

“Clinical Study of Memory Disorders in Aging Patients with Associated Cardiovascular, Neurological, Neurobehavioral and Metabolic Diseases”. A Review

Castejón OJ¹, Carrero-González Carmen Maria², Judith Cristina Martínez Royert², Galindez P³, Salones de Castejón M³, Sierra-Carrero Leandro Luis⁴ and Silvia Juliana Prada Soto⁵

¹Instituto de Investigaciones Biológicas “Drs. Orlando Castejón and Haydee Viloría de Castejón

²Facultad de Ciencias de la Salud. Universidad Simón Bolívar. Barranquilla, Colombia

³Instituto de Neurociencias Clínicas, Fundación Castejón, Hogar Clínica San Rafael. Maracaibo, Venezuela

⁴Department of Medicine, Health Sciences Division, Universidad del Norte and Hospital Universidad del Norte, Barranquilla, Colombia

⁵Fundacion Universitaria San Martin, Colombia

ABSTRACT

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%), neurosensory disorders (28%), as diminution of visual and hearing acuity, dizziness (26%), Parkinson disease (34%), Alzheimer disease (21%), gait disturbances (10%), vertigo (10%), cervicgia 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 sphincter 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%), neurosensory disorders (28%), as diminution of visual and hearing acuity, dizziness (26%), and Parkinson disease (34%).

*Corresponding author

Carmen Carrero González, Facultad de Ciencias de la Salud. Universidad Simón Bolívar. Barranquilla, Colombia, E-mail: carmen.carrero@unisimonbolivar.edu.co

Received: February 28, 2021; **Accepted:** March 09, 2021; **Published:** March 18, 2021

Keywords: Memory, 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 cognitive impairment (MCI) to vascular dementia [1]. According to Cicconetti et al., age, sex, family history and educational level, and risk factors, such as hypertension, hypercholesterolemia, diabetes mellitus and tobacco, might contribute to degenerative forms of cognitive impairment [2]. Vascular cognitive impairment (VCI) incorporates the complex interactions between vascular aetiologies, risk factors and cellular changes within the brain and cognition [3]. Neuropsychiatric symptoms were common in patients with vascular cognitive impairment (VCI) 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 leukoencephalopathy, multilacunar state, 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 elder 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 leukoencephalopathy 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.

Results

Case Report study

Case 1: ZC, 72 years-old, F. Disorders of memory in 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 and sleep, 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 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, osteotendinous areflexia. 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 hypoacusia. Diagnosis: Amnestic mild cognitive impairment. Cervicalgia. Insomnia. Hypoacusia. Loss of sphincter control.

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

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

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

Diagnosis: 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, 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, holocraneal 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 cerebro-vascular 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,

Case 14: JP, 75 years-old, F. Loss of semantic memory, dizziness, vertigo, cephalic fronto-temporo-occipital, 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, holocraneal cephalic, 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 dyspnoea, heart failure, blood hypertension, dyslipidaemia, and venous insufficiency.

Diagnosis: Alzheimer disease. Heart failure Blood hypertension, dyslipidaemia, and venous insufficiency

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

Diagnosis: Amnestic mild cognitive impairment. Heart 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. Heart failure,

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

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

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

Diagnosis: Loss of semantic memory. Seizures (partial epilepsy). 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: Mixed Alzheimer disease. Small vessel disease Blood hypertension, Sleep disorders.

Case 23: JP, 65 years-old, F. Episodic memory disorders, fronto-parieto-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, agoraphobia, 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, 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, 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. Loss of memory and Parkinson disease.

Diagnosis: Amnestic mild cognitive impairment, 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 wheel chair 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, severe dizziness, neck pain, language disturbances, sleep disorder, calcifications of aortic arch artery, constipation, bradycardia. TAC showed severe cortical atrophy according to age, and cortical calcifications, periventricular hipodensity suggestive of leukoaraiosis leukoencephalopathy, granulomatous in nature. Sinusal arrhythmia, and constipation.

Diagnosis: Amnestic Mild cognitive impairment. 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 leukoencephalopathy, 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 leukoencephalopathy.

