FINAL REPORT

STOPPING THE DETERIORATION OF ADHEISVE ARACHNOIDITIS

By

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This study shows that adhesive arachnoiditis is not a hopeless, untreatable disease and that it can be contained and controlled to counter deterioration.

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ADHESIVE ARACHNOIDITIS (AA) STUDY AND EDUCATION PROJECT

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PURPOSE

This study was primarily done to determine reasons why clinical deterioration may occur in adhesive arachnoiditis (AA) and to identify measures that may stop or slow deterioration. To date the onset of deterioration, characterized by lower extremity paralysis, fatigue, mental impairments, bladder/bowel dysfunction, severe pain, physical weakness, and wasting has been irreversible. Deterioration can be slow or rapid and occur in a short time span. The end result has historically produced a bed/house bound state, inability to care for oneself, wasting, and premature death. Terminus has historically been due to overwhelming infection, cardiac arrest, or adrenal failure. Secondary reasons for this study are to confirm the validity of the diagnostic screen of the seven major symptoms and the presence of Epstein-Barr Virus (EBV) reactivation and autoimmunity. Also, this study is to identify the forerunner or precursor conditions of AA. Of great importance is that this study is either to confirm, alter, or enhance the three-component medical protocol to treat AA. The three components are: (10 control of pain; (2) suppress inflammation and autoimmunity; (3) regenerate damaged tissue.

TABLE ONE
PURPOSES OF STUDY

Primary
1. Determine cause(s) of deterioration
2. Identify measures to retard or stop deterioration

Secondary
1. Confirm diagnostic validity of seven major symptoms
2. Confirm EBV reactivation and autoimmunity as a causative factor in AA
3. Identify preceding or precursor conditions
4. Confirm use of three-component medical protocol

METHODS

In November 2024, a written notice was issued to the recipients of our AA bulletin that has been published 2 to 4 times a month for the past two years. Approximately 4400 contacts receive the bulletins. It is estimated that at least half the recipients have the disease and the other half are medical providers. Included among the bulletin recipients are approximately 1500 persons who have had magnetic resonance imaging (MRI) reviewed by us and over 200 who have had Epstein-Barr Virus (EBV) blood tests interpreted by us. A participant had to have AA documented by MRI to be eligible for the study. Within two days after soliciting participants for this study, several dozen applied. We took the first 50 willing participants and gave them questionnaires to complete and return along with their EBV blood test results. Five of the participants submitted incomplete questionnaires and were not included in the final study group. A total of 45 persons with MRI-documented AA and EBV blood tests, therefore, constitute the participants of this study.

BASIC CLINICAL CHARACTERISTICS

The breakdown of females to males was about 2 to 1 which is a typical ration with AA. The average age was 54 years and the average length of the disease was 8.9 years. (Table Two)

INCITING EVENTS AND PREDISPOSING CONDITIONS

Participants were asked about events that may have incited or predisposed them to develop AA. The most remarkable finding was that every participant reported multiple events and/or medical conditions in their life which can compromise the immune system and make a person susceptible to EBV reactivation and autoimmunity. Over 1/3 of the participants had a recognized autoimmune disease such as systemic lupus erythematosus, rheumatoid arthritis, Sjogren's syndrome, or psoriatic arthritis. (Table Three)

TABLE TWO
N=45
BASIC BACKGOUND PROFILE
DEMOGRAPHICS

Females	30 (66.7%)
Males	15 (33.3%)
Age range (in yrs)	33-75 (average 56.4)
Yrs w/AA	.5-33 (average 8.9)

TABLE THREE
INCITING EVENTS AND PREDISPOSING CONDITIONS

Inciting Event	
Trauma	40 (88.9%)
Severe infection	15 (33.3%)
Psychologic event	15 (33.3%)
Infectious mononucleosis	14 (31.1%)
Contaminated spinal fluid	10 (22.2%)
Predisposing Condition	
Anatomic spine abnormality	27 (60.0%)
Autoimmune disease	16 (35.6%)
Ehlers-Danlos syndrome	13 (28.9%)

The 45 study participants all reported that they had at least one inciting event and predisposing medical condition prior to developing AA. This indicates that AA was the result of multiple risk factors and reasons for the development of immunocompromise and EBV reactivation and autoimmunity.

