Supraventricular arrhythmias after myocardial infarction

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INTRODUCTION

Supraventricular arrhythmias, other than atrial fibrillation (AF) or flutter, are relatively uncommon in the periinfarction period. Their occurrence often indicates myocardial dysfunction and they may, by themselves, cause congestive heart failure or exacerbate ongoing myocardial ischemia.

The incidence, mechanism, and treatment of supraventricular arrhythmias (particularly sinus bradycardia, sinus tachycardia, and AF) occurring after myocardial infarction (MI) will be reviewed here. The focus of the discussion here is on supraventricular arrhythmias in the setting of MI caused by acute atherothrombotic coronary artery disease. However, there are some clinical settings in which supraventricular arrhythmia may cause MI (such as when AF causes left atrial thrombus that embolizes to a coronary artery, or in the setting of a supraventricular tachycardia causing demand ischemia and MI). (See "Diagnosis of acute myocardial infarction" and "Diagnosis of acute myocardial infarction", section on 'Definitions'.)

The following related topics are discussed separately: conduction disturbances after MI, ventricular arrhythmias during MI, and risk stratification of patients after MI. (See "Conduction abnormalities after myocardial infarction" and "Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features" and "Risk stratification after acute ST-elevation myocardial infarction".)

SINUS BRADYCARDIA

Sinus bradycardia, defined as less than 50 to 60 beats per minute (bpm), occurs in 15 to 25 percent of patients after acute MI [1-3]. It has the following clinical characteristics [1,2,4]:

- It is frequently seen with inferior wall infarctions, since the right coronary artery supplies the sinoatrial node (SA) in approximately 60 percent of people.
- It is most often transient, particularly for sinus bradycardia occurring within the first six hours; such arrhythmias typically resolve within 24 hours.
- It is usually caused by increased vagal tone, often seen with inferior MI. In some cases, this may be the result of diaphragmatic irritation. Other less common causes include ischemia of the SA node and as a reperfusion arrhythmia following fibrinolysis [5].
- It is due to medications (beta blockade, calcium channel blocker, or digoxin).

Treatment — If therapy is necessary due to hemodynamic compromise or ischemia, sinus bradycardia following an acute MI usually responds well to intravenous atropine (0.6 to 1.0 mg in the majority of cases) [6,7]. Persistent bradycardia with hemodynamic compromise despite intravenous atropine warrants consideration of temporary cardiac pacing [6,7]. Atrial or sequential atrioventricular (AV) pacing is superior to ventricular pacing, particularly if there is an associated right ventricular MI [8].

Permanent pacing is not typically necessary in patients with sinus bradycardia after an MI, because in most cases the bradyarrhythmia is transient [7]. Any decision to implant a permanent cardiac pacemaker should be delayed for several days of observation. (See "Permanent cardiac pacing: Overview of devices and indications".)

Mortality as a result of periinfarction sinus bradycardia is rare [1]. Its significance is related to the associated reduction in cardiac output and coronary perfusion, thereby exacerbating ischemia, and to the possible development of an escape rhythm with possible AV dissociation causing hemodynamic compromise [9].

SINUS TACHYCARDIA

Sinus tachycardia occurs in approximately 30 to 40 percent of acute MIs [10]. Although this is seen on presentation, the heart rate usually declines over time to a level that reflects the degree of activation of the sympathetic nervous system. Patients with persistent sinus tachycardia

usually have larger infarcts that are more often anterior [10] and a marked impairment in left ventricular (LV) function, which is associated with substantial morbidity, a high early mortality, and an increased 30-day mortality [10,11]. In addition, sinus tachycardia may increase the size of ischemic injury and infarction due at least in part to increased oxygen consumption. In some cases, sinus tachycardia may be the result of associated pericarditis.

Therapeutic efforts should be targeted at identification and management of the underlying cause. However, most patients will receive such therapy independent of the tachycardia since early beta blocker administration is part of routine management of acute MI. Care should be exercised in the utilization of beta blockers early in an infarction, especially in the presence of large anterior MIs, hypotension, or pulmonary congestion. (See "Acute myocardial infarction: Role of beta blocker therapy".)

