

Acute ST-Elevation Myocardial Infarction in a 32-Year-Old Male Less than 2 Months after a Normal Coronary Computed Tomography Angiography

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1. Abstract

1.1. Background: Acute myocardial infarction (AMI) is an uncommon finding in young patients under age 35 and determination of etiological factors can be challenging. Coronavirus disease-2019 (COVID-19) and misuse of androgenic anabolic steroids (AAS) have both been associated with thrombotic events.

1.2. Case Summary: We report the case of a 32-year-old male with a family history of premature coronary artery disease (CAD), smoking and misuse of AAS, who two months prior to admission was assessed with a coronary computed tomography angiography (CCTA) with normal findings and coronary calcium score of zero. The patient was admitted with symptoms of ST-segment elevation myocardial infarction (STEMI) and concurrent COVID-19 infection. Due to age, history and paraclinical findings, pericarditis was initially suspected. Coronary angiography showed mid-segment stenosis of 50% at the LAD, evaluated by optical coherence tomography (OCT) showing signs of plaque rupture with local thrombus formation. Percutaneous Coronary Intervention was performed, with implantation of one drug eluting stent in the LAD.

1.3. Conclusion: Our case highlights that misuse of AAS and COVID-19 infection both constitutes predisposing risk factors to thrombotic events, even in young individuals with a very low atherosclerotic disease burden.

2. Introduction

In the rare cases of Acute Myocardial Infarction (AMI) in patients under age 35, etiological factors are less studied, but are suggested to be an accumulation of cardiometabolic risk factors, such as male gender, obesity, hypercholesterolemia, smoking and family history of premature Coronary Artery Disease (CAD). An association between misuse of anabolic steroids and AMI have also been reported in younger individuals. Likewise, a transient increase in risk of AMI have been reported in relation to influenza, pneumonia, and other chest infections [1-4], and recent studies indicate a pathophysiological association between Coronavirus disease-2019 (COVID-19) and acute coronary syndromes [5-7].

Coronary angiography is regarded as the gold standard for evaluation of acute coronary artery stenosis. Coronary computed tomography angiography (CCTA) is an alternative diagnostic tool to detect coronary atherosclerotic plaques and CAD, due to a high negative predictive value, and is recommended in the European Society of Cardiology guideline for the assessment of CAD in patients with suspected stable angina pectoris [8].

Herein, we report the case of a 32-year-old male presenting with an acute anterior ST-elevation myocardial infarction (STEMI) and concurrent COVID-19 infection, despite a CCTA with normal conditions performed two months priorly.

3. Case Presentation

A 32-year-old previously healthy man presented to the emergency department due to chest pain. The patient reported daily use of anabolic steroids but denied use of other substances including cocaine. He had no history of hypertension, diabetes, heart disease, dyslipidemia, autoimmune disease, nor of prescribed medication. He also reported active smoking for approximately 10 years, 5-6 cigarettes per day, heavy drinking twice a month and a family history of CAD, with his father having an AMI at 48 years of age. He reported drinking large amounts of alcohol the evening prior to admission. Also, the patient was PCR-tested positive for COVID-19 five days prior to admission, although asymptomatic. Due to two prior episodes with chest pain and palpitations, the patient had received a CCTA two months before, showing normal coronary conditions with a calcium score of 0.

Upon admission, he reported an acute onset during sleep and a two-hour history of intermittent retrosternal discomfort with radiating pain and burning sensation to the left arm, hand, and fingers. The pain was of compressing nature and had an intermittent intensity of 5 to 9 on a numeric rating scale (NRS). He refused shortness of breath, fever and coughing but reported some nausea. Physical examination was normal. Due to symptoms and presentation of ST-segment elevations in ambulance electrocardiogram (ECG), an oral bolus of acetylsalicylic acid was administered, and the patient was conferred by ambulance staff with an invasive center regarding coronary angiography but was directed to a local emergency department for further initial tests.