Diagnosis: Memory disturbances. High blood pressure. Depression

Case 33: CI, 78 years old, F. Disorders of semantic and working memory, tremor in both hands, gait disorders, speech difficulties, decrease hearing acuity, tinnitus, weight loss, and sleep disorders, **Diagnosis:** Alzheimer disease and parkinsonism. Sleep disorders. Neurosensory disorders

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

Diagnosis: Semantic and working memory disorders. Parkinson disease. Arthritis. Depression, Anxiety, Constipation

Disorder of semantic and working memory and Parkinson disease, Depression. 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 hearth failure.

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

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

Interpretation of Results

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%), 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 observed 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%).

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%), neurosensory disorders (28%), as diminution of visual and hearing acuity, dizziness (26%), We found eleven patients with diagnosis of Amnestic mild cognitive impairment, ten cases with Parkinson diseases, nine cases with Alzheimer disease, five cases with diabetes, three cases with arthritis, and one posttraumatic and semantic loss of memory,

We have found the following clinical mixed subtypes: five patients with a mixed Alzheimer and Parkinson disease syndrome, two cases with Alzheimer disease and diabetes, and one case with Parkinson disease and vascular demencia, The following NMR images were found: subcortical and supratentorial leukoencephalopathy, and cervical and lumbar spine pathology.

Discussion

Amnestic mild cognitive impairment

In the present study we have found 11 patients (28%) with Amnestic mild cognitive impairment, a condition often preceding AD, and currently used as a diagnostic criterion for AD [10,11].

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 [12]. Amnestic mild cognitive impairment showed decreased amplitude of low-frequency fluctuations in the bilateral precuneus/posterior cingulate cortices, bilateral frontoinsular cortices, left occipitotemporal cortex, and right supramarginal gyrus, and

increased in the right lingual gyrus, left middle occipital gyrus, left hippocampus, and left inferior temporal gyrus [13]. Vipin et al. demonstrated differential functional and structural network changes between Amnesic mild cognitive impairment and AD patients with and without cerebrovascular disease [14].

Memory disorders and cardiovascular diseases

In the present study we have reported memory disorders in patients with cardiovascular diseases and blood hypertension (82%) of patients examined. Toledo et al., postulate related the memory decline to cerebral ischemia, neuroinflammation, oxidative stress, and mitochondrial and DNA alterations [8]. Gottesman et al. postulated that hypertension is a potential cause of cognitive decline and dementia, and its greatest influence on cognition may occur in middle age [15].

Memory disorders and sleep disorders

We have observed memory disorder and sleep disorders (50%) in the 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 [16]. According to Andersson et al., stress, sleep, sensory sensitivity, depression, and negative life events are observed in patients presenting memory disorders [17]. 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 [18]. Fortier-Brochu and Morin found clinically significant alterations in attention and episodic memory in individuals with insomnia [19]. Insomnia is characterized by difficulty initiating and maintaining sleep, along with dissatisfaction with sleep quality or quantity [20, 21]. 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 [22].

Feld and Born analyzed the excitatory neurotransmitter glutamate that plays a prominent role in inducing synaptic consolidation, the inhibitory GABAergic system, 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 [23].

Memory disorders and neurobehavioral disorders

We have herein reported memory disorders in patients with neurobehavioral disorders (44%), such as depression, anxiety, aggression, and vascular dementia. 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 [24].

Executive function refers to cognitive processes that control and integrate other cognitive activities such as episodic memory [25].

Semantic memory and working 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 [26]. Semantic memory is the component of long-term memory that stores our concepts about the world. 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 [27]. This disruption was observed in the present study due to 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, heart failure and blood hypertension, and depression, anxiety and sleep disorder. According to Laisney et al., 2004 [28], the progressive deterioration begins at the level of the concept attributes, and further involves the concepts themselves, and implicates the left posterior temporal region in semantic processing for pictures, abstract words, and concrete words [28, 33].

Language disorders in Parkinson disease

We have here in reported memory disorders and language disorders in 36% of patients studied. 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, mono-loudness, reduced stress, imprecise consonants, and inappropriate silences [29].

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, reading activate both in postcentral and in precentral/prefrontal regions of the hemisphere cortices [30].

