

A-I, apolipoprotein A-II, fibrinogen, lysozyme, and gelsolin.

Children of affected patients have a 50% chance of inheriting the abnormal gene. DNA testing is recommended when the child becomes an adult.

DIAGNOSIS

The diagnosis of amyloidosis is made by a tissue biopsy that is treated with a special stain, Congo, red and examined microscopically. There are other non invasive tests available such as the Serum FLC Assay Test. Further testing is required to determine the amyloid type. The physician will first determine if the amyloid is of the most common AL type by checking for an abnormality of plasma cells in the bone marrow or light chains in the blood and urine. To determine other types more sophisticated pathology or genetic testing is required.

TREATMENT OPTIONS

Treatment depends on which type of amyloid protein is present in the body, where it is being deposited, and how it is affecting organ(s). It may range from pills, to injected chemotherapy, to stem cell transplants. Clinical trials are available to patients for some types. On rare occasions, organ transplants are performed.

AMYLOIDOSIS SUPPORT GROUPS INC.

501 (C) 3 Non Profit Corporation

This organization has been formed for the purpose of starting Amyloidosis Support Groups where needed,

- to have all Amyloidosis Support Groups be self sustaining through donations (general, memorial, note cards, and others)
- to fund and maintain libraries of educational materials for all support groups
- to fund group projects that will help in spreading awareness and fundraising
- to help finance the annual educational retreat for training new and current group leaders and facilitators.

Muriel Finkel, President

Jennifer L. Celano, Vice President

Elinda Lado, Secretary

Stephen D. Finkel, Treasurer

Sue Anne Smith, Director of Awareness

Maurice Price Jr., Director of

Fundraising

La Donna Stockstill, Honorary Co Founder

Amyloidosis Support Groups

ASG www.amyloidosisupport.com

***For Information on Note Cards, polo shirts, pins, wristbands and other awareness items that help raise funds for the support groups and their endeavors while spreading awareness, please check web-site* or call toll free USA 866-404-7539 630-350-7539**

AMYLOIDOSIS

**Amyloidosis
(pronounced am-i-loid'-osis)
is an organ system disease
caused by deposits of
amyloid protein fibrils in
one or more organs
causing the organ to
malfunction.**

**The heart, kidneys, nervous
system and gastrointestinal tract
are most often affected.**



The support we provide includes information resources from the medical community involved in the research and treatment of Amyloidosis.

Support group meetings have been held in California, Texas, Arizona, Florida, Colorado, Illinois, Massachusetts, Pennsylvania, Michigan, Minnesota, Nevada, Oregon, New York, Maryland, and others.

Our website has extensive information and links to other websites providing further information:

WHAT IS AMYLOIDOSIS?

The amyloidoses are a group of diseases that result from a protein folding abnormality that causes deposits of amyloid fibrils in organs throughout the body and leads to organ dysfunction.

There are three major systemic types, each caused by a different misfolded protein. All are very different. The most common is AL, A for amyloid and L for the light chain that is the abnormal part of an antibody made in the bone marrow in patients with this disease. A second type is AA, in which the amyloid protein comes from serum amyloid A, a protein that is elevated in inflammatory disease. Hereditary forms comprise the last category and are characterized by deposits of a variety of different misfolded proteins.

Amyloidosis was first described in the 19th century, but only in recent years have significant advances been made in identification of types and their diagnosis and treatment.

PRIMARY AMYLOIDOSIS (AL)

An abnormal antibody light chain produced in the bone marrow by a plasma cell is the cause of primary amyloidosis. Amyloidosis of this type can occur occasionally with multiple myeloma, also a disease of plasma cells.

Amyloid deposits are the light chain protein and often occur in multiple organs in a short period of time. Without treatment this disorder can be serious and sometimes fatal. The survival rates can vary for each patient.

The organs most often involved in AL amyloidosis include the heart, kidneys, nervous system, and gastrointestinal tract. Symptoms may include shortness of breath, fatigue, edema (swelling of ankles and legs), dizziness upon standing, a feeling of fullness in the stomach, diarrhea, weight loss, enlarged tongue, numbness of the legs and arms, and protein in the urine (often marked by frothing or bubbles).

AL amyloidosis is the most common type and is usually treated with chemotherapy and/or SCT to kill the abnormal plasma cells, or to slow production of the abnormal light chain. Trials of the use of different classes of drugs for treatment are underway.

SECONDARY AMYLOIDOSIS (AA)

Secondary amyloidosis is caused by a long-term serious chronic infection such as TB or osteomyelitis, or inflammatory disease such as rheumatoid arthritis or familial Mediterranean fever. The amyloid deposits in this type of amyloidosis are made up of AA protein, a protein that is markedly elevated in the body during

inflammation.

The disease progression is usually slow, and treatment of the underlying chronic infection or inflammatory disease can slow down or stop the progression of further amyloid deposits. Survival is measured in years.

In secondary (AA) amyloidosis the organ most often involved is the kidneys, but GI tract, liver, and heart can be involved as the disease progresses.

Symptoms include protein in the urine, edema, weight loss and fatigue.

HEREDITARY OR FAMILIAL AMYLOIDOSIS

Inherited mutations of several different genes lead to familial amyloidosis. The most common is an inherited transthyretin (TTR) mutation. This form is called Amyloid Transthyretin (ATTR). There are 100 different mutations of TTR that can cause amyloidosis. Some mutations can occur in many ethnic groups, while others are, thus far, very rare, occurring in only one or two families. Although the abnormal protein is present from birth, disease usually begins in mid-life. Symptoms are a little different for each mutation, and include neuropathy, gastrointestinal dysfunction, cardiomyopathy, vitreous opacities, and renal disease.

Other proteins that may have mutations leading to amyloidosis are apolipoprotein