

**Introduction**

Inflammatory bowel diseases (IBD) are chronic inflammatory disorders of the gastrointestinal (GI) tract that comprise CD and UC. IBD is a lifelong chronic, disabling and progressive disease. Nonbiological drug therapies such as steroids and immunomodulators improve symptoms but do not stop the underlying inflammatory process or the disease course. The introduction of TNF-α antagonists has considerably improved the management of IBD, resulting in steroid-sparing treatment, fewer hospitalisations and surgeries, greater clinical remission and mucosal healing rates, and improved quality of life and work productivity. However, approximately one third of patients are primary nonresponders to TNF-α antagonists, and another 30–40% of primary responders eventually lose response. Infusion reactions to infliximab are known but are fortunately uncommon. In addition, TNF-α antagonists may be associated with a number of serious and potentially life-threatening adverse events such as malignancies or opportunistic infections. Hence, new therapeutic strategies are urgently needed in both TNF-α antagonist-naïve and failure patients.

**The role of αβ7, integrin in IBD pathogenesis**

While the exact mechanisms behind IBD are not entirely understood, it is thought that multiple inflammatory cell types, including neutrophils, macrophages, dendritic cells and lymphocytes are involved, with lymphocytes playing a central role in the induction and maintenance of chronic inflammation in the lamina propria. T lymphocytes enter the GI tract via distinct molecular mechanisms not seen in other peripheral tissues. This infiltration process is coordinated by the interaction between adhesion and signalling molecules on the surface of T lymphocytes (integrins, selectins, and chemokine receptors) and their corresponding ligands on the endothelium. In particular, the αβ7 integrin binds to mucosal addressin cell adhesion molecule 1 (MAdCAM-1) on endothelial cells, allowing memory T lymphocytes to invade the GI tract (Figure 1). The high recruitment of T lymphocytes and subsequent cytokine production has been shown to be key in the pathogenesis of IBD by affecting the endothelial barrier and inducing cell apoptosis in endothelial cells. Therefore, αβ7 integrin is an ideal therapeutic target in IBD.

**Vedolizumab has a gut-selective mechanism of action**

Vedolizumab is a humanised monoclonal antibody that exclusively blocks the α4β7 integrin on peripheral T lymphocytes and inhibits adhesion of the lymphocyte to MAdCAM-1 (Figure 2). In doing so, vedolizumab modulates inflammation in the GI tract without inducing the systemic immunosuppression, such as fatal progressive multifocal leukoencephalopathy (PML), associated with monoclonal antibodies that are dual inhibitors of α4 and αβ7 integrins. Vedolizumab does not affect levels of T lymphocytes in the cerebrospinal fluid of healthy volunteers after a single dose, nor does it inhibit immune surveillance of the central nervous system (CNS) in non-human primates. To date, there have been no reported cases of PML in patients treated with vedolizumab following over 3000 subjects recruited into clinical trials and several thousand following the drug’s regulatory approval across the world. The precise targeting of vedolizumab to lymphocyte trafficking systems within the gut may provide an improved risk–benefit profile.
Vedolizumab does not bind to the majority of memory CD4+ T lymphocytes (60%), neutrophils, α4β7-integrin, and 13% of patients, respectively. Vedolizumab selectively inhibits adhesion of α4β7-integrin to MAdCAM-1, mucosal addressin cell adhesion molecule; PSGL-1, P-selectin glycoprotein ligand; VCAM-1, vascular cell adhesion molecule I

Vedolizumab’s unique gut-selective mechanism of action is largely attributable to four properties: 

