Myasthenia Gravis

Myasthenia gravis (MG) is the most common primary disorder of neuromuscular transmission. The usual cause is an acquired immunological abnormality, but some cases result from genetic abnormalities at the neuromuscular junction. Much has been learned about the pathophysiology and immunopathology of myasthenia gravis during the past 20 years. What was once a relatively obscure condition of interest primarily to neurologists is now the best characterized and understood autoimmune disease. Patients with myasthenia gravis come to the physician complaining of specific muscle weakness and not of generalized fatigue. Ocular motor disturbances, ptosis or diplopia, are the initial symptom of myasthenia gravis in two-thirds of patients; almost all had both symptoms within 2 years. Oropharyngeal muscle weakness, difficulty chewing, swallowing, or talking, is the initial symptom in one-sixth of patients, and limb weakness in only 10%. Initial weakness is rarely limited to single muscle groups such as neck or finger extensors or hip flexors. The severity of weakness fluctuates during the day, usually being least severe in the morning and worse as the day progresses, especially after prolonged use of affected muscles.

Muscular Dystrophy

Muscular dystrophy is a group of inherited diseases that are characterized by weakness and wasting away of muscle tissue, with or without the breakdown of nerve tissue. There are nine types of muscular dystrophy, but in each type there is an eventual loss of strength, increasing disability, and possible deformity.

The most well known of the muscular dystrophies is Duchenne muscular dystrophy (DMD), followed by Becker muscular dystrophy (BMD). They cause similar patterns of weakness and disability and are inherited in the same way, although weakness and disability are more severe in DMD. Becker dystrophy is often classified as a less severe form of Duchenne dystrophy. They both are due to defects of the same gene, the normal function of which is to enable muscle fibers to make a particular chemical substance, a protein called dystrophin. Muscle fibers in people affected with DMD are extremely deficient in dystrophin, but in BMD the deficiency is less severe.

Listed below are the nine different types of muscular dystrophy. Each type differs in the muscles affected, the age of onset, and its rate of progress.

Duchenne: age at onset: two to six years; symptoms include general muscle weakness and wasting; affects pelvis, upper arms, and upper legs; eventually involves all voluntary muscles; survival beyond age 20 is rare.

Becker: age at onset: two to 16 years; symptoms are almost identical to Duchenne but less severe; progresses more slowly than Duchenne: survival into middle age.

Congenital: age at onset: birth; symptoms include general muscle weakness and possible joint deformities; disease progresses slowly; shortened life span.

Distal: age at onset: 40 to 60 years; symptoms include weakness and wasting of muscles of the hands, forearms, and lower legs; progress is slow; rarely leads to total incapacity.

Emery-Dreifuss: age at onset: child-
hood to early teens; symptoms include weakness and wasting of shoulder, upper arm, and shin muscles; joint deformities are common; progress is slow; sudden death may occur from cardiac problems.

Facioscapulohumeral: age at onset: teens to early adults; symptoms include facial muscle weakness and weakness with some wasting of shoulders and upper arms; progress is slow, with periods of rapid deterioration; life span may be many decades after onset.

Limb-Girdle: age at onset: late childhood to middle age; symptoms include weakness and wasting, affecting shoulder girdle and pelvic girdle first; progress is slow; death is usually due to cardiopulmonary complications.

Myotonic: age at onset: 20 to 40 years; symptoms include weakness of all muscle groups accompanied by delayed relaxation of muscles after contraction; affects face, feet, hands, and neck first; progress is slow, sometimes spanning 50 to 60 years.

Oculopharyngeal: age at onset: 40 to 70 years; symptoms affect muscles of eyelids and throat causing weakening of throat muscles, which in time causes inability to swallow and emaciation from lack of food; progress is slow.

Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS) is a disease of the parts of the nervous system that control voluntary muscle movement. The word amyotrophic means “without muscle nourishment,” and refers to the loss of signals the nerves normally send to the muscles. Lateral means “to the side,” and refers to the location of the damage in the spinal cord. Sclerosis means “hardened,” and refers to the hardened nature of the spinal cord in advanced ALS. In the United States, ALS is also called Lou Gehrig’s disease, after the Yankees baseball player who died from it in 1941. In Britain and elsewhere in the world, ALS is often called motor neuron disease, in reference to the cells that are lost in this disorder.

