D-Dimer Plasma Levels in NSTE-ACS Patient

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Background: *Acute Coronary Syndrome (ACS) occurs when a vulnerable plaque ruptures and induces platelet aggregation and coagulation process at the rupture. Thrombogenesis is the final process that forms a clot in the coronary lumen causing myocardial injury. Plasma D-dimer, a primary degradation product and circulating marker of fibrin turnover, serves as a direct marker of ongoing fibrinolysis in site of coronary artery occlusion.*
Objective: *To determine the correlation between plasma D-dimer levels and severity of coronary artery obstruction based on angiographic data that is composed of the number of coronary arteries affected and the percentage of maximum stenosis of coronary artery lumen in non-ST elevation ACS (NSTE-ACS) patients.*
Material and Method: *NSTE-ACS patients who admitted in Siriraj hospital during June 2009 and March 2010 were enrolled. Conditions that increased plasma D-dimer other than NSTE-ACS were excluded. Demographic characteristics were assessed by a standardized questionnaire. Plasma D-dimer was measured and coronary angiography was performed to evaluate severity of coronary artery stenosis.*
Results: *Total of 74 NSTE-ACS patients were enrolled (29 in unstable angina and 45 in non-ST elevation myocardial infarction). Mean age of these patients (54.1% in female and 45.9% in male) were 66 years. D-Dimer was significantly increased with the number of coronary arteries affected ($p = 0.03$). In non-significant and single coronary artery disease (CAD) patients, median D-dimer was 406 (178-2,788) mcg/L. In multivessel CAD, median D-dimer was 941 (131-7,110) mcg/L. D-dimer levels had a trend to be increased with percentage of maximum stenosis of coronary artery lumen; atheromatosis, ($p = 0.30$). In mild and moderate atheromatosis (coronary artery stenosis < 70%), median D-dimer was 479 (182-5,902) mcg/L while median D-dimer was 789 (131-7,110) mcg/L in severe atheromatosis (coronary artery stenosis > 70%). Moreover, plasma D-dimer levels correlated with complication of NSTE-ACS (Congestive heart failure; $p < 0.001$, arrhythmia; $p = 0.007$ and death; $p = 0.009$) and was increased in patients who underwent treatment with CABG more often than those who received PCI and medication treatment alone. D-dimer also correlated with serum creatinine ($r = 0.517$, $p < 0.001$), creatinine clearance ($r = -0.463$, $p < 0.001$), troponin-T level ($r = 0.381$, $p < 0.001$) and left ventricular ejection fraction ($r = -0.368$, $p = 0.002$).*
Conclusion: *D-dimer is useful coagulation marker use to evaluate extent of coronary affected and may predict in-hospital CV complication. However, other conditions that increased plasma D-dimer also excluded.*

Keywords: *D-dimer, Acute coronary syndrome*

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Acute coronary syndrome (ACS) was divided into categories by electrocardiogram (ECG) finding. The first group is ST elevation Acute Coronary Syndrome (STE-ACS). The Second is Non-ST elevation Acute Coronary Syndrome (NSTE-ACS). NSTE-ACS is composed of unstable angina (UA) and non-ST segment elevation myocardial infarction (NSTEMI). Differentiation between UA and NSTEMI used a cardiac biomarker. ACS occurs when a vulnerable plaque ruptures that induces platelet aggregation and coagulation process at the site, blocking coronary blood flow and causing ischemic injury to the myocardium. Thrombogenesis is the final process whereby the exposed tissue factor triggers the coagulation pathway, and the freshly formed clot fills in the coronary artery lumen. Fibrinolysis process will develop for counterbalancing thrombogenesis in normal human hematostasis physiology. D-dimer, primary degradation product of cross-linked fibrin, considered as a global marker of turnover of cross-linked fibrin and activation of the hemostatic system. D-dimer in ACS serves as a direct marker of ongoing fibrinolysis process in the

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site of coronary artery occlusion.