DETERIORATION STATUS OF PARTICIPANTS

Each participant was asked to report their perception of their deterioration status. (Table Four) Fourteen of the 45 (31.1%) reported that their deterioration had stopped, was improving, or stable. The majority: 26 (57.8%) perceived that they were slowly deteriorating and five (11.1%) stated they were rapidly deteriorating. These reports are most significant as they suggest that deterioration of AA can be stopped or reversed. Also, the different groups provide the ability to analyze and possibly extract information on "what works."

TABLE FOUR
DETERIORATION STATUS

Stable (neither better or worse)	5 (11.1%)
Steadily improving	3 (6.7%)
Deterioration has stopped	6 (13.3%)
Slowly deteriorating	26 (57.8%)
Rapid deterioration	5 (11.1%)

FUNTIONAL STATUS

The functional status reported by participants was compatible with their long-standing (8.9 years) duration of the disease. Only about half (25; 55.6%) reported that they were fully functional in that they could move their arms and legs and were neither bed nor housebound. Also, about half had to use a cane, walker, or wheelchair (24; 53.3%).

MAJOR CLINICAL SYMPTOMS

In the past two years we have developed a screening test of seven symptoms common to AA. The purpose of the test is to identify persons with AA at an early clinical stage so they can confirm the diagnosis with a contrast MRI. The seven symptoms are shown here. Use of this screening test was supported in that 43 of the 45 (95.6%) participants had four or more of the seven major symptoms.

TABLE FIVE
THE SEVEN MAJOR SYMPTOMS OF AA

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 sensations such as crawling insects or water dripping down one or both legs?
3	Do you ever have burning pains in your feet and/or groin/crotch area?
4	Does your pain temporarily lessen when you stand or recline?
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, dizziness, or blurred vision?

EPSTEIN-BARR VIRUS (EBV) REACTIVATON AND AUTOIMMUNE STATUS

All 45 participants submitted some EBV test results. The two major antibodies of EBV infection are viral capsid antigen antibody (VCA) and nuclear antigen antibody (EBNA). These two tests were submitted by all 45 participants. Tests for infectious mononucleosis and current reactivation were only submitted by some participants. One participant had chronic mononucleosis, and some had current reactivation as indicated by the test results they submitted.

The VCA was elevated above normal in all 45 participants indicating that one or more reactivations had occurred in their lifetime. Forty-four (97.8%) had elevated EBNA leads suggesting that autoantibodies and autoimmunity was present.

PAIN CONTROL STATUS AND CONTROL

Severe, intractable pain is a historic and almost universal complication of AA. Twenty-two (48.9%) stated they had enough pain control to function and an additional eight (17.8%) not only were able to function but had some pain free hours. (Table Six) Unfortunately about 1/3 of the participants reported such poor pain control that they suffered impairments of physical and mental functions. Only 27 (60%) used opioids and five (11.1%) used low dose naltrexone. Overall, pain control seemed deficient in the group as a whole, since opioids have been essential in the majority of AA cases.

TABLE SIX
PAIN CONTROL STATUS

Well enough to function	22 (48.9%)
Some pain free hours in addition to function	8 (17.8%)
Poor pain control and can't physically/mentally function	15 (33.3%)
No. who used opioids	27 (60.0%)
No. who used low dose naltrexone	5 (11.1%)
No. who used both opioids and LDN	1 (2.2%)

HOW PARTICIPANTS DETERMINED DETERIORATION

Participants were asked how they determined whether or not they were deteriorating. All participants cited more pain. Other than increased pain, participants reported a number of other symptoms that they consider to be a sign of deterioration (See Table below).

