CLINICAL ARTICLE

Transient Paralysis Shortly after Anterior Cervical Corpectomy and Fusion

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Objective: To report three cases of transient paralysis shortly after (within 4 hours) anterior cervical corpectomy and fusion (ACCF), and investigate the possible causes.

Methods: Clinical and radiological data of three cases (two men and one woman, aged 41–61 years) were analyzed retrospectively. All three patients underwent ACCF for cervical spondylotic myelopathy. The decompressed segments were located in C₅, C₆ and C₅ + C_{6–7} discs, respectively. Paralysis occurred from 30 minutes to 4 hours after surgery. In two cases the paralysis was complete; it was incomplete in the third. All patients received immediate dehydration, neurotrophic drugs and high-dose methylprednisolone therapy upon recognition of their paralysis. Meanwhile, cervical MRIs were performed and showed no significant hematomas compressing the cervical spinal cord; spinal cord edema was clearly evident in all cases.

Results: In two cases the paralysis resolved within 2 hours of diagnosis and immediate medication. In the third case, because the neurological symptoms were incompletely resolved 24 hours after beginning medication, a second laminoplasty was performed. During decompression, tremendous pressure was released from the cervical spinal cord. The neurological symptoms had resolved completely by 1 week after decompression.

Conclusion: The precise cause for transient paralysis after these anterior cervical surgeries is not yet clear. Spinal cord ischemia-reperfusion injury is generally regarded as the most likely cause. Therefore, a combination of cervical spinal cord edema and limited anterior decompression space may have been the main contributing factors to the paralysis reported here. Early diagnosis and early intervention to relieve the paralysis can restore spinal cord function and result in a satisfactory prognosis.

Key words: Anterior; Cervical; Decompression; Paralysis

Introduction

Postoperative paraplegia consequent to cervical spinal cord injury is one of its most serious complications and a most worrisome issue for both doctors and patients. Free postoperative movement of limbs is always a great relief to doctors. Sometimes, however, patients move their limbs freely after surgery for a short time, then, for no obvious reason, paralysis suddenly develops. Such paraplegia often resolves miraculously within a few hours of timely diagnosis and treatment. Immediate MRI often fails to identify an explanation for this phenomenon. This article reports comprehensive data on three cases of

transient paralysis occurring shortly after anterior cervical corpectomy and fusion (ACCF), analyzes the possible causes and discusses published reports that clarify the pathogenesis.

Clinical Data

Case 1

A 41-year-old man complained of numbness and weakness in his upper and lower limbs for 3 months. Physical examination (PE) determined that the strength in his limbs was normal. Sensation was diminished on the ulnar sides of both hands.

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Disclosure: No funds were received in support of this work. No benefits in any form have been, or will be, received from a commercial party related directly or indirectly to the subject of this manuscript.

Received 19 July 2012; accepted 5 November 2012

Fig. 1 Preoperative T₂WI MRI showed obvious spinal cord compression at C₅₋₆ level with high intensity zone (case 1).

Triceps tendon reflexes were absent bilaterally, as were both biceps tendon reflexes and the radial periosteal reflex on the right side. Lower limb muscle tone was increased, the right knee tendon reflex was absent, and there was hyperreflexia on the left. He had bilateral Achilles tendon reflex hyperreflexia, bilateral patellar clonus and positive ankle clonus. Both Hoffman and Babinski signs were positive bilaterally. A preoperative MRI showed C₃–C₇ disc degeneration with herniation and grade II C₅₋₆ spinal stenosis (Fig. 1).

The patient underwent C₆ corpectomy, iliac crest graft and plate fixation under general anesthesia. During surgery, adhesions between the dura, ossified posterior longitudinal ligament and herniated discs were identified and completely removed. Immediately postoperatively, the patients had normal sensory and motor functions in all limbs. However, 30 minutes later, numerous red papules appeared on his scalp, face, chest and upper limbs. A provisional diagnosis of allergy to a narcotic drug was made and a dose of diphenhydramine administered i.m. One minute after this injection, bilateral complete paralysis occurred below the nipple plane. No physiological or pathological reflexes could be elicited. Mannitol

and high-dose methylprednisolone were given rapidly. Cervical MRI showed the fixation to be in a satisfactory position and no obvious hematoma compressing the spinal cord. However, no cerebrospinal fluid was visible around the spinal cord (Fig. 2). The paralysis miraculously resolved completely 40 minutes after its occurrence.