From data of Thai ACS registry, August 2002-2005, incidence of NSTE-ACS was about 60% from all ACS patients. These patients were admitted in hospitals and great expense was incurred for investigation and treatment. Because ECG in NSTE-ACS patients has variation and cannot predict severity of disease, other investigations were done in the quest for proper management. Several studies found relationships between D-dimer, biochemical markers of thrombus formation, and coronary artery disease (CAD). D-dimer was increased in stable CAD, acute coronary syndrome and atherosclerotic diseases due to atherothrombosis etiology. D-dimer was significantly higher in acute ischemic events[1]. D-dimer level at admission to the emergency department may serve as an additional tool to predict the magnitude of UA in patients with a normal ECG[2]. D-dimer correlated with severity of atherosclerosis in patients with stable angina after acute myocardial infarction[3]. Severe atherosclerosis of coronary artery increased fibrinolytic activity and then increased D-dimer level[3]. Otherwise, plaque progression in CAD is composed of atherosclerotic plaque growth, necrotic core progression, microruptures and destabilization of plaques induced D-dimer production[4,5]. So, plasma D-dimer may be used to evaluate severity of CAD.

Objectives

To determine the correlation between plasma D-dimer levels and severity of coronary artery obstruction based on angiographic data in non-ST elevation ACS (NSTE-ACS) patients. Severity of coronary artery disease from coronary angiography in this study composed of the number of coronary arteries affected and the percentage of maximum stenosis of coronary artery lumen.

Material and Method

Study Population

The protocol of this study had approval from Ethics Committees of Siriraj Hospital. NSTE-ACS patients who admitted in Siriraj Hospital were recruited between June 2009 and March 2010. Unstable angina was defined as ischemic chest pain at rest that lasted at least 20 min, new-onset angina of at least Canadian Classification Score class III severity, or accelerating angina, with no evidence of myocardial necrosis by enzymatic techniques[6]. NSTEMI was defined by the same criteria of UA but also exhibited increased level of cardiac biomarkers[6]. Patients were informed about the aims of the study and those who agreed with the study signed informed consent forms.

Demographic characteristics were assessed by a standardized questionnaire included age, sex, body mass index, coronary risk factors, underlying disease, drug intake, electrocardiograph, echocardiography etc. Several physiological or clinical conditions may lead to an increase in level of D-dimer. Patients younger than 18 years were excluded, as were patients who had received anticoagulation treatment or had primary coagulopathy, malignancy, surgery, or infection in the month preceding the study, or an abnormal liver or renal function test (Creatinine clearance < 30 ml/min) and history of deep vein thrombosis or pulmonary embolism in the last year.

Laboratory data

General laboratory data were measured during admission depend on patients conditions, such as fasting blood sugar, blood chemistry, cardiac biomarkers, lipid profile etc. Plasma D-dimer (mcg/L of Fibrinogen Equivalent Unit) was measured as quick as when disease was confirmed and invasive strategy was chosen. Blood sample for D-dimer needs 3 ml in 3.2% buffered sodium citrate tube, centrifuged at 3,000 rpm for 15 min within 4 hr. Plasma fibrin D-dimer was assayed in quantitative data, by enzyme immunoassay and enzyme linked fluorescent assay (ELFA) technique. Using the commercial kits and assay of Vidas D-dimer Exclusion™(DD2) (BioM'erieux, Lyon,USA) in central laboratory of Siriraj hospital. Range of detected plasma D-dimer was 45-10000 mcg/L of Fibrinogen Equivalent Unit (FEU). Sensitivity and specificity of this technique is similar as ELISA technique which is the gold standard[7].

Precision of equipment

Plasma n = 80	Concentration (ng/ml) (FEU)	Within run	Reproducibility
		% CV*	% CV
Level 1	264	5.0	5.7
Level 2	549	3.9	5.8
Level 3	7,283	5.3	7.1

Angiographic data

All patients in this study underwent coronary catheterization during admission when invasive strategy was chosen depending on indications.

Findings from the angiographic outcome, interpreted by at least two cardiologists who were blinded to the D-dimer results, were classified in two categories. The first category was number of coronary arteries affected, classified in 3 subgroups composed of single, double or triple vessels disease. Left main coronary artery was grouped with double vessel disease.

The second category was percentage of maximum stenosis of coronary arteries lumen, classified in 3 subgroups which consisted of mild, moderate and severe atheromatosis. Atheromatosis was defined as diffuse atheromatous disease of the coronary arteries. Mild atheromatosis was defined less than 30% stenosis of coronary artery lumen. Moderate and severe atheromatosis was defined 30-70% and more than 70% stenosis of coronary artery lumen respectively. Maximum percentage of coronary artery occlusion was obtained by Digital Quantitative Coronary Angiography (DQCA) technique.

