Neuropsychological Assessment in Stroke Patients

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Summary
Cerebral Vascular Accident (CVA) is the disease that kills the most in Brazil and causes the most disability in the world and occurs when the vessels that supply blood to the brain clog or rupture, thus causing paralysis of the affected area. Because this accident happens in the brain, many patients are left with consequences for the rest of their lives, so neuropsychological assessment was created in order to determine how much these injuries are harming the patient. The general objective of this study was to discuss neuropsychological assessment in stroke patients. The research method used was literature review, where reliable databases were searched for studies already published on the subject with the aim of compiling information and discussing among the authors. Therefore, it is concluded that more studies must be carried out to determine exactly what the injuries are, their degrees and thus be able to give the patient a better quality of life.
Key words: Stroke. Neuropsychological Assessment; Cognitive Deficit.

Abstract
Cerebral Vascular Accident (CVA) is the disease that kills the most in Brazil and the one that causes the most disability in the world. It happens when the vessels that carry blood to the brain clog or rupture, thus causing paralysis in the affected area. Because this accident happens in the brain, many patients are left with sequelae for the rest of their lives, so the neuropsychological assessment was created in order to determine how much these injuries are harming the patient. The general objective of this study was to discuss the neuropsychological assessment in stroke patients. The research method used was the literature review, which searched in reliable databases for studies already published on the subject in order to compile information and discuss among authors. Thus, it is concluded that more studies should be carried out to accurately determine which injuries, their degrees, and thus be able to provide the patient with a better quality of life.
Keywords:CVA. Neuropsychological Assessment. Cognitive Impairment.

1. Introduction

Stroke is the second leading cause of death and a common cause of physical and cognitive disability in patients in developed countries (LOPEZ*et. al*., 2006; DONNAN*et. al* ., 2008). The importance of stroke treatment after discharge is increasingly

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recognized for being able to reduce the burden of stroke and ensure independence. Doctors are challenged to assess patients in need of care, particularly when considering that the population over 65 years of age is the fastest growing group in Western countries (KINSELLA, 2001), and age is an important risk factor for stroke. and consequent dementia (PENDLEBURY; ROTHWELL, 2009).

Cognitive impairment that emerges after stroke is an increasingly recognized factor in long-term disability. The prevalence of cognitive impairment varies between studies depending on assessment methods, definitions, or sample characteristics. Often, screening instruments such as the Mini-Mental State Examination (MMSE) (FOLSTEIN; FOLSTEIN; MCHUGH, 1975) or the Montreal Cognitive Assessment (MoCa) (NASREDDINE*et. al.*, 2005) are applied, although they seem inadequate to identify subtle or specific cognitive deficits (NYS*et. al.*, 2005). Neuropsychological assessment reveals that deficits in executive functioning, attention, mental processing speed, visual perception and construction capacity are common illnesses in subacute and chronic patients (LESNIAK*et. al.*, 2008; NYS*et. al.*, 2005a; STEPHENS*et. al.*, 2004; HOCHSTENBACH; DEN OTTER; MULDER, 2003; SACHDEV*et. al.*, 2009).

The risk of continued cognitive decline and dementia after stroke is often stated. However, several authors have emphasized the multiple evolutionary trends in cognitive changes in chronic stroke patients (DEL SER*et. al.*, 2005; BALLARD*et. al.*, 2003). They found an annual conversion to dementia of 8–13% in the first year after stroke. Furthermore, results indicated that a large proportion of patients (50%) demonstrated improvement in cognitive functioning (BALLARD*et. al.*, 2003) or stable loss (78%) (DEL SER*et. al.*, 2005). Two recent longitudinal studies have demonstrated that stroke increases the risk of persistent cognitive decline, particularly in executive functioning (BARKER-COLLO*et. al.*, 2010; LEVINE*et. al.*, 2015). One time-varying factor that may influence cognitive performance over time is the level of depressive symptoms.

