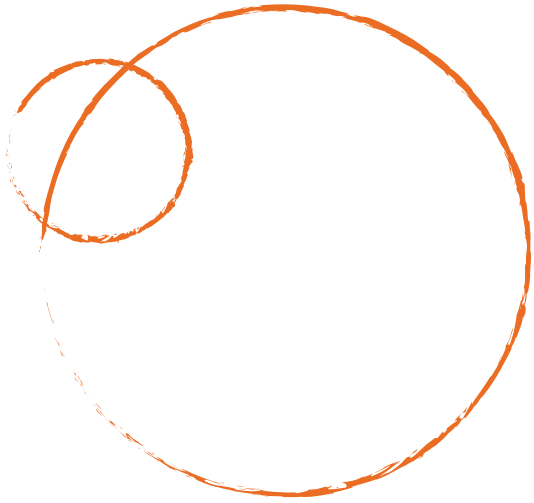
Fact Sheet

Acute Disseminated Encephalomyelitis

ADEM



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Acute Disseminated Encephalomyelitis (ADEM) is a rare inflammatory demyelinating disease of the central nervous system. ADEM is thought to be an autoimmune disorder in which the body's immune system mistakenly attacks its own brain tissue, triggered by an environmental stimulus in genetically susceptible individuals. In most cases, ADEM is believed to be triggered by a response to an infection, or much less likely, to a vaccination. For this reason, ADEM has sometimes been referred to as post-infectious or post-immunization acute disseminated encephalomyelitis.

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Epidemiology

According to a study published in 2008, the estimated incidence of ADEM in California is 0.4 per 100,000 population per year, and there are approximately 3 to 6 ADEM cases seen each year at regional medical centers in the US, UK, and Australia.[1] ADEM is more common in children and adolescents than it is in adults, and there does not seem to be a higher incidence of ADEM among males or females, nor does there seem to be a higher frequency among any particular ethnic group.

Post-infectious

In approximately 50-75 percent of ADEM cases, the inflammatory attack is preceded by a viral or bacterial infection. There have been a large number of viruses associated with these infections, including but not limited to measles, mumps, rubella, varicella zoster, Epstein-Barr, cytomegalovirus, herpes simplex, hepatitis A, influenza, and enterovirus infections. A seasonal distribution has been observed showing that most ADEM cases occur in the winter and spring. The inflammatory syndrome and neurological symptoms often begin within a couple of weeks after the viral or bacterial illness. No infectious agent is isolated in most cases.

Post-immunization

Less than 5 percent of ADEM cases follow immunization.[1] Although a temporal association between immunization and inflammatory demyelination has been reported, a direct causal relationship has not been proven. Post-vaccinal ADEM has been associated with immunization for rabies, hepatitis B, influenza, Japanese B encephalitis, diphtheria/pertussis/tetanus, measles, mumps, rubella, pneumococcus, polio, smallpox, and varicella. Currently, the measles, mumps, and rubella vaccinations are most commonly associated with post-vaccinal ADEM. The incidence of ADEM associated with the live measles vaccination is 1 to 2 per million. Neurologic symptoms typically appear 4 to 13 days after a vaccination.

Signs and Symptoms

The initial neurological symptoms of ADEM may include fever, headache, and vomiting. Encephalopathy (abnormal functioning of the brain) is a characteristic feature of ADEM. It usually develops rapidly and results in symptoms such as altered level of consciousness, acute cognitive dysfunction, behavioral changes, and seizures in about a third of cases. The altered consciousness can range greatly from sleepiness to lethargy to coma.

