improved when he was given high-dose corticosteroids. He was successfully weaned from the ventilator on day 6 after intubation (which is very good for ARDS). Given that steroids are not greatly beneficial in the treatment of ARDS [3], it is likely that the improvement of the respiratory symptoms in this patient also resulted from the prompt suppression of the inflammatory systemic response by corticosteroids. This, together with appropriate mechanical ventilation, was probably the main reason for a successful outcome. Tacrolimus may have had little or no role in the patient's recovery as it is slow-acting and unlikely to have been playing a significant role for several weeks.

In severe interstitial lung disease associated with polymyositis/dermatomyositis that is refractory to corticosteroids, cyclophosphamide has been the treatment of choice in many centres. Tacrolimus has not been widely investigated, it is overly expensive and important adverse events (more nephrotoxicity and neurotoxicity compared with ciclosporin) are common. However, tacrolimus could be reserved for those refractory cases who respond poorly to standard combination regimen.

Finally, could any of the clinical situations be explained by drug side-effects, such as the pneumonitis and rhabdomyolysis that can occur with levofloxacin. Further details of the patient history would also be helpful. Has the pulmonary disease and myositis remained in remission and what therapy is currently being used? We would also encourage the diagnosis of myositis using electromyography and a muscle biopsy.

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STATEMENT OF INTEREST

None declared.

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From the authors:

We thank our colleagues for their letter, providing the opportunity to underline the interest in the relevant case study [1].

Until recently, it was not well recognised that interstitial lung disease in polymyositis patients with anti-Jo-1 can present an acute onset with a severe outcome, as recently reported [2, 3].

Acute respiratory distress syndromes respond to a physiological definition, which does not always present in connection with diffuse alveolar damage but can be related to other processes. In our case report, we show an acute respiratory distress syndrome (ARDS) due to a nonspecific interstitial pneumonitis (NSIP), proven by an open lung biopsy. If an ARDS is properly diagnosed by such specific examinations, a more specific therapy may be applied. In our case, the presence of anti-Jo-1 antibodies with a predominant CD8 cellular NSIP and myositis allowed a precise diagnosis.

As shown in our study, this entity is corticosteroid sensitive but such treatment is often insufficient and makes an additional therapy with immunosuppressors necessary [2]. Cyclophosphamide is still frequently recommended, despite its partial efficiency and high toxicity limiting its use beyond 6–12 months. Our case report underlines again the usefulness of calcineurin inhibitors in this T-cell-mediated disease, which has also been demonstrated previously [3, 4]. The toxicity of these drugs, in particular of tacrolimus, is limited if drug monitoring is performed as it is in transplantation [5], currently allowing rather well-tolerated treatments for >10 yrs. The rapid improvement of gas exchanges and myositis should allow challenge of the concept that calcineurin inhibitors do not act quickly in settings where the cellular NSIP has not yet been followed by an irreversible lung fibrosis. A long-term improvement has previously been described in 13 patients with severe interstitial diseases [3], who did not respond to more classical immunosuppressors. In these cases, early treatment with tacrolimus is suggested before lung fibrosis is established and we can propose monitoring of the blood while levels of the drug are between 5 and 10 ng·mL⁻¹, in order to optimise the long-term incidence of side effects [5].

In our case and that reported by WILKES *et al.* [3], low dose steroids and tacrolimus were applied without using other immunosuppressive drugs in order to avoid opportunistic infection. According to WILKES *et al.* [3], patients received this treatment for an average duration of 51 months. Our patient returned abroad and follow-up was lost after 8 months of successful treatment. In our clinic, such good and long-term follow-up has been experienced in further cases but has not yet been published.

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STATEMENT OF INTEREST

None declared.

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Predicting outcome of nasal surgery in patients with obstructive sleep apnoea

To the Editors:

In a recent randomised sham-surgery-controlled trial, KOUTSOURELAKIS *et al.* [1] demonstrated that nasal surgery for fixed nasal obstruction did not improve, or even worsened, sleep-disordered breathing in a majority of 27 patients with obstructive sleep apnoea (OSA) syndrome.

Based on previous studies showing that the oral breathing route predisposes to OSA [2], KOUTSOURELAKIS *et al.* [1] compared the proportion of nasal breathing during sleep in responders and nonresponders to nasal surgery.

This *post hoc* analysis revealed that four responders spent a low proportion of the pre-operative sleep study with nasal breathing, as assessed *via* nasal cannula/pressure transducer and oral thermistor measurements. KOUTSOURELAKIS *et al.* [1] concluded that analysis of the oral–nasal breathing route during sleep might help to identify favourable candidates for nasal surgery among OSA patients with fixed nasal obstruction. As KOUTSOURELAKIS *et al.* [1] pointed out, their technique for assessing impaired nasal breathing during sleep was not ideal since it could not quantify nasal ventilation, and conventional rhinomanometry was also unhelpful in predicting the success of nasal surgery.

It has previously been shown that nasal resistance (and nasal airflow) is highly variable over time, with changes in body position and during sleep [3]. The cyclical changes in side-predominance of nasal airflow over time (the nasal cycles) additionally contribute to the high variability of nasal resistance, particularly in the presence of anatomical obstacles. It is, therefore, unsurprising that the outcome of nasal surgery cannot be reliably predicted from measurement of nasal resistance by rhinomanometry during wakefulness [4] or from qualitative oral–nasal flow recordings. An unobtrusive technique based on a bilateral nasal cannula/pressure transducer system that permits side-selective quantitative measurements of nasal airflow and conductance over the course of a night has recently been developed and validated [3, 5].

We suggest that future studies on the effects of nasal surgery or pharmacological treatment on sleep-disordered breathing

should include continuous quantitative nocturnal nasal conductance measurements for the accurate assessment of a subject's nasal patency as a means of identifying potential responders to treatment.

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STATEMENT OF INTEREST

None declared.

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From the authors:

We would like to thank M. Kohler and K.E. Bloch for their interest and comments about our recent paper [1] and we appreciate the opportunity to make some remarks in reply to their letter.