Post-stroke depression (PSD) is reported to occur with prevalence rates between 5 and 64%, with this variation depending on the study population, timing of post-stroke assessment, and assessment instruments (HOSKING; MARSH; FRIEDMAN, 2000;*et. al.*, 2013). PSD is known to be related to mortality, reduced functional outcome and quality of life (PAN*et. al.*, 2011; CHEMERINSKI; ROBINSON;

KOSIER, 2001; STURM*et. al.*, 2004), and may be linked to several psychological factors and mechanisms. Regarding demographic variables and their association with depression in stroke patients, there appears to be no clear pattern. Most studies seem to reveal that advanced age is not associated with depression in stroke, although not all can support these findings (CHEMERINSKI; ROBINSON; KOSIER, 2001; BARKER-COLLO, 2007). Furthermore, and in some contrast to the general population, female stroke patients do not appear to be more affected by depression than men (HOSKING; MARSH; FRIEDMAN, 2000). PSD has been found to be related to cognitive impairment such as attention, memory, visual perception and construction, and language (KAUHANEN*et. al.*, 1999; NYS*et. al.*, 2005b). However, the direction of whether cognitive impairment leads to depression or whether PSD leads to impairment is still being debated (HACKETT*et. al.*, 2005; MURATA; KIMURA; ROBINSON, 2000).

In clinical settings, self-reports of cognitive function are common in stroke treatment, and it has been shown that patients frequently report reduced mental rhythm, memory deficits, and difficulties concentrating (DUITS*et. al.*, 2008; HOCHSTENBACH; PRIGATANO; MULDER, 2005). However, it was not always possible to confirm these complaints with empirical data (VAN RIJISBERGEN*et. al.*, 2014). It has been shown that subjective cognitive complaints (SCC) are associated with factors such as satisfaction with the social network, self-reported emotional difficulties, and depression and neuroticism symptom scores (TOOMELA*et. al.*, 2004; PASSIER*et. al.*, 2010; LAMB*et. al.*, 2013). Van Rijsbergen*et. al.* (2014) concluded in their review the frequent occurrence of SCC after stroke. Even though they could not determine a clear pattern relating CCE to objective cognitive performance or depressive symptoms, they emphasized that patients with CCE should be monitored by physicians as it may be indicative of cognitive decline.

The Stroke Impact Scale (SIS) was developed to take into account patients' and caregivers' perspectives on the impact of stroke on health and functional status. Includes a separate scale on memory and thinking function. To date, few studies have combined measures of cognitive performance and self-reported functioning on the SIS Memory and Thinking subscale (mtSIS).

Therefore, this article is justified as it brings an important implication for the health area, as it aims to resolve several doubts related to how cognitively damaging a stroke can be.

This study has the general objective of discussing neuropsychological assessment in patients with stroke and specific objectives: a) Conceptualize cerebrovascular accident (CVA); b) Determine their degrees and c) Understand the cognitive deficits that stroke can cause in its victims. Thus, there is the guiding question: How important is neuropsychological assessment in stroke patients?

2 Theoretical Foundation

2.1 Cerebral Vascular Accident – Stroke

One of the main causes of death and physical disability worldwide is cerebrovascular accident (CVA). In the United States of America, on average 5,000 people have a stroke per year (OLIVEIRA; ANDRADE, 2001).

Of this number, 150,000 die from stroke per year. There are more than three million stroke survivors in the US; and the costs per year arise from the loss of productivity with these patients, thus exceeding 18 billion dollars. Each year, around 50,000 Americans suffer transient ischemic attacks (TIAs), of which almost a third develop a stroke as a result.

Transient Ischemic Attack (TIA) is defined as a rapid episode of loss of brain function, due to ischemia, which can be located in a portion of the central nervous system, supplied by a certain vascular system (right or left carotid, or vertebrobasilar) , and which has no other cause.

By arbitrariness, deficits last less than 24 hours. TIAs typically last a short time (average 2 to 15 minutes). Thus, if there are abrupt episodes, lasting a few seconds, they may not be TIAs (OLIVEIRA; ANDRADE, 2001).

However, there are unusual conditions that go beyond this. This difference is fundamental for efficient secondary prevention. Cooperative studies show that in symptomatic patients who have more than 70% carotid stenosis, endarterectomy works very well to reduce the risk of a subsequent ipsilateral stroke.

