Case Report

กรณีศึกษา

Fatal Coronary Artery Thrombosis in a Middle-Aged Thai Man: A Case Report

การเสียชีวิตในชายไทยวัยกลางคนจากลิ่มเลือดอุดตันในหลอดเลือดหัวใจ : กรณีศึกษา

Warataporn Sithicharoon, M.D.*, Nantana Choomchuay, M.D.**

Department of Forensic Medicine, **Department of Pathology,Faculty of Medicine, Srinakharinwirot University, Nakhon Nayok 26120, Thailand

วรัทพร สิทธิจรูญ พ.บ.†, นันทนา ชุมช่วย พ.บ..††,

†ภาควิชานิติเวชศาสตร์ และ ††ภาควิชาพยาธิวิทยา คณะแพทยศาสตร์ มหาวิทยาลัยศรีนครินทรวิโรฒ นครนายก ประเทศไทย 26120

Abstract

Coronary artery thrombosis is the occurrence of a thrombus in a coronary artery. When the occlusion is not corrected within an appropriate time, the blood supply to cardiac muscle will be insufficient resulting in ineffective cardiac contractility or myocardial infarction. We report a case of sudden death of a 38-year old man dead by acute myocardial infarction from thrombosis of the right coronary artery without the history of angina or cardiac symptom.

Keywords: Coronary artery thrombosis, sudden death, autopsy

บทคัดย่อ

โรคลิ่มเลือดอุดตันหลอดเลือดหัวใจ เกิดจากการมีลิ่มเลือดเกิดขึ้นในหลอดเลือดหัวใจโคโรนารี่ ซึ่งหากไม่ได้รับการรักษาภายในช่วงเวลาที่เหมาะสม กล้ามเนื้อหัวใจจะไม่ได้รับเลือดอย่างเพียงพอ ส่งผลให้การบีบตัวของกล้ามเนื้อหัวใจไม่มีประสิทธิภาพ หรือกล้ามเนื้อหัวใจตาย การศึกษานี้ได้รายงานกรณีการเสียชีวิตอย่างกระทันหันของชายไทย อายุ 38 ปี ที่เกิดจากกล้ามเนื้อหัวใจตายเฉียบพลันจากการอุดตันของลิ่มเลือดในหลอดเลือดหัวใจโคโรนารี่แขนงขวา โดยไม่มีประวัติเจ็บแน่นหน้าอก หรืออาการของโรคหัวใจมาก่อน

คำสำคัญ: โรคลิ่มเลือดอุดตันหลอดเลือดหัวใจ, การเสียชีวิตอย่างกระทันหัน, การตรวจศพ

Introduction

Atherosclerosis is the major cause of coronary heart disease (CHD), which is the most common cause of death, and may start around the age of 20 and develop gradually with an increasing age.[1] An acute condition of CHD is typically precipitated by thrombosis occurring at the site of atherosclerotic plaque disruption. The subsequent thrombus formation is the most important mechanism leading to acute myocardial infarction (AMI).[2] Clinical manifestations of CHD include stable or unstable angina pectoris,

myocardial infarction, cardiac arrhythmias, congestive heart failure, and/or sudden death. Coronary thrombosis almost always brings about severe chest pain behind the sternum, often radiating towards the left arm. Conversely, plaque rupture leading to a coronary thrombosis often occurs in someone with no previous history of angina.[3]

Risk factors of the progression to coronary thrombosis include: family history of atherosclerosis, high cholesterol levels, elevated concentration of low-density lipoprotein (LDL), hypertension, smoking[1,4], diabetes mellitus[5], socioeconomic and lifestyle factors. Emotional stress or physical exertion can trigger coronary events.[6] Mortality rate from CHD increases with advancing age[7], and age-adjusted CHD rates are higher in men compared with women. Men develop CHD approximately 10 to 15 years earlier than women.[8] Smoking is more prevalent and hypertension less controlled in men. Overweight and obesity appear to be more potent risk factors in men, and the higher prevalence of abdominal obesity in men increases their risk for CHD.[2]

