REPORT

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SPINAL CANAL INFLAMMATORY DISEASE

AN UNDERLYING CAUSE OF PAINFUL SPINE DISORDERS

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Working Definition: Spinal canal inflammatory disease is a process that spreads inflammation to multiple tissues in and around the spine and which may resuls in such painful conditions as degenerated discs, Tarlov cysts, tethered cord, and adhesive arachnoiditis.

Conclusion: There is a subgroup of back pain patients who develop multiple inflammatory spine disorders over a considerable time period. We conclude that spinal canal inflammation is an underlying disease, and that the disease has an autoimmune component.

INTRODUCTION

Approximately 8 years ago I founded "Arachnoiditis Hope" with the mission to bring diagnosis and treatment of this horrible disease to every community. Arachnoiditis was medically defined in 1873 as "inflammation of the arachnoid membrane" which is the inner lining of the spinal canal cover. AA was rightfully called the devil's disease" in the 1800's as it was characterized by excruciating back pain, leg paralysis, incontinence, malnutrition, profound weakness, a bed-bound state, and premature death unless suicide was chosen first. AA occurs when two spinal tissues, the cauda equina and arachnoid, become inflamed, form adhesions, and fuse into a clump or mass of nerve roots attached to the arachnoid. In our early efforts we established a system whereby patients with AA could send us their magnetic resonance imaging (MRI) results, laboratory tests, and clinical profile. We reviewed the material and gave the patient and their medical practitioner our ideas in building a treatment program. To date, I and my associates have reviewed over 3,000 cases from over 60 countries.

When we started, we had only a vague notion as to how the inflammation in the cauda equina and arachnoid began. Most cases seemed to follow a medical procedure such as an epidural injection or surgery, but others had followed an injury, infection, or simply happened with no apparent cause. As we began to study AA cases it became abundantly clear that these cases didn't just have inflammation in their cauda equina and arachnoid but in other spinal canal tissues. The first observation was that essentially all AA cases had inflamed, degenerated discs. As time went on, we began to realize that AA was the end stage or culmination of a chronic inflammatory process in the various tissues that make up the spinal canal. The new contrast MRIs allowed visualization of tissue destruction caused by inflammation as well as the development of cysts and fibrosus.

Essentially, all AA cases we have reviewed have suffered back pain for some time and have been given several diagnoses. (Table One) Failed back surgery syndrome is a common label. They had been unsuccessfully treated with the standard back treatments. It became clear that inflammation had initiated in the spinal canal and slowly but progressively over time spread to multiple tissues and produced severe pain and a variety of neurologic impairments. In summary, SCID is a clinical entity that we have identified. It may produce a number of painful spinal conditions including adhesive arachnoiditis. It appears to be a source of profound pain and disability that has not previously been conceptualized, identified, or specifically treated. This report describes our investigation and findings to date.

A DISEASE APPEARS

When we began to intently study adhesive arachnoiditis (AA), we were under the impression that AA was a stand-alone, singular disease. It was caused by spine trauma, medical procedures, and

infections such as Lyme disease. The first shock was that AA patients had universally been ill for a long time. Oftentimes for years. They all had experienced a variety of spinal conditions such as herniated discs, tethered cord, and Tarlov cysts. The number of medical procedures they had undergone was astounding. (See Table) One fact that was clear. The person with AA also had other painful, debilitating spinal conditions. AA was one complication of a disease that had multiple complications. It turns out that most complications, such as degenerated discs, like AA, had been clinically identified as a singular entity and treated as such although they were complications of an underlying disease. All of the singular conditions had chronic inflammation as a basic cause. Given this inflammatory base, we have simply labeled the disease "spinal canal inflammatory disease."

In summary, AA cases all have other inflammatory conditions that may pre-date AA. Table Two lists those that are most common.

