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Ischemic Acute Myocardial Infarction In A Young Male C And S Protein Deficiencies That Run In Families

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Abstract

We describe a 33-year-old man whose acute anterolateral myocardial infarction (MI) was the first cardiac symptom of hereditary protein C and S deficits. The MI was brought on by thrombotic blockage of the left anterior coronary artery (LAD).

This case study demonstrates that acute coronary syndrome in young people may result from conditions other than ruptured atherosclerotic plaque. For doctors to investigate various etiologies in the differential diagnosis, such as hypercoagulable state in young patients with myocardial infarctions, they must have this knowledge. Once the diagnosis has been made, it will be possible to manage the situation more effectively and prevent recurrent incidents. Additionally, family screening can result in a preventive strategy for those who carry this mutation.

Introduction

Since 15-20% of these people have normal or nearly normal coronary arteries (N/NNCAs), the absence of thermodynamically significant coronary artery disease in this age range is not as uncommon as the occurrence of MI in patients under the age of 40. Due to the significant damage that results from its rapid development and lack of collateral coronary circulation, it is linked to devastating effects. Preventing recurrence requires accurate diagnosis and care of the underlying cause. Numerous other potential contributing factors have been discovered in young individuals with myocardial infarction in addition to the conventional risk factors for atherosclerosis. These include the use of oral contraceptives by young women, the use of cocaine and marijuana, a mutation in factor V called Leiden, and a lack of protein C [1–5].

Case Presentation
A 33-year-old man who had previously been diagnosed with protein C and S deficiency, pulmonary embolism, and tobacco use presented to the emergency room after witnessing a sudden cardiac arrest that was followed by chest pain while he was walking around at home and then turned into a seizure. His mother, who was diagnosed with a deep vein thrombosis, and his brother, who had a myocardial infraction in his 30s, both have a family history of clotting disorders. He smokes a half-pack of cigarettes per day on average, and he occasionally drinks alcohol and uses marijuana.

In 2007, after receiving treatment for a pulmonary embolism and protein C and S deficits with warfarin for six months, he decided not to continue receiving systemic anticoagulation for the subsequent eight years. He had agonal breathing and tense posture when EMS arrived, and a cardiac monitor showed a rhythm consistent with ventricular fibrillation. Advanced cardiovascular life support (ACLS) protocol was immediately started, and he received one round of 200-joule defibrillation before being taken to the emergency room.

During the trip, there were two further ventricular fibrillation episodes treated successfully with two rounds of 200 J defibrillation. To safeguard his airway, he underwent intubation. An intubated male patient who was not responding to questions during a physical examination had blood pressure that was 123/67mm Hg and a heart rate of 74.S3 was audible on auscultation and saturation was at 100% with supplemental oxygen at 50%; there was no sign of volume overload. Initial laboratory testing revealed leukocytosis of 21,100 k/l with troponin levels of 0.03 ng/ml, peaking at 15.61 ng/ml. Drug tests on urine came up negative.

Discussion

Only 2-8% of people who experience an acute myocardial infarction are under the age of 40; this makes acute coronary syndrome in young adults a rare illness [6,7]. Usually, males are more affected than female represent just 6% of this age group [8].

The most common cause of acute coronary syndrome in young people is atherosclerosis; however, many other variables, including insulin resistance, diabetes, family lipid disorders, hypertension, smoking, and HIV infection, can hasten and accelerate this process [9]. Vasospasm, illicit drug use, congenital coronary artery malformations, spontaneous coronary artery dissection, and hypercoagulable states, such as our case, are non-atherosclerotic causes of acute coronary syndromes.

When there is no evidence of coronary illness, doctors should consider an inherited thrombophilia, especially in children who arrive with acute coronary syndrome.Only one (0.4%) of 255 consecutive patients who had survived a STEMI and were under 35 years old were found to have a protein C deficiency (10) Proteins C and S play a part in inactivating the clothing factors Va and VIIa that contribute to thrombin production. In their absence, people are far more likely to develop pulmonary

embolism and venous thrombosis.

This case study demonstrates the need of diagnosing protein C and S deficiency in young individuals with myocardial infarction in order to treat them more effectively and prevent future attacks.

Additionally, family screening can result in a preventive strategy for those who carry this mutation. Patients and concerned family members must be made aware of the dangers, symptoms, and signs of thrombosis as well as the value of preventative measures. Decisions regarding the prescription of antithrombotic prophylaxis may also be influenced by education and counselling concerning the patient's carrier status.

Conclusion

Young adults with normal coronaries and no significant underlying risk factors for coronary artery disease are susceptible to sudden myocardial infarction caused by a hypercoagulable state brought on by protein C and Sdeficiency without the proper anticoagulant medication. It is essential to increase patient awareness. Comprehensive cardiovascular events could be avoided with family screening.

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