Case 2

A 61-year-old man complained of numbness of the upper limbs and unsteady gait for 2 months with bladder weakness for 1 month. On PE, he had diminished sensation in the upper limbs, especially on the ulnar sides of the hands. He had grade 4 muscle strength for wrist extension, elbow extension and flexion bilaterally. Biceps and triceps tendon reflexes were normal. The Hoffmann sign was (+) bilaterally, he had decreased sensation in the lower limbs below the knees and increased muscle tone with normal strength in his lower limbs. There was hyperreflexia of both patellar tendon reflexes, normal Achilles tendon reflexes, bilateral ankle clonus (+) and bilateral negative Babinski signs. An MRI showed C₂–C₇ disc herniation with spinal cord edema from C₄ to C₆ (Fig. 3).

The patient underwent C₅ corpectomy, C₆₋₇ discectomy, mesh grafting and plate fixation under general anesthesia. During surgery, severe adhesions between the dura and posterior longitudinal ligament were noted, making decompression of the spinal cord anteriorly a lengthy procedure. Postoperatively, the patient could move his limbs normally and felt much improved. 40 minutes later, he complained of sudden and severe neck pain, numbness of the limbs and difficulty breathing. On PE his blood pressure was 148/102 mm Hg, pulse 104/min and temperature 37.2°C. He had abdominal breathing, complete sensory loss below the shoulder plane and no muscle strength in his limbs. Physiological reflexes were absent and Hoffman and Babinski signs positive bilaterally. Mannitol and high-dose methylprednisolone were administered rapidly. A cervical MRI showed satisfactory position of the fixation, minor hematoma formation anterior to the spinal cord at the level of C₅ and obvious spinal cord edema with no high intensity in the spinal cord (Fig. 4). Because the patient had difficulty breathing, he was prepared for emergency surgery. Just before anesthesia was to be initiated (1 hour after onset of paralysis), chest breathing and sensation gradually returned and his muscle strength in the limbs improved to grade 2–3. Emergency surgery was cancelled. Two hours after onset of paralysis, his limb function had returned to the initial postoperative level except that there was only partial recovery of strength in the right upper and lower limbs. One week after surgery, his neurological function remained stable and MRI T₂WI images showed high intensity in the spinal cord from C₂ to C₅, indicating residual spinal cord edema (Fig. 5). Two weeks after surgery, muscle strength in his limbs had recovered completely and he had numbness only in his right upper and lower limbs.

Case 3

A 58-year-old woman complained of neck and shoulder pain for 3 years and numbness and weakness in her left upper limb

Fig. 2 Images from an MRI immediately after onset of paralysis showing spinal cord edema without obvious hematoma (case 1).

for 1 month. On PE she had a limited range of motion in the cervical spine, normal sensation in the limbs, grade 4 strength in the extensors of the left wrist and elbow and flexors of the fingers, and positive Hoffman and Babinski's signs bilaterally. A MRI showed C₄–C₆ disc herniation.

The patient underwent C₅ corpectomy and mesh fusion under general anesthesia. During surgery, adhesions between the dura, ossified posterior longitudinal ligament and herniated disc were noted and removed completely. Postoperatively, the patient had normal sensory and motor functions in her limbs. She felt well and then fell asleep. When she woke 4 hours sleep later, she was paralyzed. On PE she had decreased strength in her limbs; the most severely affected being the left lower limb at grade 0. Sensation was decreased below the nipple plane bilaterally. Mannitol and high-dose methylprednisolone were administered rapidly. An MRI showed spinal cord edema and no hematoma formation (Fig. 6). After medication her neurological function recovered partially; the muscle strength in her limbs improving to grade 2–3. An additional posterior decompression was recommended. 24 hours after onset of paralysis, the patient agreed to undergo a C₃–C₆ laminoplasty under general anesthesia. Because of the enormous pressure within the spinal canal, when the lamina was opened with a rongeur, a 3 mm gap in the lamina burst open accompanied by a muffled bang. Obvious bulging of the spinal cord was apparent after decompression. Dehydration and neu-

rotrophic drugs were continued after surgery and her neurological symptoms had resolved completely by 1 week after decompression (Fig. 7).

