The authors have declared no conflicts of Disclosure statement for assessing risk and managing MT infection in patients who are immunocompromised. The current national guidelines Caution should be exercised for both MT and NTM infection in the use of therapy as part of the RHAPSODY trial protocol, but it was not made on clinical grounds. The patient did not develop significant rapid deterioration in an immunocompromised patient, more frequently affecting those with chronic lung disease such as chronic obstructive pulmonary disease or bronchiectasis (which was not present in this patient). NTM are also ubiquitous throughout the environment [3] and identification of these organisms, particularly if confined to a positive mycobacterium PCR, would not necessarily confirm these as the causative organism even in the absence of tuberculosis mycobacterium. We agree, however, that a positive PCR result for Mycobacterium tuberculosis (MT) would have been of interest, but it was not locally available. Because of rapid deterioration in an immunocompromised patient, the decision to treat empirically for tuberculosis was essentially made on clinical grounds. The patient did not develop significant evidence of side effects (e.g. liver toxicity) and, hence, in the absence of microbiological confirmation, risks of inadequate treatment outweighed the risks of continuing treatment.

If this case does in fact represent NTM infection then these opportunistic organisms may potentially cause significant disease in patients treated with anti-TNF. Duration of treatment in NTM is often longer than for MT, however; in this case, the cause of immunosuppression was withdrawn. The patient was followed up during and after the completion of treatment and has not suffered relapse.

We would agree with Mangat et al. that an ELISPOT test is potentially more informative in immunocompromised patients. Our patient did have this test prior to commencing anti-TNF therapy as part of the RHAPSODY trial protocol, but it was not available locally when she became unwell. From the limited head-to-head comparison data available, the pooled sensitivity of TB-SPOT-TB, while greater than Quantiferon Gold, is still not complete. Recent national guidelines for tuberculosis diagnosis [4] and an NHS Health Technology Assessment [5] have reviewed the use of γ-IFN assays in latent tuberculosis and briefly considered the use of γ-IFN release assays to rule out active infection. Caution should be exercised for both MT and NTM infection in the immunocompromised patient. The current national guidelines for assessing risk and managing MT infection in patients who are due to start anti-TNF therapy do not yet consider these issues [6].

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of doubling the risk of flares and the difficulty of regaining control but with close monitoring and a gradual reduction of therapy it is intuitive to manage RA patients on the lowest doses, of the fewest DMARDs that the individual patient requires to maintain remission. Clearly a lack of evidence is not evidence against this approach and modern studies are needed.

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Comment on: British Society for Rheumatology and British Health Professionals in Rheumatology guideline for the management of rheumatoid arthritis (after the first 2 years): reply

Sir, We thank McLaren and colleagues [1] for raising a difficult issue, which we all wrestled with when developing the BSR/BHR guidelines [2] and also with the recently published NICE RA Guidelines [3]. The issue over what constitutes optimal treatment is an ongoing area of concern for all. It has been clear from calls to the National Rheumatoid Arthritis Society (NRAS) helpline, annual NRAS surveys to patients and data from the general practice research database (4) that sub-optimal treatment for RA is still a significant problem. After much debate during the development of the NICE guidelines, we concluded that there was sufficient evidence to recommend aggressive multiple therapy for newly diagnosed patients combined with tight control using monthly disease assessment. The emphasis should shift towards addressing the risk of inadequately controlled disease rather than side effects of strictly monitored multiple therapies, especially when faced with a patient who is reluctant to take ‘toxic’ drugs.

Equally, we concluded in the NICE guidelines, that we should cautiously explore the possibility of reducing drug/s once the patient had been stable for some considerable time with the strict proviso that arrangements be in place for prompt review should symptoms worsen. This approach has to be done with full and active participation by the patient. It does appear that the BSR guidelines are at odds with the NICE RA Guidelines on this point. The problem is that we have attempted to provide guidance in the absence of ideal evidence. The real issue is what constitutes remission [5]. Use of plain radiography is most common for assessing progression even though MRI and ultrasound may be more effective [6], but this is still an area of active research. It may be intuitive to manage RA with the lowest doses of the fewest DMARDS possible, but we have considered the evidence available and remain concerned that reduction of DMARDs increases the risk of flare and may worsen comorbidity (especially cardiovascular risk). However, we have to balance this with the central role for patients in choosing how they wish to manage their disease.

In summary, it is a balance that needs to be tailored to each individual patient. On the one hand we want to aggressively control inflammation for the sake of joints and comorbidities. On the other hand, if a patient would be just as well controlled on lower doses of DMARDs, then we should strive for that in negotiation with the patient, and bearing in mind the data that suggest even joints in ‘remission’ may still progress, and even low levels of increased CRP may contribute to comorbidities in the long run. Above all, we need to accept the principle that RA does not totally disappear once established, and the overwhelming majority of patients will need some sort of DMARD for ever.

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