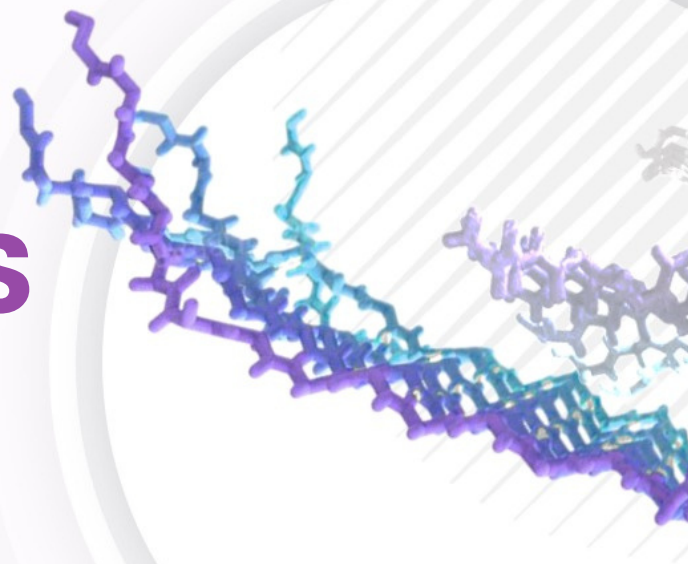
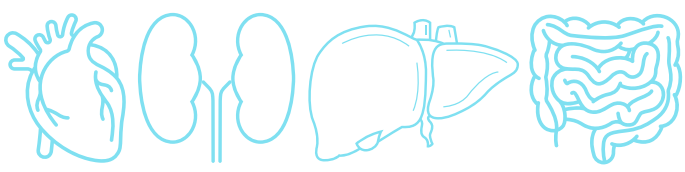
SYSTEMIC AMYLOIDOSIS

Everything You Need to Know



17
Unique Types

Systemic amyloidosis is a group of progressive and debilitating rare diseases, in which toxic amyloid, a protein that has misfolded, builds up in the body in and around organs, stopping them from functioning properly. Amyloidosis results in organ failure, high mortality rates, and high healthcare costs.



4 Most Common Types

There are more than 30 precursor proteins that cause amyloidosis, and 17 cause systemic disease.[1] The four most common types of systemic amyloidosis are:

Wild-type Transthyretin Amyloidosis (wtATTR)
Mainly manifests in the heart and in ligaments and tendons

Hereditary Transthyretin Amyloidosis (hATTR)
Manifests in the heart, nerves, GI and kidneys

Light Chain Amyloidosis (AL or primary)
Manifests in almost every organ, but >70% of patients have cardiac and/or kidney involvement [2]

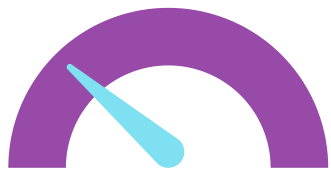
Leukocyte Chemotactic Factor 2 Amyloidosis (ALECT2)
Manifests mainly in the kidneys and the liver

Early Diagnosis is Critical

Amyloidosis leads to poor and declining quality of life, particularly in later stage patients. If left unchecked, toxic amyloid will continue to build up in and around your organs, resulting in organ failure, and is often fatal.[4]

There is no approved amyloid-specific diagnosis agent. Patients undergo numerous diagnostic tests and it can take months, and often years, for a patient to be diagnosed. [5]

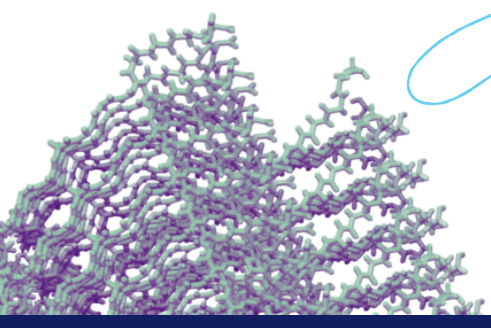
>80%
of patients never receive a diagnosis [3]



25%
of patients wait > 5 years for a diagnosis [4]

Current Treatment Options

Therapies currently used for the treatment of amyloidosis target precursor protein production, reducing the formation of new amyloid. These therapies may slow the progression of the disease but do not reduce or remove existing amyloid from the body.[6]



25%
of hATTR patients are hospitalized within 1 year of diagnosis [7]

3-5
years survival, on average, due to delays in diagnosis [8]

References: 1. Benson MD, Buxbaum JN, Eisenberg DS, et al. Amyloid. 2018;25(4):215-219. **2.** Sanchorawala V, McCausland KL, White MK, et al. Br J Haematol. 2017;179(3):461-470. **3.** Lousadal, Comenzo RI, Landau H, et al. Adv Ther. 2015;32:920-928. **4.** Hester LL, Gifkins DM, Bellew K, et al. Eur J Haematol. 2021;107(4):428-435. **5.** Baker KR, Rice L. Methodist Debakey Cardiovasc J. 2012;8(3):3-7. doi:10.14797/mdcj-8-3-3. 7. **6.** Richards DB, Cookson LM, Barton SV, et al. Sci TranslMed. 2018;10(422):eaan3128. **7.** Reddy SR, Chang E, Tarbox MH, et al. Neurol Ther.2020;9(2):473-482. **8.** Barrett CD, Dobos K, Liedtke M, et al. JACC Heart Fail. 2019;7(11):958-966.

attralus

March 2022