ATRIAL FIBRILLATION

The following section will review the incidence, pathophysiology, clinical characteristics, and treatment of the atrial tachyarrhythmias that occur after acute MI. AF is the most common of these.

The overall incidence of atrial tachyarrhythmias in the periinfarction period ranges from 6 to 20 percent [9,10,12-17]. These arrhythmias primarily occur within the first 72 hours after infarction; however, only 3 percent are noted in the very early (less than three-hour) phase [12].

AF is common both during hospitalization and after discharge for acute MI and in both time periods has prognostic significance. The incidence during hospitalization has been reported to be between 5 and 18 percent and is more likely in individuals with heart failure, kidney disease, hypertension, diabetes, and pulmonary disease [13,14,18]. As expected with these risk factors, 30-day mortality in patients who develop AF is increased [13]. Long-term mortality is also increased in patients with in-hospital AF compared to those without [9,13,15,16,19-22].

Information concerning the longer-term incidence and prognosis of patients who develop AF after discharge comes from the CARISMA study, which evaluated the long-term development of arrhythmias in 271 patients with acute MI and an LV ejection fraction less than 40 percent [18]. These individuals underwent placement of an insertable cardiac monitor (ICM; also sometimes referred to as implantable cardiac monitor or implantable loop recorder) and were monitored for the development of AF lasting more than 16 beats. (See "Ambulatory ECG monitoring", section on 'Insertable cardiac monitor' and "Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features", section on 'Late arrhythmias'.)

The following findings were noted during two years of follow-up:

- Ninety-five individuals (39 percent) developed AF. Nearly half of these occurred by two months and nearly 80 percent by one year. Most of the AF events were asymptomatic.
- The duration of the AF event(s) was more than 30 seconds in slightly more than half of these.
- New onset AF was associated with a significantly increased risk of major cardiovascular events (Unadjusted hazard ratio [HR] 2.04). AF remained a significant predictor of adverse outcomes even after adjustment. The risk was significantly increased in those individuals whose duration of AF was more than 30 seconds (HR 2.73, 95% CI 1.25-5.50), but not in those with episodes lasting less than 30 seconds (HR 1.17, 95% CI 0.35-3.92).

Pathophysiology — The increase in the prevalence of AF during an acute MI has been ascribed to one or more of the following factors: atrial dysfunction (due to atrial ischemia, which is rare; infarction, which is rare; or atrial stretching due to heart failure with elevation in left atrial pressures, which is the most common etiology) [23], sympathetic stimulation, pericarditis [24], inflammatory state [25], atheromatous disease of the arteries supplying the sinoatrial (SA) and AV nodes and the left atrium (which is a rare cause for AF) [10,12,21,26], and iatrogenic factors such as positive inotropic agents.

Prevention — Statin therapy, possibly due to an antiinflammatory effect, has been associated with a reduction in AF recurrences in patients with lone AF, ischemic heart disease, and after cardiac bypass surgery. (See "Antiarrhythmic drugs to maintain sinus rhythm in patients with atrial fibrillation: Clinical trials", section on 'Statins'.)

In a retrospective study of over 3300 patients presenting with acute MI and in sinus rhythm, statin therapy (prescription within 48 hours of hospitalization) was associated with a reduced risk of AF (odds ratio 0.64, 95% CI 0.45-0.92) [27].

However, we do not recommend initiation of statin therapy in patients with MI to solely prevent AF. Almost all such patients should be on statin therapy for other reasons. (See "Low-density lipoprotein-cholesterol (LDL-C) lowering after an acute coronary syndrome", section on 'Summary and recommendations'.)

