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ORIGINAL ARTICLE

Weight and BMI Patterns in a Biologicals-Treated IBD Cohort

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Abstract

Background Biologic therapies are effective at inducing and maintaining remission in people with inflammatory bowel disease (IBD). Previous studies have associated TNF-a inhibitors with weight gain, however, it is unclear if this is a class-specific effect or a manifestation of good disease control. To clarify this issue, a retrospective study was undertaken to examine weight changes over time during therapy with different biologic agents.

Methods Adult patients with IBD who received any biological therapy for at least 12 months, between 2008 and 2020, were identified at two specialised IBD services. Demographic, disease, and therapy-related data were examined.
Weight change and patterns thereof were examined for each specific therapy and relationships amongst weight outcomes and various predictive factors explored.

Results Of 294 patients (156 females), 165 received Infliximab (IFX), 68 Adalimumab (ADA), 36 Vedolizumab (VDZ) and 25 Ustekinumab (UST). There was a statistically significant weight gain over time in the IFX and VDZ groups and more weight gain in the IFX vs ADA and VDZ vs ADA at most time points.
Three weight trajectories were identified: around 95% of patients had small weight loss or a modest weight gain but 5% of patients, most of whom were on IFX had marked weight gain (24.3 kg). Having a baseline high BMI, being female, having an initiation CRP ≤ 5 or albumin > 35 reduced the odds of major weight gain.

Conclusion Weight gain in biologic treated IBD patients appears to be associated with clinical factors (male gender, high CRP, low albumin) and therapy-specific factors.

Introduction

Inflammatory bowel disease (IBD) is a group of chronic immune-mediated intestinal disorders predominantly recognised as Crohn's Disease (CD) and ulcerative colitis (UC). The pathophysiology of IBD involves complex genetic, environmental, microbial, and immune-related factors. Inadequately treated IBD can lead to serious potentially preventable complications [1. The current standard medical treatment approaches involve single agent or combination therapy with both targeted and older untargeted therapies. Commonly used targeted biological therapies include monoclonal antibodies such as anti-tumour necrosis factor-alpha (TNF-α), anti-α4β7, and anti-Interleukin-12/Interleukin-23 (IL12/23) antibodies.

TNF-α inhibitors such as Infliximab (IFX) and Adalimumab (ADA), are monoclonal antibodies that neutralize pro-inflammatory cytokines. There is an association suggested between this class (particularly IFX) and weight gain [2–5], with multiple proposed mechanisms such as improved disease activity and subsequent increase in muscle mass and reduced visceral sensitivity and hence fullness sensation [2, 6–8].However, it remains generally unclear if this is a true causal relationship or whether weight gain is simply a "desired" consequence of good disease control.

Assessment of this phenomenon can be challenging, as patients with IBD treated with IFX usually have a high baseline inflammatory burden and poor nutritional status, meaning that weight gain might simply be related to the reversal

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of gut inflammation and improved nutrition. However, it is important to note that many of the published studies [4, 5], included patients with rheumatological conditions, thus weight gain in anti-TNF-α treated patients may not be exclusively the result of improvement in gut function.

One of the largest analyses [2] of weight gain in IBD patients treated with anti-TNF-α, reported weight changes in kilograms, without including Body Mass Index (BMI). Despite its well-described limitations notably as a determinant of body fat mass and distribution and of nutritional status [9], BMI has valid national reference data and a reported relationship with levels of adiposity, hence, measuring BMIs throughout the treatment period, is an acceptable primary tool [10] to indicate whether weight change goes in an appropriate or inappropriate direction.

Obesity is associated with an increased risk of complications in IBD and loss of response to therapy [11]. Contrary to conventional belief, up to one-third of patients with IBD are obese, which parallels rates in the general population [11]. More interestingly, the increasing incidence of CD over the last two decades parallels the obesity epidemic [12]. It has been postulated that visceral adiposity might increase the risk of developing CD, progressing to penetrating disease, and requiring surgery [13, 14]; hence, it is important for IBD clinicians to consider obesity treatment as an important element of their patient care.

