Multiple drugs

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Severe acute respiratory syndrome coronavirus 2 reinfection and off label use: case report

A 62-year-old man developed severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) reinfection during treatment with hydroxychloroquine, unspecified corticosteroids, triamcinolone and prednisone. Additionally, he received off label treatments with azithromycin, tocilizumab, hydroxychloroquine and amoxicillin/clavulanic acid for SARS-CoV-2 infection [*not all dosages stated*].

The man, who had a history of mild asthma, hypertension, dyslipidaemia, liver steatosis, hyperuricaemia and overweight was taking olmesartan medoxomil [olmesartan], hydrochlorothiazide and allopurinol. Occasionally, he received oral unspecified corticosteroids for asthma and recurrent episodes of right knee pain and swelling intra-articular slow-release triamcinolone [*initial dosage not stated*] plus hyaluronic acid. On 23 March 2020, he experienced fever, diarrhoea, anosmia, dysgeusia, cough, intense asthenia and arthromyalgias. On 24 and 25 March, his nasopharyngeal PCR tests showed that he was positive for SARS-CoV-2. He was confined at home and he had received oral hydroxychloroquine 400mg twice daily on day 1 and thereafter, 200mg twice daily for 6 days, he also received oral azithromycin 500 mg/day for 3 days. Subsequently, improvement in symptoms noted; however, the anosmia, dysgeusia, asthenia and shortness of breath were not resolved fully until mid April. On 2 April, he was negative on a COVID-19 immunoglobulin M (IgM)/immunoglobulin G (IgG) rapid test also on 7 and 8 April, 2 nasopharyngeal swab PCRs tested negative for SARS-CoV-2. Later, on 30 May, he received a 1 dose of intra-articular triamcinolone 40 mg plus hyaluronic acid in the right knee while preparing for a trekking excursion. On 28 August, after 3 days of returning to work from vacation, developed intense arthromyalgias, headache, fever, cough and dyspnoea. Thereafter, he took azithromycin 500 mg/day for 3 days, salmeterol and oral prednisone 30mg on the first day and thereafter continued with 15 mg/day. On 31 August and 1 September, he underwent 2 consecutive nasopharyngeal SARS-CoV-2 PCR tests and he was positive in both. On 2 September, he was admitted to the emergency room for worsening dyspnoea and cough. On admission, he reported chills, fever, myalgias, anosmia and ageusia. On examination, respiratory rate was 36 breaths/minute, HR was 100 beats/minute and he had bilateral inspiratory crackles. Subsequent chest radiograph revealed bilateral alveolar-interstitial infiltrates. On admission, he had lymphopenia and high C-reactive protein. Baseline arterial blood gas (ABG) analysis showed a fraction of inspired oxygen (FiO2) 0.21 of pH 7.45, partial pressure of oxygen in arterial blood (PaO2) 80.5mm Hg and partial pressure of carbon dioxide (PaCO2) 31.0mm Hg and ration of PaO2/FiO2 was 383mm Hg. On 2 September (on day 5 of post-symptoms), received remdesivir for 5 days, oral dexamethasone 6 mg/day and enoxaparin sodium [enoxaparin] and also IV amoxicillin/clavulanic acid [amoxicillin-clavulanate] which was later, stopped when urine pneumococcal and Legionella species antigens, bacterial sputum and blood cultures were negative. In the beginning, he remained on oxygen saturation of 96%–97% with nasal cannulas 2 L/minute; however, demonstrated worsening of radiologic infiltrates with the persistence of elevated inflammation markers. On 3 September, he underwent a lung CT angiogram which ruled out pulmonary thromboembolism and confirmed extensive bilateral lung infiltrates with areas of pneumonic consolidation. These all respiratory findings suggested he had life threatening bilateral pneumonia and acute respiratory distress syndrome. Subsequently, on 3 and 4 September, he administered 2 doses of IV tocilizumab 600mg and dexamethasone dose was increased to 20 mg/day. In spite of lung infiltrates progression, he remained clinically stable until 7 September. Later, his respiratory status worsened promptly, with ABG (FiO2 35%) of pH 7.45, PaO2 55mm Hg and PaCO2 38mm Hg and ratio of PaO2 /FiO2 was 157mm Hg. On 8 September, he was transferred to the respiratory intermediate care unit for hypoxaemic respiratory failure. Thereafter, he received high fl ow oxygen therapy (HFOT) 50 L/minute, 87% with an ABG of pH 7.48, PaO2 105mm Hg and PaCO2 37mm Hg (PaO2 / FiO2 121mm Hg) and the ratio of oxygen saturation index was 5.95 at 6h. As there was a progressive improvement, for the next 4 days high-flow respiratory support was maintained. Later, it was stopped, after an ABG (0.35% 30 L/minute HFOT) of pH 7.43, PaO2 102mm Hg and PaCO2 38mm Hg (PaO2/FiO2 291mm Hg). Further, dexamethasone was reduced to 6 mg/day and tapered until discontinued after 14 days. In view of favourable evaluation, he was transferred to the infectious diseases ward, where conventional oxygen support was slowly reduced and stopped on 17 September. On 18 September, he was discharged with no further complications, thereafter.

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