

Anesthesiology
80:1396-1398, 1994
© 1994 American Society of Anesthesiologists, Inc.
J. B. Lippincott Company, Philadelphia

Successful Treatment of Meige's Syndrome with Facial Nerve Block

Atsuko Kobayashi, M.D.,* Mitsuyoshi Lee, Ph.D., M.D.,† Yoshifumi Tanaka, Ph.D., M.D.‡

MEIGE'S syndrome, first described in 1910,¹ is an idiopathic dystonic disorder that consists of blepharospasm and oromandibular dystonia. Patients usually are middle-aged or elderly, and most are women. We report a case of Meige's syndrome with severe bilateral blepharospasm successfully treated with facial nerve block.

Case Report

This 70-yr-old woman complained of having had difficulty opening her eyes for the past 4 yr. Symptoms started with frequent blinking and gradually progressed to severe continuous blepharospasm that rendered the patient functionally blind. The blepharospasm, which fluctuated in intensity from day to day, was aggravated by emotional stress, bright light, television, or wind in the eyes. The movements disappeared during sleep. When we first evaluated the patient, the blepharospasm was almost continuous, and prolonged spastic contractions occurred in the oromandibular and the forehead muscles. The patient occasionally made pursing and smacking movements with her mouth, and her tongue intermittently protruded, with tremors. She was mildly depressed. There was no family history of similar movement disorders or of neurologic or psychiatric illness.

Computed tomographic scanning and magnetic resonance imaging indicated slight atrophic changes in the brain and some lacunae at the basal ganglia. Results of laboratory evaluation by electroencephalography, skull roentgenograms, and routine chemistry, hematology, and copper studies were normal. Results of analysis of cerebrospinal fluid for protein, glucose, and cell count also were normal. No abnormal reflexes were present. Meige's syndrome was diagnosed.

Anticholinergic (trihexyphenidyl) and γ -aminobutyric acid antagonists (valproate and clonazepam) and a norepinephrine agonist

(Droxidopa) had no effect. Antidopaminergic therapy (haloperidol) aggravated the symptoms.

After the blepharospasms were relieved with 0.5 ml 0.25% bupivacaine, bilateral block of the zygomatic branch of the facial nerve, developed by O'Brien,² with 0.5 ml 99.5% ethanol was administered. Because the blepharospasm recurred, the same block was repeated five times on each side. Relief of symptoms lasted for 2-3 weeks after each block.

Although symptoms on the right side improved remarkably, blepharospasm on the left side was still severe. A method of needle insertion into the facial nerve trunk from the stylomastoid foramen, developed by Wakasugi,³ on the left side was used. A 22-G, 5-cm needle, having a bevel angle of 30° against the median line, was inserted at a point about 0.5 cm anterior to the tip of the mastoid process. Laterally, the needle was inserted parallel to the forehead-philtrum line. The needle was moved forward 3 cm until pain in the distribution of the facial nerve occurred, at which time a facial palsy occurred and the blepharospasm ceased. The patient was unable to close her eyes or to whistle. The needle was maintained without any movement or any injection for 20 min. Because facial palsy without recurrence of the spasm still was observable after 20 min, no additional injections were administered, and the needle was withdrawn. The patient subsequently had no difficulty opening her eyes.

The patient has proceeded to lead a normal life. Although weakness and paresthesia initially persisted on the edge of the left side of the mouth, these symptoms resolved within 1 month, and relief of blepharospasm has thus far lasted for 7 months.

Discussion

As described by Meige,¹ median facial spasm is characterized chiefly by symmetric dystonic spasms of the facial muscles. The initial spasms usually involve the orbicularis oculi, are dysrhythmic and irregular, and have a tonic appearance. They may last for seconds or even minutes. Later, there is involvement of most of the facial muscles. Spasms are associated with retraction of the mouth, and opening of the jaw can be quite prominent. The dystonia fluctuates considerably from day to day, is aggravated by stress, is improved by sedation, and disappears during sleep.

The features of Meige's syndrome as established by Marsden⁴ include (1) unknown etiology, (2) peak age

* Assistant Professor.

† Chair, Department of Anesthesiology, Osaka-fu Saiseikai Suita Hospital, Suita City, Osaka, Japan.

‡ Professor.

Received from the Department of Anesthesiology, Kyoto Prefectural University of Medicine, Kyoto, Japan. Accepted for publication February 7, 1994.

Address reprint requests to Dr. Kobayashi: 465 Kajicho, Kawarachi-Hirokoji, Kamigyō-ku, Kyoto, 602, Japan.

Key words: Anesthetic techniques, regional: facial nerve block. Muscle, dystonia: blepharospasm; Meige's syndrome.