1. Restriction of α4β7-integrin expression to subsets of leukocytes: 
   - Vedolizumab does not bind to the majority of memory CD4+ T lymphocytes (60%), neutrophils, and most monocytes. The highest level of binding is to a subset of human peripheral blood memory CD4+ T lymphocytes that include gut-homing interleukin-17 T-helper lymphocytes. Vedolizumab also binds to eosinophils at high levels, but to naïve T-helper lymphocytes, naïve and memory cytotoxic T lymphocytes, B lymphocytes, natural killer cells and basophils at lower levels.
2. Binding specificity: 
   - Vedolizumab binds exclusively to the α4β7-integrin that is expressed in increased amounts in IBD and not to the α4β7 and α5β1-integrins and monoclonies that are required for immunosurveillance and host defense.
3. Selective antagonism: 
   - Vedolizumab selectively inhibits adhesion of α4β7-expressing cells to MAdCAM-1 and fibronectin, but not vascular cell adhesion molecule-1 (VCAM-1).
4. Gut-specificity: 
   - The role of the α4β7 integrin in immunosurveillance is restricted to the GI tract. Gut-specific immunomodulation by vedolizumab is therefore less likely to be associated with infection and/or neoplasia outside of the GI tract than anti-α4, anti-β7, and anti-p40 subunit therapeutics.

Indications
Vedolizumab is indicated for the treatment of adult patients with moderate-to-severe UC or CD who have had an inadequate response with, lost response to, or are intolerant to either conventional therapy or TNF-α antagonists.

Dosage and administration
The recommended dosage of vedolizumab is 300mg given as a 30 minute IV infusion. A three-dose induction regimen is administered at weeks 0, 2 and 6, with maintenance therapy administered every 8 weeks from week 14. Continued treatment is not recommended for patients who have not shown a clinical response by week 14. No weight- or age-based dose adjustments are required.

Pharmacokinetics
The population pharmacokinetics of vedolizumab was similar in patients with moderate-to-severe UC and CD. Pharmacokinetics was described by a two-compartment model with parallel linear and nonlinear elimination. Linear elimination half-life was 25.5 days; linear clearance was 0.159 L/day for UC and 0.155 L/day for CD; central compartment volume of distribution was 3.19L and peripheral compartment volume of distribution was 1.66L.

Adverse events
In three randomised, controlled trials in 1731 patients with UC (GEMINI 1) or CD (GEMINI 2 and 3) treated with vedolizumab or placebo for up to 52 weeks, adverse events (AEs) were reported in 84% of patients treated with vedolizumab and 78% of patients treated with placebo. Serious AEs were reported in 19% and 13% of patients, respectively. The proportion of patients who discontinued treatment due to AEs was 9% for patients treated with vedolizumab and 10% for patients treated with placebo. AEs that occurred in more than 3% of patients are shown in Table 1 and AEs of specific interest are discussed below.

Infusion-related reactions
In the GEMINI 1 and 2 studies, 4% of vedolizumab-treated patients and 3% of placebo-treated patients experienced an AE defined by the investigator as an infusion-related reaction (IRR). The majority of IRRs were mild or moderate in intensity and <1% resulted in treatment discontinuation. IRRs generally resolved with no or minimal intervention and most occurred during the infusion or within the first 2 hours. However, a few IRRs were noted after 2 hours and more than 2 days after infusion. One serious IRR was reported by a CD patient during the second infusion and was successfully managed with discontinuation of infusion and treatment with antihistamine and IV hydrocortisone.

Infections
In the GEMINI 1 and 2 studies, the rate of infections was 0.85 per patient-year in vedolizumab-treated patients and 0.70 per patient-year in placebo-treated patients. Infections consisted primarily of nasopharyngitis, upper respiratory tract infections, sinusitis, and urinary tract infections. Most patients continued on vedolizumab after the infection resolved. The rate of serious infections was 0.07 per patient-year in vedolizumab-treated patients and 0.06 per patient-year in placebo-treated patients, with no significant increase over time. In controlled and open-label studies in patients treated with vedolizumab, serious infections have included tuberculosis, sepsis (some fatal), salmonella sepsis, listeria, meningitis, and cryptomegaloviral colitis.

Liver injury
There have been reports of elevations of transaminase and/or bilirubin in patients receiving vedolizumab. In the GEMINI trials, three patients reported serious adverse reactions of hepatitis after two to five vedolizumab doses; however, it was unclear if the reactions indicated drug-induced or autoimmune aetiology. All patients recovered following discontinuation of therapy with some requiring corticosteroid treatment. In controlled trials, the incidence of ALT and AST elevations ≥3 x ULN was <2% in both the vedolizumab and placebo groups. In an open-label trial, one case of serious hepatitis was observed.

