Rapid Progression of Extensive Arachnoid Adhesion Longer Than Twelve Segments and Cysts Formation after Posterior Lumbar Interbody Fusion: Case Report

Arachnoid cysts and intradural adhesion are uncommon in postoperative complication rather than congenital condition. The adhesion and cysts are usually around surgical site but some extend to a distant place. The authors report a case of formation of arachnoid cysts and adhesion far away from original surgical site with significant clinical manifestations after posterior lumbar interbody fusion (PLIF).

A 55-year-old woman accepted PLIF and got a good recovery but her dura mater was torn and sutured during procedure. Six months after her 1st surgery, she felt rapid progressing numbness from foot to belly and weakness of both lower limbs. On examination bilateral deep tendon reflexes were hyperactive. Magnetic resonance imaging (MRI) showed intradural adhesion formed from T4 to sacral canal. Cerebrospinal fluid terminated at T4 level and there exactly the arachnoid cyst was formed. The long intradural adhesion and distant cyst formation made this case unique and the cause of neurological manifestations. Then the patient underwent intradural exploratory surgery and cyst-abdominal shunting. The cyst was actually found to be the terminal part of remaining subarachnoid space. Patient’s numbness stopped advancing upward and the strength of both lower limbs gradually improved after surgery. Therefore, arachnoid cyst and adhesion might appear at any place after spine surgery with dural rupture. Exploratory surgery and timely decompression can effectively slow down the progression of disease.

Spinal meningeal cysts are rare and have been described as “arachnoid cysts,” “pouches”, or “diverticula”. Most spinal intradural arachnoid cysts are thought to be congenital but still some acquired arachnoid cysts were reported as to be the results of trauma, hemorrhage or infection. Even fewer were caused by intradural inflammation after invasive diagnostic test and treatment.

Key Words: arachnoid cyst, arachnoid adhesion, PLIF, shunt
Case Report

A 55-year-old woman, presented with low back pain and lower limbs weakness at the outpatient clinic. Her low back radiated to the lateral part of left Dorsalis pedis, more serious after being sedentary or long standing. On examination, she had nearly normal spine mobility, but felt backache during bending more than 45°. Gait seemed normal and Lasegue’s test was negative. Her muscle strength of dorsiflexion of left foot was IV minus and others IV. The lateral side of her left thigh was moderately hypo anesthetic. Deep tendon reflexes of lower limbs existed and no pathologic reflex could be provoked. Her JOA (Low Back Pain Scoring System) scale was 15.

Magnetic Resonance Imaging was taken to verify the location and degree of lumbar spondylolisthesis. Figure 1 shows Grade II spondylolisthesis at L4-L5 level with canal stenosis. The authors decompressed the spinal canal and did posterior lumbar interbody fusion (PLIF). The dura mater tore in the process of laminoplasty but was sutured immediately. Manipulation near spinal cord was gentle and was monitored by somatosensory evoked potential (SSEP) and motor evoked potential (MEP). Patient’s backache increased with some numbness on her...
left instep after surgery. Three months of regular follow up showed good recovery and the JOA score improved to 8 points. Her muscle power was IV+ on her both lower limbs in last follow up.

6-month after surgery she felt rapidly progressing numbness from feet to xiphoid within two weeks. Physical examination found significant loss of superficial sense. Abdominal reflex disappeared, muscle strength also decreased to III for both lower limbs. Deep tendon reflexes of both lower limbs were hyperactive. Pathological reflex was not evoked. MRI demonstrates that lumbar cistern was nearly dry (Figure 2). Arachnoid cyst formed at T4 level and the border of spinal cord below it was thickened (Figure 3). T1-weighted enhanced STIR image shows that CSF terminated at T4 level and the spinal cord below was wrapped by high signal (Figure 4). CT myelography could not be done because of pain caused by needle insertion.

**Treatment Strategy**

There were two pathogenic factors resulting in her clinical manifestation. Firstly, the intradural cyst no matter in or out of subarachnoid space compressed the spinal cord and caused upper motor neuron disorder for both limbs. Secondly, long extent of adhesion was closely related to the rapidly progressing ascending numbness but it was hard to judge the sequence of these two factors because of lack of 3-month follow up. As patient desired for symptomatic relief and prevention of further progression of the disease, we planned to perform cystectomy and intradural exploratory operation as soon as possible. We considered the cyst to be the terminal of the remaining arachnoid sac like a pouch not only just an isolated capsule. The adhesion went upwards gradually from the original surgical site to T4 level so as to “push” the terminal of the arachnoid sac to a higher position. The pulsatile flow of CSF was disturbed and the cyst was inflated by high-tension pulsation. Thus, we prepared cyst-sac shunt as well.

