Clinical Study of Memory Disorders in Aging Patients and Associated Pathology

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ABSTRACT

We have observed semantic memory and episodic memory disorders in patients ranging from 40 to 92 years-old (100%), with associated cardiovascular diseases and blood hypertension (82%), sleep disorders (50%), neurobehavioral disorders (44%), such as depression, anxiety, aggression, and vascular dementia, disorders of language (36%), neurosensory disorders (28%), as diminution of visual and hearing acuity, dizziness (26%), Parkinson disease (34%), Alzheimer disease (21%), gait disturbances (10%), vertigo (10%), cervicalgia and cervicogenic headache (10%) trigeminal neuralgia (2%). We found as comorbidities the following non-nervous diseases: metabolic diseases as diabetes (21%) and hypothyroidism (5%), gastrointestinal pathology (21%), such as constipation, loss of sphincter control, and gastritis, arthritis (13%), prostatic hypertrophy (1%) and loss of weight (1%). A detailed discussion of every pathological condition is provided.

Keywords
Memory disorders, Aging patients, Cardiovascular, Neurological, Neurobehavioral and metabolic diseases

Introduction

Vascular Cognitive Impairment (VCI) was proposed as an umbrella term to include subjects affected with any degree of cognitive impairment resulting from Cerebrovascular Disease (CVD), ranging from Mild Mognitive Impairment (MCI) to vascular dementia [1]. According to Ciconnetti et al. [2], age, sex, family history, educational level, and risk factors, such as hypertension, hypercholesterolemia, diabetes mellitus and tobacco, might contribute to degenerative forms of cognitive impairment. Vascular Cognitive Impairment (VCI) incorporates the complex interactions between vascular aetologies, risk factors and cellular changes within the brain and cognition [3]. Neuropsychiatric symptoms were common in patients with vascular cognitive impairment with and without dementia. It deserves attention that neuropsychiatric symptoms as well as cognitive deficits frequently arise from cerebrovascular disease regardless of the development of dementia [4]. Neuropathologic changes associated with cognitive impairment include multifocal and/or diffuse disease and focal lesions: multi-infarct encephalopathy, white matter lesions or arteriosclerotic subcortical leukenoencephalopathy, multifocal leukenoencephalopathy, mixed cortico-subcortical type, borderline/watershed lesions, rare granular cortical atrophy, post-ischemic encephalopathy and hippocampal sclerosis. [5]. Lifestyle variables, including subjective sleep problems and stress, are factors known to affect cognition [6]. Aging is characterized by progressive memory decline that can lead to dementia when associated with neurodegeneration [7].

Heart Failure (HF) is the most common cardiovascular disease in elderly population, and it is associated with neurocognitive function decline, which represent underlying brain pathology diminishing learning and memory faculties [8]. Most old patients with nuclear resonance images of microangiopathy and leukenoencephalopathy showed loss of short and long term memory (implicit and explicit memory) [9].

In the present investigation we studied from the clinical point of view 38 patients with memory disorders or loss of memory in aging patients and their associated comorbidities and risk factors in an attempt to get deeper insight into the multifactorial factors and pathophysiological basis of memory disturbances.

Material and Methods

Case report study

Case 1: ZC, 72 years-old, F. Disorders of episodic memory, working memory and executive function, sleep disorder, fearful, depression, occipital headache, blood hypertension and erosive gastritis.

Diagnosis: Amnestic mild cognitive impairment. Disorders of memory, blood hypertension sleep disorder, depression.

Case 2: GM, 54 years-old. F. Disorders of episodic memory, trigeminal neuralgia, and stress.

Diagnosis: Disorders of memory, trigeminal neuralgia, and stress.

Case 3: VP, 52 years-old, F. Disorders of episodic and working memory, insomnia, and depression.

Diagnosis: Amnestic mild cognitive impairment, insomnia, depression.

Case 4: RE, 74 years-old, F. Semantic memory disorder. Frequent

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loss of consciousness, tinnitus, dizziness, blood hypertension, cervicalgia and hypothyroidism.

Diagnosis: Semantic memory disorder, blood hypertension, cervicalgia and hypothyroidism.

Case 5: TC, 78 years-old, M. Posttraumatic loss of memory, Brain trauma in car accident. Loss of semantic and working memory. NMR images showed brain ischemia.

Diagnosis: Posttraumatic and semantic loss of memory, brain ischemia

Case 6: MOR, 63 years-old, F. Frequent loss of episodic memory, dizziness, vertigo, bradycardia, absent tendon reflexes. Normal blood pressure.

Diagnosis: Amnestic mild cognitive impairment. Dizziness, Vertigo

Case 7: GS, 87 years-old, F. Episodic Memory disorders, Cervicalgia, loss of sphincter control, insomnia, and hypoaacusia.

Diagnosis: Amnestic mild cognitive impairment. Cervicalgia. Insomnia. Hearing loss. Loss of sphincter control.

Case 8: FMD, 74 years-old, F. Loss of semantic and working memory, tremor, bradykinesia, severe cognitive deficit, loss of consciousness, tachicardia, loss of sphincter control, anxiety, family stress, dyspnea, and hypersalivation,

Diagnosis: Alzheimer disease and Parkinsonism. Anxiety. Stress. Loss sphincter control.

Case 9: CD, 72 years-old, F. Loss of semantic and working memory, hand and body tremor, bradykinesia, blood hypertension, renal lithiasis, fatty liver, depression, bronchial asthma. Cervicalgia. Family history of Alzheimer disease.

Diagnosis: Mixed Alzheimer disease and Parkinson disease. Loss of semantic and working memory Blood hypertension. Renal lithiasis. Fatty liver. Depression. Bronchial asthma. Cervicalgia

Case 10: CS, 76 years old, M. Episodic memory disorders, temporospatial disorientation, diminution of visual acuity, blood hypertension. Prostatic cancer 15 years ago.

Diagnosis: Amnestic mild cognitive impairment. Blood hypertension and diminution of visual acuity

Case 11: EC, 76 years-old, F. Episodic memory disorders, aggression, cognitive deficit, blood hypertension, venous thrombosis.

Diagnosis: Amnestic mild cognitive impairment. Blood hypertension. Aggression. Venous thrombosis.