TABLE ONE
CLINICAL PROFILE OF
80 MRI-DOCUMENTED CASES OF ADHESIVE ARACHNOIDITIS

	NO.
1. Females	65 – 81%
2. Males	15 – 19%
3. Age Range in Years	18 to 80
4. Mean Age ± S.D.	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%

TABLE TWO
SPINE CONDITIONS COMMONLY FOUND IN AA PATIENTS

Degenerated Disc
Vertebral Arthritis
Epidural Fibrosus
Spinal Stenosis
Tethered Cord
Tarlov Cysts

NECESSITY FOR A DISEASE LABEL AFTER SPINAL PROCEDURES

Unfortunately, spinal puncture, epidural injections, and surgery may induce acute SCID which may later turn chronic and develop AA. A major reason we have had to assign the label of SCID to cases is the development of AA after spinal punctures and epidural injections. AA doesn't immediately develop, but desperate patients and clinicians need to know why severe back pain and neurologic symptoms develop after a spinal puncture. Patients and physician rightly fear AA. It is a rare occurrence but any medical procedure that intentionally or accidentally causes a non-healing hole, abrasion, or cut on the arachnoid layer of the spinal canal cover can cause acute SCID. The process is initiated because a contaminant from the skin, needles, or medication solution may irritate the arachnoid membrane producing inflammation. Also, spinal fluid is toxic to sub-surface cells of the arachnoid. Once inflammation starts, SCID is on its way.

Many of the cases are truly tragic. For example, a woman who is delivering a baby and receives spinal or epidural anesthesia is excited about the birth but ends up with a serious disease. A person on a routine emergency room visit for an infection or headache may receive a spinal tap. It is rare but epidural injection risks needle puncture and dural tears. Surgery causes significant trauma.

These cases are similar in that severe back pain occurs during or shortly after the medical procedure. Headache, vertigo, and radiating pain usually accompanies the back pain. The patient often has a perception of disaster, and they often panic.

The follow-up of these cases is, in our review of several dozen, very inadequate. Blood patches are the standard treatment, but they may worsen the situation. No treatment is given pending MRI review. Since the MRI doesn't show adhesive arachnoiditis, the patient is often told that there is "nothing wrong" with the implication that the problem is "in their head." Our review of these cases always points out MRI evidence of inflammation. We recommend a vigorous

therapeutic procedure to prevent AA and hopefully eliminate or prevent further development of AA.

IDENTIFICATION OF CHRONIC SCID

The longstanding, standard medical approach to treatment of back pain is fundamentally anatomic and mechanical. Common causes include sprain, strain, herniated discs, and pinched nerves. Treatments focus on physical exercise and surgery. Most back pain problems are resolved with the traditional anatomic-mechanical measures. A major tip-off to identify a person with chronic SCID is that they have failed to get better with standard back treatments.

To date, we have endeavored to clinically identify SCID, develop a clinical profile, and define the disease. Identification of a person with SCID is essential because the treatment isn't a one-time fix with surgery or a shot. It is a lifetime endeavor/undertaking targeted at suppressing inflammation and autoimmune assaults just as rheumatoid arthritis and lupus are treated.

To start, chronic SCID is severe back pain that doesn't respond to standard medical treatment and that demonstrates inflammation by laboratory tests and/or MRI analysis. SCID patients have a typical clinical profile. (See Table Three) Most recall when their back pain started. Most cases begin with an injury or accident that can range from seemingly minor to a debilitating blow. The first trauma is rather typical: lifting, pushing, a fall, or blow from an object. Some persons perceive their back pain started after a viral infection or Lyme disease, and rarely, a spinal infection. Another group are individuals who have a spinal puncture for diagnostic or therapeutic purposes. Many persons date their AA to an epidural injection or spine surgery. In these cases, SCID was present when the procedure was done and the procedure accelerated SCID to develop into AA.