In the acute phase of strokes, treatment involves the use of anticoagulants, with restrictions, as they can lower blood pressure too much, general clinical care and, in rare cases, the use of fibrinolytic agents. Hemorrhagic strokes (HCVAs) represent a

accounts for approximately 10% of strokes, tending to occur earlier than heart attacks. Having high blood pressure and advanced age are main risk factors for stroke (OLIVEIRA; ANDRADE, 2001). Considerations on the etiopathogenesis of AVCHs and treatment approaches for these patients are presented. A cooperative study of TIAs determined a mean duration of 14 minutes for TIAs of carotid origin and 8 minutes for those of vertebrobasilar origin. Two-thirds of ischemic episodes are reversed spontaneously, which happens within 1 hour.

When this happens, the term “cerebral infarction with transient signs” is used, when the problem found matches the neurological symptoms presented. A study was carried out in Lausanne, Switzerland, and detected 5 patients with TIAs among 75 patients with internal carotid artery occlusion.

These patients underwent a cranial computed tomography (CT) scan that showed small, deeply located infarcts that were almost indistinguishable from lacunar infarcts.

The authors believe that cerebral infarctions with transient signs have to do with incomplete cerebral necrosis, relating the well-developed collateral network supply, or even to frequent ischemia in the region of an old and silent infarction. The New York Hospital at Cornell Medical Central carried out a study and in the period from 1980 to 1986, they had 382 patients registered with TIAs, 50% of whom showed complete improvement in symptoms in less than 30 minutes and 59% in the first hour (OLIVEIRA; ANDRADE, 2001).

Longer TIA events typically show a change on CT or magnetic resonance imaging (MRI) of the skull, even more so when the latest imaging techniques (perfusion and diffusion techniques) are used. Nicolaides*et. al*. studied 149 patients with hemispherical TIAs; where 48% had an infarction on CT and 35% had an infarction in the hemisphere that corresponded to the symptoms presented.

The authors, in the same study, combined the results of seventeen studies on the frequency with which patients with hemispheric TIAs had infarctions on CT.

Of 738 patients studied, 154 (20%) had infarcts on CT. When it comes to diagnosing cerebral infarctions, MRI is more sensitive than CT (OLIVEIRA; ANDRADE, 2001).

From this study it can be concluded that: 1) TIAs and strokes are part of the same pathological process, being caused by cerebral ischemia; 2) if a

If a doctor observes a patient who presents clinical deficits due to ischemia and the symptoms last for more than an hour, it is very likely that it is a cerebral infarction. Therefore, there needs to be greater rigor in the treatment and investigation of AITs. To diagnose a TIA, it depends on the ability to collect the history of events and their interpretation, except if the doctor watches this event. Numbness and tingling are common symptoms, which do not always indicate that it is a TIA.

Patients with TIAs have a greater chance of having a cerebral infarction than the rest of the healthy population. The risk of having a stroke after a TIA is 24% to 29% over the next five years. This risk is generally 4 to 8% during the first month and 12% during the first year. The risk of TIA patients developing a stroke is increased thirteen- to sixteen-fold during the first year and approximately seven-fold during the following five years. Different subgroups have different prognoses.

Patients who have hemispheric TIAs and carotid stenosis greater than 70% have a dangerous prognosis, with a rate of developing a stroke greater than 40% in two years. Young patients and patients with symptoms of monocular visual deficit have a much better prognosis (OLIVEIRA; ANDRADE, 2001).

The biggest risk factors for cardioembolic events in the genesis of TIAs are: ventricular fibrillation, mitral stenosis, cardiac valve prostheses, recent myocardial infarction, left ventricular thrombus (mainly mobile or pedicled), atrial myxoma, infective endocarditis, dilated cardiomyopathies and marantic endocarditis; other not so important risk factors are: mitral valve prolapse, intense calcification of the mitral annulus, patent foramen ovale, atrial septal aneurysm, calcified aortic stenosis, left ventricular contrality abnormalities and atheromatous plaques in the aortic arch.