Treatment — The management of periinfarction atrial tachyarrhythmias is important, because tachycardia can increase myocardial oxygen demand, thereby exacerbating ischemia and possibly decreasing cardiac output. Atrial tachyarrhythmias may also induce or exacerbate heart failure, especially when associated with a rapid ventricular response. For sustained AF or

atrial flutter that is of new onset after the MI and is associated with hemodynamic compromise, we make the following recommendations:

- Initial treatment should consist of rate control. The first-line treatment is beta blockade. One option is intravenous metoprolol (2.5 to 5.0 mg every two to five minutes to a total of 15 mg over 10 to 15 minutes). If ineffective, intravenous calcium channel blockade (verapamil or diltiazem) should be considered.

- For unstable patients, synchronized direct current cardioversion should be considered. (See "Atrial fibrillation: Cardioversion" and "Atrial fibrillation: Cardioversion", section on 'Indications'.)

  For episodes of AF with hemodynamic compromise that do not respond to electrical cardioversion or that recur after a brief period of sinus rhythm, the use of intravenous followed by oral amiodarone to help control rate and help maintain sinus is indicated.

  The use of amiodarone or digoxin in the setting of hemodynamic compromise is recommended because these drugs are associated with less risk of worsening myocardial dysfunction than some other drugs (although some preparations of intravenous amiodarone can cause hypotension, which is the result of Tween 80, the detergent used to get amiodarone into aqueous solution) and have not been associated with increased risk of mortality post-infarction. However, in many patients hemodynamic compromise during AF is a result of the rapid ventricular rate. In such cases, amiodarone and digoxin, which slow AV conduction only gradually and after a long period of time, may not be as effective as other agents. We recommend the use of either an intravenous beta blocker or intravenous verapamil in this setting.

  Dofetilide is also an effective drug for long-term management of AF after MI to help maintain sinus rhythm. The DIAMOND-MI trial found that this drug had no effect on all-cause, cardiac, or total arrhythmic mortality compared to placebo [28]. However, dofetilide was better than placebo for reverting AF or flutter to sinus rhythm (42.3 versus 12.5 percent for placebo).

For patients with sustained AF or atrial flutter without ongoing ischemia or hemodynamic compromise, rate control is indicated. Consideration should be given to cardioversion to sinus rhythm in patients without a history of AF or atrial flutter prior to MI. (See "Control of ventricular rate in patients with atrial fibrillation who do not have heart failure: Pharmacologic therapy" and "Atrial fibrillation in adults: Use of oral anticoagulants".)

Long-term antiarrhythmic therapy may not be necessary if factors associated with recurrent AF, such as moderate to severe LV systolic dysfunction or heart failure, are absent [16,22,29]. We suggest reassessment of the patient's rhythm status in one to two months after MI. If there is no evidence of recurrent AF and if risk factors for recurrence are absent, antiarrhythmic therapy can be discontinued.

The role of anticoagulation in these patients is discussed elsewhere. (See 'Anticoagulation' below and "Prevention of embolization prior to and after restoration of sinus rhythm in atrial fibrillation".)

Anticoagulation — The optimal anticoagulation strategy for patients with MI who develop AF for the first time is unknown.

With regard to intravenous heparin, most of our experts use it in patients who will be cardioverted prior to discharge. For patients who will not be cardioverted, some of our experts use intravenous heparin until such time as the patient is successfully anticoagulated with an oral agent while others do not use heparin (particularly in patients with a low CHA_2DS-VASc score) prior to oral anticoagulation. Most of our experts recommend at least three to four weeks of oral anticoagulant, assuming the patient is not at high bleeding risk. (See "Atrial fibrillation in adults: Use of oral anticoagulants", section on 'Summary and recommendations'.)

For those patients who are discharged on anticoagulant therapy after one episode of AF, the optimal duration is unknown [30]. As the risk of bleeding is very high in patients on triple antithrombotic therapy (ie, antiplatelet therapy for those receive a stent along with anticoagulation), practitioners should carefully and repeatedly reassess the need for continued anticoagulation (and thienopyridine). This issue is discussed in detail elsewhere. (See "Coronary artery disease patients requiring combined anticoagulant and antiplatelet therapy".)

