Transient Collateral Augmentation During Coronary Arterial Spasm Associated with ST-segment Depression

MICHIHIKO TADA, M.D., MASAKAZU YAMAGISHI, M.D., KAZUHISA KODAMA, M.D.,
TSUNEHIKO KUZUYA, M.D., SHINSUKE NANTO, M.D., MICHTOSHI INOUE, M.D.,
AND HIROSHI ABE, M.D.

SUMMARY To examine the possible existence of collateral circulation during coronary artery spasm, we attempted to visualize the transient appearance of collateral vessels that could serve to salvage otherwise jeopardized ischemic areas. In three patients with vasospastic angina, total spastic obstruction of a major coronary artery was associated with transient collateral augmentation, which was supplied by the nonspastic artery during anginal period associated with ST-segment depression. These collateral vessels disappeared when the angina and ST changes resolved after nitroglycerin administration. These findings suggest that the collateral blood supply could transiently occur through preexisting vessels to perfuse the ischemic area during coronary artery spasm and that such collateral flow could have a role in preventing transmural myocardial ischemia, resulting in a lesser degree of ischemia associated with ST-segment depression.

Patients

PATIENTS with Prinzmetal's variant angina who exhibit ST-segment elevation during anginal episodes have severe coronary artery spasm. In some cases with angina pectoris with ST-segment depression, such a dynamic coronary narrowing may also play a pivotal role in provoking myocardial ischemia. Experimental and clinical results suggested that ST-segment depression might depend not only on the severity and location of spasm, but also on the extent of collateral development. Yasue et al. reported that in vasospastic angina, the existence of collateral circulation was associated more frequently with ST depression than with ST elevation. The ST-segment depression during a vasospastic attack may also result from collateral channels through which coronary flow can be established in the presence of pressure gradients created by the spasm. However, such collateral vessels, which may appear transiently, could not easily be demonstrated because of technical difficulties in visualizing simultaneously the spastic and nonspastic artery donating collateral flow. We describe three patients with exertional or rest angina pectoris in whom collateral vessels from nonspastic arteries to the ischemic area were transiently visualized during ergonovine-induced coronary artery spasm.

Case Reports and Methods

Case 1

A 42-year-old man who had had a 6-month history of exertional and rest angina was admitted to our clinic. His anginal attacks were spontaneously relieved within a few minutes, but were sometimes accompanied by cold sweating and nausea. Physical findings were normal. An ECG, which showed incomplete right bundle branch block without any significant ST-segment deviation during nonanginal periods (fig. 1A), revealed ST-segment depression (0.05–0.1 mV) in leads II, III, aVF, V₅ and V₆ when the patient had spontaneous anginal episodes (fig. 1B). Coronary arteriography was performed using the Judkins technique in the right anterior oblique view. Using the arterial percutaneous catheter introducer (HEMQUET, USCI), the left and right coronary catheters could be changed within 30 seconds, allowing us to obtain both left and right coronary arteriograms almost simultaneously. ECGs were continuously recorded in the standard 12 leads with x-ray transparent carbon fiber electrodes (X-transrode, Nihon Kohden). Initial right coronary arteriography revealed a 90% narrowing of the proximal right coronary artery (RCA). Left coronary arteriography revealed faint visualization of the distal RCA through collateral vessels from the left coronary system. However, the peripheral RCA was not completely opacified by such jeopardized vessels (fig. 2A). To provoke coronary artery spasm, ergonovine maleate, 0.1 mg, was administered intravenously. Five minutes after ergonovine, the patient had chest oppression and ST-segment depression (0.05–0.1 mV) in leads II, III, aVF, V₅ and V₆. Under these conditions, the RCA was totally occluded at the site of the previous proximal narrowing. Left coronary arteriography, performed within 30 seconds after right coronary arteriography, revealed well-developed collateral channels from the left anterior descending coronary artery (LAD) to the right posterior descending artery through septal and diagonal branches (fig. 2B). After sublingual nitroglycerin (NTG), 0.3 mg, the chest oppression and ST changes resolved. Right coronary arteriography showed release of the spastic obstruction with visualization of the 90% proximal RCA narrowing. The collateral vessels that had been transiently augmented during the anginal period were no longer present in the left coronary arteriogram (fig. 2C).
Case 2