TABLE SEVEN
DETERIORATION SIGNS PERCEIVED BY PARTICIPANTS

Increased pain
Paralysis of feet and legs
Loss of bladder/bowel control
Loss of mental capacities
Loss of sexual capacity
Weakness
Fatigue
Anorexia
Muscle wasting
Bed/couch bound

PAINFUL CONDITIONS THAT PRECEDED AA

Participants were asked to recall and identify painful and/or other conditions that preceded the onset of AA. A Table of 27 conditions were listed. No participants had experienced pudendal neuropathy or pancreatitis. The most reported conditions were degenerated discs, migraine headaches, sciatica, and arthritis. The Table shown here lists the conditions experienced by participants.

The painful conditions that preceded AA are all ones that have been historically associated with autoimmunity and/or inflammation. In this situation EBV reactivation and autoimmunity was a likely cause of some if not most of the conditions.

TABLE EIGHT
PAINFUL CONDITIONS EXPERIENCED PRIOR TO AA
N=45

CONDITION	NO. & %
Degenerated discs	30 (66.7%)
Migraine headaches	27 (60.0%
Sciatica	24 (53.3%
Arthritis	23 (51.1%)
Neuropathy in legs/feet	18 (40.0%)
Spinal fluid leak	17 (37.8%)
Irritable bowel	16 (35.6%)
Temporal mandibular joint pain (TMJ)	13 (28.9%)
Carpel tunnel	13 (28.9%)
Tarlov cyst	12 (26.7%)
Bursitis	10 (22.2%)
Plantar fasciitis	9 (20.0%)
Myofascial pain syndrome	8 (17.8%)
Tendonitis	7 (15.6%)
Tethered cord	7 (15.6%)
Fibromyalgia	6 (13.3%)
Small fiber neuropathy	6 (13.3%)
Abdominal/pelvic adhesions	5 (11.1%)
Burning mouth	4 (8.9%)
Thyroiditis	4 (8.9%)
Piriformis syndrome	3 (6.7%)
Costochondritis	3 (6.7%)
Brachial plexus neuropathy	2 (4.4%)
Myalgic encephalomyelitis	2 (4.4%)
Psoriatic arthritis	2 (4.4%)
Trigeminal neuralgia	2 (4.4%)
Crohn's disease	1 (2.2%)

The listed conditions were reported by participants to have preceded adhesive arachnoiditis.

PARTICIPANTS WITHOUT DETERIORATION

Fourteen (14; 31.1%) of the participants stated they were stable and stopped or reversed deterioration. They are reviewed here to identify some therapeutic measures they used that probably helped to halt or reverse deterioration. Their treatment programs were quite comprehensive. The three-components of the standard medical protocol were present and targeted pain, inflammation, and EBV reactivation and autoimmunity. All 14 reported that they had good enough pain relief to mentally and physically function. All took opioids or low dose naltrexone. All took one or more of the medicinals reported to prevent EBV reactivation. Curcumin/turmeric was the most used dietary supplement. Five of the 14 used an antiviral agent. (Table Nine)

All 14 participants who reported to be stable, improving, or ceased deterioration reported good enough pain control to function. Good pain control appeared to be a factor in stopping or

slowing deterioration. Of the 5 persons who reported rapid deterioration only one reported good pain control. Below is a Table showing some of the medical measures used by the 14 participants who didn't have slow or rapid deterioration.