Statistical analysis

The statistical evaluation was carried out using the Statistical Package for the Social Sciences (SPSS 16.0). Results were given as mean $\pm$ standard deviations, median (min-max), Kruskal-Wallis-test 1-way ANOVA. The spread of constant characteristics between the two groups was compared using the Mann-Whitney U-test, and more than 2 groups using Kruskal-Wallis-test. The correlation between constant characteristics was determined using the Spearman rank-correlation coefficients. Pearson correlation coefficient (r) and the significance for it (p) were calculated between the D-dimer level and the clinical variables. P-values less than or equal to 0.05 were considered statistically significant.

Definition of correlation coefficient in this study is little or no relationship (r = 0-0.25), fair degree relationship (r = 0.25-0.50), moderate to good relationship (r = 0.50-0.75) and very good or excellent relationship (r = 0.75-1.00)[8].

Results

A total of 74 NSTE-ACS patients who were admitted to Siriraj hospital were enrolled (39.2% UA and 60.8% NSTEMI). Mean age of these patients (54.1% in female and 45.9% in male) were 66.07 $\pm$ 11 years. Most patients had diabetes mellitus (52.7%), hypertension (83%) and dyslipidemia (74%). Mean LDL was 106 $\pm$ 44 mg/dl. Other cardiovascular risk factor such as smoking (23%), ischemic stroke (4.1%) and family history of cardiovascular disease (5.4%) were found in some patients. Mean body mass index of these patients was 24.57 $\pm$ 4.24 kg/m^2. Most patients had normal kidney function (Creatinine clearance of 66.1 $\pm$ 30.34 ml/min). Diabetic patients in this study had HbA1c of 7.27 $\pm$ 1.88%.

NSTE-ACS patients came to Siriraj hospital due to angina (78.4%) and dyspnea (18.9%) respectively. Presenting ECG of these patients were ST segment depression (36.5%) and T wave inversion (35.1%). Other ECG presentations included Q wave, right bundle branch block, left bundle branch block and non significant ECG which were found in 28.38%. NSTE-ACS patients in this study had normal left ventricular ejection function (LVEF = 54.21 $\pm$ 17.17%). Others clinical parameter and laboratory data were shown in Table 1 and 2.

Most NSTE-ACS patients in this study were intermediate to high risk (Mean TIMI risk score 3.3 $\pm$ 1.4). Median length of hospital stay of NSTE-ACS in this study was 7 (1-90) days. Mean duration of plasma D-dimer collection after admission was 41 $\pm$ 4.7 hours (63 $\pm$ 6 hours after onset of symptom). Mean duration of coronary angiography after admission was 5.4 days. D-dimer was statistically significantly correlated with

Table 1. Baseline Characteristics*

Characteristics	Value*
Age (years)	66 $\pm$ 11
Male	34 (45.9%)
NSTEMI	45 (60.8%)
Diabetes Mellitus	35 (52.7%)
Hypertension	62 (83.0%)
Dyslipidemia	55 (74.0%)
Smoking	17 (23.0%)
Ischemic stroke	3 (4.1%)
Family History of Cardiovascular diseases	4 (5.4%)
Symptom	
-Angina	58 (78.4%)
-Dyspnea	14 (18.9%)
-Others	2 (2.7%)
ECG	
-ST depression	27 (36.5%)
-T wave inversion	26 (35.1%)
-Others	21 (28.4%)
Length of stay (days)	Median (0:90)
BMI (kg/m^2)	24.6 $\pm$ 4.2

*Mean $\pm$ SD All other values are n (%).
Abbreviations: ECG, electrocardiography; BMI, body mass index

number of coronary arteries affected (p = 0.03) as shown in Table 3 and Fig. 1. D-dimer was increased in double and triple vessels disease more than in non-significant and single vessel disease. Median D-dimer of double and triple vessel disease was 941 (131-7,110) mcg/L. Median D-dimer of non-significant and single vessel disease was 406 (178-2,788) mcg/L.

