinize rica ederim.	
Prof. Dr. Cumhuri TAŞ Girişimsel Olmayan Araştırmalar Etik Kurulu Başkanı	

APPENDIX 2. Voluntary Consent Form Used in the Study

INFORMED VOLUNTARY CONSENT FORM (IVCF)

TITLE OF THE STUDY:

Neuropsychological Characteristics of Depression Comorbidity in Dementia Patients

You are asked to participate in a research study, the details of which are given below. The decision whether or not to participate in the study is entirely yours. Before deciding whether or not to take part, it is important that you understand why the study is being done, how your information will be used, what the study involves, the possible benefits and risks, and any aspects that may cause discomfort. Please take time to read the following information carefully. If you decide to participate in the study, please sign the **Consent to Participate Form**. You are free to leave the study at any time. You will not be paid for participating in the study or asked for any financial contribution/material contribution. / All materials to be used in the study and all expenses to be incurred will be covered by the researcher (can be one of two sentences)

SUBJECT AND PURPOSE OF THE STUDY:

Dementia and depression are two important psychiatric conditions that are common among the elderly population. Both conditions can significantly affect an individual's quality of life and daily functioning. The most important common feature of dementia and depression in old age is that both involve losses in the cognitive domain. In both conditions, cognitive functions are affected differently. This study investigates the neuropsychological characteristics of comorbid depression in patients with dementia.

STUDY PROCEDURES:

If you participate in this study, you will be given six forms to fill out. The first one will ask for some sociodemographic information. This form was prepared by the researchers. The other five forms are cognitive tests. You are expected to fill in all forms completely.

WHAT ARE THE POSSIBLE BENEFITS OF PARTICIPATING IN THE STUDY?

If you participate in the study, you can help us add data and improve existing knowledge by providing support to the literature on this topic

HOW WILL MY PERSONAL INFORMATION BE USED?

Name, surname or any data that can decipher your person will not be used or disclosed.

PERSONS TO CONTACT FOR QUESTIONS AND PROBLEMS:

Psychologist Nisa Ülkü Ece ELÇİN

mail

number

Prof. Dr. Cumhur TAŞ

mail

number

CONSENT TO PARTICIPATE

I have discussed the above information in detail with the researcher and he answered all my questions. I have read and understood this informed consent document. I agree to participate in this study and sign this consent document of my own free will. This consent does not override any relevant laws and regulations. The researcher has given me a copy of this document for my safe-keeping, including the points to be considered during the study.

<i>Voluntary Name And Surname:</i>		<i>Date and Signature:</i>
<i>Phone Number:</i>		

<i>Guardian (If any) Name and Surname:</i>		<i>Date and Signature:</i>
<i>Phone Number:</i>		

<i>Researcher² Name and Surname:</i>		<i>Date and Signature:</i>
<i>Address and Phone Number:</i>		

1: The person who witnessed the volunteer's information process from beginning to end

2: The person who informs the volunteer about the research

APPENDIX 3. Sociodemographic Data Form

DATE OF EVALUATION:

TIME:

PATIENT'S	
NAME	
SURNAME	
GENDER	Female () Male ()
DATE OF BIRTH AND AGE	
EDUCATIONAL BACKGROUND	Uneducated () Literate () Primary school () Secondary school () High School () Undergraduate () Master's- Doctorate ()
MARITAL STATUS:	Single () Married ()... number of children Widowed-Divorced ()
DOMINANT HAND	Right () Left ()
HEARING LOSS	Yes ()...Nature No ()
LOSS OF VISION	Yes ()...Nature No ()
CHRONIC DISEASE	HT () DM () Thyroid ()... Feature..... Other.....
PAST SURGERIES	
ALCOHOL AND TOBACCO USE:	Alcohol ()... Per week.... Monthly..... In a year..... Cigarettes ()... per day.....
PSYCHIATRIC DIAGNOSIS OR FAMILY HISTORY, IF ANY	
NEUROLOGICAL DIAGNOSIS, IF ANY:	Dementia () Other..... MS () Huntington () Parkinson ()
FAMILY STORY:	Yes ()... Degree of closeness..... Attribute..... No ()
HEAD TRAUMA	Yes ()..... Attribute..... No ()
COMPLAINTS:	

