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TREATMENT PERSPECTIVES FOR POSTERIOR CIRCULATION STROKE



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ABSTRACT

Posterior circulation stroke accounts for about 20% of ischemic strokes. Unlike the anterior circulation, the posterior circulation depends on one main artery. The basilar artery supplies the entire brainstem, the cerebellum, parts of the thalamus on both sides and the occipital lobes. The clinical presentation of basilar artery occlusion is highly variable ranging from transient ischemic attacks (TIAs) or minor stroke to rapidly progressive brainstem dysfunction or coma at onset. These differences in presentation most likely represent differences in etiology. This article focuses on various treatment perspectives for Posterior Circulation Strokes.

KEYWORDS

Stroke TIA Posterior Circulation Ischemic

INTRODUCTION

Posterior circulation stroke accounts for about 20% of ischemic strokes. Unlike the anterior circulation, the posterior circulation depends on one main artery. The basilar artery supplies the entire brainstem, the cerebellum, parts of the thalamus on both sides and the occipital lobes. The clinical presentation of basilar artery occlusion is highly variable ranging from transient ischemic attacks (TIAs) or minor stroke to rapidly progressive brainstem dysfunction or coma at onset. These differences in presentation most likely represent differences in etiology. Basilar artery occlusion caused by atherosclerosis is probably more likely to present with prior TIAs and may do well if the progression of the stenosis is slow enabling collaterals to form, or may do poorly because of failure of collaterals. Embolism from extra cranial vertebral artery stenosis, the heart or the aortic arch might more likely present with a maximum deficit from onset caused by the acuteness of the basilar artery occlusion. Patients with this type of occlusion may do well due to spontaneous recanalization or presence of optimal collateral flow in the absence of atherosclerotic disease or may do poorly because of lack of time to develop sufficient collateralization.

In a disease with such a variety of presentations and etiologies differences in patient selection are likely to influence outcome. Numerous case series have failed to identify a superior treatment strategy in patients with basilar artery occlusion. The overall case fatality rate is 40 to 50% in patients treated with conventional treatment (antiplatelet or heparin), intravenous (IV) thrombolysis or intra-arterial (IA) thrombolysis ^[1-6]. The interpretation of outcome results has been as varied as the outcome results themselves. Data in these case series have been collected retrospectively on small numbers of selected patient using different definitions and treatment strategies. Due to the retrospective nature of these case series data collection has been limited to a few basic items making multivariable analysis impossible. There has been only one attempt to perform a randomized trial (Australasian Urokinase, which was terminated because of a low recruitment rate^[7].

Treatment options in basilar artery occlusion

Due to the absence of a clearly effective therapy, current treatment approaches for acute basilar artery occlusion differ considerably among stroke centers both within and between countries. Many stroke centers use an "all inclusive" treatment protocol for patients with acute stroke that does not distinguish between anterior and posterior circulation stroke, based on results from randomized treatment trials of patients with mainly anterior circulation stroke. There have been several randomized treatment trials studying the efficacy and safety of IV recombinant tissue plasminogen activator (rtPA) in patients with acute ischemic stroke^[8-11]. The results of these trials suggest a potential benefit of IV thrombolysis up to 6 hours from symptom onset, but this benefit is clearly greater the sooner a patient is treated^[11]. IV rtPA received approval from the U.S. Food and Drug Administration (FDA) and conditional approval from the European Agency for the Evaluation of Medicinal Products (EMEA) for the treatment of acute ischemic stroke within 3 hours of symptom onset in 1996. Furthermore, there has been one randomized trial showing the efficacy and safety of IA thrombolysis in anterior circulation stroke within the 0-6 hours time window and several smaller trials showing the feasibility of a combined IV-IA approach^[12-14].

Patients with posterior circulation stroke were either excluded from the randomized IV thrombolysis trials, or their number was small or undetermined ^[8-10]. The largest dataset on outcome of patients with basilar artery occlusion treated with IV thrombolysis is a case series of 50 patients ^[4]. The protocol allowed treatment up to 12 hours from stroke onset for patients with sudden onset of decreased level of consciousness or tetraparesis and up to 48 hours in patients with a progressive stroke with a lesser deficit.