All treatment methods must be used so that together they can allow an adequate diagnosis regarding the degree of stenosis, presence and characteristics of plaques, presence of ulcers, dissections or fibromuscular dysplasia.

The characteristics of this plaque can significantly change the resulting ischemic events. Echolucent and heterogeneous plaques have a high lipid content, or intraplaque hemorrhage, which can end up producing ulceration of the plaque, leading to greater embolic potential (OLIVEIRA; ANDRADE, 2001).

When studying asymptomatic patients with carotid artery disease, only around 20% to 30% of them had echolucent plaques, in contrast to symptomatic patients, where echolucent plaques were equivalent to 70%. The most striking feature of the board in the evaluation

of the risk of new events is the percentage of stenosis in the proximal portion of the internal carotid artery. This applies to both symptomatic and asymptomatic patients. AITs are emergency patients. They are to cerebral infarction what unstable angina is to acute myocardial infarction. There are several controversies regarding the best approach to controlling the most recent AITs. Some authors are in favor of using immediate intravenous heparin for patients with a high risk of new TIAs, or with severe or more frequent TIAs (OLIVEIRA; ANDRADE, 2001).

However, no larger study has been carried out to compare the action of heparin to antiplatelet agents. The probable or known etiology of the episodes must be taken into account. Acetylsalicylic acid and ticlopidine are allies in preventing a stroke after a TIA.

In an analysis of the relative benefit, side effects and costs of the drug, acetylsalicylic acid is considered as a drug of choice to initiate antithrombotic treatment.

The permitted dose of acetylsalicylic acid is controversial, as it is accepted that 325mg per day is ideal for adequate protection with minimal side effects. Clopidogrel can also be used successfully, but the cost is much higher.

The use of “statins” to control carotid plaques needs further study; However, preliminary data shows that this type of drug plays a very important role in preventing ulcers and controlling the growth of atheromatous plaques. The antioxidant effects of these medications on coronary plaques are already well known (OLIVEIRA; ANDRADE, 2001).

Strokes will be classified according to the etiological mechanism involved, into: atherothrombotic, cardioembolic, lacunar, hemodynamic and venous. Differentiating these characteristics is essential for efficient secondary prevention.

Atherothrombotic cerebral infarction occurs when atherosclerosis is involved in certain sites of the intra and extracranial arteries, which leads to occlusion or occlusion of one of its branches. Two main mechanisms are known that lead to atherosclerosis and heart attack.

If there is an atherosclerotic plaque with obstruction of the arterial lumen or fragmentation of the plaque or a lump superimposed on it, with arterio-arterial embolism, then thrombosis may occur. History of TIAs and carotid bruits are more normal in patients with atherothrombotic infarction than in other types of strokes. The clinical diagnosis is based on the finding of arterial stenosis or occlusion due to atherosclerosis in one or more sites. The heart attack can

be of very small origin and almost impossible to distinguish from those with cardioembolic origin. It has been shown, through anatomopathological studies, that the distribution of atherosclerotic lesions does not happen by chance along the cerebral arterial network (OLIVEIRA; ANDRADE, 2001). The carotid arterial system is affected first at the carotid bifurcation, then at the carotid siphon, and then at the M1 segment of the middle cerebral artery. In the vertebrobasilar system, the most affected are the first and fourth segments of the vertebral arteries and the first segment of the basilar artery.

The factors that trigger these lesions to become symptomatic are not well known, but what is known is that stenoses greater than 70% are linear and associated with an increased risk of distal cerebral infarction. Corporate studies show that in symptomatic patients with more than 70% carotid stenosis, endarterectomy is very effective in reducing the risk of a subsequent ipsilateral stroke.

However, a team of vascular surgeons should be known and recommended if the risk of surgery is less than 3%. The indications for performing an endarterectomy depend on surgical risk, both for patients with asymptomatic and symptomatic carotid atherosclerotic disease.

These indications must take into account the degree of stenosis and the type of plaque. Having ulcers in the plaque, as shown by angiography, has been an important marker for the risk of having a subsequent stroke (OLIVEIRA; ANDRADE, 2001).

