Original Article

PCI

Acute Type A Aortic Dissection Presenting as ST-Segment Elevation Myocardial Infarction Referred for Primary Percutaneous Coronary Intervention

Jian-Liung Wang,[1]* Chun-Chi Chen,[2]* Chao-Yung Wang,[2] Ming-Jer Hsieh,[2] Shang-Hung Chang,[2] Cheng-Hung Lee,[2] Dong-Yi Chen[2] and I-Chang Hsieh[2]

Background: When acute aortic dissection is complicated with acute myocardial infarction, the diagnosis of dissection can be problematic. In these cases, patients might be treated with primary percutaneous coronary intervention (PCI) and suffer acatastrophic outcome. However, there are few reports or algorithm to facilitate the accurate management of this clinical situation.

Methods: We evaluated 385 consecutive patients who underwent primary PCI arising from an initial diagnosis of STEMI at our hospitalbetween January 2006 and March 2011. Clinical characteristics, coronary angiographic findings, and outcomes were obtained from medical charts and databases.

Results: Five patients (1.3%) with STEMI secondary to aortic dissection were identified. All patients (100%) had sudden-onset of chest pain and a history of hypertension without diabetes or hyperlipidemia. An increased resistance while advancing the diagnostic catheter was reported by the operators in 3 of 5 patients (60%). Aortography performed by manual contrast-medium injection showed the discrepancy in the diameter between the aortic root and the ascending aorta in 4 patients (100%), and ascending aortic intimal flap dissections were noted in 3 patients (75%). Alternating appearance and disappearance of the coronary artery ostium was observed in 2 patients, and bedside echocardiography showed intimal flap extension inall 4 patients (100%) who underwent this examination. The mortality rate at 30days was 40%.

Conclusions: We construct an algorithm that incorporated factors including careful history evaluation, bedside echocardiography, resistance encountered while advancing a catheter, and findings of aortography performed with manual injection,which could b evaluable for this clinical situation.

[1]Section of Cardiology, Department of Medicine, Landseed Hospital; [2]Section of Cardiology, Department of Medicine, Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Taoyuan, Taiwan.
Address correspondence and reprint requests to: Dr. I-Chang Hsieh, Section of Cardiology, Department of Medicine, Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Percutaneous Coronary Intervention Center of Chang Gung Memorial Hospital, No. 199, Tung Hwa North Road, Taipei, Taiwan. Tel: 886-3-328-1200 ext. 8115; Fax: 886-3-328-1451; E-mail: jlwangkl@gmail.com
*These authors contributed equally to this project and should be considered co-first authors.

INTRODUCTION

Rapid and accurate diagnosis of ST-segment elevation myocardial infarction (STEMI) is crucial for effective treatment. While serious efforts have been made to shorten door-to-balloon time,[1,2] some patients who are initially diagnosed with STEMI actually have conditions other than myocardial infarction, such as Brugada syndrome, subarachnoidal hemorrhage, myocarditis, pericarditis and aortic dissection.[3] Acute myocardial infarction, related to the extension of the dissection flap into

the ostium of the coronary artery, develops in 1-2% of cases with acute type A aortic dissection. Typically, myocardial infarction develops more frequently than aortic dissection in a clinical setting. Therefore, when acute dissection is complicated with acute myocardial infarction, the correct diagnosis of dissection can be elusive and these patients might instead be treated with primary percutaneous coronary intervention (PCI).[4] In these cases, the outcome can be catastrophic.[5]

The current American College of Cardiology/American Heart Association (ACC/AHA) guidelines for the management of patients with STEMI recommend that the possibility of aortic dissection be excluded in the initial patient evaluation, when a patient's preliminary medical history and imaging results suggest that a differential diagnosis may be appropriate.[6] Although case reports have described these aortic dissection circumstances in patients and their associated high mortality rate, there has been no clearly presented clinical picture with systematic analysis for these patients, and the angiographic procedure in this clinical scenario has not been fully explored as well.[7-11] Here, we report the incidence, clinical characteristics, coronary angiographic findings, peri-procedural adjunctive tools, and outcomes in patients who received primary PCI because of an initial diagnosis of STEMI, but who were eventually diagnosed with aortic dissection; this study offers a reasonable clinical approach through use of an effective algorithm that could reduce such misdiagnosis and subsequent PCI treatment.

