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Amyloidosis-Associated Heart Disease: An Echocardiogram's Score

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INTRODUCTION

The misfolding of a specific protein precursor is the most common cause of cardiac amyloidosis, depending on the type of amyloidosis. Examples of protein precursors include immunoglobulin-derived light chains and mutations in transthyretin. When the protein is misfolded, it forms amyloids, which are insoluble sheets of beta-pleated protein. The body is unable to break down the protein aggregation known as amyloid. The majority of amyloids are fibrils, with the P component, apolipoprotein, collagen, fibronectin, and laminin also present.

DESCRIPTION

The P component, a pentameric protein, slows the body's clearance of amyloid fibrils by stabilizing them. Amyloid deposits can form in the heart, liver, kidneys, spleen, adrenal glands, and bones, among other organs. Deposits in the heart's extracellular space may cause the ventricles to become constrained. The restricted ventricular motion impairs the heart's ability to pump effectively, which in turn causes the various signs and symptoms of cardiac amyloidosis. The safe and non-invasive method of echocardiography can be used to evaluate structural and functional heart disease. Amyloidosis is characterized by thickening of the ventricle and valvular structures, enlargement of the biatrium, and a restricted filling pattern. Systolic function is normal to slightly diminished, and diastolic filling is diminished. An echo can be used to evaluate the prognosis of the disease by measuring the various strains on the heart. The heart's function is affected in specific ways by amyloidosis. Echocardiography can identify this particular pattern, which is 90%-95% sensitive and 80%-85% specific for cardiac amyloidosis. Echocardiography can only be used to suggest the presence of the disease, not to confirm its diagnosis, unless the condition is late-stage amyloidosis. The gastrointestinal system can accumulate amyloid proteins from a wide range of amyloid disorders, which can manifest in a variety of ways depending on the degree of organ involvement. Possible symptoms include weight loss, diarrhoea, abdominal pain, heartburn, and GI bleeding. Amyloidosis can lead to spleen enlargement, jaundice, fatty stool, anorexia, and fluid build-up in the abdomen. The liver and other accessory digestive organs may also be affected. When amyloid proteins accumulate in the liver, one third of people experience elevations in serum aminotransferases and alkaline phosphatase, two biomarkers of liver injury. Liver enlargement is common. On the other hand, spleen enlargement only affects 5% of people. Howell-Jolly bodies can be seen on a blood smear in 24% of people with amyloidosis due to splenic dysfunction. Due to its ease of acquisition, the subcutaneous abdominal fat, also known as a "fat pad biopsy," is the first-line biopsy site. A tissue sample can be taken from the affected internal organ or biopsied. Because it is not 100% sensitive and may produce false negatives, the diagnosis of amyloidosis cannot be ruled out by a negative abdominal fat biopsy result.

CONCLUSION

However, a direct biopsy of the affected organ may not be necessary if other less invasive methods of biopsy, such as rectal mucosa, salivary gland, lip, or bone marrow biopsy, which can provide a diagnosis in up to 85% of cases, are sufficient. Due to the amyloid deposition in the joints, there will be fewer signals in T1 and T2 weighted MRI images. In amyloidoma, gadolinium injection will result in low T1 and T2 signals. There are several methods for identifying the type of amyloid protein: The detection of abnormal proteins in the blood; specific antibodies that bind to the amyloid in the tissue; or the protein is taken out, and each of its amino acids is found. Immunohistochemistry is able to identify AA amyloidosis most of the time, but it can miss many cases of AL amyloidosis.

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