In addition to encephalopathy, other common neurologic signs of ADEM include long tract pyramidal signs (decreased voluntary movement), acute hemiparesis (muscle weakness on one side of the body), cerebellar ataxia (decreased coordination), and cranial neuropathies (abnormalities in eye and face movement). ADEM is multifocal, which means inflammation can occur in the brain, optic nerves (optic neuritis or ON) and/or spinal cord (transverse myelitis, or TM). Thus, a child or adult with ADEM can have the symptoms of ON such as impaired vision and eye pain, and/or TM such as motor and sensory abnormalities. TM symptoms may depend on the severity and the level of the attack in the spinal cord. These can include impaired breathing, bowel and

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bladder dysfunction, paralysis or muscle weakness, spasticity, paresthesias, or nerve pain. ADEM symptoms can persist for a few days or several weeks. Peak severity of the syndrome is typically reached within 4 to 7 days, though acute symptoms may last 2 to 4 weeks.

Diagnosis

The diagnosis of ADEM is based on clinical and radiologic characteristics. Unfortunately, there is no specific biological marker or confirmatory test to specifically identify the disorder, nor have there been large-scale, randomized, controlled studies focused on the diagnosis and treatment of ADEM. Decisions about the diagnosis and treatment of this disorder are based primarily on the opinions of experts. Since decisions are often based on clinical judgment, management of ADEM by a healthcare provider who is familiar with the syndrome is critically important.

A diagnosis of ADEM is considered when individuals develop multifocal neurological abnormalities such as confusion, excessive irritability, or altered level of consciousness (encephalopathy), especially if the onset of symptoms occurs within 1 to 2 weeks after a viral/bacterial infection or, rarely, after a vaccination. Physicians must rule out that there is a direct infection of the central nervous system as opposed to an infection that subsequently triggers a misdirected immune response. Should a direct infection be suspected, an antibiotic and/or antiviral drug should be initiated to treat the presumed infection.

Laboratory studies include a complete blood count and blood and cerebrospinal fluid (CSF) cultures, and serological (antibody) studies are performed on blood and CSF to detect bacterial and viral organisms. Additionally, viral cultures can be obtained from nasopharynx and stool. Testing for myelin oligodendrocyte glycoprotein (MOG) and neuromyelitis optica (NMO)/aquaporin-4 antibodies should be completed to evaluate for ADEM mimics that can cause relapsing demyelination.

A lumbar puncture is also performed to evaluate for evidence of inflammation in the cerebrospinal fluid (CSF). Common findings include pleocytosis (increased white blood cell count) and/or increased protein concentration, but normal CSF findings do not exclude the diagnosis of ADEM. Increased and unique antibody production in the CSF can be assessed with measuring IgG index and oligoclonal bands, respectively. While the latter is most commonly associated with multiple sclerosis (MS), these tests can sometimes be abnormal in ADEM as well.

An MRI of the brain and spine is important to establish a diagnosis of ADEM. Abnormalities are best defined by T2-weighted images, FLAIR sequences, and contrast-enhanced MRI with gadolinium. Abnormalities on MRI usually vary in

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location. Lesions associated with ADEM tend to be bilateral, asymmetric, and have poorly defined margins. Multiple lesions in the deep and subcortical white matter are common. Involvement of cortical and deep gray matter structures can also be seen, especially among children. ADEM lesions are typically large, with diameters ranging from <5 mm to 5 cm, and multiple in number, while presentations with few and/or small lesions are also possible. Additionally, brainstem and spinal cord abnormalities on MRI are common in ADEM. In the spinal cord, large confluent intramedullary lesions extending over multiple vertebral segments of the spinal cord are typical.

It is possible that the MRI may be normal early in the course of the illness and abnormalities only be visualized on subsequent imaging studies. After the acute period, physicians often will repeat the MRI, usually 3-6 months after ADEM presentation, to follow up on the evolution of prior brain lesions and survey for new lesions, which could change the diagnosis from ADEM to multiphasic ADEM (see below) or MS.

In making the diagnosis of ADEM, it is important to consider other inflammatory demyelinating disorders such as multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), and MOG antibody disease (MOGAD), in which an ADEM-like presentation may be the first manifestation of a relapsing, demyelinating disease.

