Clarithromycin/hydroxychloroquine/methylprednisolone

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Off-label use and multivessel spontaneous coronary artery dissection: case report

A 35-year-old man developed multivessel spontaneous coronary artery dissection (SCAD) while receiving treatment with methylprednisolone for Serpiginous choroiditis. He also received off-label treatment with clarithromycin and hydroxychloroquine for Coronavirus disease 2019 (COVID-19) [*not all routes and dosages stated*].

The man was admitted to the emergency department in the morning of 8 May 2020 due to episodes of pressure pain behind the sternum, lasting from 2–5 min to 1h; the pain radiated to the left upper limb, arose after gardening works and persisted both at rest and during moderate physical exertion. Upon anamnesis, it was found that since April 2019 he had blurred vision; he was observed by an ophthalmologist with a diagnosis Serpiginous choroiditis of both the eyes. He initially received pulse therapy methylprednisolone in a cumulative dose of 1000mg and then he was initiated on oral methylprednisolone at 4 mg/day. On 20 April 2020, he experienced weakness, fever, nasal congestion, anosmia, dry cough and chest congestion. A PCR for COVID-19 was found to be positive. He was hospitalised with a diagnosis of Coronavirus disease 2019 (mild case) from 21 April 2020 to 3 May 2020. During hospitalisation for COVID-2019, he received off-label therapy with hydroxychloroquine and clarithromycin. Smoking for 26 years and increased weight (overweight) were identified as the underlying cardiovascular risk factors. He denied the use of psychoactive substances (cocaine, etc.). Investigations on admission to the emergency department on 8 May 2020 revealed the following: BP 126/78mm Hg, HR 84 beats per minute, RR 18 /minute, oxygen saturation 99% on room air and a normal level of high-sensitive cardiac troponin. He was given loading doses of aspirin [acetylsalicylic acid], clopidogrel and heparin. An ECG revealed slight ST-segment elevation in the right precordial leads with reciprocal changes in the lateral wall of left ventricle, suggesting acute coronary syndrome with ST-segment elevation. As a result, he underwent a coronary angiography and a linear dissection of ramus intermedius was established over 20mm with stenosis of the lumen up to 80% in diameter against the background of thrombolysis in myocardial infarction (TIMI) 3 blood flow in all coronary arteries. It was decided to stent ramus intermedius dissection area with a drug coated sirolimus stent and a successful stenting was performed. He subsequently received triple antithrombotic therapy comprising aspirin, clopidogrel and heparin [unfractionated heparin]. At 7:20pm of the same day, while admitted in the ICU, he again had anginal pain syndrome without significant effect from nitroglycerin; the pain was relieved with morphine. A repeat ECG showed ST segment elevation in the leads II, III and aVF with reciprocal changes. Given the appearance of ST-segment elevation in the leads from the inferior wall of the left ventricle, he underwent a repeat angiography which showed that the stented segment and other sections of the left coronary artery (LCA) had no stenosis. Blood flow along the right coronary artery (RCA) TIMI 0–1 with occlusion in the distal section. Also, stenosis of the initial and middle sections of the RCA was detected (up to 60%–75%) over the entire length according to the type of possible spasm in comparison with the initial coronary angiograms. After two injections of contrast into the RCA and treatment with nitroglycerin, recanalisation was observed with the restoration of the initial caliber of the artery with visualisation of linear dissections throughout the initial and middle sections. According to the control angiography, after 15 min, a decrease in the length of the dissected segment in the initial and middle sections of the RCA and posterior lateral branch of the RCA was established. Taking into account the TIMI 3 blood flow, extended lesions of the RCA and the posterolateral branch of the RCA and spontaneous positive dynamics in relation to this dissected segment, conservative management were applied. Over the next 72h, his condition remained stable and chest pain did not recur. In the morning of 14 May 2020, he again felt a pain behind the sternum, lasting up to 10–15 min and stopping on its own. An ECG showed that there were no significant dynamic changes from previous ECGs. It was decided to conduct control angiography which showed that there was no change either in the RCA or in the posterolateral branch of the RCA in comparison with the previous examination. The stented segment of the ramus intermedius LCA was passable; however, in one of the ramus intermedius branches, immediately after its bifurcation, stenosis of up to 85% in diameter with a length of 20–25mm was detected, which was absent in the previous two angiograms.

Following intracoronary administration of 2mL of 0.01% nitroglycerin, there were no changes in the degree of stenosis in the affected artery. Anginal pain did not recur. He was discharged on 20 May 2020. During the hospitalisation, he was consulted by a rheumatologist and additional examinations were performed (antinuclear antibodies, HLA-B27 test, rheumatoid factor and antibodies to β2-macroglobulin were found to be negative), which allowed the rheumatologist to exclude the presence of chronic systemic inflammatory disease. A CT scan of the chest from April 2020 also showed no pathological changes.

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