

Isolated posterior acute myocardial infarction presenting to an emergency department: diagnosis and emergent fibrinolytic therapy

到急症室求診的單一急性後心肌梗塞：診斷及緊急纖維蛋白溶解治療

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Objectives: Isolated posterior acute myocardial infarction (AMI) is rare and possibly underdiagnosed. The incidence of misdiagnosis in the emergency department (ED) is unknown. Delayed diagnosis may prevent timely treatment, particularly emergent fibrinolytic therapy. We describe the experience of an urban ED on this rare condition. **Methodology:** A six years and seven months case series of isolated posterior AMI of initial presentation (as identified by inpatient discharge/death ICD-9-CM diagnosis code) was studied. Patients not admitted from the ED, those who developed isolated posterior AMI only after admission and/or those with concomitant ST segment elevation AMI involving other anatomical locations of the heart (e.g. inferior or lateral walls), were excluded. **Results:** Eleven cases were included in the study. All the nine cases with electrocardiograms available for review demonstrated features consistent with isolated posterior AMI. Eight out of the eleven (72.7%) cases were correctly diagnosed as isolated posterior AMI in the ED. The other three cases were treated as non-ST elevation myocardial infarction (NSTEMI). Nevertheless, their lack of the typical symptoms of acute coronary syndrome and delayed presentation (more than 12 hours) precluded them from fibrinolytics. Three of the eleven cases received fibrinolytics (all streptokinase). All three cases survived to discharge and there were no haemorrhagic complications. None of the cases underwent emergent percutaneous coronary intervention. **Conclusion:** The majority of cases with isolated posterior AMI (72.7%) were diagnosed in the ED. Although three cases were interpreted as NSTEMI, the use of fibrinolytic reperfusion therapy was not affected. (Hong Kong j.emerg.med. 2008;15:27-35)

目的：單一的急性後心肌梗塞是罕見的及有可能診斷不出。急症室的誤診率不詳。延誤診斷可能妨礙及時的治理，特別是緊急纖維蛋白溶解治療。本文描述一所市區急症室對這罕見病症的經驗。**方法系統：**這研究為期六年零七個月，是一個包括最初呈現單一急性後心肌梗塞的病人個案系列（由住院病人出院／死亡時的 ICD-9-CM 診斷代碼識別），並排除不是經急症室入院的病人，入院後才呈現單一急性後心肌梗塞的病人，及伴隨有涉及其他心臟體位 ST 段提升急性心肌梗塞的病人（例如：下方或側面心肌壁）。**結果：**研究包括 11 個案。其中 9 個可以提供心電圖作審查的個案，都一致顯示單一急性後心肌梗塞的特徵。11 個案的其中 8 個（72.7%）在急症室正確地診斷為單一急性後心肌梗塞。其他 3 個個案作「非 ST 段提升心肌梗塞」治理。然而，他們沒有急性冠動脈綜合徵的典型症狀及延遲就診（超過 12 小時），排除了他們接受纖維蛋白溶解劑的治療。11 個案的其中 3 個接受了纖維蛋

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白溶解劑（全部是鏈球菌激酶）。所有 3 個個案生存至出院及沒有出血的併發症，沒有個案接受緊急經皮冠動脈介入治療。**總結：**大多數的單一急性後心肌梗塞個案（72.7%）都在急症室診斷出。雖然 3 個個案被判斷為「非 ST 段提升心肌梗塞」，但沒有影響使用纖維蛋白溶解劑，回復灌流的治療。

Keywords: Coronary disease, fibrinolytic agents

關鍵詞：冠心病、纖維蛋白溶解劑

Introduction

Acute posterior wall myocardial infarction occurs in up to 20% of acute myocardial infarctions (AMIs), with the vast majority occurring along with inferior or lateral AMI.^{1,2} However, isolated posterior AMI is rare and possibly underdiagnosed. As an urban emergency department (ED), we were concerned about the incidence of misdiagnosis as delayed diagnosis may prevent timely treatment, particularly emergent fibrinolytic therapy. Through a case series, we describe the experience of an urban ED on this rare condition.

Subjects and methods

True posterior wall myocardial infarction cases between 20 May 1999 and 31 December 2006 were identified by searching from the hospital's inpatient discharge summaries using ICD-9-CM diagnosis code 410.6 (true posterior wall AMI) and 410.61 (true posterior wall AMI, initial presentation). This method of case selection using inpatient discharge diagnosis would have missed out those cases which were seen in the ED but not admitted due to death, at-own-risk discharge or transferred to another hospital. A review of duplicate death certificates dispensed from the ED as well as the ED's computerised documentation records of AMI cases seen within the study period was conducted to ensure all cases of true posterior AMI were collected.