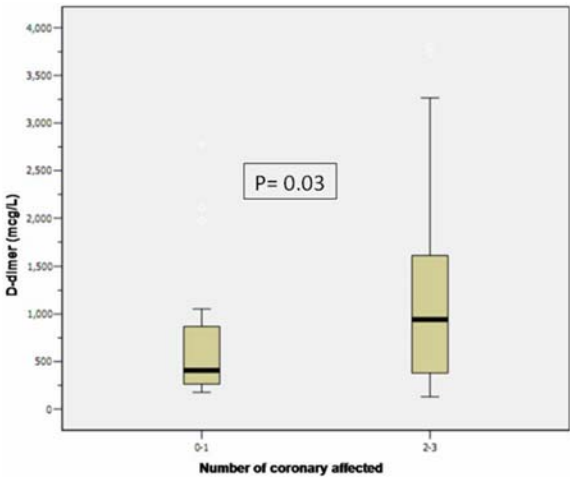
D-dimer was statistically significant when correlated with complication of NSTE-ACS. D-dimer was increased significantly when CHF, arrhythmia and death presented as shown in Table 3.

Table 2. Laboratory Findings, Clinical and cardiac catheterization During Current Hospitalization

Characteristics	Value*
Creatinine Clearance (ml/min)	66.1 ± 30.3
HbA1c (%)	7.3 ± 1.9
Cholesterol (mg/dl)	172.6 ± 50.0
Triglyceride (mg/dl)	135.7 ± 69.4
HDL (mg/dl)	42.8 ± 13.1
LDL (mg/dl)	106.0 ± 44.0
LVEF (%)	54.2 ± 17.2

Abbreviations: HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; LDL, low-density lipoprotein; IHD, ischemic heart disease; LVEF, left ventricular ejection fraction

D-dimer levels had a trend to increase with percentage of maximum stenosis of coronary artery lumen (p = 0.30) as shown in Table 3. In mild and moderate atheromatosis (coronary artery stenosis < 70%), median D-dimer was 479 (182-5,902) mcg/L. In severe atheromatosis (coronary artery stenosis > 70%),



0-1 Non significant and single vessel disease
2-3 Double and triple vessel disease

Fig. 1 Comparison between D-dimer (mcg/L) and number of coronary artery affected

Table 3. Comparison between D-dimer and angiographic variables and complication

Parameters	Characteristics	n	D-dimer (mcg/L)*	p
Number of coronary arteries affected	0-1 vessel	25	406 (178-2,788)	0.03
	2-3 vessels	49	941 (131-7,110)	
Percentage of stenosis of CAD (Atheromatosis)	Mild/Moderate (stenosis <70%)	19	479 (182-5,902)	0.30
	Severe (stenosis >70%)	55	789 (131-7,110)	
Treatment	Medications	11	479 (182-2,754)	0.25
	PCI	31	466 (178-6,988)	
	CABG	29	1,015 (131-7,110)	
Complication	CHF	31	1,475 (200-7,110)	< 0.001
	No CHF	43	385 (131-3,813)	
	Arrhythmia	2	5,422 (3733-7,110)	0.007
	No arrhythmia	72	550 (131-6,988)	
	Death	2	5,118 (3,265-7,110)	0.009
	No death	72	550 (131-6,988)	

*Median (min-max)
Abbreviations: PCI, Percutaneous Coronary Intervention; CABG, Coronary Artery Bypass Graft; CHF, congestive heart failure
0-1 Non significant and single vessel disease
2-3 Double and triple vessel disease

Table 4. Spearman Rank Correlation Coefficients Between Plasma D-Dimer Levels and multiple variables

Parameters	Results	
	r	p
Creatinine (mg/dl)	0.517	< 0.001
Troponin T(ng/ml)	0.381	< 0.001
Age (years)	0.229	0.05
TIMI risk score	0.123	0.3
HbA1C (%)	0.116	0.50
CKMB (ng/ml)	0.074	0.53
BMI(kg/m^2)	0.034	0.77
Creatinine clearance (ml/min)	-0.463	< 0.001
LVEF (%)	-0.368	0.002

Abbreviations: TIMI, thrombolytic myocardial infarction; BMI, body mass index; HbA1c, hemoglobin A1c; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction HbA1c, hemoglobin A1c

median D-dimer was 789 (131-7,110) mcg/L.

D-dimer levels had a trend to be increased in patients who underwent treatment with CABG more than in those who received PCI and medication treatment alone ($p = 0.25$), as shown in Table 3. Patients that received CABG treatment had D-dimer of 1,015 (131-7,110) mcg/L. D-dimer in PCI and medication patients was 466 (178-6,988) mcg/L and 479 (182-2,754), respectively.

