International Journal of Trend in Scientific Research and Development (IJTSRD)
Volume: 3 | Issue: 3 | Mar-Apr 2019 Available Online: www.ijtsrd.com e-ISSN: 2456 - 6470

Ischemic Brain Vascular Accident: Acute Phase Management Acute Ischemic Stroke: Early Management

Renato Serquiz E Pinheiro[1], Yanny Cinara T Ernesto[2], Irami Araújo-Neto[2], Fausto Pierdoná Guzen[3], Amália Cinthia Meneses Do Rêgo[3], Irami Araújo-Filho[3,4]

[1]Professor of the Medicine Course of the Potiguar University – UnP, Natal, Brazil
[2]Student of the Medicine Course of the Potiguar University – UnP, Natal, Brazil
[3]Graduate Program in Biotechnology of the University Potiguar - UnP, Natal, Brazil
[4]Associate Professor II, Department of Surgery – UFRN, Natal, Brazil

How to cite this paper: Renato Serquiz E Pinheiro | Yanny Cinara T Ernesto | Irami Araújo-Neto | Fausto Pierdoná Guzen | Amália Cinthia Meneses Do Rêgo | Irami Araújo-Filho "Ischemic Brain Vascular Accident: Acute Phase Management" Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN: 2456-6470, Volume-3 | Issue-3, April 2019, pp.1712-1718, URL: https://www.ijtsrd.com/papers/ijtsrd23499.pdf

IJTSRD23499

ABSTRACT

Objective: To address, in a practical way, the acute treatment of ischemic cerebrovascular accident (CVA) based on the scientific recommendations latest.

Methods: A bibliographic search was performed in the PubMed, Scopus, Scielo and Uptodate database from January/2012 to April/2018, using the descriptors "stroke", "early management", "therapeutic", "intravenous thrombolysis", "combined treatment", "mechanical thrombectomy" and its combinations. The selection of the articles was made by listing those of greater relevance according to the proposed theme, both in the foreign and Brazilian literature, in a non-systematic way.

Results: Intravenous thrombolysis with recombinant tissue plasminogen activator (rtPA) within 4.5 hours of onset of symptoms is considered the therapy of choice in eligible patients. According to the new guidelines, mechanical thrombectomy can be performed within 24h and, for prevention of subsequent ischemic events, revascularization between 48h and seven days of the index event in candidate patients is reasonable.

Conclusions: As an essential cause of death and disability in the world, acute ischemic stroke treatment has advanced rapidly in recent years, improving therapeutic methods and their combinations. In clinical practice, recognizing, stratifying and listing, quickly and effectively, the best therapy for stroke patients is paramount.

KEYWORDS: *"stroke", "early management", "therapeutic", "intravenous thrombolysis", "combined treatment", "mechanical thrombectomy".*

INTRODUCTION

According to the American Heart Association and American Stroke Association (AHA/ ASA), any irreversible cerebral, spinal or retinal marrow attributable to ischemia is defined as stroke, based on neuropathological evidence, neuroimaging and/or clinic of permanent injury; when imaging or pathology is not available, is recognized by the persistence of symptoms for more than 24h[1]. Transient ischemic attack (TIA) is a temporary episode of neurological dysfunction caused by focal cerebral, spinal cord or retinal ischemia without evidence of acute infarction on imaging examinations[2].

According to Heart Disease and Stroke Statistics 2018, every year about 795,000 people have a stroke, being one in every 40 seconds. Approximately 610,000 of these are the first AVE event, and 185,000 are recurrent events. Tree of all cases, approximately 87% are ischemic, and 10% are intraparenchymal hemorrhages, while 3% are subarachnoid hemorrhages[3-5].

Throughout life, women have a higher risk of stroke so that, each year, there are about 55,000 more cases of stroke in women than in men, according to NINDS[4]. In the United States, between 2005 and 2015, the mortality rate decreased by about 21%; but even so, when considered separately from other cardiovascular diseases, stroke is the fifth cause of death[3]. Such mortality rates are higher after a hemorrhagic stroke (67%) than the ischemic stroke (57%), according to ARIC dados data[3,4].

In Brazil, despite the decline in mortality rates, the second cause of death and the first cause of disability in the country, which creates extraordinary economic and social impact. Data from the Ministry of Health 2013 indicate an annual

incidence of 108 cases per 100,000 inhabitants, the mortality rate at 30 days of 18% and 12 months of 31%, with a recurrence rate after one year of 16%[5].

