Abstract: **P262**

Extracorporeal life support in a bone marrow transplant recipient with presumed gvhd-related fulminant myocarditis.

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Cardiac involvement by GvHD is extremely rare. Here we illustrate the case of a patient with cardiogenic shock due to a GvHD-related fulminant myocarditis. A 35-yr-old woman with a T-cell ALL presented to our ER for drowsiness. She had received an allogeneic hematopoietic stem cell transplant 4 months earlier, had a history of cocaine-addiction and had not been compliant with cyclosporine and antivirals for the past 2 weeks. A CMV-related pneumonia was suspected and the patient was hospitalized. Due to the onset of anuria and hypotension, she was transferred to the ICU where the echocardiogram showed a severe pericardial effusion conditioning cardiac tamponade and a severely depressed LV function(EF 20%) with increased parietal thickness(Figures). After intubation, the patient underwent a successful pericardiocentesis. Due to severe pleural effusion, two pleural drains were positioned. To maintain an adequate organ perfusion, inotropic support with dobutamine and noradrenalin was begun. Troponin levels and inflammatory markers were elevated so a fulminant myocarditis was suspected. Cultural, serologic and tests for autoimmunity were all negative. Flow cytometry of the pericardial fluid showed no evidence of leukaemia. Urinalysis revealed an active sediment. Acute GvHD was hypothesized as the cause, even if rare, of the myocarditis, the renal dysfunction, the thrombocytopenia and the polyserositis, given also the low cyclosporine levels on admission. Accordingly, the patient was treated with broad-spectrum antimicrobial agents, cyclosporine and methylprednisolone(1 mg/kg/day). An EMB was not performed because of severe thrombocytopenia. Cocaine-related, giant cell and infectious myocarditis were possible alternative diagnoses, but none of them could explain the involvement of the kidneys, bone marrow and serous layers. Due to persistent haemodynamic instability(anuria and increase of lactates) a v-a ECMO was positioned. The patient received continuous hemofiltration to treat fluid overload. A haemodynamic improvement and a progressive myocardial recovery were observed, so, after 6 days, the ECMO support was removed and an IABP was positioned, while a coronary angiography confirmed the absence of CAD. The patient underwent a progressive weaning from inotropes and the IABP was removed after 4 days. The last echo showed a remarkable improvement of the LV function (EF 38%). The patient, extubated and stable, was transferred to the Hematology ward. Our case-report represents a unique case of GvHD-related fulminant myocarditis, with concomitant serous, renal and marrow involvement, causing initially an obstructive shock and then a cardiogenic shock. Inotropic and mechanical circulatory support were used to stabilize the patient and improve organ perfusion, while we targeted the putative cause with steroids and cyclosporine. Although histologic confirmation is lacking, the response to the etiologic therapy is an "ex adiuvantibus" diagnostic confirmation.