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Predictors of Clinical Response and Remission at One year among a Multicenter Cohort of Patients with Inflammatory Bowel Disease Treated with Vedolizumab

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Abstract

Background—Vedolizumab (VDZ) has demonstrated long term efficacy in Crohn's disease (CD) and ulcerative colitis (UC) in phase III trials.

Aims—Our aim was to evaluate the efficacy of VDZ at week 54 in inflammatory bowel disease (IBD) in a multicenter cohort of patients.

Methods—Adult patients completing induction therapy with VDZ were eligible for this study. Clinical response and remission was assessed using the Harvey Bradshaw index (HBI) for CD, the simple clinical colitis activity index (SCCAI) for UC and physician assessment.

Results—Among 136 total patients (96 CD and 40 UC), 76 (56%) demonstrated clinical response or remission at week 54. In univariate analysis, for patients with CD concomitant initiation of immunomodulator therapy (2.71, 95% CI 1.11 – 6.57), the addition of an immunomodulator (OR 11.49, 3.16 – 41.75) and CRP <3 (4.92, 95% CI 1.99 – 12.15) were associated with increased odds of clinical response or remission at week 54. For UC patients hospitalization after VDZ induction was associated with decreased odds of response or remission at week 54 (OR 0.22, 95%CI 0.05–0.88). On multivariate analysis in CD, addition of an immunomodulator (OR 8.33, 95% CI 2.15–32.26) remained significant predictors of clinical response or remission at week 54.

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Conclusions—Among a multicenter cohort of patients with IBD demonstrating primary response to VDZ, the addition of combination therapy with an immunomodulator is a significant predictor of clinical response or remission at week 54 in patients with CD.

Keywords

Vedolizumab; Crohn's disease; ulcerative colitis; combination therapy; immunomodulator; inflammatory bowel disease

Introduction

Although the introduction of anti-tumor necrosis factor α (anti-TNF) therapy has significantly changed the approach to management of patients with Inflammatory Bowel Disease (IBD), up to 35% of patients with Ulcerative Colitis (UC) and 30% of patients with Crohn's Disease (CD) can demonstrate primary non-response to these therapies.[1] Additionally, over 60% of patients treated with anti-TNF therapy will not maintain remission at 1 year after anti-TNF initiation.[2–5] The low rate of long term remission is further complicated by the lower rates of response to second or third anti-TNF therapies among patients who lose response to their first anti-TNF. [6]

Vedolizumab (VDZ) is a gut selective α4β7 integrin antibody that blocks leukocyte trafficking to the gut mucosa. Unlike an older integrin inhibitor Natalizumab that was associated with reactivation of JC virus and development of progressive multifocal leukoencephalopathy, VDZ binds specifically to the α4β7 integrin, and neither binds to or inhibits the function of the α4β1 or αEβ7 integrins. [7] As a gut selective α4β7 integrin antibody, VDZ has demonstrated efficacy in inducing and maintaining remission among patients with CD[8] and UC, [9] without association with any cases of progressive multifocal leukoencephalopathy in a large pooled analysis of six clinical studies evaluating the safety of VDZ. [10]

While the long-term efficacy of VDZ in the treatment of patients with UC and CD has previously been demonstrated in the GEMINI studies, [8,9] patients enrolled in clinical trials may not be wholly representative of those encountered in clinical practice. Further information regarding the long term efficacy of VDZ is of critical importance, as it could significantly impact positioning of biologic therapy among patients with both UC and CD. Our primary aim was to identify specific clinical predictors of long term clinical response and remission among a large multicenter cohort of patients with IBD treated with VDZ. Additionally, we sought to evaluate the efficacy and durability of VDZ, as determined by clinical response and clinical remission at week 54.

Methods

This study included adult patients from 2 major academic hospitals in Boston, MA: Brigham and Women's Hospital (BWH) and Massachusetts General Hospital (MGH). Following demonstration of clinical response or remission after a 14 week induction period, VDZ was administered intravenously at every 8 weeks thereafter at a dose of 300 mg. Outcomes were

assessed at week 54, at which point patients had received 5 maintenance infusions after induction which should be adequate to assess for response.

Inclusion Criteria

All patients ≥ 18 years of age, who successfully completed induction therapy with VDZ and were receiving maintenance therapy for treatment of CD or UC were considered for inclusion in this study.