CASE REPORTS

of onset in the 6th decade, (3) greater frequency in women than in men, (4) no history of neurologic or psychiatric illness (although some patients, such as ours, are depressed at the onset of illness) and no history of exposure to neuroleptic medication, (5) no family history of a similar illness, (6) a chronic and disabling course, (7) no consistent abnormalities found during examinations and laboratory evaluations, (8) no intellectual, extrapyramidal, cerebellar, or sensory deficits developing during the course of the illness, and (9) lack of spontaneous remission and a poor response to medication.

The pathophysiologic cause of this disease is unknown. Because of its fluctuating course and aggravation by stress, some authors consider Meige's syndrome to be psychiatric in nature. Consequently, psychotherapy, behavioral therapy, and biofeedback have been used in treatment, but with disappointing results. Torsa and Klawans⁵ found that apomorphine and haloperidol attenuated the dystonic spasms, that physostigmine aggravated them, and that levodopa had no effect. Torsa and Lai⁶ suggested that striatal dopaminergic preponderance and cholinergic hyperfunction are associated with this syndrome. Stahl and Berger⁷ found that the dystonia was improved by administration of dopamine receptor blockers or acetylcholine receptor blockers, whereas physostigmine and levodopa aggravated the symptoms. They hypothesized the presence of relative dopamine hyperactivity and relative acetylcholine hyperactivity, a pathologic condition unique among movement disorders involving the basal ganglia. Weiner and colleagues⁸ reported the development of Meige's syndrome after long-term neuroleptic therapy and concluded that dopaminergic mechanisms play a role in its onset. Other authors^{6,9,10} consider Meige's syndrome to be related to a dysfunction of the basal ganglia.

Because little is known of its pathogenesis, the treatment of Meige's syndrome is not yet established. Forty-three patients with the clinical characteristics of Meige's syndrome were studied in therapeutic trials by Gollomp and coworkers.¹¹ In 21 (49%) patients, sustained relief of symptoms was obtained either with an antidopaminergic, anticholinergic, or acetylcholine agonist or with a γ -aminobutyric acid agonist. γ -Aminobutyric acid is involved in the physiologic functioning of the basal ganglia.

However, long-term neuroleptic therapy has serious side effects. In our patient, facial nerve block was more effective than drug therapy and lacked serious side effects. The putative mechanism of facial nerve block is

that local anesthetics, neurolytic agents, and needle insertion may block somatomotor nerves to relieve severe skeletal muscle spasm and may reduce hyperactivity of the facial nerve. We assume that facial nerve block interrupts not only efferent limbs but also afferent limbs of the abnormal reflex mechanism, because the relief of spasms outlasts, by weeks or even months, the transient pharmacologic action of local anesthetics or the effect of needle insertion.

Yuda¹² reported that recovery from postblock paresis occurred an average of 1.3 months after needle insertion blocks and that the average duration of spasm relief was more than 9 months. He also investigated complications of facial nerve block in 11,660 blocks performed in 4,462 patients with facial spasm. A method of needle insertion into the facial nerve trunk from the stylomastoid foramen developed by Wakasugi³ was performed in each case. If the spasm recurred during the block, 0.03 ml 99.5% alcohol was administered. Complications included pain during the block (18–29%), postblock pain (15%), postblock hypersecretion (16%) and hyposalivation (2%) of the lacrimal gland, facial tenderness (6%), blurred vision (5%), disturbance of taste (4%), and tinnitus (2%). Complications occurring in fewer than 1% included nausea and vomiting, nystagmus, and hypoacusis. The most drastic complication, prolonged hearing loss, was reported only in the patients treated with neurolytic agents.

Facial nerve block by a needle insertion technique may benefit a subgroup of patients resistant to other forms of therapy. We conclude that facial nerve block may be effective in treating severe cases of Meige's syndrome.

References

1. Meige H: Les convulsions de la face, une forme clinique de convulsion faciale, bilaterale et mediane. *Rev Neurol (Paris)* 21: 437–443, 1910
2. O'Brien CS: Local anesthesia in ophthalmic surgery. *JAMA* 90: 8–13, 1928
3. Wakasugi B: Facial nerve block in the treatment of facial spasm. *Arch Otolaryngol Head Neck Surg* 95:356–359, 1972
4. Marsden CD: Blepharospasm-omandibular dystonia syndrome (Brueghel's syndrome): A variant of adult-onset torsion dystonia? *J Neurol Neurosurg Psychiatry* 39:1204–1209, 1976
5. Torsa ES, Klawans HL: Meige's disease: A clinical form of facial convulsion, bilateral and medial. *Arch Neurol* 36:635–637, 1979
6. Torsa ES, Lai C: Meige disease: Striatal dopaminergic preponderance. *Neurology* 29:1126–1130, 1979

CASE REPORTS

7. Stahl SM, Berger PA: Bromocriptine, physostigmine, and neurotransmitter mechanisms in the dystonias. *Neurology* 32:889-892, 1982