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 [31]. White matter thorn-shaped astrocyte clusters have been associated with atypical language presentation of Alzheimer disease [32]. Studies of sentence comprehension deficits in Parkinson's disease (PD) patients suggest that language processing involves circuits connecting subcortical and cortical regions [33].

Memory disorders and Parkinsonism disorders

We have found two cases with Parkinsonism (5%). Parkinsonism, the clinical term for a disorder with prominent bradykinesia 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) [34].

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 [35]. Previous epidemiologic studies indicate that diabetes mellitus is associated an increased risk of developing Alzheimer disease in people who do not have dementia [36]. 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 [37]. 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 [38]. Repetitions might imply also an associated memory disorder.

Memory Disorders and Alzheimer Diseases

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 [39] (Kirova et al., 2015). The early progression continuum of Alzheimer's disease has been considered to advance through subjective cognitive decline, non-amnesic mild cognitive impairment, and amnesic mild cognitive impairment [40].

High blood pressure and Alzheimer disease was observed in six cases in the present study Rochoy et al. highlighted a possible association of AD with intracranial hypertension, which has also been related to pathological manifestations of Alzheimer's disease (senile plaques, neurofibrillary tangles, hippocampal atrophy) [40]. 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 [41, 42].

Neuroscience correlate of Alzheimer disease

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 [11] (Bayram et al., 2018). The medial temporal lobe (MTL) 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 [43].

Memory disorders and neurosensory disorders

Age-related hearing loss (ARHL) is one of the most common health conditions affecting older adults [44]. Results from a number of epidemiological and laboratory studies have demonstrated a significant link between ARHL and cognitive decline. Further systematic studies are needed to establish a precise relationship between memory disorders and neurosensory disorders.

Memory 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 [45].

Memory disorders and renal diseases

Jones et al., studied the nature of impairments of memory in patients with end-stage renal disease (ESRD) [46]. 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 regions

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 [47]. Albuminuria predicted worse memory function at 12 years follow-up [48].

Malnutrition and memory disorders

We have found two cases of loss weight and malnutrition (1%). Kuźma et al. suggest an association between severe vitamin D deficiency and visual memory decline [49]. Malnutrition produces subcellular 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 [50]. 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 [51]. Rigorous study of Korsakoff Syndrome amnesia and associated memory disorders of other etiologies provide evidence for distinct mnemonic component processes and neural networks imperative for normal declarative and nondeclarative memory abilities and for mnemonic processes spared in KS, from whence emerged the appreciation that memory is not a unitary function [52].

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 [53]. Fronto-parietal white matter, namely the corpus callosum and cingulum, continued to predict executive functions after accounting for global grey matter atrophy [54].

According to Kennedy and Raz, multiple regions of interest such as genu and splenium of corpus callosum, internal capsule limbs, prefrontal, temporal, superior/posterior parietal, occipital white matter are related to processing speed, working memory, inhibition, task switching, and episodic memory [55].

Studies that combine MRI with cognitive measures suggest that such age-related reductions in white matter integrity may produce a disconnection state that underlies some of the age-related performance declines in age-sensitive cognitive domains associated with episodic memory, executive functions, and information processing speed [56].

The hippocampus is an important limbic structure closely related to memory function. Volume changes in this region might be considered as a biomarker for dementia disorders. Additionally, several hippocampal subfield volumes were significantly associated with memory scores, further highlighting the key role of the hippocampus in age-related memory decline, several hippocampal subfield volumes were significantly associated with memory scores, further highlighting the key role of the hippocampus in age-related memory decline. These regions could be used to assess the risk of memory decline across the adult lifespan [57].

The multiple system atrophy (MSA)-mild cognitive impairment (MCI) patients showed more widespread impairment of hippocampal subfields compared with the Parkinson disease (PD)-mild cognitive impairment (MCI) group, involving trisynaptic loop and amygdala-hippocampus interactions [58].

Patients with Parkinson disease (PDP)-mild cognitive impairment (MCI) exclusively exhibited atrophy in the right entorhinal cortex (ENT). The right entorhinal cortex (ENT) may subserve as a biomarker in early, drug-naive PD-MCI, which shed light on the neural underpinnings of the disease [59].