MATERIALS AND METHODS

The Cardiovascular Atherosclerosis and Percutaneous Transluminal Interventions (CAPTAIN) registry is a prospective, physician-initiated, single-center observational database that has been maintained since November 1995. Patients who had STEMI and received primary PCI between January 2006 and March 2011 were evaluated for initial screening from this registry. ST elevation was defined as the presence of ST segment elevation ≥ 1 mm in two or more contiguous leads in limb or precordial leads. Primary PCI is a catheter-based reperfusion therapy and was performed by a trans-femoral approach in the majority of cases; a trans-radial ap-

proach was used in some stable cases. Medical professionals were placed on stand-by status in the hospital and patients were moved to the cardiac catheterization laboratory where they underwent angiography without delay. According to the recommendations in ACC/AHA guidelines, we ensured that the door-to-balloon time of primary PCI was less than 90 minutes whenever possible. If patients were later diagnosed with aortic dissection, their detailed history, laboratory data, and coronary angiographic images were reviewed retrospectively. Stanford type A aortic dissection was defined as the involvement of the ascending aorta and aortic arch, proximal to the left subclavian artery. In these patients with type A aortic dissection, the diagnostic and therapeutic modality, coronary angiographic findings and in-hospital mortality rate were obtained. This study was approved by the Institutional Review Board of this hospital.

RESULTS

In this study, we evaluated 385 consecutive patients who underwent primary PCI because of an initial diagnosis of STEMI at our hospital between January 2006 and March 2011. A total of 380 patients were diagnosed with STEMI without dissection, and 88.4% was male. Hypertension was found in 172 patients (45.3%), 76 patients (20%) with diabetes mellitus and 177 patients (46.6%) with smoking. Among this study group, the overall mortality rate was 2.6%. In that group of patients with actual STEMI, patients had less hypertension and mortality, more hyperlipidemia and more coronary artery disease than those patients with aortic dissection (Table 1).

A final diagnosis of acute type A aortic dissection was made in 5 patients (1.3%; 3 men and 2 women; Table 2). All 3 male patients were less than 50 years of age, and the female patients were between 63 and 72 years of age. All of the 5 patients (100%) had a history of hypertension without diabetes or dyslipidemia, and 2 patients (40%) had a history of smoking.

All 5 patients (100%) presented with sudden chest pain. However, no radiating pain to the back was reported. One patient (20%) had out-of-hospital cardiac arrest and received successful cardiopulmonary cerebral

resuscitation (CPCR). Hypotension with mean arterial pressure less than 60 mmHg or systolic blood pressure

Table 1. Comparison of clinical characteristics between patients with STEMI and aortic dissection with STEMI

	STEMI (n = 380)	Dissection with STEMI (n = 5)	p value
Age (%)	60.3 ± 13.4	53.8 ± 13.0	NS
Male (%)	336 (88.4)	3 (60)	NS
Hypertension (%)	172 (45.3)	5 (100)	< 0.05
Diabetes (%)	76 (20)	0	NS
Family history (%)	8 (2.1)	0	NS
Current smoker (%)	177 (46.6)	2 (40)	NS
Hyperlipidemia (%)	178 (47.7)	0	< 0.05
CAD (%)	379 (99.7)	2 (40)	< 0.05
Culprit lesion			
LM	6 (1.6)	0	NS
LAD	214 (56.3)	1 (20)	NS
LCX	30 (7.9)	0	NS
RCA	129 (33.9)	1 (20)	NS
Coronary spasm	1 (0.2)	0	NS
In-hospital mortality	10 (2.6)	2 (40)	< 0.05

