Journal of Cardiology 18: 307–317, 1988

Primary coronary artery dissection: Its incidence, mode of the onset and prognostic evaluation

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Summary

The incidence, mode of the onset and prognosis of primary coronary artery dissection in 1,445 consecutive patients with myocardial infarction undergoing coronary angiography were elucidated in the present study. Primary coronary artery dissection was observed in four patients (0.28%). The first case was a 28-year-old man, who developed angina at rest, followed by inferior myocardial infarction. His coronary angiogram showed dual lumina in the proximal to distal segments of the right coronary artery, which were separated by a flap. A left ventriculogram showed severe impairment of contraction (akinesis) in its inferior segment. Six years later, he was classified as New York Heart Association (NYHA) functional class I. The second case, a 54-year-old man, developed vasospastic angina followed by inferior myocardial infarction. His coronary angiogram showed a similar dissection from the proximal to distal segments of the right coronary artery. A left ventriculogram showed akinesis of the inferior segment and a coronary angiogram five years later showed marked resolution of the dissection. Twelve years after the infarction, he was classified as NYHA functional class I. The third case, a 46-year-old woman, experienced sudden onset of inferior myocardial infarction. Her coronary angiogram showed dissection from the middle to distal segments, and the posterior descending branch of the right coronary artery. A left ventriculogram showed akinesis of the inferior segment, and three years later, she was asymptomatic. The fourth case, a 28-year-old woman, developed anterior myocardial infarction following delivery. Her coronary angiogram revealed dissection from the proximal to middle segments of

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Received for publication June 22, 1987; accepted September 2, 1987 (Ref. No. 34–31)

the left anterior descending artery (segments 6 and 7). A left ventriculogram showed akinesis in the anteroseptal segment and dyskinesis in the apical segment. She died suddenly four years after her myocardial infarction. Thus, primary coronary artery dissection is not extremely rare and it may have been associated with coronary vasospasm in at least two of these four cases.

Key words
Primary coronary artery dissection Vasospasm Coronary angiography

Introduction

Acute myocardial infarction generally occurs secondary to atherosclerotic obstruction of the coronary arteries. Other causes of myocardial ischemia includes coronary artery dissection. One of the most common coronary artery dissections is that associated with aortic dissection.[1,2] Women appear to be more frequently affected, especially during the peripartum period.[3~16] Coronary artery dissection reportedly occurs secondary to atherosclerosis,[17] trauma, the postpartum state,[3~16] cystic medial necrosis,[18~20] and when iatrogenically-induced.[21~25] Primary coronary artery dissection, which is limited to the coronary artery system, may be non-iatrogenic and non-traumatic, and has been reported to be extremely rare.[7,11,23,26] Since the first description by Pretty in 1931,[27] approximately 60 additional cases have been reported, in most of which the diagnosis was established postmortem. Angiographic diagnoses of dissections are extremely rare; only 14 such cases have been previously reported in the literatures.[1,8,12,14,16,26,28~35] This is a report of four cases of primary coronary artery dissection diagnosed by selective coronary arteriography. These four cases were selected from 1,445 consecutive patients with angiographic examinations for myocardial infarction at the Toranomon Hospital, Tokyo. Clarification of incidence, mode of the onset and prognosis of primary coronary artery dissections were attempted.

Case reports

Case 1:

This 28-year-old man, had experienced anginal pain at rest during mornings, of three weeks duration. He was admitted to a hospital on July 26, 1980, with a chief complaint of severe substernal pain which developed when he was playing baseball early in that morning. At 21 years of age he underwent resection of an astrocytoma. He had smoked cigarettes for the previous 10 years, but had no history of hypertension. His mother and paternal grandfather both died of myocardial infarction. Admission electrocardiography showed evidence of acute inferior myocardial infarction. He was discharged three weeks post-infarction without complications.