Malignancy
Overall, results from clinical trials to date do not suggest an increased risk for malignancy with vedolizumab treatment; however, the number of malignancies was small and long-term exposure was limited. Long-term safety evaluations are ongoing.

Immunogenicity
In the GEMINI 1 and 2 studies, vedolizumab showed an immunogenicity rate of 4%. Five percent of patients with IRR were persistently anti-vedolizumab antibody-positive. Overall, there was no apparent correlation of anti-vedolizumab antibody development to clinical response or AEs. However, the number of patients that developed anti-vedolizumab antibodies was too limited to make a definitive assessment.

Progressive multifocal leukoencephalopathy
Vedolizumab has no known systemic immunosuppressive activity and no cases of PML have been identified among patients with at least 24 months of exposure to vedolizumab. However, the risk of PML cannot be ruled out. Some integrin antagonists and some systemic immunosuppressive agents have been associated with PML, a rare and often fatal opportunistic infection of the CNS caused by the John Cunningham (JC) virus.
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**Ulcerative colitis**

**GEMINI 1**

GEMINI 1 enrolled adult patients with active UC (Mayo Clinic score 6–12 with endoscopic subscore ≥2) who had failed conventional therapy (including corticosteroids or immunomodulators) or TNF-α antagonists. TNF-α antagonist failure patients included those with inadequate response (primary non-responders), loss of response (secondary non-responders) or those who were intolerant. Approximately 40% of patients had failed prior TNF-α antagonist therapy.

**Induction**

The primary endpoint in the randomised induction phase (n=374) was the proportion of patients with clinical response (reduction in Mayo Clinic score of ≥3 points and ≥30% from baseline) and no accompanying decrease in rectal bleeding subscale of ≥1 point or absolute rectal bleeding score of ≤1 point at week 6. The secondary endpoints were clinical remission at week 6 (Mayo Clinic score of ≤2 points and no individual subscore >1 point) and mucosal healing at week 6 (endoscopic subscore of ≤1 point).

Significantly more patients treated with vedolizumab compared to placebo achieved clinical response, clinical remission, and mucosal healing at week 6 (Table 2). In exploratory analyses, the beneficial effect of vedolizumab on clinical response, remission and mucosal healing was observed in patients who had failed prior TNF-α antagonist therapy and in those who were TNF-α antagonist naive.

**Maintenance**

The primary endpoint in the maintenance phase (n=373) was the proportion of patients in clinical remission at week 52. The secondary endpoints were durable clinical response (clinical response at both weeks 6 and 52), durable clinical remission (clinical remission at both weeks 6 and 52), mucosal healing at week 52 (Mayo endoscopic subscore of ≤1 point), and corticosteroid-free remission at week 52.

Significantly more patients treated with vedolizumab compared to placebo achieved clinical remission at week 52 (Table 2). In addition, significantly more patients treated with vedolizumab demonstrated durable clinical response and remission, mucosal healing, and corticosteroid-free remission.

**Table 1. AEs occurring in ≥3% of vedolizumab-treated patients with UC and CD (GEMINI 1 and 2)**

<table>
<thead>
<tr>
<th>Adverse reaction (%)</th>
<th>Placebo (n=297)</th>
<th>Vedolizumab (n=1434)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper respiratory tract infection</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>Fatigue</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Cough</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Bronchitis</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Influenza</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Back pain</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Rash</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Pruritus</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Sinusitis</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Oropharyngeal pain</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Pain in extremities</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