We, therefore, conducted this study to:

- Compare weight and BMI changes from baseline during therapy with different biologic agents.
- Examine different weight patterns over time and assess for possible clinical characteristics associating with each subgroup.
- Determine whether weight change reflects the extent of suppression of systemic indices of inflammation (CRP) and remission state.

Methods

Patient Cohort and Characteristics

Adult patients who received any biologic therapy for IBD for at least 12 months between 2008 and 2020, were identified from prospectively maintained medical records at two hospital-based IBD centers.

Extracted data included: demographics; weight and BMI at baseline, 6, 12, 24, and 48 months; IBD type and phenotype; IBD treatment site (Royal Adelaide Hospital/Logan Hospital); disease duration, baseline endoscopy, follow up endoscopy (if available), hemoglobin (Hb), C-reactive protein (CRP), albumin (alb); monotherapy or combination therapy; initial steroid therapy and biologic dosing schedule.

Treatment Schedules and Assessment Times

Common causes for primary and secondary non-response include inadequate dosing that can be identified through assessment of drug and anti-drug antibody levels. Both dose intensification from 5 mg/kg [15] to 7.5 mg/kg or 10 mg/kg 8 weekly and increased infusion frequency to 4 weekly or 6 weekly are widely accepted strategies to optimize IFX levels [15].

Standard ADA maintenance therapy is given in 40 mg subcutaneous (SC) injections every other week [16]; if escalation is needed, the dose can be increased to 60 or 80 mg or the frequency can be increased to weekly.

The recommended standard maintenance dose of VDZ is 300 mg IV every 8 weeks that can be escalated to 300 mg 6 or 4 weekly in case of inadequate response [17, 18].

For Ustekinumab, after weight-based IV induction therapy, maintenance is usually with 90 mg SC injections every 8 weeks, [19] frequency can be increased to 4 or 6 weekly if escalation is needed.

We note that in Australia, clinical remission is a prerequisite for ongoing pharmaceutical benefit scheme (PBS) subsidy of any of the biologic therapies.

Data and Sources

Data was collected from prospectively made entries into hospitals' electronic medical records and specific IBD databases. Endoscopy data prior to initiation of biological therapy was collected from "Provation" endoscopy electronic record system. Due to the lack of standardized IBD reporting among endoscopists and for the simplicity of analysis, endoscopic disease activity for both CD and UC was recorded as in remission, mild, moderate, or severe. For CD, when the Simple endoscopic score for Crohn's Disease (SES-CD) was reported by the endoscopist [20], the following decoding applied: 0–2 (remission), 3–6 (mild disease), 7–15 (moderate disease), > 15 severe disease; otherwise, the description reported by the endoscopist was recorded. For UC, when the Mayo score was reported by the endoscopist [21], the following decoding applied: 0 (remission), 1 (mild disease), 2 (moderate disease), 3 (severe disease); otherwise, we recorded the description used by the endoscopist.

Patients with any missing data but follow up endoscopy were excluded from the study.

Statistical Analysis

Weight change from baseline was examined with a linear mixed-effects model, including the interaction of treatment group and time period. Covariates of baseline weight,

IBD treatment site, Hb, alb, and CRP were included in an adjusted model. To control for repeated measurements over time, a compound symmetry covariance structure was used. The "IBD treatment site" covariate was included as a fixed effect to adjust for clustering on hospital.

For the subset of patients who had a follow-up colonoscopy, a linear mixed-effect model, adjusted for repeated measures, was applied to examine weight change from baseline, predictors being presence or absence of endoscopic remission, treatment group, time period, baseline weight, and IBD treatment site. Assumptions of a linear model were found to be upheld by inspection of scatter plots and histograms of residuals and predicted values (Supp Fig. 1).

To describe the course of weight change over the first 48 months of biologic therapy, a latent class analysis was applied. The best-fitting model was identified on the basis of the Bayesian Information Criterion (BIC) and its interpretability. Patients were assigned to trajectory groups using the maximum probability rule. Univariate and multivariable ordinal logistic regressions were used to assess factors associated with membership of each group.

