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Case Report

Intractable Coronary Artery Spasm and Acute Intracoronary Thrombosis Induced by Guide Wire During Intervention Salvaged by Emergent Coronary Bypass Graft — A Case Report

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Abstract: Coronary artery spasm induced by guide wire during coronary intervention is not unusual. However there has been no reported incidence of intractable coronary spasm that could not be relieved by pharmacological or mechanical interventions. We report such a case of intractable coronary spasm and acute thrombosis. Emergent coronary artery bypass graft (CABG) is proposed as a strategic approach to alleviate such an event.

Key Words: Coronary angiography; Nitroglycerin.

Introduction

Coronary artery spasm has been reported in 1-5% of balloon angioplasty procedures.[1,2] The majority of coronary spasms can be relieved by intracoronary nitroglycerin and/or calcium antagonist. Previous case reports have mentioned guide wire-induced coronary spasm which was refractory to intracoronary nitroglycerin and/or calcium antagonist or intravenous nitroglycerin but which resolved promptly and completely after withdrawal of the guide wire from the coronary artery.[3] We present a case who underwent successful percutaneous transluminal coronary angioplasty (PTCA) but then developed intractable coronary vasospasm and acute coronary thrombosis.

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Case Presentation

A 57-year-old man with type 2 Diabetes Mellitus and exertional chest tightness was admitted for coronary study. Cardiac catheterization was performed from the right femoral artery. The left coronary angiogram revealed 99% tubular stenosis of the junction of proximal and middle left anterior descending artery (LAD) with intracoronary thrombus, 85% stenosis of the distal left anterior descending artery just beyond the second diagonal branch, and 95% stenosis of the first diagonal branch (Fig. 1). The right coronary angiogram revealed 75% stenosis of the proxi-

Fig. 1. Left coronary angiogram showed thrombus-containing lesion with 75-99% tubular stenosis at the junction of proximal and middle left anterior descending artery (arrow) in right anterior cranial oblique projection.

Fig. 2. Immediately following PTCA, there was no residual thrombus at the junction of proximal and middle left anterior descending artery but severe vasospasm was noted distal to the intial target lesion (arrows).

mal right coronary artery. Left coronary angioplasty was performed with a 7 Fr. JL-4 guiding catheter (Schneider, Guider), a 0.014-inch Choice wire (Boston) and a balloon catheter (Racer) 3.0 mm in diameter and 18 mm in length. Before angioplasty, 12,500 units of intravenous heparin and 200 ug of intracoronary nitroglycerin were given. The guide wire crossed the LAD target lesions easily. Then the lesion was dilated by inflating the balloon at 8 barrs for 120 seconds. Repeat left coronary angiogram revealed no filling defect at the junction of proximal and middle LAD, but there was severe vasospasm at the distal LAD (Fig.2). Pharmacological interventions with intracoronary nitroglycerin (isosorbide

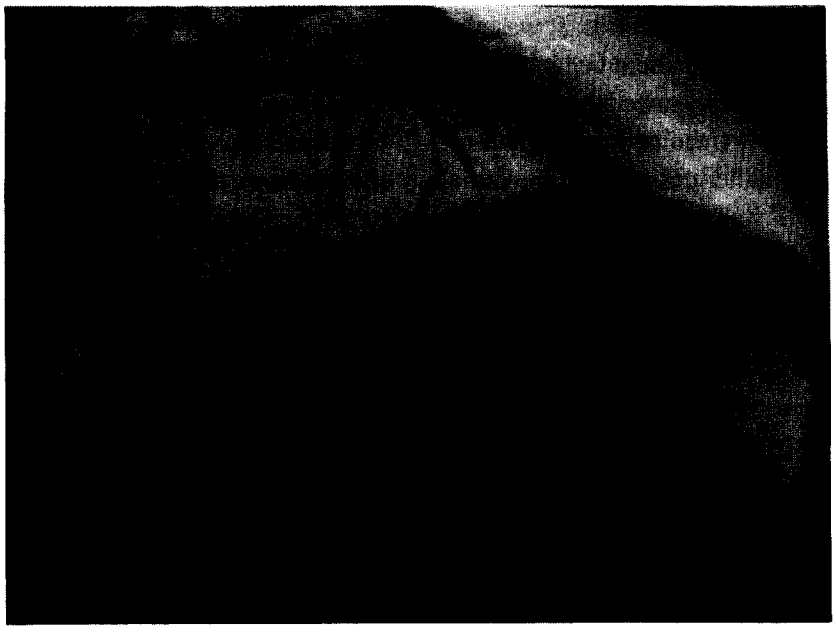

Fig. 3. Reappeared intracoronary thrombus was seen at the initial target lesion (arrow).

