Case report

Delayed occurrence of spinal arachnoiditis following a caudal block

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Context: Spinal arachnoiditis is a rare disease caused by fibrosis and adhesion of the arachnoid membrane due to chronic inflammation. The causes of arachnoiditis are infection, spinal surgery, intraspinal injection of steroid or myelography dye, and spinal anesthesia.

Method: Case report.

Findings: A 60-year-old woman presented with progressive weakness and sensory change of both legs and urinary symptoms. She had received a single caudal block 6 months before symptom onset. Magnetic resonance imaging of the thoraco-lumbar spine showed an intradural extramedullary tumor at the T5–T7 level. She underwent laminectomy and tumor resection. The pathological finding was arachnoiditis. After surgery, a rehabilitation program of strengthening exercises of both lower extremities and gait training was started. At 2-month follow-up, she was able to walk with orthoses and performed daily activities with minimal assistance.

Conclusion: Symptoms of spinal arachnoiditis occurred 6 months after a single caudal block in this woman. Clinicians should be aware of this possible delayed complication.

Keywords: Arachnoiditis, Myelopathy, Intrathecal injection, Myelography, Caudal block, Paraplegia, Neurogenic bowel, Neurogenic bladder, Rehabilitation, Spinal cord injuries, Spinal infection

Introduction

The arachnoid membrane is the central membrane of the three membrane layers that enclose the brain and spinal cord. Chronic inflammation of this membrane results in fibrosis and thickening, and leads to adhesions to pia and dura mater, which in turn cause spinal arachnoiditis. Spinal arachnoiditis is a rare disease,¹ which causes the following symptoms; back pain radiating pain to the legs, urinary symptoms, and sensory impairment. Spinal arachnoiditis can be diagnosed by myelography, computed tomography, and by magnetic resonance imaging (MRI) in combination with clinical symptoms and history taking.^{1,2}

Many reports have been issued on the etiologies of arachnoiditis, which include infection, intraspinal injections of steroid, anesthetic, or myelography dye, spinal surgery, spinal cord tumors, and idiopathic.^{1–3} We report a case of spinal arachnoiditis that occurred 6 months after a single caudal block.

Case report

A 60-year-old woman presented with weakness of both legs of 4 months duration. Six years earlier, she developed low-back and bilateral-buttock pain, which were treated conservatively at a local hospital. In addition, 10 months earlier she had received a caudal epidural block (bupivacaine 0.5 mg and triamcinolone 40 mg) at the sacral hiatus for chronic low-back pain. No short-term complications were noted. She did not undergo spinal surgery or myelography, and was not administered spinal anesthesia. There were no symptoms or signs of infection.

Four months earlier, she noticed sensory changes and hypothermia of both legs and urinary symptoms (urgency, frequency, and incontinence). Several days later, she experienced weakness of both legs. One month prior to presentation, she had been able to walk independently, but complained of a shuffling gait. At that time, she underwent a diagnostic workup that included laboratory examination, MRI, and cerebrospinal fluid (CSF) study. The following day, bilateral leg weakness rapidly worsened, and she was no longer

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able to walk, and lost sensation for urination and defecation.

Laboratory data showed normal white and red blood cell counts and erythrocyte sedimentation rate; CSF study findings were negative for WBCs and RBCs. In addition, fungal and AFB stains and cultures were negative. However, MRI of the thoraco-lumbar spine showed spinal cord edema and high-signal intensity at T8–L1. She was diagnosed with transverse myelitis, and treated with steroids and rehabilitation. However, she showed no improvement after a month, and thus, was transferred to our hospital.

At admission, the patient had no urinary or defecation sensation, and a laboratory examination failed to reveal any symptoms or signs of infection. During the neurological examination, manual muscle testing showed 2/5 to 3/5 for both lower extremities (Table 1), deep tendon reflex was increased, and no pathological reflexes were present. Follow-up MRI of the thoraco-lumbar spine showed high-signal intensity of T8 to the conus medullaris, suggesting myelopathy, and an intradural extramedullary tumor was seen on T5–T7. The mass was suspected to be an arachnoid cyst (Fig. 1). Laminectomy and tumor resection were performed, the pathological finding was arachnoiditis.

Thirty-six days after surgery, the patient was transferred to our rehabilitation department for comprehensive rehabilitation. At the time of transfer, manual muscle testing showed 2/5 to 3–/5 for both lower extremities, and a sensory examination showed reduced sensitivity to light touch and pain below the T12 dermatome level but intact perianal sensation. Both knee jerks were hyper-reflexive, Babinski's reflex was positive on the right and ankle clonus was positive on both sides. The patient was unable to stand unaided and required moderate (25–50%) assistance to cope with basic daily activities. To strengthen her muscles, active assisted range of motion exercises and functional electrical stimulation were started. Standing training and daily living activity training were initiated. At 15 days after transfer, she started gait training with a walker and at 45 days after transfer, she was able to

walk 50 m with the walker. However, at that time, she experienced new sudden onset weakness of both lower extremities and MRI of the thoraco-lumbar spine showed high-signal change of the T7–T8 cord, suggesting recurrent arachnoiditis. Intravenous dexamethasone (tapering 20–5 mg/day) and naloxone (6 mg/day) and 20% mannitol (500 mg/day) were administered for 10 days, and 1 week after discontinuation (52 days after transfer), her lower-extremity power started to recover. After 6 weeks of rehabilitation, she was able to walk 50 m with bilateral mono canes and to perform daily activities with minimal assistance (0–25%).