Case 12: EM, 65 years-old, M. Episodic and working memory disorders. Dizziness, vertigo, holocranial headache, cervicalgia, diminution of visual acuity, blood hypertension and anaemia.

Diagnosis: Amnestic mild cognitive impairment. Cervicogenic Headache. Blood hypertension and Anaemia.

Case 13: IVS, 74 years-old, F. Loss of semantic memory, ischemic cerebrovascular accident four years ago, cardiac insufficiency, mental confusion, temporospatial disorientation, thrombophlebitis and eczematous skin lesion of left leg.

Diagnosis: Amnestic mild cognitive impairment, cardiac insufficiency, mental confusion and eczematous skin lesion.

Case 14: JP, 75 years-old, F. Loss of semantic memory, dizziness, vertigo, frontal-temporal-occipital headache, diabetes type II, blood hypertension, loss of body weight, and sleep disorders.

Diagnosis: Alzheimer disease, Headache. Diabetes. Malnutrition. Sleep disorders.

Case 15: LG, 81 years old, F. Loss of semantic and working memory, blood hypertension, insomnia, holocranial cephalae, diabetes. Family history of Alzheimer disease.

Diagnosis: Alzheimer disease. Blood hypertension. Insomnia, Headache. Diabetes.

Case 16: LR, 69 years-old, F. Loss of semantic and working memory, asthenia, headache, resting and effort dysnoea, hearth failure, blood hypertension, dislipidaemia, and venous insufficiency.

Diagnosis: Alzheimer disease. Hearth failure Blood hypertension, dislipidaemia, and venous insufficiency

Case 17: LR, 64 years-old, F. Episodic and working memory disorders, blood hypertension, gait disturbances, diabetes, hypothyroidism and hearth failure.

Diagnosis: Amnestic mild cognitive impairment. Hearth failure, Diabetes, Hypothyroidism

Case 18: MAF, 72 years-old, F. Episodic memory disorders, tremor, anxiety, and heart arrhythmia.

Diagnosis: Episodic memory disorder. Parkinson disease, Anxiety. Hearth failure.

Case 19: MM, 78 years-old, F. Semantic memory disorders, tremor in right arm, bradykinesia, loss of equilibrium, blood hypertension, hearth surgery 13 years ago, sleep disorders and nightmares, and depression.

Diagnosis: Semantic memory disorder. Parkinson disease, blood hypertension. Hearth failure. Sleep disorders and nightmares. Depression.

Case 20: MM, 72 years old. F. Loss of semantic memory, seizures, loss of consciousness, deficit visual acuity, cephalae, dizziness, blood hypertension and diabetes.

Diagnosis: Loss of semantic memory. Seizures. Blood hypertension. Diabetes. Headache.

Case 21: ML, 79 years-old, F. Episodic memory and sleep disorders, reiterative speech, mood disorders, glaucoma, and intentional tremor.

Diagnosis: Amnestic mild cognitive impairment. Language and sleep disorders.

Case 22: CG, 71 years-old, F. Loss of semantic and working memory since four years ago, blood hypertension, sleep disorders, NMR images showed leukoencephalopathy and microangiopathy.

Diagnosis: Alzheimer disease. Small vessel disease Blood hypertension, Sleep disorders.

Case 23: JP, 65 years-old, F. Episodic memory disorders, frontal-parietal-occipital pulsatile headache, dizziness, fine tremor, diabetes, blood hypertension, diminution of visual acuity and sleep disorders.

Diagnosis: Amnestic mild cognitive impairment. Headache. Diabetes. Blood hypertension

Case 24: RR, 77 years-old, M. Semantic memory disorders. Tremor of arms and hands, gait disturbances, loss of body weight, loss of sphincter control, visual hallucinations. Diminution of visual acuity, agarophobia, sleep disorders.

Diagnosis: Semantic memory disorder. Simultaneous Parkinson disease and Vascular demencia, and sleep disorders.
Case 25: MEP, 58 years-old, F. Loss of episodic memory, occipital heaviness, tremor tongue, bradykinesia, speech disorders, tension headache, and gait disorders.

Diagnosis: Loss of episodic memory. Parkinson disease. Headache

Case 26: CI, 75 years-old, F. Loss of semantic and working memory, tremor in both hands, sleep disorders, tinnitus, and speech disorder.

Diagnosis: Alzheimer disease and parkinsonism. Language and sleep disorder and tinnitus.

Case 27: DV, M. 65. Semantic memory disorders, tremor in both hands, language and speech disorders, gait disturbances, insomnia, increased salivation, and prostatic hypertrophy.

Diagnosis: Parkinson disease. Insomnia. Prostatic hypertrophy

Case 28: JR, 84 years-old, M. Semantic and working memory disorders, right hand tremor, language disorder, Patient in a wheelchair by generalized arthritis, diabetes, loss of sphincter control.

Diagnosis: Semantic and working memory disorder, Diabetes, Parkinson disease. Arthritis.

Case 29: LR, 70 years-old, F. Disorders of semantic and working memory, tremor in both hands, congenital arthropathy, gait disorders, depression, anxiety, constipation.

Diagnosis: Disorders of semantic and working memory. Parkinson disease. Congenital arthropathy, Depression. Anxiety

Case 30: AP. 92 years old, M. Loss of episodic memory and preserved semantic long term memory, elevated systolic blood pressure and low diastolic pressure, Sinusal arrhythmia bradycardia, severe dizziness, neck pain, language disturbances, sleep disorder, constipation, NMR showed severe cortical atrophy according to age, and cortical calcifications, periventricular hypodensity suggestive of leukoaraiosis leukoencephalopathy, granulomatous in nature.

Diagnosis: Amnestic mild cognitive impairment. Hypertension. Cervicalgia. Sleep disorders Vascular disease. Constipation

Case 31: MM, 44 years old, F. Loss of semantic and working memory, chronic headache, Depression, High blood pressure, dyslipidaemia, NMR images showed subcortical and supratentorial leuкоencephalopathy, degenerative cervical and lumbar discopathy.

Diagnosis: Alzheimer disease. Chronic cervicogenic headache, depression, High blood pressure.

Case 32: MF, 44 years old, F. Semantic memory disturbances, high blood pressure, depression, hypoacusia, and dyslipidemia. NMR showed supratentorial subcortical leuкоencephalopathy.

