THE WAY FORWARD

FOR ADHESIVE ARACHNOIDITIS

CONFRONTING AN URGENT PUBLIC HEALTH ISSUE

Summary

Adhesive arachnoiditis (AA) has become an urgent public health issue. It now affects millions and is increasing in number. Unlike other causes of back pain, AA is an inflammatory disease inside the spinal canal which, if untreated and unchecked, may cause unbearable pain, dysfunction of legs and internal organs, dementia, a generalized autoimmune disorder, and adrenal failure. All its complications can lead to a suffering, shortened lifespan. This report calls for immediate action to diagnose, treat, and prevent AA.

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INTRODUCTION

A leadership conference was held in Gulfport, Mississippi in November 2019 to develop a consensus and "way forward" for adhesive arachnoiditis (AA) of the lumbar-sacral spine. * Until now AA has been considered a rare disease. It was originally described in the mid-1800's but began to re-emerge about 10 years ago as a growing and significant disease. AA is now an urgent public health issue due to the large numbers of persons who have it, and if left untreated, AA is usually a tragic, catastrophic, and life-shortening disease. In contrast, compared to common back problems, AA causes neuroinflammatory disease of the spinal canal that impairs multiple organs, causes autoimmunity, creates endocrine abnormalities, dementia, and early death. Unfortunately, palliative care and intractable pain clinics have recently started to identify growing numbers of patients with AA, as most have severe pain. There are now so many persons with AA that they have formed self-help and support groups primarily through social media numbering in thousands of participants. Like many other public health issues, AA has unexpectedly and "silently" emerged.[1] The purpose of the Leadership Conference was to bring together leaders of support and advocacy groups to share beliefs and experiences and develop a "way forward" to confront this new and urgent public health issue. This report summarizes the consensus of the Leaders, the current status of the disease, and some future recommendations to more vigorously confront AA.

HOW MANY PERSONS HAVE AA?

By our investigation and research, we estimate that at least 1.75 to 2.75 million, and possibly as many as 7-8 million adult Americans now have AA. The majority are not yet aware they have the disease. Our arrival at this estimate is based on the referenced epidemiologic studies.[2-28]

First, it is clear that chronic back pain is rising in prevalence in the general population, so AA has to be rising as back pain is a basic symptom of AA.[2,7,9,13,23,24,25] For example, it is estimated that from 2000 to 2007, the total number of adults in the USA with chronic back pain increased by 64% from 78 to 12.8 million.[24] In a study by Richard Nahin of the National Institute of Health, 14.4 million adults were classified as having the most severe, disabling form of pain.[19] An additional 25.4 million adults had chronic pain in a slightly less severe category. Both groups had almost daily pain severe enough to interfere with functions of normal living. About 50 to 80% of adults who claim severe pain have chronic back pain as their cause.[18] This translates to between 7 and 11 million adults who have back pain in the most severe category. At this time it is unclear as to the precise underlying causes of the 7 to 11 million adults who have severe, disabling back pain, but AA is a significant cause.[26] Other causes may include herniated discs, spondylolisthesis, osteoporosis, rheumatoid spondylitis, and scoliosis, but AA must be considered a major, if not the major cause of severe, disabling back pain. In fact, all the common spine conditions listed here may develop into AA. AA is almost always preceded by an anatomic, structural abnormality of the spine. For this reason, our working, conservative estimate is that at least 25% of the 7 to 11 million adults have AA. This translates to a prevalence of AA of between 1.75 and 2.75 million adults.

**This report only refers to lumbar-sacral arachnoiditis and not to other anatomic locations such as cervical, thoracic, and brain arachnoiditis.*

Other studies suggest that our estimate may be low. Dahlhamer and colleagues from the US Center for Disease Control estimated that in 2016 8.0% or 19.6 million US adults had "high impact chronic pain".[5] The same calculations done above would indicate that 9.8 to 15.68 million adults have high impact chronic back pain. If only 25% of the high impact back pain adults have AA, this puts the prevalence at 2.45 to 3.92 million adults. Pitcher and colleagues from the National Institute of Health determined the prevalence of high-impact chronic pain in the general population.[20] High impact was defined as pain that is daily and disabling. They found that 4.8% of the US adult population or approximately 10.6 million adults in 2011 had this condition. We have found that AA is now the major (64.0%) cause of persons who require high dose opioids.[26] In summary, we do not have a specific prevalence percentage of adults with AA in the US general population, but our analysis conservatively supports a number of at least 1.75 million adults with AA.