Diagnostic Criteria

An important paper published by the International Pediatric Multiple Sclerosis Study Group proposed diagnostic criteria and provided treatment recommendations to facilitate improved medical decision-making and formalize research on ADEM in children.[2]

The major criteria include:

- A first clinical attack of central nervous system demyelinating disease with acute or subacute onset, polysymptomatic neurologic features, and encephalopathy
- Brain MRI showing focal or multifocal lesions, predominantly involving the white matter, without evidence of previous white matter changes
- Encephalopathy as a presenting symptom, with the onset of encephalopathy corresponding with the occurrence of the disease state (encephalopathy is defined to include behavioral changes, such as lethargy or irritability, or severe changes in the level of consciousness such as coma)

These features help distinguish ADEM from other clinically isolated syndromes, which have a greater risk for recurrence and subsequent diagnosis of MS. The authors of the publication define three different categories of ADEM:

- Monophasic ADEM is a one-time episode that can develop over a period for as long as three months. Any new or changing symptoms within this three-month period is considered as one event. Symptoms that might occur during an oral steroid taper or within one month of the completion of the taper are also classified as one single episode. Recurrent and multiphasic ADEM episodes must occur more than three months after the initial event and more than one month after the completion of steroids.

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- Recurrent ADEM is defined as a subsequent attack that involves the same symptoms that occurred during the initial attack. The MRI findings tend to be similar to the initial attack, and there are no lesions, but there could be an enlargement of the lesions from the original episode.
- Multiphasic ADEM is defined as an attack that involves new areas of the central nervous system from the initial or previous attacks. There must be signs of encephalopathy, but symptoms and neuroimaging findings are in different areas from the initial attack. There might be new lesions evident on MRI, and there might also be evidence of partial or complete resolution of the lesions associated with the first episode.

The International Pediatric MS Study Group authors also provide guidance on the variables that might distinguish ADEM and MS. ADEM more frequently occurs among younger age groups (<10 years), and there does not seem to be a difference between male versus female incidence. MS occurs more frequently in adolescents and young adults, and the incidence is higher for females than for males. A prior flu-like illness is reported in ADEM but is less common with MS. Encephalopathy is required to satisfy the diagnosis of ADEM, while it is rare in initial presentations of MS. Seizures can occur in ADEM but is not typical with MS. A single event in ADEM is defined as symptoms confined to three months, while in MS, discrete events are separated by at least four weeks. Large lesions involving gray and white matter are frequently evident from MRI in ADEM and rare in MS, which usually manifests as smaller, well-defined, ovoid lesions. MRI can reveal contrast enhancement in both ADEM- and MS-related lesions. Over time, lesions may regress and even resolve completely in ADEM, while in MS, old lesions often evolve but do not disappear, and new lesions develop over time if the disease is untreated. Significant CSF pleocytosis (presence of a greater number of cells than normal) is variable in ADEM but extremely rare in MS (white blood cell count is almost always <50). Finally, the presence of oligoclonal bands in the spinal fluid is variable in ADEM and frequently found in MS.

3 Acute Treatments

Treatment strategies for ADEM are largely derived from opinions of experienced clinicians, descriptive cohort studies, and/or reports from expert committees. Standard of care treatments in acute ADEM have not been proven by randomized, placebo-controlled trials.[3] Since patients with ADEM usually present with fever, meningeal signs, acute encephalopathy, and evidence of inflammation in blood and CSF, it is important to first consider use of antibiotic and/or antiviral therapies (i.e., acyclovir) until an infectious cause is ruled out. High dose intravenous corticosteroids for 3-5 days is considered to be the first-line treatment for ADEM and can be used concurrently with antibiotics and acyclovir. Plasma Exchange (PLEX) should be considered in very aggressive forms of ADEM or if there is limited response to corticosteroids. Intravenous immunoglobulin (IVIG) can also be considered if PLEX is not available or there are contraindications to PLEX. The strength of evidence for the recommendation of corticosteroids and PLEX are graded as moderate. The strength of evidence for a recommendation of IVIG is poor.