An initial multivariable logistic model was performed by including all predictors with P value < 0.2 on univariate regression. Backward elimination was performed, removing one covariate with the highest P-value, one model at a time until all covariates had a P value < 0.2 [22].

The statistical software used was SAS 9.4 (SAS Institute Inc., Cary, NC, USA).

Ethics Approval

The Central Adelaide local health Network (CALHN) Research services (South Australia) and Metro South Health (MSH) Manager, Research Integrity and compliance (Queensland) reviewed the project and confirmed that the project meets criteria for audit and none of the triggers for consideration of ethical review are present and therefore formal ethics approval was not required. Publication approval was provided by the CALHN and MSH research committees.

Results

General Cohort Description

A total of 807 patients on biological therapy were initially screened for inclusion in the study, 513 patients were

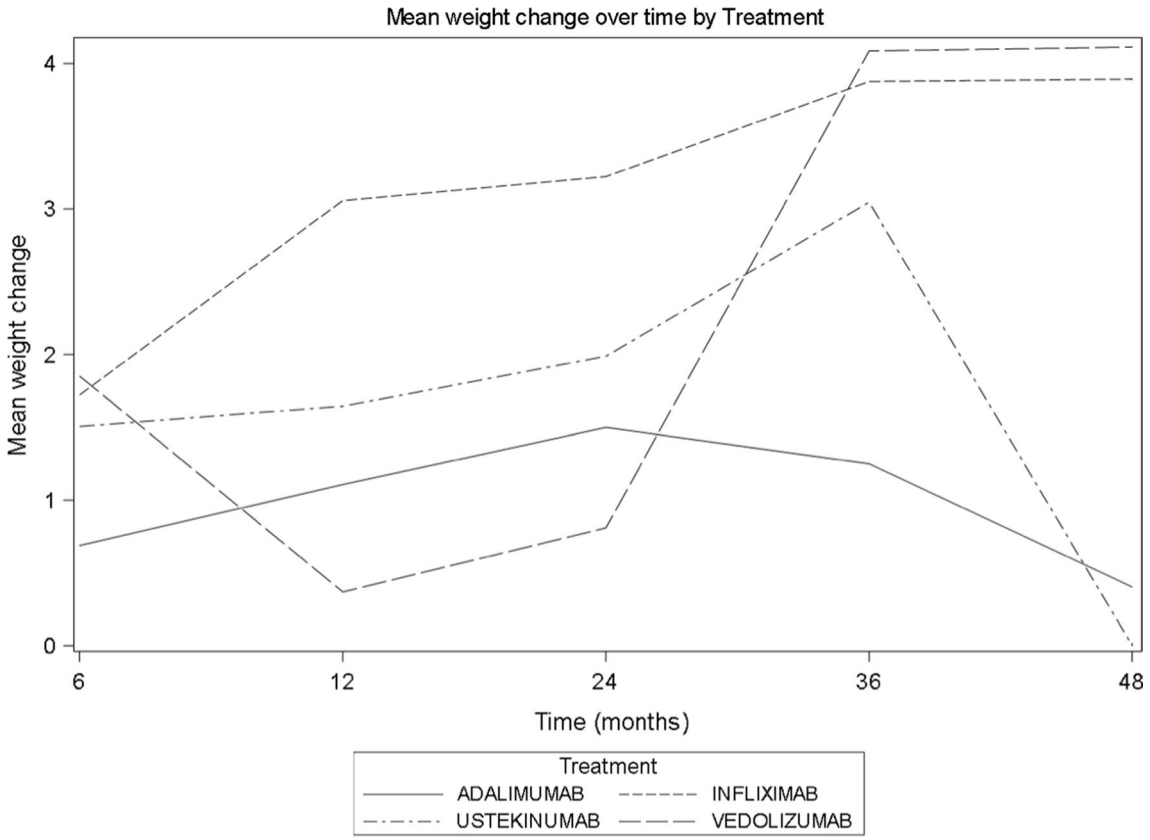

Fig. 1 Mean weight change over a period of 48 months in the 4 different biological groups

excluded due to short duration of therapy and any missing data (height, weight at a certain time point, inflammatory markers), leaving 294 patients for analysis (Supp Fig. 2). Follow-up colonoscopy data were available for 116 patients within 18 months of induction therapy.