Fig. 4. Following emergent CABG, the distal vasospasm of the left anterior descending artery had receded (arrows).

dinitrate 400 ug), intracoronary verapamil (200 ug), and intravenous nitroglycerin (100 ug/min) failed to recede the vasospasm. We used a 3.0 mm racer balloon to dilate every distal LAD spasm by inflating the balloon at 4 barrs for 60 seconds (arrows shown in Fig. 2). Nonetheless, the coronary spasm still could not be terminated by short, low- pressure balloon inflations. Finally, a long balloon (Schneider, Malvina, 2.0 to 3.2 mm in diameter, 60 mm in length) was used in an attempt to abate distal LAD spasm by inflating

the balloon up to 2 barrs for 60 seconds. It was also ineffective. Unfortunately, the patient began to complain of chest tightness. EKG showed 2mm ST segment elevation at lead V4. Coronary angiogram revealed a recurrent but smaller thrombus in the initial target lesion when ACT was more than 300 seconds (Fig. 3). Emergent CABG was therefore performed under intraaortic balloon pump support due to intractable coronary spasm and recurrent acute thrombus formation. The LAD did not develop significant spasm during the operation. Post-operative coronary angiogram showed patent distal left anterior descending artery (Fig. 4).

Discussion

The reported incidence of coronary artery vasospasm ranges between 1.3% and 4.2%.[4,5] The exact mechanism of coronary artery vasospasm during coronary intervention is unknown. Balloon angioplasty can potentially disrupt the endothelium and induce vasoconstriction by increasing the level of serotonin and thromboxane A2 and retarding the formation of endothelium-derived relaxing factor.[6,7] Guide wire-induced vasoconstriction is another proposed mechanism. To the best of our knowledge, there has been no reported incidence of severe coronary artery spasm during PTCA which was refractory to intracoronary nitroglycerin and verapamil or intravenous nitroglycerin. Choice wire is a hydrophilic rather than sprung wire. Its surface is smoother than sprung wire. As it passes through vessel lumen, the frictional force and contact surface between the wire and the endothelium is low. Moreover, wire tip-induced spasm is confined to a point. The possibility of wire tip-induced diffuse spasm is very low. In our case, balloons were passed into distal LAD 3 times. We believe intracoronary thrombosis of the junction of proximal and middle LAD resulted from severe distal spasm and blood stasis. Masafumi et al. reported withdrawal of guide wire was the only method to relieve guide wire-induced coronary vasospasm during PTCA.[3] But Keithe et al. found that short, low-pressure balloon inflations combined with intracoronary nitrate would abate coronary spasm.[8] In this case, intracoronary thrombus formation progressed rapidly and promptly over the initial culprit lesion and caused TIMI grade 0 antegrade flow. Once the guide wire was removed, the distal LAD might have been totally occluded by thrombus. Therefore we attempted to terminate the spasm by short, low-pressure balloon inflations instead of withdrawing the guide wire or infusing glycoprotein IIb/IIIa.

Conclusion

We presented a rare complication, guide wire-induced coronary vasospasm with early intracoronary thrombus during PTCA. All available medication and low-pressure balloon inflations were unable to relieve the vasospasm. Although the removal of guide wire might relieve the vasospasm completely and promptly, when early thrombosis develops due to flow stasis, emergent CABG should be considered as an alternative treatment strategy.

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經皮冠狀動脈血管整形術導線引發冠狀動脈痙攣暨冠狀動脈血栓 — 病例報告

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摘 要: 經皮內冠狀動脈血管整型術中導引導線引發血管痙攣的病例並不少，但大多可用注射藥物如硝化甘油和鈣離子阻斷劑緩解，本文報告一位 57 歲男性患者因心絞痛住院接受經皮冠狀動脈整型術，因導引導線引發嚴重冠狀動脈痙攣，雖立即經冠狀動脈導管注射藥物，乃無法舒解，最後合併冠狀動脈血栓，患者立刻接受繞道手術。當經皮內冠狀動脈血管整型術之導引導線引發嚴重血管痙攣時，如無法經由藥物緩解，緊急繞道手術是必需考慮的。

關鍵詞: 冠狀動脈攝影；硝化甘油。

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