LAD, left anterior descending artery; LCX, left circumflex artery; LM, left main; RCA, right coronary artery; STEMI, ST-segment elevation myocardial infarction.

less than 90 mmHg was noted in 4 patients (80%). Ventricular tachycardia (VT) developed in 2 patients (40%). ST-segment elevation over inferior leads (Lead II, III, aVF) and precordial leads (Lead V1 to V6) was detected in 1 patient (20%, Figure 1) and 4 patients (80%), respectively. Thereafter, portable anterior/posterior (AP) chest radiographs was obtained before primary PCI, but mediastinal widening was equivocal in 3 patients (60%) and normal mediastinal diameter with normal cardiac contour was noted in 2 patients (40%).

Primary PCI was performed via a trans-femoral approach in all 5 patients. In three of the patients (60%), operators found an increased resistance while advancing the catheter within the aorta retrogradely. Therefore, selective coronary angiography was stopped in these patients because of suspected aortic dissection. Aortography was immediately performed by manually injecting contrast medium (20 cc) through a pigtail catheter which revealed dissection with initimal flap over the ascending aorta. A total of 4 patients underwent manual aortographic study, including one patient with persistent chest pain and unstable hemodynamics after percutaneous transluminal coronary angioplasty (PTCA).

Table 2. Clinical characteristics and outcomes of patients

|	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age/sex	46/M	46/M	63/F	72/F	42/M
ECG leads of ST elevation	V1-6+VT	III, aVF, V5-6	V5-6, aVL	V1-2, III	V1-2+VT
BP(MAP) mmHg	94/29 (50)	79/49 (59)	88/50 (62)	80/56 (64)	116/63 (80)
Diabetes mellitus/hypertension	-/+	-/+	-/+	-/+	-/+
Dyslipidemia/smoking	-/+	-/-	-/-	-/-	-/+
Abrupt chest pain	Yes	Yes	Yes	Yes	Yes
Advancing resistance of catheter	Yes	Yes	No	No	Yes
Manual aortogram	+	+	+	Not done	+
Ratio of root/AsAo	1.46 (true lumen)	0.68 (false lumen)	2.27 (true lumen)	Not done	1.9 (true lumen)
Intimal flap	+	+	-	Not done	+
Selective coronary angiography	-	-	+	+	-
Coronary artery disease	Grossly normal in aortography	Grossly normal in aortography	Yes, LAD 60-70% with intervention	Yes, RCA 40-50% without intervention	Grossly normal in aortography
Alternating appearance and disappearance of coronary arteries			+	+	
Intimal flap in echocardiography	+	+	Not done	+	+
Other diagnostic tool	CT	CT	TEE	CT	CT
Surgery	No	Yes	Yes	Yes	Yes
Outcome	Mortal	Survival	Mortal	Survival	Survival

