Case Report

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Percutaneous Coronary Intervention in Patient with Protein C Deficiency

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Abstract:

The incidence of acute STEMI in patients with protein C deficiency is rare. Treatment of acute STEMI in patients with coagulation disorders is like that of the general population. Medication after the occluded artery has been performed by balloon and stent placement, especially for patients who take direct oral anticoagulants (DOACs) and still have thromboembolism. It is something that needs to be planned for treatment and further study.

Keywords: Acute STEMI, Protein C deficiency, Percutaneous Coronary Intervention

Introduction

The incidence of acute coronary syndrome (ACS) in developed countries tends to decrease due to the control of risk factors.[1,2] For Thailand, there is a tendency for deaths from ACS to increase steadily every year. In 2011 there was a death rate of 22.5 per 100,000 population, rising to 31.8 in 2017. However, the mortality rate in hospitals, especially ST-elevation myocardial infarction (STEMI) patients, decreased steadily from 17% in 2002 to about 10% in 2018.[3]

Acute ST-segment elevation myocardial infarction (STEMI) in the context of protein C deficiency is a rare. Protein C deficiency is a medical condition that affects blood coagulation and is considered an inherited thrombophilia, which means there is an increased tendency to develop abnormal blood clots. Protein C is a natural anticoagulant that regulates blood clotting and prevents excessive clot formation. When a blood vessel is injured, complex reactions occur to form a blood clot, which helps stop bleeding. However, this clotting process must be carefully balanced to prevent excessive clot formation, which can lead to harmful blockages in blood vessels (thrombosis).[4] When it occurs in the setting of acute STEMI, it can further complicate in management of the condition. In individuals with protein C deficiency, there is a reduced amount or impaired function of protein C, making it harder to regulate the clotting process. This deficiency can be either congenital (inherited from one or both parents)[5] or acquired (developed later in life due to other medical conditions or treatments).[6] Symptoms and complications of protein C deficiency can vary from mild to severe. They may include deep vein thrombosis (DVT), pulmonary embolism

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(PE), skin necrosis, and increased risk of miscarriage in pregnant women.[7-10]

Treatment for protein C deficiency aims to prevent clot formation. It may involve anticoagulant medications such as warfarin or direct oral anticoagulants (DOACs).[11,12] Supportive measures such as compression stockings may be recommended to improve blood flow in the legs and reduce the risk of DVT.[13]

Case report

A 44-year-old man presented with acute cardiac chest pain for about three hours. He had underlying ischemic stroke, protein C deficiency, hypertension and dyslipidemia. Current medications included Apixaban (5 mg) 1 tab orally BID pc, Manidipine (20 mg) 1 tab orally OD pc, Losartan (100 mg) 1 tab orally OD pc, and Atorvastatin (40 mg) 1 tab orally OD HS. Physical examination revealed an unremarkable study. Electrocardiography (ECG) showed ST segment elevation at II, III, and aVF as in Figure 1. Echocardiography showed inferior wall hypokinesia, no valvular heart disease with LVEF 61%. Coronary angiography showed an occluded lesion at the right coronary artery (RCA) as in Figure 2. The left coronary artery was not shown obvious stenosis. The RCA was engaged with a 6-French extra-backup catheter (RBU3.5), and then a Runthrough hypercoat wire (Terumo) was passed to the distal part of the RCA. PCI was done using a Sapphire II pro balloon 2.0 × 20 mm and Terumo Ryurei SC balloon 3.0 × 20 mm pre-dilatation at mid to distal RCA lesion. IVUS showed many intraluminal thrombi with minimal plaque at the endothelium. Blood clot was removed by a Thrombuster Pro catheter. The mid to distal RCA lesion was stented with 4.0×9 mm, 3.5 × 36 mm, and 3.5 × 19 mm Biometrix alpha stents. Post stent angiogram shows in Figure 3.