**Design of phase III GEMINI trials in UC and CD**

The clinical efficacy of vedolizumab in UC and CD has been demonstrated in a number of clinical trials, with the key studies being the phase III GEMINI trials. These placebo-controlled studies were designed to assess the efficacy and safety of vedolizumab in the induction of clinical response and remission in patients with moderate-to-severe active UC and CD. GEMINI 1 and 2 also assessed the efficacy and safety of vedolizumab in the maintenance of remission in patients achieving an initial response to therapy. All three studies followed a similar design (Figure 3). In each study, an initial cohort (Cohort 1) was randomised to IV vedolizumab 300mg at week 0 and 2, with the primary clinical endpoints evaluated at week 6. In GEMINI 1 and 2, patients who met week 6 response criteria were then entered into maintenance studies and randomised to placebo or vedolizumab every 4 or 8 weeks. In order to achieve sufficient statistical power, the group entering maintenance studies comprised Cohort 1 responders as well as responders from an additional cohort given open-label vedolizumab at week 0 and 2 (Cohort 2). Non-responders from both cohorts continued to receive vedolizumab every 4 weeks, whilst placebo patients from the induction study continued on placebo in the maintenance study. Neither of these two groups were included in the intention-to-treat analysis, but contributed to the safety and exploratory endpoint analyses. Maintenance studies reported outcomes at 52 weeks, with patients deriving clinical benefit invited to enter an ongoing, open-label long-term safety and efficacy follow-up study (GEMINI-LTS).

**Figure 3. Design of the GEMINI trials**

*Design of the GEMINI trials*
Table 2. Outcome measures at week 6 and 52 in GEMINI 1²⁰

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Placebo (n=126)</th>
<th>Vedolizumab q4w (n=126)</th>
<th>p value ⁹</th>
<th>Vedolizumab q8w (n=122)</th>
<th>p value ¹⁰</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical response (%)</td>
<td>15.9</td>
<td>44.8</td>
<td>&lt;0.001</td>
<td>41.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Durable clinical response (%)</td>
<td>23.8</td>
<td>52.0</td>
<td>&lt;0.001</td>
<td>56.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Durable clinical remission (%)</td>
<td>8.7</td>
<td>24.0</td>
<td>0.001</td>
<td>20.5</td>
<td>0.008</td>
</tr>
<tr>
<td>Mucosal healing (%)</td>
<td>19.8</td>
<td>56.0</td>
<td>&lt;0.001</td>
<td>51.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Corticosteroid-free remission</td>
<td>13.9</td>
<td>45.2</td>
<td>&lt;0.001</td>
<td>31.4</td>
<td>0.01</td>
</tr>
</tbody>
</table>

* placebo vs vedolizumab q4w  ‡ placebo vs vedolizumab q8w

In the GEMINI 1 study, the induction regimen was administered at weeks 0 and 2 and maintenance dosing started at week 6. However, exploratory analyses suggest a higher rate of long term clinical response and remission will be achieved with a 0, 2 and 6 week induction regimen followed by maintenance treatment every 8 weeks for patients who demonstrate a clinical response 6 to 8 weeks after completion of the induction regimen.²⁰ Relatively few patients (10 placebo recipients and 8 vedolizumab recipients) had major UC-related events (colectomy, UC-related hospitalisation, or UC-related procedure).²⁰ However, the proportion of patients who experienced these major UC-related events was lower among patients who received vedolizumab (9% in each vedolizumab group) compared with those who received placebo (8%).²⁰

Expert comment on GEMINI 1

Inhibition of lymphocyte trafficking through blockade of gut-selective integrins has certainly become an attractive therapeutic target. Several new study compounds are under investigation for the treatment of UC and CD to take advantage of the efficacy and safety of this mechanism. For UC in particular, the induction and maintenance of remission data lend support to their use in those failing conventional therapy. Even when recruiting severe UC patients (41% of subjects had failed one or more anti-TNF agent and were taking a median of 20mg of prednisolone), the clinical response rate of 47% was 22% above that of placebo (p<0.001) after only two infusions. The mucosal healing rate at the end of induction was 41%, 16% above that of the placebo group. These data demonstrate excellent induction efficacy in a difficult-to-treat UC cohort. The maintenance clinical remission rates of 45% and 42% for 4-weekly and 8-weekly infusions respectively, were significantly higher than the placebo rate of 16% (p<0.001). The mucosal healing rates of 56% and 52% with 4- and 8-weekly vedolizumab infusions compared to 20% for the placebo group (p<0.001) is equally impressive using this objective endpoint. The main comparator of vedolizumab is infliximab. Data from anti-TNF trials suggest that they have a more rapid onset of action. But the trade-off of greater immunogenicity, systemic immunosuppression, relatively high loss of response rates and lymphoma risk-recognitions with anti-TNF agents supports the earlier introduction of vedolizumab in UC. The proviso is the need to ensure induction is well-managed with other agents such as corticosteroids.