AsAo, ascending aorta; CT, computed tomography; LAD, left anterior descending artery; RCA, right coronary artery; TEE, transesophageal echocardiography; VT, ventricular tachycardia.
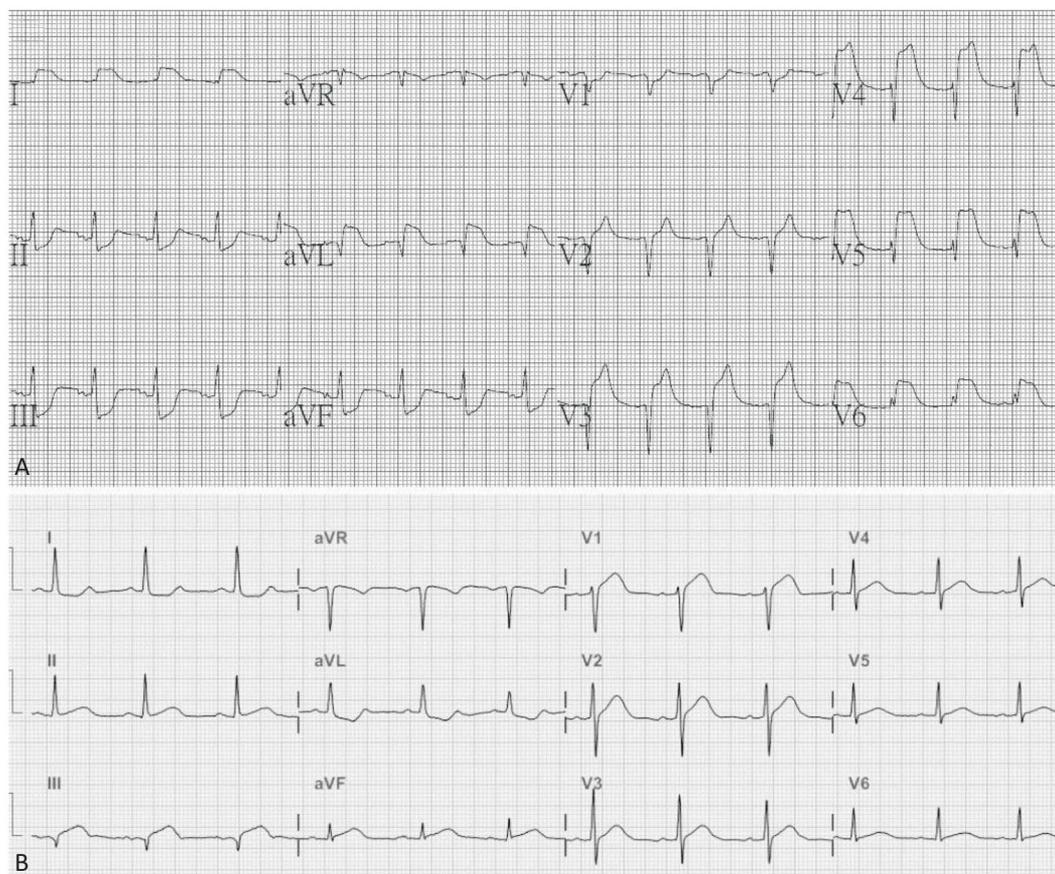
Figure 1. *ST-segment elevation over precordial leads (Lead V1 to V6) in upper ECG (A) and inferior leads (Lead III, aVF) in lower ECG (B).*

Aortography also revealed a discrepancy in the diameter between the aortic root and the ascending aorta. The ratios of the diameter of the aortic root to those of the ascending aorta were 1.46, 0.68, 2.27 and 1.9 in the 4 patients (Figure 2, A and B). Three of the 4 patients ratio were greater than 1.0, and the diagnostic catheter tips were placed in the true aortic lumen in all 3 patients. One patient whose ratio was less than 1.0 had the catheter tip placed in the false lumen.

In the 3 patients who did not receive selective coronary angiography, their manual aortography showed that coronary arteries were grossly normal without focal stenosis or total occlusion. Selective coronary angiography was performed on the other 2 patients. One of the two patients received PTCA without stent implantation. However, chest pain with unstable hemodynamic status persisted which led the operator to suspect aortic dissection. Therefore, manual aortogram was performed and showed a discrepancy in the diameter between the
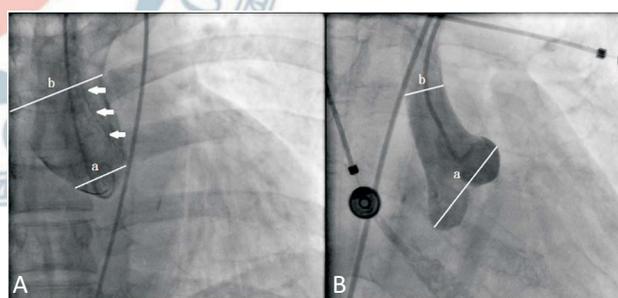
Figure 2. *(A) In the second patient, manual aortography revealed a large intimal flap (arrowhead) from the aortic root to the ascending aorta. The ratio of the diameters of the aortic root (a) and ascending aorta (b) is 0.68 (a = 26.0 mm and b = 38.0 mm); it indicated that the pigtail catheter is placed in the false lumen. (B) In the third patient, the ratio of the diameters of the aortic root and ascending aorta is 2.27 (a = 35.7 mm, b = 15.7 mm); it indicated the catheter is placed in the true lumen.*