The size and extent of the lesion have been closely related to neurological evolution. Using conventional angiography, the size of the ulcer can be given by multiplying the length by the width of the ulcer in millimeters. Thus, ulcers that measure <10mm2 are called “A” ulcers, ulcers that vary from 10mm2 to 40mm2 are called “B” ulcers, and ulcers that exceed 40mm2 are called “C” ulcers.

The fact of having a “C” ulcer, even if it does not have a carotid stenosis along with it, identifies a group of patients who have a risk of having a stroke of 7.5% per year. If the ulcers are type “A”, there is no relationship with an increased risk of having a stroke.

However, “B” ulcers are subject to many controversies, since some authors relate them to a risk of stroke of 4.5% per year, while others do not find any relationship (OLIVEIRA; ANDRADE, 2001).

Arterio-arterial embolism from aortic plaques has been evaluated more recently. More or less 40% of cerebral infarctions have an unknown cause, or are limited to stenosis

carotid artery less than 70%, or even small heart defects, which are not even accepted as a definitive cause.

With the advent of transesophageal echocardiography, plaques in the aortic arch began to be detected more easily. Several recent studies have established a statistical relationship between atherosclerotic plaques in the aortic arch and stroke (OLIVEIRA; ANDRADE, 2001). In patients who have a plaque larger than 5mm, when compared with a control group, a risk of developing stroke, myocardial infarction or peripheral embolism of 33% in 2 years is found; however, if only retinal events and strokes are analyzed, the risk is 16% in 2 years, since this risk is 7% in the control group without atheromas. Atherosclerotic plaques ≥ 4mm thick in the ascending aorta and proximal aortic arch were found in 14.4% of patients with cerebral infarction and in 2% of the control group ($p < 0.001$).

A strong association was found between plaques that are 1mm to 3.9mm thick and carotid stenosis, but with plaques greater than 4mm thick, the risk of having a stroke increases from 5 to more than 13 times. This large increase was only noticed for plaques greater than 4 mm thick, located close to the ostium of the left subclavian artery, and not plaques distal to the ostium. It can be believed that plaques more than 4 mm thick have thrombotic material superimposed on ulcerated plaques in their composition.

Therefore, aortic plaques that are more than 4mm thick are a major risk factor for cerebral infarctions, with a possible etiological relationship in some patients. The way to approach these patients in a therapeutic way has not yet been determined.

There are numerous reports of cases of thrombolysis or surgical removal of atheromas found by transesophageal echocardiography. An alternative is anticoagulation, even though there are still those who doubt that it is completely safe, since the anticoagulant can help cause microembolization of cholesterol crystals to remove the thrombus over an ulcerated plaque (OLIVEIRA; ANDRADE, 2001).

The objective of anticoagulants and antiplatelet agents to prevent cerebral infarctions due to plaque thrombi in the aortic arch still requires further study. Infarcts in border areas, which are also called infarcts in watershed areas, secondary to an aerodynamic mechanism, are much more difficult to occur. They may occur due to the association with critical stenosis of the

carotid arteries, or hemodynamic disorders, hypotension or bradyarrhythmia. Its real prevalence has not yet been estimated, but the more patients who have experienced long periods of hypotension are investigated, such as when undergoing major surgery, examples of this type of stroke can be found (OLIVEIRA; ANDRADE, 2001) .

The way to control blood pressure has not yet been established. A slight or moderate increase in blood pressure is frequently seen in patients who have suffered strokes, in a transient manner, and appearing to be a type of compensatory mechanism due to the loss of brain autoregulation, lasting a few days, and generally no need to apply any type of treatment.

Some events end up helping to increase blood pressure, such as stress, pain, bladder fullness, increased intracranial pressure, a physiological response to hypoxia; the pressure drops spontaneously when these changes are corrected.

However, the loss of autoregulation of cerebral blood flow, which occurs in the region where the ischemia arrived, can be considered and worsen the injury with an eventual reduction in blood pressure, reducing perfusion.

Patients with chronic hypertension have autoregulation of cerebral blood flow regulated with higher blood pressure levels, and a sudden reduction can cause greater damage to the brain (OLIVEIRA; ANDRADE, 2001).