The data available from case series of patients with basilar artery occlusion do not suggest a significant difference in treatment response in patients with posterior circulation stroke as compared with stroke in the anterior circulation. Nevertheless, many centers allow for a 6 to 12 hours treatment delay in patients with basilar artery occlusion resulting in severe neurological deficit and up to several days for patients with a progressive stroke with a lesser deficit. As in anterior circulation stroke, time to treatment is probably the most important determinant of a favorable outcome in patients with basilar artery occlusion. If extension of the treatment window in unselected basilar artery occlusion patients beyond 6 hours offers a benefit, this is likely to be small.

Whether advanced imaging methods will help to identify patients with symptomatic basilar artery occlusion who will benefit from treatment in delayed time windows is unknown. Preliminary data suggest that such a strategy may be useful in anterior circulation stroke patients using diffusion- and perfusion magnetic resonance imaging ^[15-18]. However, these imaging techniques in the posterior circulation are very little explored ^[19].

Current practice Data from the BASICS registry

The ongoing Basilar Artery International Cooperation Study (BASICS) is a worldwide registry of patients with symptomatic radiologically confirmed basilar artery occlusion. The registry started in November 2002. To date 500 patients have been registered by 47

Volume-8 | Issue-8 | August - 2019

participating centers in Europe, North- and South America and Australia.

Clinical presentation

More than 50% of patients had prior warning signs such as transient ischemic attacks, minor stroke or less specific prodromes. Four percent of patients presented with a transient ischemic attack (TIA) as only manifestation of their basilar artery occlusion, 15% with fluctuating symptoms without full recovery in between episodes, 40% with a progressive stroke and 40% with a maximum deficit from onset. Forty-four percent of patients were in a comatose state at time of treatment, 10% locked-in, 9% tetraplegic and 37% had a minor stroke (defined as anything less than comatose, locked-in or tetraplegic).

Treatment received

Fifty percent of patients have been treated with endovascular therapy. Most by IA thrombolysis with or without additional IV Heparin, IV Abciximab, IV thrombolysis, mechanical thrombectomy, or percutaneous transluminal angioplasty with or without stenting. A small number of patients (5%) have been treated by mechanical thrombectomy alone. Thirty-one percent have been treated with antiplatelets and or IV heparin. Only 10% have been treated with IV thrombolysis alone.

Etiology

Most occlusions were thought to be caused by large vessel atherosclerotic disease (37%) or had an embolic cause (32%). Dissection (5%) and vasculitis (< 1%) seem an infrequent cause of basilar artery occlusion.

Outcome

The overall outcome seems comparable to the outcome of previously published case series with a mortality of 46%. Treatment related complications were seen in 9% of patients with symptomatic cerebral hemorrhage (5.6% overall) as the most frequent complication.

Summary

The limited knowledge we have on the outcome in patients with basilar artery occlusion is based on case series and one small randomized trial. Similar outcomes are reported in patients treated with conventional treatment, IV thrombolysis or IA thrombolysis. This does not mean that there is no difference in efficacy among treatment modalities. Due to mayor differences in patient selection and lack of randomization no conclusions can be drawn from these studies in respect to efficacy of treatment. Analyzing the combined outcome results of patients treated with IA thrombolysis it becomes apparent that recanalization is the most important predictor of good outcome. Recanalization was established in 65% of patients treated with thrombolysis. Recanalization is a strong predictor of good outcome (OR 16; CI 6.4-42). Survival is 60% among re-canalized patients and 13% in non-recanalized patients.

The main purpose of acute treatment of patients with basilar artery occlusion should therefore be the recanalization of the blocked artery. Anti-platelets and anticoagulation probably play an important role in the prevention of clot growth, but are unlikely to establish recanalization when given alone. IV thrombolysis in anterior circulation stroke has shown improvement in recanalization rates. However, the treatment modality of choice in patients which such a high mortality and morbidity seems to be the endovascular approach in which the treatment is directly aimed at recanalization and the way of treatment can be adjusted to the specific problem encountered.