A supportive survey is the percentage of persons who develop AA after lumbosacral injections. Friedly et al reviewed lumbosacral injections in the Medicare population between 1994 to 2001.[10] Lumbosacral injections increased 271% during this period going from 553 of 100,000 in 1994 to 2055 of 100,00 in 2001. There were over 44 million beneficiaries in 2004. Assuming about 40 million beneficiaries in 2001, there were a total of 822,000 lumbosacral injections given that year. Some experts believe that 6 to 16% of persons who have these injections will develop AA.[8,22] This means that 49,320 to 131,520 Medicare beneficiaries developed AA in just 2001. If this rate was maintained over the past 19 years, the number of Medicare beneficiaries with AA would range between 937,000 and 2,500,000. While these figures are a rough estimate, certainly support numbers of at least 1.75 to 2.75 million adults with AA.

It is also critical to point out that not only have lumbosacral injections greatly increased, so have lumbar infusions and laminectomies. Between 1998 and 2008, primary lumbar fusions increased by about 170% and laminectomies increased about 11%.[21] Unfortunately, at least 20 to 40% of persons who undergo these surgeries do not achieve much pain relief.[3,4]

It is interesting and perhaps worth noting that arachnoiditis was previously called, in 2002, by Dr. Antonio Aldrete, a "silent epidemic".[1] Today the epidemic is apparently shedding its silence.

CONSENSUS POSITIONS 2020

Listed here are 17 consensus positions drafted to address needs and a "Way Forward".

1. AA is an urgent public health issue that affects at least 1.75 to 2.75 and possibly as many as 7-8 million adults. It can no longer be considered a rare disease.

2. AA is a treatable, inflammatory disease of the lower spinal canal. The notion that "nothing can be done" and that afflicted persons must "learn to live with it" are rejected.

3. AA is grossly underdiagnosed. Most persons who have AA are not yet aware they have it. All medical practitioners should know the symptoms and complications that call for a clinical and diagnostic evaluation.

4. Persons with AA need diagnosis and treatment as early as possible after onset of the disease to retard progression and complications.

5. AA has specific, typical symptoms such as those listed here and should be known to all medical practitioners: (1) severe radiating back pain; (2) water/bugs sensation on skin; (3) urination dysfunction; (4) burning feet; (5) headache/blurred vision; and (6) electric shock sensation on turning or bending. A recommended screening test to be used by medical practitioners is given in these proceedings.

6. AA is a neuroinflammatory disease of the cauda equina nerve roots and the arachnoid/dural covering of the spinal canal. It has its own International Classification of Diseases Code (ICD) G03.9. Therefore, AA treatment should be reimbursed by governmental and private health insurance plans.

7. Common sense medical treatment should include medical and physiologic measures including a 3-component medical protocol: (1) suppression of neuroinflammation, (2) promotion of neuroregeneration, and (3) pain control, and special physiologic measures to enhance spinal fluid flow, reduce retained electricity, increase oxygen intake, and maintain function of arms and legs.

8. Emergency therapeutic administration of potent anti-neuroinflammatory medications such as methylprednisolone and ketorolac should be immediately administered if a person develops AA-type symptoms after a surgery, lumbar puncture, lumbar injection, or epidural injection.

9. AA is now so common that treatment must be carried out in a variety of ambulatory health care settings, by a number of medical practitioners in every community.

10. Treatment benefits often outweigh the risks of medications used to treat AA.

11. Persons with AA must have a vigorous self-care program.

12. Implanted spinal cord electrical stimulators, intrathecal opioid infusion devices, and the experimental treatments such as stem cells should be “add-on” or last resort treatments to basic, common sense medical treatment of suppression of neuroinflammation, promotion of neuroregeneration, and symptomatic pain care.

13. Persons with AA who seek disability or worker’s compensation awards should seek the help of attorneys who specialize in these claims.

14. Invasive spinal interventions and surgeries, both diagnostic and therapeutic, should only be instituted as a last resort in persons known to have AA.

15. Palliative care must be provided for AA patients if they are in the catastrophic stage of AA with nutritional wasting and a bed-bound state.

16. Educational seminars for persons with AA, families, and medical practitioners should be started to facilitate the spread and awareness of current knowledge about AA.