In cases where hypertension is more severe, reducing blood pressure must be done slowly and carefully. The use of antihypertensive medication is indicated when mean arterial pressure is higher than 130 mmHg or when systolic blood pressure is higher than 220 mmHg. When blood pressure is associated with hemorrhagic transformation, myocardial infarction, thoracic aortic dissection or secondary renal failure, parental drugs should be used, such as enalapril or labetalol.

For arterial hypertension associated with intracranial hypertension to be controlled, drugs that increase cerebral vasodilation (sodium nitroprusside) and drugs that cause a rapid drop in blood pressure, such as sublingual calcium blockers, should not be used. . For most patients, beta-blockers or angiotensin-converting enzyme inhibitors are sufficient. Diuretics should also be

avoided because they cause hypovolemia and seriously worsen the neurological condition (OLIVEIRA; ANDRADE, 2001).

Arterial hypotension is a very difficult thing to see, while hypovolemia is much more common. The treatment priority in the early phase of stroke is to correct hypovolemia and normalize cardiac output. While high blood pressure is being treated, it must be known whether or not the patient can use thrombolytics. If the response is positive, then different drugs will be prescribed as treatment.

Research into the treatment of stroke using thrombolytic or fibrinolytic agents began when these medications began to be used to treat acute myocardial infarction. After important studies, the use of thrombolytic agents for stroke has become possible and much safer. However, this study also showed the failure to use thrombolytics for isolated cases of cerebral infarction, opening other controlled studies with streptokinase and rt-PA.

Through these studies, limits and conditions for safer thrombolytic treatment were established. The NINDS study obtained the best result, and its methods were used as a basis for the American Academy of Neurology and the American Heart Association to demonstrate essential criteria and procedures for treatment to be safer and more effective (OLIVEIRA; ANDRADE, 2001).

It is important to be aware of the considerable number of intracerebral hemorrhages obtained in all multicenter studies, both with the test drug and the placebo, which shows that it is necessary to improve the patient selection method.

Knowing and checking the criteria of this protocol are very important prerequisites for this treatment to be safe. Thrombolysis can only be performed when a doctor specialized in stroke and capable of interpreting a head CT gives the final diagnosis (OLIVEIRA; ANDRADE, 2001).

Studies measuring the head CT interpretation capacity of emergency physicians, neurologists and radiologists showed that even the latter were not skilled enough to localize an intraparenchymal hemorrhage, which hinders the adequate selection of patients for thrombolysis. It is very important that there is specific training for doctors in the area of cerebrovascular diseases, so that the diagnosis can be improved.

In places where stroke treatment is carried out with thrombolytic agents, whether anywhere in the world, which greatly limits the trained team to provide a correct assessment

and starting treatment safely, is the delay for the patient suffering from a stroke to reach the hospital. It is normally not recommended to lower blood pressure (BP) in patients who are in the acute phase of stroke, however several studies have already shown that high BP, above certain levels, is very strongly associated with hemorrhagic transformation and negative results when thrombolytic treatment is used, so in this case it is recommended that BP be lowered only to an adequate level so that thrombolytic treatment can begin (OLIVEIRA; ANDRADE, 2001).

After RT-PA infusion, blood pressure needs to be maintained below the indicated levels for at least 24 hours. Intra-arterial injection of thrombolytics is an effective alternative in the treatment of stroke in the acute phase, being advantageous as it allows a greater local concentration of the agent and a lower systemic concentration of it. Without forgetting the other advantage, which is safer patient selection, having the occluded branch of the artery demonstrated through angiography.

With this study we realized that the time factor is very important and very limited for the indication of thrombolytics, becoming more critical, since for an angiographic study to be carried out, as all the normal procedures of a hospitalization, necessary clinical and neurological evaluations they usually take longer than the maximum tolerance for treatment.

AVCH accounts for 10% of the total number of strokes, and generally happens faster than heart attacks. They are much more common than subarachnoid hemorrhage and much more aggressive than cerebral infarction. Some studies show a mortality rate of 30% to 50% in the first month, with half of these deaths occurring in the first two days (OLIVEIRA; ANDRADE, 2001).

