CE

A Comprehensive Review of Spinal Arachnoiditis

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Spinal arachnoiditis is an insidious disease caused by an inflammatory process of the arachnoid membrane resulting from many possible causes, such as myelograms with oil-based radiographic contract agents and multiple back surgeries. Diagnosis is based on symptoms and magnetic resonance imaging. Arachnoiditis can also mimic the symptoms of other diseases, such as spinal cord tumors, cauda equina syndrome, arachnoiditis ossificans, and syringomyelia. Unfortunately, there is no cure, only treatment of the chronic symptoms. It is an incurable disease that can cause minor to severe symptoms from unexplained rashes to neurologic defects.

Anatomy

Spinal arachnoiditis is a disease characterized by inflammation and scarring of the arachnoid membrane of the spinal cord. Three membranes, the arachnoid, the dura, and the pia cover, protect and provide cushion for the spinal cord and spinal nerve roots. Damage occurs when an inflammatory response triggers fibrinous exudates, which results in the nerve roots adhering to themselves and/or the thecal sac. Dense collagenous adhesions form from the fibrocytes produced during postinflammatory repair (see Figure 1). (Toribatake, Baba, Maezawa, Umeda, & Tomita, 1995). The arachnoid membrane, the central membrane layer, is extremely thin and fragile. It does not have innervation or vascularization, thus the healing process is difficult. The healing process of the arachnoid membrane is believed to be similar to that of other serous membranes, such as the peritoneal or pleural membrane. The constant flow of cerebrospinal fluid also interferes with the healing process because it washes away the phagocytes and enzymes that prevent the formation of the scar tissue precipitated by inflammation (Delamarter, Ross, Masaryk, Modic, & Bohlman, 1990; Ross et al., 1987). Quiles et al. showed specific pathologic abnormalities in long-standing cases, including lack of dural pulsation; meningeal thickening; absence of cerebral spinal fluid; nerve roots embedded in thick, fibrous tissue; fibrosis; and hyalinization of the arachnoid membrane (Delamarter, 1990; Sharma, et al., 1997).

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Symptoms

Arachnoiditis symptoms are difficult to distinguish from other types of neural compressive diseases, because many of the symptoms are similar and the disease onset can be months to years from the initial injury. Some common symptoms described by patients are:

- Burning pain in the low back that may radiate down the legs
- Pain that persists even at rest
- Urinary urgency, frequency, and incontinence
- Spasms of the back and legs
- Burning in the ankles and feet
- Unexplained skin rashes and itching
- Loss of sensation below the area afflicted
- Partial or complete paralysis of the lower extremities
- Myelograms can frequently induce the same symptoms of numbness, burning, and stinging below the effected site
- Neurologic deficits

Causes

Possible causes of spinal arachnoiditis include infections, trauma, spinal cord contamination, spinal cord tumors, and genetics. Infections can include syphilis, meningitis, tuberculosis, staphylococcus aureus, and candida. Trauma to the spinal cord can be in the form of multiple spine surgeries, epidural injections, intervertebral disk