Nine month later, on March 12, 1981, this patient was referred to the Toranomon Hospital for further evaluation of his coronary artery disease. Physical examination revealed an apparently healthy man, 170 cm in height and weighing 61 kg. His pulse was normal and his blood pressure was 120/82 mmHg. There was a fourth heart sound at the apex without a significant murmur. There was a surgical scar on his scalp. Four days post-admission, he underwent left cardiac catheterization and selective coronary angiography by the Sones technique. Left-sided cardiac catheterization showed normal pressures within the aorta (135/85 mmHg) and the left ventricle (135/13 mmHg). Right coronary arteriography (**Fig. 1**) showed dual lumina in the proximal to distal segments separated by a flap of the intima and/or the media. A left coronary arteriogram showed no obstructive disease. Biplane left ventriculography demonstrated severe impairment of contraction (akinesis) in the inferior wall.

After convalescence he returned to normal activity, and is now classified as New York Heart Association (NYHA) functional class I, seven years post-myocardial infarction.

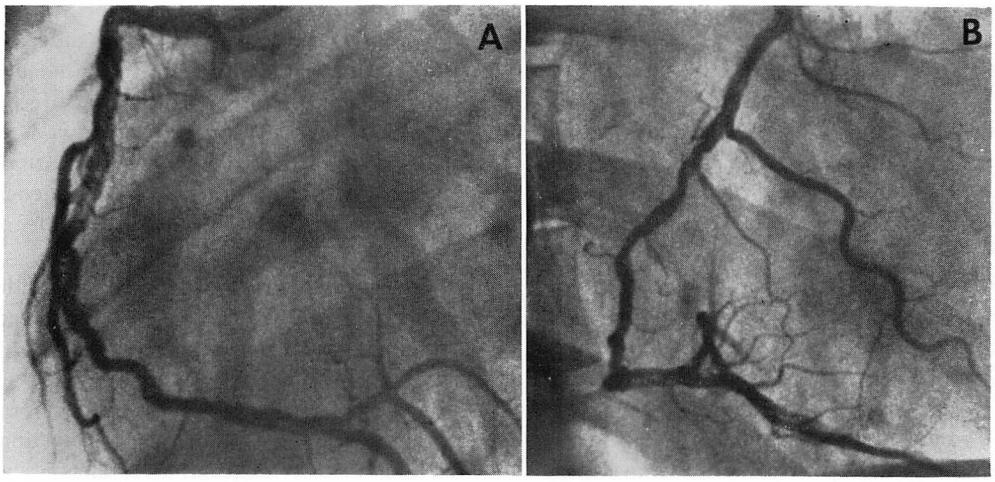

Fig. 1. Right coronary arteriograms obtained in Case 1.
A: Left anterior oblique (LAO) projection. B: Right anterior oblique (RAO) projection.
Dual lumina in the proximal to distal segments are separated by a flap of the intima and/or the media.

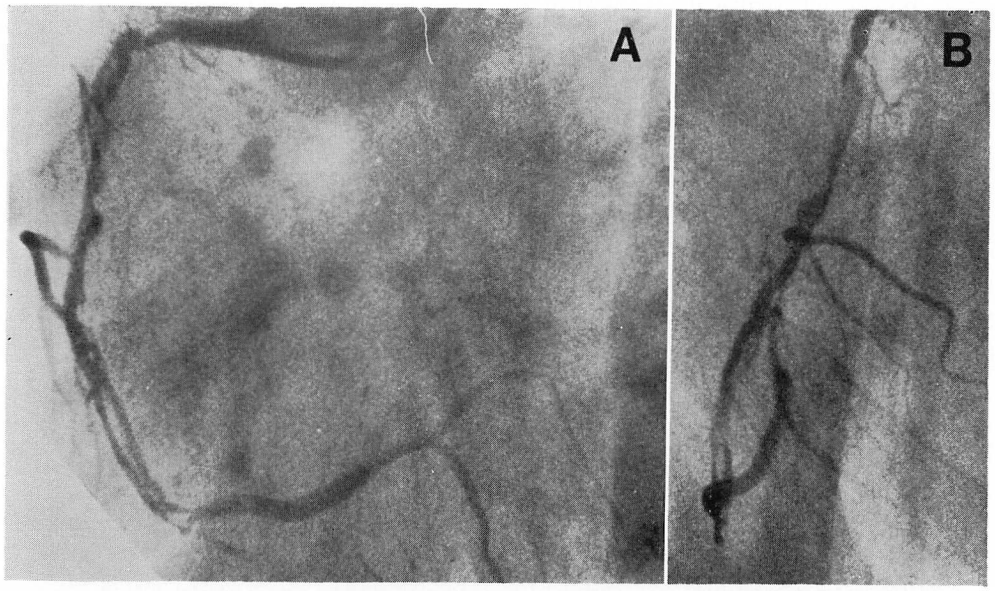

Fig. 2. First right coronary arteriograms obtained in Case 2.
A: LAO projection. B: RAO projection.
There are severe wall irregularities with dual lumina in the proximal to distal segments.

