

ADHESIVE ARACHNOIDITIS CLINICAL UPDATE 2020

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ABSTRACT

Adhesive arachnoiditis (AA) is an inflammatory disease of the lower spinal canal that involves cauda equina nerve roots and the arachnoid-dural covering (meninges) of the spinal canal. While once a rare disease, it is now emerging in every community. Diagnostic and treatment protocols have been developed and are presented here. A clinical profile of 80 MRI-confirmed cases show that AA is primarily a disease of mid to late-age females. The most common underlying cause is a structural disorder of the spine. Genetic connective tissue/collagen disorders of the Ehlers-Danlos Syndrome type appear to be the second most common cause of AA. Multiple surgeries and spinal interventions were common in these cases. A symptom profile of AA is quite uniform and includes pain relief on change of position, jerks or tremors in the legs, urinary dysfunction, and sensations of insects crawling or water dripping on the skin. Treatment specifically directed at suppressing neuroinflammation, promoting tissue regeneration, and providing aggressive pain management is the most successful approach for halting the progression of the disease, restoring function, and enhancing patient quality of life.

INTRODUCTION

Adhesive arachnoiditis (AA) is an inflammatory disease that occurs inside the lumbar and sacral regions of the spinal canal.¹⁻⁶ The disease inflames nerve roots of the cauda equina and the arachnoid-dural covering (meninges) of the spinal canal. This inflammation produces adhesions that merge or “glue” these two separate anatomic structures together into an inflammatory-adhesive mass inside the spinal canal. Multiple mass areas can form, and one or more of these areas can enlarge, entrap nerve roots, and block normal spinal fluid flow. Unless this inflammation is controlled and suppressed, it causes dysfunction and death to the entrapped nerve roots which results in, intractable pain, multiple neurologic impairments, and autoimmune manifestations. Progressive enlargement of the mass or masses may occur in a manner analogous to the growth of a cancerous tumor. AA requires aggressive anti-inflammatory treatment and pain control.

Arachnoiditis simply means inflammation of the arachnoid layer of the meninges. It may occur in other areas of the central nervous system (CNS) but as used in this report only refers to arachnoid inflammation in the lumbar-sacral area.

In the past, AA has been considered a rare disease. This is no longer the case. An analysis of multiple, publicly available, scientific epidemiologic surveys of people with back pain suggests that 1.75 to 7.0 million adult Americans have AA.⁷⁻¹³ Regardless of the precise number, it is now clear that AA is found in every community and medical practice in the modern world.

Consequently, it has become incumbent on every primary care and pain practitioner to be able to identify persons with AA and participate actively in the care of these individuals.

Since AA has become one, if not the most, common reason people develop severe intractable pain, the authors have established a research and education project to study AA and develop first generation diagnostic and treatment guides for medical practitioners. Our latest observations and recommendations are presented in this report. To date, we have been able to analyze medical information from over 300 cases confirmed by magnetic resonance imaging

(MRI), and observe treatment results from over 200 cases. This report summarizes our observations and findings to date.

CAUSES OF TODAY'S AA CASES

In the 19th century AA was known as a dread disease causing unbearable suffering and early death. The initiating causes in the 19th century were usually tuberculosis or syphilis, which had invaded the arachnoid-dura meninges covering. In the 20th century the most common cause was the use of insoluble irritating oil-based dyes called myelograms that were directly injected into the spinal fluid to enhance x-ray visualization of the spine. Today the major cause is spine disorders that alter the natural anatomic structure and posture of the spinal column. The most common spinal condition which causes AA is the protrusion of lumbar and sacral intervertebral discs into the spinal canal causing narrowing (stenosis) of the canal along with compression and displacement of the free-floating nerve roots of the cauda equina. Stenosis of the spinal canal plus nerve root compression eventually cause the irritation, friction, and inflammation, that leads to adhesion formation. Other spine disorders very common among adults in modern society include scoliosis, spondylolisthesis, osteoporosis, and arthritis. All these disorders have the potential to cause the cauda equina nerve roots to undergo the compression and inflammatory process that leads to AA. These conditions appear to be related to sedentary lifestyle, inactivity, obesity, and longer lifespan.¹⁴⁻¹⁵ Next to spinal conditions our experience has been that genetic connective tissue/collagen disorders of the Ehlers-Danlos Syndrome (EDS) type are the most common conditions which can lead to AA. Trauma, autoimmune disorders, Lyme disease, and possibly viral, fungal and bacterial infections make up an estimated 10-15% of cases.^{16,17} Although surgery and epidural corticoid injections are often cited as the cause of AA, they are actually co-factors, as the person who receives surgery and/or epidural injections has some underlying spinal disorder that medically indicates the need for surgery or an epidural corticoid injection. Clinical data collected by us suggests that spine surgeries and epidural injections done on some individual patients may be excessive and even causative of AA.^{11,18}