17. More research is needed to identify and improve upon the current treatment protocols to identify and more effectively utilize the anti-neuroinflammatory and neuroregenerative agents.

THE DISEASE

AA is a neuroinflammatory disease of the lower spinal canal which causes the nerve roots of the cauda equina to merge with and adhere to the arachnoid/dural covering (meninges) of the spinal canal. The underlying, causative factor is the formation of adhesions that act as "glue" to attach the two anatomical structures forming a bundle or mass of tissue. The inflammation and adhesions which cause attachment of the two anatomic structures entraps and destroys nerve root tissue and produces varying degrees of pain. The merger creates an inflammatory, adhesive mass that is formed within the spinal canal that interferes with spinal fluid flow and may erode the arachnoid/dural covering and seep spinal fluid into the non-neural tissues that surround the spinal column. If left untreated, the inflammatory, adhesive mass may mimic a cancer in that it continues to grow and may progressively entrap and destroy more and more neural tissue. This can result in lower extremity paraparesis, bladder, bowel, and sexual dysfunction, headaches, blurred vision, autoimmune reactions, endocrine abnormalities, and overwhelming pain that may greatly inhibit activities of daily living. Early death from autoimmune deficiencies, overwhelming infections and endocrine failure may result. The mass may initially grow without producing many more symptoms, but at some point, too many nerve roots are destroyed so immense pain and disability may unexpectedly appear. It is now recommended that a person with AA take anti-neuroinflammatory agents to prevent the inflammatory mass from enlarging and causing more neurologic complications.

IMPETUS AND REASONS FOR THE CONFERENCE

The basic reason for the conference is that AA is an urgent public health issue. Leaders have increasingly observed that persons with AA are growing in number. AA can no longer be considered a "rare" disease as it is now being recognized in every community and in essentially every medical practice. Those persons who are afflicted are obtaining various degrees of relief and recovery with new treatment protocols that have been urgently developed to counter the disease. This is a major turning point because there has been the belief for over a century that "nothing can be done for AA". In fact, the general medical belief and message to those afflicted has been to take whatever symptomatic pain medications may be available, since nothing else

can apparently be done beyond relieving the pain of AA. In other words, “live with it, accept it, and expect a short life”.

The treatment experiences and input of persons who have AA as voiced through social media has influenced the leaders of the conference to expressly reject this old notion. Consequently, a major consensus message of the conference is to broadcast the observation that AA is treatable, and that the notion that, “nothing can be done”, or “there is no proven treatment” is to be explicitly rejected.

BRIEF HISTORY OF AA

AA was first described in the mid-1800’s when inflammation and adhesions were found in the spinal canal of victims who died with the disease. Medical dictionaries described the disease after about 1860. The main cause of AA in the 19th century was primarily the infections of tuberculosis and syphilis. These diseases were nearly eradicated in the 20th century due to the discovery of antibiotics. Between about 1930 and 1990 AA was rarely diagnosed and observed. When it was observed it was due to the injection of oil-based dyes into the spinal canal to enhance x-rays known as myelograms.

Magnetic Resonance Imaging (MRI) was invented in 1987. It essentially eliminated the rare case caused by oil-based dyes. Consequently, AA became known as a rare disease and it was listed on the registry of the National Organization of Rare Disorders (NORD).

Due to the rarity of AA since the advent of MRI’s in the late 20th century, few physicians have been aware of it, much less know how to diagnose and treat it. This is a challenge that must be met as AA has begun to re-emerge and increase in incidence and prevalence in the 21st century due to various factors. Action to begin diagnosis and treatment of AA is urgently needed.

WHY AA IS INCREASING

Although the precise incidence and prevalence of AA is unknown, it appears to be increasing at a progressive rate throughout the modern world.[2,9,23,25] The major reasons why AA is increasing are aging; sedentary lifestyles, structural anatomic abnormalities that support the spinal canal, and the increased use of spinal surgeries, and lumbosacral injections (Table One).

Another reason for the seeming increase in incidence and prevalence is that AA is now being recognized much more often. Contrast MRI technology allows a diagnosis of AA to be made when previous generations of MRI and myelograms were less efficient. AA has a rather unique set of symptoms that allows persons with the disease, and medical practitioners to suspect and evaluate for its presence.

SPECIFIC CAUSES OF ADHESIVE ARACHNOIDITIS

In the past decade, the specific basic causes of AA have been identified and are listed here. An individual may have more than one causative condition.

