well understood. The optimal management for these patients is unclear, in particular whether recommendations for conventional treatment for LVSD can be extrapolated from the general population. These patients may be turned down for transplant on the basis of increased perioperative morbidity and mortality.
Discussion: It is thought that a prolonged exposure to uremic toxins, particularly "middle molecules", in dialysis patients may lead directly to cardiac myocyte dysfunction and LV impairment. Dialysis induced variations in blood volume and haemodynamics can also significantly affect systolic function. We have seen an improvement in EF in the post-transplant period in a number of our patients with non-ischaemic cardiomyopathy. We therefore postulate that in addition to conventional medical therapy for LV dysfunction, treatment regimes should also include optimising fluid management and early consideration for renal transplantation.
Conclusions: We have successfully demonstrated that renal transplantation can be safely performed in selected patients with LVSD and results in the improvement of EF, LV systolic dimensions and symptoms of heart failure. These patients, traditionally considered high risk for surgery, may benefit from early renal transplantation and demonstrate reversibility of their LV dysfunction.

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A patient with human immunodeficiency virus infection and acute decompensated heart failure; never forget the pericardium

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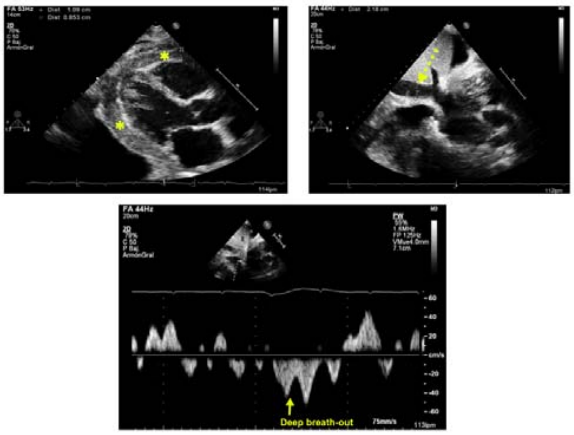
A 35-year-old male patient with past medical history of Human Immunodeficiency Virus (HIV) infection non-compliant to antiretroviral therapy (ARV) presented complaining of 2 moths of dyspnoea on exertion, fever, night chills and weight loss. M. tuberculosis was found from cultures and genome detection through protein chain reaction (PCR) in bronchial samples, pericardial fluid and pericardium samples obtained from a pericardial window. Disseminated tuberculosis infection was diagnosed and therapy was started.
He was readmitted due to dyspnoea, ortopnea, paroxysmal nocturnal dyspnoea, abdominal pain and anasarca. Exploration revealed bilateral jugular vein dissension, hepatojugular reflux, reduced breath sounds reduced, hepatomegaly, ascites and bilateral lower leg pitting edema. Electrocardiogram showed generalized low voltage, echocardiography proved pericardial thickening up to 13 mm, mild pericardial effusion, restrictive diastolic dysfunction, bilatearal atrium enlargement, interventricular interdependence with inspiration, inversion of the flow diastolic in suprahepatic veins in exhalation, reduction of left ventricular inflow (E wave) >25% with inspiration and IVC dilation. Left and right ventricle systolic function were preserved.

Microbiological test.	
Adenosine deaminase in pericardial fluid	86 U/L (Reference: 0 to 10)
M. tuberculosis culture in pericardium biopsy	Positive
M. tuberculosis culture in pericardial fluid	Positive
M. tuberculosisprotein chain reaction in pericardial fluid	Positive
Pericardium biopsy	Granuloma formation with multinucleate cells and caveating necrosis.
M. tuberculosis culture in bronchoalveolar lavage	Positive
M. tuberculosis protein chain reaction in bronchoalveolar lavage	Positive
CD4 positive lymphocytes	294/ul
HIV Viral load	6595 copies/ml

HIV: human immunodeficiency virus

Questions, problems or possible differential diagnosis: What is the most probable diagnosis of this patient? What would be the acute initial management? What would be the best option for the long-term management?
Answers and discussion: Final diagnosis was tuberculous constrictive pericarditis in an HIV positive patient. Tuberculosis is the main cause of pericarditis and pericardial effusion in patients living in endemic countries and in patients with HIV infection it responds for more than 90% of cases. The mortality after pericardial tuberculosis can be as high a 40% six months after diagnosis.
Acute management was based on support intervention and symptomatic relief. Long-term management of this patient was made by the cardiovascular staff. The patient had poor prognosis predictors such as cachexia and limitation to give treatment for the primary diagnosis due to patient persistent refusal to take ARV medications. Taking this into account and the high mortality and morbidity associated with pericardiectomy, the patient was discharged with follow-up
Conclusions and implications for clinical practice: Tuberculosis and HIV infection are still a dangerous combination affecting patients living in developing countries. Pericardial involvement during M. tuberculosis infection can be disastrous during acute episodes generating the necessity of invasive procedures or extensive work-up increasing risk of complications. The decision-making process is not easy and must be individualized to each patient because medical therapy and surgical interventions also have the potential risks.



Echocardiographic findings

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First report of heart failure due to zika myocarditis

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Introduction. Zika virus infection (ZIKV), is a mosquito-borne disease that has become a global health hazard. There are no reports of cardiovascular manifestations, heart failure (HF) or arrhythmias associated with ZIKV disease. Case report: A 51 year-old man from Venezuela, previously asymptomatic and with a history of well-controlled arterial hypertension and type 2 diabetes mellitus that in a recent medical evaluation had a normal electrocardiogram (ECG) and echocardiogram (TTE) with a left ventricular ejection fraction (EF) of 68 %. On May 2016, he developed low-grade fever, polyarthralgia, widespread pruritic maculopapular rash, conjunctivitis and myalgia. These clinical manifestations were resolved in 2 days. Five days after the onset of this acute episode, however, he was admitted to the hospital with rapid progressive dyspnea, rated according to the New York Heart Association (NYHA) as class IV. The ECG, five days after onset, showed a new left-bundle branch block and on the TTE a severely reduced EF of 16%. A coronary angiogram was performed which was normal. A holter recording showed frequent atrial and ventricular premature beats. A cardiac MRI (cMRI) exhibited severe impaired left ventricular systolic function (EF 16%), hiperintensive inferior wall signal on T2-weighted and lateral subepicardial enhancement in late gadolinium sequence. ZIKV-induced myocarditis was diagnosed based on the following criteria: i) ZIKV RNA was detected in serum with the use of reverse-transcriptase polymerase chain reaction (RT-PCR) and Zika virus-specific IgM antibody; ii) Potential simultaneous infection including Dengue, Chikungunya, HIV, and other virus or parasitic infections such as Chagas were ruled out; iii) Clinical, TTE, ECG and cMRI evidence of myocarditis was detected; iv) Normal coronary arteries on angiogram. The patient was initially treated with intravenous furosemide and oral digoxin for a period of one month, along with enalapril, carvedilol and eplerenone and his condition improved to NYHA Class II-III and then, after discontinuing enalapril and administering sacubutril/valsartan titrated