Surviving a stressful MIBI scan

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ABSTRACT

Dipyridamole/technetium sestamibi scans (more commonly known as MIBI scans, an acronym for methoxyisobutyl isonitrile) are used commonly for the diagnosis and risk stratification of coronary artery disease. Adverse events from MIBI scans are extremely rare. We present the case of a 64-year-old man who was successfully resuscitated after two asystolic episodes following dipyridamole infusion for a MIBI scan. The second asystolic episode occurred in the emergency department 40 minutes after the patient had been transferred from the Cardiac Stress Test Laboratory. To our knowledge, there are no previous reports of patients having two discrete asystolic episodes or an asystolic episode as delayed as we report after a MIBI scan. Our case illustrates why emergency physicians should be aware of the potential for asystole following MIBI scanning and why aminophylline, the antidote for dipyridamole, should be readily available in emergency departments that could see patients after pharmacologic stress testing. Patients who become asystolic following dipyridamole infusion likely require prolonged cardiac monitoring, given the potential for further episodes after periods of hemodynamic stability.

RÉSUMÉ

Les scintigraphies au dipyridamole-technétium sestamibi (mieux connues sous le nom de scintigraphies au MIBI, acronyme anglais de méthoxy-isobutyl-isonitrile) servent souvent au diagnostic et à la classification du risque de maladie coronarienne. Les événements indésirables découlant des scintigraphies au MIBI sont extrêmement rares. Nous exposons le cas d'un homme de 64 ans qui a été réanimé avec succès à la suite de deux épisodes d'asystole, consécutifs à la perfusion de dipyridamole en vue d'une scintigraphie au MIBI. Le deuxième épisode est survenu au service des urgences, 40 minutes après la mutation du patient du laboratoire d'épreuves d'effort cardiaque. À notre connaissance, il n'existe pas de rapport antérieur de deux épisodes distincts d'asystole ou tout au moins d'un épisode d'asystole survenu aussi tardivement après une scintigraphie au MIBI. Le cas décrit démontre très bien pourquoi les médecins d'urgence devraient être conscients du risque d'asystole à la suite d'une scintigraphie au MIBI et pourquoi il faudrait que les services d'urgence susceptibles de recevoir des patients après une épreuve d'effort médicamenteuse aient à la portée de la main de l'aminophylline, l'antidote du dipyridamole. Il ne fait aucun doute que les patients qui subissent un épisode d'asystole après une perfusion de dipyridamole doivent faire l'objet d'une surveillance cardiaque prolongée, compte tenu du risque d'épisodes ultérieurs d'arrêt cardiaque après des périodes de stabilité hémodynamique.

Keywords: aminophylline, asystole, dipyridamole, MIBI

Dipyridamole/technetium sestamibi scans (more commonly known as MIBI scans, an acronym for methoxyisobutyl isonitrile) are used commonly for the diagnosis and risk stratification of coronary artery disease. Adverse events from MIBI scans are extrenely rare. The MIBI scan consists of an infusion of dipyridamole 0.56 mg/kg over 4 minutes, although higher doses are used at some centres; the radionuclide is injected between 7 and 9 minutes after initiation of the dipyridamole infusion.[1,2] We present the case of a 64-year-old man who was successfully resuscitated after two asystolic episodes following dipyridamole infusion for a MIBI scan.

CASE

A 64-year-old man presented to the emergency department (ED) after successful resuscitation following cardiac arrest in the nuclear medicine department. He had been referred for a dipyridamole stress MIBI after an abnormal electrocardiogram (ECG), taken during a routine life insurance examination. The ECG had revealed Q waves in leads V1 and V2, a left

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anterior hemiblock, premature ventricular contractions, and voltage criteria for left ventricular hypertrophy (Figure 1). The man had a history of hypertension and previous borderline hyperglycemia, which had resolved with weight loss. He was taking ramipril, hydrochlorothiazide, aspirin, and a multivitamin and had no known allergies.

