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AcuteST-ElevationMyocardialInfarctionina32-Year-OldMaleLessthan2Months after a Normal Coronary Computed TomographyAngiography

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1. Abstract

Background: Acute myocardial infarction (AMI) is an uncommonfindinginyoungpatientsunderage35anddeterminati on ofetiologicalfactorscanbechallenging.Coronavirusdisease-2019 (COVID-19) and misuse of androgenic anabolic steroids (AAS) have both been associated with thrombotic events.

Case Summary: We report the case of a 32-year-old male withafamilyhistoryofprematurecoronaryarterydisease(CAD), smoking and misuse ofAAS, who two months prior to admission wasassessedwithacoronarycomputedtomographyangiograp hy (CCTA)withnormalfindingsandcoronarycalciumscoreofzer o. Thepatientwasadmittedwithsymptomsof ST-segmentelevation myocardial infarction (STEMI) and concurrent COVID-19 in- fection. Due to age, history and paraclinical findings, pericarditis was initially suspected. Coronary angiography showed mid-seg- ment 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,withimplantationofonedrugelutingstentintheLAD.

Conclusion: Our case highlights that misuse of AAS and COVID-19 infection both constitutes predisposing risk factors to thromboticevents,eveninyoungindividualswithaverylowatherosclerotic disease burden.

2. Introduction

IntherarecasesofAcuteMyocardialInfarction(AMI)inpatients under age 35, etiological factors are less studied, but are suggest- ed to be an accumulation of cardiometabolic risk factors, such as male gender, obesity, hypercholesterolemia, smoking and family historyofprematureCoronaryArteryDisease(CAD).Anassociationbetweenmisuseofanabolicsteroidsand AMIhavealsobeen reported in younger individuals. Likewise, a transient increase in risk ofAMI have been reported in relation to influenza, pneumo- nia, and other chest infections [1-4], and recent studies indicate a pathophysiologicalassociationbetweenCoronavirusdisease-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 todetectcoronaryatherosclerotic plaques and CAD, due to a high negative predictive value, and is recommended in the European SocietyofCardiologyguidelinefortheassessmentofCADinpatients with suspected stable angina pectoris [8].

Herein, we report the case of a 32-year-old male presenting with anacuteanteriorST-elevationmyocardialinfarction(STEMI)and concurrentCOVID-19infection,despiteaCCTAwith normalconditions performed two months priorly.

3. Case Presentation

A32-year-oldpreviouslyhealthymanpresentedtotheemergency department due to chest pain. The patient reported daily use of anabolicsteroidsbutdenieduseofothersubstancesincludingcocaine. 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 cigarettesperday,heavydrinkingtwiceamonthandafamilyhis- tory of CAD, with his father having an AMI at 48 years of age. He reported drinking large amounts of alcohol the evening prior toadmission.Also,thepatientwasPCR-testedpositiveforCOV- ID-19fivedayspriortoadmission,althoughasymptomatic.Dueto twopriorepisodeswithchestpainandpalpitations,thepatienthad 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-hourhistoryofintermittentretrosternaldiscomfortwithradiatingpainandburningsensationtotheleftarm,hand,andfingers. Thepainwasofcompressingnatureandhadanintermittentintensityof5to9onanumericratingscale(NRS).Herefusedshortness of breath, fever and coughing but reported some nausea. Physical examination was normal. Due to symptoms and presentation of ST-segmentelevationsinambulanceelectrocardiogram(ECG),an oralbolusofacetylsalicylicacidwasadministered,andthepatient was conferred by ambulance staff with an invasive center regard- ing 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 cholesterol1.9mmol/L,low-densitylipoprotein-cholesterollevelunmeasurable,high-densitylipoprotein-cholesterol<0.52mmol/L, and triglycerides 1.04 mmol/L. Initial ECG testing demonstrated sinusrhythm,heartrate63beatsperminute,ST-elevationsinlead II, II, aVF and V3-V6 (1-2 mm) and ST-depressions in aVR and aVL (1 mm) (Figure 1). Bedside echocardiography showed normalconditionswithapreservedLeftVentricularEjectionFraction (LVEF)andnopericardialeffusion.Pericarditiswassuspecteddue toage,history,diffuseST-elevationsinECGthoughnoPRdepres- sion 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/Land further dynamical changes were observed in ECG withextensiveST-elevationsinII,III,aVFandV3-V6increasing 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, chestpain was persistent and the cardiac troponin level had increasedto 181 ng/L, thus intravenous nitroglycerin and subcutaneous fondaparinuxwasadministered.Thepatientwasre-conferredwith invasivecenterandreceivedasubacutecoronaryangiographythe nextday,approximately28hoursfromadmission.Thecardiactroponin 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) showingsignsofplaquerupturewithalocalthrombus(Figure3). Percutaneous Coronary Intervention (PCI) was performed, with theimplantationofonedrugelutingstentintheLAD.IntheRCA, an insignificant post ostial plaque with a 25% stenosis was observed, with no further actions required. The patient received an oralprasugrelbolusandinfusionofefibatidefor18hoursandwas subsequentlyprescribedprasugrel10mgonceadayfor12months and life-long treatment with acetylsalicylic acid, together with atorvastatin.Ondischarge,echocardiographyshowedapreserved 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.