Crohn’s disease

GEMINI 2²¹ and GEMINI 3²² enrolled adult patients with moderate-to-severe CD (CD Activity Index [CDAI] score of 220–450) who had failed at least one conventional therapy (including corticosteroids or immunomodulators) or had failed TNF-α antagonists. TNF-α antagonist failure patients included those with inadequate response (primary non-responders), loss of response (secondary non-responders) or those who were intolerant.

GEMINI 2

Induction

In the randomised induction phase of GEMINI 2 (n=368) the two primary endpoints were the proportion of patients in clinical remission (CDAI score ≤150 points) at week 6 and the proportion of patients with enhanced clinical response (≥100-point decrease in CDAI score from baseline) at week 6. Approximately 50% of patients had failed prior TNF-α antagonist therapy. A significantly higher percentage of patients treated with vedolizumab achieved clinical response compared to placebo at week 6 (Figure 4). The difference in the percentage of patients who demonstrated enhanced clinical response was however, not statistically significant at week 6. The beneficial effect of vedolizumab on clinical remission was similar in patients naive to TNF-α antagonist exposure as well as in those who had failed prior TNF-α antagonist therapy.

Maintenance

To evaluate efficacy at week 52, 461 patients who achieved clinical response (≥70-point decrease in CDAI score from baseline) at week 6, were randomised to maintenance therapy as described above. The primary endpoint was the proportion of patients in clinical remission at week 52. The secondary endpoints were enhanced clinical response, corticosteroid-free remission, and durable clinical remission (clinical remission ≥80% of study visits, including final visit at week 52). Significantly more patients treated with vedolizumab compared to placebo achieved clinical remission at week 52 (Figure 4). In addition, significantly more patients treated with vedolizumab demonstrated enhanced clinical response and corticosteroid-free clinical remission. The between-group difference was not significant for durable clinical remission.

Figure 4. Outcome measures at week 6 and 52 in GEMINI 2²¹

Week 6 (induction)

Week 52 (maintenance)
In the GEMINI 2 study, the induction regimen was administered at weeks 0 and 2 and maintenance dosing started at week 6. However, similar to the GEMINI 1 study, exploratory analyses suggest a higher rate of long term clinical response with a 0, 2 and 6 week induction regimen followed by maintenance treatment every 8 weeks for patients who demonstrate a clinical response 6 to 8 weeks after completion of the induction regimen.18

**Expert comment on GEMINI 2**

Results from the GEMINI 2 study for CD differ to those of the GEMINI 1 study for UC. The co-primary endpoint of clinical remission was statistically significant whilst that of clinical response (CDAI drop of at least 100 points) was not. CDAI drop of 100 is a tough endpoint given that only two infusions were given. The time to response for CD may be longer than UC given transmural inflammation in CD may mean a higher number of lymphocytes need to be cleared prior to the blockade of new lymphocytes traffick into the gut can take effect. The choice of CDAI 100 also means that those with milder disease on entry need to demonstrate a CDAI drop far below that of remission (CDAI of 150) before meeting this endpoint. The CD cases recruited were quite refractory with a median of 8–9 years of disease, 48% failing one or more anti-TNF agent and median prednisolone dose of 20mg on entry. It was always going to be difficult to meet primary endpoints after only two infusions as early as week 6. The remission rates at week 52, however, are impressive at 36% and 39% with 4- and 8-weekly infusions compared to placebo of 22% (p<0.004). Being on concomitant immunosuppressive and/or prior anti-TNF use did not predict for a better or poorer response. At present there is no advice regarding whether to continue or to cease immunomodulator co-therapy. However, immunogenicity appears to be lower than with anti-TNF agents.