Because of the importance of rapid and effective management of these acute patients, the new AHA/ASA recommendations consider the development of regional stroke care systems so that they can provide early emergency care, including administration of intravenous alteplase (rtPA IV); perform more advanced care, such as comprehensive care endovascular treatment; when necessary, facilitate the rapid transport to advanced centers[6]. In addition to participating in a database of stroke to improve adherence to treatment guidelines and the quality of results. In hospitals that do not have neurologists on call, telestroke assessments are recommended for screening patients eligible for intravenous thrombolysis or transfer for mechanical thrombectomy[7].

Methods

This is a literature review study, whose bibliographic research was carried out in Pub Med, Scopus, Scielo and Up to date databases. The descriptors used were "stroke", "early management", "therapeutic", "intravenous thrombolysis", "combined treatment", "mechanical thrombectomy" and their combinations through Boolean operators AND and OR. As inclusion criteria, were selected articles published between January/2012 to April /2018, in English and Portuguese; clinical trials (clinical trial), guidelines (guideline), systematic reviews (systematic review and review). Articles that addressed thrombolytic treatment in diseases other than ischemic stroke were excluded. The selection of articles was made by listing those of greater relevance according to the proposed theme, both in the foreign and Brazilian literature, in a non-systematic way.

Discussion

Initial evaluation

The initial assessment of a patient with a possible stroke is similar to that of other critical patients: immediate airway stabilization, respiration, and circulation (ABCs). This is quickly followed by an assessment of deficits of neurological and possible comorbidities. The general objective is to not only identify patients with possible stroke but also exclude other diagnoses, identify other conditions that require immediate intervention and determine possible causes of stroke for early secondary prevention. It is recommended to use a stroke evaluation scale, preferably NIHSS (National Institutes of Health Stroke Scale), score ranging from 0 to 42, with higher scores indicating more massive deficits. (Class I, Level of Evidence B)[1,6].

A limited number of hematological exams and biochemical tests are recommended during the initial emergency evaluation, but the only laboratory outcome required before fibrinolysis therapy is the assessment of blood glucose. (Class I, Level of Evidence B) [1,6,7]. Besides, an emergency neuroimaging examination is recommended before initiating any specific treatment to treat acute ischemic stroke. (Class I, Level of evidence A) [6-8].

In most cases, unconfined skull computed tomography (CT) will provide the information needed to make emergency management decisions, since it excludes parenchymal hemorrhage and may assess other exclusion criteria for thrombolytic therapy, such as hypoatenance (> 1/3 of the cerebral hemisphere), as well as helping to discriminate non-vascular causes of neurological symptoms (e.g., brain tumors)[8]. However, it is relatively insensitive in detecting acute and small cortical or sub cortical infarctions, especially in the posterior fossa within the first 24h [9,10].

Compared with CT scan of the skull, the advantages of magnetic resonance imaging (MRI) include the ability to detect posterior circulation strokes (cerebellum and brainstem), differentiate cerebral infarctions from inflammatory/demyelinating lesions, distinguish between acute and chronic ischemia, as well as other differential diagnoses. Limitations include cost, relatively limited availability and relatively long duration of the exam (usually>30min), increased vulnerability to movement artifact and patient contraindications, such as claustrophobia, cardiac pacemaker, patient confusion, or metal implants. MRI is more sensitive to the presence of ischemia, but in most institutions, unconfined skull CT remains the most recommended initial neuroimaging examination because of its immediate wide availability, acquisition speed, and relative ease of interpretation [1-3,6].

Noncontrast cranial CT should be obtained within 20min of the patient's arrival in the emergency department and candidates for intravenous fibrinolysis , the imaging of the brain should be interpreted within 45min after the arrival of the patient in the emergency room by a physician with experience in reading CT/MRI (Class I, Level of Evidence C)[6-8,10].

Signs of cerebral ischemia in the first hours after the onset of symptoms in the cCSC of the skull:

- Loss of gray matter differentiation: it may manifest as a loss of distinction between basal ganglia nuclei (lenticular obscuration) or as a mixture of the densities of the cortex and underlying white matter in the insula (insular tape) and on the convexities (sign of the cortical tape);
- Blurring of the brain grooves;
- Hyperdensity of the middle cerebral artery (ACM);
- "Spot" hyperdense of ACM: may represent a clot within a branch of ACM that possibly means the best destination for intravenous rtPA.

General support

Positioning and monitoring

Patient positioning may influence oxygen saturation, cerebral perfusion pressure, mean MV flow velocity, and intracranial pressure (ICP). In no hypoxemic patients able to tolerate rest, a supine position is recommended given the advantage of offering better cerebral perfusion. Patients at risk of airway obstruction or aspiration and those with suspected elevated PIC should have the bed head elevated from 15°-30° while maintaining centered positioning of the head and neck free of compression[11].