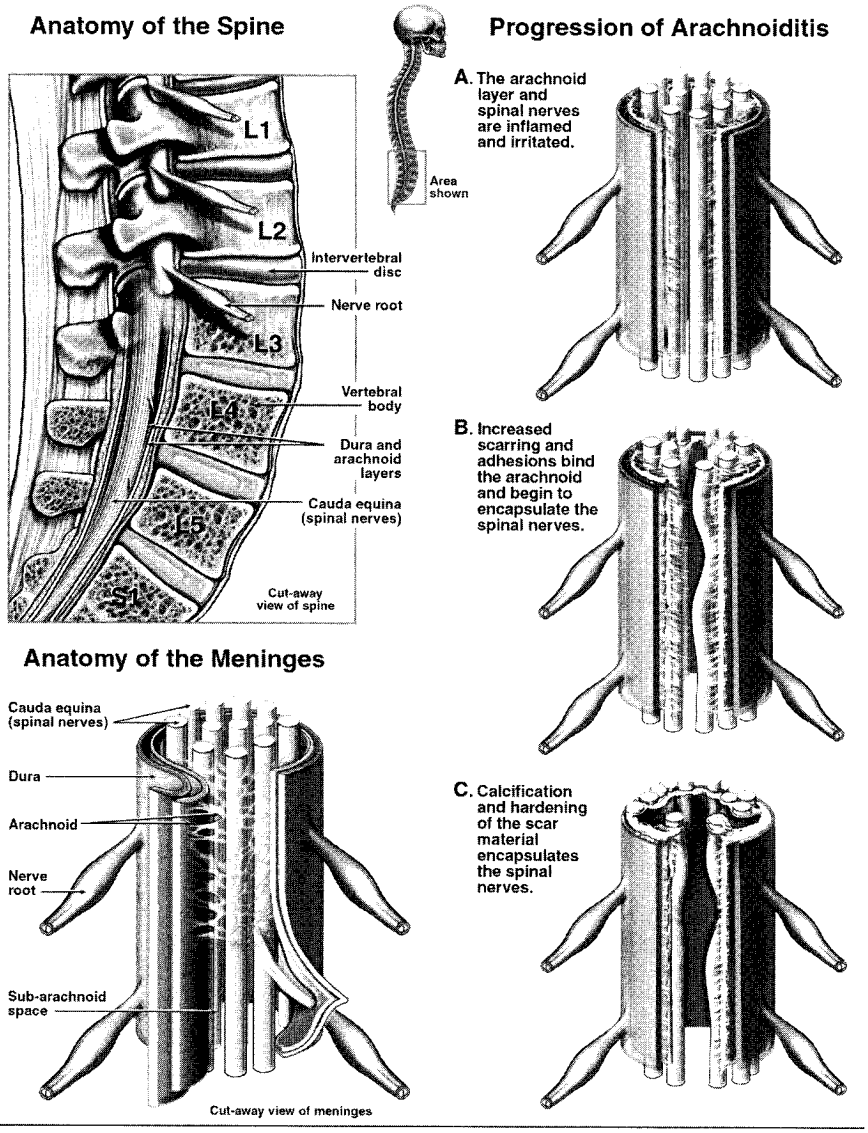

FIGURE 1. Description of the progression of arachnoiditis (Used with permission, Nucleus Communications, 2003).

disease, or epidural anesthesia. Contamination of the spinal cord by introduction of foreign substances, such as steroid injections, radiographic contrast agents, or blood products, may also lead to arachnoiditis. However, the medications are not always the culprit, rather the method of injection, such as injection into the subarachnoid space; the preservatives used in the medications; or contaminants can cause inflammatory reactions (Delamarter et al., 1990).

Genetics

Genetics may also play a role in the disease; familial tendencies toward keloid-like scarring, fibrolytic defects, and idiopathic causes have all been suggested. (Shiraishi, Crock, & Reynolds, 1995). Only a few familial cases have been documented. In one family, six members spanning three generations were documented with no primary event found in any of the patients that could precipitate the disease. The onset of arachnoiditis symptoms was in adulthood and varied from age 18 to age 60 and occurred earlier in the succeeding generations. Symptoms included bladder dysfunction, spastic paraparesis, and radicular complaints of pain and numbness. The genetic trait, as found in this family, is believed to be autosomal dominant (Shikata, Yamamuro, Iida, & Sugimoto, 1989). Still, other studies suggest a fibrinolytic defect as a possible cause of arachnoiditis. Jayson, Keegan, Million, and Tomlinson (1984) have studied the fibrin activity in patients with chronic low back pain and found that those with spondylosis, proven arachnoiditis, and nonspecific back pain exhibited a prolongation of the euglobulin clot lysis time, a decrease in fibrin-plate lysis area and plasminogen levels, and an increase in the levels of fibrinolytic inhibitors *a*2 antiplasmin and *a*2 macroglobulin. These defects prevent the clearing of fibrin from areas of chronic inflammation and result in scarring (Shikata et al., 1989).

Myelograms

In the past, myelograms, used for diagnosing spinal stenosis of multiple etiologies, used oil-based radiographic contrast agents, such as Pantopaque and lipiodol. These dyes have been well documented to cause arachnoiditis, especially when they are not aspirated from the dural space after the procedure (Roca, Moreta, Ubierna, Caceres, & Gomez, 1993; Ross, 1987). The residual oil forms globules, which can become enmeshed in dense scar tissue. Studies have shown that the oily residue remaining after myelograms can result in an irritation reaction and inflammation, which thickens and scars the membranes covering the nerve roots. Recent research has led to the use of isotonic water-soluble dyes, which are less likely to cause arachnoiditis (Roca et al., 1993; Ross, 1987; Toribatake et al., 1995).

