

Metabolic Changes after Revascularization in a Patient with Innominate Artery Occlusion by Localized in vivo Proton Magnetic Resonance Spectroscopy

Localized in vivo proton magnetic resonance spectroscopy ($^1\text{H-MRS}$) has been used to measure the metabolic status of the human brain in a non-invasive manner; thus, it is often called "a non-invasive biochemical assay". MRS is more sensitive than magnetic resonance imaging (MRI) in detecting ischemic damage by measuring the metabolic changes that occur prior to the anatomic changes. We report a patient who presented with innominate artery occlusion and symptoms of posterior circulation insufficiency and showed favorable metabolic changes by $^1\text{H-MRS}$ after revascularization. He showed no visible lesion in brain MRI, but in $^1\text{H-MRS}$, decreased N-acetylaspartate (NAA) signal was noted in a resting state. After revascularization, both symptomatic improvement and recovery of NAA signal were observed. $^1\text{H-MRS}$ may provide valuable clinical information in diagnosis and management of cerebral hypoperfusion at a much earlier stage prior to the anatomic changes.

Key Words : *Magnetic Resonance Spectroscopy; Brachiocephalic trunk; Arterial Occlusive Diseases; Reperfusion*

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INTRODUCTION

Although occlusive lesions of the arch vessels are not uncommon among patients with atherosclerosis, these lesions are encountered less frequently than carotid bifurcation lesions and account for fewer than 5% of the surgical procedures performed on the cerebral vasculature. Most patients with arch vessel lesions are asymptomatic, because of the rich collateral circulation of the head and shoulder girdle. When symptoms occur, they are usually flow-related, and are quite variable. Neurological symptoms are most common and are related to the degree of posterior circulation insufficiency in patients with a subclavian or innominate artery disease.

Localized in vivo proton magnetic resonance spectroscopy ($^1\text{H-MRS}$) has been used to measure the metabolic status of the human brain in a non-invasive manner; thus, it is often called "a non-invasive biochemical assay" (1). MRS is more sensitive than magnetic resonance imaging (MRI) in detecting ischemic damage by measuring the metabolic changes that occur prior to the anatomic changes. Therefore, $^1\text{H-MRS}$ can aid the diagnosis and management of cerebral hypoperfusion at a much earlier stage.

This report describes a patient with innominate artery occlusion and symptoms of posterior circulation insufficiency who showed metabolic changes by $^1\text{H-MRS}$ after revascularization.

CASE REPORT

A 61-yr-old male was admitted to our department because of intermittent dizziness, blurred vision, and right arm claudication of 6 yr duration. He had never suffered from a cerebral event. MRI, carotid duplex scan (ATL HDI 3000: Advanced Technology Laboratories High Definition Imaging 3000, Bothell, Washington, U.S.A.), and four-vessel cerebral angiography studies were undertaken to assess the posterior cerebral circulation. The MRI showed no visible lesion. Carotid duplex scan showed a damped waveform and markedly decreased volume flow of the right common carotid artery (60 mL/min) and reversed flow of the ipsilateral vertebral artery (Fig. 1). Four-vessel cerebral angiography exhibited complete occlusion of the innominate artery and a retrograde flow from the right vertebral artery into the common carotid and subclavian artery (Fig. 2). Duplex and angiographic findings were consistent with the subclavian steal syndrome caused by complete occlusion of the innominate artery. $^1\text{H-MRS}$ was performed on a GE 1.5T SIGNA system equipped with shielded gradients and proton brain examination (PROBE) package (General Electric Medical System, Milwaukee, U.S.A., Version 5.4) in a resting state, after informed consent. Image-guided stimulated echo acquisition mode (STEAM)-spectra were obtained from 7-10 mL voxels in the middle cerebral artery territory and cerebellum of both hemispheres (Fig. 3A).