REFERENCES-

- Zeumer H, Hacke W, Ringelstein EB. Local intraarterial thrombolysis in vertebrobasilar thromboembolic disease. Am J Neuroradiol 1983;4:401-404.
- Hacke W, Zeumer H, Ferbert A, Bruckmann H, del Zoppo GJ. Intra-arterial thrombolytic therapy improves outcome in patients with acute vertebrobasilar occlusive disease. Stroke 1988:19:1216-1222.
- Arnold M, Nedeltchev K, Schroth G, et al. Clinical and radiological predictors of recanalization and outcome of 40 patients with acute basilar artery occlusion treated with intra-arterial thrombolysis. J Neurol Neurosurg Psychiatry 2004;75:857-862.
- Lindsberg PJ, Soinne L, Tatlisumak T, et al. Long-term outcome after intravenous thrombolysis of basilar artery occlusion. JAMA 2004;292:1862-1866.
- Lindsberg PJ, Mattle HP. Therapy of basilar artery occlusion. A systematic analysis comparing intra-arterial and intravenous thrombolysis. Stroke 2006;37(3):922-928.
 Schonewille WJ, Algra A, Serena J, Molina CA, Kappelle LJ. Outcome in patients with
- Schonewille WJ, Algra A, Serena J, Molina CA, Kappelle LJ. Outcome in patients with basilar artery occlusion treated conventionally. J. Neurol Neurosurg Psychiatry 2005;76:1238-1241.
- Macleod MR, Davis SM, Mitchell PJ, et al. Results of a multicentre, randomized controlled trial of intra-arterial urokinase in the treatment of acute posterior circulation ischemic stroke. Cerebrovasc Dis 2005;20(1):12-17.
 - Hacke W, Kaste M, Fieschi C, et al. Intravenous thrombolysis with recombinant tissue

plasminogen activator for acute hemispheric stroke. The European Cooperative Acute Stroke Study (ECASS). J Am Med Assoc. 1995; 274: 1017-1025.

- Hacke W, Kaste M, Fieschi C, et al. Randomised double-blind placebocontrolled trial of thrombolytic therapy with intravenous alteplase in acute ischaemic stroke (ECASS II). Second European-Australasian Acute Stroke Study investigators. Lancet. 1998; 352: 1245-1251.
- Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Eng I J Med 1995; 333: 1581-1587.
- Hacke W, Donnan G, Fieschi C, et al. Association of outcome with early stroke treatment: pooled analysis of ATLANTIS, ECASS, and NINDS rtPA Stroke Trials. Lancet 2004; 363: 768-774.
 Furlan A, Higashida R, Wechsler L, et al. for the PROACT Investigators. Intra-arterial
- Furlan A, Higashida R, Wechsler L, et al. for the PROACT Investigators. Intra-arterial Prourokinase for acute ischemic stroke. The PROACT II Study: A randomized controlled trial. JAMA 1999;282:2003-2011.
- Lewandowski CA, Frankel M, Toomsick TA, et al. and the EMS bridging trial investigators. Combined intravenous and intra-arterial r-TPA versus intra-arterial therapy of acute ischaemic stroke. Emergency Management of Stroke (EMS) bridging trial. Stroke 1999;30:2598-2605.
- The IMS study investigators. Combined intravenous and intra-arterial recanalization for acute ischemic stroke: The interventional management of stroke study. Stroke 2004;35:5004-912.
- Hacke W, Albers G, Al-Rawi Y, et al. The Desmoteplase in Acute Ischemic Stroke trial (DIAS): a phase II MRI-based 9-hour window acute stroke thrombolysis trial with intravenous desmoteplase. Stroke. 2005; 36: 66-73.
- Ribo M, Molina CA, Rovira A, et 211. Safety and efficacy of intravenous tissue plasminogen activator stroke treatment in the 3-to 6-hour window using multimodal transcranial Doppler/MRI selection protocol. Stroke 2005;36: 602-606.
- Albers GW Thijs VN, Wechsler L, et al. MRI profiles predict clinical response to early reperfusion: the DEFUSE study. Ann Neurol 2006;60:508-517.
- Furlan AJ, Eyding D, Albers GW, et al. Dose Escalation of Desmoteplase for Acute Ischemic Stroke (DEDAS): evidence of safety and efficacy 3 to 9 hours after stroke onset. Stroke 2006;37:1227-1231.
- Ostrem JL, Saver JL, Alger JR, et al. Acute basilar artery occlusion: diffusion-perfusion MRI characterization of tissue salvage in patients receiving intra-arterial stroke therapies. Stroke. 2004; 35: 30-34.