Discussion

Summary of Characteristics of Cases

The above three cases of paralysis after anterior cervical surgery have some things in common as well as unique individual characteristics. The similarities we noted are as follows: (i) paralysis occurred after anterior cervical decompression and fusion surgeries during which removal of severe adhesions to the dura prolonged the procedures considerably; (ii) immediately postoperatively, neurological function was quite good, paralysis developing suddenly within 1–4 hours; (iii) immediate MRIs showed obvious spinal cord edema rather than hematoma formation; and (iv) after early medication or surgical intervention, spinal function recovered completely. The differences were in (i) the recovery process: the paralysis of cases 1 and 2 resolved completely within 2 hours, whereas, that of case 3 had only resolved partially by 24 hours after its onset; (ii) extent of paralysis: this was complete in cases 1 and 2 and incomplete in case 3; and (iii) treatment: medication was all that cases 1 and 2 required, whereas case 3 underwent posterior decompression in addition to receiving medication.

Fig. 3 Preoperative T₂WI MRI showed C₄₋₇ disc herniation with high intensity zone in spinal cord (case 2).

Analysis of Causes of the Paralysis

Paralysis after cervical spinal surgery has multiple causes, including compression of the spinal cord resulting from poorly implanted fixation or hematoma formation, spinal cord edema and ischemia reperfusion injury. Postoperative radiological examinations can detect poorly implanted fixation and hematomas in time for immediate revision to restore spinal function. Otherwise unexplained paralysis is often attributed to ischemic reperfusion injury to the spinal cord. Ischemia reperfusion injury occurs when blood flow is restored to previously ischemic tissues and organs, damaging them. Its occurrence and severity correlate closely with tissue ischemia time, extent of ischemic tissue and the affected tissue's oxygen requirements^{1,2}. Neurons in the spinal cord are extremely sensitive to ischemia and hypoxia; such sensitivity may be a prerequisite for spinal cord ischemia reperfusion injury. Its mechanisms, which are multi-factorial and multi-channel, include: (i) local changes in the plasma during spinal cord ischemia, in particular Ca²⁺ overload mediated by a cascade of axon disintegration and changes caused by fibrosis; (ii) many oxygen free radicals, which mediate per-

oxidation reactions; and (iii) presence of excitatory amino acids; glutamate is involved in calcium-mediated nerve cell apoptosis.

According to published reports, the incidence of spinal cord ischemia reperfusion in thoracic and abdominal aortic surgeries is 0.4%–18%, that is, 2.87%–3.89% of all spinal surgery^{3,4}. They proposed diagnostic criteria for spinal cord ischemia reperfusion injury are: (i) clear underlying causes of spinal cord compression, such as cervical or thoracic disc herniation or stenosis; (ii) surgery for spinal cord decompression; (iii) paralysis, which is often gradually progressive, occurring within 8 hours of surgery, usually within 1–3 hours; (iv) motor and sensory dysfunction from lower to upper limbs which, when rapidly developing and extreme, may result in death; (v) radiological and other investigations rule out spinal cord compression caused by mechanical factors such as hematoma and spinal cord or brain lesions above the level of decompression; and (vi) timely high-dose methylprednisolone, dehydration and neurotrophic drugs can quickly restore spinal function completely or partially.

Fig. 4 Image from an MRI immediately after onset of paralysis showing spinal cord edema with hematoma formation at the operated level (case 2).

Fig. 5 One week after surgery, MRI image showing decreased spinal cord edema and hematoma are still present (case 2).