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It should be noted that no studies have compared IVIG treatment with corticosteroids or plasma exchange, and there is debate over whether PLEX or IVIG should be used at the beginning, or only when corticosteroids fail to work.

Prognosis and Management

The prognosis for most individuals with ADEM is good. The recovery process may take place over four to six weeks, but the majority of those with ADEM make a fairly significant recovery. Between 60 to 90 percent are left with no neurological deficits. Those who do have residual symptoms can have residual weakness or sensory disturbances, bowel/bladder dysfunction, recurrent headaches, and behavioral and neurocognitive problems. Interestingly, the location of lesions and the extent of inflammatory lesions do not appear to have any predictive value in regard to outcome. Typically, follow-up MRIs show complete or partial resolution of abnormalities in the majority of ADEM cases. Those who are positive for MOG antibodies are unlikely to be later diagnosed with multiple sclerosis or NMOSD, but may have relapses of ADEM, optic neuritis, and transverse myelitis.

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Long-term clinical follow-up and sequential imaging by MRI are helpful to increase confidence of a diagnosis of ADEM. Should there be clinical relapses with evidence of new MRI lesions, it is not compatible with a diagnosis of monophasic ADEM, and depending on the clinical and imaging features, the diagnosis may be modified to either multiphasic ADEM or MS. Although consensus is lacking, some physicians recommend that those with ADEM receive follow-up MRIs for a period of up to five years to ensure that there is no new inflammatory activity after the initial ADEM attack (i.e., to exclude the diagnosis of MS.)

After the acute phase, rehabilitative care to improve functional skills and prevent secondary complications of immobility involves both psychological and physical accommodations. There is very little written in the medical literature specifically dealing with rehabilitation after ADEM. However, much has been written regarding recovery from spinal cord injury (SCI) in general, and this literature applies. The physical issues include bowel and bladder management, sexual dysfunction, maintenance of skin integrity, spasticity, activities of daily living (i.e., dressing), mobility, and pain.

It is important to begin occupational and physical therapies early during the course of recovery to prevent the inactivity-related problems of skin breakdown and soft tissue contractures that lead to a decreased range of motion. Assessment and fitting for splints designed to passively maintain an optimal position for limbs that cannot be actively moved is an important part of the management at this stage.

The long-term management of ADEM requires attention to a number of issues. These are the residual effects of any spinal cord or brain injury, including ADEM. In addition

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to chronic medical problems, there are the ongoing issues of ordering the appropriate equipment, reentry into school, re-socialization into the community, and coping with the psychological effects of this condition by the patients and their families. During the early recovery period, family education is essential to develop a strategic plan for dealing with the challenges to independence following return to the community.

Neurocognitive Issues

Individuals who have had ADEM may have difficulties with attention, short-term memory, decision making and processing speed that impacts their ability to return to their prior level of functioning in school and their occupation. Neuropsychological evaluation may be helpful in quantifying these cognitive deficits and providing recommendations for work and school accommodations such as extended time for assignments and rest breaks.

Visual Issues

For patients who have had optic nerve inflammation, residual vision loss can be experienced. Patients can possibly note blurred vision, loss of color vision, difficulty with depth perception and glares or halos around lights at night. Furthermore, patients who fully recover vision after optic neuritis may experience transient returns of blurred vision during times of stress, exertion, or heat exposure.

