Favorable outcome of acute basilar artery occlusion : A report of two cases

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Abstract

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Abstract

Basilar artery occlusion (BAO) represents 1–4% of ischemic strokes and includes variable symptoms, resulting in delayed diagnosis. Treatment is not codified and outcome depends on the severity of clinical presentation and extent of the occlusion. We present a case of two patients with BAO and we discuss treatment protocols.

Key words : Basilar artery occlusion, Posterior circulation strokes , treatment

Key Clinical Message : codify therapeutic protocol of Basilar artery occlusion

Introduction

Posterior circulation strokes account for about 15% to 20% of all ischemic strokes (1). Basilar artery occlusion (BAO) is a subset of this category, representing 1% to 4% of all ischemic strokes (8). Diagnosing a BAO can be difficult and mortality rate can reach 80%–90% if not promptly treated (4). The treatment of patients with acute BAO is uncertain and prognosis depends on the initial stroke severity and time to therapy .

Here we present two cases of BAO and we discuss treatment in order to codify therapeutic protocol.

Observation :

Case 1 :

A 42-year-old man, without pathological medical history, presented to the emergency with acute left-sided weakness associated with dysarthria on january 2019. He was admitted in stroke unit of our department. On initial examination, vital signs were normal. He was drowsy with limited speech and he had a left hemiplegia. Cranial nerve testing showed bilateral horizontal gaze palsy with a right peripheral facial palsy and an inexhaustible nystagmus. His calculated National Institutes of Health Stroke Scale (NIHSS) was 14 and Modified Rankin Scale (MRS) was 4.

A Stroke Alert was activated. The worsening was rapid, the patient developed swallowing difficulties and emergent intubation was performed. Non contrast head computed tomography (CT) showed hyperdensity of the basilar artery. Brain Magnetic resonance imaging (MRI) with angiography (MRA) revealed hyperintensities in the pons and left cerebellar hemisphere as well as a thrombosis in the basilar artery with extension into the right vertebral artery (Figure 1).

Patient received thrombolysis with i.v. alteplase approximately 17 hours after symptoms onset.

An exhaustive etiological assessment was carried out including a24-hour ECG Holter monitoring and a transthoracic echocardiogram which were normal. Auto immune antibodies were evaluated in serum: anti nuclear antibody (AAN), C-antineutrophil cytoplasmic antibodies (ANCA), anti DNA, anti SSA, SSB, anticardiolipin antibodies were negative.The activities of proteins C and S, antithrombin III, lupus anticoagulants, mutation for Factor V Leiden , MTHFR mutation and Homocysteine level were normal.

Treatment with acenocoumarol (Sintrom) was started 24 hours after thrombolysis. Rehabilitation was initiated. After one year, his clinical condition gradually improved, he has a mild hemiparesis with dysarthria. NIHSS was 5 and MRS was 2.

Case 2 :

A 65-year-old man, with a history of type 2 diabetes mellitus, hyperlipidemia, and hypertension presented to the emergency department with sudden onset of right arm and leg weakness. The patient had gradually worsened and he presented a dyarthria and swallowing difficulty.

On examination, His initial Glasgow Coma Scale score was 13. He was dysarthric and had a flaccid right hemiplegia. Sensation was preserved. The remainder of the neurological examination including cranial nerves was normal. His NIHSS was 10 and MRS was 5.

Brain MRI with angiography revealed a pontine infarction due to an acute basilar artery occlusion (Figure 2).

Anticoagulant treatment was initiated on the third day of symptoms. We first introduced low-dose subcutaneous heparin calcium with activated partial thromboplastin time (aPTT) range between 2 and 3. Acenocoumarol (Sintrom) was introduced a few days later.

The outcome was favorable and the patient resumed walking after 12 months, though he has residual hemiparesis and dysarthria. NIHSS was 6 and MRS was 3.

Discussion :

BAO presents as a subset of the larger category of posterior circulation strokes, associated with a poor prognosis and a high mortality rate carrying $> 80\%$ without treatment (1,4).

The severity of presentation can vary from isolated cranial nerve palsies to tetraplegia, locked-in state, or coma (1).Our patients presented a severe hemiplegia with dyarthria and swallowing difficulty. Brain magnetic resonance imaging (MRI) with angiography is the diagnostic imaging modality of choice which confirm occlusion of the artery.

Once the diagnosis has been obtained, the next goal is to recanalise the artery. However There is no consensus for the best management of BAO for the lack of prospective studies which compare the different therapeutic strategies and identify the most effective (4).

Emergent arterial recanalization has improved functional outcomes and reduced mortality (5,6). Median time to treatment with IV tissue-type plasminogen activator (IV t-PA) was approximately 12 hours (1,5). Some interventionalists may consider revascularization up to 24 hours after symptom onset (7). Our patient recived IV t-PA 17 hours after onset of symptoms. Mechanical thrombectomy is another therapeutic alternative (8,9), which was not carried out for our patients due to lack of resources.

In addition to recanalization therapy, anticoagulation is indicated in these patients to prevent reocclusion of Basilar Artery (10). A study showed an improved outcome in thrombolysed patients treated with heparin (10).

Our protocol was to introduce acenocoumarol (Sintrom) from the first 24 hours after thrombolysis and then overlap with the sintrom. Good functional results were obtained with no increased risk of intracranial hemorrhage.

To conclude, our objective was to try to codify the treatment of BAO in order to improve the vital and functional prognosis of these patients.

Our results join those of previous studies on the delay of thrombolysis which can reach up to 24 hours after the onset of symptoms.

We insist on intensive treatment and we propose to initiate anticoagulant treatment in hyperacute settings to improve outcomes of patients.

Conclusion :

BAO has a poor clinical outcomes with high morbidity and mortality. Arterial recanalization and early introduction of anticoagulant may be associated with good outcomes. Therapeutic decisions must be individualized to improve clinical prognosis.Further research should focus on quicker diagnosis, appropriate imaging, and finding the best treatment strategy for this patient populatio

The authors declare that there is no conflict of interest

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