On the whole, the three cases reported in this article conform to the above six criteria. However, ischemia reperfusion injury cannot explain some of their characteristics. The enormous pressure released from the spinal canal during revision of case 3 particularly impressed the surgeons. We postulated that this pressure was due to spinal cord edema consequent on damage to the spinal cord during removal of extensive adhesions anterior to the dura. That the space provided by corpectomy is relatively small compared to that provided by posterior decompression could have been a compounding factor. In addition, after mesh grafting the once opened spinal canal was again closed. A swollen spinal cord in a closed canal together with possible compression from postoperative hematoma will increase pressure on the spinal cord. Under such circumstances, the spinal cord may not only be deprived of blood reperfusion, but also be more severely ischemic than it was preoperatively. Also, when the laminae of case 3 were opened, the spinal cord bulged dramatically as the pressure was almost instantaneously released. Spinal function recovered quite well postoperatively. Recently, there has been much debate about the mechanism of ischemia reperfusion injury. Ondrejicková *et al.* established a rabbit model of ischemia reperfusion injury by blocking the abdominal aorta for 25 minutes, then allowing blood flow reperfusion for 60–120 minutes⁵. They confirmed that injury occurred only in the ischemic phase, whereas in the reperfusion phase a tissue repair process started in the spinal cord. If reperfusion was extended to 120 minutes, the metabolic disorder caused by

ischemia was significantly less severe. Torg *et al.* reported 110 cases of transient paralysis related to sports injuries involving the cervical spine⁶. Developmental cervical spinal stenosis and degenerative cervical disease, such as cervical disc herniation or cervical ossification of the posterior longitudinal ligament, are proven risk factors for this condition. The authors believe that transient paralysis after anterior cervical spinal surgery may be related to spinal cord edema and the limited decompression space inherent to anterior cervical decompression procedures, rather than the ischemia reperfusion injury to which it is usually attributed.

Treatment of Paralysis

In postoperative patients, intensive care and regular checking of spinal function are of great importance. Once paralysis occurs, early diagnosis and early intervention are essential to restoring spinal function. If the cervical spinal cord is under high pressure for a long time nerve cell atrophy and necrosis may occur, leading to the tragic consequences of permanent paralysis. The first step in intervention involves dehydration, neurotrophic drugs and high-dose methylprednisolone. Additional surgical intervention depends on the patient's reaction to the medication. If the medication is not as effective as expected, posterior decompression to restore spinal cord function should be considered. A good prognosis can be expected if this condition is diagnosed and treated properly.

Fig. 6 Image from an MRI immediately after onset of paralysis showing spinal cord edema without obvious hematoma (case 3).

Fig. 7 After a second laminoplasty, an MRI image showing decreased cord edema and high intensity signal in the cord (case 3).

References

1. Tomes DJ, Agrawal SK. Role of Na(+)-Ca(2+) exchanger after traumatic or hypoxic/ischemic injury to spinal cord white matter. *Spine J*, 2002, 2: 35–40.
2. Asiedu-Gyekye IJ, Vaktorovich A. The “no-reflow” phenomenon in cerebral circulation. *Med Sci Monit*, 2003, 9: BR394–BR397.
3. Jiang X, Shi E, Nakajima Y, et al. Postconditioning, a series of brief interruptions of early reperfusion, prevents neurologic injury after spinal cord ischemia. *Ann Surg*, 2006, 244: 148–153.
4. Back MR, Bandyk M, Bradner M, et al. Critical analysis of outcome determinants affecting repair of intact aneurysms involving the visceral aorta. *Ann Vasc Surg*, 2005, 19: 648–656.
5. Ondrejicková O, Stolz S, Ziegelhöffer A, et al. Postischemic reperfusion of the spinal cord: prolonged reperfusion alleviates the metabolic alterations induced by 25 min ischemia in the cervical and thoracolumbal segments. *Gen Physiol Biophys*, 2000, 19: 415–426.
6. Torg JS, Corcoran TA, Thibault LE, et al. Cervical cord neurapraxia: classification, pathomechanics, morbidity, and management guidelines. *J Neurosurg*, 1997, 87: 843–850.