The ED was staffed by board certified emergency physicians (EPs) and medical officers (MOs). MOs would routinely approach the floor EP-on-duty for advice on interpreting ECG. The clinicians were not required to follow any specific ECG criteria for diagnosis of isolated acute posterior AMI. Suspicious

or equivocal cases would be discussed with the cardiologist-on-duty by telephone consultation and faxing of ECG. The initiation of thrombolysis for AMI would follow the cardiologist's opinion. Patients who presented within 12 hours of symptom onset with ECG changes would have fulfilled the criteria for consideration of fibrinolytic therapy.

Information collected from the case series for analysis included basic patient demographics, initial ED ECG characteristics, cardiac enzyme results, usage of fibrinolytic and incidence of haemorrhagic complications, survival to discharge, and cardiac catheterisation findings.

All calculations and description of variables were performed by using SPSS version 10.0 (SPSS Inc, Chicago, IL).

Results

Thirty-four hospitalised cases were selected by ICD coding. Subsequently, those patients not admitted from the ED (none), those who developed posterior AMI only after admission (3 cases) and/or those with diagnosis of concomitant ST segment elevation AMI involving another anatomical location of the heart e.g. inferior/lateral walls (20 cases), were excluded. In total, 11 cases of isolated posterior AMI were included in the study.

A review of duplicate death certificates dispensed from the ED during the study period did not discover additional cases. The ED's computerised documentation system broadly categorised all AMIs under ICD-9-CM code 410 and a search resulted in 3,778 cases within the study period. Among these, 26

AMI cases that were not admitted either due to at-own-risk discharge or transfer to another hospital were reviewed; none of them had isolated posterior AMI.

The median age of the 11 patients was 62, with age range between 31 to 89 years old. The majority were males with only 2 females. There were 9 Chinese and 2 Malay. Six out of the eleven cases had history of ischaemic heart disease (IHD), 3 of whom also had previous myocardial infarctions; 1 inferior, 1 anteroseptal and 1 non-ST segment elevation AMI (NSTEMI) of uncertain territory. The other 5 cases without IHD history each had at least one cardiovascular risk factor like diabetes mellitus, hypertension or dyslipidemia. About half of the cases were smokers.

Only 5 patients had definite angina from the history. Of the other 6 cases that did not have chest pain, all had dyspnoea. Among those with dyspnoea, two had pneumonia and one was a terminally-ill lung cancer patient. Three cases were suspected to have heart failure, having clinical findings of lung crepitations and pulmonary congestion on chest X-rays.

All except 2 cases have initial ED ECGs available for review (Table 1). The 9 ECGs had features of isolated posterior AMI without abnormal ST segment elevation. Specifically, all of them displayed the following: tall and wide R wave in V_2 (V_2 R/S ≥ 1 , V_2 R wave ≥ 0.04 sec) and upright T waves in two or more contiguous right precordial leads (V_1 to V_3). Almost all cases demonstrated ST depression of ≥ 0.2 mV (2 mm) with upright T waves in two or more contiguous anterior precordial leads (V_1 to V_4). Two cases lost the initial ED ECGs for review, the ECG descriptions by ED doctors were as such: case no. 10 – anterior ischaemia, suspected posterior infarct, U wave; case no. 11 – ST depression V_1 to V_2 and V_4 to V_6 . None of the 11 cases had posterior ECG leads V_8 and V_9 recorded in the ED.

Isolated posterior AMI was diagnosed at the ED in 8 out of the 11 cases. The remaining three cases, cases no. 6, 7 and 9 were admitted as non-ST elevation myocardial infarction (NSTEMI). All 3 had elevated initial serum troponin T (TnT). Cases no. 6 and 7 did not have typical symptoms of acute coronary syndrome (ACS) and case no. 9 was symptomatic for more than

Table 1. Electrocardiographic data in nine isolated posterior AMI patients

	Patient number								
	1	2	3	4	5	6*	7*	8	9*
Age	62	42	56	48	52	77	89	31	75
Gender	M	M	M	M	M	M	M	M	M
V_1 R wave ≥ 0.04 sec	+	+	+	+	-	-	-	+	-
V_2 R wave ≥ 0.04 sec	+	+	+	+	+	+	+	+	+
V_1 R/S ≥ 1	-	+	+	-	-	-	-	+	+
V_2 R/S ≥ 1	+	+	+	+	+	+	+	+	+
Upright T waves in 2 or more contiguous right precordial leads (V_1 to V_3)	+	+	+	+	+	+	+	+	+
V_1 ST depression	+	+	-	-	-	-	+	-	+
V_2 ST depression	+	+	+	+	+	+	+	-	+
ST depression ≥ 0.2 mV (2 mm) with upright T waves in 2 or more contiguous anterior precordial leads (V_1 to V_4)	+	+	+	+	+	+	+	-	+

* Admitted from emergency department as NSTEMI

12 hours. Case no. 9 presented with dyspnoea and decreased effort tolerance for two days. He did not have angina and was clinically in Killip Class 2 heart failure. He was treated with antiplatelet agents (aspirin, clopidogrel), intravenous nitroglycerin, frusemide and low molecular weight heparin (fraxiparine) in the ED.