Spinal tuberculosis, another cause of arachnoiditis, is most common in third-world countries but is becoming more prevalent in developed nations because of the presence of the human immunodeficiency virus (HIV). Definitive diagnosis can be made using cerebral spinal fluid (CSF) findings of elevated lymphocytes, low glucose levels, and elevated protein levels; symptoms of tuberculosis; myelograms; and MRI or computer-assisted tomography (CAT) scan findings, which may include filling defects and spinal block (de La Blanchardi'ere, Stern, Molina, Lesprit, Gasnault, de Kerviler et al., 1996; Jayson et al., 1984). Isolation of acid-fast bacilli (AFB), however, from the CSF cannot be an early or reliable indicator of tuberculosis because many times it is not present until long after the onset of the disease and after the progres-

sion of edema and scarring has produced irreparable harm. However, long-term illness and spinal damage do not have to be the outcomes of this disease. Treatment initiated early in the disease process can avoid neurologic deficits and the complications of arachnoiditis (Carroll & Wiesel, 1992; Duke & Hashimoto, 1974; Tetsworth & Ferguson, 1986). Treatment includes the use of typical antitubercular drugs and steroids to decrease the inflammatory response (Carroll & Wiesel, 1992; Tetsworth & Ferguson, 1986; Chandra, Jagdish, Sen, Siddhartha, Ravi, Madail et al., 1989).

Treatment of spinal arachnoiditis is difficult for the physician and the patient, because complete pain relief and the alleviation of symptoms remains impossible in most cases.

Radiologic Diagnosis

Visual diagnosis of arachnoiditis is most commonly made using CAT scan (using intravenous contrast dye) and MRI, thus a trend toward noninvasive diagnosis without the potential exacerbation or further irritation of the arachnoid membrane secondary to the direct introduction of dyes to the spinal membranes (i.e., myelograms). The use of MRIs for diagnosis has been divided into classifications based on the appearance of the arachnoiditis. Jorgensen et al. have devised two groups. Type I: the "sleeveless" or short caudal sac appearance results from the adhesion of the roots within the meninges. Type II: partial blockage from scar tissue results in irregular filling defects, producing an appearance of narrowing, shortening, and occlusion in the thecal sac. Type I is believed to indicate mild disease and type II more extensive disease (Shiraishi et al., 1995; Toribatake, 1995;). A second classification system has been developed by Delamarter to further divide the disease divided into three groups and has also been used to correlate the extent of the disease from mild to severe:

- Group I—appears as an adhesive mass of adherent roots centrally in the thecal sac, considered mild arachnoiditis.
- Group II—the empty sac, the roots are adhered peripherally to the meninges.
- Group III—a soft-tissue mass replaces the subarachnoid space. (This corresponds to Jorgensen type 2)

The use of axial TI-weighted images is the most efficient imaging sequence to diagnose arachnoiditis and allows the radiographer a clear image to distinguish the patterns of nerve root changes as described.

Finally, a third method of classification has been developed by Wilkinson, which divided the myelographic appearances into four groups that correlate with specific physiologic and anatomic appearances.

- Type I: A unilateral focal defect centered on the nerve root exit pouch adjacent to the intervertebral disc space.
- Type II: An annular or a circumferential defect with a bilateral notch and only filiform passage of the medium.
- Type III: Complete transverse obstruction with the picture of stalagmites, candle-guttering, or paintbrush defects.
- Type IV: An infundibuliform cul-de-sac with loss of radicular striation, a vitreous appearance, and cutting-off of the root sleeves.

The appearance of the MRI has also been correlated to the physical symptoms of the patients. Those patients exhibiting mild defects radiographically may only have symptoms with the presence of a secondary lesion, such as a herniated disc or spinal stenosis. Patients with more severe disease of types II and III were more likely to have leg pain, absent lese ague sign, weakness, and neurologic deficits. Finally, those with type IV exhibited dysesthesia. (Shiraishi et al., 1995) The advent of MRI and CT scans has decreased the need for use of myelograms in diagnosing this disease, thus helping to eliminate a potential cause of arachnoiditis (Ross et al., 1987).