TABLE NINE
MEDICAL TREATMENT OF PARTICIPANTS WITHOUT DETERIORATION
N=14

Had good enough pain control to function	14
Used low dose naltrexone and/or opioids	14
Used dietary supplements known to prevent Epstein-Barr reactivation	13
Used curcumin/turmeric as a supplement	10
Used a periodic corticosteroid	8
Used periodic ketorolac	6
Used peptides	6
Used palmitoylethanolamide (PEA)	6
Used an antiviral (Valtrex® or Acyclovir®)	5
Used both a corticosteroid and ketorolac	5

PARTICIPANTS WITH RAPID DETERIORATION

Only one person with rapid deterioration reported pain control good enough to function although four used opioids. The use of anti-inflammatory and antiviral medicinals were seemingly not used as much as those who reported no deterioration, but the number of subjects is too small to make solid comparisons. EBV reactivation appeared to be a factor in 4 of the 5 cases. One had currently active EBV reactivation, and one had chronic infectious mononucleosis. Two of the rapid cases had EBV viral capsid and antinuclear antibody levels above laboratory test limits. This suggests severe EBV autoimmunity. One case had low or negative EBV antibody levels and did not show current reactivation. There was no obvious cause of this participants' rapid deterioration except that she had poor pain control.

TABLE TEN
SOME CHARACTERISTICS OF PARTICIPANTS WITH RAPID DETERIORATION
N=5

Use of opioids for pain relief	4
Uncontrolled pain	4
Used one or more EBV reactivation preventive medicinals	3
Used a corticosteroid	2
Used ketorolac	1
Used an antiviral agent	0

SLOW DETERIORATION PARTICIPANTS

A majority (26 of 45; 57.8%) of the participants reported they were slowly deteriorating. Review of these cases showed that all took some medicinals from each of the three traditional treatment components: (1) suppression of autoimmunity and inflammation, (2) regeneration of damaged tissues, and (3) pain control. Some had only minor attempts at EBV prevention and treatment.

Eleven (11) of the 26 (42.3%), however, reported pain control as too poor to function. In general, it was believed that most cases could improve their medical treatment. It is unknown how much these participants could have benefitted from enhanced treatment.

ANALYSIS OF FINDINGS

There are essentially no publications on the treatment of AA. Over the past eight years we have developed treatment protocols for AA based on personal clinical experience plus reports and cases reviewed by our Foundations AA study and education project. These protocols have been widely adopted as anecdotal reports claim therapeutic success with these protocols in many persons who have AA. Despite these reports we, to date, have little systematic data to guide future recommendations.

As stated in the background section of this report, the typical history and clinical course of AA has been one of continual, downhill progression with no known remedies to stop it. This study is a small and preliminary effort to identify some measures to slow or stop deterioration. Caution is warranted in the interpretation of this study as it involves a relatively small number of participants who were simply the first ones to answer a solicitation.

This study, despite its caution and shortcomings, answers some basic questions about AA treatment. First and foremost is that deterioration of AA can be stopped or even reversed. About a third (14 of 45; 31.1%) stated they had stopped deterioration, were stable, or even improved. Only 5 of 45 (11.1%) reported they were rapidly deteriorating, which has historically been the usual clinical course. The remainder reported slow rather than rapid deterioration, and this finding was probably due to treatment.

Although no singular treatment agent stood out, those participants who stopped or held off deterioration had good pain control and took preventive and suppressive measures to control EBV reactivation and autoimmunity. The five participants with rapid deterioration did neither.

Helpful information was obtained on some secondary concerns and issues. Only one participant out of 45 did not show EBV antibodies compatible with recurrent reactivation and autoimmunity. This is essentially an identical finding to our prior EBV surveys in persons with AA. Participant reported several precipitating events and/or predisposing medical conditions that likely caused immune-suppression and EBV reactivation. Participants reported one or more medical conditions prior to AA that have often been associated with autoimmunity and/or inflammation. These findings suggest that participants had an ongoing, chronic autoimmune process that caused painful conditions prior to the development of AA.

Forty three of the 45 participants reported four or more of the seven major symptoms of AA. This is additional confirmation that these seven symptoms provide a diagnostic screening test for AA that should be confirmed by magnetic resonance imaging (MRI).