Diagnosis: Memory disturbances. High blood pressure. Depression

Case 33: CI, 78 years old, F. Disorders of semantic and working memory, tremor in both hands, bradykinesia, gait disorders, speech difficulties, decrease hearing acuity, tinnitus, weight loss, and sleep disorders,

Diagnosis: Alzheimer disease and Parkinson disease. Sleep disorders. Neurosensory disorders

Case 34: JR, 70 years-old, F. Semantic and working memory disorders. Tremor in both hands, edema of lower extremities, digit arthritis, gait disturbances, depression, anxiety, constipation, blood hypertension, protrusion of spine disk L5-S1.

Diagnosis: Disorder of semantic and working memory and Parkinson disease, Depression, Anxiety. Blood hypertension, Arthritis

Case 35: JL, 84 years old, M. Semantic memory disorders. Tremor in right hand since three years ago, diabetes, arthritis, disorders of language and blood hypertension.

Diagnosis: Semantic memory disorders. Parkinson disease. Diabetes. Blood hypertension

Case 36: MV, 69 years-old, F. Loss of episodic memory. Doppler showed moderate carotid obstruction.

Diagnosis: Amnestic mild cognitive impairment. Loss of episodic memory. Vascular disease

Case 37: LMR, 69 years old, F. Loss of semantic and working memory, blood hypertension, dyslipidemia, headache, effort dysnoea, cardiac and venous insufficiency.

Diagnosis: Alzheimer disease, Loss of semantic and working memory, blood hypertension and heart failure.

Case 38: DV, 69 years old, M. Semantic memory disorders, dizziness, speech difficulties, asthenia, depression, sleep disorders, gait disturbances, hypersalivation, prostatic hypertrophy and protrusion of spine disk L5-S1.

Diagnosis: Semantic memory disorders. Depression. Anxiety. Sleep disorders. Blood hypertension. Prostatic hypertrophy.

Results

Interpretation of results

We have observed semantic memory and episodic memory disorders in patients ranging from 40 to 92 years-old (100%), mainly in female patients, associated to cardiovascular diseases and blood hypertension (82%), sleep disorders (50%), neurobehavioral disorders, such as depression, anxiety, aggression, and vascular demencia (44%), disorders of language (36%), neurosensory disorders, such as diminution of visual and hearing acuity (28%), dizziness (26%), Parkinson disease (34%), Alzheimer disease (21%), gait disturbances (10%), vertigo (10%), cervicalgia and cervicogenic headache (10%), neurosensory disorders (5%). We observed as comorbidities the following non-nervous diseases: metabolic diseases as diabetes (21%) and hypothyroidism (5%), gastrointestinal pathology, such as constipation, loss of sphincter control, and gastritis (21%), arthritis (13%), trigeminal neuralgia (2%), and allergic disease like asthma bronchial (2%), prostatic hypertrophy (1%) and loss of weight (1%).

According to their high frequency the most risk factors associated to memory disorders are cardiovascular diseases and blood hypertension (82%), sleep disorders (50%), neurobehavioral disorders (44%), such as depression, anxiety, aggression, and vascular demencia, disorders of language (36%), neurosensory disorders (28%), such as diminution of visual and hearing acuity, dizziness (26%).

We have found the following clinical mixed subtypes: five patients with mixed Alzheimer and Parkinson disease syndrome (13%), two cases with Alzheimer disease and diabetes (2%), and one case with Parkinson disease and vascular demencia (1%).
The NMR images showed brain ischemia, subcortical and supratentorial leukoencephalopathy, and cervical and lumbosacral spine pathology.

**Discussion**

**Memory disorders and cardiovascular diseases**

In the present study we have reported memory disorders in patients with cardiovascular diseases and blood hypertension (82%). Toledo et al. [8], emphasized on related memory decline in cerebral ischemia, neuroinflammation, oxidative stress, mitochondrial and DNA alterations. Castejón et al. [9], reported a clinical and neuroimaging study of thirty three patients with vascular dementia in neurological, psychiatric and cardiovascular diseases exhibiting microangiopathy and leukoencephalopathy. Gottesman et al. [10], postulated that hypertension is a potential cause of cognitive decline and dementia, and its greatest influence on cognition may occur in middle age.

**Memory disorders in parkinson and alzheimer diseases**

In the present paper we have found semantic and working memory disorders in 21 cases (55%), including patients with Parkinson disease (34%) and Alzheimer disease (21%). Semantic memory refers to our long-term knowledge of word and object meaning. Semantic memory is a dynamic system whose effectiveness relies on the coordination of multiple components distributed across a large network of cortical regions [11]. The disruption of semantic memory as a result of brain damage may have profound negative consequences on an individual’s ability to name objects and process concepts [12]. This disruption was observed in patients with posttraumatic car accidents, Parkinson disease, Alzheimer disease, mixed syndromes of Parkinson and Alzheimer disease, Alzheimer disease and diabetes, and Parkinson disease and vascular dementia. Parkinson disease and diabetes, ischemic cerebro-vascular accident, diabetes and hypothyroidism, hearth failure and blood hypertension, and depression, anxiety and sleep disorder. According to Laisney et al. [13], the progressive deterioration begins at the level of the concept attributes, and further involves the concepts themselves, and implicates the left posterior temporal region involved in semantic processing for pictures, abstract words, and concrete words.

**Memory disorders and sleep disorders**

We have observed memory disorder and sleep disorders in 50% of patients examined. Sleep is known to facilitate the consolidation of memories learned before sleep as well as the acquisition of new memories to be learned after sleep. Neurotransmitters such as noradrenaline and glutamate likewise facilitates memory processing during sleep [14]. According to Andersson et al. [15], stress, sleep, sensory sensitivity, depression, and negative life events are observed in patients presenting memory disorders. Hypertension, diabetes mellitus, renal failure, respiratory diseases such as asthma, immune disorders, gastroesophageal reflux disease, physical disability, dementia, pain, depression, and anxiety are all associated with sleep disturbances. [16]. Fortier-Brochu and Morin [17], found clinically significant alterations in attention and episodic memory in individuals with insomnia. Insomnia is characterized by difficulty initiating and maintaining sleep, along with dissatisfaction with sleep quality or quantity [18,19].