FORGETFULNESS	Difficulty Finding Words () Forgetting and confusing names () Remember where you put things () Don't forget the items to be taken () Loss of Location-Direction () Specify.....
DURATION OF FORGETFULNESS COMPLAINT	
LOSS OF BALANCE:	Yes () Duration..... No ()
TREMOR:	Yes () Right ().... Left () Both hands () No ()
PAST STROKE	Yes ()... Type.....Duration.... None ()
SLEEP:	Less ().....duration..... Excessive sleep ().....duration..... Normal ().... duration.....
DID S/HE SLEEP WELL LAST NIGHT?	Yes () No ()... Less than Normal ()
APPETITE:	Normal () Less () More ()
WHEN WAS THE LAST TIME S/HE DRANK TEA-COFFEE-COLA-GARBAGE:	1 hour ago () 2 hours ago () 3 hours ago () 4 hours ago () 5 hours ago () Other:.....
PERSONAL CARE SKILLS:	Unassisted () Assisted (due to physical limitation) () With help ()
PREVIOUS NEUROPSYCHOLOGICAL EVALUATION RESULTS, IF ANY:	
WITH WHOM S/HE LIVES	alone () spouse () spouse and child () with child ()
SOCIAL LIFE	Book Reading ()..... per day..... content..... Regular hobby ()..... ..frequency.....content..... Friend Chats()..... frequency..... Shopping ().....frequency.... Hiking.....().... Frequency....

APPENDIX 4. Standardized Mini Mental State Examination

STANDARDIZE MINI MENTAL TEST

Name/Surname : Age:

Education (year) : Dominant Hand :

Date : T-Score :

Orientation (Total Points of 10)

What year are we in ()
What season are we in ()
What month are we in ()
What day of the month is it today ()
What day are we in ()
What country do we live in ()
What city are you in right now ()
What district are you in right now ()
What building are you in right now ()
What floor are you on in this building ()
(1 point is given for each item.)

Recording Memory (Total points of 3)

Listen carefully to the three names I am about to tell you and repeat them after I finish (table, flag, dress) (20 seconds, 1 point for each correct name).

..... ()

Attention and Calculation (Total Points of 5)

Go backwards from 100, subtracting 7..

Continue until you hear stop.

(1 point for each correct operation) (100, 93, 86, 79, 72, 65)

..... ()

Remembrance (Total Points of 3)

Do you remember the words you repeated above? Say what you remember (table, flag, dress)

(1 point is given for each correct name).

..... ()

Language (Total Points of 9)

1. What are the names of the objects you see (clock, pen) (20 seconds, 1 point for each correct name) (Total points of 2)

..... ()

2. Listen carefully to the sentence I am going to tell you now and repeat it after I finish. "If and but I don't want to" (10 seconds, 1 point for correct and complete sentence).

..... ()

3. Now I am going to ask you to do something, listen to me carefully and do as I say, "Take the paper on the table with your right/left hand, fold it in half with both hands and put it on the floor please". (30 seconds is allowed, 1 point is given for each correct action).

..... ()

4. Now I will give you a sentence. *Read it and do what it says (1 point for correct action).

..... ()

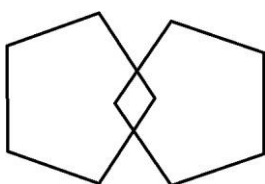
5. Now write a meaningful sentence that comes to your mind on the paper I will give you (30 seconds is allowed, 1 point is given for a meaningful sentence).

..... ()

6. Draw the same shape as I will show you. *(1 minute time is given, 1 point is given for the number of sides, complete shape)

***Please show the sentence and figure on the back page.**

CLOSE YOUR EYES



Notes:

.....

.....

.....

.....

.....

.....

.....

