volume in 1 s (FEV₁) reduced to 1.7 litres (46.8% of predicted) and FEV₁/forced vital capacity (FVC) ratio 55.9%. Since his RA responded favourably on adalimumab and there was not enough evidence to ascribe his respiratory complaints to this drug, it was decided to rechallenge him with adalimumab. During the third administration of adalimumab he still used prednisolone 20 mg/day and inhaled budesonide/formoterol twice daily. On the third day following adalimumab administration he developed dyspnoea, wheezing and a reduction in his PEF to 290 l/min. His PEF went back to baseline after 2 weeks.

Figure 1 The patient was instructed to monitor his peak expiratory flow (PEF) twice a day. The first 2 days after the third adalimumab administration he had a stable PEF of approximately 450 l/min. On the third day he developed dyspnoea, wheezing and a reduction in his PEF to 290 l/min. His PEF went back to baseline after 2 weeks.

Bennett et al reported a patient with presumed adalimumab-induced asthma. They hypothesised that once the tumour necrosis factor (TNF)α blocking adalimumab was introduced the T helper cell (Th)1 response characteristic for RA was suppressed, allowing the Th2-activated pathway to express itself as asthma. However, patients with asthma have upregulation of the TNFα axis so one would expect suppression of asthmatic inflammation. Since Bennett and colleagues assumed that the pathophysiological mechanism causing adalimumab was a direct effect of the TNFα blockade, they suggested a class effect and decided not to treat their patient with other TNFα blocking agents. Our patient used etanercept, adalimumab and infliximab within a short time frame and only reacted to adalimumab. This case therefore refutes the hypothesis that the asthmatic response is caused by blockage of TNFα, as well as the existence of a class effect. Symptoms started 3 days after drug administration which is not compatible with anaphylaxis. A delayed-type T-cell mediated hypersensitivity reaction would be more likely.

At present it is unclear how often adalimumab has induced asthma-like symptoms. Because the use of adalimumab is increasing, this adverse event may become more prevalent in the future. We are the first to report adalimumab-induced bronchospasm with a positive rechallenge on the agent itself and negative challenges on etanercept and infliximab. We believe it is justified to make a switch to a different TNFα blocker under strict medical observation.

REFERENCES

Renal impairment in cystic fibrosis

We read with interest the paper by Bertenshaw et al on the incidence of acute renal failure (ARF) in patients with cystic fibrosis (CF) and would like to supplement these results with our findings. With increasing survival and therefore consideration for lung transplantation, monitoring adult patients with CF for renal impairment is assuming increasing importance (creatinine clearance <50 mg/ml/min is a contraindication to lung transplantation). Since Bennett and colleagues assumed that the pathophysiological mechanism causing adalimumab was a direct effect of the TNFα blockade, they suggested a class effect and decided not to treat their patient with other TNFα blocking agents. Our patient used etanercept, adalimumab and infliximab within a short time frame and only reacted to adalimumab. This case therefore refutes the hypothesis that the asthmatic response is caused by blockage of TNFα, as well as the existence of a class effect. Symptoms started 3 days after drug administration which is not compatible with anaphylaxis. A delayed-type T-cell mediated hypersensitivity reaction would be more likely.

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