The patient arrived at the emergency department 2 hours after symptom onset and initial laboratory tests revealed a cardiac troponin I level of 11 ng/L (reference interval <45 ng/L, high-sensitivity cardiac TnI Siemens Atellica), normal levels of all organ biomarkers, including hematology, infection parameters and kidney-, liver- and coagulation function, normal d-dimer, total cholesterol 1.9 mmol/L, low-density lipoprotein-cholesterol level unmeasurable, high-density lipoprotein-cholesterol <0.52 mmol/L,

and triglycerides 1.04 mmol/L. Initial ECG testing demonstrated sinus rhythm, heart rate 63 beats per minute, ST-elevations in lead II, III, aVF and V3-V6 (1-2 mm) and ST-depressions in aVR and aVL (1 mm) (Figure 1). Bedside echocardiography showed normal conditions with a preserved Left Ventricular Ejection Fraction (LVEF) and no pericardial effusion. Pericarditis was suspected due to age, history, diffuse ST-elevations in ECG though no PR depression was observed, normal LVEF and troponin level. The patient was admitted to the in-house cardiological department for further observation. Three hours after admission, troponin had increased to 90 ng/L and further dynamical changes were observed in ECG with extensive ST-elevations in II, III, aVF and V3-V6 increasing to a range of 1-4 mm (Figure 2). Oral nitroglycerin was administered, with instant effect on chest pain and a subsequent decrease of ST-elevations on ECG. After six hours of admission, chest pain was persistent and the cardiac troponin level had increased to 181 ng/L, thus intravenous nitroglycerin and subcutaneous fondaparinux was administered. The patient was re-conferred with invasive center and received a subacute coronary angiography the next day, approximately 28 hours from admission. The cardiac troponin I level peaked at 70800 ng/L after 32 hours of admission. Coronary angiography revealed a mid-segment stenosis of 50% at the LAD, evaluated by optical coherence tomography (OCT) showing signs of plaque rupture with a local thrombus (Figure 3). Percutaneous Coronary Intervention (PCI) was performed, with the implantation of one drug eluting stent in the LAD. In the RCA, an insignificant post ostial plaque with a 25% stenosis was observed, with no further actions required. The patient received an oral prasugrel bolus and infusion of efbatide for 18 hours and was subsequently prescribed prasugrel 10 mg once a day for 12 months and life-long treatment with acetylsalicylic acid, together with atorvastatin. On discharge, echocardiography showed a preserved LVEF. The following 12 months, the patient contacted the emergency department more than 15 times due to chest pain, although without any indications of further coronary events.

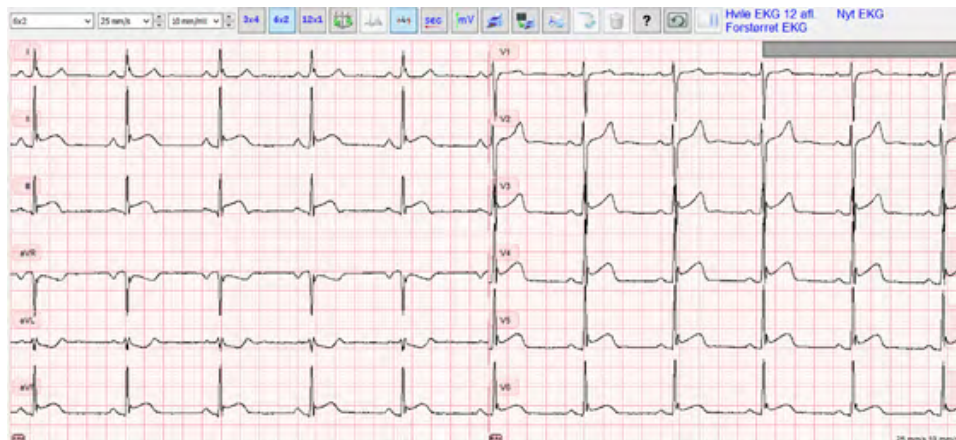


Figure 1: Initial 12-lead ECG testing demonstrated sinus rhythm, heart rate 63 bpm, ST-elevations in lead II, III, aVF and V3-V6 (1-2 mm) and ST-depressions in aVR and aVL (1 mm).