Exclusion Criteria

Patients <18 years of age were excluded from this study. Any patient that experienced primary non-response to VDZ initiation was not eligible for this study. Primary non-response was defined as no clinical response to VDZ during the initial 14 week induction period. Patients with prior history of surgery resulting in an ileal pouch anal anastomosis or permanent stoma were also excluded. No other exclusion criteria were defined.

Data Collection Protocol

All patients initiated on therapy with VDZ were retrospectively assessed by chart review at weeks 0, 2, 6, 14, and through week 54. In addition, the treating provider was asked to assess the clinical response to VDZ at the end of induction at week 14 and week 54 as no response, clinical response, or clinical remission. Additional data including laboratory tests and other medication usage was recorded from chart review. Data collected from the record of each visit included the Harvey Bradshaw Index (HBI) for CD, [11] the Simple Clinical Colitis Activity Index (SCCAI), [12 serum C-reactive protein (CRP) and changes to medication. Clinical response and clinical remission at week 54 were defined by HBI, SCCAI, and provider evaluation. Clinical response and remission were mutually exclusive.

Variables, Outcomes, and Definitions

The electronic medical record was reviewed to confirm the diagnosis of IBD, as well as demographic and disease related variables including age, gender, IBD subtype, surgical history, disease duration and distribution, prior biologic therapies, concomitant immunosuppression, and smoking history. Medication additions that occurred during the study period were also noted as were dose adjustments.

The primary clinical outcome was defined as clinical response or clinical remission at week 54. Clinical response was defined as either a decrease in HBI ≥ 3 or SCCAI ≥ 3 from baseline or by physician assessment of clinical response. Clinical remission was defined as HBI ≤ 4 or SCCAI ≤ 2 or by physician assessment of clinical remission.

Statistical Analysis

Continuous variables are reported as means with corresponding standard deviations and are compared using the student t test. Categorical variables are reported as proportions with corresponding percentages and are compared using the Chi Square Test and Fisher Exact Test as appropriate. Univariate analysis was performed to identify predictors of clinical response and remission at week 54. Multivariable logistic regression analysis was then utilized to control for potential confounders. A prior selection of variables felt to be related

to response or remission was used for the creation of the final multivariable logistic regression model. Odds Ratios (OR) and 95% Confidence Intervals (CI) are presented. In the univariate and multivariable analyses, CD and UC were analyzed separately, given concerns of potential differential results in the two subtypes of IBD. All statistical analyses were performed using SAS (version 9.4) statistical software (SAS Institute, Cary, NC, USA).

Results

There were a total of 136 patients in the database who demonstrated initial response or clinical remission to VDZ at week 14 and were thus eligible for inclusion in this study. Among these 136 patients, 73% of the patients with IBD who were initial responders to induction therapy with VDZ remained on therapy with VDZ at week 54. When evaluating only patients with UC, 88% remained on therapy with VDZ at week 54 while 67% of patients with CD remained on therapy at week 54 ($p = 0.019$). Among all patients with IBD, 56% demonstrated clinical response or remission at week 54. When comparing clinical response or remission among patients with UC to patients with CD, there was no significant difference (65% vs 52%, $p = 0.188$). A comparison of baseline demographic and clinical characteristics between patients demonstrating clinical response or remission at week 54 to those with no response or remission is seen in Table 1.

In the univariate analysis of patients with CD, concomitant initiation of immunomodulator therapy (2.71, 95% CI 1.11 – 6.57), the addition of an immunomodulator after induction (OR 11.49, 95% CI 3.16 – 41.75), and a CRP <3mg/L at week 54 (OR 4.92, 95% CI 1.99–12.15) were associated with a statistically significant increased odds of clinical response or remission at week 54, while hospitalization after induction (OR 0.41, 95% CI 0.18 – 0.92) was associated with decreased odds of clinical response or remission (Table 2). Of note, a CRP < 3 was considered normal at both institutions. Age, male sex, CRP at week 14, the presence of perianal disease, smoking status, and surgery prior to initiation of VDZ were not significant predictors of clinical response or remission at week 54 in our population of patients with CD (Table 2). Among patients with UC, only the need for hospitalization after induction with VDZ was significantly associated with a decrease in the odds of clinical response or remission at week 54 (OR 0.22, 95% CI 0.05 – 0.88, Table 2).