Degenerative Spine Conditions

Common spine disorders including chronic herniated discs, stenosis, osteoporosis, scoliosis, and vertebral arthritis may, over time, cause nerve roots in the cauda equina to rub and be compressed together causing friction ("sand-paper effect"), which results in inflammation and adhesions.

Genetic Connective/Collagen Disorders

The arachnoid and pia mater layers of the spinal covering are thin, soft, and easily damaged, because they are composed primarily of very soft and frail tissue compared to such tissues as tendons and bone. Genetic or connective tissue/collagen disorders that cause tissue fragility and degeneration, particularly Ehlers-Danlos and Marfan Syndromes, may cause collagen dissolution and tissue micro-tears in the arachnoid layer which leads to inflammation, cysts, (Tarlov), and adhesions.

Autoimmune Disease

Some AA patients have autoimmune disease such as systemic lupus, psoriasis, or rheumatoid arthritis. AA may be a direct result of autoimmunity.

Infections

Various bacterial and fungal infections are known causes of AA. Cases of disseminated coccidioidomycosis (Valley Fever), various bacteria, and Lyme have been reported to cause AA. It is interesting that the epidemic of Lyme Disease has somewhat paralleled the rise of AA. Some viruses are also highly suspected to cause AA, as many persons with AA have very high viral titers to various viruses. It may be that some viruses such as Epstein Barr, cytomegalus, and others are cloistered in cauda equina nerve roots and activate with trauma or other physiologic insult to the lower spine and cause AA.

Trauma

A puncture, tear, or trauma injury to the arachnoid-dural covering of the spinal canal lining from an accident, needle puncture, or chemical irritant may initiate AA. Inflammation and adhesions of the arachnoid lining may later capture the nerve roots that are close to the inflamed site and form an adhesive mass.

Epidural Injections

It is clear that epidural injections may cause AA. Just how common this may occur and if there are underlying conditions that should contraindicate an epidural injection are unclear. The clarification and classification of this critical issue is an immediate need as the rise in epidural injections and AA seem to have occurred in parallel.

Surgical Interventions

"Failed Back Syndrome" is a recognized outcome of many invasive spinal surgeries. The cause of this outcome may be variable, but some "failed back" cases are caused by AA. The percentage of patients given this label, however, is unknown. It is highly possible that the "failed" and "painful" aspect is due to neuroinflammation that may be active or has already destroyed nerve tissue.

MAJOR COMPLICATIONS

The complications of AA stem from the basic neuroinflammatory process and mass inside the spinal canal that entraps and destroys nerve roots, obstructs spinal fluid flow, and erodes the arachnoid/dural spinal covering allowing seepage of fluid into non-neural tissues that surround the spinal column. Here are the major pathologic changes and complications of AA.

1. Nerve root entrapment and destruction: pain which causes endocrine disturbances, leg, ankle, foot weakness and paralysis, and bladder, bowel, and sexual dysfunction
2. Spinal fluid flow impairments: headache, dizziness/vertigo, imbalance, blurred vision, and mental impairment
3. Spinal fluid seepage: pain, shrinkage of paraspinal tissue, decreased arm extension and posture deformities, and autoimmune manifestations

AA is historically known to cause an early, horrible death. Without effective treatment this may happen. Early death in AA occurs due one or more of these AA complications: autoimmunity, adrenal failure, cardiac arrest, and sepsis.

DIAGNOSTIC SCREENING

AA of the lumbar-sacral spine usually causes a rather standard set of symptoms. This is because the anatomic location of the inflammatory process and mass that entraps cauda equina nerve roots is usually at, or very near, the junction of the lumbar five (L5) and sacral (S1) vertebrae. This joint is mobile and subject to structural displacement due to various pressures put on it, especially sitting. Nerve roots that pass through this area have typical innervation to the skin and tissue of the lower extremities and bladder. Since those nerve roots are compressed with sitting, persons with AA usually find relief by standing or reclining. The ability to stand for long periods may also be limited.

Common symptoms include the sensation of water dripping or insects crawling on the legs or trunk, burning feet or ankles, and bladder dysfunction in either starting or stopping urination. Other symptoms such as headaches, blurred vision, dizziness, weakness, or vertigo may also be present. Due to this typical symptom profile, a short, written screen has been developed and shown here. (Table Two) Conferees firmly believe that all medical practitioners should know the typical symptoms of AA and evaluate persons for AA when they voice these symptoms.