Of the 294 patients included, 165 were on IFX, 68 on ADA, 36 on VDZ, and 25 on UST. In total, 179 patients remained on biological therapy at 48 months (Supp Fig. 3).

Patient characteristics are summarized in Table 1.

There was a trend for weight gain in the whole cohort with mean weight of 77.4 kg (SD19.10) at induction and 80.0 kg (18.72) at 48 months ($P = 0.14$).

The mean BMI at induction was 26.58 kg/m^2(SD 6.01) classifying our cohort as overweight on average and was 27.08 kg/m^2 (SD 5.37) at 48 months($P = 0.36$).

At induction of therapy; 27.5% of patients were obese (BMI ≥ 30 kg/m^2), 23.5% were overweight (BMI ≥ 25–29.9 kg/m^2), 38.1% had a normal weight (BMI ≥ 18.5–24.9 kg/m^2) and 10.9% of patients were underweight (BMI < 18.5 kg/m^2).

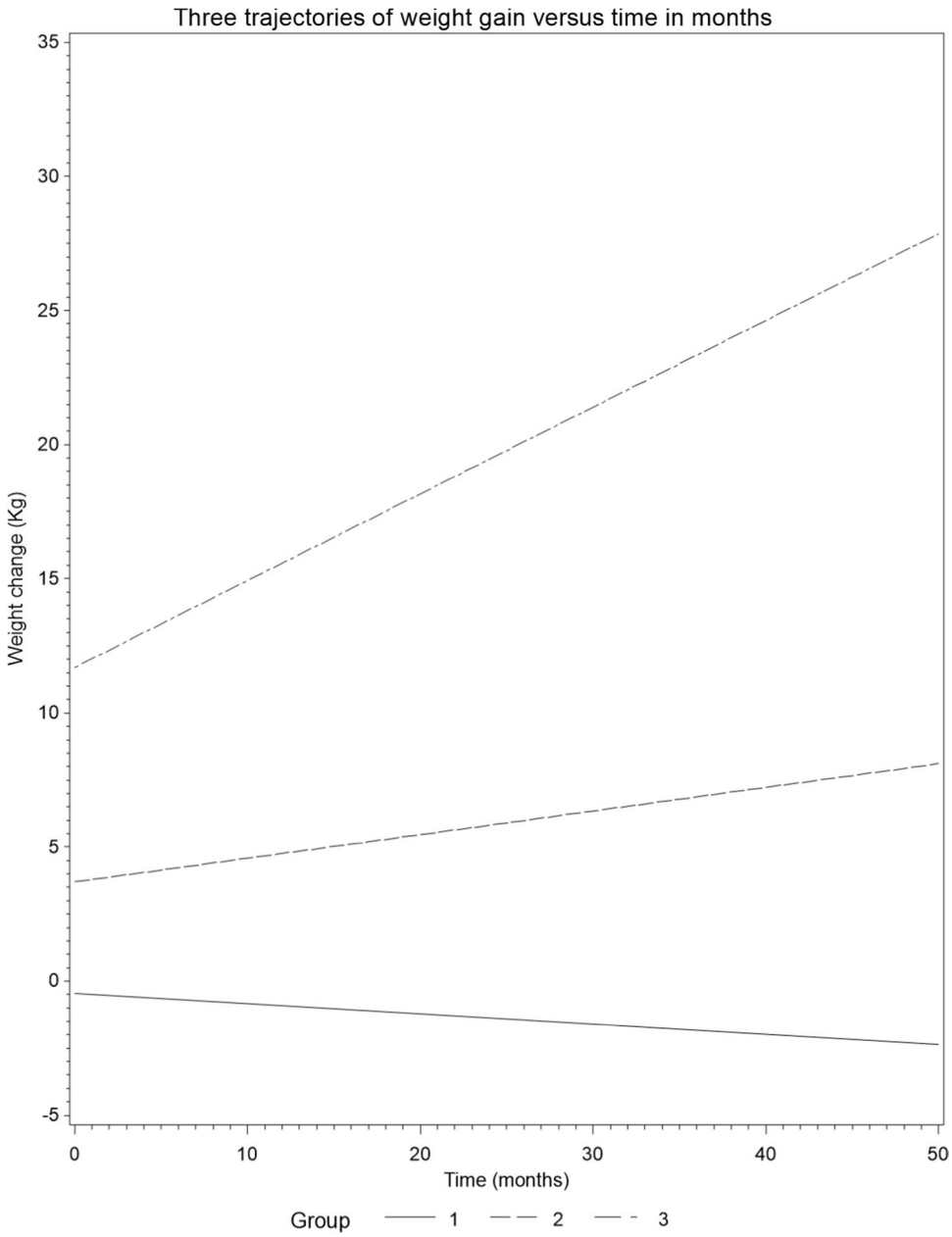

Fig. 2 Three different trajectories of weight gain over time in our cohort of patient

Table 1 Patient characteristics at baseline

Characteristic	Mean (SD)	Median (IQR) (IQR
Age	42.76 (14.0)	41.00 (31, 53)
Disease duration	13.4 (9.6)	11 (7, 18)
Characteristic	Frequency	Percentage
Gender		
Female	156	53.1
Male	138	46.9
IBD Type		
UC	22	7.4
CD	272	92.5
L1/L3	194	
L2	78	
IBD Treatment Site		
RAH	210	71.4
LGH	84	28.5
Steroids at baseline		
Yes	56	20.3
No	221	79.7
Monotherapy	149	50.7
Combination therapy	145	49.3
CRP		
≤ 5	156	53.1
> 5	138	46.9
Albumin		
≤ 35	102	34.7
> 35	192	65.3
Hb		
< 120	74	25.2
≥ 120	220	74.8

Weight and BMI Patterns in Different Biological Groups

Adjusting for baseline weight, treatment site, CRP, Alb, and Hb and controlling for repeated measurements over time (interaction *P* value = 0.026), the weight pattern differed over time between the 4 biological classes included in the study (Fig. 1).