8. Weiner WJ, Nausieda PA, Glantz RH: Meige syndrome (blepharospasm-oro-mandibular dystonia) after long-term neuroleptic therapy. *Neurology* 31:1555-1556, 1981

9. Casey DE: Pharmacology of blepharospasm-oro-mandibular dystonia syndrome. *Neurology* 30:690-695, 1980

10. Ortiz A: Neuropharmacological profile of Meige's disease: Overview and a case report. *Clin Neuropharmacol* 6:297-304, 1983

11. Gollomp SM, Fahn S, Burke RE: Therapeutic trials in Meige syndrome. *Advances in Neurology*: vol. 37. *Experimental Therapeutics of Movement Disorders*. Edited by Fahn S, Calne DB, Shoulson I. New York, Raven Press, 1983, pp 207-213

12. Yuda S: Complications of facial nerve block [in Japanese]. *J Pain Clin* 4:377-386, 1983

Anesthesiology
80:1398-1400, 1994
© 1994 American Society of Anesthesiologists, Inc.
J. B. Lippincott Company, Philadelphia

Hepatic Venous Outflow Obstruction during Hepatic Resection Diagnosed by Transesophageal Echocardiography

Andre M. De Wolf, M.D.,* Victor L. Scott, M.D.,† Yoogoo Kang, M.D.,‡ Moyses Mandel, M.D.,† Juan Madariaga, M.D.§

HEPATIC resection can be complicated by massive blood loss, bile duct complications, dysfunction of the residual liver, and postoperative bleeding. Obstruction of hepatic vessels, an uncommon surgical complication of liver surgery, may be life-threatening and may be difficult to diagnose clinically. We present a case of hepatic venous outflow obstruction after right trisegmentectomy that was diagnosed intraoperatively by transesophageal echocardiography (TEE), allowing immediate surgical repair. TEE also allowed us to confirm normal flow after repair.

Case Report

A 75-yr-old woman with a bile duct tumor underwent excision of the bile duct and right trisegmentectomy. Past medical history was

* Associate Professor of Anesthesiology and Critical Care Medicine.

† Assistant Professor of Anesthesiology and Critical Care Medicine.

‡ Professor of Anesthesiology and Critical Care Medicine.

§ Assistant Professor of Surgery.

Received from the Department of Anesthesiology and Critical Care Medicine and the Department of Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania. Accepted for publication February 7, 1994.

Address reprint requests to Dr. De Wolf: Department of Anesthesiology and Critical Care Medicine, Room C-222, Presbyterian University Hospital, University of Pittsburgh Medical Center, 200 Lothrop Street, Pittsburgh, Pennsylvania 15213-2582.

Key words: Liver, hepatic vein: obstruction. Monitoring: transesophageal echocardiography. Surgery: liver resection.

significant for hypertension controlled with enalaprilat and nifedipine. Monitoring included invasive determination of arterial pressure, central venous pressure, pulmonary arterial pressure, and thermol dilution cardiac output. A lumbar epidural catheter was placed before induction of anesthesia for postoperative analgesia. Anesthetics and adjuvants included thiopental, fentanyl, vecuronium, pancuronium, and isoflurane in oxygen and air.

Overall, the patient was hemodynamically stable, although several hypotensive episodes occurred because of blood loss and manipulation of the major blood vessels. Total transfusion requirements were 14 U packed red blood cells and 11 U fresh frozen plasma. The cardiovascular system was supported by intermittent infusion of dopamine (to a maximum of $10 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) and epinephrine ($0.05 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) for about 30 min. During dissection, the left hepatic vein was injured, and after the inflow and outflow had been controlled for approximately 30 min, it was repaired.

After resection, the left lateral segment of the liver appeared congested. When a decrease in central venous pressure from 19 to 13 mmHg and manipulation of the left hepatic lobe by the surgeon (in an attempt to relieve possible kinking of the hepatic vein) failed to reduce the hepatic congestion, a TEE probe (Sonos 1000, Hewlett-Packard, Andover, MA) was placed (fig. 1). Echocardiographic evaluation of the heart did not reveal enlargement of the right atrium or right ventricle. When the inferior vena cava was inspected, the junction of the left hepatic vein and the inferior vena cava could not be visualized. Color Doppler sonography of the hepatic vein showed a mosaic pattern, indicating blood flow turbulence that was very likely the result of partial vessel obstruction (fig. 2A). Hepatic venous outflow obstruction caused by surgical narrowing of the vessel was diagnosed (fig. 1 inset). A homologous "jump" graft was placed between the intrahepatic left hepatic vein, which was very superficial to the raw surface of the liver, and the inferior vena cava (fig. 1 inset). Nonturbulent flow through the jump graft was clearly demonstrated by color Doppler sonography (fig. 2B), and subsequently, the congestion of the left lateral segment diminished.

The patient recovered from the procedure and left the hospital 1 week after her operation.