P053  FULMINANT PURPURA IN A PATIENT WITH SARS-COV-2

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Background/Aims
According to available data children with SARS-CoV-2 has asymptomatic or a milder clinical course, but there are known cases of multisystem inflammatory syndrome.

Methods
We observed a case of fulminant purpura in a boy with SARS-CoV-2. A three year old boy had febrile fever, vomiting, abdominal pain for no apparent reason. On the 2nd day this boy had joint pain, petechial rashes, fever and abdominal pain persisted. The patient was hospitalized in the pediatric Department, where he was diagnosed with hemorrhagic vasculitis, there was prescribed therapy with glucocorticosteroids (1 mg/kg per day), dipyridamole (2.5 mg/kg per day), oral cephalosporin. Against the background of ongoing therapy, the child’s condition became worse. On the 5th day of the disease appeared necrosis in the area of the ankle joints, heels, and toes, patient was transferred to the pediatric Department of Children’s Clinical Regional Hospital.

Results
At the time of hospitalization, the child’s condition was severe; the boy had persistent high fever, petechial rash on the torso and legs, necrosis in the area of the left ankle, outer surface of the right ankle and in the area of both heels, edema of the feet and hands. The boy was in a forced position, couldn’t not walk and had intense pain in the
shoulders, elbows, wrists, hips, knees and ankles. Blood tests revealed anemia (HGB 73 g/L), thrombocytopenia (PLT 120 x 10⁹/L), leukocytosis (WBC 23 x 10⁹/L), ESR 60 mm/h; in the clinical analysis of urine - proteinuria. In the blood test was detected significantly increased levels of CRP (129 mg/L), ferritin (637 ng/mL), triglycerides (1.39 mmol/L), hypoproteinemim (48 g/L), hypoalbuminemia (31 g/L); the level of ALT, AST, LDH was normal. The level of D-dimer was 4750ng/mL, fibrinogen - 8.8 g/L, antithrombin III - 156%, protein C - 64%, coagulation Factor IX activity - 150%, coagulation Factor VIII activity - 289%. An increase of antinuclear antibodies, cardiolipins, phospholipids and ANCA were excluded. A nasopharyngeal swab was negative for SARS-CoV-2 in this patient (IgG antibodies directed toward SARS-CoV-2 were absent). An echocardiogram showed no coronary artery abnormalities, echocardiogram was normal, a chest CT and MRI of the brain also were normal. The diagnosis was fulminant purpura in a patient with SARS-CoV-2. This boy received heparin, IVIG 2g/kg, dexamethasone 10 mg per meter of body surface a day, antibiotic therapy was continued. As a result fever, petechial rash and necrosis regressed. After 4 weeks we detected IgG antibodies directed toward SARS-CoV-2.

Conclusion
the current case demonstrates the possibility of developing a multi-system inflammatory syndrome like fulfilminant purpura in patients who had SARS-CoV-2.

Disclosure
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