After a month, 10% show an independent life, and after six months, this number rises to 20%. High blood pressure and advanced age are the main risk factors for stroke. There is a small preference for young and middle-aged black men. There is also this preference with Asians, compared to Westerners.

Dietary factors also affect the AVCH rate. Japanese men, residents of Japan show a higher incidence of AVCH than those who moved to the USA. Black people in the US also show a much higher risk than white people.

AVCHs, when attributed to hypertension, typically occur in the basal ganglia, thalamus, pons and cerebellum. These areas are supplied by small-caliber vessels, direct branches to wider vessels, thus more vulnerable to the effects of

pressure. These areas are also the sites of lacunar infarcts, as this same vascular disease secondary to arterial hypertension is responsible for both (OLIVEIRA; ANDRADE, 2001).

A higher risk of mortality from stroke is related to low cholesterol levels (<160mg/dl). The reason for this to happen is unknown. Other types of later studies were related to a higher incidence of stroke at low cholesterol levels and diastolic hypertension. Alcohol consumption and the occurrence of AVCH are related in a more complex way.

New studies suggest a behavior similar to a stroke, having a protective effect for daily consumption of up to 60g/day. The main pathophysiological changes for the genesis of AVCH occur in small arteries and arterioles due to the effect of arterial hypertension. However, other causes have been found during hemorrhagic strokes.

Other causes that are not so common are arteriovenous malformations, aneurysms, coagulation disorders and the use of anticoagulants or thrombolytics, hemorrhagic transformations of heart attacks and bleeding from tumors, or even drug abuse (OLIVEIRA; ANDRADE, 2001).

A cause that doctors hardly notice is the chronic use of antiplatelet agents, especially aspirin, which facilitates a hemorrhagic stroke in a patient who is medicated preventively, even more so if trying to avoid a stroke or an ischemic event in someone else. organ. The number of patients who use aspirin is considerable, often serving to primarily prevent cardiocirculatory ischemic events.

Arteriovenous malformations, aneurysms and tumors may be detected. Usually, attempts are made to determine that the cause of the bleeding is due to its location, following the idea that deep capsulonuclear bleedings are attributed to microvascular changes secondary to arterial hypertension, since lobar bleedings are caused by amyloid angiopathy, such as We already have the chance to mention them previously. A presumed but unproven diagnosis shows a greater risk of resorting to the patient (OLIVEIRA; ANDRADE, 2001).

When analyzing angiograms in patients who suffered stroke, changes were found in 84% of them (32/38). In 19% of patients, unsuspected arteriovenous malformations were detected, and in 5%, aneurysms. It has been shown, through a prospective study, that cerebral angiography has a minimal chance of achieving

distinguish changes in patients who have suffered stroke and who are over 45 years of age, with putaminal or posterior fossa thalamic bleeding, and a history of hypertension.

2.2 Neuropsychological Assessment in Stroke Patients

The choice of the Rey Complex Figure Test (RCFT) may have contributed to findings that indicate a high impairment in the visual memory domain. The test requires visuomotor skills during the copy condition (the score used to represent the visuoconstructive domain) and subsequently reproducing/memorizing a complex line drawing (visual memory domain). Thus, the failure may arise from constructive deficiency, visual impairment or spatial memory or an interaction of both, or even other factors (LEZAK; HOWIESON; LORING, 2004). Through this complexity, the RCFT can therefore tap slightly different abilities than a visual memory test in which items must simply be remembered.

Barker-Collo*et. al*. (2010) including the RCFT in a 5-year follow-up study found that visual memory scores were within 1 SD of the normative data, while group average copy function was clearly impaired being >2 SD below the normative data. They suggest that the impaired copy score is related to the executive functions that are required when copying the picture. As suggested by Barker-Collo*et. al*. (2010) contributed to visual memory impairment.

The findings showing impairment in executive functioning and processing speed are generally in line with previous findings (NYS*et. al*., 2005a; STEPHENS*et. al*., 2004; SACHDEV*et. al*., 2009). In particular, the finding that executive impairment occurred more frequently in the older age group is notable.