The BMI change over time is significantly different between treatment groups only when we don't adjust for markers of inflammation.

Applying the same adjustments, there was a statistically significant weight gain in the IFX group and the VDZ subgroups and a significant BMI increment in the IFX group.

Over a period of 48 months, the mean weight and BMI gain in the IFX treated groups were respectively 2.4 kg (95% CI: 1.4 kg, 3.4 kg) and 0.77 kg/ m^2 (95% CI: 0.43 kg/ m^2, 1.1 kg/ m^2).

There was found to be a significantly greater weight and BMI gains over time in the IFX group compared with the ADA group (mean difference = 0.06, 95% CI: 0.002, 0.11, *P* value = 0.0437). (Table 2).

Weight Patterns and Associated Clinical Characteristics

An inverse association between baseline weight and weight change was found. This held true whilst adjusting for the interaction between treatment group and time period, Hb, CRP, albumin, and IBD unit, and controlling for repeated measurements over time (global P value = 0.0164). For every 10 kg increase in baseline weight, the weight change from baseline decreases by half a kilogram (estimate = – 0.05, 95% CI: – 0.09, – 0.01). Thus heavier patients, on average, gain less weight than lighter patients. (Table 2).

Table 2 Linear mixed-effects model of Weight change from baseline versus interaction of time period and treatment (continuous), and adjusted for baseline weight, hospital, Hb, albumin, and CRP, controlling for repeated measurements over time