Cardiac monitoring is recommended to detect atrial fibrillation and other potentially dangerous cardiac arrhythmias that would require emergency cardiac interventions. Cardiac monitoring should be performed for at least the first 24h. (Class I, Level of Evidence B)[6].

Supplemental oxygenation

Hypoxia often appears after stroke and is defined as <96% oxygen saturation for a period higher than 5min. Supplemental oxygen may be beneficial in patients with severe cerebrovascular accidents, and oxygen

administration to hypoxemic patients is recommended in order to maintain oxygen saturation> 94%. (Class I, Level of evidence C) [1,9]. When oxygen treatment is indicated, it is reasonable to use the least invasive method possible to achieve normoxia.

Body temperature

Approximately one-third of stroke patients will be hyperthermic (temperature> 37.6°C) in the first hours after the onset of the disease[1]. In these cases, use of acetaminophen or dipyrone is indicated, since such condition is associated with worse neurological prognosis, possibly secondary to increased metabolic demands, increased release of neurotransmitters, and increased free radical production. Hyperthermia may be secondary to a cause of stroke, such as infective endocarditis, or may represent a complication such as pneumonia, urinary tract infection (UTI), or sepsis. That way, it is essential that there is always better research[8].

Although strong experimental and clinical evidence indicates that induced hypothermia can protect the brain in the presence of global hypoxia or ischemia, even after cardiac arrest, the utility of hypothermia-induced for the treatment of patients with ischemic stroke has not yet been well established and more studies[12,13].

Intravenous fluids

Hypovolemia may predispose to hypoperfusion and exacerbate ischemic brain injury, cause renal impairment, and potentiate thrombosis. Hypervolemia may exacerbate ischemic cerebral edema and increase myocardial stress. Therefore, euvolemia is desirable. Is recommended intravenous fluid in those who have impaired swallowing [1,6,13].

Hypotonic solutions, such as 5% glycosated serum or 0.45% saline, are distributed in intracellular spaces and may exacerbate ischemic cerebral edema. Isotonic solutions, such as 0.9% saline, are more evenly distributed in the extracellular (interstitial and intravascular) spaces, and thus preferable. The administered volume (about 100 mL/hour) should be considered individually, based on basal hydration and possible comorbidities[8].

Glycemia

Hypoglycemia during an acute ischemic stroke is rare and probably related to antidiabetic medications. However, if left untreated, severe or prolonged hypoglycemia can result in permanent damage to the brain. Thus, low levels (<60 mg/dL) should be corrected urgently[14,15].

Hyperglycemia is common during the acute phase and is related, in part, to a non-fasting state and partly to a stress reaction with impaired glucose metabolism. It is recommended to treat hyperglycemia to achieve blood glucose levels between 140 and 180mg/dL to prevent hypoglycemia in patients with acute ischemic stroke (Class IIa; Level of Evidence C)[6].

Blood pressure

Theoretically, moderate arterial hypertension during acute ischemic stroke may be advantageous, improving cerebral perfusion of ischemic tissue, or may be detrimental by exacerbation of edema and hemorrhagic transformation of ischemic tissue. Extreme arterial hypotension is detrimental because it reduces perfusion to multiple organs, especially the ischemic brain, exacerbating the ischemic injury[6,16].

The BP approach will depend on the treatment of choice for the patient. In patients NOT candidates for fibrinolytic therapy:

- If PA> 220 x 120 mmHg: administer antihypertensive medications (metoprolol, esmolol or sodium nitroprusside) with the aim of reducing the blood pressure value by around 15% in 24 hours[1,8].
- If PA <220 x 120 mmHg: conservative treatment, except in cases of acute myocardial infarction, acute pulmonary edema, aortic dissection, acute renal failure or hypertensive encephalopathy (acute or subacute onset of lethargy, headache, confusion, visual disturbances or seizures)[8].

The recommendation not to lower blood pressure during the initial 24h of acute ischemic stroke unless the blood pressure is higher than 220x120 mmHg or there is a concomitant specific medical condition that would benefit from the reduction in blood pressure remains applicable. (Class I, Level of evidence C)[1,6]. For patients candidates for fibrinolytic therapy, see below under "Intravenous Thrombolysis".

Anticoagulation

Early administration of anticoagulants does not decrease the risk of early recurrent stroke (including among patients with cardioembolic sources), does not interrupt early neurological worsening, or improve outcomes after the acute ischemic event. In addition, it increases the risk of bleeding complications. Thus, urgent anticoagulation is not recommended in acute ischemic stroke (Class I; Evidence Level A) [10].

