

que não desenvolveram doença relacionada, mas que foram acometidos de COVID-19. Tratam-se dos seguintes pacientes: sexo feminino, 64 a, com diagnóstico de HTLV há 4 anos, ao apresentar quadro clínico de sarna crostosa, estrongiloidíase e infecção grave secundária as lesões cutâneas. O outro paciente de sexo masculino, 72 anos, portador de HTLV, diagnosticado durante triagem sorológica há 4 anos, sem nunca desenvolver evento clínico infeccioso. Os dois são seguidos regularmente no ambulatório de hematologia e não apresentaram alterações significativas aos hemogramas e bioquímicas de rotina no seguimento. Durante a pandemia atual, os dois pacientes foram contaminados pelo COVID-19, porém os sinais e sintomas de ambos foram leves como coriza, congestão nasal, anosmia e febre. Não houve quadro clínico respiratório (queda da saturação de oxigênio, taquidispneia) e nem necessidade de internamento hospitalar. Os pacientes foram apenas medicados com sintomáticos e os exames laboratoriais não apresentaram exacerbação de linfócitos e não apresentaram desenvolvimento clínico de aspectos das doenças do HTLV (neuropatia ou leucemia) e nem apresentaram outras infecções associadas. Geralmente, ao serem avaliados casos na literatura que relacional covid-19 e portadores de HTLV, ainda os dados clínicos são escassos e os relatos estão mais relacionados a casos de infecção pelo vírus da imunodeficiência humana (HIV); sendo assim, cabe aos profissionais de saúde aconselhamento de prevenção em relação ao COVID-19, a fim de que os portadores não sejam contaminados, uma vez que ainda não há tratamentos efetivos para erradicação e controle viral quando se abordam questões do HTLV, sendo na maioria das vezes desfechos desfavoráveis com taxas de mortalidade e morbidade bastante elevadas. Neste relato, os casos abordados servem para colocar em evidência a importância e a endemidade do HTLV, porém os questionamentos ainda permanecem como uma lacuna na literatura científica, sendo necessário estudo com maior número de participantes para elucidar essa coinfeção.

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#### IMMUNE THROMBOCYTOPENIA PURPURA ASSOCIATED WITH NOVEL CORONAVIRUS INFECTION

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Thrombocytopenia is usually multifactorial as its pathogenesis is likely to be more complex than the conventional model of platelet consumption associated with thrombin-mediated platelet activation. The use of antibiotics, antivirals, heparin, and other commonly used agents, as well as haemodialysis, may contribute in some cases. Furthermore, platelet production may be affected by direct viral insult to the bone marrow or reduced effect of thrombopoietin. The

current COVID-19 pandemic, caused by a novel coronavirus (SARS-CoV-2), poses dilemmas for the investigation of thrombocytopenia. Thrombocytopenia was noted in up to one third COVID-19-hospitalized patients. Besides that, platelet count was lower in those with very severe disease: the lower the platelet count, the worse the prognosis. Mortality may also increase with progressively lower platelet counts. In most COVID-19 cases of thrombocytopenia, the platelet count does not fall below  $100 \times 10^9/L$ . Severe degree of thrombocytopenia ( $< 20 \times 10^9/L$ , or a sudden drop  $> 50\%$  over 24-48 h) is likely to indicate an immune aetiology. Autoimmune thrombocytopenia is a diagnosis of exclusion, there being no confirmative test. Herein we describe a rare case of immune thrombocytopenia purpura (ITP) associated with COVID-19. A previously healthy and nullipara 23-years-old woman visited the Emergency Department after 2 days of retroorbital headache, sneezing, dry cough, sore throat, shivers, and non-measured fever. She also complained petechiae and spontaneous mild gingival hemorrhage. Due to local epidemics, dengue infection was considered, although NS1 fast-test was non-reagent and C-reactive protein was normal. Her blood count was normal, as the test performed almost 10 months before. Three days later (Jul/30/2020), she was admitted in the hospital wards, without any complain except from worsening of the petechiae. Her platelet count dropped from  $231.9 \times 10^3/mm^3$  to  $5.8 \times 10^3/mm^3$ , and platelet concentrate was transfused. Dengue serology was IgM negative and IgG positive, and she confirmed previous 2 episodes of dengue infection. RT-PCR on naso-/oropharynx swab detected SARS-CoV-2. Reticulocyte count was normal and no schistocyte was observed in blood smear. Clotting assays and fibrinogen were normal, and D-dimers were slightly increased. She had microscopic hematuria with normal renal function tests. Total and fractionated bilirubin, and transaminases were normal, although lactic dehydrogenase was slightly elevated. Thyroid function was normal. Protein electrophoresis was normal. Folate and vitamin B12 levels were normal. Thoracic computed tomography was normal. Anti-nuclear antibodies, syphilis, human immunodeficiency virus, and hepatitis C virus tests were non-reagent. She was immunized against hepatitis B virus. Anticardiolipin antibodies were negative, but direct Coombs test and lupus anticoagulant assay were positive. Prednisone 1 mg/kg was administered for 5 days (Jul/01-05/2020). Platelet count increased and petechiae solved. She was discharged home on Jul/05/2020, when her platelet count was  $191.9 \times 10^3/mm^3$ . She had  $207.0 \times 10^3$  platelets/ $mm^3$  on her last visit, when prednisone was 0.1 mg/kg (Jul/07/2020). Although thrombocytopenia may be a predictor of worse prognosis in COVID-19, no relationship between ITP and outcome was described yet. Currently, after excluding other causes of (severe) thrombocytopenia, the treatment of COVID-19-associated ITP should be similar the treatment of non-COVID-19 individuals.

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