Outcome	Predictor/Interaction	Comparison	Mean difference (95% CI)	Comparison *P* value	Interaction/Global *P* value
Weight change from baseline	Time period*Treatment	Slope ADALIMUMAB	– 0.01 (– 0.06, 0.04)	0.7786	0.0264
		Slope INFLIXIMAB	0.05 (0.03, 0.07)	< .0001	
		Slope USTEKINUMAB	0.05 (– 0.03, 0.13)	0.1960	
		Slope VEDOLIZUMAB	0.06 (0.01, 0.11)	0.0147	
		Slope INFLIXIMAB vs slope ADALIMUMAB	0.06 (0.002, 0.11)	0.0437	
		Slope INFLIXIMAB vs slope USTEKINUMAB	– 0.001 (– 0.08, 0.08)	0.9651	
		Slope INFLIXIMAB vs slope VEDOLIZUMAB	– 0.01 (– 0.06, 0.04)	0.6935	
		Slope ADALIMUMAB vs slope USTEKINUMAB	– 0.058 (– 0.15, 0.03)	0.2174	
		Slope ADALIMUMAB vs slope VEDOLIZUMAB	– 0.07 (-0.14, 0.003)	0.0611	
		Slope USTEKINUMAB vs slope VEDOLIZUMAB	-0.01 (– 0.10, 0.08)	0.8522	
	Hospital	LOGAN vs RAH	1.04 (– 0.76, 2.82)		0.2550
	Baseline weight		– 0.05 (– 0.09, -0.01)		0.0164
	CRP (binary)	< = 5 vs > 5	– 1.10 (– 2.71. 0.50)		0.1777
	Albumin (binary)	> = 36 vs < 36	– 1.67 (– 3.41, 0.07)		0.0693
	Hb (binary)	> = 120 vs < 120	– 0.50 – -2.52, 1.51)		0.5625

In the subgroup with follow-up colonoscopy data, endoscopic remission did not appear to have a statistically significant impact on weight change, adjusting for time period, treatment group, baseline weight, and hospital, and controlling for repeated measurements over time (global *P* value = 0.755).

A latent class analysis was performed aiming to assign each of the 294 patients to one of three weight trajectory groups: weight loss (57.4%), modest weight gain (37.8%), and marked weight gain (4.8%) (Fig. 2).

Out of the 14 patients with marked weight gain, 11 were on IFX.

The respective mean weight changes from baseline to 48 months per group, were – 2.41 kg (SD ± 4.47), + 7.09 kg (4.64), and + 25.8 kg (5.41).

The respective mean BMIs from baseline to 48 months were: 27.49 (SD 6.27) to 26.53 (SD 5.4); 25.68 (SD 4.95) to 27.65 (SD 5.16) and 21.1 (SD 4.79) to 29.8 (SD 5.16).

There was a statistically significant association between Group affiliation and the following predictors on univariate logistic regression analysis (Table 3):

(i) Gender (global *P* value = 0.0031): Females had a lesser probability of being in the 'marked weight gain' group than males (OR = 0.50, 95% CI: 0.31, 0.79).

(ii) CRP (global *P* value = 0.0187): Patients with a CRP ≤ 5 were 43% less likely to be in the 'marked weight gain' group than patients with a CRP > 5 (OR = 0.57, 95% CI: 0.36, 0.91).

(iii) Albumin (global *P* value = 0.0053): Patients with an albumin > 35 were 50% less likely to be in the 'marked weight gain' group than patients with an albumin ≤ 35(OR = 0.50, 95% CI: 0.31, 0.82)

(iv) Baseline BMI (global *P* value = 0.0001): For every one unit increase in BMI, the odds of being in the 'marked weight gain' group decreased by 8% (OR = 0.92, 95% CI: 0.88, 0.96).

There was no statistically significant association between various weight trajectories and: age (global *P* = 0.13), disease type (CD Vs UC; global *P* = 0.63), disease duration (global *P* = 0.16), steroid therapy at baseline (global *P* = 0.74), dose-escalated therapy (global *P* = 0.95) or combination versus monotherapy (global *P* = 0.80). Nor was small bowel involvement in CD associated with a specific weight trajectory. (L1/L3 vs L2 only; global *P* = 0.33).