A functional brain network, termed the parietal memory network (PMN), has been shown to reflect the familiarity of stimuli in both memory encoding and retrieval. The integrity as an intrinsic connectivity network for the PMN was significantly decreased in AD [60].

Cortical lesions cause disturbances in short-term memory. Other cortical lesions disturb the retrieval of previously well-established semantic and episodic memories. Frontal cortex dysfunction seems to be related to a memory syndrome caused by a breakdown in the ability to plan and carry out elaborative processing, and the amnesic syndrome(s), caused by limbic system or diencephalic lesions [61].

Cellular and molecular correlates of memory disorders

Studies of postmortem brain tissue from AD and PD patients have provided evidence for increased levels of oxidative stress, mitochondrial dysfunction and impaired glucose uptake in vulnerable neuronal populations. Studies of animal and cell culture models of AD and PD suggest that increased levels of oxidative stress (membrane lipid peroxidation, in particular) may disrupt neuronal energy metabolism and ion homeostasis, by impairing the function of membrane ion-motive ATPases and glucose and glutamate transporters. Such oxidative and metabolic compromise may thereby render neurons vulnerable to excitotoxicity and apoptosis [62].

Transcriptome wide changes have been assessed extensively during the progression of neurodegenerative diseases Alternative polyadenylation (APA) occurs in over 70% of human protein coding genes and it has recently been recognised as a critical regulator of gene expression during disease. Deregulation of APA may play a significant role in neurodegeneration by altering the expression of genes including UBR1 and OGDHL in AD, LONP1 in PD and UCHL1 in ALS. [63].

Mixed clinical syndromes of Parkinson and Alzheimer disease subtypes

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 [64]. 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 [65]. 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), 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 [66].

Joyce et al (1997) [67] 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. The loss of striatal dopamine transporter sites in AD/parkinsonism may be related to the clinical parkinsonian symptoms. 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 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 [68, 69] (Jorge et al., 2009, Lee et al., 2018). Patients with RA had a significantly worse outcome in verbal fluency ($p < 0.05$), logic memory ($p < 0.05$) and short memory ($p < 0.05$) [70]. Short-term memory, immediate and delayed episodic recall [71]. The Reumathoid disease process, inflammation and demyelination, is associated with cognitive deficits observed with RA [72].

Memory disorders and allergic diseases

We have not found previous reports on memory disorders in patients with allergic diseases.

Memory disorders and prostatic diseases

We have found two cases with memory disorders and prostatic hypertrophy. Jarzowski et al., 2019 [73] reported delayed memory dysfunction in patients undergoing both surgical and adjunct therapy for radical prostatectomy [73].

Conclusions

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 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 observed 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%). 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%), neurosensory disorders (28%), as diminution of visual and hearing acuity, dizziness (26%), and Parkinson disease (34%), A detailed discussion on the relationship of memory disorder and the related associated pathological entities is analyzed.

Conflict of interests: the authors declare that they have no conflicts of interest in the preparation of the manuscript.