Before the MIBI scanning, the patient felt well and had normal vital signs. According to the usual scan protocol at our centre, dipyridamole 0.56 mg/kg was administered intravenously over 4 minutes. Near the end of the infusion, the patient experienced some retrosternal discomfort. At 18 seconds postinfusion, he developed bradycardia, and at 30 seconds postinfusion, he became asystolic and unconscious. Following administration of 100 mg of intravenous aminophylline, the patient had return of spontaneous circulation and consciousness and his vital signs normalized. The asystolic episode lasted 15 seconds, during which time no chest compressions were administered.

Post–cardiac arrest, while still in the Cardiac Stress Test Laboratory, the patient was assessed by the intensive care unit team and deemed to be stable. As the scanning had been performed on an outpatient basis, he was transferred to the ED for further management.

On ED presentation, the patient was brought directly into the resuscitation area. He was alert and oriented, with a regular pulse of 82 beats/min and a blood pressure of 115/65 mm Hg. Approximately 40 minutes following the initial asystolic episode, the patient rapidly became progressively bradycardic. He subsequently became asystolic and unconscious. CPR was initiated, and 1 mg of atropine was administered intravenously. The patient had return of spontaneous circulation within approximately 30 seconds and

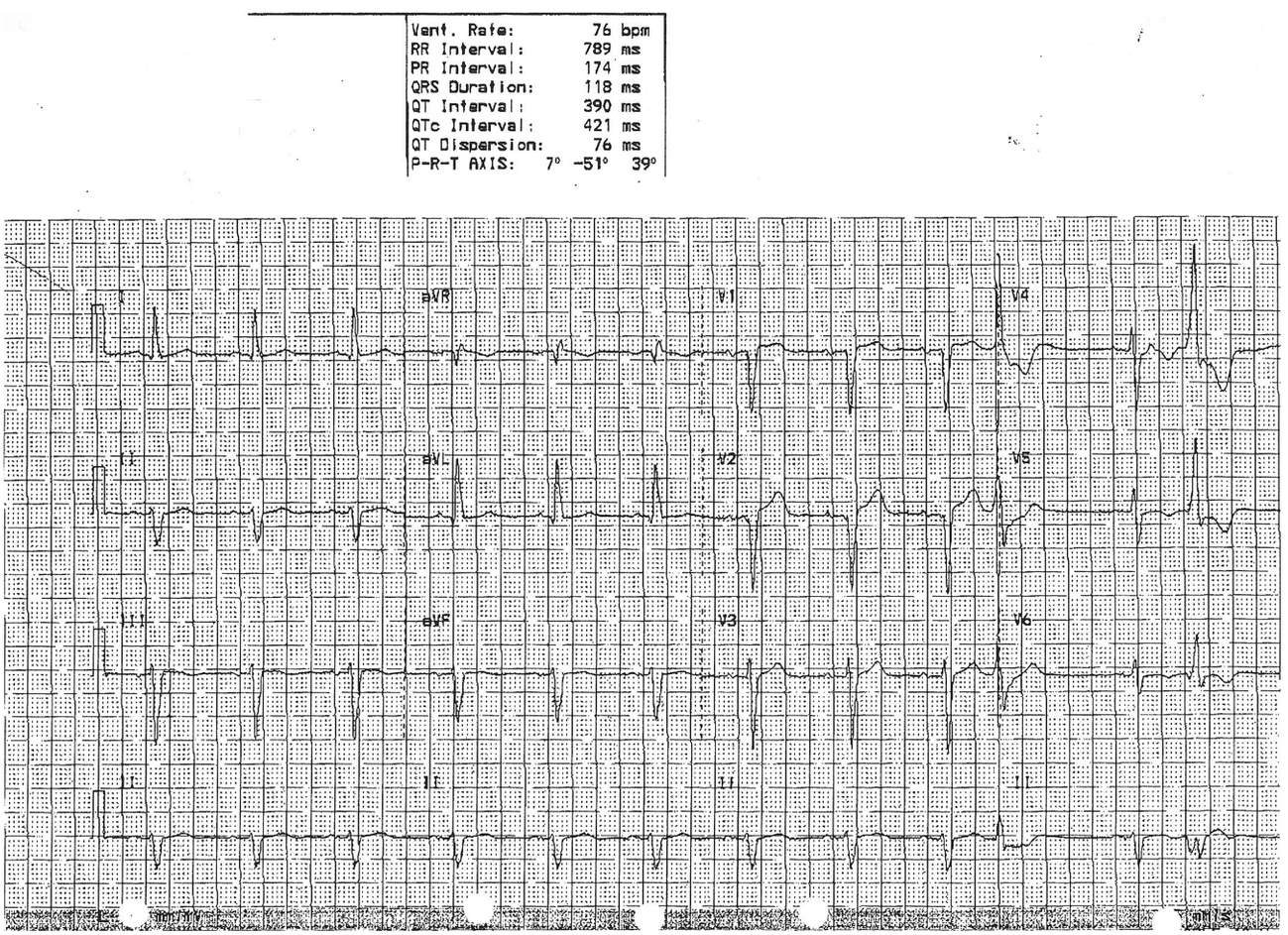

Figure 1. The patient's pre-MIBI electrocardiogram.

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regained consciousness almost immediately following this. A second dose of 100 mg aminophylline was administered intravenously as soon as it became available (approximately 10 minutes after return of spontaneous circulation).

Cardiology was consulted, and the patient was admitted for further assessment and observation. He had no further asystolic episodes and did not require any additional doses of aminophylline. An ECG the following day showed nonspecific T wave abnormalities in leads V4–V6. Serial troponin T assays were negative; however, a D-dimer assay was positive. Computed tomographic (CT) pulmonary angiography showed no evidence of a pulmonary embolism. A dobutamine MIBI scan showed left ventricular dilation with an ejection fraction of 49% and mild inferior wall reversibility.

