LETTER TO THE EDITOR

A rare case of pericarditis, complication of infliximab treatment for Crohn’s disease

Dear Sir,

Infliximab, a monoclonal anti-TNFα antibody, is a widely used drug in the treatment of inflammatory bowel disease. Various adverse reactions including infusion reactions, reactivation of tuberculosis, serum sickness, hematologic or bronchogenic malignancies are attributed to infliximab therapy. Pericarditis as one of the adverse reactions is very rarely reported. We present a case of pericarditis that occurred in a patient with refractory ulcerative colitis soon after initiating infliximab therapy.

A 59 year old Vietnamese male presented with a 2-week history of hematochezia associated with intermittent episodes of abdominal pain relieved by defecation. He was having about 8–10 bowel motions per day which was interfering with his daily activities. His did not have any significant past medical problems, but for 30 pack years history of smoking. On examination, he was afebrile and with normal vital signs. His physical examination showed mild generalized abdominal tenderness on deep palpation. Examination of other systems was negative for any other abnormality. His initial abdominal CT scan showed bowel thickening suggestive of ulcerative colitis. This was further confirmed with colonoscopy and biopsy. His initial treatment with mesalamine and steroids failed to show any significant improvement. Hence intravenous infliximab (5 mg/kg) every 2 weeks was initiated to control his symptoms. Prior to the initiation of the third dose, the patient presented to the emergency room with left-sided chest pain, which was sharp, non-radiating and non-exertional. Patient denied any preceding viral prodromal symptoms. A 12-lead EKG showed saddle shaped global ST elevation that was consistent with pericarditis (Fig. 1). His laboratory data showed a hemoglobin count of 12.2 g/dl [14–18 g/dl], white blood cell count of 6800/ml [4000–11,000/ml], platelet count of 387,000/ml [150,000–450,000/ml], C-reactive protein of 0.5 mg/l [0–5 mg/l] and erythrocyte sedimentation rate of –32 mm/h [20–30 mm/h]. His echocardiogram of heart showed an EF of 60% with no other acute abnormalities. In view of mildly elevated troponins in setting of chest pain, he underwent a coronary angiogram which was normal. His viral serology markers for adenovirus, cytomegalovirus (CMV), Coxsackie virus and Epstein-Barr virus (EBV) were negative. His serum anti nuclear antibody (ANA), anti double stranded DNA (anti ds-DNA), anti-histone antibodies and rheumatoid factor were negative. He was admitted with a diagnosis of possible drug induced pericarditis likely secondary to infliximab therapy which was stopped immediately. NSAIDs were commenced and clinical improvement was observed in the next 2 days. Patient was discharged on the third day with a plan to continue NSAID for 2 weeks.

Adalimumab was later initiated in an attempt to control the ulcerative colitis-related symptoms during his outpatient visit, and there was no recurrence of pericarditis either clinically or electrocardiographically.

Infliximab is a chimeric anti-TNFα antibody generally used as a second line agent when the conventional treatment fails to yield the desired outcome in inflammatory bowel disease. Infliximab therapy is associated with various side effects including infusion reactions, reactivation of infections (tuberculosis, endemic fungal infections, gram positive bacterial infections like peptostreptococcus, listeria,2–4) serum sickness, and hematological as well as bronchogenic malignancies.5 Also, therapy with infliximab is associated with a mortality risk of 1%.6 In our case, the absence of any prodromal illness along with negative viral and rheumatologic markers points towards a non-infectious cause for his pericarditis. The temporal association between starting infliximab and the onset of symptoms, and resolution of symptoms after stopping of the medication was striking. Electrocardiogram changes resolved in 4 weeks after stopping of infliximab, thus proving that infliximab is the most likely culprit for the etiology of pericarditis.

The exact mechanism of infliximab causing pericarditis is not clearly known. Infliximab may have a pro-inflammatory activity in certain tissues including the pericardium. One of the postulated theories is that delayed reactions can cause a serum sickness like reaction (type 3 hypersensitivity), in which pericarditis could have been a component. Development of pericarditis after initiating anti-TNF α therapy (etanercept or infliximab) or even DMARD (sulfasalazine or hydroxychloroquin) therapy for rheumatoid arthritis has been studied in a case series.7 However the authors have concluded that the development of pericarditis could be a part of the disease spectrum caused by rheumatoid arthritis, rather than from the treatment with the drugs. Development of pericarditis...
and subsequently drug induced lupus after restarting the drug for treating Crohn’s disease has been previously reported. In our case, our patient (who was treated for Crohn’s disease as well) did not have any features clinical or serological features of drug induced lupus. Thus, infliximab may rarely cause pericarditis although the exact molecular pathogenesis is not clearly discovered yet.

Infliximab is widely used agent control of inflammatory bowel disease can rarely be associated with pericarditis. Physicians need to be cognizant about this fact and should counsel patient about this rare but clinically significant complication. Pericarditis can sometimes be life threatening and hence cautious monitoring is needed while a patient is on this therapy.

Conflict of interest statement

None for any of the authors.

References