Although parenteral anticoagulation with unfractionated heparin or low molecular weight heparin is not recommended within the first 48h after anticoagulation, anticoagulation is essential as a secondary prevention of stroke in cases of paroxysmal or persistent AF. Ischemic stroke caused by acute myocardial infarction (AMI) and evidence of mural thrombus in the left ventricle; dilated cardiomyopathy; patent foramen oval; cardiac valve disease; AVE by dissection of cervical arteries; acquired thrombophilia; antibody-antiphospholipid syndrome, among others [11].

For stable patients with small to moderate infarction, warfarin can be started 24h after stroke and new anticoagulants after 48h because they have the fastest anticoagulant effect. While in those with large infarcts, poorly controlled hypertension or other bleeding conditions, it is generally recommended to delay anticoagulation, depending on the condition, for up to 2 weeks[15,16].

The use of warfarin in an adjusted dose (INR 2-3) is preferred for patients with previous use of warfarin and relatively easy to control INR. In those with prosthetic heart valves; patients with rheumatic mitral valve disease; mitral stenosis of any origin or other valve lesions associated with moderate to severe heart failure that may lead to valve replacement in the near future. If there is social contraindication (difficulty in adhering to INR control), prophylaxis should be done with double antiplatelet platelet (ASA 100mg/ day + clopidogrel 75mg/day) [11].

Regarding the new oral anticoagulants, rivaroxaban (20mg/day) is not inferior to warfarin for the prevention of stroke and/or thromboembolic phenomena in patients with non-valvular AF, whereas apixaban (5mg-2x/day) and dabigatran (150mg-2x/day) proved superior concerning then[13]. Regarding the occurrence of bleeding, reduced incidence of intracranial hemorrhage with new oral anticoagulants. Other advantages include convenience (no need for routine INR testing), few food and drug interactions. Disadvantages include lack of efficacy and safety data in patients with severe chronic kidney disease, lack of readily available monitoring of blood levels and compliance, higher cost and the potential for unplanned side effects [17,18].

Antiplatelet

Oral administration of aspirin (initial dose of 325mg) within 48h after stroke is recommended for the treatment of most patients (Class I; the level of evidence A) [1,6], as there is a reduction in stroke recurrent disease and, consequently, a statistically significant decline in mortality. It is indicated mainly for cases of atherothrombotic stroke since cardioembolic stroke has greater benefit with anticoagulation. In addition, after the acute phase, the oral use of AAS even at low doses (e.g., 100mg/day) is enshrined in the secondary prophylaxis of stroke/TIA[19,20].

There is no evidence to support the routine administration of any other antiplatelet agents Platelet alone or in combination with other substances in the treatment of acute ischemic stroke (Class II, Level of evidence B)[10]. However, clopidogrel (75mg-daily) and ticlopidine (2x-250mg/day) are indicated for the prevention secondary to the AVE/TIA or as an alternative when the AAS is contraindicated, with clopidogrel being a better alternative because of its similar efficacy to ASA and the lower profile of serious adverse events when compared to ticlopidine[14].

There is controversy regarding the recommendation of the double antiaggregation platelet aggregation. The CHANCE trial (clopidogrel + aspirin for 21 days, followed by clopidogrel alone for a total of 90 days) showed a reduction in the risk of subsequent vascular ischemic events without change in the risk of bleeding among patients with high-risk TIA ($ABCD^2 \geq 4$) or minor ischemic stroke (NIHSS ≤ 3) that are initially seen within 24h after the onset of symptoms, but other studies are still ongoing[15].

Statins

Although studies have not shown a reduction in mortality with the use of statins in the acute phase of ischemic stroke, patients already on statins at the time of stroke should continue to take them. According to the Brazilian directive of acute treatment of the AVE, the administration of statins after 48h of stroke is recommended (Class I, Level of Evidence B)[10], being secondary prevention for both patients with hypercholesterolemia (LDL target <100mg/dL), as well as patients with ischemic stroke or TIA of presumably atherothrombotic origin, even if they present normal cholesterol [21,22].

For patients with high vascular risk, the target is LDL<70mg/dL (established cardiovascular disease associated with multiple primary and poorly controlled risk factors, especially diabetes mellitus; multiple risk factors for metabolic syndrome, mainly triglycerides>200mg/dL and HDL<40mg/dL, coronary disease, carotid stenosis, atherothrombotic stroke) [11,23].