Case 2:

This 54-year-old man had a history of mild hypertension, heavy cigarette smoking, and frequent episodes of "crushing" chest pain, by which he was awakened from sleep during the previous six months. He had experinced no recent exertional chest pain, but on February 28, 1975, he experienced severe substernal pain after excessive ingestion of alcohol, and was examined at that time at a clinic where an inferior myocardial infarction was diagnosed. On March 4, 1975, he was referred to the Toranomon Hospital for further evaluation and treatment. His blood pressure was 150/92 mmHg; his heart rate, 74/min. The admission electrocardiogram revealed abnormal Q waves, and T wave inversions in the inferior leads. His hospital course was uneventful. On April 25, he underwent selective coronary angiography. His right coronary arteriogram (**Fig. 2**) showed marked wall irregularities with dual lumina in the proximal to distal segments. Left coronary arteriography was normal. Biplane left ventriculography demonstrated akinesis in the inferior segment.

On July 7, 1980, the patient underwent repeat cardiac catheterization, which showed evidence of an akinetic segment in the inferior wall. The left coronary arteriogram was normal. The previously noted right coronary arterial dissection had resolved with a well-demarcated perivascular pocket impinging on the wall of the vessel (**Fig. 3**).

On February 26, 1987, the patient underwent a third cardiac catheterization. A left ventriculogram revealed similar evidence of akinesis in the inferior wall. A left coronary arteriogram was normal. A right coronary arteriogram revealed further resolution of the dissection (**Fig. 4**). There was evidence of a vasospastic diathesis in both coronary arteries after the intravenous administration of 0.2 mg ergonovine (**Fig. 5**).

Twelve years after the myocardial infarction, he was classified as NYHA class I, and was being maintained on diltiazem.

Case 3:

This 46-year-old woman who had a history

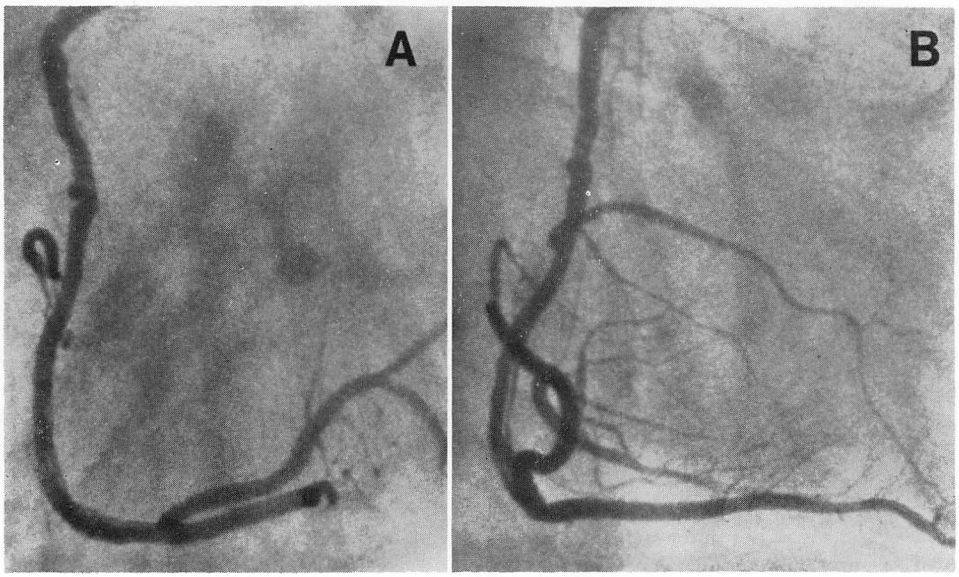

Fig. 3. Second right coronary arteriograms obtained in Case 2 five years later.
A: LAO projection. B: RAO projection.
The previously noted right coronary artery dissection has resolved markedly with a well-demarcated perivascular pocket impinging on the wall of the vessel.