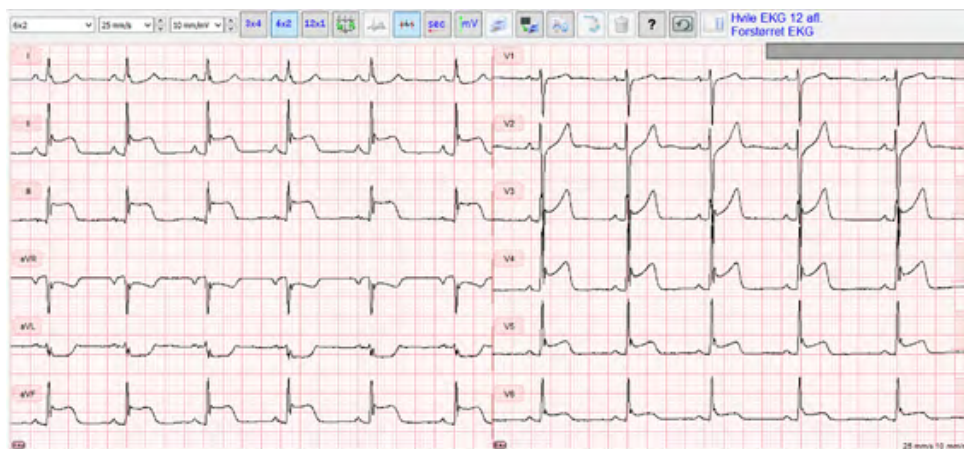


Figure 2: 12-lead ECG recorded 3 hours after admission showing sinus rhythm, heart rate 68 bpm, extensive ST-elevations in inferior leads (II, III and aVF) of 4 mm and in anterior-lateral leads (V3-V6) of 1-4 mm and ST-depressions in aVR and aVL of 1-2 mm.

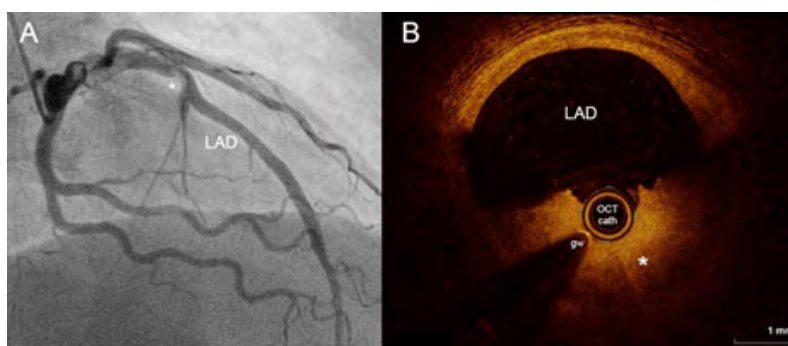


Figure 3: Panel A: Coronary angiogram showing stenosis (*) in proximal left anterior descending artery (LAD). Panel B: Intravascular imaging with optical coherence tomography at the site of the stenosis. Acute thrombus (*) is seen partially obstructing the lumen of the LAD. OCT cath: imaging catheter, gw: guide wire.

4. Discussion

We present a case of STEMI in a COVID-19 positive young male with cardiac risk factors of family history of CAD, smoking and misuse of anabolic steroids, but with a normal CCTA and a calcium score of zero two months prior to admission. AMI in young individuals under 35 years are very rare [9, 10]. The patient developed STEMI despite not having hypertension, diabetes mellitus and hypercholesterolemia, including hyperlipoproteinemia(a) and with a recent normal CCTA.

Non-invasive anatomical visualization of the coronary artery lumen, assessing coronary artery stenoses, is possible with CCTA, which has a high negative predictive value and thus provides a high accuracy for the exclusion of obstructive CAD [8, 11]. Coronary Artery Calcium (CAC) scoring, using noncontrast computed tomography, is a clinically noninvasive estimate of CAD burden, especially due to calcified plaques. Reportedly, among symptomatic patients with stable angina pectoris, a CAC score of zero identifies low-risk patients with very low adverse event rates [12, 13]. However, non-calcified plaques are reportedly more frequent in young individuals, causing doubt regarding the sensitivity of CAC scoring in this age group [14]. Similar doubt regarding sensitivity of CCTA detection of non-calcified plaques in young individuals, however, has not been confirmed searching the literature.

Villines et al. found an adverse event rate of 0.8% within a median of 2.1 years in patients with a CAC score of zero and non-obstructive CAD verified on CCTA [15]. Thus, AMI in young individuals under 35 years are very rare [10], especially with a recent normal CCTA, as presented here. This knowledge, together with findings of diffuse ST-elevations in several leads in the ECG, preserved LVEF and initial normal cardiac troponin levels, caused clinicians to suspect pericarditis initially.