Bladder Function

Bladder function is almost always at least transiently impaired in patients with inflammation in the spinal cord (TM). Immediately after the onset of TM, there is frequently a period of transient loss or depression of neural activity below the involved spinal cord lesion, referred to as "spinal shock," which lasts about 3 weeks. Following this period, two general problems can affect the bladder. The bladder can become overly sensitive and empty after only a small amount of urine has collected, or relatively insensitive, causing the bladder to become over extended and overflow. An overly distended bladder increases the likelihood of urinary tract infections and, in time, may threaten the health of the kidneys. Depending on the dysfunction, treatment options include timed voiding, medicines, external catheters for males (a catheter connected to a condom), padding for women, intermittent internal self-catheterization, an indwelling catheter, or electrical stimulation. Surgical options may be appropriate for some people. Common bladder problems include incontinence, frequency, nocturia (frequent urination at night), hesitancy, and retention. Treating incontinence, frequency, and nocturia is often easier than treating hesitancy and retention, where clean intermittent urinary catheterizations are the basic component to success. Working with a good urologist is imperative to prevent potential serious complications, particularly one who understands spinal cord disease. Urodynamic testing is necessary to determine urine retention to check risk for urinary tract infections, particularly if there is a history of UTIs to guide the urologist in terms of the best management.

Bowel Function

Another major area of concern is effective management of bowel function. A common problem in spinal cord injury is difficulty with evacuation of stool, although fecal incontinence can also occur. The neurologic pathways for defecation are similar to those of the bladder. Many lacking voluntary control of the bowel may still be able to achieve continence by diet, strategic use of stool softeners and fiber, and the

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technique of rectal stimulation. Other aids include suppositories, anal irrigation, and oral medications. A high-fiber diet, adequate and timely fluid intake, and medications to regulate bowel evacuations are the basic components of success. Regular evaluations by medical specialists for adjustment of the bowel program are recommended to prevent potentially serious complications. There are some surgical options, although this is rarely necessary.

Sexual Dysfunction

Sexual dysfunction involves similar innervation and analogous syndromes as those found in bladder dysfunction. Treatment of sexual dysfunction in adults with ADEM should take into account baseline function before the onset of ADEM. Of the utmost importance is adequate education and counseling about the known physical and neurologic changes that ADEM has on sexual functioning. Because of the similarities in innervation between sexual and bladder function, patients with sexual dysfunction should be encouraged to empty their bladders before sexual stimulation to prevent inopportune incontinence. The mainstays of treatment of erectile dysfunction in men are inhibitors of cGMP phosphodiesterase, type 5, which will allow most men with TM to achieve adequate erections for success in intercourse through a combination of reflex and/or psychogenic mechanisms. Although less effective in women, these same types of medications have been shown capable of enhancing a woman's sexual functioning. The most commonly used oral erectile dysfunction drugs are Viagra (sildenafil), Levitra (vardenafil), and Cialis (tadalafil). Although sexual experience is impacted by spinal cord injury, sensual experience and even orgasm are still possible. Lubricants and aids to erection and ejaculation (for fertility) are available. Adjustment to altered sexuality is aided by an attitude of permissive experimentation, as the previous methods and habits may no longer serve.

Skin Breakdown

Skin breakdown occurs if the skin is exposed to pressure for a significant amount of time, without sensation or the strength to shift position as necessary. Sitting position should be changed at least every 15 minutes. This can be accomplished by standing, by lifting the body up while pushing down on armrests, or by just leaning and weight shifting. Wheelchairs can be supplied with either power mechanisms of recline or tilt-in-space to redistribute weight bearing. A variety of wheelchair cushions are available to minimize sitting pressure. Redness that does not blanch when finger pressure is applied may signal the beginning of a pressure ulcer. Good nutrition, vitamin C, and avoidance of moisture all contribute to healthy skin. Pressure ulcers are much easier to prevent than to heal.

Spasticity

Spasticity means stiffness or muscle spasms and is often a very difficult problem to manage. Some stiffness in our muscles is necessary in order to control our movement, but when they become too tight, the result can range from slightly bothersome stiffness (particularly upon wakening) to uncontrollably painful spasms. When the latter occurs, small triggers such as changes in position, temperature, humidity, or presence of infections can cause this painful spasticity. The key goal is to remain flexible with exercise, a daily stretching routine, and a bracing program with splints, as needed. These splints are commonly used at the ankles, wrists or elbows. Also recommended are appropriate strengthening programs for the weaker of the spastic