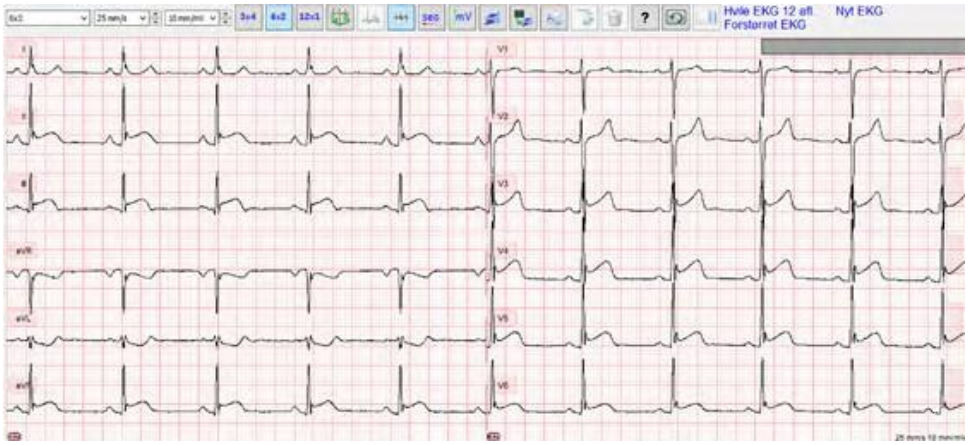

Figure1: Initial12-leadECGtestingdemonstratedsinusrhythm,heartrate63bpm,ST-elevationsinleadII,II,aVFandV3-V6(1-2mm)andST-de- pressions in aVR and aVL (1 mm).

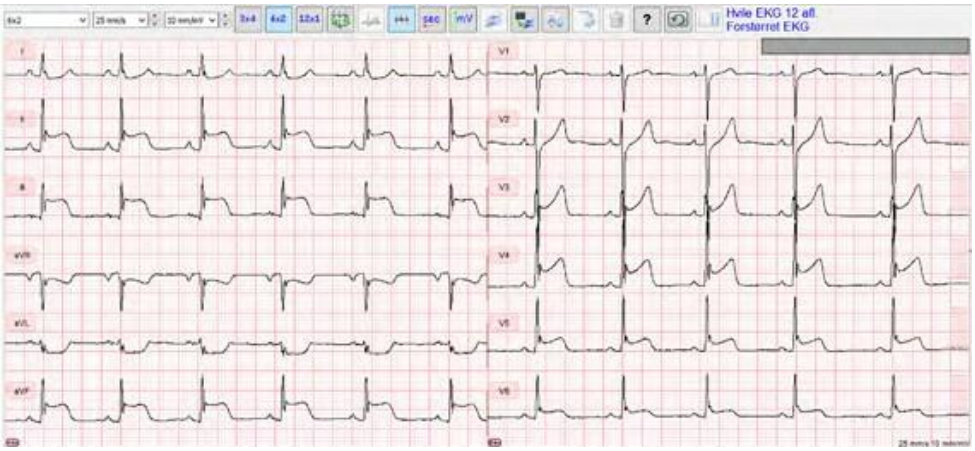

Figure2: 12-leadECGrecorded3hoursafteradmissionshowingsinusrhythm,heartrate68bpm,extensiveST-elevationsininferiorleads(II,IIIand aVF) of 4 mm and in anterior-lateral leads (V3-V6) of 1-4 mm and ST-depressions in aVR and aVLof 1-2 mm.