Among the eight cases diagnosed as isolated posterior AMI in the ED; three cases received fibrinolytics (Table 2), five did not (Table 3). All those who received emergent fibrinolytic therapy presented to the ED within 12 hours from symptom onset with their initial ECGs fulfilling the American College of Emergency Physicians (ACEP) recommended ECG indications for emergent fibrinolytic reperfusion therapy in suspected true posterior AMI.³ Streptokinase was utilised in all

cases. No haemorrhagic complications occurred. None of the patients who were offered fibrinolytic refused therapy.

None of the patients underwent emergent percutaneous coronary intervention or had malignant arrhythmia requiring treatment while staying in the ED. For all cases, the decision on the level of care (intensive care unit or general ward) preceding disposition was determined following a discussion with the cardiologist-on-duty. Three cases (cases no. 6, 10 and 11) did not survive to discharge. The primary cause of death for cases 6 and 10 were metastatic lung cancer and pneumonia respectively. The remaining case was signed up as AMI. All 3 cases who received fibrinolytic survived to discharge. Six out of the eleven cases had

Table 2. Characteristics of the three patients who received fibrinolytic therapy in the emergency department

	Patient number		
	1	2	5
Age	62	42	52
Gender	M	M	M
Angina pectoris	+	+	+
Dyspnoea	-	+	-
Symptoms less than 12 hours	+	+	+
ST depression ≥ 0.2 mV (2 mm) with upright T waves in 2 or more contiguous anterior precordial leads (V_1 to V_4)**	+	+	+

**ACEP recommended ECG indications for emergent fibrinolytic reperfusion therapy in suspected true posterior AMI

Table 3. Characteristics of the five ED-identified isolated posterior AMI patients who did not receive fibrinolytic therapy in the emergency department

	Patient number				
	3	4*	8	10	11
Age	56	48	31	89	83
Gender	M	M	M	F	F
Angina pectoris	-	+	+	-	-
Dyspnoea	+	+	-	+	+
Symptoms less than 12 hours	-	+	-	+	+
ST depression ≥ 0.2 mV (2 mm) with upright T waves in 2 or more contiguous anterior precordial leads (V_1 to V_4)**	+	+	-	?	?

* Lung auscultation was clear; ** ACEP recommended ECG indications for emergent fibrinolytic reperfusion therapy in suspected true posterior AMI; ? ECG not available for review

cardiac catheterisation done within two months following the acute episode, 3 of which were performed during the same admission for AMI (Table 4). Left circumflex coronary artery (LCX) occlusion was demonstrated in all the patients who underwent cardiac catheterisation.

Discussion

Acute posterior wall MI occurs in up to 20% of AMIs, with the vast majority occurring along with inferior or lateral AMI.^{1,2} On the other hand, the incidence of isolated posterior AMI is considered very low¹ and it

is possibly underdiagnosed. Posterior MI refers to infarction of the posterior or posterobasal wall of the left ventricle. It is mostly due to left circumflex (LCX) artery occlusion,^{4,5} or that of a dominant right coronary artery (RCA)⁶ and its posterior descending branch.

In isolated posterior AMI, characteristic ST elevation and Q wave are absent on the conventional 12-lead ECG. Instead, the ECG abnormalities include the following (in leads V₁, V₂ or V₃): (1) horizontal ST depression with tall, upright T waves; (2) a tall, wide R wave; and (3) an R/S wave ratio greater than 1.0 in lead V₂.^{2,7} This is because leads V₁ to V₃ face the endocardial surface of the posterior wall of the left