Patients with recurrent episodes of AF, especially if persistent or permanent (which is often the result of LV dysfunction and heart failure post-MI), should be treated with oral anticoagulants long term. (See "Atrial fibrillation: Overview and management of new-onset atrial fibrillation", section on 'Classification and terminology' and "Atrial fibrillation in adults: Use of oral anticoagulants" and "Acute coronary syndrome: Oral anticoagulation in medically treated patients".)

PSVT

Paroxysmal supraventricular tachycardia (PSVT) occurs in less than 10 percent of patients after an acute MI, but may require aggressive management due to a rapid ventricular rate [31]. We

suggest the following sequence of therapeutic measures [7]:

- Carotid sinus massage or a valsalva maneuver (both of which increase vagal tone and alter AV nodal properties). (See "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation", section on 'Carotid sinus massage'.)
- Intravenous adenosine (6 mg over one to two seconds; if no response, 12 mg one to two minutes later; may repeat 12 mg dose if needed). We suggest adenosine be used only in patients who have been revascularized. An external defibrillator should be available since a small percentage of patients develop ventricular fibrillation from adenosine likely related to a coronary steal phenomenon.
- Intravenous beta blockade with metoprolol (2.5 to 5.0 mg every two to five minutes to a total of 15 mg over 10 to 15 minutes) or esmolol.
- Our authors and reviewers also suggest that either intravenous verapamil, amiodarone, diltiazem, or cardioversion may be considered.

NONPAROXYSMAL JUNCTIONAL TACHYCARDIA

Junctional tachycardia (or an accelerated junctional tachycardia) is an arrhythmia arising from a discrete focus within the AV node or His bundle. The mechanism is thought to be one of enhanced automaticity rather than reentry. In adults, this rhythm, generally called nonparoxysmal junctional tachycardia, and is an uncommon arrhythmia associated with acute MI [32-34]. The same arrhythmia may also be seen with digitalis intoxication [35,36].

Atrial activity during junctional tachycardia is variable. Retrograde atrial activation may occur, with a P wave that either follows each QRS complex or is concealed in the QRS complex, as with AV nodal reentrant tachycardia. If retrograde conduction does not occur, independent atrial activity may be seen, with complete AV dissociation that must be distinguished from AV dissociation due to complete heart block (in complete heart block, the atrial rate exceeds the ventricular rate, while with an accelerated junctional tachycardia the atrial rate is slower than the ventricular rate). (See "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation", section on 'Undetectable P waves'.)

Nonparoxysmal junctional tachycardia is typically transient, occurring within the first 48 hours of infarction and developing and terminating gradually. The rate of a junctional tachycardia is generally slightly above 100 bpm, whereas reentrant paroxysmal supraventricular tachycardias (PSVTs) are generally faster. No specific antiarrhythmic therapy is indicated [7].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Non-ST-elevation acute coronary syndromes (non-ST-elevation myocardial infarction)" and "Society guideline links: ST-elevation myocardial infarction (STEMI)".)

SUMMARY

- Supraventricular arrhythmias, other than atrial fibrillation (AF) or atrial flutter, are relatively uncommon in the periinfarction period.
- The incidence of in-hospital AF after myocardial infarction (MI) ranges from 5 to 18 percent. The development of AF is associated with a worse prognosis.
- Sinus bradycardia, defined as less than 50 to 60 beats per minute, occurs in 15 to 25 percent of patients after acute MI and is most often due to increased vagal tone. (See 'Sinus bradycardia' above.)
- Sinus tachycardia occurs in approximately 30 to 40 percent of acute MIs. Patients with persistent sinus tachycardia usually have larger infarcts that are more often anterior, associated with a marked impairment in left ventricular function, and a high, early, and 30-day mortality. (See 'Sinus tachycardia' above.)
- Paroxysmal supraventricular tachycardia (PSVT) occurs in less than 10 percent of patients after an acute MI, but may require aggressive management due to a rapid ventricular rate. (See 'PSVT' above.)
- Nonparoxysmal junctional tachycardia is typically transient, occurring within the first 48 hours of infarction and developing and terminating gradually. No specific antiarrhythmic therapy is indicated.

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