It was emphasized that executive dysfunction and reduced processing speed frequently occur in individuals with vascular cognitive impairment (NORDLUND*et. al* ., 2007) who may present additional cognitive decline and progression to dementia (SACHDEV*et. al*., 2009). We can only speculate that this applies to some of the patients.

Still, he highlights the need for longitudinal studies including comprehensive cognitive assessment in stroke patients. This importance is further emphasized and

expanded by results showing significant correlations between symptoms of depression, executive function, processing speed and language production.

The proportion of patients reporting symptoms of depression and anxiety (13–14%, respectively) was considerably lower compared to those reported by Ayerbe *et. al*. (2011) (28% depression and 33% anxiety) or Kauhanen*et. al*. (1999) (42%) and more similar to those of Astrom*et. al*. (1993) (16% depression). However, Ayerbe*et. al*. (2011) found that cognitive impairment (based on the MMSE) predicted depression over a 5-year follow-up and showed that the prevalence of depression even doubles in these individuals.

Language production had the strongest inverse relationship with depression. In other words, reduced word production indicated more symptoms of depression. Despite requiring knowledge and semantic processing, these tests, in particular letter fluency, are shown to achieve a lot of executive functioning, since retrieval strategies must be initiated, the answers given must be kept in mind to avoid repetitions (working memory) and semantically closed words should be avoided (LUO; LUK; BIALYSTOK, 2010).

It has previously been shown that executive dysfunction is closely associated with activities of daily living (POHJASVAARA*et. al*., 2002) and accelerated cognitive decline (LEVINE *et. al*., 2015). The clear link between depression and executive functioning emphasizes the importance of monitoring these patients to prevent them from ending up in a vicious circle.

Analyzes showing that the mtSIS subscale correlated significantly with the cognitive domains of language, executive function, and memory are novel and noteworthy. To our knowledge, no previous studies have combined the mtSIS with neuropsychological assessment.

At first, it should be recognized that self-reported functioning based on the mtSIS was quite positive, as indicated by a high median. This appears to be in some contrast to previous studies that demonstrated cognitive complaints in more than 50% of stroke patients (HOCHSTENBACH; PRIGATANO; MULDER, 2005).

Duncan*et. al*. (2003) found that the items were very easy to answer and would only detect difficulties in severely impaired stroke patients. There are significant positive correlations between the mtSIS and executive functioning and language production, although 4 of the 7 mtSIS questions relate to memory function.

This indicates that patients who report good cognitive functioning also perform equally well.

However, sensitivity and specificity for the mtSIS across all cognitive domains were critically low. Although the lack of empirical evidence between objective and self-reported cognitive function is not a new phenomenon, it indicates that clinicians should be cautious when using the mtSIS to identify patients with cognitive impairment.

Thus, the commitment may not have been as prominent as it needed to be mentioned. In fact, there is a significant correlation between mtSIS scores and the number of impaired domains. This may imply that a certain "threshold" of disability must be crossed before it affects routine daily activities frequently enough to be observed and reported by the patient. However, the sensitivity problem was commented on by Duncan*et. al*. (2003) even in a more heterogeneous group of patients. In his review of subjective cognitive complaints, van Rijsbergen*et. al*. (2014) highlighted the urgent need for more information about post-stroke complaints and their multifaceted underlying reasons.

Analyzes revealed that additional referral bias is possible since the study includes subjects with fairly mild stroke (as indicated by the NIHSS) and subjects who are able to undergo neuropsychological assessment. Thus, patients with more severe stroke and more severe cognitive impairment and/or depression are underrepresented.

Final considerations

Cognitive impairment in chronic stroke patients occurs frequently and persistently in the first year after stroke. It is important to provide a more comprehensive neuropsychological assessment in addition to screening measures to detect cognitive deficits as well as their interaction with depressive symptoms.

The results underscore the fact that stroke patients must be monitored and that longitudinal data are needed. Providing a more comprehensive assessment for stroke patients allows doctors to provide appropriate treatment and identify individuals who need additional care. Doctors should be aware of possible compromises, even if patients do not report them.

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