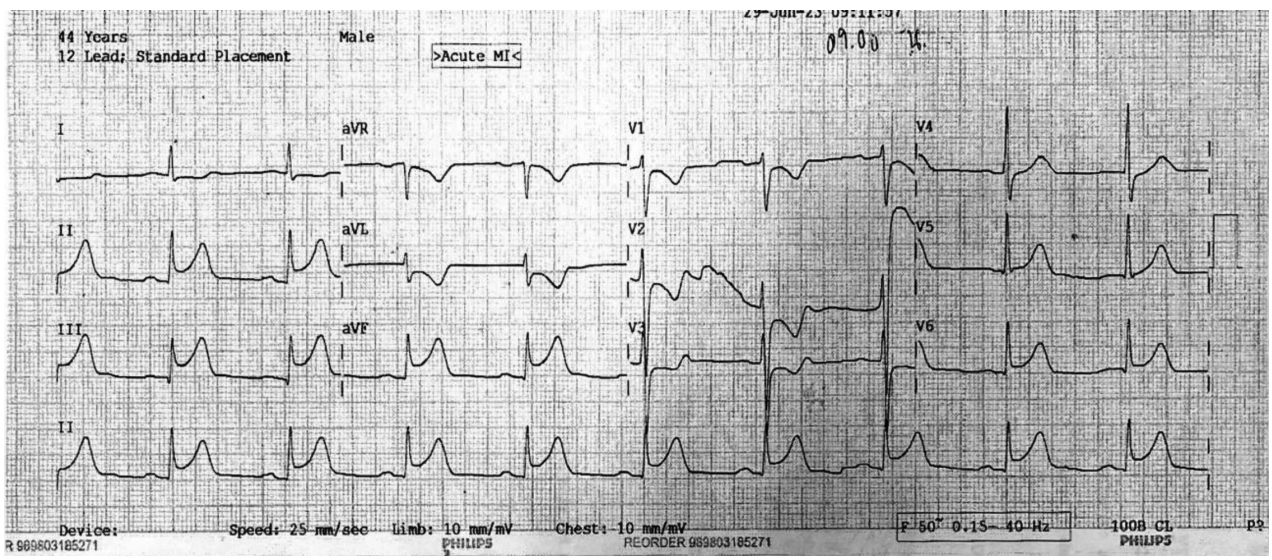

Figure 1 ECG shows ST segment elevation at II, III, and aVF

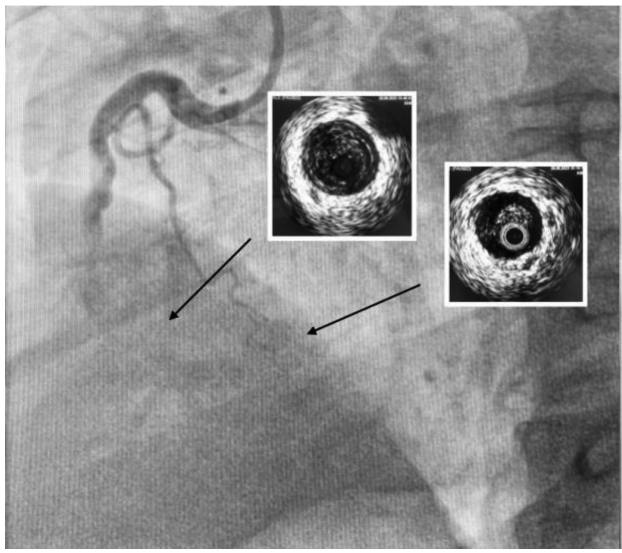

Figure 2 Coronary angiography shows occluded lesion at the right coronary artery (RCA)