Multivariate logistic regression analysis was performed assessing the outcome of clinical response or remission at week 54. In a multivariate analysis of patients with CD, the addition of an immumodulator after induction remained a significant predictor of clinical response or remission at week 54 (OR 8.33, 95% CI 2.15 – 32.26, Table 3). In a multivariate analysis of patients with UC, there were no significant predictors of clinical response or remission, although the need for hospitalization while on vedolizumab therapy demonstrated a trend towards decreased odds of response or remission (OR 0.28, 95% CI 0.06 – 1.21, Table 3).

Among those patients who failed VDZ between weeks 14 and 54, VDZ was discontinued due to a flare of disease requiring a change in therapy in 36% of patients. Twenty-eight percent of patients discontinued VDZ due to worsening disease that required IBD related surgery while 11% of patients stopped VDZ therapy due to an adverse drug reaction. The drug reactions experienced were a type III hypersensitivity reaction consisting of back pain

and shortness of breath, butterfly rash, joint pain, and asthma. After discontinuation of VDZ, 22% of patients were started on an anti-TNF agent, 11% were initiated on ustekinumab, and 8% were initiated on steroid therapy. Of note, 40% of patients experienced a hospitalization after the induction period. However, 63% of patients who were hospitalized remained on VDZ at one year and therefore hospitalization itself was not considered VDZ failure.

It is important to note that of the 99 patients who remained on VDZ at week 54 only 76 (77%) were found to have clinical response or be in clinical remission at week 54. Twenty-three patients remained on VDZ despite not meeting the definitions for response or remission. The reasons cited by both physicians and patients included: fear of surgery, partial response and lack of other options for therapy.

Discussion

Among 136 patients with IBD, we were able to identify several clinically meaningful predictors of clinical response or remission including the addition of immunomodulator therapy and the lack of need for hospitalization after VDZ initiation. Among a population of patients who demonstrated an initial response to VDZ at week 14, a significant portion of patients with both UC (65%) and CD (52%) demonstrated continued clinical response or remission at week 54.

The benefits of long term clinical response and remission among patients with UC and CD treated with VDZ were originally demonstrated in the GEMINI-1[9] and GEMINI-2[8] trials respectively. While a prior “real-world” study from our two academic medical centers have demonstrated that VDZ was both effective and safe as an induction therapy for patients with both CD and UC, [13] further evidence of the longer duration of efficacy of VDZ in this population is an important finding. We chose to evaluate the end point of clinical response or remission at week 54, at which point patients had received 5 maintenance infusions after induction which should be adequate to assess for response.

In the GEMINI-1 study, among patients treated with every 8 week dosing of VDZ, age ≥ 35 years, treatment with prior anti-TNF or immunosuppressive failure, male sex, and a baseline fecal calprotectin of > 500 μg/g were associated with some of the most significant differences in clinical remission at 52 weeks when compared to placebo. [9] In the GEMINI-2 study, among patients treated with every 8 week dosing of VDZ, male gender, baseline fecal calprotectin of > 500 μg/g, ileocolonic disease location, and prior anti-TNF failure were among the factors associated with significant differences in clinical remission at 52 weeks when compared to placebo. [8] In the univariate analysis of our population, there was no significant difference in clinical response or remission among patients with CD or UC who had received prior treatment with more than 1 biologic. Similarly, there was no significant difference when comparing male or female sex. Among patients with CD however, a lower CRP was associated with a significant increase in odds of clinical response or remission at week 54.

In a population pharmacokinetic and pharmacodynamic analysis, use of concomitant immunosuppression was not associated with any clinically meaningful impact. [16] In a

separate real-world consortium of academic medical centers where 54% of patients were treated with concomitant immunomodulators or concomitant immunomodulators and steroids, no differences were noted when comparing clinical response or clinical remission among those patients treated with concomitant immunosuppression and those treated with VDZ alone. [17] When analyzed in phase III trials, concomitant use of immunosuppressive therapy with VDZ in patients with both CD and UC was associated with a reduction in immunogenicity, though there was no overall effect on efficacy of VDZ at 52 weeks. [8,9,14,15] In our evaluation of patients with CD, the use of combination immunosuppressive therapies was associated with a significant benefit in the univariate analyses, including both concomitant immunomodulator therapy at induction and when those agents were added after the induction period. Given the improved odds of clinical response or remission at week 54 among CD patients who had an immunomodulator added after induction when analyzed in multivariate analysis, the addition of immunomodulator therapy may represent a salvage therapy for patients demonstrating less than adequate response after induction with VDZ or may offer a pharmacokinetic benefit similar to that seen with other biologic agents such as anti-TNF therapies. [18]