Correlation between plasma D-Dimer Levels and multiple variables by Spearman Rank Correlation Coefficients was found as shown in Table 4. Moderate to good correlation between plasma D-dimer and serum creatinine ($r = 0.517$, $p < 0.001$) was found. There was fair correlation between plasma D-dimer and troponin-T level ($r = 0.381$, $p < 0.001$), creatinine clearance ($r = -0.463$, $p < 0.001$) and left ventricular ejection fraction ($r = -0.368$, $p = 0.002$). When D-dimer increased, it was associated with increase of serum creatinine and Tn-T level and also a decrease of creatinine clearance and LVEF.

Discussion

ACS is characterized by rupture or erosion of vulnerable atherosclerotic plaque, Exposure of the procoagulant lipid core to circulating blood established thrombus formation. These result in the support of the contribution of intravascular fibrin in coronary arteries. As coronary thrombus proceeds to myocardial necrosis, it is possible that coagulation markers such as D-dimer are sensitive markers of ACS. D-dimer potentially rises earlier than markers of myocardial necrosis such as troponin-T. However, evaluation of plasma D-dimer in this study aims to evaluate severity and extent of coronary artery occlusion in NSTE-ACS patients.

D-dimer was correlated significantly with number of coronary arteries affected but insignificantly in percentage of maximum stenosis of coronary artery lumen (atheromatosis). If plasma D-dimer level is high in NSTE-ACS patients without other physiologic conditions that involve fibrinolytic pathway, multiple coronary arteries involvement should be suspected. Plasma D-dimer may be used as prognostic factor in NSTE-ACS patients due to extent of CAD which affected survival[9]. Invasive strategy by coronary angiography may play an important role and be the preferred treatment in these patients. The patients who had high plasma D-dimer also had a tendency to be treated with CABG more than PCI or medication alone. Physicians should be more aware of complications such as CHF, arrhythmia and death more than for low plasma D-dimer patients.

D-dimer can be used to predict cardiovascular events during hospitalization in NSTE-ACS patients. D-dimer is rising when CHF, arrhythmia and death was found. But further study or long term study should be done to confirm this hypothesis due to low event rate in this study.

D-dimer insignificantly correlated with percentage of maximum stenosis of coronary artery lumen (atheromatosis) in this study because most NSTE-ACS patients had chronic coronary artery lesions that form few thrombi. Thus plasma D-dimer was not as high as in the acute condition.

An hypothesis of pathophysiology of NSTE-ACS disease, clot bound formation, can not be evaluated since most patients in this study went to the catheterization laboratory so late (mean time to catheterization laboratory about 5.4 days) that thrombus was lysed during reception of anticoagulant drugs. Only one patient was seen with thrombus in the distal circumflex coronary artery due to a time to cardiac catheterization of about 10 hours.

Limitations

Because the number of patients in this study was smaller than expected, the results may not represent true outcomes. Thus the result of correlation between D-dimer and maximum percentage of coronary artery

stenosis (atheromatosis) should be further investigated using large sample size or increased study duration to confirm our hypothesis. Correlation between D-dimer and complications of NSTE-ACS should be studied further in a large population cohort.

Since most patients in this study were intermediate to high risk (Mean TIMI risk score 3.3 $\pm$ 1.4), the results in this study may represent this group only. Otherwise, plasma D-dimer was collected too late in some patients due to decision making delay of patients not yet deciding to allow invasive strategy treatment. Additionally duration to coronary angiography was too long because of long weekend and many queues.

Conclusion

Plasma D-dimer levels in NSTE-ACS patients was statistically significant when correlated with multivessel CAD, troponin-T level, kidney function, left ventricular ejection fraction and complications. Otherwise, plasma D-dimer had a trend to be increased in severe atheromatosis patients and those receiving CABG treatment.

D-dimer is the coagulation marker which rises earlier the marker of myocardial ischemia and is used to evaluate the extent of coronary arteries affected and may predict in-hospital cardiovascular complication. However, other conditions that increased plasma D-dimer should also be earlier excluded.

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Potential conflicts of interest

None.

