REPORT

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PREVENTION AND TREATMENT OF EPSTEIN-BARR VIRUS IN ADHESIVE ARACHNOIDITIS

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Adhesive arachnoiditis (AA) is a very serious, chronic, inflammatory disease in which cauda equina nerve roots become adhered ("glued") to the arachnoid membrane by adhesions. The disease is characterized by severe intractable pain and neurologic impairments of bladder, bowel, sex organs and lower extremities. Left untreated, the disease is progressive and may result in severe debilitation, a bed-bound state, and premature death. Although AA may be initiated by injury, trauma or infection such as Lyme disease, the chronic, unremitting inflammation that maintains AA appears to have a different origin or cause. To date, the only logical cause of the unremitting, progressive inflammation of AA that we have identified is related to the Epstein-Barr virus (EBV). This report provides our current recommendation on who should be tested and treated for EBV.

Who Should Be Tested?

Any person with MRI-documented AA who has intractable pain should be tested for EBV antibodies.

Which Cases of AA Should Be Treated?

1. Persons with MRI-documented AA whose two main EBV antibodies, which are listed here, are at least two times above normal range:
    a. Viral capsid antigen antibody (VCA)
    b. EBV nuclear antigen antibody (EBNA)
2. Person has intractable pain defined here as constant pain likely due to glial cell dysfunction ("centralized pain").[2]

The EBV Pathologic Process

A major characteristic of the herpes class of viruses (EBV is herpes 4) is that they may colonize body tissues and remain asymptomatic for long periods. Examples are herpes labialis and herpes zoster (shingles). If EBV reactivates it may cause two pathologic processes that can lead to AA:

1. Colonize the arachnoid membrane and/or cauda equina nerve roots
2. Develop autoimmunity with serum autoantibodies or local tissue cells that are biologically altered (term is mimicry)

Either of these two processes may individually or collectively cause tissue degeneration over a long period of time before symptoms become apparent. This is the situation with the cancers produced by EBV.[3] If the arachnoid membrane or cauda equina nerves have been injured or traumatized by an accident or medical procedure such as an epidural injection, they may become

more prone to EBV colonization and/or an autoimmune process which leads to inflammation to be followed by tissue degeneration.

Three Step Medical Protocol in AA Patients

Step 1- Prevent Future Reactivation: A number of vitamins, minerals, and herbals have been found in laboratory studies to prohibit reactivation of EBV.[1] See Attached Tabel. We recommend that two or more of these medicinals be used to prevent future EBV reactivation.

Step 2-Monolaurin or Thymosin: These 2 medicinals clinically appear to suppress EBV activity in viruses that have colonized in tissue or may have reactivated.

Step 3-Ivermectin: Ivermectin is used to eliminate EBV and prevent colonies of EBV from developing in the arachnoid membrane, cauda equina, intervertebral discs, and glial cells in the central nervous system. A dose of 6 to 12 mg daily for 5 to 7 days followed by 3 to 6 mg on 1 to 2 days a week is the basic recommendation. Some persons with AA find that ivermectin provides significant pain relief due to its anti-inflammatory properties, so they take it more often than two days a week. Also, some persons take a daily dose of 12.0 mg. If ivermectin fails to improve symptoms after 6 to 8 weeks, discontinuation should be considered.

Some Specifics of EBV Testing and Treatments

Medical practitioners and patients need to be aware of these specific aspects of EBV testing and treatment.

1. EBV autoimmunity and colonization have no specific laboratory test. Autoimmunity and colonization may have occurred in the distant past and be severe even if the laboratory tests for active EBV, VCA IgM, polymer chain reaction (PCR) DNA, and early EBNA, are negative.
2. Antiviral drugs are recommended if laboratory tests indicate current reactivation, (positive PCR-DNA or early EBNA). (See Table.)
3. EBV antibody tests (VCA, EBNA) will usually not reduce with treatment as they are fixed, protective antibodies.
4. Reactivation of EBV is difficult to stop. There are no published test results showing that early EBNA or positive PCR-DNA tests have been converted from active to inactive, regardless of treatment.
5. Many clinical decisions on medications such as ivermectin will necessarily have to be made on the patient's report of symptom improvement. Cessation of ivermectin and antiviral agents should be considered if no clinical improvement is observed after 6 to 8 weeks of therapy.

6. EBV autoimmunity and colonization both cause considerable inflammation in and outside the central nervous system. Anti-inflammatory medications such as diclofenac, methylprednisolone, pentoxifylline, and metformin may play a tissue protection role.

Tables Preventives

TABLE Medicinals For Prevention of Epstein-Barr Virus Reactivation	
Vitamin C	Astragalus
Vitamin D	Curcumin
Resveratrol	Andrographis
Luteolin	Cimetidine
Selenium	Lysine
Zinc	

Standard Antiviral Agents

Acyclovir (Zovirax®, 400 to 1000 mg a day)
Valacyclovir (Valtrex®, 500 to 1000 mg a day)
Famciclovir (Famvir®, 500 to 1000 mg a day)

References

1. Kerr J. Epstein-Barr virus (EBV) reactivation and therapeutic inhibitors. *J Clin Pathol* 2019;0:1-8.
2. Jakhmola S, Jho HM. Glial cell response to Epstein-Barr virus infection: a plausible contribution to virus-associated inflammatory reactions in the brain. *Virology* 2021;559:182-195
3. Tennant F. Handbook for Epstein-Barr virus: testing and treatment in severe chronic pain conditions. 2024 *Tennant Foundation, West Covina, Calif.*