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Cardiac Amyloidosis is to Understanding the Silent Threat to the Heart

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INTRODUCTION

This progressive disease can lead to significant structural and functional abnormalities, ultimately impairing cardiac function. Despite its rarity, cardiac amyloidosis poses a serious threat to patient health, as it can mimic other more common cardiac disorders and is often diagnosed at an advanced stage. This article aims to provide an in-depth understanding of cardiac amyloidosis, including its pathophysiology, clinical presentation, diagnostic approaches, and available treatment options. AL amyloidosis, also known as primary amyloidosis, is associated with plasma cell dyscrasias such as multiple myeloma, while ATTR amyloidosis, previously referred to as senile systemic amyloidosis, is related to the misfolding of transthyretin protein. The deposition of amyloid fibrils leads to structural changes in the heart, causing thickening and stiffness of the cardiac walls, interfering with the normal filling and contraction of the heart chambers.

DESCRIPTION

Over time, this can result in heart failure, arrhythmias, and other cardiovascular complications. Furthermore, the infiltrative nature of amyloid deposits can also affect other organs, such as the kidneys, liver, and peripheral nerves. Cardiac amyloidosis presents with a wide range of symptoms, making diagnosis challenging. Patients may experience fatigue, shortness of breath, peripheral edema, palpitations, and chest pain. However, these symptoms are nonspecific and can be mistaken for other cardiac conditions, leading to delayed diagnosis. To establish a diagnosis, a multidisciplinary approach is crucial. A thorough medical history, physical examination, and appropriate laboratory tests can provide initial clues. Blood tests may reveal abnormal protein levels or evidence of organ dysfunction. Imaging techniques, such as echocardiography, cardiac Magnetic Resonance Imaging (MRI), and nuclear imaging, can help identify characteristic findings associated with cardiac amyloidosis, such as thickened ventricular walls, restrictive filling patterns, and myocardial uptake of amyloid tracers. Definitive diagnosis often requires histopathological examination of the affected tissue. Endomyocardial biopsy or biopsy of other involved organs may be performed to obtain tissue samples for Congo red staining and immunohistochemistry. However, due to the patchy nature of amyloid deposition, false-negative results are possible, necessitating careful interpretation of biopsy findings in the clinical context. The management of cardiac amyloidosis depends on the type and extent of amyloid deposition, as well as the presence of associated organ involvement. The primary goal of treatment is to slow the progression of the disease, relieve symptoms, and improve quality of life. For patients with AL amyloidosis, systemic therapy targeting the underlying plasma cell dyscrasia is the cornerstone of treatment. Chemotherapy regimens, including proteasome inhibitors, immunomodulatory agents, and monoclonal antibodies, are used to reduce the production of abnormal light chains.

CONCLUSION

Hematopoietic stem cell transplantation may be considered for eligible patients. In addition to systemic therapy, supportive measures such as diuretics, angiotensin-converting enzyme inhibitors, and beta-blockers are employed to manage heart failure symptoms. In ATTR amyloidosis, treatment strategies differ based on the presence of mutant or wild-type transthyretin. For patients with hereditary ATTR amyloidosis, targeted therapies such as RNA interference agents and small molecule stabilizers are being developed to inhibit the production of abnormal transthyretin or prevent its misfolding. Liver transplantation, which replaces the mutant transthyretin-producing liver with a healthy one, can halt the progression of the disease in suitable candidates.

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