Among statins, rosuvastatin (10mg/day dose), with the same degree of safety and tolerability and at a lower dose, promotes stabilization of atheroma plaques and a more effective reduction of LDL levels associated with an increase in levels of HDL, compared to atorvastatin (10 mg/day), simvastatin (20 mg/day) and pravastatin (20 mg/day), thus being the statin of choice[16].

Intravenous thrombolysis

Intravenous administration of recombinant tissue-type plasminogen activator (rtPA) remains the only pharmacological therapy approved by the Food and Drugs Administration (FDA) for the treatment of acute ischemic stroke. Clinical trials with streptokinase were interrupted prematurely due to unacceptably high bleeding rates; therefore, this drug should not be used[10]. Other fibrinolytic agents administered intravenously, including reteplase, urokinase, anistreplase, and staphylokinase, have not been extensively tested.

For greater safety, the application of rtPA (IV) should respect inclusion and exclusion criteria. Certain factors interfere with the risk/benefit of thrombolytic therapy, but there is no absolute contraindication to its use[10]:

- Age> 80 years;
- Baseline NIHSS score> 22;
- Hyperglycemia.

In patients eligible for intravenous administration of rtPA, the benefit of therapy is time-dependent, and treatment should be started as soon as possible. Needle-time (bolus administration time) should ideally be 60 minutes after arrival at the hospital. (Class I, Level of evidence A – Table 1)[6,10].

Table1. Inclusion and exclusion criteria for treatment of ischemic BIRD with rtPA IV

Inclusion criteria
BIRD in any territory ischemic injury
Onset of symptoms < 4.5 hours (If symptoms are observed upon awakening, one must consider the last time in which the patient was observed normal); CT/MRI without evidence of bleeding.
Age ≥ 18 years
Exclusion criteria
Significant head trauma or ischemic BIRD in the last 3 months
Clinical suspicion of subarachnoid hemorrhage or acute dissection of aorta
Arterial puncture in the last 7 days incompressible; Lumbar puncture in the last 7 days;
History of previous intracranial hemorrhage or cerebral vascular malformation

PAS ≥ 185 mm Hg or PAD≥110 mm Hg (in 3 occasions with 10 minute intervals) antihypertensive treatment refractory; Speedy recovery and full of signs and symptoms in the period before the opening of the Thrombolysis; Mild neurological deficits (no significant functional impact); Major surgery or invasive procedure within the last 2 weeks;
Bleeding from the gastrointestinal tract or urinary tract in the last 21 days or history of esophageal varices.
Prolonged coagulopathy with TP (INR > 1.7), TTPA high or < 100 000 platelets/mm ^{3}
Use of heparin within 48 hours, resulting in TTPA greater than the upper limit of normal.
Current use of anticoagulant with INR > 1.7 – TP >15 seconds.
< 50 blood glucose mg/dL with reversal of symptoms after correction Evidence of endocarditis/septic embolus or pregnancy Acute myocardial infarction within the last 3 months
TC with early hipodensidade > 1/3 the territory of the ACM

Source: Malik S, 2012.

The pressure control has fundamental importance in thrombolytic treatment, aiming to minimize hemorrhagic complications. The infusion of rt-PA should not be started before controlling for blood pressure.[1] If SBP between 185 and 220 mmHg or DBP between 110 and 140mmHg (Table 2)[11]:

- Metoprolol (1 amp = 5 mL = 5mL): apply (IV) 5 mg every 10 min, being 1 mL/min. Maximum 20mg;
- Esmolol (1amp = 2500mg = 10mL): dilute 1 amp in 240mL of 0.9% SF and apply (IV) 0.5mg/Kg in 1 minute. Then a continuous infusion of 0.05 - 3.0mg/kg/min (start at the lowest dose and adjust every 4 minutes, repeating the dose of attack and increasing the infusion until the desired BP is achieved).