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muscles acting on a joint and an aerobic conditioning regimen. These interventions are supported by adjunctive measures that include antispasticity drugs (e.g., diazepam, baclofen, dantrolene, tizanidine), therapeutic botulinum toxin injections, and serial casting. In cases where spasticity is severe, a baclofen pump, which provides the medication directly to the spinal cord, may be considered. The therapeutic goal is to improve the function of the individual in performing specific activities of daily living (i.e., feeding, dressing, bathing, hygiene, mobility) by improving the available joint range of motion, teaching effective compensatory strategies, and relieving pain. Left untreated, severe spasticity can lead to shortening of the affected muscle or joint called contractures, further impacting mobility, rehabilitation, and independence.

Pain

Changes in sensation often occur and can manifest as lack of sensation, or numbness, as well as painful sensations called neuropathic pain. This pain is described in many different ways, including burning, squeezing, stabbing, or tingling. Having the sensation of pain means the nerve signal is getting through, but in an inappropriate way. While this can get better over time, there is a long list of medications to treat these symptoms. The same medication doesn't work for everyone, so the trial and error of finding the right medication can be frustrating. Alternative therapies such as acupuncture and meditation have also been utilized, with varying success.

While the body is constantly working toward repair, once damage is done to the central nervous system, there will always be evidence of this damage, usually evidenced on an MRI. Clinical fluctuations of old symptoms, particularly in the setting of infection, stress, heat (Uhthoff's phenomenon), menstrual cycle, or anything that increases core body temperature or throws the body off of its normal course are also possible. It is important to note that this is not inflammatory driven and therefore in no way represents worsening of the condition.

The first step in treating pain effectively is obtaining an accurate diagnosis. Unfortunately, this can be very difficult. Causes of pain include muscle strain from using the body in an unaccustomed manner, nerve compression (i.e., compression of the ulnar nerve at the elbow due to excessive pressure from resting the elbow on an armrest continuously) or dysfunction of the spinal cord from the damage caused by the inflammatory attack. Muscle pain might be treated with analgesics, such as acetaminophen (Tylenol), non-steroidal, anti-inflammatory drugs such as naproxen or ibuprofen (Naprosyn, Aleve, Motrin), or modalities such as heat or cold. Nerve compression might be treated with repositioning and padding (i.e., an elbow pad for an ulnar nerve compression).

Nerve pain can be a significant challenge to find effective treatment. Nerve messages traveling through the damaged portion of the spinal cord may become scrambled and misinterpreted by the brain as pain. Besides the treatments listed above, certain antidepressants such as amitriptyline (Elavil), or anticonvulsants, such as carbamazepine, phenytoin, or gabapentin (Tegretol, Dilantin, Neurontin) may be helpful. Stress and depression should also be addressed, since these conditions make pain harder to tolerate.

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Depression

Individuals with ADEM should be educated about the effect of ADEM on mood regulation and routinely screened for the development of symptoms consistent with clinical depression. Warning signs that should prompt a complete evaluation for depression include failure to progress with rehabilitation and self-care, worsening fixed low mood, pervasive decreased interest, and/or social and professional withdrawal. A preoccupation with death or suicidal thoughts constitutes a true psychiatric emergency and should lead to prompt evaluation and treatment. Depression in ADEM is similar to the other neurologic symptoms patients endure, which are mediated by the effects of the immune system on the brain. Depression is not due to personal weakness or the inability to "cope." It can have devastating consequences; not only can depression worsen physical disability (such as fatigue, pain, and decreased concentration) but it can have lethal consequences. Despite the severity of the clinical presentation of depression in ADEM, there is a very robust response to combined aggressive psychopharmacologic and psychotherapeutic interventions. With appropriate recognition and treatment of ADEM depression, complete symptom remission is standard.