References

1. Román GC, Sachdev P, Royall DR, Bullock RA, Orgogozo JM, et al. (2004) Vascular cognitive disorder: a new diagnostic category updating vascular cognitive impairment and vascular dementia. *J Neurol Sci* 226: 81-87.
2. Cicconetti P, Riolo N, Priami C, Tafaro L, Ettore E (2004) Risk factors for cognitive impairment. *Recenti Prog Med* 95: 535-545.
3. Erkinjuntti T (2007) Vascular cognitive deterioration and stroke. *Cerebrovasc Dis* 1: 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: 86-91.
5. Jellinger KA (2008) Morphologic diagnosis of "vascular dementia" - a critical update. *J Neurol Sci* 270: 1-12.
6. Miley-Akerstedt A, Jelic V, Marklund K, Walles H, Åkerstedt T, 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: 439-452.
7. Jawaid A, Woldemichael BT, Kremer EA, Laferriere F, Gaur N, et al. (2019) Memory Decline and Its Reversal in Aging and Neurodegeneration Involve miR-183/96/182 Biogenesis. *Mol Neurobiol* 56: 3451-3462.
8. Toledo C, Andrade DC, Díaz HS, Inestrosa NC, Del Rio R (2019) Neurocognitive Disorders in Heart Failure: Novel Pathophysiological Mechanisms Underpinning Memory Loss and Learning Impairment. *Mol Neurobiol* 56: 8035-8051.
9. Castejón OJ, Carrero González CM, Lastre-Amell G, Leal J, Galindez P, 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* 3: 01-13.
10. Nasrouei S, Rattel JA, Liedlgruber M, Marksteiner J, Wilhelm FH (2019) Fear acquisition and extinction deficits in amnesic mild cognitive impairment and early Alzheimer's disease. *Neurobiol Aging* S0197-4580 30392-6.
11. 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: 395-413.
12. Brueggen K, Kasper E, Dyrba M, Bruno D, Pomara N, Ewers M, et al. (2016) The Primacy Effect in Amnesic Mild Cognitive Impairment: Associations with Hippocampal Functional Connectivity. *Front Aging Neurosci* 8: 244.
13. Pan P, Zhu L, Yu T, Shi H, Zhang B, et al. (2019) Aberrant spontaneous low-frequency brain activity in amnesic mild cognitive impairment: A meta-analysis of resting-state fMRI studies. *Ageing Res Rev* 35:12-21.
14. Vipin A, Loke YM, Liu S, Hilal S, Shim HY, et al. (2018) Venketasubramanian N, Chen CL2, Zhou J. Cerebrovascular disease influences functional and structural network connectivity in patients with amnesic mild cognitive impairment and Alzheimer's disease. *Alzheimers Res Ther* 10: 82.
15. Gottesman RF, Schneider AL, Albert M, Alonso A, Bandeen-Roche K, et al. (2014) Midlife hypertension and 20-year cognitive change: the atherosclerosis risk in communities neurocognitive study. *JAMA Neurol* 71: 1218-1227.
16. Diekelmann S (2014) Sleep for cognitive enhancement. *Front Syst Neurosci* 8: 46.
17. Andersson C, Marklund K, Walles H, Hagman G, Miley-Akerstedt (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: 196-206.
18. Gulia KK, Kumar VM (2018) Sleep disorders in the elderly: a growing challenge. *Psychogeriatrics* 18: 155-165.
19. Fortier-Brochu E, Morin CM (2014) Cognitive impairment in individuals with insomnia: clinical significance and correlates. *Sleep* 37: 1787-1798.
20. Brownlow JA, Miller KE, Gehrman PR (2020) Insomnia and cognitive performance. *Sleep Med Clin* 15: 71-76.
21. Momin RR, Ketvertis K. Primary Insomnia. StatPearls Publishing; 2020.
22. Grau-Rivera O., Operto G, Falcón C, Sánchez-Benavides G, Cacciaglia R, et al. (2020) Association between insomnia and cognitive performance, gray matter volume, and white matter microstructure in cognitively unimpaired adults. *Alzheimers Res Ther* 12:4.
23. Feld GB, Born J (2020) Neurochemical mechanisms for memory processing during sleep: basic findings in humans and neuropsychiatric implications. *Neuropsychopharmacology* 45: 31-44.
24. Sultzer DL, Leskin LP, Melrose RJ, Harwood DG, Narvaez TA, et al. (2014) Neurobiology of delusions, memory, and insight in Alzheimer disease. *Am J Geriatr Psychiatry* 22: 1346-1355.
25. Fossati P, Coyette F, Ergis AM, Allilaire JF (2002) Influence of age and executive functioning on verbal memory in patients with depression. *J Affect Disord* 68: 261-271.
26. Antonucci SM, Reilly J (2008) Semantic memory and language processing: a primer. *Semin Speech Lang* 29: 5-17.
27. Chertkow H, Whatmough C, Saumier D, Duong A (2008) Cognitive neuroscience studies of semantic memory in Alzheimer's disease. *Prog Brain Res* 169: 393-407.
28. Laisney M, Giffard B, Eustache F (2004) Semantic memory in Alzheimer's disease: contributions of semantic priming. *Psychol Neuropsychiatr Vieil* 2: 107-115.
29. Martinez-Sánchez F (2010) Speech and voice disorders in Parkinson's disease. *Rev Neurol* 51: 542-550.
30. Ingvar DH (1983) Serial aspects of language and speech related to prefrontal cortical activity. A selective review. *Hum Neurobiol* 2:177-189.
31. Critchley EM (1981) Speech disorders of Parkinsonism: a review. *J Neurol Neurosurg Psychiatry* 44: 751-758.
32. Resende EPF, Nolan AL, Petersen C, Ehrenberg AJ, Spina S, et al. (2020) Language and spatial dysfunction in Alzheimer disease with white matter thorn-shaped astrocytes: Astrocytic tau, cognitive function, and Alzheimer disease. *Neurology*.2020pii:10.1212/WNL.0000000000008937.
33. Hochstadt J, Nakano H, Lieberman P, Friedman J (2006) The roles of sequencing and verbal working memory in sentence comprehension deficits in Parkinson's disease. *Brain Lang* 97: 243-257.