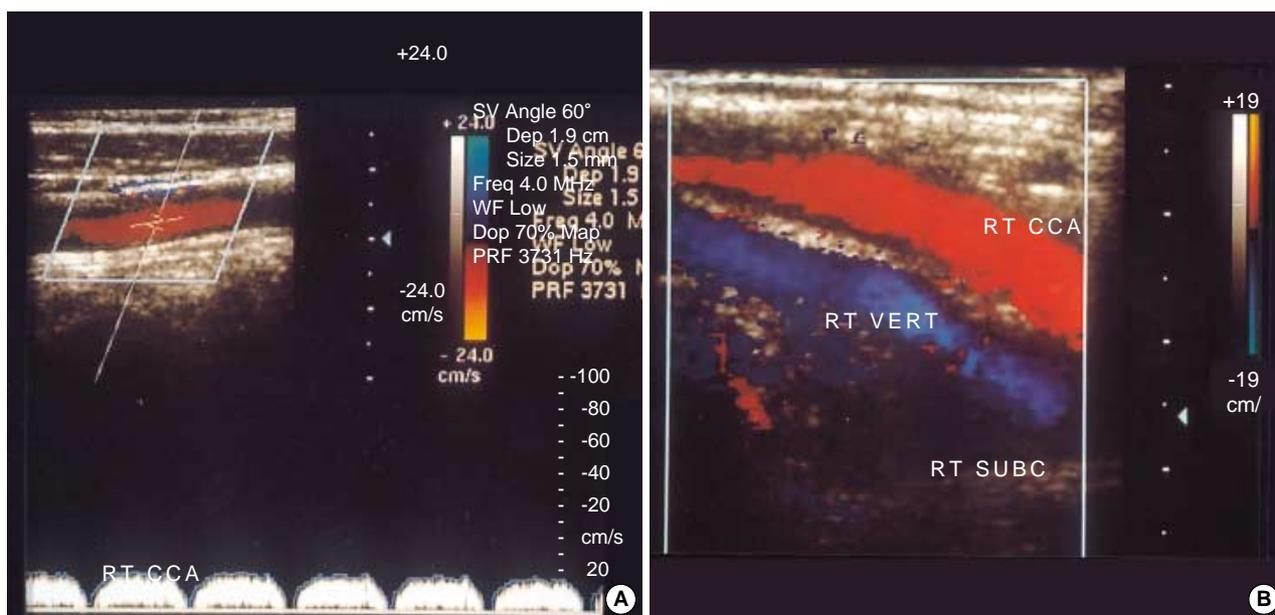


Fig. 1. Preoperative carotid duplex scan shows (A) a damped waveform and markedly decreased volume flow of the right common carotid artery (RT CCA), and (B) reversed flow of the ipsilateral vertebral artery (RT VERT).

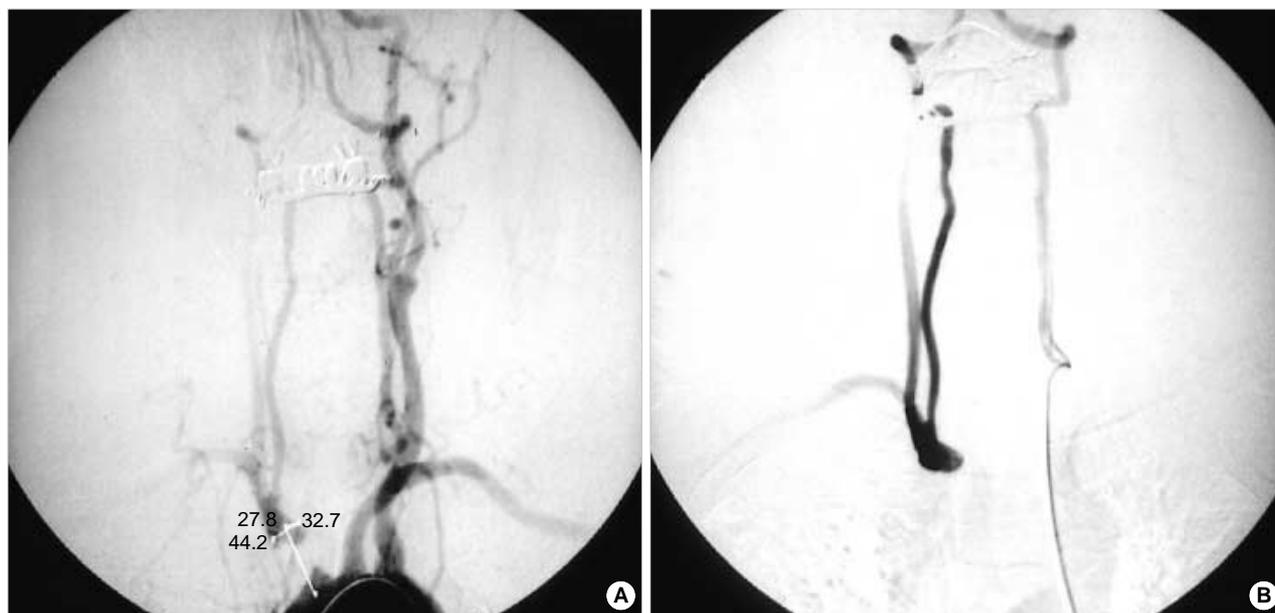


Fig. 2. Transfemoral four-vessel cerebral angiography shows (A) complete occlusion of the innominate artery and (B) retrograde flow from the right vertebral artery into the common carotid and subclavian artery.