A 46-year-old man visited our clinic with typical features of rest angina. He was well until 4 months before, when he began to complain of anterior chest pain while asleep. His attacks were relieved with NTG and were never provoked by exercise. Administration of nifedipine, 40 mg/day, has completely abolished the anginal complaints. Physical findings were normal. The ECG, which was normal during the nonanginal period, did not reveal appreciable ST deviation when the patient had spontaneous anginal episodes. The stenotic lesion of the LAD was suspected in the initial left coronary arteriogram. The mid-RCA had minor luminal irregularities (fig. 3A). After provocation with ergonovine, 0.2 mg, the patient had chest pain associated with ST-segment depression (0.05–0.2 mV) in leads II, III, aVF, V5, and V6. Under these conditions, the LAD was totally occluded near the site where the narrowing had been suspected. The subsequently performed right coronary arteriogram showed significantly developed collateral vessels, by which the mid-LAD was apparently visualized (fig. 3B). After NTG, the chest pain and ST changes resolved. The left coronary arteriogram in the right anterior oblique view with craniocaudal projection showed release of the spastic obstruction with visualization of the 70% LAD narrowing. Under these conditions, the right coronary arteriogram no longer exhibited the collateral vessels that had been transiently visualized during angina (fig. 3C).

Case 3

A 53-year-old man who had been suffering from rest
angina for 5 years was admitted to our clinic. His attacks usually occurred at midnight and early in the morning despite isosorbide dinitrate, 20 mg/day. Physical findings were normal. The ECG, which showed slight ST-segment depression (0.05 mV) in leads V$_5$ and V$_6$ (fig. 4A) during the nonanginal periods, revealed further ST depression (0.1-0.3 mV) in leads I, II, III, aV$_F$ and V$_s$-V$_o$ when the patient had spontaneous angina (fig. 4B). The coronary arteriogram showed no stenotic lesions in the left and right coronary systems when angina was absent. Under these conditions, septal perforating branches were faintly visualized in the right coronary arteriogram. However, no left coronary segment could be opacified by such collateral vessels (fig. 5A). After provocation with ergonovine, 0.2 mg, the patient had chest pain associated with ST-segment depression (0.05-0.2 mV) in leads II, III, aV$_F$ and V$_s$-V$_o$. At this time, the left coronary arteriogram exhibited 90% narrowing of the left main trunk, total occlusion of the LAD and 70% narrowing of the left circumflex (LCx) (fig. 5B). After NTG, the chest pain and ST changes temporarily resolved. Five minutes later, without additional ergonovine, the patient again had chest pain associated with ST-segment depression, as observed before. Under these conditions, the right coronary arteriogram demonstrated well-developed collateral vessels, opacifying the LAD through septal perforating branches (fig. 5B). After another dose of NTG, there was complete resolution of left coronary spasm, resulting in relief of angina and ST-segment depression. The collateral vessels were no longer demonstrated in the subsequently right coronary arteriogram (fig. 5C). The patient had no significant (>50%) left coronary nar-
FIGURE 4. ECGs taken during a nonanginal period (A) and during spontaneous episode of chest pain (B) from patient 3. (A) The ECG showed slight ST depression (0.05 mV) in leads V4 and V5. (B) The ECG revealed marked ST depression (0.1–0.3 mV) in leads I, II, III, aVF, and V3–V6 when the patient had spontaneous angina.

Rowing in multiple views, including the craniocaudal projection (fig. 6).

In the present study, ST-segment elevation, which represents transmural myocardial ischemia, was not observed during spontaneous or ergonovine-induced angina. Rest angina was relieved by nifedipine, 40 mg/day, and isosorbide dinitrate, 20 mg/day.

Discussion

In these three patients with vasospastic angina, a spastic obstruction of the major coronary artery was associated with transient augmentation of collateral flow that was supplied by the nonspastic coronary artery. Such an instantaneous supplementation of collateral flow to the ischemic area may account for the paradoxical ST-segment deviation during coronary artery spasm.