aortic root and the ascending aorta. This patient was sent for emergency surgery where transesophageal echocardiography revealed type A aortic dissection. In

the other one patient (the fourth patient), hypotension with mild coronary artery plaques in selective coronary angiography led the operator to consider the possibility of aortic dissection. Emergency bedside echocardiography was performed and showed intimal flap in the ascending aorta. In this study, bedside transthoracic echocardiography was done in 4 patients and all showed an intimal flap over the aortic root (100%). In these 2 patients, coronary angiography was reviewed afterwards and showed alternating appearance and disappearance of coronary artery ostium due to compression by the dissected false lumen (Figure 3).

The diagnosis of acute type A aortic dissection in 4 patients was confirmed by computed tomography (CT), and one patient was diagnosed by means of intraoperative transesophageal echocardiography.

Thereafter, the standard Bentall operation was performed on the four patients and two expired (mortality rate: 40%).

DISCUSSION

In patients with STEMI, primary PCI is the most effective myocardial reperfusion therapy. In these cases, the ACC/AHA guidelines recommend that the door-to-balloon time must be less than 90 min for treatment to be most effective. Therefore, the processing time of primary PCI, including the door-to-ECG time, the door-to-catheterization room time, and puncture-to-balloon time, should be as short as possible. Patients who are diagnosed as STEMI by emergency department (ED) physicians are often rushed to the catheterization room for primary PCI. However, these patients may actually have an aortic dissection. In this study, the incidence of aortic dissection manifesting as STEMI was 1.3% in patients receiving primary PCI.

Acute aortic dissection is the most catastrophic event that can affect the aorta, and the associated early mortality rate is very high, with 1-2% per hour in the first few hours after the occurrence of dissection.[4] Early diagnosis is essential for survival; however, early recognition of the condition is challenging for primary physicians because the symptoms of aortic dissection can be variable and may mimic those of common conditions. The current ACC/AHA guidelines classify the targeted
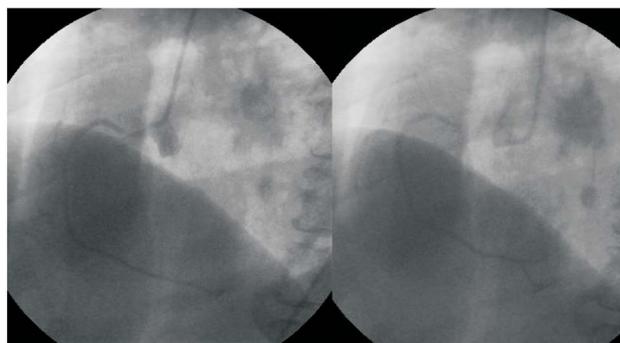
Figure 3. *Aortography showed alternating appearance and disappearance of coronary artery ostium because of compression by the dissected false lumen. Coronary angiogram at the left side showed proximal RCA stenosis, and subsequent disappearance of RCA ostium resulted from compression by dissecting flap in the same scene as was shown at the right side. RCA, right coronary artery.*

obtaining of patient history as a class I recommendation and suggest that aortic dissection should be considered in the initial evaluation of STEMI.[6]

Typical chest pain in acute MI includes sudden and prolonged substernal chest pain, and might present simultaneously with sweating, dyspnea, radiating pain to the left arm or neck. Some patients might have recent effort-related chest tightness. Typical chest pain in aortic dissection includes sudden severe chest pain described as tearing, stabbing, sharp in character or radiating pain to the back. However, sometimes it is difficult to differentiate the two conditions clinically because both diseases manifest as sudden chest pain, and pain radiating from chest to back occurs in approximately 17% of aortic dissection patients. All the patients in our study presented with sudden severe chest pain without tearing or radiation pain.