Fig. 3B demonstrates the ^1H -MR spectrum acquired from the right cerebellum. The spectrum showed a decreased N-acetylaspartate (NAA) signal as compared to the contralateral side (Table 1). Axillary-to-axillary bypass with 8-mm externally supported polytetrafluoroethylene (PTFE) graft was performed without complications. At 10 days after surgery, carotid duplex scan and ^1H -MRS were done for the evaluation of hemodynamic changes and the metabolic status of the

right cerebellum. Postoperative carotid duplex scan showed a normal arterial waveform of the right common carotid artery with a markedly increased volume flow (400 mL/min) and a to-and-fro waveform of the ipsilateral vertebral artery (Fig. 4). Postoperative ^1H -MR spectra were acquired from the same region of the middle cerebral artery territory and cerebellum of both hemispheres. The spectra showed increased NAA values in the right middle cerebral artery territory and cerebel-

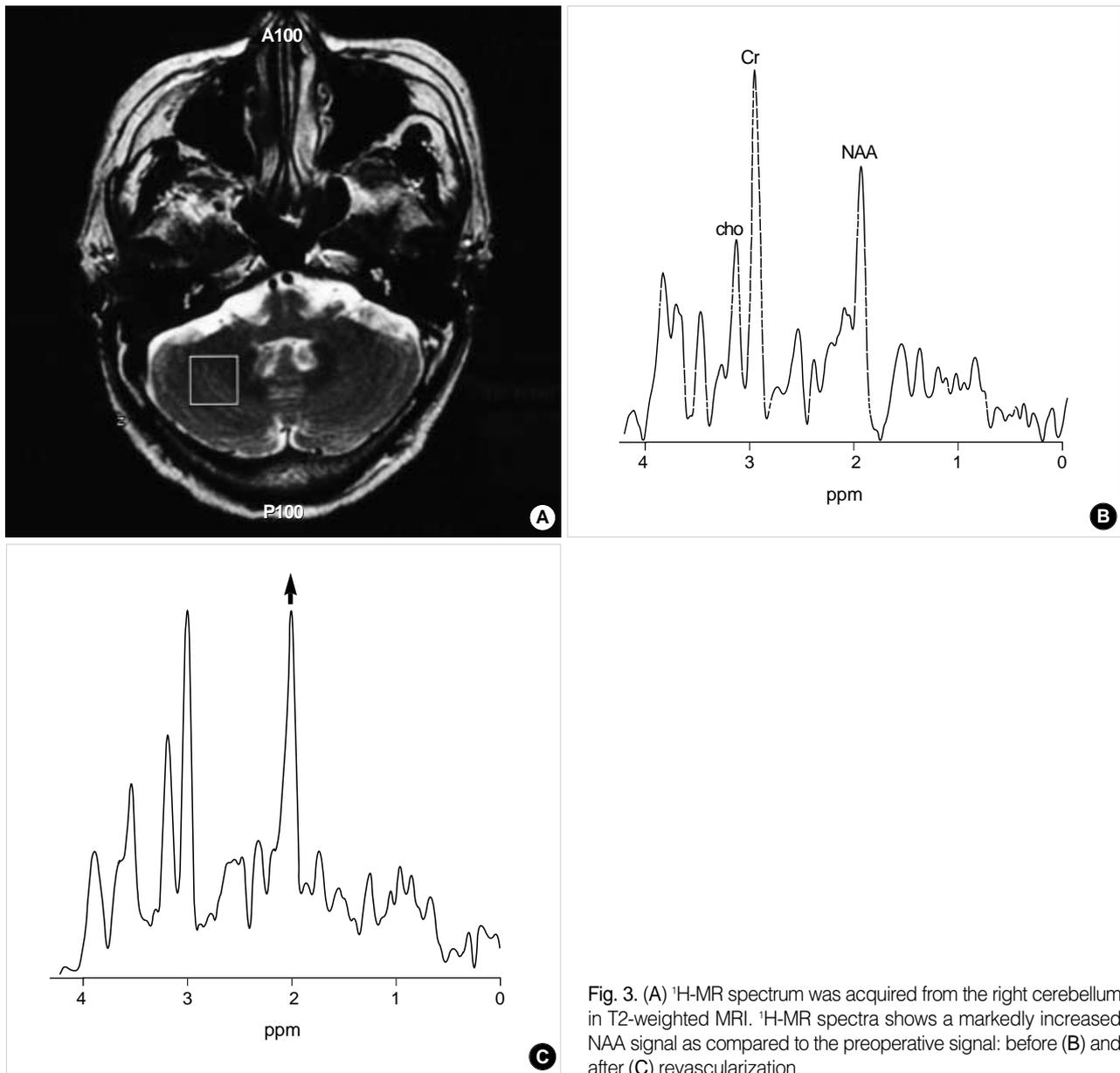


Fig. 3. (A) ¹H-MR spectrum was acquired from the right cerebellum in T2-weighted MRI. ¹H-MR spectra shows a markedly increased NAA signal as compared to the preoperative signal: before (B) and after (C) revascularization.

lum as compared to the preoperative values (Fig. 3C, Table 1).

DISCUSSION

Neurospectroscopy is defined as the field of study resulting from MRS examination of the human brain (1). It measures neuronal markers, energy and redox states, specific fuels of tissue respiration, maturation, and possibly myelination. It provides diagnostic patterns of altered neurochemistry. Positron emission tomography (PET), and to a lesser extent single photon emission computed tomography (SPECT), magnetic resonance angiography (MRA), functional magnetic resonance imaging (fMRI), and “diffusion-imaging” address

blood flow, glucose turnover, and oxygen consumption. However, until the advent of nuclear magnetic resonance, no direct noninvasive assay of the cerebral metabolites was available. There was no neuronal marker, no astrocyte marker, and no technique to directly determine the energy metabolism. These gaps are now filled by neurospectroscopy. With increased clinical experience of neurospectroscopy, an increasingly useful metabolic information emerges. Single-voxel ¹H-MRS can measure the cerebral metabolites for patients with various diseases (1-3).

Of the many expected and new resonances now identified in the clinical practice of neuro-MRS, none has created more diagnostic information than NAA. Multiple lines of evidence suggest that NAA can be used as a neuronal marker as it is