The vasospastic obstruction that occurs in the proximal coronary artery was usually accompanied by ST-segment elevation due to transmural myocardial ischemia. However, it has been suggested that ST-segment depression, often observed during vasospastic
attacks, occurs as the result of subendocardial ischemia due to either an incomplete occlusion of a large coronary artery, a complete occlusion of a small coronary artery or patent collateral circulation. The existence of collateral channels, through which coronary blood flow could be augmented in the presence of pressure gradients created by the spasm, may also result in ST-segment depression. However, to our knowledge, no attempts have been made to angiographically visualize such collateral vessels that could transiently perfuse the ischemic area.

Our angiographic procedures, in which the left and right coronary arteriograms could be obtained almost simultaneously, enabled us to demonstrate that the total vasospastic obstruction of the major coronary artery was accompanied by significant augmentation of the collateral supply, which had been faintly or never visualized during nonanginal period. Such collateral vessels completely disappeared when the angina and ST changes resolved after NTG. These observations indicate that transient collateral supply could occur through preexisting vessels during coronary artery spasm.

Gensini had described such a dynamic visualization of collateral vessels in Prinzmetal’s variant angina, suggesting that the pressure gradients abruptly produced between the spastic and nonspastic coronary arteries might induce the collateral flow. These forms of collateral appearance were considered not to be the artifactual demonstration of collateral channels, which had been visualized in coronary arteriograms with forceful injection of the contrast material in a wedged position, because a medium backflux from each coronary ostium was shown during our study and such pitfalls have seldom been demonstrated.

In vasospastic angina, collateral vessels may develop in the presence of major coronary stenosis as well as in other types of coronary artery disease. When such collateral vessels are present, transmural myocardial ischemia might not be produced even if a focal (not diffuse) spasm has totally occluded a large coronary artery at the stenotic segment. The present report suggests that in patients with vasospastic angina whose coronary arteries exhibit apparently well maintained perfusion during nonanginal periods, recurrent vasospastic obstructions may augment collateral flow. The finding that the ECGs did not exhibit ST-segment elevation despite total spastic obstructions in major coronary arteries might be related to the existence of such collateral augmentation. One might speculate that the transiently augmented collateral flow could salvage otherwise jeopardized ischemic areas, resulting in nontransmural myocardial ischemia associated with ST-segment depression, although whether ST-segment depression represents a lesser degree of myocardial ischemia than ST-segment elevation is controversial. Further studies may demonstrate the significance of such a dynamic development of collateral circulation in determining ST-segment deviation during coronary artery spasm.

Using ergonovine maleate, we could visualize the

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**Figure 6.** Left coronary arteriogram in the left anterior oblique view with craniocaudal projection after nitroglycerin administration in patient 3. No significant (≥ 50%) narrowing was found in the left coronary artery.

**Figure 5.** Coronary arteriograms and simultaneously recorded ECGs from patient 3. (A) In the control state, left (upper) and right (lower) coronary arteriograms did not reveal significant (≥ 50%) stenotic lesions. Septal perforating branches were faintly visible on the right coronary arteriogram. (B) During angina associated with ST depression (0.05–0.2 mV) in leads II, III, aV, and V–V, spasm totally occluded the left anterior descending artery (LAD) (upper). After nitroglycerin administration, the angina and ST changes resolved temporarily. The patient, however, had chest pain associated with ST depression 5 minutes later. Under these conditions, the LAD was opacified by collateral flow from the right coronary system (arrows, lower). (C) When another dose of nitroglycerin abolished the angina and ST changes, the left coronary arteriography showed release of the spastic obstruction (upper) and the collateral vessels were no longer visualized in the subsequent right coronary arteriogram (lower).
transient appearance of collateral vessels that could perfuse the area where the total vasospastic obstruction would have provoked severe myocardial ischemia. However, several reports indicate that ergonovine can provoke irreversible coronary occlusion, which results in acute myocardial infarction or sudden death.\textsuperscript{21, 22} As suggested by Heupler,\textsuperscript{23} careful patient selection and well-prepared laboratory procedures should prevent these complications, facilitating an effective documentation of coronary arterial dysfunction that could not be demonstrated by conventional methods.

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