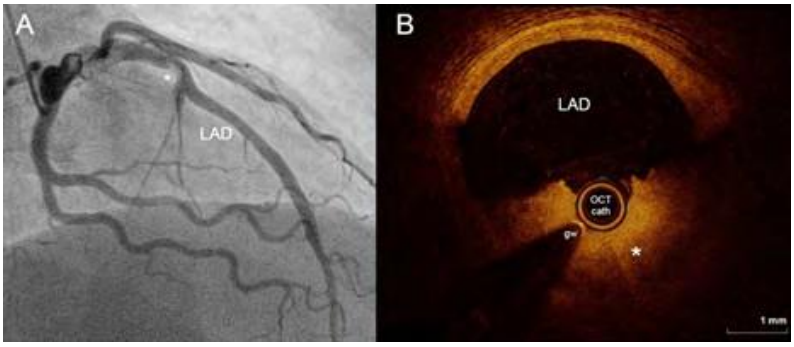

Figure 3: PanelA: Coronary angiogram showing stenosis (*) in proximal left anterior descending artery (LAD). Panel B: Intravascular imaging with optical coherence tomography at the site of the stenosis.Acute thrombus (*) is seen partially obstructing the lumen of the LAD. OCT cath: imaging catheter,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

4. Discussion

WepresentacaseofSTEMIinaCOVID-19positiveyoungmale with cardiac risk factors of family history of CAD, smoking and misuse of anabolic steroids, but with a normal CCTAand a calcium score of zero two months prior to admission. AMI in young individualsunder35yearsareveryrare[9,10].Thepatientdevel- oped STEMI despite not having hypertension, diabetes mellitus andhypercholesterolemia,includinghyperlipoproteinemia(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 highaccuracyfortheexclusionofobstructiveCAD[8,11].CoronaryArtery 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-calcifiedplaquesarereportedlymorefrequent in young individuals, causing doubt regarding the sensitivity of CAC scoring in this age group [14]. Similar doubt regarding sensitivityofCCTAdetectionofnon-calcifiedplaquesinyoungindividuals,however, hasnot beenconfirmed searching theliterature. Villinesetal.foundanadverseeventrateof0.8%withinamedian of2.1yearsinpatientswithaCACscoreofzeroandnon-obstructiveCADverifiedonCCTA[15].Thus,AMIinyoungindividuals under 35 years are very rare [10], especially with a recent normal CCTA,aspresentedhere.Thisknowledge,togetherwithfindings of diffuse ST-elevations in several leads in the ECG, preserved LVEFandinitialnormalcardiactroponinlevels,causedclinicians 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,rangingfromAMIandstroketosuddencardiacdeath [16-22].Thepathophysiologyissuggestedtoinvolvedyslipidem- ia (elevated LDL and reduced HDL cholesterol) causing acceleratedatherosclerosis,coronarythrombosisduetoincreasedplatelet aggregation and coronary vasospasm [18]. Although our patient hadalowleveloftotalcholesterolatadmission,hehadcholesterol parameterssimilartothosedescribedintheliteratureformisuseof AAStwomonthsprior,withtotalcholesterolof5.1mmol/L,HDL of 0.67 mmol/Land LDLof 3.4 mmol/L, suggesting some degree of dyslipidemia in this patient.

The patient also had an ongoing infection with COVID-19, althoughasymptomatic.COVID-19hasbeenshowntopredispose

patients to a prothrombotic state [7, 23]. The underlying etiology is probably caused by direct endothelial cell damage and platelet aggregationduetoanexaggeratedinflammatoryresponsecausing intimalinflammation[24,25].ArecentlargeSwedishstudyfound anincreasedCOVID-19associatedriskofAMIwithanincidence rateratioof8.44inthefirstweekofinfection[26].Anotherstudy foundstrongindicationofahigherintracoronarythrombusburden inCOVID-19patientspresentingwithSTEMI[27].Typicalfindingsofhypercoagulability,althoughprimarilyobservedincritical ill patients with COVID-19, are increased D-dimer, a modest decreaseinplateletcount,prolongedprothrombintimeandelevated lactate dehydrogenase [23]. These findings were not observed in our patient although prothrombin time was not measured. Also, our patient was asymptomatic.

5. Conclusion

EvidencesuggeststhatboththemisuseofAASandinfectionwith COVID-19isstronglyassociatedwithvasculartromboticevents. 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 coronaryangiographywithintravascularimagingshowedobstructiveplaqueruptureinLADandnon-obstructiveplaqueformation inRCA,noneofwhichwasobservedonCCTA.Ourcaseservesto illustratethatmisuseofAASandCOVID-19infectionbothconstitutespredisposingriskfactorstothromboticevents,eveninyoung individuals with low atherosclerotic disease burden. Also, clinicians should be aware that in rare cases,AMI can be seen despite a recent normal CCTA.

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