Monitoring of the lateral spread response in the endovascular treatment of a hemifacial spasm caused by an unruptured vertebral artery aneurysm

Case report

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The lateral spread response (LSR) is used in the electrophysiological diagnosis of a hemifacial spasm or for monitoring during microvascular decompression. The authors used LSRs for intraoperative monitoring during endovascular surgery in a rare case of vertebral artery (VA) aneurysm that caused intractable hemifacial spasm.

A 49-year-old woman presented with a right hemifacial spasm that had persisted for 9 months. No other clinical symptom was observed. Vertebral artery angiography revealed a saccular aneurysm of the right VA. Magnetic resonance (MR) imaging demonstrated that the aneurysm was compressing the root exit zone of the right facial nerve. Endovascular treatment of the VA aneurysm was performed while monitoring the patient’s LSRs. During occlusion of the VA at sites distal and proximal to the aneurysm, the LSRs temporarily disappeared and then reappeared with a higher amplitude than those measured preceding their disappearance. The hemifacial spasm alleviated gradually and disappeared completely 6 months after treatment. The LSRs changed in parallel with the improvement in the patient’s hemifacial spasms and eventually disappeared. No recurrence of symptoms has been noticed as of 18 months postoperatively.

This is the first report of the use of LSR monitoring during endovascular surgery for an intracranial aneurysm that causes hemifacial spasm. Intraoperative and postoperative changes in the LSRs provided useful information regarding the pathophysiology of hemifacial spasm.

KEY WORDS: endovascular therapy • hemifacial spasm • lateral spread response • unruptured aneurysm • vertebral artery
eventful. Her hemifacial spasm alleviated gradually and disappeared completely 6 months after treatment. The amplitude of the LSRs also decreased gradually at 1 and 3 months after the procedure, in parallel with alleviation of her hemifacial spasm, and disappeared 6 months postoperatively. Follow-up MR imaging depicted the gradually decreasing size of the aneurysm. At the same time that the patient’s hemifacial spasm and LSRs disappeared, MR images confirmed that the aneurysm had retracted completely from the facial nerve (Fig. 3). No recurrence of symptoms has been noticed 18 months after the endovascular procedure.

**Discussion**

Hemifacial spasm is usually caused by compression of the facial nerve, usually by branches of the VA and basilar artery, or by the elongated VA itself. Gross pathological lesions such as tumors, arteriovenous malformations, or aneurysms in the cerebellopontine angle can also cause hemifacial spasm. A VA aneurysm is a rare cause of hemifacial spasm. Although placement of a clip on the aneurysm has generally been performed as treatment for hemifacial spasm, two groups have recently reported on patients who underwent endovascular surgery for aneurysms causing hemifacial spasm. In one report, endovascular proximal obliteration of the VA for hemifacial spasm, caused by a dissecting aneurysm located at the vertebrobasilar junction, resulted in the gradual improvement and disappearance of the patient’s hemifacial spasm. In the other report, a fusiform aneurysm of the left VA compressed the RExZ of the facial nerve, resulting in a good outcome. In our patient, a saccular VA aneurysm compressed the facial nerve, leading to intractable hemifacial spasm. Previously investigators have shown that intraaneurysm embolization of unruptured VA aneurysms can also cause hemifacial spasm.
Lateral spread response during endovascular surgery for aneurysm

For an aneurysm that compresses the facial nerve, the clinical course after endovascular treatment is obviously different from that after direct surgery. Previous reports indicate that most patients experience immediate relief of symptoms after direct surgery. Besides the present case there have been only two other reported cases of endovascular treatment for hemifacial spasm. Together these three patients experienced a gradual improvement over time, culminating in complete disappearance of their symptoms approximately 6 months after treatment. It is conceivable that the gradual decrease in facial nerve compression after intravascular treatment of the aneurysm parallels the gradual improvement in the abnormal excitatory circuit at the RexZ, thus resulting in alleviation of the hemifacial spasm. Indeed, both the hemifacial spasm and the LSRS in our patient completely disappeared at almost the same time that MR imaging revealed shrinkage of the aneurysm away from the facial nerve.

To our knowledge, this is the first report of the use of the LSRS as a monitoring tool during endovascular surgery for an intracranial aneurysm that causes hemifacial spasm. It is intriguing that the LSRS changed dramatically during occlusion at sites distal and proximal to the VA. These notable changes in the responses may be attributed to subtle changes in blood pressure against the aneurysm wall during VA embolization at the site distal or the one proximal to the aneurysm affected the compression strength against the facial nerve, consequently leading to changes in the LSRS waveforms.

Some debate continues regarding whether the LSRS originate from the facial motor nucleus or from peripheral compression sites. Recently, we used a double-stimulation technique to show that the LSRS probably arise from vascular compression sites where a cross-transmission response seems to occur, rather than from facial motor neurons. Additionally, we treated a patient whose LSRS disappeared after decompression of the offending vessel and reappeared after replacement of the vessel. In the present case the LSRS dramatically changed while the VA was being trapped by intravascular embolization. If the site of abnormal cross-transmission is in the facial nucleus, the LSRS should not change immediately in response to manipulation of the offending vessel or aneurysm causing the hemifacial spasm. These findings support the cross-transmission theory regarding the pathophysiological origin of LSRS.

References


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Fig. 3. Magnetic resonance image obtained 6 months after surgery, demonstrating the decreased size of the aneurysm as it shrinks away from the facial nerve.