Case presentation

Case outline and history

A Thai 38-year-old man attending medical care at Health Promotion Clinic, HRH Princess Maha Chakri Sirindhorn Medical Center complained about 2-weeks duration of cough. His vital signs were normal. The physical examination was unremarkable except for some wheezing of both lungs. Blood tests revealed high Cholesterol (203 mg/dL) and LDL-direct (155 mg/dL). Chest roentgenography showed no pulmonary infiltration. The primary diagnosis was bronchitis, and then he was treated in emergency room with nebulizer of Berodual 1:3, and discharged 2 hours later. However, He had an appointment to follow up at OPD of Internal Medicine within 3 days. 15 hours later, he drove back to the hospital with clinical history of chest pain that was presented 2 hours before arrival. He gave an additional history of severe chest pain that located to the apex of the heart with poorly explained the symptoms. His vital signs were stable. He underwent EKG that revealed ST elevation in V1 and V2. Then, EKG were repeated one hour later and showed insignificant ischemic patterns. The cardiac biomarkers were not elevated. After EKG examination, he suddenly collapsed and had no vital signs. Advanced cardiac life support was performed for 1 hour. Finally, he was declared death 2 hours later.

External findings

The body height was 175 cm. and the body weight was 80 kg. with body mass index (BMI) of 26.1 kg/m2. There was non-fully developed rigidity with unfixed lividity of the body. The neck veins

were engorged. The chest wall showed a large abrasion and contusion, 10 cm in width and 30 cm in length, resulting from defibrillator pad injuries. There were no other external injuries.

Internal findings

Intercostal muscles were contused. The heart weighing 400 grams was slightly enlarged, and showed generalized petechial hemorrhage with a white patch, 3 cm. in diameter, at epicardium. The thickness of the right and the left ventricles were 0.5 and 2 cm, respectively. The papillary muscles of the left ventricle showed dark mottling area with white discoloration. Cut surfaces showed heterogeneous hemorrhagic patchy area. The right ventricle showed scattered, small whitish patches. The right coronary artery showed atherosclerotic plaque with recent thrombosis at the mid part, 1 cm. in length (fig. 1A). The proximal part of left anterior descending artery showed complete luminal occlusion (fig. 1B).

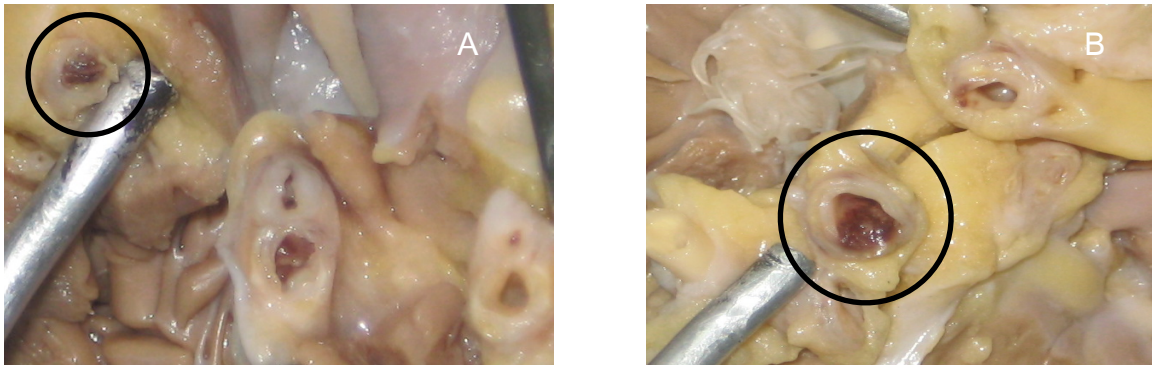

Fig.1 Arterial occlusion by a recent thrombus. (A) Right coronary artery, (B) left anterior descending artery.