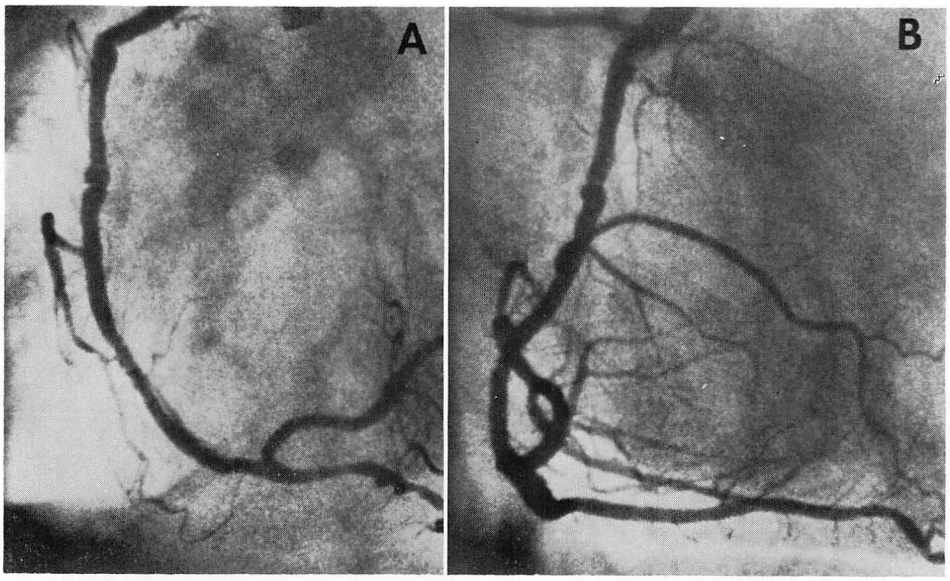

Fig. 4. Third right coronary arteriograms obtained in Case 2 twelve years later.
A: LAO projection. B: RAO projection.
They show further resolution of dissection.

of chronic cigarette smoking and atrial fibrillation, was admitted to a hospital on May 21, 1984, after the sudden onset of "choking" chest pain. Her admission electrocardiogram showed ST segment elevation in the inferior leads, and atrial fibrillation. On the first hospital day she refused further medication and discharged herself from the hospital. On October 26, 1984, she was examined at the Tornanomon Hospital because of chest discomfort after drinking alcohol. She was diagnosed as having atrial fibrillation and placed on digitalis therapy.

On April 22, 1986, she was admitted for further evaluation of her heart disease. She had no prior history of hypertension, diabetes or any lipid disorder, and no family history of coronary heart disease. Her physical examination revealed a healthy woman, 163 cm tall and weighing 59 kg. Her blood pressure was 114/82 mmHg; her pulse 78 /min and absolutely irregular. There were no significant murmurs. On April 24, she underwent selective coronary angiography. Her right coronary arteriogram showed dual lumina in the middle to distal segments, and the proximal portion of the posterior descending branch was separated by a flap (**Fig. 6**). Her left coronary arteriogram was free of any obstructive disease. Her left ventrculogram showed akinesis in the inferior segment.

One year after the cardiac catheterization she was asymptomatic, on digitalis.

Case 4:

This 28-year-old woman, gravida IV, para II, was admitted to a hospital because of loss of consciousness due to a toxemic pregnancy. She underwent a Caesarean section resulting in a stillbirth on August 9, 1976. The following day she experienced severe anterior chest pain and subsequent dyspnea. She was transferred to the emergency room where serial electrocardiograms and serum enzyme levels confirmed acute anterior myocardial infarction complicated by congestive heart failure, which responded favorably to medical therapy.