Ongoing problems typically include ordering the appropriate equipment, dealing with re-entry into school, work, and community, and coping with the psychological effects of this condition on both those diagnosed with ADEM and their families. Being saddened or demoralized by the diagnosis of ADEM is appropriate. The inability to move past this grief in a reasonable period of time such that it interferes with relationships and functional living needs to be addressed and treated. Many fear that depression reflects on oneself as an inadequate ability to cope with their diagnosis and feel weak. But it is not a personal strength issue, and depression is very much a physiological manifestation and treatable. Both talking to a psychiatrist/psychologist and medication management can be beneficial, and some studies indicate a synergistic effect of combining the two. Depression can rebound and can at times become more resistant to treatment.

Fatigue

Fatigue is the lack of mental and/or physical energy. Fatigue can be a direct result of a disease process (primary fatigue) or an indirect result (secondary fatigue). In ADEM, fatigue is more often thought to be a result of secondary fatigue. Examples of secondary fatigue include fatigue from medications, depression, stress, poor sleep patterns, infections, or changes in walking, which increase energy requirements. The key is to try to identify the underlying cause of the fatigue – for example, if one is not sleeping well because of pain, bladder dysfunction, or depression, this needs to be identified and addressed; not getting consistent sleep will worsen every other aspect of ADEM! If too much energy is exerted due to changes in walking, physical therapy can help identify better body mechanics that will help conserve energy. When nothing else can be identified as contributing to fatigue, REST is recommended! Conserving energy such that activities are planned and paced can allow for these activities to be more enjoyable rather than stressful. Also, reorganizing home and office can help to reduce the amount of wasted energy exerted so that energy can be saved up for activities that are enjoyable. Also, exercise routines incorporated in the day can actually help build stamina and reduce fatigue in the long-run – it's also

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a great stress reducer! Pilates, yoga, and swimming are great, but the key is to find something enjoyable and not overdo it.

Rehabilitation and Activities of Daily Living

An appropriate strengthening program and an aerobic conditioning regimen are recommended. The effects on mobility as a result of ADEM can vary widely, however, from paralysis to mild weakness. Either way, physical therapy is instrumental in returning function. Because physical therapists deal with many different types of injuries and diseases, it is ideal to work with one who has a particular interest in spinal cord rehabilitation when possible. Assistive devices may be necessary for weakness – it can be difficult and oftentimes humbling to take the necessary step of using an assistive device, but when faced with the alternative of broken hips, heads, and the downstream effects of lost wages or jobs, it is an important and sometimes indispensable step in maintaining independence. It is also always very important to remember to exercise, as tolerated, in order to maintain physical health and stamina.

Individuals with ADEM may find ordinary tasks such as dressing, bathing, grooming, and eating very difficult. Many of these obstacles can be mastered with training and specialized equipment. For example, long handled sponges can make bathing easier, as can grab bars, portable bath seats, and hand-held shower heads. For dressing, elastic shoelaces can eliminate the need to tie shoes, while other devices can aid in donning socks. Occupational therapists are specialists in assessing equipment needs and helping people with limited function perform activities of daily living. A home assessment by an experienced professional is often helpful.

Physical therapists assist with mobility. Besides teaching people to walk and transfer more easily, they can recommend mobility aids. This includes everything from canes (single point vs. small quad cane vs. large quad cane) to walkers (static vs. rolling vs. rollator) and braces. For a custom-fabricated orthotic (brace), an orthotist is necessary. Careful thought should go into deciding whether the brace should be an ankle-foot orthosis, whether it should be flexible or stiff, and what angle the foot portion should be in relationship to the calf portion. Some will benefit by a knee-ankle foot orthosis. Each person should be evaluated individually. The best results occur when a physician coordinates the team so that the therapists and orthotists are united on what is to be achieved. The physician best trained to take this role is the physiatrist.

Additional Resources

Myelitis Helpline
srna.ngo/helpline

For questions about our organization and rare neuroimmune disorders, visit the Myelitis Helpline, an online tool developed by SRNA.

Resource Library
srna.ngo/resources

To access up-to-date resources on rare neuroimmune disorders, which include symposium videos, magazines, podcast recordings, published research summaries, information sheets and relevant external resources, visit our Resource Library.

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