

## Methylprednisolone/tocilizumab

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**Cytomegalovirus gastritis following off label use: case report**

A 33-year-old man developed cytomegalovirus (CMV) gastritis following off label treatment with methylprednisolone and tocilizumab for COVID-19 [*routes and dosages not stated*].

The man presented to a hospital with odynophagia, persistent dysphagia, and epigastric pain and discomfort following discharge from COVID-19 related hospitalisation for a month. It was reported that, his current symptoms had started 1 week previously and were aggravated by receiving of solids or liquids. Moreover, he complained of a new-onset symptom of feeling sick. About 35 days prior to the current presentation, he was diagnosed with COVID-19 and was hospitalised. From hospitalisation day 3, his oxygen saturation decreased, and he started receiving noninvasive positive pressure ventilation. During the hospitalisation, he had received remdesivir, off label methylprednisolone and two doses of off label tocilizumab for COVID-19. Additionally, he had received unspecified broad-spectrum antibiotics and unspecified supportive therapy. It was reported that, in the final days of his hospitalisation (day 28–30), he developed mild epigastric pain; hence, he received an unspecified proton pump inhibitor. Afterwards, he was discharged. However, after 5 days from the discharge (day 35), he developed new symptoms including dysphagia and epigastric discomfort; hence, he was admitted (current presentation). On current presentation, his vital signs were stable. His paraclinical workup showed a normal white blood cell count with a decreased lymphocyte count and thrombocytopenia. His other routine laboratory parameters were within normal limits. Other immune system tests including human immunodeficiency virus antibody serology, complement and immunoglobulin levels and, flow-cytometric analysis of lymphocyte subpopulations were not suggestive of an underlying immunodeficiency disorder. Hence, to determine the cause of his low level of platelets, laboratory tests including peripheral blood smear, viral markers of hepatitis B surface antigen and hepatitis C virus antibody, lupus markers including antinuclear and antideoxyribonucleic acid antibodies and color Doppler sonography of the hepatic portal system were performed; however, no remarkable findings were concluded. Since, he reported that he had white oral mucosal lesions that were observed after his first hospital discharge, a primary gastrointestinal candidiasis was suspected. Therefore, his treatment was started with nystatin and fluconazole. On day 2 from the current admission, endoscopy revealed mild inflammation along with a few whitish exudates in the proximal and distal third of the oesophagus, and a large gastric ulcer from the fundus extending to the body of the stomach. The lesion had inflamed, elevated borders; however, no bleeding was noted. A black exudate covering the gastric ulcer was observed, which was highly suggestive of mucormycosis. Subsequently, brushing for KOH and cytology tests were performed, and from the margins of the lesion a biopsy was obtained. Meanwhile, amphotericin-B-liposomal [AmBiosme; liposomal amphotericin B] was started. Subsequently, paranasal and abdominopelvic CT scans did not reveal any extragastrointestinal involvement. Thereafter, histopathological assessment of the biopsy specimens revealed an acute inflammatory process along with granulation tissue formation; however, no evidence of mucormycosis or malignant cells was observed. After 8 days (day 44), he received another endoscopy, which revealed mild inflammation in the distal third of the oesophagus and numerous large active ulcers with congested borders in the body of the stomach, but without the previous black coating. Subsequent, biopsies from the antrum and body mucosa revealed cytopathic effects indicative of CMV infection including cell and nuclear enlargement and nuclear inclusion bodies. Thereafter, immunohistochemical (IHC) study confirmed CMV gastritis. Off label treatment with methylprednisolone and tocilizumab was considered as risk factor for the development of CMV gastritis [*durations of treatments to reaction onset not stated*].

Therefore, the man's treatment was started with ganciclovir. After about 72 hours, his dysphagia and abdominal pain decreased, and his symptoms improved. He received ganciclovir for 3 weeks. After 3 weeks, a new endoscopy revealed a healed ulcer in the body of stomach. Moreover, after CMV treatment, his platelet level was also normalised.