On December 6, 1976, she was referred to the Toranomon Hospital for further evaluation. She had a history of hypertension of 10 years

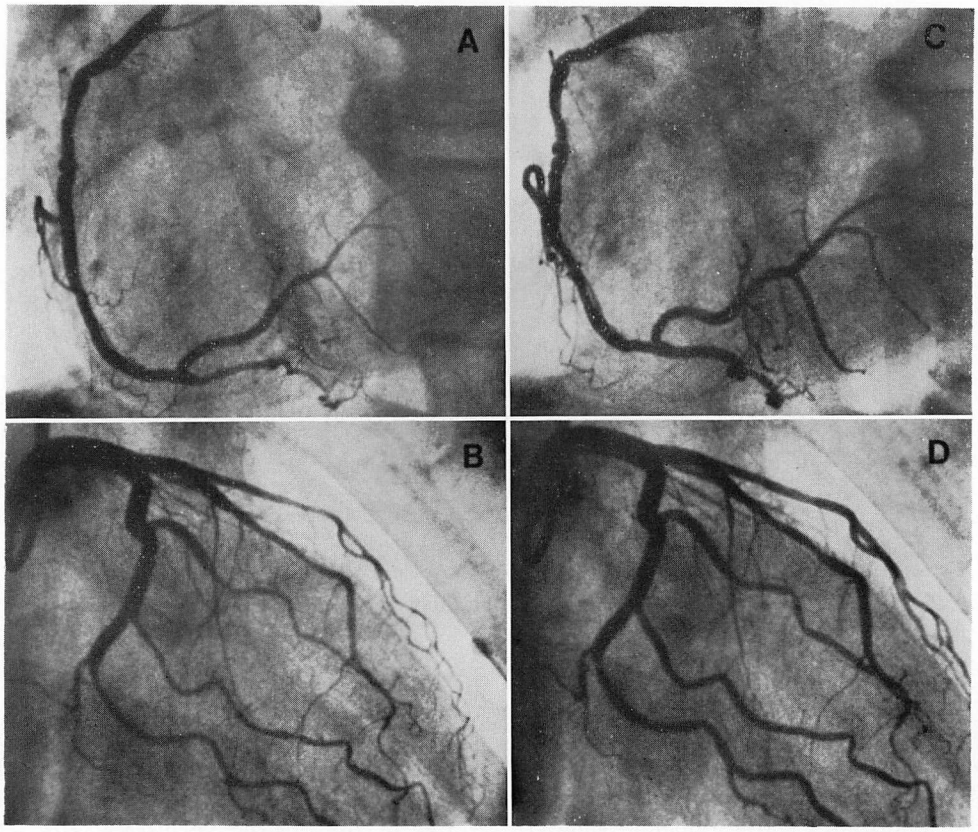

Fig. 5. Selective coronary arteriograms with ergonovine provocation test obtained in Case 2.

A: Right coronary artery in the LAO projection after intravenous injection of 0.2 mg ergonovine.
B: Left coronary artery in the RAO projection after intravenous injection of 0.2 mg ergonovine.
C: Right coronary artery in the LAO projection after sublingual administration of 0.3 mg nitroglycerin.
D: Left coronary artery in the RAO projection after sublingual administration of 0.3 mg nitroglycerin.
Right and left coronary arteriograms after intravenous injection of 0.2 mg ergonovine demonstrate vasospastic diathesis in comparison with those after sublingual administration of 0.3 mg nitroglycerin.

duration and mild hyperlipidemia. She had been diagnosed as having toxemic pregnancy three months before her myocardial infarction. On December 9, 1976, she underwent left-sided cardiac catheterization and selective coronary angiography. The left ventricular pressure was 162/37 mmHg. The right coronary artery was normal. The left coronary arteriogram showed a flap-line defect from the proximal to middle segments (**Fig. 7**). The left ventriculogram demonstrated akinesis in the anteroseptal segment, dyskinesis in the apical segment, and enlargement of the left ventricle.

The patient had ventricular arrhythmias after her myocardial infarction; however, she experienced no chest pain. On October 20, 1979, while attending her father's funeral, she suddenly died.