Differential Diagnosis

The differential diagnoses often associated with spinal arachnoiditis are spinal cord tumors, cauda equina syndrome, arachnoiditis ossificans, syringomyelia, and failed back syndrome. These complications are usually found in more severe cases of the disease.

Spinal Cord Tumor

In advanced arachnoiditis, the symptoms may mimic other diseases, as was seen by Vloeberghs, Herregodts, Stadnik, Goossens, and D'Haens (1992). In this case, a spinal tumor was believed to be the cause of the patient's symptoms and radiographic imaging could not differentiate the diagnosis from that of a tumor, so surgery was scheduled. A laminectomy from D9 to L3 was performed. Upon opening the dural sac, a dense fibrous tissue was found that encased the cauda equina, the terminal cone, and part of the spinal cord, but no tumor was found. The tissue was mostly avascular, without infection or hemorrhage. Multiple biopsies were taken, and cysts were found but not dissected. The patient had neurologic improvement after surgery and for the following year but had recurrent symptoms the next year similar to those before the surgery. This case illustrates the need for careful diagnosis in evaluating cases of arachnoiditis that may imitate other spinal diseases or a spinal cord tumor (Martin & Yuan, 1996). The chronic compression exhibited by spinal cord tumors is another causative mechanism of arachnoiditis (Brammah & Jayson, 1994).

Complications

Cauda Equina Syndrome

Cauda equina syndrome is considered a late symptom of arachnoiditis, with symptoms of bowel and bladder dysfunction, paresthesias, and paralysis of the lower limbs. Scarring results in the inability of the caudal sac to respond to the changes in CSF pressure and in enlarged arachnoid diverticula and bone erosion. The flow of spinal fluid and vascular circulation is inter-

rupted. Nerve root damage is caused by compression injuries from long-standing arachnoiditis and ischemic changes resulting from the loss of elasticity of the caudal sac. Presently, the treatment for long-standing cauda equina syndrome is to treat the symptoms. No improvement has been observed with the use of anti-inflammatory drugs (Martin & Yuan, 1996). Surgical lysis has also been performed; however, the symptoms often recur with the reformation of adhesions, thus, it is rarely used. If, however, the symptoms of cauda equina syndrome are closely related to a lumbar disc surgery, it is possible that the artery of Adamkiewicz may be injured or cauterized and a hematoma may have occurred. In this instance, a myelogram or MRI imaging should be performed quickly to confirm or deny the diagnosis, because the paralysis can be permanent if it is not treated immediately. If there is such an injury, it is imperative to return to surgery quickly. According to Carroll & Wiesel (1992) when performing surgery, it is important to handle the tissues of the spinal cord with care and to maintain a dry, controlled exposure to prevent cauda equina syndrome. Hematoma formation is also a concern, and the insertion of a Hemovac drain is recommended if there is any suspicion of bleeding from the immediate postoperative site (Jackson & Isherwood, 1994).

Arachnoiditis Ossificans

Arachnoiditis Ossificans, another complication of arachnoiditis, refers to the calcification and ossification of the spinal meninges from chronic changes associated with arachnoiditis. An intrathecal bony metaplasia of the arachnoid membrane occurs, causing compression of the spinal nerve roots resulting in severe symptoms of arachnoiditis, including irrepressible pain, paraparesis, and bowel and bladder incontinence (Tetsworth & Ferguson, 1986; Martin & Yuan, 1996). The causes of arachnoiditis ossification are believed to be the same as for arachnoiditis; however, the cause of ossification is not fully understood. Toribatake et al. (1995) found a localized increase in clusters of arachnoid cells surrounding the ossified lesion, accompanied by a proliferation of osteoblasts.

If the patient is symptomatic, the treatment is surgical resection of the ossified plaques as soon as possible if there is any hope of reversing the symptoms related to the nerve compression injuries. A microsurgical technique is recommended to resect the ossified plaque, with watertight closure of the dura mater. However, the importance of complete imaging before surgery cannot be over stressed, so complete resections of all areas of ossified plaque are removed initially. Repeat surgical procedures can lead to recurrence of the original problem (Fritsch, Heisel, & Stefan, 1996; Klekamp, Batzdorf, Samii, & Bothe, 1994).