The day after the initial MIBI scan, the patient was discharged home with an appointment for outpatient follow-up.

DISCUSSION

Dipyridamole is commonly used to facilitate pharmacologic stress testing. It indirectly increases the interstitial concentration of adenosine by inhibiting both adenosine deaminase and the cellular reuptake of adenosine, particularly by erythrocytes.[3] Adenosine, in turn, acts as a coronary vasodilator. Stenosed arteries, which are already partially dilated at baseline, dilate relatively less than healthy arteries in the presence of adenosine. This difference in flow, and thus volume of radiotracer, is visible with gamma cameras.

If coronary arteries are stenosed to such a degree that collateral circulation has developed, adenosine can cause a steal phenomenon. In its presence, pressure is decreased downstream from collaterals; therefore, flow is decreased through the collaterals themselves. This can lead to ischemia and, on occasion, infarction.[4]

Adenosine has other effects, in addition to coronary vasodilation. It can cause systemic vasodilation, and most patients develop a compensatory sinus tachycardia as a result.[5] Bronchospasm is another potential side effect.[2,6] Adenosine also blocks sinoatrial and atrioventricular conduction, especially when given in bolus doses—hence its role in treating supraventricular tachycardia.

Dipyridamole is heavily protein bound and primarily metabolized by the liver. Its concentration decreases in a triexponential fashion following intravenous infusion, with half-lives of approximately 12 minutes, 62 minutes, and 11.6 hours.[7,8] Its peak cardiac vasodilatory effect occurs 9 minutes after infusion.[6]

Aminophylline is an antidote for dipyridamole as it antagonizes the adenosine receptor. The usual dose range for reversal of side effects from dipyridamole is 100 to 240 mg intravenous push. Aminophylline has a terminal half-life of approximately 7 hours, shorter than that of dipyridamole.[6,9]

