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Cover Page

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# Cardiogenic Shock Following Dipyridamole Administration for Myocardial Perfusion Imaging

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**Word Count** 981

**Case Presentation:**

This 52-year-old gentleman with a prior history of hypertension, transient ischemic attacks and prior myocardial infarction 24-years ago presented to our hospital with a two week history of worsening exertional angina. He was brought to the nuclear laboratory for a dipyridamole myocardial perfusion study. Resting images were taken following the injection of 290 MBq of intravenous Tc-99m tetrofosmin (figure 1). Dipyridamole was infused at 0.56 mg/kg over four minutes. A few minutes after completion of the infusion the patient's initial blood pressure declined from 120/70 mm Hg to 70/40 mm Hg. This was associated with profound ST-depression in multiple ECG leads (figure 2), chest pain, dyspnea and overt signs of congestive heart failure. He was given intravenous aminophylline (125 mg) immediately with improvement in his blood pressure to 90 mm Hg systolic and partial resolution of the ST depression on his ECG. Owing to his unstable condition, stress images were deferred and the patient was shifted immediately to the cardiac catheterization laboratory where he was electively intubated for worsening heart failure and hypoxemia. Coronary angiography revealed critical left main coronary artery disease (figure 3; *single arrow*) with poor antegrade flow in the left anterior descending (LAD) artery (figure 3; *small arrows*) and complete occlusion of the left circumflex (LCx) artery. The right coronary artery also had a critical lesion proximally (figure 4; *single arrow*) with collateral flow to the LCx (figure 4; *double arrows*) and the LAD (figure 4; *arrowheads*). An intra-aortic balloon pump was inserted and the patient was taken for emergent coronary artery bypass surgery. He received vein grafts to the LAD, right coronary and marginal arteries. His post-operative course was relatively uneventful and he was discharged on the seventh day after the operation. Creatine-kinase (MB) prior to surgery

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3 was 30.6 ng/ml (0-3.97). Left ventricular function following discharge was 40-45% by  
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5 echocardiography.  
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### 11 **Discussion:**

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17 Intravenous dipyridamole is a widely used agent for pharmacologic stress myocardial perfusion  
18 imaging (MPI). Dipyridamole induces a differential degree of coronary vasodilatation in beds  
19 supplied by normal and stenosed epicardial coronary arteries. As the vessels subtended by a  
20 stenosed coronary artery are significantly dilated at rest, further dilatation (and increase in flow) in  
21 response to a vasodilator like dipyridamole is limited. On the other hand, vessels in the normal  
22 areas undergo significantly more vasodilation (and increase in flow) leading to heterogeneity of  
23 perfusion and, hence, a difference in uptake of flow-dependent radioisotopes like technetium  
24 following dipyridamole administration. Thus, although regions supplied by stenotic epicardial  
25 arteries appear “ischemic” on MPI due to differential blood flow (and radiotracer uptake), in the  
26 majority of cases true tissue ischemia does not occur – in fact, due to some degree of additional  
27 vasodilatation in response to dipyridamole (over the resting state) coronary blood flow actually  
28 increases in these underperfused regions <sup>1</sup>.  
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49 The exception to this is when the phenomenon of “coronary steal” occurs, whereby an unfavorable  
50 redistribution of blood flow takes place from underperfused areas towards better perfused regions  
51 <sup>2,3</sup>. This has been described in association with collateral dependent myocardium, which becomes  
52 ischemic following coronary vasodilation because of a decrease in pressure in the distal bed of the  
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3 feeder vessel. Perfusion to the collateralized myocardium declines as blood flow takes the path of  
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5 "least resistance". Estimates suggest that this phenomenon can occur in as many as 45% of  
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7 patients with severe coronary artery disease <sup>4</sup> and is frequently associated with ischemic ST-  
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9 segment depression <sup>3</sup>. There are a number of reports in the literature of severe transient ischemia  
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11 induced during intravenous <sup>5</sup> and oral <sup>6,7</sup> dipyridamole administration requiring reversal of the effect  
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13 with intravenous aminophylline as well as intravenous nitrates. However, the overall incidence is  
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15 extremely low. In a large series on the adverse effects of dipyridamole MPI published by  
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17 Ranhosky et al., <sup>8</sup> four out of 3911 patients (0.10%) suffered a major adverse cardiac event (two  
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19 fatal and two nonfatal myocardial infarctions). Three of these four patients had a previous history  
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21 of worsening angina. More recently, in a report on 73,806 patients who underwent intravenous  
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23 dipyridamole MPI <sup>9</sup>, there were seven cardiac deaths (0.95 per 10,000) and 13 nonfatal myocardial  
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25 infarctions (1.76 per 10,000). Although transient hypotension can occur in approximately 5% of  
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27 subjects <sup>8</sup>, to date there are no formal reports of cardiogenic shock precipitated by intravenous  
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29 dipyridamole. In our patient the left coronary system was essentially occluded and was extensively  
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31 collateralized by the right coronary artery. While it is possible that dipyridamole induced  
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33 hypotension may have triggered an ischemic cascade, it is likely that dipyridamole caused a  
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35 significant degree of "steal" in the left coronary system leading to acute severe ischemia, heart  
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37 failure and shock. In retrospect, our patient did give a history of significantly worsening angina  
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39 (including resting symptoms) over the last few weeks but during the initial assessment he under-  
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41 reported his symptoms. Perhaps if the true symptomatology had been known, he may have been  
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43 scheduled directly for coronary angiography. However, at the time of the test, the patient was not  
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45 having active symptoms of angina.  
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3 To the best of our knowledge, this is the first report describing cardiogenic shock occurring  
4 following intravenous dipyridamole administration. That said, it is likely that at least some of the  
5 patients who suffered myocardial infarctions in the reports by Ranhosky <sup>8</sup> and Lette <sup>9</sup> also  
6 developed cardiogenic shock. Nevertheless, our report highlights an important (and potentially  
7 fatal) complication of dipyridamole MPI. Although fatal complications are extremely rare, especially  
8 with the advent of aggressive coronary revascularization (Rahnosky's paper <sup>8</sup>, for example,  
9 included patients who were tested between 1978 and 1985 when aggressive coronary intervention  
10 was not in common practice) it is essential to be cognizant of the possibility and the presence of  
11 appropriate backup services is important to prevent catastrophic events. This is of particular  
12 relevance to office-based nuclear cardiology facilities. This report also reemphasizes the fact that  
13 patients with active unstable angina should not undergo dipyridamole MPI unless stabilized.  
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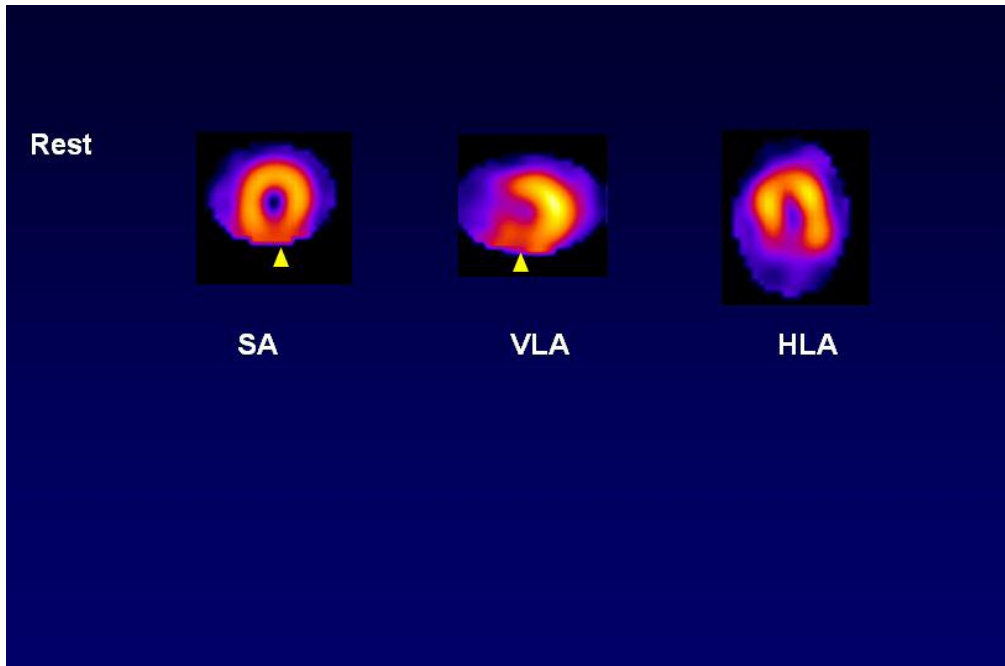