The patient reported a daily misuse of Androgenic Anabolic Steroids (AAS). Several case reports of AMI in young individuals suggest an association between the use of AAS and vascular thrombosis, ranging from AMI and stroke to sudden cardiac death [16-22]. The pathophysiology is suggested to involve dyslipidemia (elevated LDL and reduced HDL cholesterol) causing accelerated atherosclerosis, coronary thrombosis due to increased platelet aggregation and coronary vasospasm [18]. Although our patient had a low level of total cholesterol at admission, he had cholesterol parameters similar to those described in the literature for misuse of AAS two months prior, with total cholesterol of 5.1 mmol/L, HDL of 0.67 mmol/L and LDL of 3.4 mmol/L, suggesting some degree of dyslipidemia in this patient.

The patient also had an ongoing infection with COVID-19, although asymptomatic. COVID-19 has been shown to predispose

patients to a prothrombotic state [7, 23]. The underlying etiology is probably caused by direct endothelial cell damage and platelet aggregation due to an exaggerated inflammatory response causing intimal inflammation [24, 25]. A recent large Swedish study found an increased COVID-19 associated risk of AMI with an incidence rate ratio of 8.44 in the first week of infection [26]. Another study found strong indication of a higher intracoronary thrombus burden in COVID-19 patients presenting with STEMI [27]. Typical findings of hypercoagulability, although primarily observed in critical ill patients with COVID-19, are increased D-dimer, a modest decrease in platelet count, prolonged prothrombin time and elevated lactate dehydrogenase [23]. These findings were not observed in our patient although prothrombin time was not measured. Also, our patient was asymptomatic.

5. Conclusion

Evidence suggests that both the misuse of AAS and infection with COVID-19 is strongly associated with vascular thrombotic events. We propose that the cause of the AMI was a result of reported risk factors including family history of CAD and smoking with contribution of AAS use and COVID-19. The case is special as coronary angiography with intravascular imaging showed obstructive plaque rupture in LAD and non-obstructive plaque formation in RCA, none of which was observed on CCTA. Our case serves to illustrate that misuse of AAS and COVID-19 infection both constitutes predisposing risk factors to thrombotic events, even in young individuals with low atherosclerotic disease burden. Also, clinicians should be aware that in rare cases, AMI can be seen despite a recent normal CCTA.

References

- Warren-Gash C, Hayward AC, Hemingway H, Denaxas S, Thomas SL, Timmis AD, et al. Influenza infection and risk of acute myocardial infarction in England and Wales: a CALIBER self-controlled case series study. *J Infect Dis*. 2012; 206(11): 1652-9.
- Clayton TC, Thompson M, Meade TW. Recent respiratory infection and risk of cardiovascular disease: case-control study through a general practice database. *Eur Heart J*. 2008; 29(1): 96-103.
- Smeeth L, Thomas SL, Hall AJ, Hubbard R, Farrington P, Vallance P. Risk of myocardial infarction and stroke after acute infection or vaccination. *N Engl J Med*. 2004; 351(25): 2611-8.
- Kwong JC, Schwartz KL, Campitelli MA, Chung H, Crowcroft NS, Karnauchow T, et al. Acute Myocardial Infarction after Laboratory-Confirmed Influenza Infection. *N Engl J Med*. 2018; 378(4): 345-53.
- Modin D, Claggett B, Sindet-Pedersen C, Lassen MCH, Skaarup KG, Jensen JUS, et al. Acute COVID-19 and the Incidence of Ischemic Stroke and Acute Myocardial Infarction. *Circulation*. 2020; 142(21): 2080-2.
- Mafham M, Baigent C. What is the association of COVID-19 with heart attacks and strokes? *Lancet*. 2021; 398(10300): 561-3.
- Salabei JK, Asnake ZT, Ismail ZH, Charles K, Stanger G-T, Abdul-lahi AH, et al. COVID-19 and the cardiovascular system: an update. *Am J Med Sci*. 2022; 364(2): 139-47.
- Knuuti J, Wijns W, Saraste A, Capodanno D, Barbato E, Funck-Brentano C, et al. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes: The Task Force for the diagnosis and management of chronic coronary syndromes of the European Society of Cardiology (ESC). *Eur Heart J* [Internet]. 2020; 41(3): 407-77.
- Liu X, Luo Y. Progress in the studies on the risk factors of acute myocardial infarction in patients under 35 years old. *Zhong nan da xue xue bao Yi xue ban, J Cent South Univ Med Sci*. 2020; 45(7): 856-61.
- Jortveit J, Pripp AH, Langørgen J, Halvorsen S. Incidence, risk factors and outcome of young patients with myocardial infarction. *Heart*. 2020; 106(18): 1420-6.
- Knuuti J, Ballo H, Juarez-Orozco LE, Saraste A, Kolh P, Rutjes AWS, et al. The performance of noninvasive tests to rule-in and rule-out significant coronary artery stenosis in patients with stable angina: a meta-analysis focused on post-test disease probability. *Eur Heart J*. 2018; 39(35): 3322-30.
- Gulati M, Levy PD, Mukherjee D, Amsterdam E, Bhatt DL, Birtcher KK, et al. 2021 AHA/ACC/AASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*. 2021; 144(22): e368-454.
- Budoff MJ, Mayrhofer T, Ferencik M, Bittner D, Lee KL, Lu MT, et al. Prognostic Value of Coronary Artery Calcium in the PROMISE Study (Prospective Multicenter Imaging Study for Evaluation of Chest Pain). *Circulation*. 2017; 136(21): 1993-2005.
- Feuchtner GM, Beyer C, Langer C, Bleckwenn S, Senoner T, Barbieri F, et al. The Atherosclerotic Profile of a Young Symptomatic Population between 19 and 49 Years: Coronary Computed Tomography Angiography or Coronary Artery Calcium Score? *J Cardiovasc Dev Dis*. 2021; 8(11).
- Villines TC, Hulten EA, Shaw LJ, Goyal M, Dunning A, Achenbach S, et al. Prevalence and severity of coronary artery disease and adverse events among symptomatic patients with coronary artery calcification scores of zero undergoing coronary computed tomography angiography: results from the CONFIRM (Coronary CT Angiography Eval. *J Am Coll Cardiol*. 2011; 58(24): 2533-40.
- McNutt RA, Ferenchick GS, Kirlin PC, Hamlin NJ. Acute myocardial infarction in a 22-year-old world class weight lifter using anabolic steroids. *Am J Cardiol*. 1988; 62(1): 164.
- Wysoczanski M, Rachko M, Bergmann SR. Acute myocardial infarction in a young man using anabolic steroids. *Angiology*. 2008; 59(3): 376-8.
- Christou GA, Christou KA, Nikas DN, Goudevenos JA. Acute myo-