CLINICAL PROFILE

No clinical data on AA has been published in recent years.^{18,19} As a component of our research and education project, we compiled a basic clinical profile from 80 cases who had MRI-confirmed AA. (Table One) These patients sent us their magnetic resonance imaging (MRI's) with a clinical history to help develop a profile which is described and summarized in Table One. Females outnumbered males, and the age of these patients was generally in the older ranges. Sixty-One (76.3%) of the cases had one or more spinal conditions as a predisposing cause. Over half (53.8%) had one or more spinal surgeries and 69 (86.3%) had one or more epidural injections.

The majority (over 55%) reported the symptoms of burning feet, tremors or jerks, the sensation of crawling insects and/or water dripping, difficulty starting urination or defecation and blurred vision. This symptom profile is important in identifying AA in patients who report back pain. Sixty-six (66; 82.5%) among the 80 cases in the clinical profile reported here indicated their pain was always constant.

COMPLICATIONS OF ADHESIVE ARACHNOIDITIS

AA has some serious complications that are briefly described here: (1) neurologic impairments; (2) spinal fluid flow obstruction; (3) spinal fluid “seepage”; (4) autoimmune sequelae, (5) anatomic derangement; and (6) intractable pain syndrome. (Table Two)

1. Neurologic Impairments

The most common site for nerve root inflammation and destruction is around the lumbar-sacral junction (e.g. L5-S1). Nerve roots that transverse this area connect directly to the lower extremities, bladder, bowel, and sex organs. The lower extremities may suffer a variety of neurologic leg, ankle, or foot impairments including weakness and immobility (“foot-drop”). In severe cases outright paraparesis or paralysis may occur requiring the person with the disease to use a walker, cane, or wheelchair. Bladder impairment is almost uniform. Impairments include hesitancy, urgency, overflow incontinence, leaking, and even the necessity of self-catherization. Paresthesias of the lower trunk and extremities are common and include the feelings of insects crawling or water dripping on the skin. Table One lists some of the most common symptoms derived from 80 persons with confirmed AA.

2. Spinal Fluid Flow Obstruction

Unfortunately, the inflammatory-adhesion process causes the nerve roots to clump and a tumor or mass to form when the clump attaches to the arachnoid-dura covering. This inflammatory -adhesive mass acts like a cancerous tumor. It may grow, expand, and capture more and more nerve roots. The net result is more pain and neurologic dysfunction. Inflammatory-adhesive masses most often occur near the lumbar-sacral spine junction (L5-S1) which is the joint that must endure the most pressure when we sit. Therefore, persons with AA can’t usually sit very long without causing themselves great pain as they are compressing the inflammatory-adhesive mass that is inside the spinal canal.

There may be multiple small masses inside the spinal canal caused by the inflammatory-adhesive process of AA. They act like boulders or a dam in a stream or river. Spinal fluid constantly flows inside the spinal canal to bring nutrients, lubricate the spinal cord and nerve roots, and carry away any toxins, including inflammation waste products and dead cellular material.^{20,21} These intraspinal masses may interfere with spinal fluid flow, causing such symptoms as headache, blurred vision, dysphoria (opposite of euphoria) and ringing in the ears or tinnitus.