Although the need for hospitalization is a predictor that may not be modifiable, recognition of the need for hospitalization after the induction period as a predictor of worse outcomes at week 54 is potentially clinically meaningful. A majority of the patients hospitalized remained on VDZ therapy, and thus the need for inpatient admission alone should not be viewed as a failure of VDZ. Patients requiring hospitalization after induction may represent a population where therapy modification or closer monitoring may be of benefit.

Overall, the rates of clinical response or remission were relatively high in this cohort, which is also noteworthy given the concern of a potential bias given the presumed burden of illness associated with patients being seen in two tertiary care referral centers. A considerable proportion of patients with both UC (26%) and CD (22%) remained on VDZ therapy at week 54, despite no evidence of clinical response or remission. Given that both the response and failure populations documented prior therapy with a mean of approximately 2 biologic therapies, this may indicate a hesitancy to switch therapies given prior failures. Particularly among patients with UC, the continued therapy with VDZ could represent an attempt to avoid colectomy, although this area likely needs further exploration.

Our study has multiple limitations that should be outlined. The most significant limitation is that our study was conducted with retrospective data collection. We collected data from two different centers, and although we would expect the patient populations to be similar given the geographic similarities and similarities between medical centers, heterogeneity remains in the collection of data and potentially in the populations of the two centers. Additionally the smaller population of patients with UC may partially explain the inability to identify significant predictors of clinical response and/or remission at 54 weeks.

It has previously been established that many patients with IBD would not be eligible for clinical trials assessing biologic therapies. [20] For this reason, we feel that a real-world assessment of both the efficacy of VDZ as a maintenance therapy in the treatment of patients with IBD, as well as an evaluation of the predictors of response and remission to VDZ at

week 54 is important. The appropriate positioning of biologics in the therapeutic armamentarium of patients with UC and CD and choices between individual biologic agents remains a topic of considerable discussion. [21–24] Earlier introduction of VDZ in the treatment of some patients may be appropriate, however strategies for the appropriate selection of these patients have not been identified to this point.

In a population of patients with IBD that have previously been refractory to prior therapy including anti-TNF agents, VDZ is an effective maintenance therapy with sustained response at week 54. Among patients with CD that demonstrated clinical response or remission to VDZ during the 14 week induction period, the addition of immunomodulator therapy is a significant predictor of clinical response or remission at week 54. These findings may help in guiding clinical practice, particularly regarding the use of combination therapy with a thiopurine or MTX among patients initiated on VDZ.

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Table 1

Baseline Characteristics of Patients demonstrating response or remission with Vedolizumab at week 54 compared to those patients with no response or remission