During the operation, vertebral lamina from T2 to T6 was removed by ultrasonic osteotome. We found the cyst was hyper tense and located at the ventral side of spinal cord in T4 level with its downstream arachnoid sac dried up (Figure 5). After opening dural sac, we noticed that the arachnoid mater stuck with the pia mater without any space or CSF between them (Figure 6). The twin-layer sleeve-like membrane (dural and arachnoid mater) wrapped spinal cord tightly that neural transmission was impossible. Then we opened the cystic wall and found plenty of CSF which was under pressure and kept on flowing out. The roof of cyst connected with subarachnoid space by a one-way-permissive valve and its bottom was made by adhesion of arachnoid mater and pia mater. Then, we implanted a catheter between the cyst and abdominal cavity (Figure 7). The pressure threshold of catheter was 70mmH2o for lying position and 270mmH2o for standing upright. Before closure of dural sac, careful irrigation with normal saline and local glucocorticoid injection was administered with the purpose of preventing further inflammatory reaction. Vertebral lamina was implanted back with mini plates. No changes were observed in motor evoked potential and somatosensory evoked potential during surgery. Pathologists’ final report announced neither infection nor tumor.

**Postoperative Course**

Following an uneventful 4-hour procedure, the patient was transferred to the post-anesthetic care unit. On her immediate postoperative exam, no further paralysis or hypoesthesia was noted. 3 days after surgery her muscle strength of lower limbs went down due to edema and recovered to preoperative level in 12 days. On regular post-operative follow-up, her superficial sensation did not deteriorate but gradually recovered in the following 3 months. The muscle strength of her both legs stayed at III level.
Discussion

Existence of both distant arachnoid cyst beyond surgical site and extensive subarachnoid adhesion in one case after lumbar surgery is very rare. Even if intraoperative dural sac tear might cause inflammatory reaction and later cyst, few cases have been published. Following the patient up, we found the intradural structure and spinal function was normal before first surgery and no infection occurred during first hospitalization. The initial pathogenic factor was possibly dural mater tear which was iatrogenic. The general incidence of dural tear is 7.3%. Although several studies have shown that accidental durotomy that is identified intraoperatively and properly addressed does not have a detrimental effect on long-term outcome. We still considered opened dural sac, blood and tissue debris might have irritated immune system of arachnoid membrane and caused inflammation. Infection following lumbar interbody fusion has been reported 0-9% of patients. During first surgery, our patient was normal in temperature and WBC count and was free from meningeal irritation though local infection without clinical manifestation could still exist.

The second surgery was to decompress spinal cord from the cyst by inserting a shunt even if the over-tight and over-long adhesion could not be released. Extensive arachnoid adhesion and cyst were two interdependent causes of neurological damage for this patient. Cyst explained hyperactive deep tendon reflexes of low extremities and absence of abdominal reflex. Patient’s rapid progressing numbness rising from feet to belly was mostly caused by sleeve-like adhesion of intra-arachnoid structure which possibly grew upwards in the meantime. We found the bottom of cyst was not independent but was composed of adhesion of arachnoid mater and pia mater and the ceiling of it was one-way-permissive valve that just allowed CSF to pulse in so that the cyst was hyper tense. Relieving compression as soon as possible was necessary, since it is more direct and etiological. CSF dried up because of extensive adhesion and upstream cyst blocking CSF transfer from cerebral ventricle to lumbar cistern but did not affect the absorptive function of arachnoid villi and meningeal sheaths. Reduced CSF led to material exchange disorder, disturbance of PH and collision buffering. Luckily, the blood supply of spinal cord and cauda equine was not affected so that nutrition exchange through blood brain barrier was partly preserved. That’s why her neurological function recovered after surgery.

In summary, any accidental opening of dural mater might possibly cause intradural inflammation, so doctors need careful manipulation. Subarachnoid adhesion is very likely to obstruct CSF flow and produce hyper tense cysts that might compress spinal cord badly. If adhesion and compression both exist, removing compression should be considered first. When vertebral canal is opened, complete hemostasis is needed. If dural sac tears unexpectedly, it is highly suggested blocking the hole by nonirritating gauze temporarily to prevent blood inflowing and CSF outflowing and thoroughly flushing the wound ahead of suture. Postoperative steroid and antibiotics can be administered if necessary.

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References

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