Syringomyelia

Syringomyelia, or cyst of the spinal cord, is a rare complication of arachnoiditis, which can also be associated with a Chiari malformation and is believed to be a congenital defect. A Chiari malformation is characterized by the cerebellar tonsils' downward displacement beneath the foramen magnum into the cervical spinal canal, which blocks the flow of CSF. The malformation can, but does not always, lead to the development of a cyst. Surgically, the malformation can be treated by craniocervical decompression.

Syringomyelia can also be caused by arachnoiditis when the scarring causes changes in the flow of CSF and/or the vascular supply (Bourne, 1990; Vleoberghs et al., 1992). The blockage may also be mistaken for a spinal cord tumor. Treatment has included shunting of the syrinx to the subarachnoid, pleural, or peritoneal space, but this has been associated with poor long-term outcomes, especially in the advanced cases of arachnoiditis when severe symptoms have already developed. Continued close observation of these patients is necessary, because displacement or blockage of the shunt is not uncommon. Shunting cannot reverse the symptoms; it only prevents the progression of more severe complications (Vleoberghs et al., 1992).

Failed Back Syndrome

Failed back surgery syndrome (FBSS) is the continuation or return of previous back pain symptoms after primary back surgery. Arachnoiditis is believed to be common in FBSS (Dolan, 1993). These patients often present with histories of repeated lumbar surgeries and myelograms and physical symptoms that include sensory defects and irrepressible pain. The success of repeated lumbar surgeries in patients with FBSS is poor. After surgery, those patients with spinal arachnoiditis typically have only 1 to 6 months of pain relief before an insidious return of the previous pain and symptoms. Patients who have already developed scar tissue are not considered good candidates for surgery, because the scar tissue generally returns after the procedure in even greater amounts (Dolan, 1993; Ghirlanzoni, Marazzi, Pareyson, Olivieri, & Bracchi, 1989).

Care of the Patient

Treatment of spinal arachnoiditis is difficult for the physician and the patient, because complete pain relief and the alleviation of symptoms remains impossible in most cases. Methods of treatment include pain management with narcotics, steroids and spinal cord stimulation, and, in severe cases, surgical removal of scar tissue. Surgical intervention is regarded as a last resort after all other methods of conservative treatment have been exhausted.

Direct Spinal Cord Stimulation

Direct spinal cord stimulation (DCS) has provided an alternative to narcotics in long-term pain control. The mechanism of pain relief has not been established, but it is believed to be synaptic inhibition. Patients report an average reduction of their pain by 50% in studies of up to 7 years after the insertion of the device. The DCS is inserted in the surgical setting. Because of a limited success rate of approximately 50%, a 5- to 7-day trial with a temporary device is necessary. Two temporary electrodes are placed subcutaneously with local anesthetic, and the device is enabled. The patient then evaluates the device for several days, and the device can be easily removed if pain increases or does not change. A paresthesia is needed to cover the affected area of pain and, because the patient has been given only local anesthesia, he or she can assist in

this evaluation. Once it is established that the patient will benefit from the device, a partial laminectomy is scheduled, the electrodes are placed at the appropriate level for the patient, a subcutaneous tunnel is formed to the soft tissue at the superior aspect of the gluteal area, and the generator is inserted (Ghirlanzoni et al., 1989).

Microlysis

Surgical treatment of spinal arachnoiditis is difficult and considered a last resort, especially for patients with advanced disease, because the prevention of further scarring is imperative. Surgical techniques have included microsurgical lysis with a possible shunt, laminectomy, dural decompressive grafting, and posterolateral fusion with instrumentation (Brammah & Jayson, 1994). Shikata et al. (1989) describe a surgical method for the treatment of arachnoiditis that includes a laminectomy and microlysis, with a shunt placed when needed to connect the affected area with the normal site below, followed by dural decompressive grafting with Lyodura and posterolateral fusion with instrumentation. The addition of fusion was made because their study noted a marked improvement in those patients receiving fusion in addition to the laminectomy and microlysis. Their study also suggests a correlation between spinal mobility and the development of arachnoiditis. This method of surgical treatment produced an 80% success rate, with an average follow-up period of more than 4 years. Microsurgical lysis and laminectomy offer another alternative to treating the disease but often result in only temporary improvement for those patients with milder arachnoiditis disease and often no improvement in those with more severe arachnoiditis. Wilkinson reported only a 75% improvement postoperatively in 17 patients, 18% of whom having new neurologic deficits after the procedure and only 50% maintaining the improvements at the 1-year follow-up (Brammah & Jayson, 1994; Ghirlanzoni et al., 1989; Martin & Yuan, 1996; Shiraishi, 1995).