Table 4. Cardiac catheterization findings in six isolated posterior AMI cases

Case number	Coronary vessel			Remark
	LM/LAD	LCX	RCA	
1	Minor irregularities	Proximal – 2 discrete lesions 80% and 50% Distal – diffuse 70%	Dominant In-stent stenosis 40% Distal – 50%	EF 40% Double vessel disease Inferior AMI 5 years prior
2	LM normal LAD – small, distal disease	Co-dominant Proximal lesion 85%	Co-dominant Large PL1 and PL2 wall injury noted	PTCA planned for LCX
4	Normal	OM1 – small OM2 – proximal discrete lesion 90%, distal discrete lesion 80%	Minor irregularities with slow flow	PTCA planned for OM2
5	LM – distal 30% LAD – 100% at D1	Large OM1 – proximal lesion 70% LCX – proximal 40%, distal 100% LCX fills PDA	Mid 100% Proximal conus branch supplies LAD!	EF 35% TVD for CABG
8	Mid LAD – mild myocardial bridging	Distal – 100%, TIMI 2 flow to distal OM via collaterals from RCA	Minor luminal irregularities	
9	LAD – distal 50-60%, proximal 70% diffuse calcification	Proximal OM – 100%	Mid 40-50%	

CABG – coronary artery bypass graft; D – diagonal artery; EF – ejection fraction; LAD – left anterior descending artery; LCX – left circumflex artery; LM – left main artery; OM – obtuse marginal artery; PDA – posterior descending artery; PL – posterolateral artery; PTCA – percutaneous transluminal coronary angioplasty; RCA – right coronary artery; TIMI – Thrombolysis In Myocardial Infarction; TVD – triple vessel disease

ventricle. As these leads record from the opposite side of the heart instead of directly over the infarct, the changes of posterior infarction are reversed in these leads.⁸ The 9 ECGs available for review in our series had these features (Table 1). Specifically, all of them displayed the following: tall and wide R wave in V_2 (V_2 R/S ≥ 1 , V_2 R wave ≥ 0.04 sec) and upright T waves in two or more contiguous right precordial leads (V_1 to V_3). It is helpful to turn the ECG upside down and look at it from the back through the paper (Figures 1 & 2). The changes in V_1 and V_2 , which might be overlooked at first glance, will be seen as abnormal Q waves, ST segment elevation, and increased T wave inversion.⁹

If additional ECG leads are utilised, $V_{8,9}$ may show >1 mm ST segment elevation.² Numerous studies have shown using additional ECG leads in selective cases increases the sensitivity without sacrificing the specificity of detecting acute posterior wall AMI.^{1,2} Posterior leads can also help to identify LCX

involvement. In one study, a team prospectively studied electrocardiographic changes during balloon occlusion of single-vessel RCA and LCX. ST elevation was always seen in inferior leads in the RCA group and in posterior leads in the LCX group. Thus, posterior leads helped differentiating RCA versus LCX as the infarct-related artery.¹⁰ Our findings indicated that the majority of isolated posterior AMI cases (8 out of 11 cases) were correctly diagnosed at our ED, without acquiring posterior ECG leads $V_{8,9}$. In fact, there was enough confidence to commence fibrinolytics in three cases following discussion with the cardiologist-on-duty. The placement of additional leads $V_{8,9}$ is not widely practiced in our ED. The actual reason is uncertain but we suspect that ED staff perceives it to be cumbersome to conduct or lacks the knowledge to do so.

Other than true posterior wall AMI, ST segment depression on conventional 12-lead ECG in the ACS population may indicate one of three diagnoses: myocardial ischaemia (without infarction), reciprocal



Figure 1. ECG showing tall R wave and ST depression in leads V_1 to V_3 in one patient.



Figure 2. Same ECG (Figure 1) when flipped vertically, shows characteristic Q waves and ST elevation.

ST segment change in the setting of AMI, and NSTEMI (formerly the non-Q wave AMI).⁶ Mukharji et al¹¹ noted that of all patients with inferior AMI, 80% demonstrated anterior ST segment depression in leads V₁₋₃. Other possible mimics of a posterior MI ECG include causes of tall R waves in V₁ – such as right ventricular hypertrophy, right bundle branch block, Wolff-Parkinson-White syndrome and normal variants - which can be differentiated since they do not cause ST segment elevation or significant Q waves in leads V₇-V₉.¹²

The most stringent criterion for posterior infarction, R wave duration ≥ 0.04 sec and R \geq S in lead V₁, showed a very high specificity (>99%), a high positive predictive value (91%), but a low sensitivity (36%).¹³ Only 3 cases (cases no. 2, 3 and 8) in our series fulfilled these criteria.

In a study to assess the ability of junior doctors to interpret ECGs which had immediate clinical relevance, difficulty in the interpretation of posterior myocardial infarction was noted.¹⁴ We did not analyse the work experience of the doctors who managed individual cases but junior doctors in our ED had ready help from the senior doctors and cardiologists. In this setting, three cases (with elevated initial serum TnT and no abnormal ST segment elevation) were misinterpreted as NSTEMI; 2 of these cases did not have typical symptoms of acute coronary syndrome, and the other presented with symptoms for more than 12 hours' duration. This would have precluded them from fibrinolytic therapy even if correct diagnoses were made. The use of additional posterior ECG leads V₈₋₉ would have been particularly useful in distinguishing between NSTEMI (anterior territory) and isolated posterior AMI as the cause for precordial ST segment depression in these cases.