34. Dickson DW (2012) Parkinson's disease and Parkinsonism: neuropathology. *Cold Spring Harb Perspect Med* 2). pii: a009258.
35. Cukierman T, Gerstein HC, Williamson JD (2005) Cognitive decline and dementia in diabetes--systematic overview of prospective observational studies. *Diabetologia* 48: 2460-2469.
36. Sanz C, Andrieu S, Sinclair A, Hanaire H, Vellas B, et al. (2009) Diabetes is associated with a slower rate of cognitive decline in Alzheimer disease. *Neurology* 73: 1359-1366.
37. Barbagallo M, Dominguez LJ (2014) Type 2 diabetes mellitus and Alzheimer's disease. *World J Diabetes* 5: 889-893.
38. Perlmutter LC, Tun P, Sizer N, McGlinchey RE, Nathan DM (1987) Age and diabetes related changes in verbal fluency. *Exp Aging Res*. 1987 Spring-Summer; 13: 9-14.
39. 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.
40. Rochoy M, Bordet R, Gautier S, Chazard E (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: e0220174.
41. Skoog I, Gustafson D (2006) Update on hypertension and Alzheimer's disease. *Neurol Res* 28: 605-611.
42. Paglieri C, Bisbocci D, Caserta M, Rabbia F, Bertello C, et al. (2008) Hypertension and cognitive function. *Clin Exp Hypertens* 30: 701-710.
43. Sperling RA, Dickerson BC, Pihlajamaki M, Vannini P, LaViolette PS, et al. (2010) Functional alterations in memory networks in early Alzheimer's disease. *Neuromolecular Med* 12: 27-43.
44. Uchida Y, Sugiura S, Nishita Y, Saji N, Sone M, et al. (2019) Age-related hearing loss and cognitive decline - The potential mechanisms linking the two. *uris Nasus Larynx* 46: 1-9.
45. González-Arancibia C, Urrutia-Piñones J, Illanes-González J, Martínez-Pinto J, Sotomayor-Zárate R, et al. (2019) Do your gut microbes affect your brain dopamine?. *Psychopharmacology (Berl)* 236: 1611-1622.
46. Jones DJ, Harris JP, Vaux E, Hadid R, Kean R, et al. (2015) The nature of impairments of memory in patients with end-stage renal disease (ESRD). *Physiol Behav* 147: 324-333.
47. Huang L, Yang L, Wu P, Yan X, Luo L, 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: 1975-1981.
48. Sacre JW, Magliano DJ, Zimmet PZ, Polkinghorne KR, Chadban SJ, et al. (2019) Anstey KJ, Shaw JE. Associations of Chronic Kidney Disease Markers with Cognitive Function: A 12-Year Follow-Up Study. *J Alzheimers Dis* 70: S19-S30.
49. Kuźma E, Soni M, Littlejohns TJ, Ranson JM, van Schoor NM, et al. (2016) Vitamin D and Memory Decline: Two Population-Based Prospective Studies. *J Alzheimers Dis* 50: 1099-1108.
50. Castro-Chavira SA, Aguilar-Vázquez AR, Martínez-Chávez Y, Palma L, Padilla-Gómez E, et al. (2016) Effects of chronic malnourishment and aging on the ultrastructure of pyramidal cells of the dorsal hippocampus. *Nutr Neurosci* 19: 329-336.
51. Moon Y, Moon WJ, Kwon H, Lee JM, Han SH (2015) Vitamin D deficiency disrupts neuronal integrity in cognitively impaired patients. *J Alzheimers Dis* 45: 1089-1096.
52. Fama R, Pitel AL, Sullivan EV (2012) Anterograde episodic memory in Korsakoff syndrome. *Neuropsychol Rev* 22: 93-104.
53. Bauer E, Toepper M, Gebhardt H, Gallhofer B, Sammer G (2020) The significance of caudate volume for age-related associative memory decline. *Brain Res* 1622: 137-148.