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ระดับ D-dimer ในผู้ป่วยโรคหัวใจขาดเลือดเฉียบพลันชนิด ST ไม่ยก

นิศารัตน์ เจริญศรี, สุวัจชัย พรรัตนรังสี

ภูมิหลัง: *ภาวะหัวใจขาดเลือดเฉียบพลันเกิดจากการแตกเฉียบพลันของตะกรันในหลอดเลือดหัวใจ แล้วกระตุ้นการเกาะตัวของเกล็ดเลือดและกลไกการแข็งตัวของเลือด นำไปสู่การเกิดลิ่มเลือดซึ่งอุดตันหลอดเลือด แล้วทำให้เกิดกล้ามเนื้อหัวใจตาย D-dimer เป็นผลผลิตจากการสลายของลิ่มเลือด ซึ่งแสดงถึงกระบวนการสลายของลิ่มเลือดที่เกิดขึ้นในหลอดเลือดที่อุดตันนั้น*

วัตถุประสงค์: *เพื่อเปรียบเทียบความสัมพันธ์ระหว่างระดับ D-dimer ในเลือดและความรุนแรงของโรคหลอดเลือดหัวใจจากการสวนหัวใจ โดยดูที่จำนวนหลอดเลือดที่ตีบและอัตราส่วนรอยตีบในผู้ป่วยหัวใจขาดเลือดเฉียบพลันชนิด ST segment ไม่ยก*

วัสดุและวิธีการ: *ทำการศึกษาผู้ป่วยที่รับตัวเข้ารักษาในโรงพยาบาลศิริราชด้วยโรคหัวใจขาดเลือดเฉียบพลันชนิด ST ไม่ยก ตั้งแต่มิถุนายน พ.ศ. 2552 ถึงมีนาคม พ.ศ. 2553 โดยทำการคัดแยกสาเหตุที่ทำให้ระดับ D-dimer สูงขึ้นออก ข้อมูลทั่วไปจะถูกเก็บโดยการใช้แบบสอบถาม D-dimer จะได้รับการตรวจก่อนการทำการตรวจสวนหัวใจ*

ผลการศึกษา: *ผู้ป่วย 74 ราย โดย 29 รายเป็นภาวะเจ็บหน้าอกแปรผัน และ 45 ราย เป็นกล้ามเนื้อหัวใจขาดเลือดเฉียบพลันชนิด ST ไม่ยกเป็นผู้ป่วยหญิง 54.1% อายุเฉลี่ยของผู้ป่วย 66 ปี D-dimer มีระดับเพิ่มขึ้น โดยมีความสัมพันธ์อย่างมีนัยสำคัญทางสถิติกับจำนวนหลอดเลือด coronary ที่มีรอยตีบ ($p = 0.03$) โดยค่ากลางของ D-dimer ในกลุ่มหลอดเลือดที่ตีบไม่เกิน 1 เส้น คือ 406 (178-2788) mcg/L ขณะที่ค่าดังกล่าวในกลุ่มตีบ 2 หรือ 3 เส้น คือ 941 (131-7,110) mcg/L นอกจากนี้ระดับของ D-dimer มีแนวโน้มสูงขึ้นตามความรุนแรงของโรควัดจากอัตราส่วนรอยตีบ ที่มากที่สุด ($p = 0.30$) โดย D-dimer มีค่ากลาง 479 (182-5,902) mcg/L ในกลุ่มที่รอยตีบน้อยกว่า 70% และ 789 (131-7,110) mcg/L ในกลุ่มที่รอยตีบเกิน 70% นอกจากนี้ค่า D-dimer ยังมีความสัมพันธ์กับอัตราการเกิดผลลัพธ์ทางคลินิกในผู้ป่วยด้วย เช่น ภาวะน้ำท่วมปอด ($p < 0.001$) หัวใจเต้นผิดจังหวะ ($p = 0.007$) และเสียชีวิต ($p < 0.009$) D-dimer ยังสูงขึ้นในผู้ป่วยที่รักษาโดยการผ่าตัดบายพาส เมื่อเปรียบเทียบกับการรักษาวิธีอื่นพบความสัมพันธ์ ของ D-dimer กับค่า creatinine ($p < 0.001$), creatinine clearance ($p < 0.001$), troponin-T ($p < 0.001$) และ left ventricular ejection fraction ($p = 0.002$)*

สรุป: *D-dimer เป็นสารในกลไกการแข็งตัวของเลือดที่สามารถใช้ในการประเมินความรุนแรงของหลอดเลือดโคโรนารีที่ตีบ และคาดการณ์ถึงผลแทรกซ้อนทางคลินิกในผู้ป่วยโรคหัวใจขาดเลือดเฉียบพลันชนิด ST ไม่ยก*