Both lungs showed interlobar petechial hemorrhages, congestion and edema. The brain weighed 1,500 grams and was markedly congested and edematous. The cerebral arteries showed atherosclerosis. The liver weighed 1,600 grams and presented marked congestion. Stomach contained 20 ml. of dark brown heterogeneous fluid. Both congested kidneys weighed 400 grams and showed congestion with petechial hemorrhages. The other internal organs showed congestion.

Histopathological findings

The right coronary and left anterior descending arteries revealed a recent thrombus with narrowing of the lumen that caused acute myocardial infarction (fig. 2A, 2B). There was thickened tunica intima comprising proliferative fibroblasts and glycoprotein.

Discrete areas of fibrosis were seen in the thickened left ventricular wall, right ventricle and subendocardium (fig. 2C, 2D, 2E). The myocardial cells revealed hypertrophy with enlarged and hyperchromatic nuclei. Evidence of acute myocardial infarction was seen as irregular contraction bands necrosis with mild interstitial edema and neutrophil infiltration. Both lungs showed diffuse

capillary congestion with focal pulmonary edema (fig. 2F) and hemorrhage. Kidneys and spleen showed marked vascular congestion. The liver showed moderate degree of hydropic degeneration and mild fatty change.

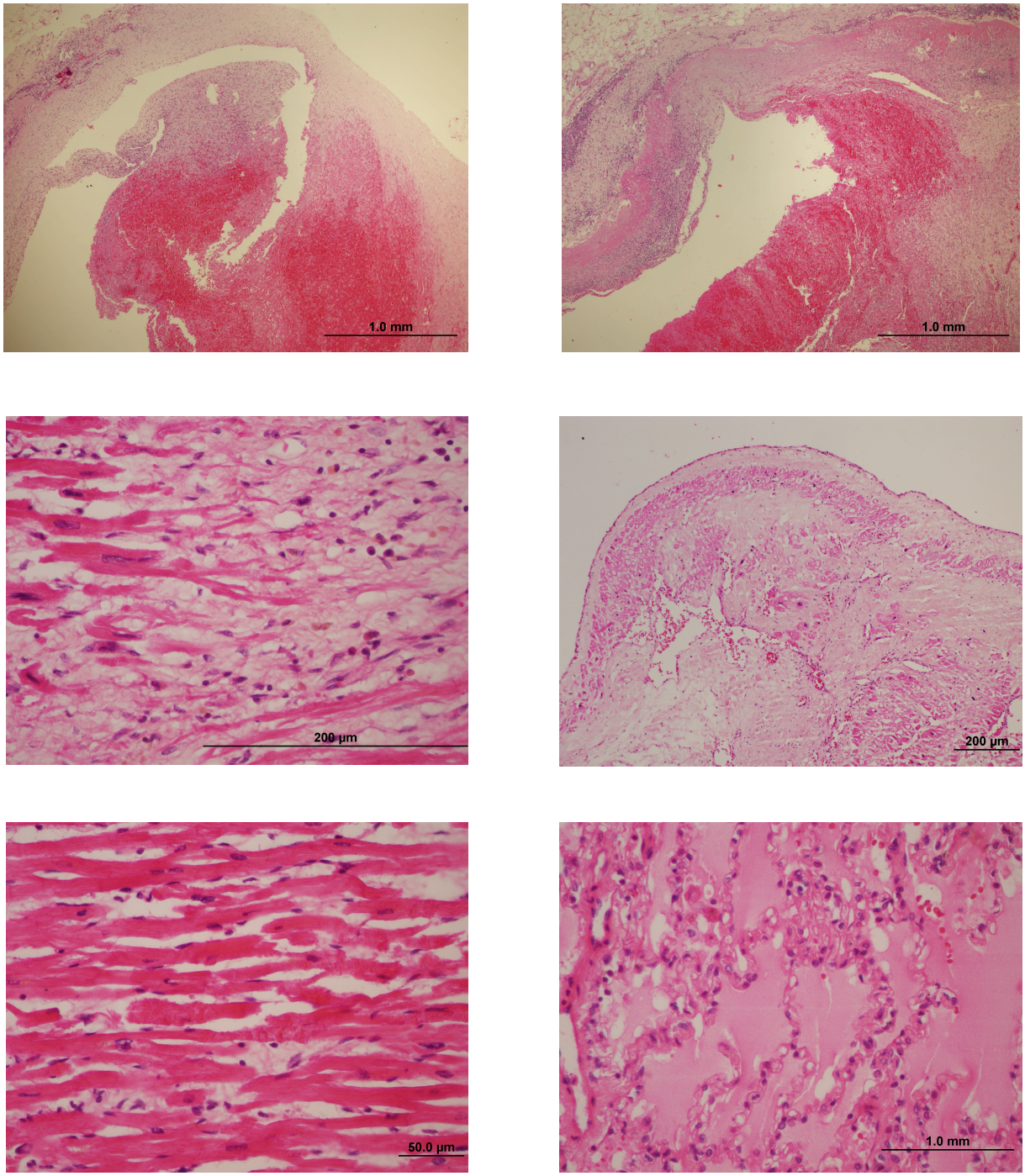

Fig. 2 Histopathology. Thrombus in right coronary artery (A) and left anterior descending artery (B), healed myocardial infarction in left ventricular wall (C), and subendocardium (D), recent myocardial infarction (E), diffuse pulmonary edema (F).

Toxicological findings

Peripheral blood alcohol concentration was negative. Other toxicological examinations revealed negative reaction for all items tested.

Discussion

This case was a 38-year-old man dying from acute coronary thrombosis with the evidence of chronic myocardial infarction. His risk factors for atherosclerosis were a history of cigarette smoking, hypercholesterolemia and high LDL-C. From pervious study, A.P. Burke et al1 claimed that it was a risk factor in 75% of men with acute thrombosis, and the serum total cholesterol was markedly elevated. The LDL-cholesterol was also the independent risk factor to demonstrate significant concurrent.