Discussion

The prognosis is poor for patients with spinal arachnoiditis. There is no cure, and surgical intervention may provide only temporary relief, with the scarring returning after a short period of time. The patients are at risk for depression, suicide, and drug and alcohol abuse as a result of enduring pain for years at a time and the disabilities associated with the disease. The lifespan of the patient may be decreased as much as 12 years. Prevention of the disease is difficult and cannot be reliable if there are, in fact, genetic factors, but it has been stated that careful handling of the neural elements during surgery and the prevention of trauma to the spinal meninges from puncture, hematoma, and exposure to foreign substances may reduce the incidence of spinal arachnoiditis.

References

Bourne, I. (1990). Lumbo-sacral adhesive arachnoiditis: A review. *Journal of the Royal Society of Medicine, 83,* 262–265.

Brammah, T., & Jayson, M. (1994) Syringomyelia as a complication of spinal arachnoiditis. *Spine, 29,* 2603–2605.

Burton, C. V. (1978). Lumbosacral arachnoiditis. *Spine, 3,* 24–30.

Carroll, S. E., & Wiesel, S. (1992) Neurologic complications and lumbar laminectomy: A standardized approach to the multiply-operated spine. *Clinical Orthopaedics and Related Research, 284,* 14–23.

Chandra, J., Sen, S., Madail Ravi, R. N., et al. (1989) Tubercular spinal arachnoiditis with radiculomyelopathy. *The Indian Journal of Pediatrics, 56,* 670–674.

de La Blanchardi'ere, A., Stern, J. B., Molina, J. M., Lesprit, P., Gasnault J., de Kerviler, E., et al. (1996). Spinal tuberculosis arachnoiditis. *Presse Med, 25,* 1333–1335.

Delamarter, R., Ross, J., Masaryk, T., Modic, M., & Bohlman, H. (1990). Diagnosis of lumbar arachnoiditis by magnetic resonance imaging. *Spine, 15,* 4304–4310.

Dolan, R. (1993). Spinal adhesive arachnoiditis. *Surgical Neurology, 39,* 479–484.

Duke, R., & Hashimoto, S. (1974). Familial spinal arachnoiditis. *Archives of Neurology, 30,* 300–303.

Fritsch, E., Heisel, J., & Stefan, R. (1996). The failed back surgery syndrome. Reasons, intraoperative findings, and long-term results: A report of 182 operative treatments. *Spine, 21,* 626–633.

Ghirlanzoni, A., Marazzi, R., Pareyson, D., Olivieri, A., & Bracchi, M. (1989). Epidural anesthesia and spinal arachnoiditis. *Anesthesia, 44,* 317–321.

Guyer, D., Wiltse, L., Eskay, M., & Guyer, B. (1989). The long-range prognosis of arachnoiditis. *Spine, 14,* 1332–1340.

Jackson, A., & Isherwood, I. (1994). Does degenerative disease of the lumbar spine cause arachnoiditis? A magnetic resonance study and review of the literature. *The British Journal of Radiology, 67,* 840–847.

Jayson, M., Keegan, A., Million, R., & Tomlinson, I. (1984). A fibrinolytic defect in chronic back pain syndromes. *Lancet, 2,* 1186–1187.

Klekamp, J., Batzdorf, U., Samii, M., & Bothe, H. (1997). Treatment of syringomyelia associated with arachnoid scarring caused by arachnoiditis or trauma. *Journal of Neurosurgery, 86,* 233–240.

Martin, R., & Yuan, H. (1996) Neurosurgical care of the spinal epidural, subdural and intramedullary abscesses and arachnoiditis. *Orthopedic Clinics of North America, 27,* 125–136.

Phadke, R., Kohli, A., Jain, V., Gupta, R., Kumark, S., & Gujral, R. B. (1994). Tuberculous radiculomyelitis (arachnoiditis): Myelographic (and CT myelographic) appearances. *Australian Radiology, 38,* 10–16.