3. Spinal Fluid “Seepage”

The term arachnoiditis simply means inflammation of the arachnoid layer of the meninges or spinal canal covering.

When inflamed, nerve roots become attached to the arachnoid-dural covering of the lower spinal canal, inflammation involves not only the inner arachnoid layer but also the outer dural layer. Just like leakage from a “rusty pipe”, spinal fluid may chronically seep

out of the dural layer into the soft tissues that surround the spinal column. When this occurs, lower back pain develops. Paraspinal tissues may become inflamed and contract, since spinal fluid is a toxic irritant to tissues outside the spinal canal. Considerable anatomic and tissue alterations may occur (See Figures 1-3).

4. Autoimmune Sequelae

Moderate through severe stages of AA may cause autoimmune sequelae.^{22,23,24} There may include arthralgia, myalgia, carpal tunnel syndrome, and thyroiditis among autoimmune manifestations that have been observed with other diseases. The aegis of autoimmunity in AA is likely related to inflammatory waste and/or spinal fluid that reaches either the general vascular circulation or tissues outside the spinal canal. Inflammatory waste can reach the general circulation by being transported from the lower spinal canal up into the brain and neck where the glymphatic and lymphatic systems empty waste into the general circulation. AA may cause erosion of the spinal canal covering (arachnoid and dural layers) and allow chronic seepage of spinal fluid into the paraspinal soft tissue producing an inflammatory response. This can ignite an autoimmune reaction.

5. Anatomic Derangement

AA plus any underlying spine abnormality causes profound anatomic derangement of paraspinal muscles and soft tissue. The derangement is due to anatomic spine abnormalities plus chronic bending and leaning to minimize pain. The result is abnormal posture with excess stress and pressure on joints including facet and sacroiliac joints. If spinal fluid seepage has chronically occurred, paraspinal tissue may construct toward further anatomic derangement. (Figures 1-3) These complications may require soft tissue and intra-articular measures such as corticoid injections, topical lidocaine, homeopathy, electric current (TENS) or electromagnetic therapy. Physical stretching of the upper back, shoulder girdle, and hips is essential to counter the anatomic derangement found in AA patients.

6. Intractable Pain Syndrome (IPS)

IPS is defined here as a disorder that produces constant (24/7) pain, insomnia, and anorexia and has measurable, adverse impacts on the endocrine and cardiovascular systems. IPS raises blood pressure and pulse rate, alters glucose metabolism, and over stresses the pituitary-adrenal-gonadal axis to produce multiple hormonal deficiencies. IPS results from the activation of glial cells and the formation of neuroinflammation within the CNS (i.e. pain centralization).

DIAGNOSIS

A preliminary diagnosis of AA is made in a patient who has an inciting event or disorder such as herniated discs, trauma, or EDS, and who has the typical symptom profile. We recommend that all patients presenting with typical AA symptoms be screened for EDS. (Table One.) Although non-specific, persons with AA always have some physical, neurologic abnormalities of the lower extremities. Examination of the back will almost always show asymmetry, leaning, and possible contractures and skin indentation. (See Figures 1-3) There is no specific blood test.

Inflammatory markers of C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and various cytokines may elevate.²⁵ Hormonal deficiencies of cortisol, pregnenolone, dehydroepiandrosterone (DHEA) and testosterone may be present.²⁶

A contrast MRI of the lumbar-sacral spinal canal is required for a confirmatory diagnosis providing the history, symptoms, and physical examination are also compatible. (Table One.) Contrast techniques (injected dye or high-resolution MRI) differentiate cauda equina nerve roots and spinal fluid so the typical appearance of AA can be visualized.^{18,27,28}

CATEGORIZATION OF SEVERITY

Like all diseases AA has degrees of severity. We recommend a 4-level categorization that is based on clinical assessment as MRI findings are not specific enough to determine clinical severity. Our recommended classification is mild, moderate, severe, and catastrophic (Table Three). Those persons in the severe and catastrophic levels appear to have sustained permanent and irreversible neurologic damage. Consequently, they will likely not respond to anti-neuroinflammatory or neuroregenerative therapies. These individuals will usually require palliative pain care.