Discussion

Spinal arachnoiditis was first recognized as a separate disease entity in 1909 by Victor Horsley.³ Its prevalence has not been determined precisely, but it is considered a rare spinal disease,⁴ which presents with non-specific neurological signs and symptoms, such as back pain, radiating pain to the legs and/or paraplegia, and thus, it is difficult to diagnose clinically.² It is usually encountered in the thoracic or cervical region, although recently it has been reported in the lumbosacral region, which reflects a shift in the causes of arachnoiditis from mainly spinal infections to intrathecal injections and myelography dye.⁵

The initial stage of this disease is characterized by radiculopathy caused by inflammation of the pia-arachnoid mater with nerve root swelling and hyperemia. 'Arachnoiditis' follows, which is characterized by collagen deposition, a decrease in nerve root swelling, and the adherence of nerve roots to each other. 'Adhesive arachnoiditis' represents the resolution of the inflammatory process and dense collagen deposition causing complete encapsulation of nerve roots, which then undergo progressive atrophy.⁶

Possible causes of spinal arachnoiditis include infections, blood in CSF due to hemorrhage, intervertebral disk herniation, spinal surgery, spinal anesthesia, intraspinal steroid injection, myelography dye injection, and idiopathic.^{3,7,8} Intraspinal steroid injection is used

Table 1 Serial changes in manual muscle strength

	Admission		Rehabilitation start		45 days after rehabilitation		Arachnoiditis recurrence		Discharge	
	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
L2 hip Fx	3/5	3/5	2/5	2–/5	3–/5	3–/5	2+/5	2+/5	3–/5	3–/5
L3 knee Ex	3/5	3/5	2+/5	3–/5	3/5	3/5	2/5	2/5	4/5	4/5
L4 ankle DF	3/5	3/5	2/5	2/5	3–/5	3–/5	2–/5	2–/5	3/5	3/5
L5 long toe Ex	2/5	2/5	2+/5	2/5	2+/5	2+/5	2–/5	2–/5	2+/5	2+/5
S1 ankle PF	3/5	3/5	2+/5	2+/5	3–/5	3–/5	2–/5	2–/5	3+/5	4/5

Figure 1 (A) Sagittal T2-weighted magnetic resonance image of thoracolumbar spine, showing a prominent CSF space at the posterior aspect of the T5–T7 spinal cord (continuous arrow) and diffuse swelling and high signal change of the T8–L1 spinal cord (broken arrow). (B) Axial T2-weighted magnetic resonance image of T6 level showing a prominent CSF space. (C) Axial T2-weighted magnetic resonance image of T10 level showing high-signal change of spinal cord.

to relieve back pain; corticosteroid as hydrocortisone acetate, methylprednisolone acetate (MPA), or triamcinolone are commonly used. In particular, MPA has been reported to cause arachnoiditis. However, intraspinal injections may be made intrathecally or epidurally, and reported cases arachnoiditis have followed an intrathecal injection.^{9,10} Furthermore, arachnoid villi subtend the subarachnoid, subdural, and epidural spaces, and thus, inflammation caused by an epidural injection can spread. The other danger associated with epidural injections is that the epidural space is entered ‘blindly’ during injection. These anatomical features and technical errors during injection largely explain the development of arachnoiditis following epidural injection.⁹

The delayed occurrence of spinal arachnoiditis is rare but not unprecedented. Several articles have reported cases of spinal arachnoiditis with a delayed symptom onset following spine procedures or diseases. Toshihiko *et al.* reported a case of arachnoiditis caused by epidural anesthesia during childbirth in a 30-year-old woman. The patient continued to complain of radiating pain to the left leg and buttock after epidural anesthesia. Gait disturbance and hyperesthesia occurred 7 months after injection, when she was diagnosed as having arachnoiditis by MRI.¹⁰ Christian *et al.*¹¹ described four children who contracted meningitis during the neonatal period who developed myelopathy caused by chronic arachnoiditis several years later; in two of these cases, myelopathy was precipitated by a fall. Benjamin and George reviewed 68 patients with a diagnosis of spinal arachnoiditis. The probable causes of diagnosed arachnoiditis were: myelogram and surgery (60 patients), myelogram only (2 patients), and spinal anesthesia (2 patients). Of these 68 patients, 36 (53%) experienced a relatively symptom-free period of 3 months to 9 years after the spinal procedure.¹²

In our case, the patient had no history related to the known causes arachnoiditis other than a history of a caudal block for back pain control some 10 months previously (6 months before symptom onset), which implicates the caudal block.

The clinical symptoms of arachnoiditis are back and buttock pain – more commonly radiating pain. Motor paralysis and sensory impairment below the injury level and urinary symptoms develop as the disease progresses.^{1,8} The latent period after the initial trigger is reported to range from 1 to 10 years.⁵ Myelography was used for diagnosis, but currently, MRI appears to be the diagnostic tool of choice.

Spinal arachnoiditis has been treated with steroid or antituberculosis drug therapy, surgical dissection, and

by anesthetic-based pain control, but no specific treatment regimen has been established. The prognosis is poor. Guyer *et al.* reported that despite aggressive one treatment, symptom severity fluctuated, pain and functional impairment seldom improved, and average life-span was shortened by 12 years.⁴

Conclusions

Six-month delayed onset of symptoms after a caudal block is an unusual presentation of spinal arachnoiditis. Symptomatic and functional improvements in this woman were achieved through surgery, drug therapy, and comprehensive rehabilitation. Clinicians should be aware that there may be a delayed onset of symptoms of arachnoiditis.

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