Dyspnoea was present in all the cases without angina. It could be an important angina-equivalent symptom in isolated posterior AMI patients. The presence of angina may have raised the clinical suspicion of AMI; notably all three cases (100%) which were labelled as NSTEMI did not have angina.

Current evidence strongly indicates that fibrinolytic therapy should not be used routinely in patients with ST-segment depression on the 12-lead ECG unless the evaluating physician suspects isolated posterior AMI.¹⁵ Mortality rate may actually be increased by the administration of fibrinolytics in this electrocardiographically diverse patient subgroup. In the Fibrinolytic Therapy Trialists meta-analysis, mortality in patients with ST-segment depression was 15.2% in the fibrinolytic therapy group versus 13.8% in the control group. Due to this finding, the American College of Cardiology/American Heart Association guidelines for AMI categorised ST-segment depression as a class III indication for fibrinolytic drugs (i.e. no benefit with possible harm) except in patients in whom a true posterior AMI is suspected.¹⁶ Evidence based guidelines from the Seventh American College of Chest Physicians (ACCP) Conference on Antithrombotic and Thrombolytic Therapy suggest fibrinolytic therapy for patients with ischaemic symptoms characteristic of acute MI of 12 hours in duration and 12-lead ECG findings consistent with a true posterior MI (grade 2C recommendation).¹⁷ Grade 2C recommendations are based only on observational studies and have unclear risk vs. benefit. It is a very weak recommendation and other alternatives may be equally reasonable.¹⁸ ACEP Clinical Policies Subcommittee (Writing Committee) on Reperfusion Therapy in Emergency Department Patients with Suspected Acute Myocardial Infarction recommends assessing for fibrinolytic therapy in patients with clinical presentation suggestive of AMI involving the posterior left ventricular wall, who present within 12 hours of symptom onset and if ECG reveals ST depressions greater than or equal to 0.2 mV (2 mm) with upright T-waves in two or more contiguous anterior precordial leads (V₁ to V₄). This is a level B recommendation that reflects moderate clinical certainty.³ The cases in our series which received emergent fibrinolytic therapy in the ED, were compliant to the above ACEP recommendations. In addition, we felt that case no. 4 should be considered for fibrinolytic therapy, although utilisation of low molecular weight heparin is just as appropriate, until stronger evidence for fibrinolytic usage in isolated posterior AMI arises.

We encountered three cases suspected to have heart failure; one of whom had previous anteroseptal AMI. Usually true posterior AMIs are well tolerated,¹⁹ although some believe that it may be associated with a significant amount of myocardium in jeopardy.^{1,20}

Lastly, the following would be helpful in distinguishing isolated posterior AMI from among the other causes of ST segment changes: (1) detection of elevated cardiac enzymes, (2) absence of abnormal ST elevation on ECG, (3) acquiring additional posterior leads V_{8,9}, (4) turning the ECG around and looking at it from the back, (5) obtaining serial ECGs for review, (6) consultation with floor senior ED doctor or cardiologist-on-duty (by faxing ECG and telephone consult), (7) reckoning the ECG machine's computerised interpretation printout.

Limitations

As case selection relied mainly upon inpatient discharge ICD-9-CM coding, the authors recognised that cases might be missed due to miscoding. In addition, cases which were wrongly admitted as isolated posterior AMI from the ED would be missed by our selection methodology because discharge diagnosis would not reflect it. However, this was highly unlikely as all cases with AMI should have been discussed with a senior doctor or cardiologist-on-duty at the ED.

We faced problems with data collection that are inherent to studies of a retrospective nature. Some ED records had poor documentation of patient symptomatology and clinical examination. The initial ED ECGs of two cases were not available for review. Finally, the study was limited to one urban hospital in Singapore.

Conclusion

Our ED physicians were able to correctly diagnose the majority of cases (72.7%) with isolated posterior AMI. Although three cases were interpreted as NSTEMI, the use of fibrinolytic reperfusion therapy was not affected.

Additional posterior chest leads V_{8,9} were not utilised in any of the cases. They would be helpful to differentiate NSTEMI from isolated posterior AMI in suspicious cases. All cases which received emergent fibrinolytic therapy were compliant with recommendations set forth by the ACEP Clinical Policies Subcommittee for Reperfusion Therapy in ED Patients with Suspected AMI.

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