Despite the small number of participants and lack of definitive data, this study is very encouraging. The three-component medical protocol recommended by us in recent years appeared to be beneficial for most participants. It points out that AA is not hopeless and that it can be contained or controlled even though cure may not be possible.

CONCLUSIONS

Although this is a small study it establishes the fact that at least some persons with AA can be treated and deterioration abated. The traditional mantra that AA is untreatable and hopeless should be discarded. The three-component medical protocol of pain control, suppression of inflammation and autoimmunity, and regeneration of damaged tissues should be continued. The protocol component of inflammation and autoimmunity suppression should have a special focus on Epstein-Barr virus reactivation and autoimmunity.

BACKGROUND FOR THIS STUDY

BASIC CAUSE OF DETERIORATION

In the first five years of our project, we were able to determine that AA is an inflammatory disease of spinal tissues. To develop the disease, one has to simultaneously have inflammation in two tissues: (1) cauda equina nerve roots and (2) arachnoid membrane (inner lining of the canal cover). Inflammation produces sticky adhesions which glue cauda equina nerve roots to each other. Microscopic masses were formed when the two tissues stuck together. The masses were like boulders in a creek that disturb water flow. In this case, spinal fluid flow was interrupted and diverted which lead to the "head" symptoms noted above (i.e., headache, blurred vision). The microscopic masses consisting of cauda equina nerve roots and arachnoid are small "balls of fire" that are hot and painful. They entrap the nerves that connect to the feet, legs, bowel, sex organs, bladder. The fiery, inflamed microscopic masses continue to enlarge, entrap, and destroy nerve connections. Hence, the cause of continuous and progressive deterioration.

FIRST GENERATION TREATMENT APPROACH

Once we realized that AA was an intraspinal inflammatory-adhesive disease, a search began for medicinals that would suppress inflammation that is inside the spinal canal. The usual anti-inflammatory agents and the most popular corticosteroid, prednisone, were essentially of no help. Through patient reports from multiple countries and our own clinical trials, these two anti-inflammatory medicinals appeared to reduce pain and improve mobility, motivation, and a number of physiologic and mental functions: (1) ketorolac, (2) methylprednisolone (dexamethasone may substitute for methylprednisolone). These two medicinals remain the core of AA treatment.

A third agent proved very effective, the anabolic hormone, human chorionic gonadotropin (HCG). It was essentially removed from compounding pharmacies a few years ago (may be available now).

Another revelation was made early on. The micro-masses that entrapped cauda equina nerves could be somewhat neutralized with some spinal fluid flow exercises, and lower extremity paralysis could be slowed with regular walking and the extending and flexing of the feet and legs.

CAUSE OF PROGRESSIVE DETERIORATION

Progressive deterioration in AA is partially due to ongoing chronic inflammation, adhesions, and scar formation in the micro-masses. Nerves may be permanently destroyed or rendered dysfunctional. Spinal fluid flow may be more impaired.

We have learned, however, that the cause of AA is due to a disease process that originates outside the spinal canal. This process may adversely affect the pain control system in the brain and spinal cord (central nervous system-CNS). Pain may increase and overall disability may set in.

AUTOIMMUNITY

Recent research has determined that the pathologic process known as autoimmunity is the cause of the inflammation in the cauda equina and arachnoid membrane. Autoimmunity is simply defined as a process in which some element in the body attacks its own tissues and causes inflammation and destruction. At this time the autoimmunity that produces AA is almost always related to an infectious agent that is normally a harmless parasite in the body. The major parasite that appears responsible for AA is Epstein-Barr Virus (EBV), but some others may act in conjunction with EBV or by themselves. The parasite reactivates to invade tissues or produce autoimmunity after severe biologic stress (elevation of cortisol and adrenalin) such as an accident, spinal medical procedure, infection, or psychologic stress.

TAKING THE NEXT STEP IN FIGHTING DETERIORATION

We've come a long way and now know a lot about treating AA. This study has pointed out a number of areas where protocol revision and subsequent education may help the AA community.