Those persons with AA who are in the mild or moderate categories have appeared to have good response to anti-neuroinflammatory and neuroregenerative treatment. This is the reasonable assumption as these individuals do not have total irreversible neurologic damage, as do those persons in the severe and catastrophic categories. As with other diseases, the earlier the case is diagnosed and treated, the better the outcome. (Table Three)

TREATMENT

Treatment for AA consists of both physical and pharmacologic measures. Physical measures that are highly recommended are daily walks and stretching to full extension of upper and lower extremities and paraspinal muscles. The primary purpose of these measures is to prevent paralysis and contractions of paraspinal musculature. Other physical measures we recommend include water soaking, deep breathing, rocking in a rocking chair or mini-trampoline walking. Although no scientific evidence is available to support these measures, they will hopefully enhance spinal fluid flow leading to improved CNS nutrition and cleansing of inflammatory waste.

While several agents have some scientific research that suggest they suppress neuroinflammation, we currently recommend the following 3 agents as the starting treatment for AA. All are potent neuroinflammation suppressors. They also provide direct analgesic action. They are: (1) naltrexone; (2) ketorolac; and (3) corticosteroid (methylprednisolone or dexamethasone).

Pharmacologic treatment is primarily directed at control of the neuroinflammation actions of AA. In patients who are not on opioids, naltrexone is our primary drug preference as it has analgesic, as well as anti-inflammatory and autoimmune suppression properties. The starting

dosage is 0.5 to 1.0 mg given twice a day. This dosage can be raised over 4 to 6 weeks to as high as 7.0 mg given twice a day.

At this point in time, we regret we must highly recommend ketorolac and one of these corticosteroids, methylprednisolone or dexamethasone, as adjuncts to naltrexone or daily opioids if they are patient's primary pain treatment. Although a number of agents including diclofenac, metformin, indomethacin, acetazolamide, and prednisone have some anti-neuroinflammatory properties, we have not found them to be consistently effective simply because they are not as potent as either ketorolac or methylprednisolone/dexamethasone.²⁹ Also for agents to be effective in AA treatment they must cross the blood brain barrier, enter spinal fluid and act on receptors in lower spinal canal tissue. We recognize the complications that ketorolac and the 2 potent corticosteroids can cause, so we recommend these agents be used intermittently at a low dose. Examples of recommended use are: (a) 1 to 2 times a month; (b) 1 to 3 times a week avoiding two days in a row. Ketorolac must be used with great caution if used in persons over age 70, orally, or in persons with renal compromise. The preferred route of administration is IM (intramuscular) injection if possible. Regular monitoring for gastrointestinal bleeding or renal toxicity is recommended. Potential complications of AA including a shortened lifespan, outweigh the risk of low dose, intermittent use of ketorolac, methylprednisolone, or dexamethasone.

In addition to the above-noted physical and pharmacologic therapies we recommend a high protein, low carbohydrate, high content fruit/vegetable diet. Useful dietary supplements in our experience have been curcumin, pregnenolone, and B₁₂. Pain control may have to be very aggressive and potent especially in persons with AA who fall into the severe and catastrophic categories. These individuals have usually had little anti-neuroinflammatory treatment and suffer severe intractable pain with many of the complications noted above. High-dose, opioid therapy, implanted electrical stimulators, and intrathecal opioid administration may be necessary. (Table Four) It is also important to point out that AA patients in the severe and catastrophic categories may be too impaired to participate in physical measures or take pharmacologic agents recommended here.

NEW THERAPEUTIC APPROACHES

Since AA is being recognized in every community, new therapeutic measures are being attempted. Our clinical experience as well as reports received by us indicate potential with two new approaches. The first is an anabolic or neuroregenerative effort with the neurosteroids, human chorionic gonadotropin (HCG) and nandrolone. Second, intravenous infusions of ketamine, lidocaine, vitamin C, and nicotinamide adenine dinucleotide (NAD) have all been reported to us to give some patients relief for up to 3 months after a single infusion.

