CAUDA EQUINA INFLAMMATORY DISORDER (CEID): ANTECEDENT OF ADHESIVE ARACHNOIDITIS (AA)

To develop the dreadful disease of AA, one must have inflammation in both cauda equina nerve roots and the arachnoid membrane. Inflammation must produce adhesions which glue the two tissues together. Inflammation must be present in the cauda equina before symptoms of AA can develop and this condition, in itself, is a disorder that can be identified, treated, and hopefully prevented from progressing to AA.

Symptoms of CEID: The symptoms of CEID are essentially the same as AA and include pain, burning feet, urinary dysfunction, and sensation of water dripping or insects on the skin. Headache, dizziness, sweating, and fever may be present. The pain can radiate into groin, buttocks, legs, and feet. It may be temporarily relieved on standing or reclining. The pain can range from mild to severe and require symptomatic medical relief. The arachnoid membrane has no nerves, so it is essentially asymptomatic when inflamed.

Initiation of CEID: Since CEID is the antecedent to AA, it is initiated by the same causative factors as those ascribed to AA:

1. Trauma (accident, medical procedure including spinal taps and epidural injections)
2. Chemical contaminant of injection
3. Infection (Lyme, virus)
4. Autoimmune disease (lupus, psoriasis)

The Prologue or Precursor Period: CEID may exist for weeks to years before inflammation develops enough adhesions to develop AA.

MRI Findings: The major findings are swelling or enlargement of nerve roots. They may become displaced (i.e. shifted) from their normal symmetrical pattern. Some may coalesce with each other but not to the arachnoid. Other tissues, especially the arachnoid, dura, ligamentum flavum may show thickening due to inflammation and edema.

Laboratory Test: Some leukotrienes (cytokines) may elevate as well as other inflammatory markers. Epstein-Barr virus testing may show reactivation and/or autoimmunity.

Treatment: Same as AA. Initial focus should be on symptomatic pain relief and suppression of inflammation.

Summary: The diagnosis of CEID will now be applied to many persons who have symptoms but have not yet developed AA. A hope is that diagnosis and treatment of CEID will prevent AA