Careful and accurate history taking in STEMI patients with strongly suspected acute aortic dissection can assist physicians in obtaining early and correct diagnoses. More importantly, if the patient had the history of hypertension, this can be a strong indication of possible acute aortic dissection. However, onset or severity of chest pain may not always provide sufficient information to exclude aortic dissection in the ED room.

Acute aortic dissection with extension of a dissection flap into the ostium of the coronary artery has been more commonly reported in an older population, and in the right coronary artery.[4] However, in this series, 3 patients were male and less than 50 years of age. Four pa-

tients had electrocardiographic ST-segment elevation in the precordial leads, which might be secondary to the compression of the left main coronary artery by the dissected intimal flap.

The ACC/AHA guidelines also recommend patients with STEMI should have a portable X-ray,[6] but a widening mediastinum could be inconclusive from the chest A-P view. In this study, CT was used to definitively diagnose aortic dissection. Recent advances in CT have made it possible to differentiate the three potentially fatal conditions – aortic dissection, pulmonary embolism and myocardial infarction – those manifest as acute chest pain.[12]

Transthoracic echocardiography is recommended for differentiating STEMI from aortic dissection in patients in whom this distinction is initially unclear on the basis of medical history or chest radiography.[13] We recommend that, if possible, clinicians perform echocardiographic study for aortic dissection before performing primary PCI. Moreover, even if the ED physicians do not suspect aortic dissection, echocardiography is still helpful during catheterization.

During the primary PCI procedure, any resistance noted while advancing the diagnostic catheter may be helpful in the diagnosis of aortic dissection. If the catheter is placed in the true aortic lumen, the movement of the catheter is free and smooth. However, if the catheter is placed in the false lumen, the advancement of the catheter is laborious and slow, like "walking through sludge". Therefore, aortic dissection should be considered if resistance is encountered while advancing the catheter.

Aortography with manual injection of the contrast medium through a pigtail catheter is effective for detecting aortic dissection. The presence of an intimal flap and discrepancy in the diameter between the aortic root and ascending aorta from an aortogram are crucial for accurate diagnosis of aortic dissection. On the basis of our findings, we believe that if the pigtail catheter is placed in the true lumen, the ratio of the diameters of the aortic root to that of the ascending aorta will be greater than 1.0. In contrast, if the pigtail catheter is placed in the false lumen, the ratio will be less than 1.0. These differences in ratios occur because the dissected false lumen of the ascending aorta dilates and compresses the true lumen. Therefore, if the pigtail catheter

is placed in the true lumen of the aortic root, the ascending aorta compressed by the dissected lumen will be narrowed, thus resulting in a diameter ratio of greater than 1. We also found that the alternating appearance and disappearance of the coronary artery on aortography or coronary angiography is a sign of aortic dissection, because the artery will be compressed by the dissected intimal flap.

Once STEMI resulting from type A aortic dissection is confirmed, the customary definitive therapy is surgical correction. Successful left main coronary artery direct stenting as a lifesaving procedure was even reported, allowing for subsequent operation.[14]