Table 2. Intravenous rtPA

Admit the patient to an intensive care unit or bird to monitoring.
Infuse rtPA IV 0.9 mg/kg (maximum dose of 90mg), being 10% of the administered dose in bolus in the first minute and the rest over the course of 60 minutes.
If there is any suspicion of intracranial bleeding, interrupt the RTPA and CT skull without urgent contrast request, CBC, PT, TTPA, platelets and fibrinogen.
Measuring blood pressure and perform neurologic evaluations with NIHSS every 15 minutes during and after IV infusion of rtPA for 2 hours, then every 30 minutes for 6 hours, then every hour until 24 hours after treatment.
Increase the frequency of measurements of blood pressure > 180 mmHg if the PAS or if PAD>105 mmHg; administer antihypertensive medications to maintain blood pressure equal to or lower than these levels.
Keep the patient in fasting for 24 hours due to the risk of bleeding and the need for emergency surgery.
Maintain hydration with physiological saline. Just use dextrose if there is hypoglycemia (in this case, use isotonic saline: SG 5% + 20% NaCL 40 mL).
Don't pass nasoenteral probe, probe vesical or central venous catheter or arterial puncture in the first 24 hours.
Don't use heparin, platelet antiagregante or anticoagulant in the first 24 hours after administration of rtPA.
After 24 hours of treatment, thrombolytic treatment of STROKE follows the same guidelines of the patient who did not receive Thrombolysis, i.e. platelet antiagregante or anticoagulation.
Start prophylaxis for VTE with intermittent pneumatic compression or low weight heparin/24 hours pós-trombólise enoxaparin.

Source: Malik S, 2012.

It is known that the AHA/ASA recommends, as first-line drugs, labetalol, and nicardipine, but not available in the Brazilian market. In cases of asthma, cardiac insufficiency or severe cardiac function abnormalities that contraindicate the use of beta-blockers, or in cases of uncontrolled hypertension (SBP>220mmHg and DBP>140mmHg), it is suggested[11]:

- Sodium nitroprusside (1amp = 50mg): dilute in 250 ml of 5% SG and apply (IV) 0.5 to 8 µg/kg/min, adjusting if necessary every 10 minutes;

During and after treatment with rtPA, monitor BP strictly in order to maintain it less than 180x105mmHg [1,6,10].

Intra-arterial thrombolysis

The intra-arterial approach as reperfusion therapy in the acute phase of stroke is an alternative to intravenous thrombolysis. It may have advantages such as the higher thrombolytic concentration in the occlusive thrombus, higher rate of recanalization and possibility of use in patients with contraindication for intravenous chemical thrombolysis. On the other hand, the clinical benefit can be compensated by the longer time interval for the initiation of the intra-arterial procedure and by being performed in a hemodynamic center.

It is a treatment option for selected patients with ischemic stroke less than 6h in duration, due to occlusion of the middle cerebral artery, carotid or basilar (Class II, Level of Evidence B) and the drug used, by extrapolation of the studies of intravenous thrombolysis , is the rtPA (Class IV, Level of evidence C)[10].

Mechanical thrombolysis

In view of the new results of the DEFUSE-3 studies 17 and DAWN 18 the new guidelines recommend mechanical thrombectomy in eligible patients up to 16 hours after an ischemic stroke (Class I, Level of Evidence A) 6 and, based on the results of the DAWN study , the procedure is acceptable in patients with 16 24 hours after stroke (Class II, Level of evidence A)[6].

Eligible patients are those with[6,7]:

- Age ≥ 18 years;
- The onset of symptoms within 6 to 24 hours;
- Occlusion of the internal carotid artery or segment 1 of the middle cerebral artery (M1);
- Prestroke Modified Rankin Score from 0 to 1;
- The score of National Institutes of Health Stroke Scale (NIHSS) ≥ 6; 6.7
- ASPECTS ≥ 6;
- They may receive treatment (puncture in the inguinal region) within 6 hours after the onset of symptoms.

The significant advantage is that patients not eligible for thrombolysis with rtPA IV - for example, those on anticoagulants such as warfarin may still be eligible for mechanical thrombectomy. It is also important to note that thrombectomy and rtPA IV are not mutually exclusive; patients can receive both interventions if necessary. However, the mechanical thrombectomy procedure requires highly qualified professionals with at least two years of intensive training, whether radiologists, neurologists or neurosurgeons.

In Brazil, devices may be used for thrombectomy with the aim of reperfusion in acute ischemic stroke in patients with large vessel occlusion up to 8h after onset of symptoms and who are ineligible for IV thrombolysis or in those in whom IV thrombolysis failed (Class II, Level of Evidence B)[10].

Combined thrombolysis

It aims to associate the advantages of each approach: the ease and speed of administering intravenous thrombolytics with the highest rates of recanalization and potentially better prognosis of intra-arterial thrombolysis.

In individual cases with a high risk of permanent sequelae and persistent arterial occlusion, combined thrombolysis may be offered to patients with informed consent (Class IV, Level of Evidence C)[10]. However, studies have shown that there is no benefit in the functional outcome of using additional endovascular therapy compared with standard rt-PA IV therapy alone[19].