Various stem cell measures are now being attempted. At this time, however, we have no specific recommendation on their use. Our major caution with the new approaches is that they are sometimes promoted as a substitute for daily physical measures, anti-neuroinflammatory treatment, and effective pain control. Also, some persons with long-standing AA who never had the benefit of specific treatment may not benefit from treatment other than palliative care.

TREATMENT OF PARASPINAL TISSUES

The soft tissues that surround the spinal column including muscle, fat, connective tissue, and large nerves that exit the spinal column may become inflamed and painful in AA. Reasons are: (1) anatomic and postural abnormalities caused by an underlying spinal disorder; (2) neurologic impairments; and (3) chronic seepage of spinal fluid. Any number of therapies that reduce inflammation and pain in soft tissues may provide some relief and recovery. Daily extension stretching of lower back muscles, arms and legs may be essential to prevent contractures. Topical agents including lidocaine and diclofenac may be helpful.

Local corticosteroid (non-epidural) injections may be necessary for localized, painful areas. A favored treatment of ours is pulsed electromagnetic energy administration (radio wave, laser, infra-red) to not only help heal inflamed soft tissues but hopefully, the electromagnetic energy waves will penetrate deep enough to help heal the spinal canal covering (arachnoid-dura) and stop spinal fluid seepage. Individual AA patients have reported that massage, TENS, homeopathy and other soft tissue therapies are helpful.

POST INJECTION EMERGENCY TREATMENT

An unfortunate, small number of persons develop AA following a spinal tap or epidural injection. It is, therefore, critical that simple, emergency treatment be done if symptoms of AA develop within 60 days after the injection. These symptoms are increased back pain, dizziness or vertigo, leg weakness, burning skin, and urinary hesitancy, frequency or incontinence. It is essential to administer emergency treatment based strictly on symptoms, because signs of AA may not show on an MRI for up to 6 months after the injection.

The emergency treatment we recommend is simple and has not proven to cause any lasting complications:

1. 6-day methylprednisolone dose pak (Medrol®)
2. Ketorolac, 30 to 60 mg for 3 consecutive days
3. Medroxyprogesterone 10 mg twice a day for 5 days

In our hands, the emergency treatment recommended here has always stopped further progression into full blown AA, but some of the post-injection symptoms have remained. Intravenous use of methylprednisolone or dexamethasone can be a useful adjunct to this protocol. Following emergency treatment, naltrexone and the other measures described will need to be started and continued for at least a few weeks.

SUMMARY

AA is an inflammatory disease of the lower spinal canal that involves cauda equina nerve roots and the arachnoid-dural covering of the spinal canal. It can no longer be considered a rare disease as it is emerging in every community. Today's major causes of AA are degenerative spine disorders that anatomically compress cauda equina nerve roots and initiate a process of friction, inflammation, and adhesion formation. Genetic connective tissue/collagen disorders of the Ehlers-Danlos class appear to be the second most common cause.

AA should be suspected in a back-pain patient who complains of leg weakness, urinary symptoms, blurred vision, sensations of water or insects on their skin, and relief of pain on change of position such as standing or reclining from a sitting position. Elevated inflammatory markers may be present in serum. MRI's are used to confirm the diagnosis.

AA is a progressive, inflammatory disease of the spinal canal with the potential for severe neurologic impairments leading to an inability to care for oneself, immense pain, and an early death. Treatment must be aggressive and primarily directed at controlling intraspinal inflammation. The first drug preference is naltrexone in patients who do not take daily opioids, as naltrexone has analgesic, anti-inflammatory, and autoimmune suppression properties. We recommend that naltrexone be accompanied by low intermittent dosages of ketorolac and one of these two corticosteroids: methylprednisolone or dexamethasone. This is an aggressive approach to the intraspinal, inflammation-adhesion process that is the basis of AA. AA patients may have intense, intractable pain that requires the most aggressive and potent pain control measures which may include opiate medication for those who have already failed other treatments. In addition to control of intraspinal inflammation and pain control, a number of approaches are being attempted to regenerate tissue and provide some permanent relief and recovery.