We also created a new algorithm (Figure 4) for further management of this clinical challenge. First, it is imperative to carefully evaluate clinical history, including
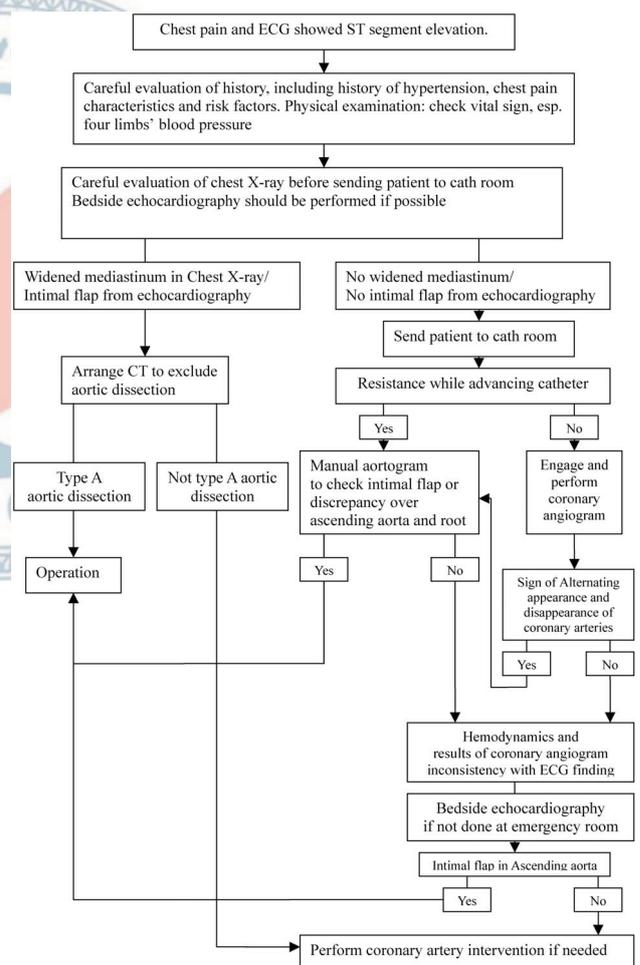

Figure 4. Algorithm for all language within the boxes, make certain only first word is capitalized. CT, computed tomography; ECG, electrocardiogram.

history of hypertension, chest pain characteristics and risk factors in patients presenting with ST elevation. It is also helpful to check the four limbs' blood pressure, especially if there are signs of obvious difference. Second, it is important to review chest X-ray before sending patients to the catheterization room. Furthermore, it is appropriate to defer the catheterization examination and arrange a timely CT if a widened mediastinum is noted. Third, it is important that the operating physician or other professional keep the diagnosis of aortic dissection in mind if resistance is noted while advancing the catheter. Manual aortographic study should be done first if aortic dissection is suspected. If the procedure is smooth and coronary angiography is then performed, the signs of alternating appearance and disappearance of the coronary arteries would likely provide the operator a impression of aortic dissection. Finally, if the hemodynamics and results of coronary angiogram are inconsistent with ECG changes, the operator should perform a bedside echocardiography to evaluate the possibility of aortic dissection.

There were certain limitations to our study. First, our study population was small, and thus it is very difficult to draw very firm conclusions from such modest patient numbers. Second, some patients with concomitant type A aortic dissection and STEMI could expire before arriving at the hospital. Additionally, some patients might have a correct diagnosis yet are still not included in our study due to typical dissecting chest pain and trivial ECG changes. Moreover, not every primary PCI case underwent a more comprehensive review for the presence of concomitant type A aortic dissection. In other words, some undiscovered type A dissection cases may exist, especially for those who died during admission. The actual rate of concomitant type A dissection may be underestimated. Third, all five reported cases were performed via a trans-femoral approach in this study. However, an increasing number of primary PCIs are performed via the trans-radial approach. Therefore, further experience and reports should be investigated in these patients. Further large population study, especially from the big data of national health insurance database, to analyze the clinical characteristics, incidence, angiographic findings and mortality might be helpful to cope with this clinical problem.