Revascularization

Noninvasive cervical vessel imaging studies are recommended within 24h of admission for patients with moderate or non-disabling ischemic stroke in the carotid artery and who are candidates for carotid endarterectomy or stenting to prevent subsequent ischemic events. If there are no contraindications, it is reasonable to perform revascularization between 48h and seven days of the index event[6].

Carotid endarterectomy

Endarterectomy brings theoretical benefits by removing the source of thromboembolic debris (thus reducing the possibility of recurring events, particularly in the case of plate "soft" or "ulcerated") and restoring normal perfusion pressure to ischemic penumbra in the brain.

For patients with a TIA or ischemic stroke in the last six months and severe stenosis of the carotid artery (70%-99%) ipsilateral documented by non-invasive imaging examination, endarterectomy is recommended that the risk of morbidity and perioperative mortality is estimated in <6%. (Class I, Level of evidence A)[20,21].

For patients with recent AIT or ischemic stroke and moderate ipsilateral carotid stenosis (50%-69%) as documented by MR or CT angiography, endarterectomy is recommended depending on patient-specific factors such as age, sex, and comorbidities, and whether the risk of perioperative morbidity and mortality is estimated to be <6%. (Class I, Level of evidence B) [20,22].

Carotid angioplasty with stent

Carotid angioplasty has emerged as a therapeutic alternative to endarterectomy for the treatment of occlusive disease of the extracranial carotid artery. The proposed advantages of angioplasty are its less invasive nature, reduced patient discomfort, and a shorter recovery period, which reflected improvements in perioperative health-related quality of life[23].

Carotid stent angioplasty is indicated as an alternative to endarterectomy for symptomatic patients with low or medium risk of complications associated with endovascular intervention when carotid artery lumen diameter is reduced by >70% for noninvasive imaging or >50% per image based on catheter and risk of periprocedural mortality is <6% (Class IIa; Level of evidence B)[20,24].

When the degree of stenosis is <50%, endarterectomy and carotid stenting angioplasty are not recommended. (Class III, Level of evidence A)[20].

Conclusion

As an essential cause of death and disability worldwide, the acute treatment of ischemic stroke has advanced rapidly in recent years, improving therapeutic methods and combinations. In clinical practice, recognizing, stratifying and listing, quickly and effectively, the best therapy for stroke patients is paramount.

References

[1] Jauch E, Saver J, Adams H, Bruno A, Connors J, Demaerschalk B et al. Guidelines for the Early Management of Patients With Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2013;44(3):870-947. doi:10.1161/str.0b013e318284056a.

[2] Kernan W, Ovbiagele B, Black H, Bravata D, Chimowitz M, Ezekowitz M et al. Guidelines for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2014;45(7): 2160-2236. doi:10.1161/str.0000000000000024.

[3] Mozaffarian D, Benjamin E, Go A, Arnett D, Blaha M, Cushman M et al. Heart Disease and Stroke Statistics—

2018 Update: A Report From the American Heart Association. Circulation. 2018;137(12):493-493.

[4] Mozaffarian D, Benjamin E, Go A, Arnett D, Blaha M, Cushman M et al. Heart Disease and Stroke Statistics—2016 Update. Circulation. 2015;133(4):38-360. doi.org/10.1161/cir.0000000000000350.

[5] Ministério da Saúde, Secretaria de Atenção à Saúde, Departamento de Ações Programáticas Estratégicas. Diretrizes de atenção à reabilitação da pessoa com acidente vascular cerebral. Brasília: Editora do Ministério da Saúde; 2013. Disponível em:http://bvsms.saude.gov.br/bvs/publicacoes/diretrizes_atencao_reabilitacao_acidente_vascular_cerebral.pdf.

[6] Powers W, Rabinstein A, Ackerson T, Adeoye O, Bambakidis N, Becker K et al. 2018 Guidelines for the Early Management of Patients With Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2018;49(3):46-99. doi:10.1161/STR.0000000000000158.

[7] Powers W, Derdeyn C, Biller J, Coffey C, Hoh B, Jauch E et al. 2015 American Heart Association/American Stroke Association Focused Update of the 2013 Guidelines for the Early Management of Patients With Acute Ischemic Stroke Regarding Endovascular Treatment. Stroke. 2015;46(10):3020-3035. doi:10.1161/str.0000000000000074.

[8] Oliveira-Filho J, Martins S, Pontes-Neto O, Longo A, Evaristo E, Carvalho J et al. Guidelines for acute ischemic stroke treatment: part I. Arq. Neuro-Psiquiatria. 2012; 70(8):621-629. doi:10.1590/S0004-282X2012000800012.

