Rare Immune Disorder of Unknown Origin

Metachromatic Leucodystrophy

Background
Metachromatic leukodystrophy (MLD) is part of a larger group of lysosomal storage diseases, some of which are progressive, inherited, and neurodegenerative disorders (MLD included). Four types of MLD occur with varying ages of onset and courses (i.e. late infantile, early juvenile, late juvenile, adult). All forms of the disease involve a progressive deterioration of motor and neurocognitive function. The typing is somewhat arbitrary because the types overlap and some cases do not fall neatly within a single type. MLD actually describes a continuum of clinical severity. As the term implies, the presence of white matter abnormalities on brain images is characteristic.

Pathophysiology
In patients, the inability to degrade sulfated glycolipids, especially the galactosyl-3-sulfate ceramides, characterizes MLD. A deficiency in the lysosomal enzyme sulfatide sulfatase (arylsulfatase A) is present in MLD. Some patients with clinical MLD have normal arylsulfatase A activity but lack an activator protein that is involved in sulfatide degradation. Both defects result in the accumulation of sulfatide compounds in neural and in nonneural tissue, such as the kidneys and gallbladder. These defects may result from a number of different mutations, and many new causative mutations have been identified.
Histologic examination of the tissues often reveals metachromatic granules. Central and peripheral myelination are abnormal, with a widespread loss of myelinated oligodendroglia in the CNS and segmental demyelination of peripheral nerves. The sulfatide accumulations produce extensive damage and result in loss of both cognitive and motor functions.

Mortality/Morbidity
Morbidity and mortality rates vary with each form of the disease. In general, young patients have the most rapidly progressive disease, whereas patients with adult onset experience a more chronic and insidious progression of disease.
Patients with the late infantile form are usually aged 4 years or younger and typically present initially with gait disturbances, loss of motor developmental milestones, optic atrophy, and diminished deep tendon reflexes. In addition, progressive loss of both motor and cognitive functions is fairly rapid, and death results within approximately 5 years after the onset of clinical symptoms

Treatment
Currently, no effective treatment is available to reverse the deterioration and loss of function that metachromatic leukodystrophy (MLD) causes. In individuals with asymptomatic late infantile and early juvenile forms of the disease, bone marrow or cord blood transplantation may stabilize neurocognitive function; however, symptoms of motor function loss frequently progress. Mildly symptomatic and asymptomatic late juvenile and adult-onset forms are more likely to be stabilized with bone marrow transplantation because of slower progression.

Case Study

The parents brought their son aged 5,5 to our center to examine his health condition. For over 3,5 years of progressive disability he was becoming progressively weaker. He could not move his legs and arms.

Clinical data (September 2009): History of disease – Metachromatic Leukodystrophy
MEDICAL IMAGING REPORT
MRI Brain
HISTORY: Known case of white matter disease, R/O metachromatic leukodystrophy.
The boy developed right arm weakness.
TECHNIQUE: sagittal, coronal, axial;
T1wi,T2wi,FLAIR,GEwi, Gd-T1wi
The study reveals diffuse decreased signal on T1wi and increased signal on T2wi of pariventricular and deep white matter of both cerebrum. The lesion shows central low- and peripheral high-SI on FLAIR.

Stripped like pattern or tigoid appearance is noted. No enhancement is demonstrated.
There is a few area of subcortical white matter involvement especially at frontal lobe.
Involvement of internal splenium of corpus callosum is suspected.
No involvement of internal capsule is seen.
There is normal signal intensity of cerebellum and brain stem.
The ventricular system and cortical sulci are not dilated.
The visualized paranasal sinus is clear.
IMPRESSION: No change of white matter disease.
Metachromatic leukodystrophy is possible.

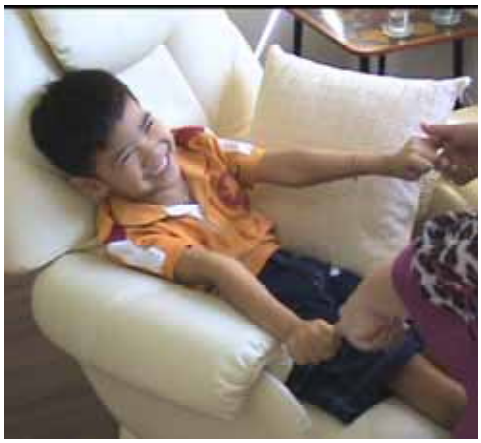
MRI T-L SPINE
History: Known case of white matter disease with right arm weakness.
Technique: axial. sagittal
T1wi,T2wi,Gd-T1wi
Findings: The study reveals normal signal of the entire spinal cord.
No evidence of abnormal enhancement is noted. The vertebral spine is also normal.
No evidence of spinal compression is noted. Impression: No detectable abnormality.
Doctor's suggestion: the illness is caused by genetic disorder. There is no cure available.