Insomnia in cognitively unimpaired adults at increased risk for AD is associated to poorer performance in some executive functions and volume changes in cortical and subcortical gray matter, including key areas involved in Alzheimer’s disease, as well as decreased white matter diffusivity [20].

Feld et al. [21], analyzed the excitatory neurotransmitter glutamate that plays a prominent role in inducing synaptic consolidation, the inhibitory GABAergic system and the strengthening memories during sleep, the dopaminergic reward system that plays a side role for enhancing relevant memories during sleep, and acetylcholine and cortisol whose low tone during slow wave sleep is crucial in supporting hippocampal-to-neocortical memory transmission.

**Memory disorders and alzheimer disease**

In the present study we have reported six patients with Alzheimer disease (21%). Numerous studies have widely demonstrated that Alzheimer’s Disease (AD) is a progressive neurodegenerative disease marked by deficits in episodic memory, working memory (WM), and executive function. Executive dysfunction in AD include poor selective and divided attention, failed inhibition of interfering stimuli, and poor manipulation skills [22]. The early progression continuum of Alzheimer’s disease has been considered to advance through subjective cognitive decline, non-amiloid mild cognitive impairment, and amnestic mild cognitive impairment [23].

High blood pressure and Alzheimer disease was observed in six cases in the present study Rochoy et al. [24], highlighted a possible association of Alzheimer’s Disease (AD) with intracranial hypertension, which has also been related to pathological manifestations of Alzheimer’s disease related with memory disorders, such as senile plaques, neurofibrillary tangles, hippocampal atrophy. Hypertension may also lead to vessel wall changes in the brain, leading to hypoperfusion, ischemia and hypoxia which may initiate the pathological process of AD [25,26].

The most established Magnetic Resonance Imaging (MRI) finding is hippocampal atrophy, which is related to memory decline and currently used as a diagnostic criterion for AD [27]. The medial temporal lobe system, the posteromedial cortices, including the precuneus and posterior cingulate, are also thought to play a key role in both memory encoding and retrieval, and are connected to the medial temporal lobe system [28].

**Mixed Clinical Syndromes of Parkinson (PD) and Alzheimer Diseases (AD)**

We have found 13% of patients with a mixed form of Parkinson Disease and Alzheimer disease. Classical forms of AD and PD, both types of lesions can coexist suggesting an increased risk of PD in patients with AD and vice versa [29]. Familial early-onset PD/AD are due to genetic factors, sometimes a single mutation in a given gene. Both diseases have neuronal loss and abnormal accumulations of specific proteins in common, but in different brain regions [30]. Parkinsonism occurs in approximately 35 to 40% of patients with Alzheimer’s Disease (AD) even with little or no neuronal degeneration in the substantia nigra, which in idiopathic Parkinson’s Disease (PD) results in the severe loss of striatal
dopamine transporter sites. It is not known if there is a loss of striatal dopamine transporter sites in AD with coexistent parkinsonism (AD/parkinsonism), in AD the loss of dopamine transporter sites was restricted to the nucleus accumbens. The loss of these sites in the AD/parkinsonism group was more extensive than in the AD group, with the most severe losses in the rostral caudate and putamen and least in the caudal caudate and putamen. In contrast, no reductions in dopamine transporter sites, tyrosine hydroxylase, and D2 autoreceptors were observed in the substantia nigra and ventral tegmental area of the AD or AD/parkinsonism. Thus, the loss of striatal dopamine transporter sites in AD/parkinsonism may be related to the clinical parkinsonian symptoms [31]. Joyce et al. [32], studied the levels of Tyrosine Hydroxylase (TH) protein, and the expression of TH and Dopamine Transporter (DAT) mRNAs, in midbrain neurons of PD, AD, and AD/Park cases. Compensatory events occur in these DA neurons in AD/Park that are similar to those in PD and that result in differential effects on mRNAs encoding TH and DAT proteins.

**Memory disorders and parkinsonism**
We have found two cases of memory disorders and parkinsonism (5%). Parkinsonism, the clinical term for a disorder with prominent bradykinia and variable associated extrapyramidal signs and symptoms, is accompanied by degeneration of the nigrostriatal dopaminergic system, with neuronal loss and reactive gliosis in the substantia nigra found at autopsy. Parkinsonism is pathologically heterogeneous, with the most common pathologic substrates related to abnormalities in the presynaptic protein α-synuclein or the microtubule binding protein tau In idiopathic Parkinson's Disease (PD), α-synuclein accumulates in neuronal perikarya (Lewy bodies) and neuronal processes (Lewy neurites) [33]. Apparently mild cognitive impairment found in Parkinson disease is related with the memory disorders observed in the patients with Parkinson disease. According to Jia et al. [34], patients with Parkinson disease (PDP-Mild Cognitive Impairment (MCI) exclusively exhibited atrophy in the right entothral cortex .may subserve as a biomarker in early, drug-naive PD-MCI, which shed light on the neural underpinnings of the disease.

**Memory disorders and neurobehavioral disorders**
We have herein reported memory disorders in patients with neurobehavioral disorders (44%), such as depression, anxiety, aggression, and vascular demencia. As above mentioned, aging is characterized by progressive memory decline that can lead to dementia when associated with neurodegeneration [7]. Delusions are partially clinically and neurobiologically linked to memory deficits but not to poor insight. Delusions in Alzheimer Disease (AD) are associated with dysfunction in specific frontal and temporal cortical regions [35]. Fossati et al. [36], postulated the executive memory decline hypothesis in young as well as old depressed patients The memory deficits in depression may be associated with both trait and state factors and raise questions about the long-term cognitive functioning of patients with recurrent affective disorders.

**Memory disorders and language disorders**
We have reported memory disorders and language disorders in 36% of patients with amnestic mild cognitive impairment, Parkinson and Alzheimer diseases. One of the features of Parkinson Disease (PD) is the alteration of voice and speech. The motor deficits associated with PD adversely affect motor control including respiration, phonation, and articulation. The speech deficits related to PD are often called hypokinetic dysarthria and can be characterized by monopitch, monoloudness, reduced stress, imprecise consonants, and inappropriate silences [37]. There is evidence that action programs for speech and language are handled specifically by the prefrontal cortex as supported by regional cerebral blood flow and metabolic rate studies. Word perception, speech, and reading activate both postcentral and in precentral/prefrontal regions of the hemisphere cortices [38].

