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Case report

The use of mechanical thrombectomy in the treatment of basilar artery occlusion – case report

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ARTICLE INFO

Article history:
Received 23 September 2014
Accepted 16 July 2015
Available online 29 July 2015

Keywords:
Stroke
Interventional radiology
Thrombectomy
Embolectomy

ABSTRACT

Occlusion of the basilar artery (BAO) is a rare cause of stroke, making up approximately 1% of all cases. Ischemic stroke within the basilar artery is associated with serious complications and high mortality (75–91%). BAO may occur initially in the form of mild prodromal symptoms with neurological disorders, the consequences of which can lead to death. For these reasons, BAO requires rapid diagnosis and treatment. We report the case of a 26-year-old man who suffered basilar artery occlusion and was treated with endovascular therapy. The patient was disqualified from intra-venous thrombolysis and endovascular treatment due to exceeding the therapeutic time window. Despite this, due to the location of ischemia and age of the patient, it was decided to proceed with a mechanical thrombectomy (TM). Vessel patency was restored using the Solitaire FR stent. Treatment continued with antiplatelet therapy. Despite a significant overshoot of the time window the procedure was successful and complete recanalization was achieved. During hospitalization, significant neurological symptom reductions were observed. There is no accurate data on which method of treatment of ischemic stroke is best for BAO. Expectations about the effectiveness of endovascular techniques are high.

Introduction

The basilar artery arises from the combination of the two vertebral arteries at the junction between the medulla oblongata and the pons. BA gives numerous branches to vascularize the brain, they are: anterior inferior cerebellar artery, artery branches to the pons, vestibular artery, superior cerebellar artery and posterior cerebral artery. Occlusion of the basilar artery is a rare cause of stroke, being approximately 1% of all cases [1]. Ischemic stroke in the territory of the basilar artery is associated with serious complications and high

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http://dx.doi.org/10.1016/j.pjnns.2015.07.004
0028-3843/

mortality (75–91%) [2]. Among the risk factors of basilar artery occlusion are: age, sex, hypertension, diabetes, smoking, hyperlipidemia, coronary artery disease, previous ischemic stroke and oral contraception [3]. BAO may present as mild prodromal symptoms, such as dizziness, headache, nausea and vomiting [3]. Other symptoms that may follow are: nystagmus, alternating syndrome, cerebellar syndrome, bulbar syndrome, circulatory and respiratory disorders, which can consequently lead to death. For these reasons, BAO requires rapid diagnosis and treatment. Recanalization of the obstructed vessel is the aim of therapy but there is no accurate data on which method of treatment of ischemic stroke is best in cases of BAO.

Case report

Symptoms

A 26-year-old patient was admitted to Hospital on 9 March 2012 at 2:45 PM. The patient was urgently transferred from another hospital (after undergoing a head MRI-DWI and being diagnosed with a brain stem ischemic stroke), in order to undergo a mechanical thrombectomy. Medical history revealed a plane journey the day before, followed by malaise and neck pain beginning in the evening. The patient woke up around 2 AM with weakend limbs (right side) and while trying to get up fell down. The patient probably suffers from epilepsy – he has been taking Amizepine since he was 3 years old, past medical history also includes hyperlipidemia IIa and hypertension. The patient has a family history, his father also suffered a ischemic stroke. Neurological examination at the time of admission stated: the patient is conscious, somnolent, understands and follows simple commands, meningeal signs absent, anartria with features of lockjaw, a tendency to turn the head and the eyes to the left, peripheral paresis of the right VII nerve, central paresis of the IX, X, XII nerve on the right side, plegia of the right leg with increased spastic muscle tone, right-sided hypoaesthesia, bilateral Babiński, NIHSS of 14 points, GCS 14 points. During the follow-up in A&E, a progression of neurological symptoms was observed – paresis of the left limbs of level 3 on Lovette's scale, NIHSS 18 points. Saturation: 96%; BP: 175/100 mmHg, body temperature: 36.9 °C.

Investigations

In order to confirm the diagnosis, the patient underwent a head CT (in transverse planes after intravenous administration of 95 mL Iomeron 350) at 3 PM (15 min after admission). The scan revealed a deficit of contrast in a segment of the basilar artery, of approximately 2 cm of length. The patient was disqualified from intravenous thrombolysis treatment due to exceeding the therapeutic time window (the duration of symptoms exceeded 4.5 h) and because of the inability to clearly determine the time of the onset of symptoms. Initially, mechanical thrombectomy was also abandoned, also due to exceeding the time window (time over 15 h). At 4:40 PM cerebrovascular digital subtraction angiography (DSA) was performed which showed basilar artery occlusion throughout it's entire length including the branches (Figs. 1–3).

The treatment

Despite contraindications, the patient underwent a mechanical thrombectomy procedure, which started at 5:20 PM. The procedure was carried out with a catheter entering the right femoral artery using the Seldinger method. A microcatheter was introduced coaxially, through which a thrombectomy Solitaire FR device was placed in the artery. The stent was inflated above the thrombus, without complete release. Then, together with the thrombus, it was pulled through the guiding catheter. During the procedure constant washing with a heparin solution in normal saline was performed, while guiding both the catheter and microcatheter. We managed to get patency in the closed segment of the basilar artery. During the procedure an Abciximab infusion was administered, which continued in the neurology ward. The procedure was performed under general anesthesia.

Control after treatment

A control DSA performed after treatment revealed the left vertebral artery and the basilar artery with its branches. The posterior brain artery was non-contrasting in the DSA but was visible in an angiography of the right common carotid artery. The primary efficacy endpoint of cerebrovascular recanalization was assessed using the TICI scale (Thrombolysis in Cerebral Infarction: 0 – no flow; 1 – the contrast agent fills segment beyond embolic trace reperfusion; 2a – partial reperfusion, 2/3 of the total area of proper vascularization; 2b – a total reperfusion but slower filling; 3 – complete reperfusion), a score of 3 was achieved. The next day a control angio-CT of the head was performed, which showed an unobstructed basilar artery throughout its course. Segmental occlusions of the BA were visible in the middle part (6.6 mm of length) and also just before the upper cerebellar branch (3 mm of length). A head CT without contrast describes the findings as hypodense, ischemic, 28 mm in diameter in the left cerebellar hemisphere and 11 mm in diameter in the left side of the brain stem. A head MRI was performed – SE/T1, T2, FLAIR, DWI sequences in transverse planes; T2 and FLAIR CUBE sequences in sagittal planes (layer thickness: 5 and 3 mm). Within the central and left part of the pons, ischemic changes (subacute/chronic) were revealed and similar changes, but of a settled character, were revealed within the lower medial part of the cerebellar hemisphere (Figs. 4 and 5).

The patient's condition after treatment

On the first day after treatment a neurological examination showed dysarthria, peripheral paresis of the right VII cranial nerve, central paresis of the IX, X, XII cranial nerves on the right side, tetraplegia with paresis of the right limbs (0/1 on Lovette's scale in the upper limb, 2 on Lovette's scale in the lower limb), paresis of the left limbs (3 on Lovette's scale), right-sided hypoaesthesia, bilateral positive Babinski. The patient's condition assessed in NIHSS was rated at 16 points. On 28th March 2012 the patient was discharged from the ward and moved to the Department of Neurological Rehabilitation SP CSK, still presenting mild dysarthria, minor peripheral paresis of the right VII cranial nerve and a retreating pyramidal syndrome of the right hand, paresis of the right upper limb