A FATAL CASE OF VASCULITIS AFTER SARS-COV-2 NEGATIVIZATION

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BACKGROUND AND AIMS: Spectrum of acute severe respiratory syndrome Coronavirus 2 (SARS-CoV-2) ranges from mild to critical and probably mortality rate is largely underestimated. Complications may represent different manifestation of a profound endothelial dysfunction and injury. Biopsies reveal macro and microvascular thrombosis involving larger and smaller vessels. Different Authors described accumulation of inflammatory cells across vascular bed, viral inclusions and apoptotic bodies across vascular bed. Endothelitis leads to loss of vessel integrity with bleedings and lumen restriction with tissue ischemia and necrosis. SARS-CoV-2 also can cause vasculitis and a systemic inflammatory vascular disease with COVID-19 associated coagulopathy. Several cases of vasculitis have been described during COVID-19 pandemy but in literature, to our knowledge, there are few cases of patients developing vasculitis after SARS-CoV-2 infection.

METHOD: A 59-year-old male patient in chronic haemodialysis with hypertension, chronic thrombocytopenia, uncertain history of type 2 diabetes mellitus and recent COVID-19 pneumonia (1 month before) was admitted to our E.R. with psycho-motor slowdown, dyspnoea, profound hypotension, diffuse and extensive purpura especially on extremities and nose with petechiae and ecchymoses, thrombocytopenia, widespread arthritis and myalgia and atrial fibrillation with high ventricular response. Laboratory tests revealed leucocytosis with elevation of inflammatory markers (GB 15.6 x 10^3/µL, CRP 6.61 mg/dl, procalcitonin 2.64 mg/ml), thrombocytopenia (PLTs 55 x 10^3/µL), Hb 11.2 g/dl, increased amylase (444 UI/L) and lipase (608 UI/L), hyperkalaemic metabolic acidosis, mild impaired blood clotting with normal fibrinogen and severe D-Dimer elevation (3453 mcg/ml), normal haptoglobin and bilirubin.

SARS-CoV-2 rapid antigen test and three molecular swab tests (at admission and during recovery) were negative. Contrast-enhanced chest-abdomen CT did not demonstrate relevant findings.

RESULTS: We suspected thrombotic thrombocytopenic purpura, COVID-19 related vasculitis or an autoimmune disease reactivation after SARS-CoV-2 or other concurrent viral infections. A peripheral blood smear excluded presence of schistocytes, HBV, HCV, HIV, c-ANCA, p-ANCA, LAC anti-mitochondrial and ENA antibodies were negative, such as complement factor C4 (C3 at low limits). IgA immunoglobulins resulted increased (952 mg/dl).

We administered intravenous (IV) hydration, analgesic therapy, broad spectrum antibiotics and Methylprednisolone 40 mg IV without benefit. Three days after clinical conditions were critical with further neurological deterioration and haemodynamic instability, consequently we started methylprednisolone 1 g/day IV for three days, followed by oral prednisone 25 mg/die with rapid improvement of clinical conditions and laboratory findings. A skin biopsy revealed a variable and discontinuous presence of C4d and IgM along capillary walls and traces of IgG, supporting the hypothesis of an immune-mediated vasculitic process. Patient was discharged in stable condition with Prednisone 25 mg/die per os.
Later we learned that patient had been hospitalized again for severe anaemia, thrombocytopenia and septic shock with fatal outcome.

CONCLUSION: SARS-CoV-2 clinical spectrum appears to be extremely varied; the direct cytolysis and the immune-mediated damage are among the most accredited hypotheses on disease physiopathology, but molecular mimicry mechanisms may also be involved. Vascular endothelium can be considered among the first targets. Vasculitis are frequently described in literature during COVID; this case, instead, is an anecdotal report of severe angitis arising after swab negativization, underlying the possibility that hyperinflammation can either cause an autoimmune syndrome de novo or trigger a flare-up of a pre-existing condition.