Currently, substantial effort is underway to improve the door-to-balloon time in PCI. Many operators might not have the experience with or even the awareness of aortic dissection due to atypical presentation, and borderline widened mediastinum in Chest X-ray may not lead a physician to perform CT. Therefore, further effort should be made to reduce the mortality rate in this patient group. We suggest that the diagnosis of aortic dissection should be kept in mind while taking patient history even if the ECG is very typical for STEMI. And while advancing the diagnostic catheter, the tactile sense of advancing resistance might give operators a hint of possible aortic dissection. If aortic dissection is strongly suspected during catheterization, manual aortogram might give operators more information, such as alternating appearance and disappearance of coronary arteries and discrepancy of the diameter between the aortic root and the ascending aorta. If STEMI caused by type A aortic dissection is confirmed during catheterization, prompt cardiovascular surgery without PCI for stenotic lesions might preserve more time for patients to undergo surgery (Figure 4 algorithm).

CONCLUSIONS

In conclusion, knowledge of pain characteristics, bedside echocardiography, resistance encountered while advancing a catheter, and findings of aortography performed with manual injection are helpful in the diagnosis and management of type A aortic dissection manifesting as STEMI.

REFERENCES

1. Sekulic M, Hassunizadeh B, McGraw S, et al. Feasibility of early emergency room notification to improve door-to-balloon times for patients with acute ST segment elevation myocardial infarction. *Catheter Cardiovasc Interv* 2005;66:316-9.
2. Ting HH, Rihal CS, Gersh BL, et al. Regional systems of care to optimize timeliness of reperfusion therapy for ST-elevation myocardial infarction. *Circulation* 2007;116:729-36.
3. Gu YL, Svilaas T, van der Horst IC, et al. Conditions mimicking acute ST-segment elevation myocardial infarction in patients referred for primary percutaneous coronary intervention. *Neth Heart J* 2008;16:325-31.
4. Elliott MA, David AM. Disease of the aorta. Braunwald's heart disease. 9th Ed., Chapter 60, 1319-23.

5. Cook J, Aeschlimann S, Fuh A, et al. Aortic dissection presenting as concomitant stroke and STEMI. *J Hum Hypertens* 2007;21: 818-21.

6. Kushner FG, Hand M, Smith SC, et al. 2009 Focused Updates: ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction (Updating the 2004 Guideline and 2007 Focused Update) and ACC/AHA/SCAI Guidelines on Percutaneous Coronary Intervention (Updating the 2005 Guideline and 2007 Focused Update). *Circulation* 2009;120:2271-306.

7. Sir JJ, Kim YI, Cho WH, et al. Acute myocardial infarction due to aortic dissection. *Intern Med* 2009;48:173.

8. Camaro C, Wouters NT, Gin MT, et al. Acute myocardial infarction with cardiogenic shock in a patient with acute aortic dissection. *Am J Emerg Med* 2009;27:899.e3-6.

9. Tarver K, Kindler H, Lythall D. Extensive aortic dissection presenting as acute inferior myocardial infarction. *Heart* 2007;93:1225.

10. Zegers ES, Gehlmann HR, Verheugt FW. Acute myocardial infarction due to an acute type A aortic dissection involving the left main coronary artery. *Neth Heart J* 2007;15:263-4.

11. Horszczaruk GJ, Roik MF, Kochman J, et al. Aortic dissection involving ostium of right coronary artery as the reason of myocardial infarction. *Eur Heart J* 2006;27:518.

12. Michael JG, Gilbert LR. Use of multislice CT for the evaluation of emergency room patients with chest pain: the So-called "Triple Rule-Out". *Catheter Cardiovasc Interv* 2008;71:92-9.

13. Luo JL, Wu CK, Lin YH, et al. Type A aortic dissection manifesting as acute myocardial infarction: still a lesson to learn. *Acta Cardiologica* 2009;64:499-504.

14. Melinda B, Gilbert G, Jacques C, et al. Left main stenting-as a bridge to surgery-for acute type A aortic dissection and anterior myocardial infarction. *Catheter Cardiovasc Interv* 2000;51:74-7.