Method

Using a powerful tool called MOgS (Molecular Organ and Gland Sequence), it is possible to detect with remarkable accuracy, the locations and the severity of these ailments. Therefore, it is possible to determine the root case of the disease even while it is still in the pre-diagnosable stage.

During the last nine years, over 5,000 cases of various muscle and joint disorders have been treated in my practice. Over 1,000 of those cases were either experiencing symptoms of Muscular Sclerosis or Muscular Dystrophy or Myasthenia Gravis without being diagnosed as such. However some people of those 1,000 were diagnosed with the above diseases from various neurological departments in hospitals, mainly from Cyprus but also throughout the world.

The MOgS Method uses Urine and Saliva samples taken from each patient to measure the precise functioning of the liver, protein absorption and distribution throughout the body, cell salt deficiencies, electrolyte imbalance, water distribution within the muscles and joints, deficiencies pertaining to muscle contraction directly and indirectly related to the nervous system. Immunity interference factors and metabolism efficiency as well as body chemistry were analyzed carefully to determine organ and gland sequence function. This unique tool has the ability to predict not only the speed of degeneration but also the speed of recovery.

The most important factors that are measured in MOgS are as follows

Sugar levels: Sugars indicate the level of energy the body is obtaining from the food

Saliva and Urine pH: The saliva pH is more significant that the urine pH but it is necessary to look at them separately because there are an infinite number of arrangements and combinations that represent different profiles. It is the balance between the acid and alkaline media of the body. When the urine and saliva pH are naturally held in correct bal-
ance, with both reading 6.4, it means that the secretions of the digestive system are operating at peak efficiency. The patient is obtaining maximum nutrition from the foods eaten. When the pH balance is altered and one or both of the pH readings moves away from 6.4, it means that the digestive secretions are out of balance. The enzymes become less efficient, which means that the nutritional processes in the body are becoming more inefficient. The farther away from perfect the balance of the two pHs travels, the more serious health problems that may develop because of improper assimilation of important nutrients. A combination of these two pH levels give us the body pH, a number indicative of the internal atmosphere of the body.

Salt levels: It is the measurement of every kind of salt, including fats and oils. We are interested in salt level because salts, organic as well as inorganic, cause the water in the body to be an electrolyte and to conduct the minute electrical charges. Too much salt magnetizes or over-ionizes the nerves of the stomach and may be the primary cause of ulcers. Salt also over-ionizes the food, causing it to cling to the walls of the colon. The patient can end up having too many mineral salts in his/her system by eating too much protein. The body converts excess protein into salts in order to eliminate them from the system. Often these excess salts are stored in the connective and fibrous tissue. Excess mineral salts cause this connective tissue to dehydrate and become rigid and stiff. The patient complains of stiff joints, strained tendons, torn cartilage and other irritating symptoms.

Nitrogen levels: There are two types of nitrogen measured: nitrate nitrogen and ammonia nitrogen. Both are by-products of protein metabolism, although the ammonia nitrogen is more toxic than the nitrate nitrogen. A high nitrogen level in MOgS indicates a buildup of toxic material in the tissues of the body. This is an indication of the amount of stress the person is experiencing due to this excess undigested protein by-product accumulation. It is likely that diseases begin with a vital organ that is central-nervous system controlled. The liver is the most likely organ to be involved because of its detoxification role. The more toxic it becomes, the less efficient it is in producing its many enzymes and amino acids. They also represent the level of undigested, trapped proteins in the body. Many diseases are caused by the failure of the body to digest proteins by an insufficient quantity of pancreatic enzymes. A reduced output of the pancreas, in most cases, is caused by a sluggish liver. Anionic substances taken regularly in the diet normally cause the liver to produce its normal output.