[9] It was interesting that he did not complain about clinical of chest pain at OPD but dyspnea. The clinical was developed 15 hours later after treatment with beta-blockers. Kohei et al study[3] showed that 60 – 70% of coronary heart disease patients had sudden death or acute myocardial infarction (MI) as the first clinical manifestation. Patients with coronary heart disease who were asymptomatic and had no warning symptoms would have an even greater risk of a fatal outcome than those with stable symptoms. The subsequent 12 leads EKG during the second visit showed insignificant ischemic pattern. The cardiac biomarkers (CPK, CK-MB, Troponin-T) were not elevated. Both of these might come from early acute stage of myocardial infarction. In previous study, Troponins were the preferred biomarkers for diagnosis of acute coronary syndrome and myocardial infarction. However, they were also elevated in the patients with left ventricular hypertrophy, heart failure, chronic kidney disease and diabetes.[4] The increase of Troponins concentrations in individuals without known CHD was related to an increased risk of death. The usefulness of Troponin-T measurement was to identify high-risk subjects in the primary prevention of CHD. In the population-based Dallas Heart Study, Troponin-T values ≥0.01 μg/L were found in 0.7% of the population and the level was rising to 100% at 6 hours.[10]

Histological findings confirmed the diagnosis of chronic ischemic heart disease by the evidence of myocardial fibrosis and recent myocardial infarction as a result of thrombosis in the right coronary and left anterior ascending coronary arteries. A.P. Burke study revealed that the incidence of population with coronary heart disease who had died suddenly from acute coronary thrombosis and the stable plaque was 52.21% and 47.79%, respectively.[1]

From this case, the physicians should recognize that the middle-aged man may die from acute coronary artery occlusion leading to acute myocardial infarction, despite of the fact that clinical

manifestation and investigations were negative. Clinical skills to find clues to diagnose coronary artery occlusion consist of history taking, physical examination, laboratory investigation and data interpretation. The treatment is limited by time and experience of physicians. If the case is not typical, a second opinion or consulting specialists is recommended.

