Cardiac Amyloidosis: Evolving Diagnosis and Management

A Scientific Statement From the American Heart Association

Cardiac amyloidosis results in a restrictive cardiomyopathy caused by extra-cellular deposition of proteins in the myocardium. The proteins have an unstable structure that causes them to misfold, aggregate, and deposit as amyloid fibrils. More than 30 proteins can form amyloid fibrils in vivo, and the clas sification is based on the precursor protein. Cardiac amyloidosis is caused mainly by misfolded monoclonal immunoglobulin light chains (ALs) from an abnormal clonal proliferation of plasma cells or transthyretin (TTR) amyloidosis (ATTR), a liver-synthesized protein previously called prealbumin that is normally involved in the transportation of the hormone thyroxine and retinol-binding protein. Given the paramount relevance of transthyretin amyloid cardiomyopathy (ATTR-CM) to the practicing cardiologist, this statement focuses on its diagnosis and management.

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