Cell Debris Levels: The cell debris count is the number of minute particles in the urine. The cells in the body are constantly dying and being replaced. When they die a large number of the cells are removed from the body through urine. For a person in perfect health, this should not exceed 40,000 parts of cell debris per liter of urine per 45 kg of body weight. However, if we are aging too fast, our cells are breaking down prematurely and the cell debris found in the urine will increase.

When your patient’s metabolism is in balance, their cells won’t wear out as rapidly and there will be less tissue and cell debris in the urine. By calculating the amount of cell debris, it is possible to see how fast your patient’s immune system is breaking down. If the body pH and sugars balances are away from normal, then a high cell debris is expected. If it does not appear, then that could represent a failure of the nervous system to properly command the kidneys, and as a result, the repair mechanism of the body would be malfunctioning.
Case Histories

In 2002, a 14-year old boy of Greek Cypriot origin came to my office accompanied by his parents. He had been diagnosed with muscular dystrophy when he was 5 years old, and had been on cortisone since then. His father had to hold him up since his feet dragged along the floor and was unable to walk without assistance. I performed the MOgS method to determine the root cause of the symptoms and disorder of the patient, and had seen the patterns that were necessary to correct. Orthomolecular therapy was given accordingly, and within 3 months time, this 14 year old boy was now able to play soccer and was able to continue his life doing things a boy his age could do. Till today, he does not experience any relapses.

In 2000, a 35 year old ex-athlete of Greek Cypriot origin, who later had a sedentary job, came to my office. He was diagnosed with muscular sclerosis. I performed the MOgS method, once again determining the root cause of his symptoms, and proceeded with orthomolecular therapy. Within six months, all his symptoms disappeared. Till today, he not only leads a perfectly normal life without symptoms, he has returned to his sport on a non-professional level.

In 2001, a 57 year old woman of Greek Cypriot origin came to my office diagnosed with myasthenia gravis. Her most prominent symptom were heavy eyelids. After performing the MOgS method, orthomolecular therapy was carried through over a three month period with great success. To this day, she has no symptoms of myasthenia gravis.

Discussion

Protomyonecrosis is a Greek term for “prior to muscle damage.” Muscular Diseases are not only psychologically terrifying to a patient but often paralyzing.

Having lived the last 9 years in Cyprus where the climate is hot most of the year, I have observed that many severe electrolyte and tissue salt deficiencies are partially implicated in muscular disorders. The climate where my practice is located makes it easier to notice the patterns in the extremes.

All cases had the same pattern in which muscular and central nervous system were both malfunctioning in the way of leading to serous muscular disorders.

All cases also had a severe protein malabsorption as well as distribution within the body, electrolyte levels were nearly non existent, they were so low that electricity could not generate enough energy to stimulate a proper muscle contraction. Simultaneously there was a great amount of dehydration not only within the muscles but also through the entire body, disabling other mobility mechanisms needed for a healthy neuro-muscular system.

The absence of cell salts made it difficult if not impossible for the cell to carry its message across to the fine areas needed to protect the body from muscle deterioration. I raise the argument that Proto-myonecrosis is the common denominator and starting point of all the above diseases mentioned.

Conclusion

Protomyonecrosis is a specific partial malfunction of the system which if left unattended, in time will cause a general malfunction of the system and specific damage or multiple damages to the muscles. It is the stage prior to any muscular paralysis in any area of the body. It’s the onset of muscle deterioration due to S.P.A.C.E factors (Severe Protein And Cell Salt Electrolyte factors). Muscular Diseases can not only be prevented but also treated with great success at an early enough stage. Focusing on the causes of the disorder from an orthomolecular point of view is the answer.

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The 2003 Conference featured two days of presentations from 30 speakers on Brain Function and Neurodegenerative Disease, Aging, Cancer, Eye Disease, and Diabetes. The international audience was much impressed by the quality and relevance of many of the lectures. On the third day, Walter Willett, M.D., winner of the 2003 Linus Pauling Institute Prize for Health Research, opened the standing-room-only public session with his talk, “The Pursuit of Optimal Diets.” The importance of advancing orthomolecular practice was closer to the forefront in this year’s conference, keeping Linus Pauling’s mission in focus.