against C. bantiana in this setting [8], and in vitro it is the most potent anti-fungal agent [9].

In the presented case, it is unclear if the cerebral abscess led to dissemination to the knee joint or vice versa. To the authors’ knowledge, this is the first report of isolation of C. bantiana from a joint.

**Rheumatology key message**

- *Cladophialophora bantiana* is a rare cause of septic arthritis; however, when disseminated it is highly fatal.

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Comment on: Obstructive sleep apnoea in relation to rheumatic disease

Sir, We read with a keen interest a recent article by Taylor-Gjevre et al. [1] regarding the association between obstructive sleep apnoea (OSA) and rheumatic disease. We are grateful to the authors for reviewing this important problem. Nevertheless, we believe that certain aspects should be mentioned.

Indeed, as was mentioned by the authors, patients with RA may be at greater risk of OSA. This association may be due not only to anatomical changes related to RA pathogenesis (such as temporomandibular joint destruction, retrognathia/micrognathia and occipitocervical lesions), but also to effects of glucocorticoids (GCs), which are used to manage RA. It is well known that an excessive amount of GC leads to a redistribution of adipose tissue to the face and neck area [2], which theoretically should increase OSA severity. Furthermore, patients with AS may be at greater risk of OSA development [3]. A theoretically plausible explanation for the association between RA and AS may be explained by the overexpression of TNF-α, which is a key pro-inflammatory biomolecule. Drugs targeting TNF-α are used with success in patients with both RA and SpA, which supports a key role of TNF-α in the pathobiology of these rheumatic diseases. Loubaki et al. [4] showed that TNF-α is abundant in the upper airway (UA) musculature. Local overexpression of TNF-α may lead to UA muscle and neural damage, with a resultant increase in OSA risk or its severity [5]. Walsh et al. [6] recruited 63 subjects with SpA and studied the impact of TNF-α inhibitors on OSA severity. These investigators showed that patients using TNF-α inhibitors had a lower frequency of OSA. However, the small study sample in their work warrants a study with a larger sample size to give a better understanding of whether this group of medicines may prevent OSA in susceptible individuals. Moreover, patients with primary SS were found to have a greater burden of OSA, which was probably explained by UA dryness with a resultant increase in collapsibility [7]. Conversely, can OSA be a risk factor for rheumatic diseases? In a retrospective epidemiological study, Kang et al. [8] showed that patients with OSA had a greater risk of development of RA compared with the general population. The same research group showed that OSA is associated with an increased risk for the development of psoriasis and PsA [9]. However, these studies did not answer the question of whether OSA severity and OSA treatment have any impact on the incidence of autoimmune diseases. Thus further studies should address these fundamental questions. The mechanistic question is how OSA can increase the occurrence of rheumatic diseases. Chronic intermittent hypoxia, which is a pathophysiological driver of OSA, leads to activation of
various pro-inflammatory molecules via hypoxia-inducible factor-1 and TNF-α [10]. Indeed, an exaggerated inflammatory response may lead to autoimmune disease incidence in susceptible individuals. A simplified sketch of the interrelationship between OSA and rheumatic diseases is presented in Fig. 1.

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Comment on: Obstructive sleep apnoea in relation to rheumatic disease: reply
Sir, We would like to thank Drs Mirrakhimov and Mirrakhimov [1] for their kind interest in our recent