Pic.19. Before Ψ-TC correction (2.02.2010)

Discussions
We observed the boy's conditions (02.02.2010):

- Left side is paralyzed
- Legs are paralyzed
- The boy developed right arm weakness and cannot hold any object in his hands, cannot control his fingers
- Arms and legs are cold
- The boy has a weak voice (whisper-like),
- Chronic throat infection
- Night sleep disorders
- Psychoses and fear to stay away from his mother even for a short time like 1-2 minutes.

No conventional treatment is available for the boy.



Pic.20. Taken on 05.02.2010 after 3 sessions of Ψ-TC

Following health Ψ-TI assessment we proposed to try 3 sessions of Ψ-TT correction.

On day 3 we have observed the following:
The boy begins to move his hands, can hold objects in both hands, speaks with stronger voice, and sleeps for longer periods at night.

"Surrender to me"- the boy said holding the doctor's fingers.
"I know, that I do not have enough power, but I want to see that you are being defeated by me! Please!" - the boy said.

Pic.21. The boy can sit up after 10 Ψ-TC sessions (18.03.2010)

Discussions 2:

Since the case looks outstanding we decided to learn more about the origin of this immune disorder and set an additional investigation of the biometrical data produced by Ψ-TT system. We spent time to collect and analyze most of the tissue samples available in the Ψ-TT database.

1. The biometrical data captured by Ψ-TI scan have shown the highest level of invasion of Mononucleosis, compromising the Lymphocytes and Eosinophils, as well as traces of Epstein Barr virus, cytomegalovirus and herpes. Following the biometrical capture we propose the model that it was possible that the boy was infected with Epstein-Barr virus, frequently referred to as EBV, which is a member of the herpes virus family and one of the most common human viruses. It later caused infectious mononucleosis, which happens 35% to 50% of the time. Thus we made a hypothesis that the boy might have gotten an infection within a period between 1,5 and 2 year old, before he developed his weaknesses. This group of viruses might be consuming the enzymes responsible for the organic reactions processing sulfur in the nerve tissue. This event may promote sulfatide accumulations causing the loss of myelinated oligodendroglia. Such defects result in the accumulation of sulfatide compounds in neural and in nonneural tissue, such as the adrenal system, kidneys and gallbladder. These defects may further result in a number of different mutations causing the loss of signals in the nerve tissue.

1.A. In order to test our hypothesis we asked the parents to try to answer a few questions:

1) How their son was born (naturally or by Caesarean section)?
2) How he felt after he was born?
3) How had he been developing until the age of one?
4) Was their son able to walk before 1,5- 2 years old?

5) Had he been sick with some high fever before he was 2 years old?

The mother answered these question as follows:
1) Her son was born by Caesarean section.
2) She remembers nothing special.
3) Her son developed normal during this time.
4) Her son could walk and run as a normal child before 2 years old.
5) The mother recalled that it was true and her son really had got a high fever before 2 years old. Thus they performed a blood test at the hospital. The doctor informed them that there was some infection in the blood of their son, but he did not specify, what kind.

Few months after this episode their son's motor and other general functions started to weaken. First, the boy felt weak to run, then to walk. Later on he could only sit up, the weakness in his legs progressed. Then he lost control over his trunk. Recently his arms developed weakness: first left then right.

On his first visit to our center we observed that he experienced difficulty controlling the muscles of the neck and head's motion as well.

Conclusions:
Ψ-TI diagnostics helped us establish the origin of a complicated immune disorder.
Ψ-TC correction method (10 sessions in 30 days) demonstrated the efficacy of Ψ-TC correction to optimize the cellular metabolism in case of Metachromatic Leukodystrophy and to restore the pathways for the neural signal transmission.

Currently the boy continues the Ψ-TC. His condition is improving and he is slowly regaining control over the trunk. His parents were advised to help him with physical exercises, swimming and massage to restore the muscle tonus of the trunk.

His parents have reported about an interesting phenomenon - their son started to read two languages (Thai and English) even though nobody was teaching him to do so. Also he instantly replies (in Thai) and comments, when he hears discussions about his health in English, though he never met foreigners before, because he mainly stays at his home due his progressive disability. His parents do not know English as well.

We think this case is unique and may be beneficial to other patients with little hope for health improvements.