ADHESIVE ARACHNOIDITIS (AA)
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SELECT CORTICOSTEROIDS ARE ESSENTIAL FOR ADHESIVE ARACHNOIDITIS (AA)

AA is a serious disease with severe suffering, neurologic impairments, and a shortened lifespan. WHY? Once inflammation starts inside the spinal canal, it apparently never, or rarely, goes totally away.

Some persons with severe asthma and rheumatoid arthritis must take a corticosteroid (CS) for years and don't experience serious side effects. We believe all persons with typical AA symptoms and documentation of the disease on an MRI must take one of two CS's – methylprednisolone or dexamethasone for the spinal canal inflammation and pain of AA.

TWO KEY POINTS	
1. Currently there is no other medication agent that consistently and predictably suppresses intraspinal canal inflammation and reduces pain.	**2. Do not expect to halt progression or have much recovery if you do not consistently take a CS.**

THERAPEUTIC DOSING FOR AA

1. Maintenance-low dose of dexamethasone (.5 to .75mg) or methylprednisolone (Medrol®) 2 to 4 mg on 2 to 5 days a week. Skip days between dosages.

An alternative to the above is a weekly or bi-monthly injection of methylprednisolone or dexamethasone. Injections are usually the answer to corticoid sensitivity or gastric upset.

2. For flares-6 Day Medrol Dose Pak or an injection of methylprednisolone or dexamethasone preferably mixed with a standard dose of injectable ketorolac.

SELECT CORTICOSTEROID

Dexamethasone and methylprednisolone are the preferred CS's because they cross the blood brain barrier, enter spinal fluid, and act on glial cells. Prednisone and hydrocortisone are not as consistently effective as dexamethasone and methylprednisolone.

AVOIDANCE OF SIDE EFFECTS

The fear of corticosteroids comes from daily use of high doses, not from low, intermittent dosages.

References
1. Takedo, etal. Effect of methylprednisolone on neuropathic pain and spinal glial activation in rats. Anesthesiology 2004;100:1249-1257.
2. Kiefer, etal. Effects of dexamethasone on microglial activation in vitro. J Neuroimmunology 1991;34:99-108.

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