APPENDIX 5. Digit Span Test

DIGIT SPAN TEST

FORWARD	SCORE	BACKWARD	SCORE
6 – 4 – 3 – 9	4	2 – 8 – 3	3
7 – 2 – 8 – 6	4	4 – 1 – 5	3
4 – 2 – 7 – 3 – 1	5	3 – 2 – 7 – 9	4
7 – 5 – 8 – 3 – 6	5	4 – 9 – 6 – 8	4
6 – 1 – 9 – 4 – 7 – 3	6	1 – 5 – 2 – 8 – 6	5
3 – 9 – 2 – 4 – 8 – 7	6	6 – 1 – 8 – 4 – 3	5
5 – 9 – 1 – 7 – 4 – 2 – 3	7	5 – 3 – 9 – 4 – 1 – 8	6
4 – 1 – 7 – 9 – 3 – 8 – 6	7	7 – 2 – 4 – 8 – 5 – 6	6
5 – 8 – 1 – 9 – 2 – 6 – 4 – 7	8	8 – 1 – 2 – 9 – 3 – 6 – 5	7
3 – 8 – 2 – 9 – 5 – 1 – 7 – 4	8	4 – 7 – 3 – 9 – 1 – 2 – 8	7
Forward Score:			
Backward Score:			

ÖKTEM VERBAL MEMORY PROCESS TEST (ÖKTEM VMPT)
(LIST A)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Score
	Drum	Curtain	Bell	Coffee	School	Mom	Garden	Hat	Moon	Farmer	Nose	Turkey	Colour	Home	River	
1																
2																
3																
4																
5																
6																
7																
8																
9																
10																
U S B																
T																
Tot. Rec.																
F. R.																

APPENDIX 7. Back Side of Öktem Verbal Memory Processes Test

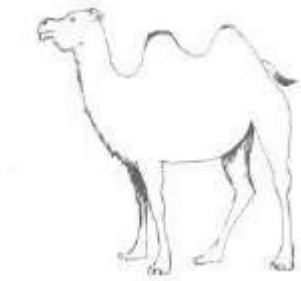
WALL	MOON
DRUM	QUINCE
ZURNA	SUN
WINDOW	FIELD
CURTAIN	FARMER
PITA	FARM
BELL	THROAT
CHAIN	NOSE
DOOR	MOUTH
TEA	TURKEY
COFFEE	COCK
BREAKFAST	DITCH
GAME	PICTURE
SCHOOL	COLOUR
TEACHER	AD
SISTER	ROOM
MOM	HAND
DAD	HOME
TREE	SEA
FLOWER	RIVER
GARDEN	STREAM
SCARF	
HAT	
CAP	

VMPT Scores	
Immediate Memory	
Learning Score	
Reaching the Criterion	
Highest Learning	
Learning Miscue Score	
Perseveration	
Inconsistency Score	
Delayed Spontaneous Recall	
Recognition	
TOTAL Recall	
False Recall Score	
False Recognition	

APPENDIX 8. Boston Naming Test Sample Images



.....



.....



.....

APPENDIX 9. Geriatric Depression Scale (GDS)

Geriatric Depression Scale

Please answer the following questions about how you felt in the last week of your life by marking the answer that applies to you.

Yes No

- 1) Are you basically satisfied with your life?
- 2) Do you still pursue many of your personal activities and interests?
- 3) Do you feel that your life is empty?
- 4) Are you often bored?
- 5) Are you hopeless about the future?
- 6) Do you ever feel disturbed by thoughts you can't get out of your head?
- 7) Are you usually in a good mood?
- 8) Are you afraid that something bad will happen to you?
- 9) Do you feel happy most of the time?
- 10) Do you often feel helpless?
- 11) Do you often feel restless and restless?
- 12) Would you rather stay at home than go out and do something new?
- 13) Do you often worry about the future?
- 14) Do you feel that your memory is weaker than most?
- 15) Do you think it is a very good thing to be alive right now?
- 16) Do you often feel sad and gloomy?
- 17) Do you feel worthless as you are now?
- 18) Do you grieve a lot about the past?
- 19) Do you find life enjoyable and exciting?
- 20) Is it difficult for you to start new projects?
- 21) Do you feel full of energy?
- 22) Do you think you are in an unsolvable situation?
- 23) Do you think most people are better off than you?
- 24) Do you often get upset over small things?
- 25) Do you often feel like crying?
- 26) Do you have difficulty concentrating?
- 27) Do you like to start your day in the morning?
- 28) Do you avoid attending social gatherings?
- 29) Is it easy for you to decide?

30) Is your mind as clear as it used to be?