Discussion

Since Pretty's first description in 1931,[27] primary, spontaneous coronary artery dissec-

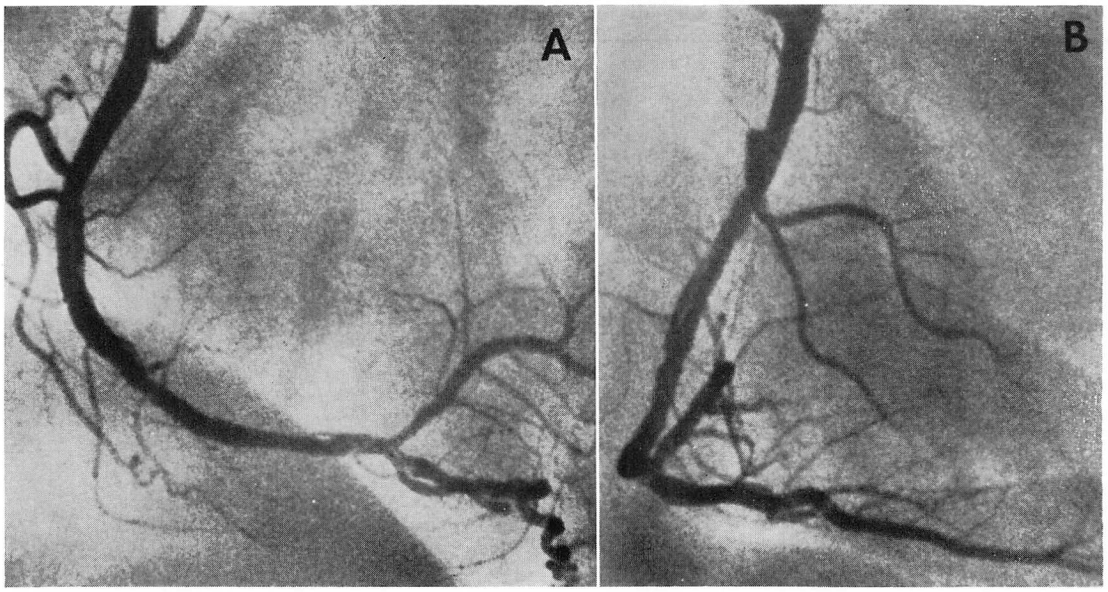

Fig. 6. Right coronary arteriograms obtained in case 3.
A: LAO projection. B: RAO projection.
There are dual lumina in the middle to distal segments and the proximal portion of the posterior descending branch separated by a flap.

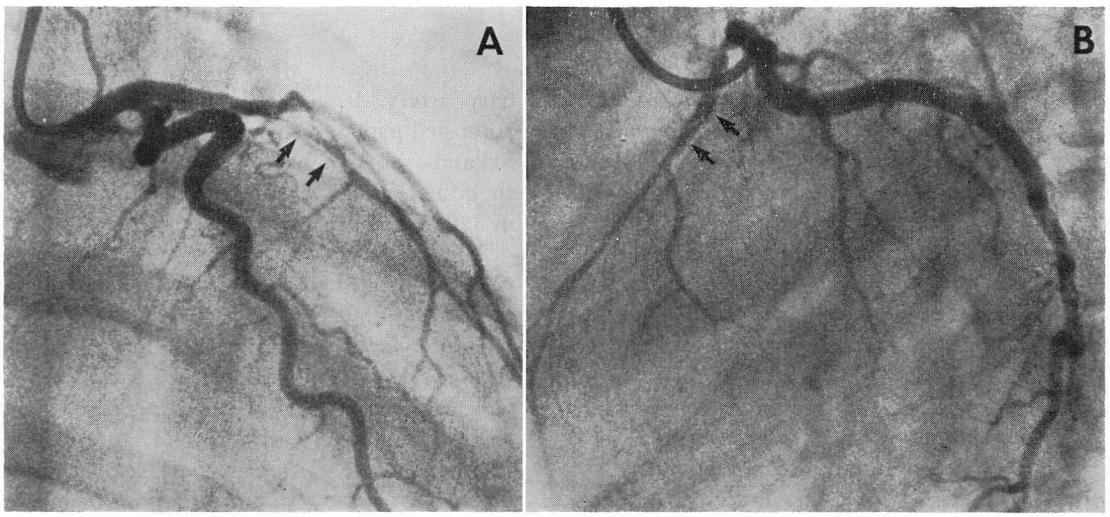

Fig. 7. Left coronary arteriograms obtained in case 4.
A: RAO projection. B: LAO porjection.
There is a flap-line defect in the proximal to middle segments (arrows).