**Conflicts of Interest:** None

For Peer Review

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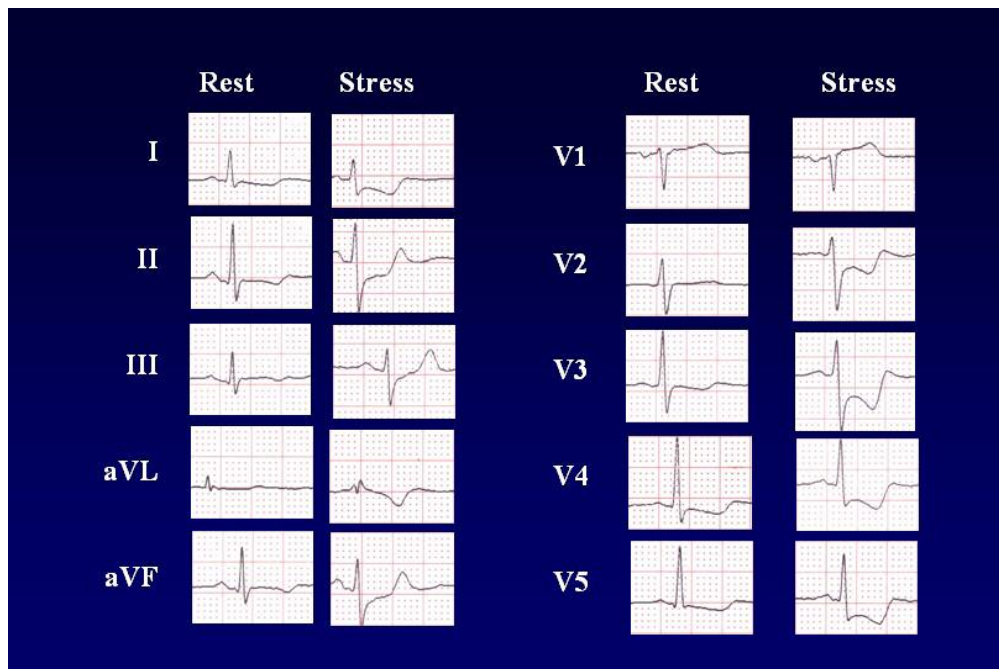
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**Resting images showing an inferior perfusion defect**  
285x190mm (72 x 72 DPI)

Review

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**Baseline and Stress ECG**  
285x190mm (72 x 72 DPI)

Review