Roca, J., Moreta, D., Ubierna, M., Caceres, E., & Gomez, J. (1993). The results of surgical treatment of lumbar Arachnoiditis. *International Orthopaedics, 17,* 77–81.

Ross, J., Masaryk, T., Modic, M., Decameter, R., Bohlman, H., Wilbur, G., et al. (1987). MR imaging of lumbar arachnoiditis. *AJR Am J Roentgenol,* 149, 1025–1032.

Sharma, A., Goyal, M., Mishra, N. K., Gupta, V., Gaikwad, S. B. (1997). MR imaging of tubercular spinal arachnoiditis. *AJR Am J Roentgenol, 168,* 807-812.

Shikata, J., Yamamuro, T., Iida, H., & Sugimoto, M. (1989). Surgical treatment for symptomatic spinal adhesive arachnoiditis. *Spine, 14,* 870–875.

Shiraishi, T., Crock, H., & Reynolds, A. (1995). Spinal arachnoiditis ossificans. Observations on its investigation and treatment. *The European Spine Journal, 4,* 60–63.

Tetsworth, K., & Ferguson, L. (1986). *Arachnoiditis Ossificans* of the cauda equina. *Spine,11,* 765–766.

Toribatake, Y., Baba, H., Maezawa, Y., Umeda, S., & Tomita, K. (1995). Symptomatic arachnoiditis ossificans of the thoracic spine. Case report. *Paraplegia, 33,* 224–227.

Vleoberghs, M., Herregodts, P., Stadnik, T., Goossens, A., & D'Haens, J. (1992). Spinal arachnoiditis mimicking a spinal cord tumor: A case report and review of the literature. *Surgical Neurology, 37,* 211–215.

CE Test

A Comprehensive Review of Spinal Arachnoiditis

Instructions:
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CE TEST QUESTIONS

GENERAL PURPOSE: To provide a comprehensive review of the causes, diagnosis, and treatment of spinal arachnoiditis.

LEARNING OBJECTIVES: After reading the article and taking this test, you will be able to:

1. List causes of spinal arachnoiditis, differential diagnoses, and complications.
2. Outline testing procedures and classification systems used to diagnose spinal arachnoiditis.
3. Describe patient care and treatment options for spinal arachnoiditis and the various complications of this condition.

1. Spinal arachnoiditis
a. is a disease characterized by inflammation and scarring of the arachnoid membrane of the spinal cord.
b. is a cyst of the spinal cord and can be associated with a Chiari malformation.
c. occurs when scarring causes changes in the flow of cerebrospinal fluid.
d. refers to the calcification and ossification of the spinal meninges.

2. In spinal arachnoiditis, damage occurs when an inflammatory response triggers
a. fibrinous exudates.
b. loss of elasticity.
c. neural compression.
d. ossified plaques.

3. Symptoms of arachnoiditis include
a. urinary retention.
b. pain relieved by rest.
c. loss of sensation above the area affected.
d. partial or complete paralysis of lower extremities.

4. Studies by Jayson et al. (1984) on the fibrin activity in chronic low back pain patients identified
a. a localized increase in clusters of arachnoid cells surrounding the ossified lesion.
b. an increase in the levels of fibrinolytic inhibitors *a2* antiplasmin and *a2* macroglobulin.
c. that the lifespan of the patient with arachnoiditis may be decreased by as much as 5 years.
d. that the artery of Adamkiewicz may be injured in patients with spondylosis.

5. Direct introduction of dyes to the spinal membranes is used for
a. axial TI-weighted images.
b. CT scans.
c. MRI imaging.
d. myelogram.

6. Visual diagnosis of arachnoiditis is most commonly made using MRI and
a. axial TI-weighted images.
b. CT scan.
c. CSF analysis.
d. myelogram.

7. According to Delamatar (1990), mild arachnoiditis occurs when
a. a soft tissue mass replaces the subarachnoid space.
b. an adhesive mass of adherent roots appears centrally in the thecal sac.
c. partial blockage from scar tissue results in irregular filling defects.
d. the roots are adhered peripherally to the meninges.

8. The most efficient imaging sequence to diagnose arachnoiditis has been shown to be
a. axial TI-weighted images.
b. CT scans.
c. MRI imaging.
d. myelograms.

9. In Wilkinson's classification system, dysesthesia is usually exhibited in patients with
a. Type I.
b. Type II.
c. Type III.
d. Type IV.