Table 1. Primary coronary artery dissection studied by coronary angiograms

Case No.	Year	Authors	Patient Age/Sex	Postpartum (days)	Artery involved	Treatment
1	1973	Forker (26)	56 M		RCA	IMA implant
2	1975	Westbrook (28)	26 F	—	LAD	Medical
3	1975	Razavi (12)	31 F	6	LCA, LAD, CX	Graft plus aneurysmectomy
4	1977	DiMatteo (8)	59 F	—	RCA	Graft
5	1977	Palank (1)	31 M		RCA	Medical
6	1978	Shaver (14)	28 F	21	LCA, LAD, CX	Medical
7	1978	Ciraulo (29)	55 M		RCA	Medical
8	1980	Molloy (30)	42 F	—	LCA, LAD, CX	Medical
9	1984	Mathieu (31)	33 F	—	LAD	Medical
10	1985	Mark (32)	50 F	—	RCA	Medical
11	1985	DeJunco (33)	52 M		RCA	Graft
12	1985	Ramamurti (34)	42 F	—	LAD	Medical
13	1986	Bonnet (17)	32 F	12, 2 mos	CX; LAD	Graft
14	1986	Vicari (35)	33 F	14	LCA, LAD, CX	Clot extrusion
15	1987	Present case 1	54 M		RCA	Medical
16		Present case 2	28 M		RCA	Medical
17		Present case 3	46 F	—	RCA	Medical
18		Present case 4	28 F	1	LAD	Medical

M=male; F=female; RCA=right coronary artery; LAD=left anterior descending artery; LCA=left coronary artery; CX=circumflex artery; IMA=internal mammary artery; mos=months.

tion has been reported to be extremely rare. The earlier cases had been incidentally diagnosed at autopsy. The clinical picture was that of acute catastrophic coronary artery disease, which was divulged either by sudden death (62%) or by acute myocardial ischemia (38%).[31] More recently, the diagnosis has been made on clinical grounds. Fourteen earlier cases have been documented by coronary arteriography (**Table 1**). As in our patients, all of their diagnoses were made during coronary angiography as initial examinations of acute myocardial infarction. We encountered four patients (0.28%) with primary coronary artery dissection among 1,445 consecutive patients with myocardial infarction who were angiographically examined. Primary coronary artery dissection may occur more frequently than previously believed. Shin et al.[36] reported two cases of primary coronary artery dissection among 45 consecutive autopsied patients having cardiac diseases in the Sakurabashi Watanabe Hospital, Osaka. This suggests that this entity may be more frequent in Japan than elsewhere.

Angiographic features common to all previously reported dissections include: (1) a thin radiolucent line, i.e., a flap, indicating partial separation of the intima; (2) filling of the dissection sac; and (3) irregularity in caliber with widened and/or narrowed segments.[31] These changes may be reversible and, in follow-up studies, this is particularly evident in dissections when reentry occurs spontaneously.[29] In case 2, we observed angiographically, resolution to relative patency of the involved segment of the right coronary artery, and partial resolution of the dissection. These angiographic findings may reflect thrombosis, retraction and

dissolution of the dissection's hematoma in a manner similar to that observed angiographically in aortic dissection.

Histopathologically, arterial dissection is a lesion in which there are dissecting hemorrhage in the media, most often between the external lamina and the media, or in the outer third of the media.[37] Compression of the true lumen is secondary to the hematoma, and frequently to thrombosis. The major consequences of dissections are stenoses, thromboses and vascular occlusions with subsequent myocardial infarction.[4]

Various hypotheses have been offered to explain the etiology of primary coronary artery dissection. Hypertension does not seem to be a factor, since it is usually absent.[11] Pre-existing coronary arteritis is often discussed,[38] but this might simply be in response to the intramural hematoma, rather than an underlying condition.[11] Histologically, cystic medial necrosis has often been reported in primary coronary artery dissection.[18~20] This includes fragmentation of reticular fibers, a decrease of acid mucopolysaccharides, hyperplasia and hypertrophy of smooth muscle fibers, and disorganization of elastic fibers.[3] These histological alterations may play an important role in the dissection. Boschetti et al. accepted cystic medial necrosis as evidence of pre-existing brittleness of the media.[18] However, Morise et al cited the same histological features in three cases of iatrogenic coronary artery dissection.[25] Recently, Bonnet et al. observed that collagen synthesis was impaired in in vitro studies of skin fibroblasts from a postpartum woman with spontaneous coronary artery dissection.[16] Reduced collagen synthesis may be responsible for alterations in mechanical function of vessel walls, leading to spontaneous coronary artery dissection. The frequency with which these dissections develop in young women, particularly during advanced pregnancy or postpartum state as observed in Case 4, may lend support to the role of hormones, especially since the association of pregnancy and aortic dissection is well known.[31,39] Coronary atherosclerosis is apparently very uncommon among cases of primary coronary artery dissection, judging from previous cases and from our observation. The case reported by Lovitt et al is exceptional.[17]