- cardial infarction in a young bodybuilder taking anabolic androgenic steroids: A case report and critical review of the literature. *Eur J Prev Cardiol.* 2016; 23(16): 1785-96.
19. Melhem AJJ, Araújo AC, Figueiredo FNS, Figueiredo DLA. Acute Myocardial Infarction in a Young Bodybuilder: A Case Report and Review of the Literature. *Am J Case Rep.* 2020; 21: e924796.
 20. Frankle MA, Eichberg R, Zachariah SB. Anabolic androgenic steroids and a stroke in an athlete: case report. *Arch Phys Med Rehabil.* 1988; 69(8): 632-3.
 21. Fineschi V, Baroldi G, Monciotti F, Paglicci Reattelli L, Turillazzi E. Anabolic steroid abuse and cardiac sudden death: a pathologic study. *Archives of pathology & laboratory medicine.* 2001; 125(2): 253-5.
 22. Kennedy MC, Lawrence C. Anabolic steroid abuse and cardiac death. *Med J Aust.* 1993; 158(5): 346-8.
 23. Levi M, Thachil J, Iba T, Levy JH. Coagulation abnormalities and thrombosis in patients with COVID-19. *Lancet Haematol.* 2020; 7(6): e438-40.
 24. Farshidfar F, Koleini N, Ardehali H. Cardiovascular complications of COVID-19. *JCI Insight.* 2021; 6(13).
 25. Masi P, Hékimian G, Lejeune M, Chommeloux J, Desnos C, Pinenton De Chambrun M, et al. Systemic Inflammatory Response Syndrome Is a Major Contributor to COVID-19-Associated Coagulopathy: Insights From a Prospective, Single-Center Cohort Study. *Circulation.* 2020; 142(6): 611-4.
 26. Katsoularis I, Fonseca-Rodríguez O, Farrington P, Lindmark K, Fors Connolly AM. Risk of acute myocardial infarction and ischaemic stroke following COVID-19 in Sweden: a self-controlled case series and matched cohort study. *Lancet.* 2021; 398(10300): 599-607.
 27. Choudry FA, Hamshere SM, Rathod KS, Akhtar MM, Archbold RA, Guttman OP, et al. High Thrombus Burden in Patients With COVID-19 Presenting With ST-Segment Elevation Myocardial Infarction. *J Am Coll Cardiol.* 2020; 76(10): 1168-76.