TABLE ONE

CLINICAL PROFILE OF 80 MRI-DOCUMENTED CASES OF ADHESIVE ARACHNOIDITIS

	<u>NO.</u>
1. Females	65 – 81%
2. Males	15 – 19%
3. Age Range in Years	18 to 80
4. Mean Age ± S.D. in Years	48.9 ± 13.7
5. No. with a Predisposing Spinal Condition*	61 – 76.3%
a. Herniated discs	44 – 55%
b. Spondylolisthesis	17 – 21.25%
c. Osteoporosis	6 – 7.5%
d. Spine arthritis	23 – 28.75%
e. Scoliosis	9 – 11.25%
f. Tarlov cysts	9 – 11.25%
6. No. with One or More Spinal Surgeries	43 – 53.8%
7. Total No. of Spine Surgeries in 43 Cases	91
8. Range of Surgeries in 43 Cases	1 to 8
9. No. Who Had 2 or More Spine Surgeries	22 – 27.5%
10. No. Who Had One or More Epidural Injections	69 – 86.3%
11. Total No. Epidural Injections in 69 Cases	236
12. Range of Epidural Injections in 69 Cases	1 to 20
13. No. Reported Over 8 Epidural Injections	16 – 20.0%
14. Symptoms and Complications Reported by Over 55% of Cases	
a. Pain Relief on standing	70 – 87.5%
b. Standing Too Long Causes Need to Lie Down	69 – 86.3%
c. Hurts to Lie Flat on Back	67 – 83.8%
d. Pain Always Present	66 – 82.5%
e. Shooting Pains, Tremors, or Jerking in Legs	64 – 78.8%
f. Burning Pains in Feet	63 – 78.8%
g. Cold Hands or Feet	58 – 72.5%
h. Crawling of Insects on Skin	58 – 72.5%
i. Water Dripping/Running Down Legs	53 – 66.3%
j. Difficulties Starting Urination/Defecation	51 – 63.8%
k. Leg Raise Hurts Back	50 – 62.5%
l. Blurred Vision	47 – 58.8%
m. Pain Behind Eyes	45 – 56.3%

+Some cases reported more than one condition.

TABLE TWO

COMPLICATIONS OF ADHESIVE ARACHNOIDITIS

- NEUROLOGIC IMPAIRMENTS (e.g. paralysis, urinary, bowel, and sexual dysfunction)
- SPINAL FLUID FLOW OBSTRUCTION (e.g. headache, blurred vision, tinnitus)
- SPINAL FLUID SEEPAGE (e.g. back pain, paraspinal tissue contractures, tissue atrophy)
- AUTOIMMUNITY (myalgias, arthralgias, mast cell activation)
- ANATOMIC DERANGEMENTS (leaning, splinting, aberrant posture, arthritic)
- INTRACTABLE PAIN SYNDROME (hormone deficiencies, hypertension, insomnia, fatigue)
- SHORTENED LIFE SPAN (cardiac arrest, adrenal failure, cachexia sepsis)

TABLE THREE

CATEGORIES OF SEVERITY

Mild:

- Full range of motion
- No back indentation or contracture
- Normal inflammatory markers
- No bladder impairment
- No MRI evidence of spinal fluid leakage or obstruction
- No hormone abnormalities
- Can sit and stand in one position for 10 minutes

Moderate:

- Full range of motion and walks without assistance
- Mild to zero lower extremity weakness
- Normal inflammatory markers
- Some bladder hesitancy, urgency, dripping
- No MRI or physical evidence of spinal fluid leakage
- Mild constant pain but no need for sleep medication
- Can sit and stand in one position for 10 minutes

Severe:

- Some range of motion impairment and needs assistance (cane or other) to ambulate
- Weakness in lower extremities with neurologic symptoms (e.g. burning feet, bugs crawling, jerking or other)
- Elevated inflammatory markers and/or hormone abnormalities
- Bladder impairment symptoms of hesitancy, urgency, or incontinence
- MRI and/or physical evidence of chronic spinal fluid leakage and/or flow obstruction
- Constant pain that impairs sleep
- Can't sit and stand in one position for 10 minutes

Catastrophic:

- Requires assistance with activities of daily living (dressing, toiletry, eating, etc.)
- Significant lower extremity impairment (needs walker, wheelchair, braces)
- Bladder impairment of hesitancy, urgency, or incontinence
- Mental deficiencies such as memory loss or reading ability
- MRI and physical evidence of chronic spinal fluid obstruction and leakage
- Elevated inflammatory markers and hormone abnormalities
- Constant pain that impairs sleep
- Can't sit or stand in one position for 10 minutes

TABLE FOUR

STARTING TREATMENT REGIMEN+

- A. Low dose naltrexone – .05 to 7.0 mg twice a day*
- B. Ketorolac – injection or troche – 15 to 60 mg on 1 to 3 days a week or bi-monthly
- C. Corticosteroid – methylprednisolone 2.0 to 4.0 mg or dexamethasone .5 to .75 mg 1 to 3 days a week or bi-monthly
- D. Dietary measures/supplements
 - a. Curcumin 900-1800 mg a day
 - b. Pregnenolone 200-250 mg a day
 - c. Diet: high protein/low carbohydrate/high vegetable-fruit
- E. Physical measures to be done daily
 - a. Walk with arm swings
 - b. Full length stretching of arms, legs and feet
 - c. Water soaking: tub, shower, jacuzzi, pool
 - d. Side-to-side leaning and stretching
- F. Pain control
 - a. Standard treatment with analgesic, neuropathic, and adrenergic/stimulant agents

*Cannot use naltrexone in patients who take opioids. Substitute diclofenac, indomethacin, acetazolamide, or metformin.

FIGURE ONE

Spinal fluid leakage and rods penetrating in AA patient.

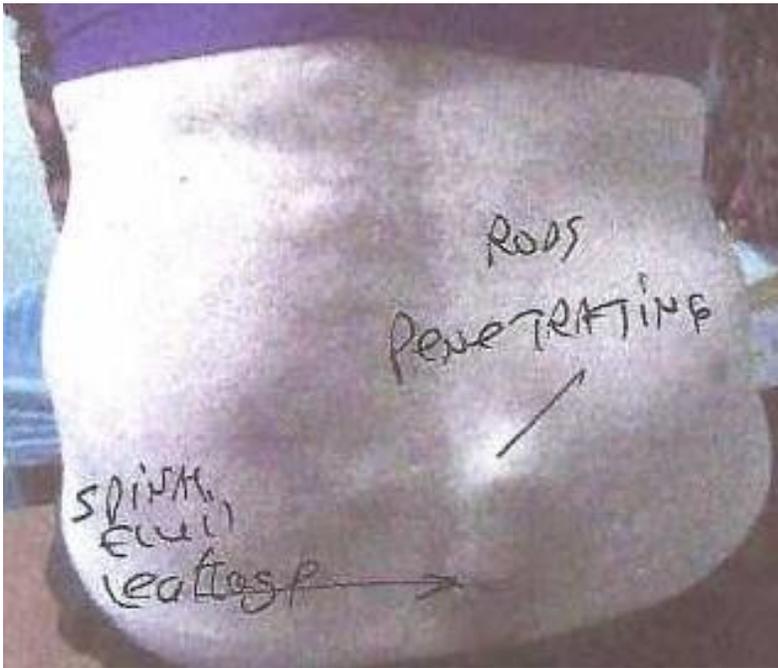


FIGURE TWO

Multiple spine surgeries in AA patient. Note the areas of tissue atrophy, and crease of left side.



FIGURE THREE

Mid-line indentation and skin contractures in an AA patient.



FIGURE FOUR

Mid-line indentation in an AA patient



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