Conclusion

It is very important to find all available courses to determine the exact cause of death. The most frequent cause of sudden death is coronary heart disease, even in the young. The postmortem diagnosis of coronary thrombosis is very important because the family of the deceased will need counseling and the first degree relatives should be suggested to perform a possible screening test to prevent other sudden deaths. In addition, the diagnosis by gross and microscopic findings may be difficult in some cases. Obviously, the forensic pathologist has a responsibility to accurately record data to identify the cause of death with reasonable degree of credibility.

Conflict of interests

The authors declare that we have no conflict of interests.

Reference

1. A.P. Burke, A. Farb, G.T. Malcom, Y.H. Liang, J. Smialek, R. Virmani. Coronary risk factors and plaque morphology in men with coronary disease who died suddenly. N Engl J Med. 1997; 336: 1276–82.
2. Jari A. Laukkanen, Sudhir Kurl, Timo A. Lakka, Tomi-Pekka Tuomainen, Rainer Rauramaa, Riitta Salonen, et al. Exercise-induced silent myocardial ischemia and coronary morbidity and mortality in middle-aged men. Journal of the American College of Cardiology. 2001; 38 (1): 72-9.
3. Kohei Wakabayashi, Cedric Delhaye, Gabriel Maluenda, Itsik Ben-Dor, Manuel A. Gonzalez, Sara D. Collins, et al. Prognosis of Asymptomatic Coronary Artery Disease after Percutaneous Coronary Intervention. The American Journal of Cardiology. 2010; 105 (11): 1507-12.
4. Toshiaki Otsuka, Tomoyuki Kawada, Chikao Ibuki, Yoshihiko Seino, Tokyo, and Chiba. Association between high-sensitivity cardiac troponin T levels and the predicted cardiovascular risk in middle-aged men without overt cardiovascular disease. American Heart Journal. 2010; 159 (6): 933-6.
5. Angelo Avogaro, Gianpaolo Fadini, Saula Vigili de Kreutzenberg, Antonio Tiengo. Coronary heart disease in diabetes. International Congress Series. 2007; 1303: 70-3.
6. Demosthenes B. Panagiotakos, Christina Chrysohoou, Christos Pitsavos, Alessandro Menotti, Anastasios Dontas, John Skoumas, et al. Forty-years (1961–2001) of all-cause and coronary heart disease mortality and its determinants: the Corfu cohort from the Seven Countries Study. International Journal of Cardiology. 2003; 90 (1): 73-9.
7. Hanyu Ni, Sean Coady, Wayne Rosamond, Aaron R. Folsom, Lloyd Chambless, Stuart D. Russell, et al. Trends from 1987 to 2004 in sudden death due to coronary heart disease: The Atherosclerosis Risk in Communities (ARIC) study. American Heart Journal. 2009; 157 (1): 46-52.

8. George Fodor, Rayka Tzerovska. Coronary heart disease: is gender important? The Journal of Men's Health & Gender. 2004; 1 (1): 32-7.
9. Victor Y. Cheng, Arik Wolak, Ariel Gutstein, Heidi Gransar, Nathan D. Wong, Damini Dey, et al. Low-Density Lipoprotein and Noncalcified Coronary Plaque Composition in Patients with Newly Diagnosed Coronary Artery Disease on Computed Tomographic Angiography. The American Journal of Cardiology. 2010; 105 (6): 761-6.
10. E. Giannitsis, M. Becker, K. Kurz. High-sensitivity cardiac troponin T for early prediction of evolving non–ST-segment elevation myocardial infarction in patients with suspected acute coronary syndrome and negative troponin results on admission. Clin Chem. 2010; 56: 642–50.