Speech disorders of Parkinsonism involve larynx, pharynx, tongue and finally lips. The integration of speech production is organized asymmetrically at thalamic level. Experimental or therapeutic lesions in the region of the inferior medial portion of ventro-lateral thalamus may influence the initiation, respiratory control, rate and prosody of speech [39]. White matter thorn-shaped astrocyte clusters have been associated with atypical language presentation of Alzheimer disease [40]. Studies of sentence comprehension deficits in Parkinson’s Disease (PD) patients suggest that language processing involves circuits connecting subcortical and cortical regions [41].

**Memory disorders and neurosensory disorders**
In the present study we have found 28% of patients with neurosensory disorders. Age-related hearing loss is one of the most common health conditions affecting older adults [42]. Results from a number of epidemiological and laboratory studies have demonstrated a significant link between age-related hearing loss and cognitive decline. According to Pu et al. [43], recent evidence shows that hippocampal theta oscillations shared neurophysiological mechanisms between language and memory related with the hippocampus and the perisylvian cortical areas, generally thought to support language processing. Bregman et al. [44], examined awareness of decline in memory and in language in individuals with Alzheimer’s Disease (AD). Their findings reflect better awareness of decline in language than of decline in memory in individuals with AD. Further systematic studies are needed to establish a precise relationship between memory disorders and neurosensory disorders.

**Amnestic mild cognitive impairment**
In the present study we have found 10 patients (28%) with Amnestic mild cognitive impairment, a condition often preceding AD [45], and currently used as a diagnostic criterion for AD [46]. According to Brueggen et al. [47], Functional Magnetic Resonance Imaging (FMRI) at resting state revealed that hippocampus functional connectivity with neocortical brain areas, including regions of the default mode network, is altered in amnestic mild cognitive impairment. Amnestic mild cognitive impairment showed decreased amplitude of low-frequency fluctuations in the bilateral precuneus/posterior cingulate cortices, bilateral frontoinsular cortices, left occipitotemporal cortex, right supramarginal gyrus, and increased amplitude of low-frequency fluctuations at the right lingual gyrus, left middle occipital gyrus, left hippocampus, and left inferior
temporal gyrus [48]. Vinp et al. [49], demonstrated differential functional and structural network changes between Amnestic mild cognitive impairment and AD patients with and without cerebrovascular disease.

Memory disorders and diabetes
We have observed memory disorders and diabetes in 21% of patients examined. People with diabetes have a greater rate of decline in cognitive function and a greater risk of cognitive decline [50]. Previous epidemiologic studies indicate that diabetes mellitus is associated with an increased risk of developing Alzheimer disease in people who do not have dementia [51]. Epidemiological and biological evidences support a link between type 2 diabetes mellitus and Alzheimer’s disease. Cognitive deficits in persons with diabetes mainly affect the areas of psychomotor efficiency, attention, learning and memory, mental flexibility and speed, and executive function. The strong epidemiological association has suggested the existence of a physiopathological link. Hyperglycemia itself is a risk factor for cognitive dysfunction and dementia. Hypoglycemia may also have deleterious effects on cognitive function [52]. Qualitative analyses of the verbal output revealed that older subjects and diabetics produced the greatest number of previously recited words (repetitions). Repetitions may signal a failure to adequately monitor behavior which in turn could contribute to cognitive decline. Repetitions might imply also an associated memory disorder [53].

Memory disturbances and gastrointestinal diseases
In our study we have found memory disturbances in patients with gastrointestinal diseases (21%) of patients examined. Patients exhibited constipation, erosive gastritis, and lack of sphincter controls. Increasing evidence shows changes in gut microbiota composition in association with psychiatric disorders, including anxiety and depression. Moreover, it has been reported that perturbations in gut microbe diversity and richness influence serotonergic, GABAergic, noradrenergic, and dopaminergic neurotransmission. Among these, dopamine is regarded as a main regulator of cognitive functions such as decision making, attention, memory, motivation, and reward [54].

Malnutrition and memory disorders
We have found two cases of loss weight and malnutrition (5%). Kuźma et al. [55], suggest an association between severe vitamin D deficiency and visual memory decline. Malnutrition produces subcortical alterations in vulnerable hippocampal pyramidal cells, and these alterations may provide an explanation for the previously reported deficient performance of malnourished animals in a spatial memory task in which aging and malnutrition were shown to impede the maintenance of long-term memory [56].

Vitamin D deficiency is associated with disruption of neuronal integrity, primarily in frontal regions. Vitamin D deficiency may lead to the loss of neuroprotective properties in cerebral ischemia and vascular lesions, contributing to memory impairment [57]. Rigorous study of Korsakoff Syndrome amnesia (KS) and associated memory disorders of other etiologies provide evidence for distinct mnemonic component processes and neural networks imperative for normal declarative and non-declarative memory abilities and for mnemonic processes spared in KS, from whence emerged the appreciation that memory is not a unitary function [58].

Memory disorders in arthritis
We have herein reported two cases of arthritis and one case of congenital arthrosis (5%) Rheumatoid Arthritis (RA) patients have deficits in memory functioning [59,60]. Patients with RA had a significantly worse outcome in verbal fluency and short memory [61], and immediate and delayed episodic recall [62]. The Rheumatoid disease process, inflammation and demyelination, is associated with cognitive deficits observed with RA [63].

Memory disorders and prostatic diseases
We have found two cases with memory disorders and prostatic hypertrophy (5%). Jarzemski et al. [64], reported delayed memory dysfunction in patients undergoing both surgical and adjunct therapy for radical prostatectomy.

Memory disorders and renal diseases
We have herein reported memory disorders and renal disease just in one patient (2%). Jones et al. [65], studied the nature of impairments of memory in patients with End-Stage Renal Disease (ESRD). They concluded that the type of processing required by the task (conceptual vs. perceptual) is more important than the type of retrieval (explicit vs. implicit) in memory failures in ESRD patients, perhaps because temporal brain regions are more susceptible to the effects of the illness than are posterior region.

Low-grade albuminuria is associated with poor memory performance, especially in the youngest old (60-69 years) and in those with shorter duration of diabetes (< 10 years). Type 2 diabetics with urinary albumin excretion in the upper normal range were also at risk for declining memory performance [66]. Albuminuria predicted worse memory function at 12 years follow-up [67].

