

Hypertrophic Cardiomyopathies

1. How early do hypertrophic cardiomyopathies develop in the following patients? Idiopathic, diabetic, alcoholic—should they be screened? How?

Question submitted by: David Abriel, Mahone Bay, Nova Scotia

There appears to be a basic misunderstanding about the cardiomyopathies (or I have totally misunderstood the question). Hypertrophic cardiomyopathy is a genetic disorder characterized by hypertrophy of the left ventricle. A subset of this is those with asymmetric hypertrophy of the interventricular septum causing outflow tract obstruction (hypertrophic obstructive cardiomyopathies).

Hypertrophic cardiomyopathies are inherited as an autosomal dominant, but mutations in genes do occur, probably explaining the lack of family history in

many cases.

Dilated cardiomyopathies, the most common group, are characterized by global dilation and thinning of the left ventricular walls and often severe systolic dysfunction leading to heart failure. Of this group, idiopathic dilated cardiomyopathy is most common and in many cases may represent “burned out” myocarditis. Diabetic dilated cardiomyopathies probably result from diffuse small coronary vessel disease and multiple small or large infarctions. Alcoholic cardiomyopathy is again a dilated cardiomyopathy, either related directly to alcohol toxicity, or secondary metabolic disturbances.

Occasionally, hypertrophic cardiomyopathies may “burn out,” leading to a dilated poorly functioning ventricle. However, dilated cardiomyopathies never become hypertrophic.

Screening for the development of hypertrophic cardiomyopathies, primarily with ECHOs, is indicated in families with known hypertrophic cardiomyopathy, a history of familial sudden death, or with unusual hypertrophy detected by ECG.

Answered by:
Dr. Wayne Warnica

Monitoring INR Levels

2. How frequently should INR levels be monitored in patients who have had consistently stable readings while on long-term warfarin therapy?

Question submitted by: Donald J. Collins-Williams, Mississauga, Ontario

While the American College of Chest Physicians recommends individualized management because the optimal frequency of INR monitoring varies according to patient compliance, dosing decisions, duration of therapy and changes in health, diet, or medications, their latest guidelines suggest a monitoring interval of no longer than four weeks (Grade 2C, the weakest recommendations available). For highly compliant patients with stable INR levels and a clear understanding of factors that influence anticoagulation (changes in

health, diet, medications), routine monitoring has been shown to be safely extended to 14 weeks. However, more frequent INR testing allows for better fine-tuning of the dosage and increases the time patients spend in the therapeutic range (2.5-3.0), optimizing stroke prevention and minimizing bleeding complications. Therefore, once a month warfarin monitoring remains a sensible interval after the therapeutic level has been consistently achieved.

Maintaining a standard routine simplifies the many

instructions that physicians give and patients receive. Additionally, monitoring has the secondary benefit of reinforcing the risks associated with warfarin and provides a further opportunity for patient education.

Resources

1. Ansell J, Hirsh J, Hylek E, et al: Pharmacology and Management of the Vitamin K Antagonists: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest 2008; 133(6 Suppl):160S-198S.
2. Lidstone V, Janes S, Stross P: INR: Intervals Of Measurement Can Safely Extend To 14 Weeks. Clin Lab Haematol 2000; 22(5):291-3.

Answered by:

Dr. Theodore Fenske

Once a month warfarin monitoring remains a sensible interval after the therapeutic level is achieved.