Historically, aminophylline had been advocated for bradyasystolic cardiac arrest, based on the assumption that such patients may have elevated levels of endogenous adenosine. Despite promising small studies, a large randomized, placebo-controlled Canadian trial by Abu-Laban and colleagues found an increase in nonsinus tachyarrhythmias but no increase in return of spontaneous circulation from aminophylline in prehospital patients with bradyasystolic cardiac arrest.[10]

Although mild side effects are not uncommon, dipyridamole is considered a relatively safe drug. In a study of 3,911 patients receiving dipyridamole intravenously during a stress test, 10 had serious adverse events: 4 had myocardial infarctions, two of which were fatal, and 6 had bronchospasm.[11] A study of dipyridamole in 10,541 echocardiograms on 9,122 patients found seven adverse events: one patient became asystolic secondary to an myocardial infarction, and two had episodes of asystole.[12] A retrospective review of patients undergoing dipyridamole stress testing found 19 adverse events, including one death, in 24,599 patients.[13] Finally, in a study involving 73,806 patients, seven cardiac deaths were described.[2] Three of these deaths were preceded by progressive hypotension. Two resulted from ventricular fibrillation, and two were caused by pulmonary edema. The overall risk of death from dipyridamole stress testing is estimated to be approximately 1 in 10,000.[2,14]

Published case reports have proposed various pathophysiologic mechanisms for asystole following dipyridamole infusion. Blumenthal and McCauley reported the case of a 71-year-old woman who suffered an asystolic arrest but had return of spontaneous circulation with aminophylline administration and chest compressions. She was found to have had an acute transmural myocardial infarction, thought to be secondary to coronary steal, as opposed to systemic hypotension precipitating ischemia.[15]

Frossard and colleagues reported two cases of asystolic arrest.[3] The first, a 50-year-old man, became

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bradycardic 1 minute into a dipyridamole infusion and then had a 39-second asystolic period that resolved when he was placed in a supine position. The second, a 49-year-old man, became bradycardic 1 minute after the infusion finished and then had an asystolic period of 15 seconds, which again resolved with supine positioning. The authors concluded that the dipyridamole likely precipitated a vagal effect in the two patients, perhaps as a result of underlying autonomic dysfunction.

Lo Mauro and colleagues reported the case of a 47-year-old man who had syncope and sinus bradycardia 3 minutes after a dipyridamole infusion finished and then became asystolic for approximately 18 seconds.[16] He had return of spontaneous circulation following chest compressions and aminophylline administration, and arrest was thought to be the result of sinus blockade by adenosine or an increase in vagal tone.

Veerana and colleagues reported the case of a 67-year-old man who had an asystolic episode lasting 20 seconds but resolved prior to the initiation of chest compressions.[17] They concluded that this was likely a direct side effect of the dipyridamole.

To our knowledge, there are no previous reports of patients having two discrete asystolic episodes or an asystolic episode as delayed as we report after a MIBI scan. It is quite likely that the asystole episodes were a result of the dipyridamole infusion. The most probable explanation is a conduction system blockade as a result of increased adenosine concentration. Increased vagal tone as a result of autonomic dysfunction or failure to compensate for systemic vasodilation in the presence of dipyridamole cannot be excluded. It is unlikely that the episodes were vasovagal; the patient was supine when the second cardiac arrest occurred, had no preceding vagal symptoms, and had not been recently exposed to any common vasovagal triggers. Myocardial infarction is unlikely as serial troponin T assays were negative, and pulmonary embolism was excluded by CT scan.

CONCLUSION

Our case illustrates why emergency physicians should be aware of the potential for asystole following MIBI scanning and why aminophylline, the antidote for dipyridamole, should be readily available in EDs that could see patients after pharmacologic stress testing. Patients who become asystolic following dipyridamole infusion likely require prolonged cardiac monitoring, given the potential for further episodes after periods of hemodynamic stability.

Competing interests: None declared.

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