APPENDIX 10. Cornell Scale for Depression in Dementia

NAME _____	AGE _____	SEX _____	DATE _____
------------	-----------	-----------	------------

Cornell Scale for Depression in Dementia

Ratings should be based on symptoms and signs occurring during the week before interview. No score should be given if symptoms result from physical disability or illness.

SCORING SYSTEM

a = Unable to evaluate	0 = Absent	Score greater than 12 = Probable Depression
1 = Mild to Intermittent	2 = Severe	

A. MOOD-RELATED SIGNS

	a	0	1	2
1. Anxiety; anxious expression, rumination, worrying	☐	☐	☐	☐
2. Sadness; sad expression, sad voice, tearfulness	☐	☐	☐	☐
3. Lack of reaction to pleasant events	☐	☐	☐	☐
4. Irritability; annoyed, short tempered	☐	☐	☐	☐

B. BEHAVIORAL DISTURBANCE

	a	0	1	2
5. Agitation; restlessness, hand wringing, hair pulling	☐	☐	☐	☐
6. Retardation; slow movements, slow speech, slow reactions	☐	☐	☐	☐
7. Multiple physical complaints (score 0 if gastrointestinal symptoms only)	☐	☐	☐	☐
8. Loss of interest; less involved in usual activities (score 0 only if change occurred acutely, i.e., in less than one month)	☐	☐	☐	☐

C. PHYSICAL SIGNS

	a	0	1	2
9. Appetite loss; eating less than usual	☐	☐	☐	☐
10. Weight loss (score 2 if greater than 5 pounds in one month)	☐	☐	☐	☐
11. Lack of energy; fatigues easily, unable to sustain activities	☐	☐	☐	☐

D. CYCLIC FUNCTIONS

	a	0	1	2
12. Diurnal variation of mood; symptoms worse in the morning	☐	☐	☐	☐
13. Difficulty falling asleep; later than usual for this individual	☐	☐	☐	☐
14. Multiple awakenings during sleep	☐	☐	☐	☐
15. Early morning awakening; earlier than usual for this individual	☐	☐	☐	☐

E. IDEATIONAL DISTURBANCE

	a	0	1	2
16. Suicidal; feels life is not worth living	☐	☐	☐	☐
17. Poor self-esteem; self-blame, self-depreciation, feelings of failure	☐	☐	☐	☐
18. Pessimism; anticipation of the worst	☐	☐	☐	☐
19. Mood congruent delusions; delusions of poverty, illness or loss	☐	☐	☐	☐

NOTES/CURRENT MEDICATIONS:	Score	
ASSESSOR:		

Instruction for use: (Cornell Dementia Depression Assessment Tool)

1. The same CNA (certified nursing assistant) should conduct the interview each time to assure consistency in the response. 2. The assessment should be based on the patient's normal weekly routine. 3. If uncertain of answers, questioning other caregivers may further define the answer. 4. Answer all questions by placing a check in the column under the appropriate-	ly numbered answer. (a=unable to evaluate, 0=absent, 1=mild to intermittent, 2=severe). 5. Add the total score for all numbers checked for each question. 6. Place the total score in the "SCORE" box and record any subjective observation notes in the "Notes/Current Medications" section. 7. Scores totaling twelve (12) points or more indicate probable depression.
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APPENDIX 11. Stroop Test TBAG Form

Section I: Reading Color Name Printed in Black Name

B	Y	R	G
G	B	Y	R
G	R	B	Y
R	G	Y	B
Y	R	G	B
R	B	Y	G

Section III: Saying Shape Color Name

G	B	Y	R
Y	R	G	B
B	G	Y	R
B	Y	R	G
R	G	B	Y
Y	G	B	R

Section II: Reading Color Printed Color

B	Y	R	G
G	B	Y	R
G	R	B	Y
R	G	Y	B
Y	R	G	B
R	B	Y	G

Section IV: Word Color without a Color

G	B	Y	R
Y	R	G	B
B	G	Y	R
B	Y	R	G
R	G	B	Y
Y	G	B	R

Section V: Word Color with Color Name

G	B	Y	R
Y	R	G	B
B	G	Y	R
B	Y	R	G
R	G	B	V
Y	G	B	R

(B = BLUE, G = GREEN, R = RED, Y = YELLOW)