EMERGENCY TREATMENT

Conferees universally reported that when they initially developed AA, no medical practitioner offered or knew how to provide emergencr treatment to possibly prevent AA. In some cases, their symptoms of AA appeared within hours following a spinal tap, epidural injection, or surgical procedure. In 2009, Dr. Antonio Aldrete reported that high dose intravenous methylprednisolone could prevent AA if administered within 60 days of the inciting event. Unfortunately, the initial symptoms of AA and MRI abnormalities after a spinal tap, epidural injection, or surgery may not occur for up to six months later. Even more unfortunate is an apparent lack of medical knowledge among medical practitioners as to what emergency treatment should be done to possibly prevent the development of a full-blown case of AA.

The conferees believe that all medical practitioners should know the fundamentals for emergency AA prevention and treatment if such symptoms as pain, vertigo, blurred vision, paraparesis, and bladder dysfunction occur after a spinal tap, epidural injection, spinal anesthesia, or spine surgery. The following prevention protocol has been developed and is now being used with some success. It can be implemented in any ambulatory medical setting.

a. Methylprednisolone (Medrol®)
b. Ketorolac
c. Medroxyprogesterone or minocycline

The emergency protocol listed above is a short-term (7 to 10 days) procedure that can be initiated if AA symptoms are present. Intravenous methylprednisolone is also a viable emergency treatment, and a combination of two may provide superior results.

TREATMENT STATUS

No specific drug or medical device has been developed that carries a United States Food and Drug Administration (FDA) label or indication for AA treatment. To deal with the urgency of the increasing prevalence of AA, clinical protocols based on treating the underlying/neuroinflammatory process inside the spinal canal began to be developed about 5 years ago. All conference leaders reported that pain relief, improved function, slowing the disease process, and an enhancement of quality of life is being recognized and achieved with this protocol effort. To some extent, the new protocols have been patterned after the treatment of rheumatoid arthritis, which is a progressive, destructive inflammatory disease of joints. Although improvement is being noted with the new protocol effort, symptom relief and well-being can be improved with research and innovation.

In general, current treatment of AA consists of 2 basic strategies: (1) a 3-component medication program and (2) specific physiologic measures.

A. Medication Protocol for Treatment

Each of the 3 components listed here has a therapeutic goal. Medication examples listed here are ones that conferees stated were popular primarily based on their personal experience and reported to be effective by participants in their social media support groups.

Component One – Suppression of Neuroinflammation

Goals

a. Prevention of nerve root entrapment and destruction
b. Prevention of spinal fluid seepage
c. Prevention of disease progression
d. Reduction of inflammatory mass
e. Reduction of pain

Some Popular Agents

a. Ketorolac
b. Methylprednisolone
c. Dexamethasone
d. Low dose naltrexone
e. Curcumin
f. Diclofenac
g. Indomethacin

Component Two – Regrowth of Damaged Nerve Tissue (Neuroregeneration)

Goals

a. Reduction of glial cell (neuroinflammation) activity
b. Regeneration of damaged nerve tissue

Some Popular Agents

a. Pregnenolone
b. Medroxyprogesterone
c. Human chorionic gonadotropin
d. Nandrolone

Component Three – Pain Control

Goals

a. Provide comfort and quality of life
b. Allow physiologic measures such as walking, stretching and dietary intake to be done

Some Popular Agents

a. Low dose naltrexone for persons not taking daily opioid drugs
b. Neuropathic (nerve damage) agents: gabapentin, topiramate, clonazepam

c. Symptomatic analgesic agents: ketamine, kratom, oxytocin, opioids, CBD products

B. Physiologic Measures for Treatment of AA

Here is the summary of physiologic measures currently being recommended. None have published scientific data, however, that document effectiveness.

- Enhancement of spinal fluid flow: walking, stretching and arm swings, rocking
- Increased oxygen: deep breathing, hyperbaric treatment, oxygen supplements
- Reduction of retained electricity: water soaking, magnet rubs, copper jewelry
- Extremity stretching and flexing: Leg, feet, arm extension, foot and hand flexing, straight leg raising

Given the fact that AA can suddenly and unexpectedly progress to cause leg, bladder, and bowel impairments, we recommend that some of the above-listed measures be done daily to possibly prevent a serious complication like leg paralysis or incontinence.