[9] Jauch EC, Cucchiara B, Adeoye O, Meurer W, Brice J, Chan YY, Gentile N, Hazinski MF. Part 11: adult stroke: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care [published correction appears in Circulation. 2011; 124:e404]. Circulation. 2010;122(suppl 3):S818–S828.

[10] Martins S, Freitas G, Pontes-Neto O, Pieri A, Moro C, Jesus P et al. Guidelines for acute ischemic stroke treatment: part II: stroke treatment. Arq. Neuro-Psiquiatria. 2012;70(11): 885-893. doi:10.1590/S0004-282X2012001100012.

[11] Ministério da Saúde, Secretaria de Atenção à Saúde, Departamento de Atenção Especializada. Manual de rotinas para atenção ao AVC. Brasília : Editora do Ministério da Saúde; 2013. http://bvsms.saude.gov.br/bvs/publicacoes/manual_rotinas_para_atencao_avc.pdfMANNING.

[12] Warren J, Daniel E, Gregory L. Atrial fibrillation: Anticoagulant therapy to prevent embolization. [Internet] Uptodate. 2018. [atualizado em 2018 may, citado 2018 apr] https://www.uptodate.com/contents/atrial-fibrillation-anticoagulant-therapy-to-prevent-embolization.

[13] Fernandes A, Andrade A, Cruz C, Oliveira E. Novos anticoagulantes orais (NOACs) na prevenção de acidente vascular encefálico (AVE) e fenômenos tromboembólicos em pacientes com fibrilação atrial. Rev Soc Bras Clin Med. 2015;13(2):98-106. http://files.bvs.br/upload/S/1679-1010/2015/v13n2/a4736.pdf.

[14] Lorga Filho A, Azmus A, Soeiro A, Quadros A, Avezum Junior A, Marques A et al. Diretrizes Brasileiras de Antiagregantes Plaquetários e Anticoagulantes em Cardiologia. Arq. Bras. Cardiol. 2013;101(suppl.3):01-95. doi:10.5935/abc.2013S00.9.

[15] Wang Y, Wang Y, Zhao X, Liu L, Wang D, Wang C et al. Clopidogrel with Aspirin in Acute Minor Stroke or Transient Ischemic Attack. New England Journal Of Medicine. 2013;369(1): 11-19. doi:10.1056/nejmoa1215340.

[16] Rubens J. Prevenção primária da doença cerebrovascular. Diagn Tratamento. 2015;20(3):88-94. http://files.bvs.br/upload/S/1413-9979/2015/v20n3/a4894.pdf.

[17] Albers G, Marks M, Kemp S, Christensen S, Tsai J, Ortega-Gutierrez S et al. Thrombectomy for Stroke at 6 to 16 Hours with Selection by Perfusion Imaging. New England Journal of Medicine. 2018;378(8):708-718. doi: 10.1056/NEJMoa1713973.

[18] Nogueira R, Jadhav A, Haussen D, Bonafe A, Budzik R, Bhuva P et al. Thrombectomy 6 to 24 Hours after Stroke with a Mismatch between Deficit and Infarct. New England Journal of Medicine. 2018;378(1):11-21. doi: 10.1056/NEJMoa1706442.

[19] Abilleira S, Ribera A, Cardona P, Rubiera M, López-Cancio E, Amaro S et al. Outcomes After Direct Thrombectomy or Combined Intravenous and Endovascular Treatment Are Not Different. Stroke. 2017;48(2):375-378. doi: 10.1161/STROKEAHA.116.015857.

[20] Kernan W, Ovbiagele B, Black H, Bravata D, Chimowitz M, Ezekowitz M et al. Guidelines for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2014; 45(7): 2160-2236. doi:10.1161/str.0000000000000024.

[21] Bensenor I, Goulart A, Szwarcwald C, Vieira M, Malta D, Lotufo P. Prevalence of stroke and associated disability in Brazil: National Health Survey - 2013. Arq. Neuro-psiquiatria. 2015;73(9):746-750. doi:10.1590/0004-282x20150115.

[22] Starostka-Tatar A, Łabuz-Roszak B, Skrzypek M, Gąsior M, Gierlotka M. Definition and treatment of stroke over the centuries. Wiad Lek. 2017;70(5):982-987.

[23] Chen C, Ding D, Starke R, Mehndiratta P, Crowley R, Liu K et al. Endovascular vs medical management of acute ischemic stroke. Neurology. 2015;85(22):1980-1990. doi: 10.1212/WNL.0000000000002176

[24] Martins H, Brandão R, Scalabrini A, Velasco I. Medicina de emergências: abordagem prática. 11. ed. Barueri, SP: Manole, 2016.