

Which meds for WPW Syndrome?

1. Which medication is best and which should be avoided when treating patients with angina in the presence of Wolff-Parkinson-White (WPW) syndrome?

Question submitted by Dr. W. Sullivan, Halifax, Nova Scotia

Conventional treatments for chronic stable angina include:

- nitrates,
- beta-blockers,
- angiotensin converting enzyme (ACE) inhibitors,
- statins,
- calcium channel blockers,
- combination therapies (beta-blockers plus nitrates),
- acetylsalicylic acid with or without clopidogrel and
- revascularization by percutaneous coronary intervention or bypass graft surgery.

Q: Are any of these antianginal treatments relatively contraindicated in WPW syndrome?

There is no scientific data that indicates that conventional beta-blockers are proarrhythmic in the setting of WPW, but similarly, there is no scientific data that confirms that conventional beta-blockers slow the ventricular response to atrial fibrillation in WPW.

However, there is clear data that verapamil and diltiazem can accelerate ventricular response to atrial fibrillation. Accordingly, if a calcium channel antagonist is important for the management of angina, a dihydropyridine would be the indicated choice (i.e., amlodipine or felodipine). There is no evidence in the literature that an interaction exists between nitrates or ACE inhibitor use and WPW syndrome.

Theoretically, any drug-induced hypotension could increase sympathetic tone, thus potentially increase the possibility of arrhythmogenesis. Anginal syndromes can be complicated by congestive heart failure. Digitalis is a conventional treatment for heart failure.

However, if angina, heart failure and WPW syndrome coexist, digitalis is contraindicated in patients with manifest WPW (persistent delta wave on the ECG). Ablation of the accessory connection, rather

than pharmacologic treatment, is the most rational approach to these patients.

Q: Are there antiarrhythmic treatments conventionally used for WPW syndrome that are relatively contraindicated in the setting of angina?

Propafenone and flecainide can be prescribed to prevent recurrent reciprocating supra-ventricular tachycardias in the setting of WPW. However, these medications are contraindicated in the setting of ischemic heart disease, particularly in patients with reduced ejection fractions and myocardial scar. Ablation of the accessory connection, rather than pharmacologic treatment, is likely the most rational approach to these patients.

Answered by:

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Does CHD cause hypertension?

2. As most hypertension has no known cause, yet causes CHD, could it be that patients' CHD is a cause of hypertension and we have got it the wrong way around?

Question submitted by Dr. W. Doyles, Sarnia, Ontario

That is an interesting hypothesis.

It is well known that hypertension and coronary heart disease (CHD) coexist. About 90% of patients with CHD have at least one major risk factor, including hypertension.

The INTERHEART study has shown that 90% of the risk of the first myocardial infarction (MI) can be attributed to nine modifiable risk factors, including hypertension. Sixty-nine per cent of patients who had their first MI also had hypertension (BP > 140/90 mmHg).

Statistics derived from the 2006 AHA report on cardiovascular disease indicate that 15% of the population has hypertension, while the prevalence of CHD is only 6.9%.

Hypertension occurs earlier than CHD, with the average age of first MI at 65.8 years for men and 70.7 years for women.

Finally, from Framingham data, even people with "prehypertension" (BP 120-139/80-89 mmHg) have a 3.5 fold higher-risk of MI and a 1.7 fold higher-risk of CHD.

Therefore, this data would suggest that hypertension is a risk factor for CHD rather than the other way around.

References

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Studies show that hypertension is a risk factor for CHD rather than the other way around.

Small vessel coronary artery disease

3. I have a patient who has been diagnosed with small vessel coronary artery disease by a local cardiologist. How common is this and what are the implications/prognosis?

Question submitted by Dr. Katherine Allen, Belleville, Ontario

Anginal chest pain is usually caused by blockages in the larger coronary arteries. The diagnosis of small vessel coronary artery disease is often inferred in patients with chest pain that has features typical of angina, yet who have normal coronary arteries.

Occasionally, stress-induced myocardial ischemia can be observed in a patient that has a normal coronary arteriogram. Some of these patients have abnormal coronary flow reserve due to functional abnormalities in the small arteries. However, other cardiac causes should be considered in those with chest pain who present with normal coronary arteriograms. Other

cardiac causes include mitral valve prolapse or extra cardiac causes, such as esophageal disease.

The prognosis of patients with chest pain and a normal coronary angiogram is excellent. Often, with the knowledge that the coronary arteries are normal, the pain becomes both less frequent and less disabling.

Answered by:

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Occasionally, stress-induced myocardial ischemia can be observed in a patient that has a normal coronary arteriogram. Some of these patients have abnormal coronary flow reserve due to functional abnormalities in the small arteries.


Reducing C-reactive protein

4. If the high-sensitivity C-reactive protein is elevated in a high-risk cardiovascular patient, what should be done to reduce this marker of inflammation?

Question submitted by Dr. Paul Stephan, Scarborough, Ontario

In patients with stable coronary artery disease, high-sensitivity C-reactive protein (CRP) has been shown to be predictive of future coronary events and of poorer short- and long-term outcomes after non-ST elevation acute coronary syndrome and myocardial infarction.^{1,2} CRP is also considered a predictor of adverse events following percutaneous coronary intervention.³

Statin therapy represents a cornerstone in the treatment of cardiovascular patients and has been shown to significantly decrease CRP, independent of reducing LDL-cholesterol. Patients with LDL levels < 1.8 mmol/L and CRP levels < 1 mg/L—after statin therapy—had fewer recurrent cardiovascular events following acute coronary syndromes in the PROVE IT-TIMI 22 study.⁴ Decreased CRP, related to

statin therapy, was also associated with slower rates of disease progression by intravascular ultrasound in the REVERSAL study.⁵ Acetylsalicylic acid (ASA), beta-blockers and thiazolidinediones have also been shown to reduce CRP levels.⁶⁻⁸ The therapeutic response to elevated levels of high sensitivity CRP in high-risk cardiovascular patients is still not entirely understood. Conventional medications, including statins, beta-blockers and ASA should continue to be part of any secondary prevention strategy. 

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Further references available—
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