It is acknowledged that treatment efforts to date are "first generation" that should be improved over time with research and experience. It is further acknowledged that we know of no controlled or randomized studies to validate or provide "evidence-based" therapy to the popular therapeutic agents and measures recommended here. All clinical experience and recommendations in this report are based on "trial and error", and reports from persons who have AA.

MAJOR NEEDS GOING FORWARD

Conferees voiced some specific needs to progress forward.

1. There is a great need for medical practitioners to diagnose the disease, provide emergency, preventive care, and render long-term treatment of AA, including palliative care for those who are in the late stages of the disease.
2. Educational seminars for persons with the disease and their families is urgently needed
3. Educational materials on diagnosis and treatment need to be developed that persons with the disease, and their family members can present to their medical practitioners.

SUMMARY

AA is an urgent public health issue that calls for prevention, diagnosis, and treatment measures. The disease is clearly increasing in prevalence due to various factors including the ability to diagnose it. It can no longer be considered a rare disease. Few medical practitioners know how to diagnose and treat AA at this time. First-generation emergency and long-term treatment protocols have been developed. Although current protocols can be improved, they are effective enough for prevention of complications and serious progression. Meaningful treatment is being reported by persons with AA. Going forward there is an urgent need for

education and training of persons with the disease, their families, and medical practitioners. A major message of the Leadership Conference is to reject the old notion that "nothing can be done so live with it, and die young in misery". Progress in recent years has clearly shown that persons with AA can obtain some relief and recovery with the new knowledge of the disease and its treatment. In contrast to other common back pain problems, AA can become a catastrophic, multi-systemic disease with a miserable, shortened lifespan. AA is clearly an urgent public health issue.

TABLE ONE

MODERN TIMES PROMOTERS OF ADHESIVE ARACHNOIDITIS

- ✓ Sedentary (sitting) lifestyle*
- ✓ Poor posture
- ✓ Non-supportive footwear
- ✓ Bucket seats
- ✓ Risk-taking sports activities
- ✓ Obesity
- ✓ Lack of exercise and walking
- ✓ Epidural injections, spinal surgery, and anesthesia
- ✓ Longer lifespan and aging
- ✓ Emergency life saving measures following accidents**
- ✓ Increased infant and childhood survival rates due to antibiotics and vaccines***

*Refers to the long hours of sitting in front of television and computers
**Life may be saved but injury left behind
***Immune deficient infants may survive and develop tissue degeneration disorders in adulthood

TABLE TWO

A SCREENING TEST FOR LUMBAR-SACRAL ADHESIVE ARACHNOIDITIS

Adhesive Arachnoiditis (AA) in the lumbar and/or sacral region of the spine is a condition that is the result of nerve roots adhering or gluing to the arachnoid layer of the spinal canal covering due to inflammation and adhesions. When this inflammatory process happens, a typical set of symptoms occur which will both identify and separate the person with AA from those with back pain due to some other cause.

If a person with lower back pain answers yes to at least four of the seven questions in the screen below, they should be immediately evaluated by a physician to confirm a potential diagnosis of AA. Previously, AA has been considered a hopeless, untreatable disease that is progressive, debilitating and life-shortening. This perception of AA is both outdated and no longer accurate. Today, AA can be specifically diagnosed and treated. The earlier in the AA disease process that both confirmation of an AA diagnosis is determined, and treatment is initiated, the better the potential for relief and hope for eventual recovery.

	QUESTIONS	Yes	No
1	In addition to chronic pain, do you ever experience sharp, stabbing pains in your lower back when you twist, turn or bend?		
2	Do you ever experience bizarre skin sensation**s** such as crawling insects or water dripping down one or both legs?		
3	Do you ever have burning, tingling, or a sensation of walking on broken glass in your feet and/or toes?		
4	Does your pain become worse while standing, sitting and/or walking?		
5	Do you have leg weakness and/or pain that radiates down one or both legs?		
6	Do you experience any bladder dysfunction such as dribbling, or difficulty when starting or stopping urination?		
7	Do you sometimes have a headache along with blurred vision?		

If you answered yes to four or more of these seven questions, you most likely have AA or some other neuroinflammatory disease of the nerve roots in your lumbar or sacral spine. Your physicians need to be informed of the results of this screening test as you need to obtain both a confirmatory diagnosis and a treatment plan that is specific for your condition.

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