Memory disorders and allergic diseases
According to Altveş et al. [68], studies have shown that the lack of microbiota diversity leads to many diseases like memory disorders, depression, stress, autism, and Alzheimer’s disease. The immune system in disease onset and pathogenesis, the role of cytokines, growth factors, and other immune signaling pathways in disease pathogenesis is still being examined. Recent genetic risk and genome-wide association studies and emerging mechanisms for three key immune pathways implicated in disease have shown that the growth factor TGF-β, the complement cascade, and the extracellular receptor TREM2 signaling pathways are important under both healthy and neurodegenerative conditions [69].

Neural correlates of memory disorders
Older adults showed reduced caudate volume relative to younger adults showing the relevance of caudate nucleus for associative memory decline in the aging brain [70]. Frontoparietal white matter, namely the corpus callosum and cingulum, continued to predict executive functions after accounting for global grey matter atrophy [71]. According to Kennedy and Raz [72], multiple regions of interest such as genu and splenium of corpus callosum, internal capsule
We have observed semantic memory and episodic memory disorders (100%) in patients ranging from 40 to 92 years-old, associated to cardiovascular diseases and blood hypertension (82%), sleep disorders (50%), neurobehavioral disorders (44%), such as depression, anxiety, aggression, and vascular demencia, disorders of language (36%), neurosensoric disorders (28%), as diminution of visual and hearing acuity, dizziness (26%), Parkinson disease (34%), Alzheimer disease (21%), gait disturbances (10%), vertigo (10%), cervicalgia and cervicogenic headache (10%) trigeminal neuralgia (2%),). We observed as comorbidities the following non-nervous diseases: metabolic diseases as diabetes (21%) and hypothyroidism (5%), gastrointestinal pathology (21%), such as constipation, loss of spinicter control, and gastritis, arthritis (13%), prostatic hypertrophy (1%) and loss of weight (1%). We consider that according to their high frequency the most risk factors associated to memory disorders are cardiovascular diseases and blood hypertension (82%), sleep disorders (50%), neurobehavioral disorders (44%), such as depression, anxiety, aggression, and vascular demencia, disorders of language (36%), neurosensoric disorders (28%), as diminution of visual and hearing acuity, dizziness (26%), and Parkinson disease (34%).

**Conflict of Interest**

The authors state that they have no conflicts of interest.

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**References**

1. Román GC, Sachdev P, Royall DR, et al. (2004) Vascular cognitive disorder: a new diagnostic category updating vascular cognitive impairment and vascular dementia. J Neurol Sci 226(1-2): pp. 81-87.
2. Cicconetti P, Riole N, Priami C, et al. (2004) Risk factors for cognitive impairment. Recenti Prog Med 95(11): pp. 535-545.
3. Erkinjuntti T (2007) Vascular cognitive deterioration and stroke. Cerebrovasc Dis 24 Suppl 1: pp. 189-194.
4. Chiu PY, Liu CH, Tsai CH (2007) Neuropsychiatric manifestations in vascular cognitive impairment patients with and without dementia. Acta Neurol Taiwan 16(2): pp. 86-91.
5. Jellinger KA (2008) Morphologic diagnosis of "vascular dementia" - a critical update. J Neurol Sci 270(1-2): pp. 1-12.
6. Miley-Akerstedt A, Jelic V, Marklund K, et al. (2018) Lifestyle Factors Are Important Contributors to Subjective Memory Complaints among Patients without Objective Memory Impairment or Positive Neurochemical Biomarkers for Alzheimer’s Disease. Dement Geriatr Cogn Dis Extra 8(3): pp. 439-452.
7. Jawaid A, Woldemichael BT, Kremer EA, et al. (2019) Memory Decline and Its Reversal in Aging and Neurodegeneration Involve miR-183/96/182 Biogenesis. Mol Neurobiol 56(5): pp. 3451-3462.
8. Toledo C, Andrade DC, Díaz HS, et al. (2019) Neurocognitive
Disorders in Heart Failure: Novel Pathophysiological Mechanisms Underpinning Memory Loss and Learning Impairment. Mol Neurobiol 56(12): pp. 8035-8051.

9. Castejón OJ, Carrero Gonzalez CM, Lastre G, et al. (2020) Clinical and neuroimaging study of thirty three patients with vascular dementia in neurological, psychiatric and cardiovascular diseases exhibiting microangiopathy and leukoencephalopathy”. EC Neurology 1(3): pp. 1-13.

10. Gottesman RF, Schneider AL, Albert M, et al. (2014) Midlife hypertension and 20-year cognitive change: the atherosclerosis risk in communities neurocognitive study. JAMA Neurol 71(10): pp. 1218-1227.

11. Antonucci SM, Reilly J (2008) Semantic memory and language processing: a primer. Semin Speech Lang 29(1): pp. 5-17.

12. Chertkow H, Whatmough C, Saumier D, et al. (2008) Cognitive neuroscience studies of semantic memory in Alzheimer's disease. Prog Brain Res169: pp. 393-407.

13. Laisney M, Giffard B, Eustache F (2004) Semantic memory in Alzheimer’s disease: contributions of semantic priming. Psychol Neuropsychiatr Viel 2(2): pp. 107-115.

14. Diekelmann S (2014) Sleep for cognitive enhancement. Front Syst Neurosci 8: p. 46.

15. Andersson C, Marklund K, Walles H, et al. (2019) A lifestyle factors and subjective cognitive impairment in patients seeking help at a memory disorder clinic: the role of negative life events. Dement Geriatr Cogn Disord 48(3-4): pp. 196-206.

16. Gulia KK, Kumar VM (2018) Sleep disorders in the elderly: a growing challenge. Psychogeriatrics 18(3): pp. 155-165.

17. Fortier-Brochu E, Morin CM (2014) Cognitive impairment in individuals with insomnia: clinical significance and correlates. Sleep 37(11): pp. 1787-1798.

18. Brownlow JA, Miller KE, Gehrman PR (2020) Insomnia and cognitive performance. Sleep Med Clin 15(1): pp. 71-76.

19. Momin RR, Ketvertis K (2020) Primary Insomnia. StatPearls Publishing.;

20. Grau-Rivera O, Operto G, Falcón C, et al. (2020) Association between insomnia and cognitive performance, grey matter volume, and white matter microstructure in cognitively unimpaired adults. Alzheimers Res Ther 12(1): p. 4.

