Semantic aphasia in a sonothrombolysed patient. A treatment without use of rt-PA

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Summary  The objectives were to describe a case of a patient with acute ischemic stroke who achieved recanalization of the occluded middle cerebral artery and a good clinical improvement after immediate application of standard medical treatment (including aspirin) plus sonothrombolysis for 2 h without use of rt-PA. After the first 24 h, a rare form of aphasia, a semantic type of aphasia was described. On day 5, the NIHSS score of the patient was 2, on day 30, his NIHSS and modified Rankin score was 0. The main factors influencing favorable outcome were discussed. Despite controversial evidence, it is worth studying the efficacy and safety of sonothrombolysis without rt-PA, but with commonly used drugs. This approach could be easily applied in eligible patients almost in every stroke unit worldwide.

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Introduction

The ultrasound energy could increase the intrinsic fibrinolysis in occluded vessels even in the absence of thrombolytic agents, such as alteplase (rt-PA) [1–5]. The results of CLOT-BUST clinical trials showed that the sonothrombolysis (STL) or the combination of rt-PA plus 2 h of continuous transcranial Doppler (TCD) was able to increase recanalization rates in acute ischemic stroke (AIS), with a trend toward better functional outcomes compared to rt-PA alone [6]. Spontaneous recanalization of the middle cerebral artery (MCA) in AIS was observed despite a poor initial thrombolysis in Brain Ischemia (TIBI) score in some patients who were not eligible for (or not treated with) thrombolysis (TL) using rt-PA [2,7–10]. The factors leading to spontaneous recanalization, its natural rate, and whether STL without rt-PA should be worth doing in large clinical series or is potentially ineffective, are still unresolved questions.

The objectives of the current report were to describe a case of a patient who suffered AIS and achieved good clinical improvement after immediate application of standard medical treatment plus STL for 2 h (transcranial color-coded duplex sonography) without use of rt-PA.

Materials and methods

A 59-year old patient presented in the emergency room with right hemiplegia and complete motor aphasia 1.5 h after he was found earlier in the morning. The exact time of his stroke onset was not known, but according to his wife, it was definitely between 2 and 6 h before the admission.
On admission, the patient had a NIHSS score of 10, and blood pressure of 150/80 mm Hg. From the past medical history, he had only mild arterial hypertension, treated with beta-blocker. The routine blood tests, ECG, and conventional computed tomography (CT) of the head were carried out (according to the Bulgarian AIS guideline). When the diagnosis of AIS was accepted after CT, the patient was given immediately aspirin 300 mg, bisoprolol 5 mg, atorvastatin 10 mg orally; normal saline 0.9% 1.5 L, pyracetam (Nootropil) 3.0 g intravenously; and fraxiparine 0.4 mL subcutaneously. The values of his complete blood count, biochemistry and lipids panel were within normal limits, except for the leucocytes count of 13.3 $\times 10^9/l$ and hematocrit of 0.54.

The extracranial and transcranial color-coded duplex sonography (TCCDS) was performed after admission in the stroke unit. The exact vessel pathology and hemodynamic status were defined in accordance to TIBI and COGIF criteria[7,11—14] using General Electric Vivid 7 Pro diagnostic TCCDS device. The extracranial ultrasound showed mild atheromatous carotid changes with higher intima—media thickness complex values bilaterally and several small hypoechoic nonstenotic plaques around the bifurcations. On TCCDS, there was evidence of occlusion in the left MCA, its proximal M1 segment. Signals from corresponding A1, P1 and C1 segments of the basal cerebral arteries were recorded as needed [13]. The monitoring of intracranial velocities and STL of the left MCA were done by manually holding a 2-MHz transducer for 2 h via transtemporal window (sample volume 5 mm, low velocity scale; broad color duplex window to be able to see A1, C1, P1 arteries; mild oscillations and adjustments along the course of affected M1 segment). The neurologic and hemodynamic status of the patient was assessed after 3, 6, and 12 h, as well as on the 1st, 3rd, 5th, 30th, and 90th day by a neurologist, speech therapist and neurosonologist.

**Results**

On starting TCCDS monitoring (minute 0), there was not any detectable signal from the initial M1 segment of MCA, TIBI scale score 0. From minute 30 (Fig. 1) until minute 120, a minimal, spike-like MCA flow occurred, with peak systolic/end diastolic velocity (PSV/EDV) 20/5 cm/s, and TIBI score was between 1 and 2. Respectively, TIBI 3 was detected on the sixth hour with a dampened MCA flow pattern and PSV/EDV 50/24 cm/s. On the first day, MCA PSV/EDV were 68/26 cm/s (Fig. 2). On the third day, there was no significant asymmetry between the two MCA arteries, with PSV/EDV of 76/40 cm/s, TIBI 5 score. On day 30, blood flow velocities of both MCAs were within normal range (Figs. 3—7).

The patient’s neurological status improved on the first day after admission. His NIHSS score changed from 10 to 5, and his complete motor aphasia resolved to a semantic one. This particular and rare type of aphasia was characterized by impaired simultant gnosis, comprehension of time relations, and constructions of affiliations, such as “‘my father’s brother is my...”. This rare type of aphasia (described first by Russian neuropsychologist A.R. Luria) is not included in all modern classifications. It is thought to be a part of the sensory aphasia [15].

On discharge at day 5, the NIHSS score of the patient was 2. On the follow-up at the first month the patient achieved complete neurological recovery. At the third month his modified Rankin Scale was 0.

**Discussion and conclusion**

The acute occlusion of the proximal MCA segment is a dangerous event that could lead to a large brain infarct, severe disability, and poor functional outcome in cases with no recanalization [9,10]. That is why we applied STL 3.5—6 h after the onset of AIS. We observed the evolution of recanalization in the occluded MCA within the first 6—12 h after the procedure [8,16]. In the presented case, two rare clinical situations deserve attention. First, the favorable outcome was achieved using simple and non-expensive treatment including STL without rt-PA. Second, there was a quick resolution of an almost complete motor aphasia to a specific semantic type that was connected to the location of the brain ischemic changes in MCA territory and their functional compensation.

The quick clinical improvement of our patient could be a result of mere chance for spontaneous recanalization [9,10]. We believe that early aspirin administration (immediately
after CT, and approximately 3.5 h after the stroke onset) was an important factor for the disease evolution and long-term prognosis [17]. Based on the evidence from a single observation, an impelling suggestion to accept that 2 h of STL combined with 300 mg aspirin given orally contributed to the mechanical breakdown of thrombus and recanalization in the left MCA. The data from a clinical study showed evidence toward better recanalization rate in patients not eligible for IV rt-PA [2,8]. However, the role of other medications given to those patients, their dosages and time of administration along with the STL was not clear as a contributing factor.

In some acute clinical presentations with unknown time of stroke onset, still there are no reliable criteria to distinguish who would benefit from TL and who would benefit from STL. Perhaps, younger age, lower initial NIHSS score, MRI mismatch and TIBI scale or COGIF grade improvements could be used as favorable prognostic parameters [4,11−13,16,18].

Despite controversial evidence in literature [1,2,5,10−12], we think that it is worth studying the efficacy and safety of this simple method, TCCDS STL without TL agent, but with aspirin or other commonly used drugs. This approach could be easily applied in eligible patients almost in every stroke unit facility worldwide.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.permed.2012.03.018.

References