54. Bettcher BM, Mungas D, Patel N, Eloffson J, Dutt S, et al. (2016) Neuroanatomical substrates of executive functions: Beyond prefrontal structures. *Neuropsychologia* 85:100-109.
55. Kennedy KM, Raz N (2000) Aging white matter and cognition: differential effects of regional variations in diffusion properties on memory, executive functions, and speed. *Neuropsychologia* 947: 916-927.
56. Gunning-Dixon FM, Brickman AM, Cheng JC, Alexopoulos GS (2009) Aging of cerebral white matter: a review of MRI findings. *Int J Geriatr Psychiatry* 24: 109-117.
57. Zheng F, Cui D, Zhang L, Zhang S, Zhao Y, et al. (2018) The volume of hippocampal subfields in relation to decline of memory recall across the adult lifespan. *Front Aging Neurosci* 10: 320.
58. Wang N, Zhang L, Yang H, Luo X, Fan G (2019) Do multiple system atrophy and Parkinson's disease show distinct patterns of volumetric alterations across hippocampal subfields? An exploratory study. *Eur Radiol* 29: 4948-4956.
59. Jia X, Wang Z, Yang T, Li Y, Gao S, et al. (2019) Entorhinal Cortex Atrophy in Early, Drug-naive Parkinson's disease with Mild Cognitive Impairment. *Aging Dis* 10: 1221-1232.
60. Hu Y, Du W, Zhang Y, Li N, Han Y, et al. (2019) Loss of Parietal Memory Network Integrity in Alzheimer's disease. *Front Aging Neurosci* 11: 67.
61. Mayes AR (1986) Learning and memory disorders and their assessment. *Neuropsychologia* 24: 25-39.
62. Mattson MP, Pedersen WA, Duan W, Culmsee C, Camandola S (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: 154-175.
63. Patel R, Brophy C, Hickling M, Neve J, Furger A (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: 60.
64. Jellinger K (1987) Neuropathological substrates of Alzheimer's disease and Parkinson's disease 24: 109-129.
65. 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: E3297.
66. Murray AM, Weihmueller FB, Marshall JF, Hurtig HI, Gottleib GL, et al. (1995) Damage to dopamine systems differs between Parkinson's disease and Alzheimer's disease with Parkinsonism. *Ann Neurol* 37: 300-312.
67. Joyce JN, Smutzer G, Whitty CJ, Myers A, Bannon MJ (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: 885-897.
68. 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: 469-477.
69. Lee JH, Kim GT, Kim YK, Lee SG (2018) Cognitive function of patients with rheumatoid arthritis is associated with disease activity but not carotid atherosclerotic changes. *Clin Exp Rheumatol* 36: 856-861.
70. Appenzeller S, Bertolo MB, Costallat LT (2004) Cognitive impairment in rheumatoid arthritis. *Methods Find Exp Clin Pharmacol* 26: 339-343.
71. Simos P, Ktistaki G, Dimitraki G, Papastefanakis E, Kougkas N, et al. (2016) Cognitive deficits early in the course of

- rheumatoid arthritis. J Clin Exp Neuropsychol 38: 820-829.
72. Hamed SA, Selim ZI, Elattar AM, Elserogy YM, Ahmed EA, et al. (2012) Assessment of biocorrelates for brain involvement in female patients with rheumatoid arthritis. Clin Rheumatol 31: 123-132.
73. Jarzowski P, Brzoszczyk B, Popiołek A, Stachowicz-Karpińska A, Gołota S, et al. (2019) Cognitive function, depression, and anxiety in patients undergoing radical prostatectomy with and without adjuvant treatment. Neuropsychiatr Dis Treat 15: 819-829.

Copyright: ©2021 Carmen Carrero González, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.