Mark et al. reported a case with variant angina and angiographic evidence of spontaneous coronary artery dissection.[32] Except for that case, there has been no report of an association between variant angina and spontaneous coronary artery dissection. However, we observed preexisting angina at rest, suggestive of variant angina, in Cases 1 and 2, who had no exertional chest pain. Furthermore, the coronary arteriogram after intravenous ergonovine in Case 2 showed vasospastic diathesis in both coronary arteries. We, therefore, assume that coronary vasospasm may have been the primary event. Variant angina is reportedly more frequent in Japan, and this relatively high frequency may in turn contribute to the relatively high prevalence of primary coronary artery dissection in Japan.

There is no established treatment for primary coronary artery dissection because this disease has been lethal in most cases. The previously reported treatment is summarized in **Table 1.** Surgical intervention was used in six patients. An internal mammar yartery was implanted in one; bypass surgery and aneurysmectomy of the left ventricle in one; clot removal in one; and isolated bypasses in three cases. In the 12 remaining cases, including our four, only medical therapy was used.

This condition reportedly has a serious prognosis; in fact, in most cases it has been diagnosed at autopsy.[31] Recently, however, the diagnosis has been made in the living by coronary angiography. In one reported case with repeated coronary arteriography during 19 months post infarction, resolution of the dissection and return of luminal patency of the coronary artery were observed.[29] In our second case, second and third serial coronary arteriograms, six and 11 years post infarction, respectively, showed partial resolution of the dissection, without complete resolution of the intimal fiap. Long-term survival in this disease has not be described

yet. Excluding our postpartum patient who died of probable ventricular arrhythmia, our three cases are currently asymptomatic three to 12 years post-myocardial infarction. Thus, the prognosis in this disease may be better than was previously believed.

Conclusion

Primary coronary artery dissection was observed in four (0.28%) of 1,445 consecutive patients with myocardial infarction angiocardiographically examined. The right coronary artery was involved in three patients; the left coronary artery, in one. Two of these four patients had pre-existing angina at rest, suggestive of vasospastic angina. Except for one postpartum patient, three of the four patients have good prognoses.

要　　約

原発性冠動脈解離の頻度，発症様式ならびに予後に関する検討

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原発性冠動脈解離の頻度，発症様式ならびに予後に関する検討を行った．

心血管造影にて心筋梗塞と診断された連続1,445例中，4例（0.28%）に原発性冠動脈解離を認めた．

症例：(1) 28歳，男性．3週間の安静時狭心症後，下壁梗塞発症．右冠動脈 Seg. 1-3 の解離と下壁の akinesis を認めた．6年後の NYHA I.

(2) 54歳，男性，7ヵ月の安静時狭心症後，下壁梗塞発症．右冠動脈 seg. 1-3 の解離と下壁の akinesis を認めた．5年後の再検査にて解離の著明な治癒傾向を認め，12年後施行された ergonovine 負荷試験にて冠スパスムを認めた．現在，NYHA I.

(3) 46歳，女性，朝食後，突然胸痛きたし，下壁梗塞発症．右冠動脈 Seg. 2-3, 4PD の解離と下壁の akinesis を認めた．3年後現在，NYHA I.

(4) 28歳，女性，妊娠中毒症にて死児娩出後，前壁梗塞発症．左冠動脈 Seg. 6-7 の解離と前壁の akinesis および心尖部の dyskinesis を認めた．4年2カ月後，突然死した．

心筋梗塞の冠動脈病変において，原発性冠動脈解離は 0.28% に見られ，発症様式から明らかに冠スパスムの関与が示唆されるものがあった．予後は，出産に関する1例を除き良好であり，再造影にても解離の治癒傾向を認めた．

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