21. Feld GB, Born J (2020) Neurochemical mechanisms for memory processing during sleep: basic findings in humans and neuropsychiatric implications. Neuropsychopharmacol 45(1): pp. 31-44.

22. Kirova AM, Bays RB, Lagalwar S (2015) Working memory and executive function decline across normal aging, mild cognitive impairment, and Alzheimer's disease. Biomed Res Int 2015: 748212.

23. Xue C, Yuan B, Yue Y, et al. (2019) Distinct disruptive patterns of default mode subnetwork connectivity across the spectrum of preclinical alzheimer's disease. Front Aging Neurosci 11: p. 307.

24. Rochoy M, Bordet R, Gautier S, et al. (2019) Factors associated with the onset of Alzheimer’s disease: Data mining in the French nationwide discharge summary database between 2008 and 2014. PLoS One 14(7): e0220174.

25. Skoog I, Gustafson D (2006) Update on hypertension and Alzheimer’s disease. Neurol Res 28(6): pp. 605-611.

26. Paglieri C, Bisbocci D, Caserta M, et al. (2008) Hypertension and cognitive function. Clin Exp Hypertens 30(8): pp. 701-710.

27. Bayram E, Caldwell JZK, Banks SJ (2018) Current understanding of magnetic resonance imaging biomarkers and memory in Alzheimer’s disease. Alzheimers Dement (N Y) 4: pp. 395-413.

28. Sperling RA, Dickerson BC, Pihlajamaki M, et al. (2010) Functional alterations in memory networks in early Alzheimer’s disease. Neuromolecular Med 12(1): pp. 27-43.

29. Jellinger K (1987) Neuropathological substrates of Alzheimer’s disease and Parkinson’s disease. J Neural Transm (Suppl.) 24: pp. 109-129.

30. Franco R, Navarro G, Martínez-Pinilla E (2019) Lessons on Differential Neuronal-Death-Vulnerability from Familial Cases of Parkinson’s and Alzheimer’s Diseases. Int J Mol Sci 20(13): E3297.

31. Murray AM, Weihmueller FB, Marshall JF, et al. (1995) Damage to dopamine systems differs between Parkinson’s disease and Alzheimer’s disease with Parkinsonism. Ann Neurol 37(3): pp. 300-312.

32. Joyce JN, Smutzer G, Whitty CJ, et al. (1997) Differential modification of dopamine transporter and tyrosine hydroxylase mRNAs in midbrain of subjects with Parkinson’s, Alzheimer’s with parkinsonism, and Alzheimer’s disease. Mov Disord 12(6): pp. 885-897.

33. Dickson DW (2012) Parkinson’s disease and parkinsonism: neuropathology. Cold Spring Harb Perspect Med 2(8): a009258.

34. Jia X, Wang Z, Yang T, et al. (2019) Entorhinal Cortex Atrophy in Early, Drug-naïve Parkinson’s Disease with Mild Cognitive Impairment. Aging Dis 10(6): pp. 1221-1232.

35. Sultzter DL, Leskin LP, Melrose RJ, et al. (2014) Neurobiology of delusions, memory, and insight in Alzheimer disease. Am J Geriatr Psychiatri 22(11): pp. 1346-1355.

36. Fossati P, Coyette F, Ergis AM, et al. (2002) Influence of age and executive functioning on verbal memory in patients with depression. J Affect Disord 68(2-3): pp. 261-271.

37. Martínez-Sánchez F (2010) Speech and voice disorders in Alzheimer’s disease. Neurol Res 28(6): pp. 605-611.

38. Ingvar DH (1983) Serial aspects of language and speech related to prefrontal cortical activity. A selective review. Hum Neurobiol 2(3): pp. 177-189.

39. Critchley EM (1981) Speech disorders of Parkinsonism: a review. J Neurol Neurosurg Psychiatr 44(9): pp. 751-758.

40. Resende EPF, Nolan AL, Petersen C, et al. (2020) Language and spatial dysfunction in Alzheimer disease with white matter thorn-shaped astrocytes: Astrocytic tau, cognitive function, and Alzheimer disease. Neurol pii: 10.1212/WNL.0000000000008937.

41. Hochstadt J, Nakano H, Lieberman P, et al. (2006) The roles of sequencing and verbal working memory in sentence comprehension deficits in Parkinson’s disease. Brain Lang 97(3): pp. 243-257.

42. Uchida Y, Sugiyama S, Nishita Y, et al. (2019) Age-related
hearing loss and cognitive decline - The potential mechanisms linking the two. uris Nasus Larynx 46(1): pp. 1-9.

43. Pu Y, Cheyne D, Sun Y, et al. (2020) Theta oscillations support the interface between language and memory. Neuroimage 215: pp. 116782.

44. Bregman N, Kavé G, Shiner T, et al. (2019) Alzheimer's Disease Neuroimaging Initiative. Dissociation in awareness of memory and language decline in Alzheimer’s disease. Int J Geriatri Psychiatr 34(4): pp. 548-554.

45. Nasrouei S, Rattel JA, Liedlgruber M, et al. (2019) Fear acquisition and extinction deficits in amnesic mild cognitive impairment and early Alzheimer’s disease. Neurobiol Aging pii: S0197-4580(19)30392-6.

46. Bayram E, Caldwell JZK, Banks SJ (2018) Current understanding of magnetic resonance imaging biomarkers and memory in Alzheimer’s disease. Review. Alzheimers Dement 4: pp. 395-413.

47. Brueggen K, Kasper E, Dyra M, et al. (2016) The Primacy Effect in Amnestic Mild Cognitive Impairment: Associations with Hippocampal Functional Connectivity. Front Aging Neurosci 8: p. 244.

48. Pan P, Zhu L, Yu T, et al. (2017) Aberrant spontaneous low-frequency brain activity in amnestic mild cognitive impairment: A meta-analysis of resting-state fMRI studies. Ageing Res Rev 35: pp. 12-21.

49. Vipin A, Loke YM, Liu S, et al. (2018) Cerebrovascular disease influences functional and structural network connectivity in patients with amnestic mild cognitive impairment and Alzheimer’s disease. Alzheimers Res Ther. 10(1): pp. 82.