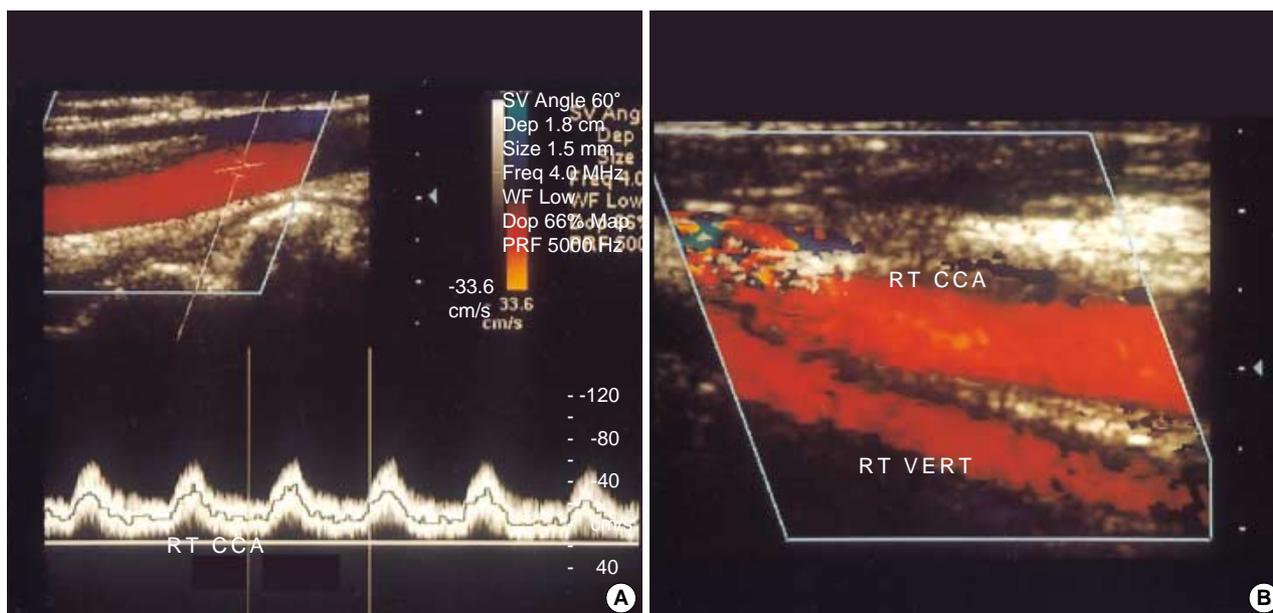


Fig. 4. Postoperative carotid duplex scan shows (A) a normal arterial waveform and a markedly increased volume flow of the right common carotid artery (RT CCA) and (B) a to-and-fro waveform of the ipsilateral vertebral artery (RT VERT).

Table 1. Metabolic changes in the middle cerebral artery territory and cerebellum of both hemispheres before and after revascularization procedure

Metabolite ratio	Rt MCA	Lt MCA	Rt Cbll	Lt Cbll
NAA/Cr, pre-	1.22	1.31	0.71	0.93
post-	1.32	1.31	0.97	0.98
Cho/Cr, pre-	0.69	0.75	0.49	0.64
post-	0.59	0.70	0.63	0.60
ml/Cr, pre-	0.58	0.57	0.32	0.39
post-	0.46	0.41	0.55	0.35

MCA; middle cerebral artery territory, Cbll; cerebellum, pre-; pre-revascularization, post-; post-revascularization, NAA; N-acetylaspartate, Cho; Choline-containing compounds, Cr; Creatine, ml; myo-Inositol.

found exclusively in neurons (4-6), and their processes in the mature brain were well previously investigated (7). In human brain spectra, *in vivo* NAA is reduced in situations known to be associated with neuronal loss (8, 9). It can begin to fall as early as two hours after the onset of focal infarction (10). The continued NAA decline over the first two weeks after stroke does not merely reflect clearing of debris from neurons killed at the initial outset, but it is an evidence of continuing neuronal loss after the acute period, possibly, in the ischemic penumbra (4). Reversible decreases in NAA emphasize that neuronal dysfunction or transient relative volume changes can also lead to a decreased level of NAA (3, 4, 11). Although we do not think that the increased NAA means the creation of new neurons, the postoperative NAA/Creatine (Cr) ratio showed a significant increase than the preoperative value, endorsing the authors' contention that the improved cerebral perfusion after revascularization will positively affect the

hemodynamically compromised brain, e. g., better survival of neuronal cells in the ischemic penumbra. Total Cr concentration is known to be relatively constant throughout the brain and tends to be relatively resistant to changes for many metabolic diseases. Therefore, it may be used as an internal reference for the other metabolic peaks of the ^1H -MR spectrum (1). The concentration of myo-Inositol (mI) fluctuates more than any of the other major compounds detected in the proton spectrum. While mI has been recognized as a cerebral osmolyte and its cellular specificity is believed to be that of an astrocyte marker, evidence is incomplete. The resonance intensity of mI has been labeled as a breakdown product of myelin, but the evidence is particularly indirect and rather weak on this point (1).

The term subclavian steal syndrome has been used to describe any condition in which there is reversal of vertebral blood flow distal to a subclavian artery lesion, and may be applied when cerebral symptoms are produced by exercise of the affected upper extremity. It is presumed that under these circumstances, the collateral circulation to the arm becomes insufficient. The decrease in the blood pressure in the vertebral system results in a "steal" of the blood from the intracranial circulation via the basilar artery. The decreased perfusion pressure in the intracranial circulation causes the transient neurological symptoms. Cessation of the arm exercise stops or lessens the "steal" from the intracranial circulation.

We described ^1H -MRS findings in a patient with innominate artery occlusion and symptoms of subclavian steal syndrome, before and after revascularization. He showed no visible lesion in brain MRI, but in ^1H -MRS, a decreased NAA signal was noted in a resting state. After revascularization, both

symptomatic improvement and recovery of NAA signal were observed. ¹H-MRS may provide valuable clinical information in diagnosis and management of cerebral hypoperfusion at a much earlier stage prior to the anatomic changes.

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