**GEMINI 3**

The GEMINI 3 study (n=416) was a second randomised, double-blind, placebo-controlled study that evaluated efficacy at week 6 and 10 in a subgroup of patients defined as having failed conventional therapy and TNF-α antagonist therapy, as well as the overall population, which also included patients who failed conventional therapy and were naïve to TNF-α antagonist therapy.19 The primary endpoint was the proportion of patients in clinical remission (CDAI score ≤150 points) at week 6 in the TNF-α antagonist failure subpopulation. In the TNF-α antagonist failure population (n=315), vedolizumab was not significantly more effective than placebo in inducing clinical remission at week 6 (15.2% vs 12.1%; p=NS). However, secondary and exploratory outcome results suggest that vedolizumab had clinically relevant activity in both TNF-α antagonist failure and -naïve patients. At week 10, a higher proportion of TNF antagonist-α failure patients treated with vedolizumab were in clinical remission than those given placebo (26.6% vs 12.1%; p<0.001). Analyses of the overall population showed more vedolizumab-treated patients than placebo-treated patients in clinical remission at week 6 (19.1% vs 12.1%; p=0.048) and week 10 (28.7% vs 13.0%; p<0.0001). These findings indicate that additional benefits of vedolizumab therapy may increase between weeks 6 and 10, regardless of previous TNF-α antagonist response, and may be associated with the effects of an extra vedolizumab dose at week 6 or with the incremental effect of time on the drug’s ability to exert a therapeutic benefit. This observation may help with the optimization of vedolizumab induction therapy in a clinical setting.

**Expert comment on GEMINI 3**

The choice of assessing the primary endpoint after only two infusions of vedolizumab at week 6 once again failed to identify differences in the remission rates with a difference of only 3% between vedolizumab and the placebo group. However, the exploratory endpoint assessment at week 10, when patients had received three infusions, was significantly higher than the placebo group. As such this induction study would recommend assessing the response rate after at least three infusions. The drug appears to have an onset of action slower than that of anti-TNF agents, especially in patients that had already failed anti-TNF agents.

**Conclusions and future directions**

Vedolizumab appears to be an important, much needed new option for patients with IBD. Standard therapies are suboptimal, with many patients not responding, relapsing, or experiencing serious side effects. In UC, vedolizumab has shown clear efficacy in inducing and maintaining remission in patients who have failed first-line treatment and are TNF-α antagonist naïve, as well as those who have failed TNF-α antagonist therapy. Results in CD are less dramatic, but still show an effect that will translate into important clinical benefits for some patients. In common with all existing IBD therapies, there is a lack of adequate biomarkers and mechanistic insights to predict the patients that will derive sustained benefit. To date, vedolizumab has shown a good level of patient acceptability and a reasonable safety profile with a low risk of immunosuppression; these are important features of vedolizumab, as IBD is a lifelong disease and many patients with the disease are young and face years of therapy. However, prospective studies are required to ascertain its long-term benefits and side effect profile in the treatment of IBD. Further trials directly comparing vedolizumab with other biological agents as well as studies evaluating cost effectiveness will be useful.

**Experts concluding comments**

Vedolizumab is the first non-anti-TNF biological agent to be available for the treatment of CD and UC. The drug can be used in anti-TNF naïve and also the more difficult anti-TNF failure groups. Response is more rapid in the UC group than in CD, and may reflect differences in the disease and the fact that the drug’s mechanism of action requires more time. A greater emphasis on inducing remission with corticosteroids is required. The benefits of this drug include the impressive steroid-free remission rates, the gut-specific targeting, the adverse reaction rates being similar to placebo and so far no cases of PML. Pharyngitis seems to be the most relevant AE in the early days of this treatment and is consistent with the expression of the α4β7 integrin at the pharyngeal mucosa. Immunogenicity also appears lower, although the relevance of antibodies to vedolizumab is so far undefined. The drug pharmacokinetics suggests weight-based dosing is not required, increasing the efficiency of reconstituting the standard dose of 300mg. Eight-weekly infusions are also convenient for patients. Further questions include whether immunomodulator co-therapy is needed to reduce loss of response in the long term. GI infections and intestinal dysplasia remain of concern and follow up post-marketing surveillance data will prove important for assessing other rarer side effects. This drug should certainly be incorporated into the management algorithm of both CD and UC.
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References


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