50. Cukierman T, Gerstein HC, Williamson JD. (2005) Cognitive decline and dementia in diabetes--systematic overview of prospective observational studies. Diabetologia 48(12): pp. 2460-2469.

51. Sanz C, Andrieu S, Sinclair A, et al. (2009) Diabetes is associated with a slower rate of cognitive decline in Alzheimer disease. NeuroU 73(17): pp. 1359-1366.

52. Barbagallo M, Dominguez LJ (2014) Type 2 diabetes mellitus and Alzheimer’s disease. World J Diabetes 5(6): pp. 889-893.

53. Perlmuter LC, Tun P, Sizer N, et al. (1987) Age and diabetes related changes in verbal fluency. Exp Aging Res 13(1-2): pp. 9-14.

54. González-Arancibia C, Urrutia-Piñones J, Illanes-González J, et al. (2019) Do your gut microbes affect your brain dopamine?. Psychopharmacology (Berl) 236(5): pp. 1611-1622.

55. Kuźma E1, Soni M, Littlejohns TJ, et al. (2016) Vitamin D and Memory Decline: Two Population-Based Prospective Studies. J Alzheimers Dis 50(4): pp. 1099-1108.

56. Castro-Chavira SA, Aguilar-Vázquez AR, Martínez-Chávez Y, et al. (2016) Effects of chronic malnourishment and aging on the ultrastructure of pyramidal cells of the dorsal hippocampus. Nutr Neurosci 19(8): pp. 329-336.

57. Moon Y, Moon WJ, Kwon H, et al. (2015) Vitamin D deficiency disrupts neuronal integrity in cognitively impaired patients. J Alzheimers Dis 45(4): pp. 1089-1096.

58. Fama R, Pitel AL, Sullivan EV (2012) Anterograde episodic memory in Korsakoff syndrome. Neuropsychol Rev 22(2): pp. 93-104.

59. Jorge LL, Gerard C, Revel M (2009) Evidences of memory dysfunction and maladaptive coping in chronic low back pain and Eur J Phys Rehabil Med 45(4): pp. 469-477.

60. Lee JH, Kim GT, Kim YK, et al. (2018) Cognitive function of patients with rheumatoid arthritis is associated with disease activity but not carotid atherosclerotic changes. Clin Exp Rheumatol 36(5): pp. 856-861.

61. Appenzeller S, Bertolo MB, Costallat LT (2004) Cognitive impairment in rheumatoid arthritis. Methods Find Exp Clin Pharmacol 26(5): pp. 339-343.

62. Simos P, Ktistagi G, Dimitraki G, et al. Cognitive deficits early in the course of rheumatoid arthritis. J Clin Exp Neuropsychol 38(7): pp. 820-829.

63. Hamed SA, Selim ZI, Elattar AM, et al. (2012) Assessment of biocorrelates for brain involvement in female patients with rheumatoid arthritis. Clin Rheumatol 31(1): pp. 123-132.

64. Jarzemski P, Brzoszczyk B, Popiolek A, et al. (2019) Cognitive function, depression, and anxiety in patients undergoing radical prostatectomy with and without adjuvant treatment. Neuropsychiatr Dis Treat 15: pp. 819-829.

65. Jones DJ, Harris JP, Vaux E, et al. (2015) The nature of impairments of memory in patients with end-stage renal disease (ESRD). Physiol Behav 147: pp. 324-333.

66. Huang L, Yang L, Wu P, et al. (2017) Low-grade albuminuria is associated with poor memory performance in the nondemented Chinese elderly with type 2 diabetes. Metab Brain Dis 32(6): pp. 1975-1981.

67. Sacre JW, Magliano DJ, Zimmet PZ, et al. (2019) Associations of Chronic Kidney Disease Markers with Cognitive Function: A 12-Year Follow-Up Study. J Alzheimers Dis 70(1): S19-S30.

68. Altveq S, Yildiz HK, Vural HC (2020) Interaction of the microbiota with the human body in health and diseases. Biosci Microbiota Food Health 39(2): pp. 23-32.

69. Hammond TR, Marsh SE, Stevens B (2019) Immune signaling in neurodegeneration. Immunity 50(4): pp. 955-974.

70. Bauer E, Toepfer M, Gebhardt H, et al. (2015) The significance of caudate volume for age-related associative memory decline. Brain Res 1622: pp. 137-148.

71. Bettcher BM, Mungas D, Patel N, et al. (2016) Neuroanatomical substrates of executive functions: Beyond prefrontal structures. Neuropsychologia 85: pp. 100-109.

72. Kennedy KM, Raz N (2009) Aging white matter and cognition: differential effects of regional variations in diffusion properties on memory, executive functions, and speed. Neuropsychologia 200947(3): pp. 916-927.

73. Gunning-Dixon FM, Brickman AM, Cheng JC, et al. (2009) Aging of cerebral white matter: a review of MRI findings. Int J Geriatri Psychiatr 24(2): pp. 109-117.

74. Zheng F, Cui D, Zhang L, et al. (2018) The volume of hippocampal subfields in relation to decline of memory recall across the adult lifespan. Front Aging Neurosci 10: p. 320.

75. Wang N, Zhang L, Yang H, et al. (2019) Do multiple system atrophy and Parkinson's disease show distinct patterns of
volumetric alterations across hippocampal subfields? An exploratory study. Eur Radiol 29(9): pp. 4948-4956.
76. Hu Y, Du W, Zhang Y, et al. (2019) Loss of Parietal Memory Network Integrity in Alzheimer’s Disease. Front Aging Neurosci 11: p. 67.
77. Mayes AR (1986) Learning and memory disorders and their assessment. Neuropsychologia 24(1): pp. 25-39.
78. Mattson MP, Pedersen WA, Duan W, et al. (1999) Cellular and molecular mechanisms underlying perturbed energy metabolism and neuronal degeneration in Alzheimer’s and Parkinson’s diseases. Ann N Y Acad Sci 893: pp. 154-175.
79. Patel R, Brophy C, Hickling M, et al. (2019) Alternative cleavage and polyadenylation of genes associated with protein turnover and mitochondrial function are deregulated in Parkinson’s, Alzheimer’s and ALS disease. BMC Med Genomics 12(1): p. 60.