中文題目：在一位胰臟癌患者，同時罹患急性肺栓塞與 ST 段上升急性心肌梗塞

英文題目：Acute pulmonary embolism concomitant with ST-elevation myocardial infarction in patient with pancreatic cancer

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Case presentation：Acute pulmonary embolism (PE) and ST-elevation myocardial infarction (STEMI) are life threatening diseases in patients with acute chest pain Although PE and STEMI have different pathophysiology, PE maybe concomitant with STEMI in rare patients. Here, we presented a 62 year-old male with pancreatic cancer had acute PE and STEMI. He presented with shortness of breath and right lower leg swelling for 2 days. Physical examination revealed heart rate 86/min, respiratory rate 18/min, blood pressed 120/80 mmHg, grade III/VI pansystolic murmur over left lower sternal border, and right lower leg swelling. The laboratory tests were as follows: white blood cell count 9600/μL, hemoglobin 13.8 g/dL, platelet 133000/μL, cardiac troponin I 0.01 μg/L, brain natriuretic peptide 20.8 ng/L, D-dimer 39.75 nmol/L, creatinine 0.96 mg/dL, protein C 87%, protein S 109%, anti-thrombin 100%, collating factor VIII 166%, anti-β2 glycoprotein 0.9 U/ml, homocysteine 11.4 μmol/L, and carcinoembryonic antigen 261.78 ng/ml. The chest computed tomography (CT) scan revealed acute pulmonary embolism involved bilateral pulmonary arteries. Due to hemodynamic stable, our patient received subcutaneous enoxaparin and combination with oral warfarin. After treated 5 days, he had sudden onset of typical cardiac chest pain and cold sweating. The 12 lead electrocardiogram showed ST-segment elevation on lead II, III, aVF. The emergent coronary angiogram showed total occlusion of middle right coronary artery. He was successfully treated with coronary intervention and stent deployment. Five days later, he was discharged and received triple antithrombotic therapy included aspirin, clopidogrel, and warfarin.

Discussion：In the aspect of pathophysiology, acute PE are frequently accompany with deep venous thrombosis and STEMI are common caused from rupture of coronary plaque. Although the incidence of patients with malignancy diagnosed deep venous thrombosis or PE are nearly 20%, patients with cancer and acute myocardial infraction (AMI) are rare. Some cases with gastro-intestinal, hematological, germinal system malignancy were reported had AMI. Dieckmann, et al. demonstrated that majority patients who diagnosed testicular cancer and AMI did not have traditional atherosclerotic risk factors and coronary plaque. Those cancer patients with AMI were probable caused from acute thromboembolic events rather than from coronary atherosclerotic stenosis. The reasons why patients with malignancy have hypercoagulable state may be that cancer cells directly act on thrombin, enhance the interaction between cancer cell and host blood cell through adhesion molecules, and activate coagulation cascade and inflammatory cytokines.

In rare cases, acute PE were be found concomitantly with STEMI. The possible mechanism are

included coagulation factor defect, hyper-homocysteinemia, and patent foramen ovale. In our presented patients with pancreatic cancer, he did not had intra-cardiac shunting, however his collating factor VIII was 166% (normal 60 to 150%). Our patient concomitant with PE and STEMI may be related to hyper-coagulation status. Triple antithrombotic therapy was expected prominent efficacy for hypercoagulability, but side effect of bleeding tendency was should be consideration.