Table 2 Baseline data* for patients developing extra-articular disease versus non-extra-articular rheumatoid arthritis patients

	ExRA	Non-ExRA	
N	22	157	p
Age; years (mean; SD)	48.4 (10.2)	51.9 (12.7)	0.21
Male/female (N)	8/14	56/100	0.89
Current smoker	10 (50%)	41 (36%)	0.22
Ever smoker	15 (75%)	70 (61%)	0.23
IgM RF positive	19 (90%)	116 (76%)	0.25
IgA RF positive	20 (95%)	115 (79%)	0.05
IgG RF positive	18 (86%)	97 (64%)	0.05
ANA positive	10 (45%)	65 (41%)	0.72
Anti-CCP positive	19 (90%)	118 (79%)	0.25
CRP, mg/l (median; IQR)	5 (0–61)	15 (0–42)	0.88
ESR, mm/h (mean; SD)	40.8 (32.5)	35.2 (27.0)	0.38
Complement (mean; SD)	10.9 (3.4)	12.0 (3.8)	0.22
C3, % of normal (mean; SD)	114.2 (11.6)	120.7 (27.3)	0.11
C4, % of normal (mean; SD)	106.1 (25.0)	129.6 (39.5)	0.03
HAQ, mean; SD	0.94 (0.65)	0.92 (0.59)	0.89

ANA, Antinuclear antibody; CCP, cyclic citrullinated peptide; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; ExRA, extra-articular rheumatoid arthritis; HAQ, Health Assessment Questionnaire; IQR, interquartile range; RF, rheumatoid factor.
*Missing data for smoking (two with ExRA and 42 non-ExRA patients), C3 and C4 (seven with ExRA and 37 non-ExRA patients), IgM RF, IgA RF, IgG RF and anti-CCP (one with ExRA, six non-ExRA patients).

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The use of the tumour necrosis factor antagonist infliximab in heart transplant recipients: two case reports

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Inflammatory cytokines, such as tumour necrosis factor alpha (TNF-α), have been implicated in the pathophysiology of heart failure, leading to the hypothesis that TNF inhibition might improve the symptoms of patients with moderate-to-severe cardiac symptoms. Recent data from the Anti-TNF Therapy Against Congestive Heart failure (ATTACH) pilot study, however, suggested that infliximab, a chimeric monoclonal antibody against TNF-α, not only failed to produce clinical benefits, but given at higher doses (10 mg/kg) was associated with a worsening of cardiac symptoms. Conversely, two large-scale trials, RECOVER and RENAISSANCE, examined the effects of infliximab and etanercept in over 2000 patients with heart failure, finding no increased risk of mortality or morbidity.[1] We describe two case reports of patients who received TNF antagonist therapy after heart transplant for heart failure. The patients had a previous history of rheumatoid arthritis, diabetes mellitus, and hypertension, as well as other conditions, including hypercholesterolemia, psoriasis and colon cancer (case 1, Hispanic man, 58 years old) and peripheral vascular disease (case 2, African-American woman, 63 years old). The use TNF antagonist therapy after heart transplant was of concern, in light of the ATTACH study results, as well as the lack of data regarding tumour recurrence in patients on TNF antagonists.

Whereas studies of patients with heart failure have correlated TNF levels with the severity of heart failure[2] and prediction of mortality,[3–5] the exact origin of elevated circulating TNF levels during myocardial ischemia and heart failure is still unclear. Interestingly, TNF has been suggested to have a protective negative inotropic action on the failing heart, contradicting the previous paradigm of TNF being harmful.[6] TNF has also been implicated in the production of heat shock proteins, enabling cells to survive stressful conditions such as ischemia.[7] Placebo-controlled studies have shown that targeted therapy antagonising TNF in patients with moderate-to-severe heart failure has resulted in increased mortality and hospitalisations and the potential induction of a new onset of heart failure or exacerbation of existing disease. It is unclear whether TNF antagonists are safe in heart transplant recipients. Compared with heart transplant recipients without rejection, plasma TNF-α levels were significantly higher in patients with moderate to severe graft rejection.[8]

In the current case study, case 1 underwent heart transplant surgery in 2002 and subsequently a partial colectomy, and was treated with eight weeks of adjuvant chemotherapy for stage 2A colon cancer, initiating infliximab in 2005. The patient has been doing well with infliximab, with no noticeable worsening of the skin disease towards the end of the six-week infusion schedule. Case 2 suffered from dilated and congestive cardiomyopathy status after heart transplantation in May 2002. The patient's rheumatoid arthritis remained active while on methotrexate and prednisone therapy. The patient began infliximab in 2006, with a good response and complete cessation of narcotic medications; however, treatment was discontinued after four months as the patient developed bed sores. Four years after transplant surgery, we observed no complications, from a cardiac standpoint, associated with infliximab treatment. This is, to our knowledge, the first case series reporting the use of TNF antagonists in heart transplant recipients.

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Effective treatment of a colchicine-resistant familial Mediterranean fever patient with anakinra

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Familial Mediterranean fever (FMF) is an autoinflammatory disorder, characterized by periodic fever and serosal inflammation, often complicated by systemic amyloidosis. Maintenance treatment of FMF with colchicine can reduce disease activity and prevent amyloidosis. Some patients, however, fail colchicine therapy. Many reports have recently

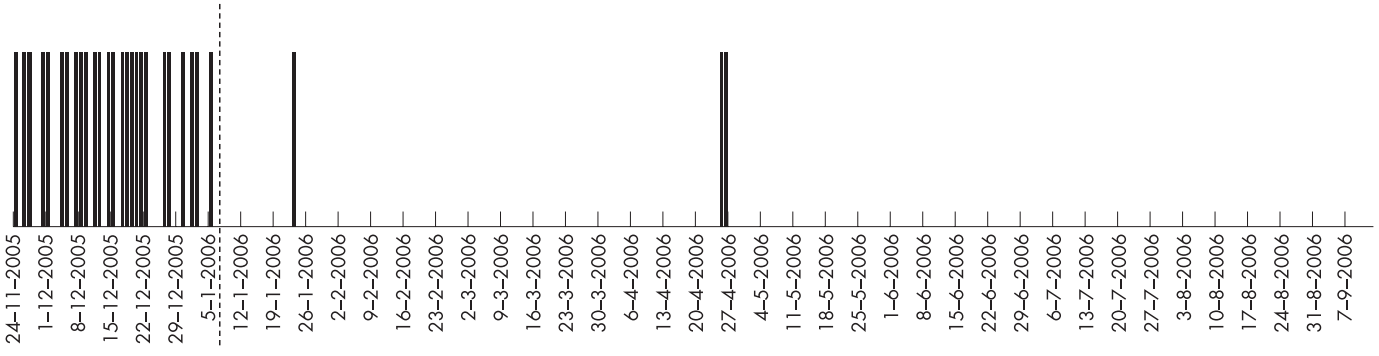

Figure 1 Disease activity before and during anakinra treatment. The bars indicate days when the patient reported being ill in her diary and/or her temperature exceeded 38°C. The vertical dotted line indicates start of treatment.