10. In cauda equina syndrome
a. treatment with antiinflammatory drugs has proved to be effective.
b. nerve root damage is caused by compression injuries from long-standing arachnoiditis.
c. the flow of spinal fluid and vascular circulation is not compromised .
d. there is no evidence of paresthesias or paralysis of the lower limbs.

11. Recommendations for treatment of symptomatic patients with arachnoiditis ossificans include
a. direct spinal cord stimulation (DCS).
b. dural decompressive grafting.
c. insertion of a Hemovac drain.
d. microsurgical resection of the ossified plaque.

12. **Which statement about syringomyelia is true?**
    a. Back surgery, including repeated lumbar surgeries, is the treatment of choice.
    b. Shunting of the syrinx to the subarachnoid, pleural, or peritoneal space is associated with poor long-term outcomes.
    c. It is a common complication of arachnoiditis.
    d. It is easily managed with the use of antiinflammatory drugs.

13. **Once it is established that the patient will benefit from DCS**
    a. a partial laminectomy is scheduled.
    b. MRI imaging should be performed.
    c. multiple biopsies are taken.
    d. surgery is scheduled to open the dural sac.

14. **Shikata et al. (1989) identified a correlation between spinal mobility and the development of**
    a. arachnoiditis.
    b. cauda equina syndrome.
    c. spinal cord tumor.
    d. tuberculosis.

15. **How many patients maintained improvement at 1 year after treatment with microsurgical lysis and laminectomy for spinal arachnoiditis?**
    a. 18%
    b. 50%
    c. 75%
    d. 80%

16. **Which statement about arachnoiditis is true?**
    a. Complete pain relief remains impossible in most cases.
    b. Surgical intervention is the initial treatment of choice.
    c. Patients' life spans are not affected by this condition.
    d. Lifestyle modifications can prevent the occurrence of this condition.

CE Enrollment Form

Orthopaedic Nursing, May/June 2003

A Comprehensive Review of Spinal Arachnoiditis

A **Registration Information:**

Last name_____ First name_____ MI____

Address_____

City_____ State_____ Zip_____

Telephone_____ Fax_____ email_____

Registration Deadline: June 30, 2005

Contact Hours: 2

Fee: NAON member: $10.00 ☐

Expiration Date: _____
from NAON membership card

Nonmembers: $20.00 ☐

❑ LPN ❑ RN ❑ CNS ❑ NP ❑ CRNA ❑ CNM ❑ other_____

Job Title_____ Specialty_____

Type of facility_____

Are you certified? ❑ Yes ❑ No

Certified by_____

State of License (1)_____ License #_____

State of License (2) _____ License#_____

Social Security #_____

❑ From time to time we make our mailing list available to outside organizations to announce special offers. Please check here if you do not wish us to release your name and address.

B **Test Answers:** Darken one for your answer to each question.

	A	B	C	D		A	B	C	D		A	B	C	D		A	B	C	D
1.	○	○	○	○	5.	○	○	○	○	9.	○	○	○	○	13.	○	○	○	○
2.	○	○	○	○	6.	○	○	○	○	10.	○	○	○	○	14.	○	○	○	○
3.	○	○	○	○	7.	○	○	○	○	11.	○	○	○	○	15.	○	○	○	○
4.	○	○	○	○	8.	○	○	○	○	12.	○	○	○	○	16.	○	○	○	○

C **Course Evaluation***

	A	B
1. Did this CE activity's learning objectives relate to its general purpose?	❑ Yes	❑ No
2. Was the journal home study format an effective way to present the material?	❑ Yes	❑ No
3. Was the content relevant to your nursing practice?	❑ Yes	❑ No

4. How long did it take you to complete this CE activity?____ hours____minutes

5. Suggestion for future topics_____

D **Two Easy Ways to Pay:**

❑ Check or money order enclosed
(Payable to Lippincott Williams & Wilkins)

❑ Charge my ❑ Mastercard ❑ Visa ❑ American Express

Card #_____ Exp. Date_____

Signature_____

*In accordance with the Iowa Board of Nursing Administrative rules governing grievances, a copy of your evaluation of the CE offering may be submitted directly to the Iowa Board of Nursing.

Need CE STAT? Visit www.NursingCenter.com for immediate results, other CE activities, and your personalized CE planner tool!