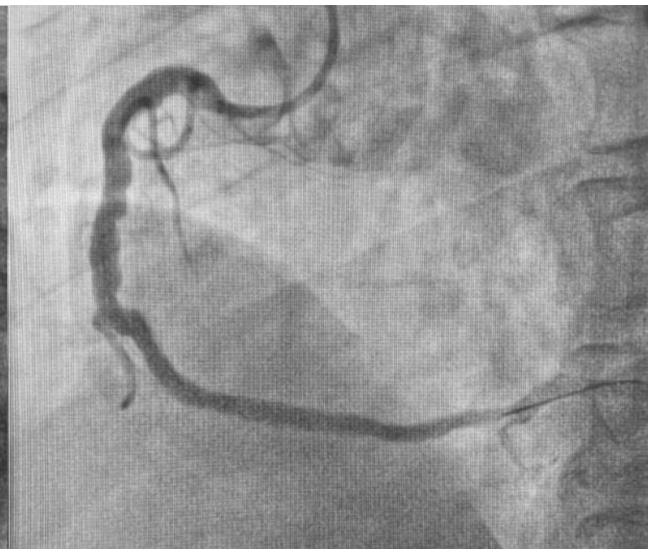

Figure 3 Post stent angiogram

Discussion

Acute ST-segment elevation myocardial infarction (STEMI) is a severe form of heart attack characterized by complete or near-complete occlusion of a coronary artery, resulting in the death of heart muscle (myocardium) due to a lack of blood supply.[14] The pathophysiology of STEMI involves several key processes.[15,16] Early intervention can significantly improve outcomes and reduce the risk of complications.[17]

Acute ST-segment elevation myocardial infarction (STEMI) in the context of protein C deficiency is a rare and complex situation. Protein C deficiency is an inherited or acquired disorder that affects the body's ability to regulate blood clotting.[18] When it occurs in the setting of acute STEMI, it can further complicate the pathophysiology and management of the condition. Here's how protein C deficiency may contribute to the development and progression of acute STEMI:

1. Hypercoagulability: Protein C is a natural anticoagulant protein that plays a crucial role in inhibiting blood clot formation. Its deficiency can lead to a prothrombotic state, making individuals more prone to developing blood clots, including those that can cause a STEMI.

2. Increased thrombus formation: In protein C deficiency, there is a higher likelihood of forming blood clots, especially in situations where there is atherosclerosis and plaque rupture, as seen in STEMI. The reduced ability to control clot formation can result in more extensive and occlusive thrombi within the coronary arteries.

3. Impaired fibrinolysis: Protein C deficiency can also affect the fibrinolytic system, which is responsible for breaking down blood clots. This impairment can make it challenging to dissolve or break apart the thrombus once it forms, further exacerbating the blockage in the coronary artery.

4. More severe clinical presentation: The combination of protein C deficiency and acute STEMI may lead to a more severe clinical presentation, with larger infarct size, increased risk of complications, and potentially worse outcomes.

5. Management challenges: Protein C deficiency poses unique challenges in the management of acute STEMI. Traditional anticoagulant therapies like heparin or

antiplatelet agents may need to be carefully adjusted or supplemented to address the underlying coagulation disorder.

Management of acute STEMI in individuals with protein C deficiency typically involves a multidisciplinary approach, including cardiologists and hematologists. The treatment may involve anticoagulant medications that are specific to protein C deficiency, in addition to standard therapies for STEMI, such as reperfusion strategies like PCI or thrombolytic therapy.[19]

DOACs (Direct oral anticoagulants) are a class of medications used to prevent and treat blood clots. They work by inhibiting certain clotting factors in the blood, thereby reducing the risk of clot formation. The DOACs include dabigatran, rivaroxaban, apixaban, and edoxaban. The connection between DOACs and protein C deficiency lies in the mechanism of action of these medications. DOACs inhibit specific clotting factors like thrombin (dabigatran) or factor Xa (rivaroxaban, apixaban, edoxaban) to prevent clot formation. However, this inhibition may exacerbate the underlying tendency to bleed in individuals with protein C deficiency, as they already have a reduced ability to regulate clotting.[12] For individuals with protein C deficiency, anticoagulant therapy requires careful consideration. Traditional anticoagulants like warfarin, which target multiple clotting factors (including vitamin K-dependent factors like protein C), have been used cautiously in these patients. The management of anticoagulation in protein C deficiency is often complex and individualized, considering factors like the severity of the deficiency, the clinical context, and the risk of bleeding versus clotting.[20]

It is essential for individuals with protein C deficiency to work closely with their healthcare provider, preferably a specialist in hematology or thrombosis, to determine the most appropriate anticoagulation strategy for their specific case. Close monitoring and regular follow-up are necessary to optimize treatment outcomes and minimize potential risks.

Conclusion

Percutaneous coronary intervention (PCI) for obstructive lesion in protein C deficiency is uncommon. Long-term anticoagulant is still necessary in patients with protein C deficiency.

Potential conflicts of interest

The author has no relevant conflict of interest to disclose.

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