	Response or Remission at One Year	No Response or Remission at One Year	p-value
Crohn's Disease	n=50	n=46	
Age (years)	42.0	36.9	0.079
Male sex	22 (44%)	21 (46%)	1.00
Duration of disease (years)	17.4 (10.3)	15.6 (11.2)	0.406
Current smoker	5 (10%)	5 (11%)	0.625
HBI score at week 14	5.1 (3.8)	8.3 (5.0)	0.160
HBI score at 1 year	4.3 (3.7)	9.4 (3.6)	0.10
CRP mg/L at week 14	11.3 (14.2)	19.6 (20.7)	0.031
CRP mg/L at week 54	11.6 (14.1)	15.3 (18.5)	0.505
Disease location			0.735
Small bowel disease	5 (10%)	2 (4%)	
Colonic disease	10 (20%)	11 (24%)	
Small bowel and colonic Disease	32 (64%)	28 (61%)	
Prior biologic therapy			
Number of prior biologics	2.4 (1.4)	2.8 (1.1)	0.082
Prior Natalizumab	9 (18%)	12 (26%)	0.459
Prior surgery for Crohn's disease	33 (66%)	30 (65%)	1.00
Perianal Disease	25 (50%)	27 (59%)	0.419
Concomitant Thiopurine or Methotrexate at Induction	20 (40%)	9 (20%)	0.045
Methotrexate or Thiopurine added after Induction	20 (40%)	3 (7%)	<0.001
Ulcerative colitis	n=26	n=14	
Age (years)	38.5 (11.6)	42.8 (14.05)	0.326
Male sex	14 (54%)	7 (50%)	1.00
Duration of disease (years)	9.3 (6.3)	8.3 (4.8)	0.618
Current smoker	2 (8%)	0	0.533
SCCAI score at week 14	2.6 (2.9)	4.0 (4.6)	0.123
SCCAI score at week 54	3.09 (2.43)	4.85 (1.77)	0.117
CRP mg/L at week 54	6.76 (13.1)	4.20 (6.0)	0.416
CRP mg/L at week 54	5.18 (6.2)	4.64 (3.6)	0.818
Disease location			0.062
Rectum and sigmoid only	5 (19%)	1 (7%)	
Left colon	4 (15%)	7 (50%)	
Pancolitis	15 (58%)	5 (36%)	
Number of prior biologics	1.81 (0.749)	2.00 (1.04)	0.504
Concomitant Thiopurine or Methotrexate at Induction	10 (38%)	5 (36%)	1.00

	Response or Remission at One Year	No Response or Remission at One Year	p-value
Methotrexate or Thiopurine added after Induction	6 (24%)	6 (33%)	0.280

Continuous variables presented as mean (Standard Deviation)

Categorical variables presented as raw number (%)

Table 2

Predictors of Clinical Response or Remission to treatment with Vedolizumab at week 54, univariate analysis

	UC		CD	
	Odds Ratio	**95% CI**	**Odds Ratio**	**95% CI**
Age – years	.96	0.92–1.01	1.03	0.99–1.06
Male sex - no (%)	1.22	0.35 – 4.27	1.09	0.50–2.38
Duration of disease – years	1.03	0.92–1.14	1.02	0.97–1.05
Perianal Disease			0.72	0.33–1.58
Prior surgery	1.62	0.15–17.1	1.06	0.46–2.41
Current smoker	>999	<0.001–>999	1.32	0.43–4.09
More than 1 Prior biologic	1.24	0.35–4.38	0.62	0.22–1.72
Prior Natalizumab			0.66	0.25–1.75
CRP <3 mg/L at week 14	1.40	0.63–3.12	0.71	0.20–2.53
CRP < 3mg/L at week 54	2.09	0.55–7.98	**4.92**	**1.99–12.15**
Concomitant Thiopurine or Methotrexate at Induction	1.05	0.28–3.93	**2.71**	**1.11–6.57**
Methotrexate or Thiopurine added after Induction	0.39	0.10 – 1.54	**11.49**	**3.16–41.75**
Hospitalization after VDZ induction	**0.22**	**0.05–0.88**	**0.41**	**0.18–0.92**
Concomitant Thiopurine at Induction	1.27	0.27–5.84	**3.32**	**1.09–10.05**
Concomitant Methotrexate at Induction	0.75	0.11–5.06	1.40	0.41–4.74
Methotrexate added after Induction	0.27	0.04–1.84	**6.46**	**1.73–24.11**
Thiopurine added after Induction	0.64	0.12–3.35	>999	<0.001–>999

Table 3

Odds of Demonstrating Clinical Response or Remission on Vedolizumab at Week 54 among Patients with Crohn's Disease or Ulcerative Colitis, Multivariable Analysis[a]

	OR	95% CI
Crohn's Disease		
Age	1.03	0.99–1.06
Male	0.85	0.34–2.13
Thiopurine or Methotrexate added after Induction	**8.33**	**2.15–32.26**
Concomitant thiopurine or methotrexate at Induction	1.89	0.66–5.38
Hospitalization while on Vedolizumab	0.57	0.23–1.42
Ulcerative Colitis		
Age	0.97	0.92–1.02
Male	1.05	0.26–4.16
Thiopurine or Methotrexate added after Induction	0.43	0.09–2.00
Concomitant thiopurine or methotrexate at Induction	1.27	0.27–5.96
Hospitalization while on Vedolizumab	0.28	0.06–1.21

[a]All variables included in final multivariable analysis are demonstrated above