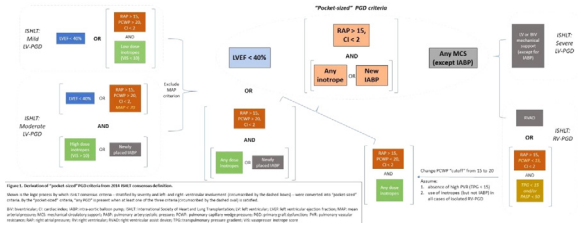
Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.



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Late Cytomegalovirus Primoinfection in a Heart Transplant Recipient After COVID-19 Vaccine

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Introduction: Cytomegalovirus [CMV] is a frequent infection in solid organ transplant recipients and the mRNA COVID-19 vaccine has been associated with transient lymphopenia, which could be a risk factor to consider for CMV replication.
Case Report: A 51-year-old man with arterial hypertension and a history of repaired complex congenital heart disease, which required a heart transplant [HT] in 2019. He had a high risk CMV missmatch (D+/R-). Routine HT follow up tests after the first year were normal, with neither signs of rejection nor allograft vascular disease. His immunosuppressive treatment consisted in Tacrolimus 5 mg once a day, Mycophelonate 500 mg twice a day, and Prednisone 5 mg a day. 77 weeks after his heart transplant, two doses of mRNA COVID-19 vaccine were inoculated separated by 28 days. After 4 days of the second dose, he developed fever, diarrhea, and general malaise. The physical findings were abdominal pain, slight hepatomegaly and several painful gum lesions. Laboratory tests showed severe lymphopenia; and elevated C-reactive protein. CMV by polymerase chain reaction was tested positive with 228.356 copies/ml. The decision was to admit him and to initiate intravenous ganciclovir 5 mg/kg twice a day and to reduce the immunosuppressive treatment. He completed a course of 15 days with ganciclovir, and then continued with valganciclovir 900 mg twice a day. Early after ganciclovir start, clinical manifestations gradually disappeared. Due to persistent lymphopenia, mycophenolate was switched to everolimus. After 60 days of antiviral treatment, CMV loading was below detection range (Image 1).
Summary: The probability of favoring CMV replicability after mRNA COVID-19 vaccine has not been yet described. We here present a case of CMV primoinfection closely related in time with a full mRNA COVID-19 vaccine administration. We hypothesize that lymphopenia and a high risk CMV could be factors to consider at the time of mRNA COVID-19 vaccine administration. A close CMV monitoring should be advised in this scenario.

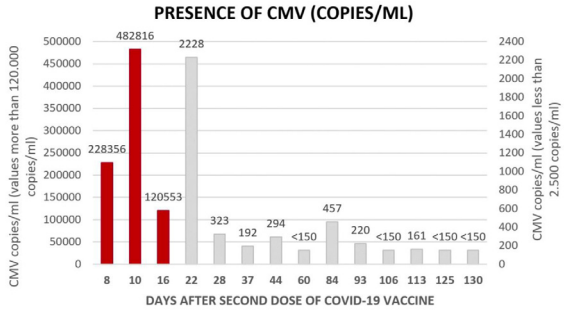

Image 1. Evolution of CMV loading detected by blood polymerase chain reaction after the full dose of mRNA COVID-19 vaccine. On the left bar, data are expressed in CMV loading > 120.000 copies/ml (red colour). On the right bar, data are expressed in CMV loading < 2.500 copies/ml (grey colour).

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Vaccine-Induced Coronay Antibodie-Mediated Rejection and Thrombosis in a Heart Transplant Pacient: A Case Report

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Introduction: Heart tansplant (HT) recipients constitute a population at risk for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Efficay and safety of SARS-CoV-2 vaccine in this population is still yet to be established. It has been described, immune thrombotic thrombocytopenia, myocarditis and Guillain-Barre syndrome in individuals who received the ChAdOx1 SARS-CoV-2 vaccine. There are very few cases of acute rejection after SARS-CoV-2 vaccination post HT patients. We will describe a several outcome of heart function vaccine-induced.
Case Report: A 46-year-old heart transplanted male since 2015, started with persistent cough 15 days after a dose of adenoviral vector-based vaccine against SARS-CoV-2. As he had increased troponin and new left ventricular dysfunction, he underwent an endomyocardial biopsy, collected an panel reactive antibodies (PRA) and started pulse dose metylprednisolone. He developed an ischemic electrocardiographic alteration with a ST elevation and the coronary angiography found a thrombosis in the anterior descending coronary artery with no success with percutaneous treatment. Endomyocardial biopsy found no acute rejection, and PRA showed de novo donor specific antibodies (DSA). Despite treatment for antibodie-mediated rejection with plasmapheresis, human immunoglobulin and rituximab, he had a cardiogenic shock, refractory to inotropic support and intra-aortic balloon pump, requiring peripheral VA ECMO. Regardless of initial hemodynamic response and partial recovery of biventricular function, patient could not stand weaning from ECMO and inotropes. After rejection therapy, PRA showed no antibodies and patient was included in HT list and had a retransplant after 16 days without complications.
Summary: To the best of our knowledge, this is the firts report of antibodie-mediated rejection in heart-transplant patient with thrombotic complication after ChAdOx1 SARS-CoV-2 vaccine. Although vaccination remains the main approach of preventing SARS-CoV-2 infeccion, transplant recipients were not included in clinical trials, so its safety remains unknown in this population. More studies are needed in order to increase knowledge about vaccine outcomes in these individuals.

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Too Much Too Soon? The Catch-22 of Catching COVID-19

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Introduction: Myocarditis has become a well-recognised cardiac complication of SARS-CoV-2 infection. Now, there are growing reports of rare incidences of myocarditis following receipt of mRNA COVID-19 vaccines.
Case Report: A previously healthy 35-year-old male tested positive for COVID-19 on 8/12/21. He never required hospitalisation and was thought to have cleared the infection by 9/5/21. Three weeks later he received the first dose of Pfizer-BioNTech BNTT162b2/Comirnaty mRNA COVID-19 vaccine. Five days later he presented to emergency with chest pain alongside myalgia, headache and cough. His troponin-T was below reference range (RR), electrocardiogram showed sinus rhythm with mild, diffuse T-wave flattening, and no evidence of pericardial effusion was seen on bedside transthoracic echocardiogram (TTE) so was discharged. Two days later he represented to hospital with fevers, vomiting, diarrhea and a maculopapular rash. His subsequent admission was complicated by rapid deterioration and his management reflected a diagnostic dilemma with wide differentials. On day 2 of admission he became haemodynamically unstable requiring vasopressor therapy with a high sensitivity (hs) troponin-I of 103ng/L (RR <26ng/L). On day 4 he developed atrial fibrillation, worsening respiratory distress, peak hs troponin-I of 1474ng/L and required intubation, direct-current cardioversion and venoarterial extracorporeal membrane oxygenation (ECMO) for intractable heart failure. TTE here showed severe global systolic impairment with a left ventricular ejection fraction of 15% and small pericardial effusion. His subsequent treatment targeted possible multiorgan sepsis with antibiotics,