Typically, persons with SCID seek chiropractic or other mechanical help from their local resources. As the disease progresses, they usually try a number of over-the-counter analgesics and anti-inflammatory agents. If these initial attempts don't bring relief, persons with SCID begin the rounds of back and spine specialists, orthopedists, neurosurgeons, neurologists, osteopaths, physical medicine, physical therapy, acupuncture, and mind/body alternative healing modalities. It is almost unimaginable the number of doctors that may consult and the variety of treatments that SCID patients attempt. Most therapies are posited as cure-alls and are focused on the physical mechanics of the spine, rather than the underlying inflammatory/autoimmune process that is silently destroying the tissues inside the spinal canal.

TABLE THREE
CLINICAL PROFILE OF SCID

- Recalls the specific back pain injury/event that initiated the disease
- No improvement with non-prescription medications or physical therapy
- Sought treatment from multiple specialists
- Failed to resolve the pain with surgery and epidural injections
- Laboratory elevations of inflammatory and autoimmune markers and Epstein-Barr antibodies
- Magnetic resonance imaging (MRI) presence of degenerated discs, arachnoid thickening, cauda equina abnormalities, and structural indications of chronic inflammation
- Temporary relief with a corticosteroid challenge (i.e., epidural injection, or 6-day Medrol® dose pak
- Severe pain is constant, often with sciatica, burning skin, and radiation into groin and legs, can often be relieved by positional changes

Note: This clinical profile includes a majority of manifestations but not necessarily all of them.

TABLE FOUR
Epstein-Barr Virus (EBV) Antibodies in 110 Patients
With Adhesive Arachnoiditis Plus Other Spine Disorders

		No. Tested	No. + Positive	%
1	EBV Viral Capsid IgG Antibody (VCA)	106	105	99.1
2	EBV Nuclear Antigen IgG Antibody (EBNA)	105	99	94.3
3	EBV Viral Capsid IgM Antibody (VCA IgM)	94	5	5.3
4	Early Nuclear Antigen IgG Antibody (EA-EBNA IgG)	41	29	70.7
A positive antibody test was elevation above the laboratories' normal limit.				

INITIATION OF INFLAMMATION AND AUTOIMMUNITY

The inflammation of SCID may exist for years after initiation and before pain begins. It is insidious and progressive and isn't extinguished by the usual NSAIDs or physical measures. Patients with SCID recall their initiating event. It is our current belief that many if not most of these cases must reactivate a parasitic virus, usually Epstein-Barr virus.

It is now clear that EBV will reactivate during biologic stress such as a back strain. EBV has now been shown to produce an autoimmune disorder and chronic inflammation.

To help determine if EBV may be an underlying causative factor in SCID, we tested 110 patients with adhesive arachnoiditis who also had other spinal disorders. Over 90% had antibody levels indicating EBV autoimmunity could have been active for several years. Seventy (70%) showed evidence of current reactivation.

At this time, we have no other plausible explanation for long-term chronic inflammation other than EBV reactivation and autoimmunity. It is interesting and supportive of this notion in that inflammation, and immune cells have been identified in degenerated discs and ligamentum flavum.

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SUMMARY

Early in our work and study of adhesive arachnoiditis (AA) we discovered that AA usually develops long after other back and spine pain conditions are in place. In effect AA is the "end of the road" of a long-term chronic inflammatory process that may cause multiple conditions such as herniated discs or Tarlov cysts long before AA develops. To date, the long, inflammatory process has had no name, label, or diagnosis. We have had to assign a diagnosis to this process as persons such as a woman who has had spinal puncture birthing anesthesia and developed severe spine pain and neurologic manifestations. Then they sought our help with the belief they had AA. In reviewing the case there was clear evidence of chronic inflammation by MRI and laboratory tests. Consequently, we simply called the condition, out of necessity, "spinal canal inflammatory disease."

To help clarify this finding, we have made our first attempt to identify, define, and clinically profile the disease. Patients with SCID can usually date the initiation of their condition as it relates to an injury or medical procedure. Rather than heal, our suggestion is that the injury reactivated the Epstein-Barr virus that formed an autoimmune disease and chronic inflammation. Just how widespread it may be in unknown at this time, but chronic inflammation in and around the spinal canal appears to be a disease that needs investigation and study.