In a multivariable model (Table 4) after adjusting for all other variables, a statistically significant inverse association remained between weight trajectory

Table 3 Univariate ordinal logistic regression of weight change Group versus various predictors

Predictor	Comparison	Odds ratio* (95% CI)	Comparison *P* value	Global *P* value
Gender	F vs M	0.50 (0.31, 0.79)		0.0031
Age		0.99 (0.97, 1.00)		0.1329
Disease type	CD vs UC	1.25 (0.51, 3.05)		0.6267
Montreal CD	L2 only vs L1 or L3	0.77 (0.45, 1.30)		0.3257
Endoscopy grading	Mild vs Moderate	1.23 (0.63, 2.40)	0.5364	0.6510
	Mild vs Severe	0.90 (0.46, 1.77)	0.7585	
	Moderate vs Severe	0.73 (0.37, 1.44)	0.3651	
Therapy	Mono vs Combo	1.06 (0.67, 1.68)		0.8020
Steroids	Yes vs No	1.10 (0.62, 1.96)		0.7394
Albumin	> 35 vs < = 35	0.50 (0.31, 0.82)		0.0053
Hb	> = 120vs < 120	0.80 (0.45, 1.40)		0.4286
CRP	< = 5 vs > 5	0.57 (0.36, 0.91)		0.0187
Hospital	LGH vs RAH	0.89 (0.53, 1.48)		0.6476
Treatment	ADA vs IFX	0.63 (0.36, 1.11)	0.1122	0.1675
	ADA vs UST	1.05 (0.41, 2.69)	0.9126	
	ADA vs VEDO	1.23 (0.52, 2.90)	0.6408	
	IFX vs UST	1.67 (0.71, 3.94)	0.2384	
	IFX vs VEDO	1.95 (0.90, 4.21)	0.0895	
	UST vs VEDO	1.16 (0.40, 3.40)	0.7807	
Initial BMI		0.92 (0.88, 0.96)		0.0001
Disease duration		0.98 (0.96, 1.01)		0.1632
Frequency of infusion	STD vs accelerated	1.01 (0.72, 1.43)		0.9551

Table 4 Multivariable ordinal logistic regression of weight change Group versus various predictors

Predictor	Comparison	Adjusted odds ratio* (95% CI)	Global *P* value
Albumin	< = 35 vs > 35	1.88 (1.09, 3.24)	0.0230
CRP	> 5 vs < = 5	1.54 (0.92, 2.57)	0.1026
Disease duration		0.98 (0.96, 1.01)	0.1867
Initial BMI		0.92 (0.88, 0.97)	0.0005
Gender	M vs F	2.04 (1.25, 3.32)	0.0045

group and: Alb (global *P* value = 0.023), initial BMI (global *P* value = 0.0005) and female gender (global *P* value = 0.0045).

Patients with an initial albumin ≤ 35 were 1.9 times more likely to be in the 'major weight gain' group than patients with an albumin > 35 (adjusted OR = 1.9, 95% CI:1.1, 3.2).

For every unit increase in initial BMI, the odds of being in the 'major weight gain' group decrease by 8% (adjusted OR = 0.92, 95% CI: 0.88, 0.97).

Males were twice as likely to be in the 'major weight gain' group than females (adjusted OR = 2.0, 0.95% CI: 1.3, 3.3).

Discussion

Despite an anecdotal trend of weight gain in our cohort of patients, a more detailed observation shows that more than half of our patients were overweight or obese at induction of therapy; subsequently more than half of the cohort lost weight over time.

That might reflect a weight change towards their ideal body weight due to reversal of inflammation and catabolic burden leading to an improved quality of life and physical activity. It is an interesting observation given that a recent prospective cohort study (23)concluded that all biologicals are associated with some degree of weight gain.

In patients who gained weight, a modest weight gain (7.09 kg) occurred in 38% of cases and significant gain (25.8 kg) occurred only in 14 patients (4.8%) of which 11 were on IFX and 2 on VDZ, upgrading the mean BMI of this subgroup from "normal" to significantly "overweight".

Weight gain appears to be a combination of class and disease effects as we found that both active disease at baseline and specific therapies such as IFX and VDZ were associated with greater weight gain than other therapies.

Patient characteristics including male gender, a lower baseline weight, and disease characteristics such as indices of active systemic inflammation (high CRP and Low

albumin) at initiation of therapy were associated with more weight gain.

Those results, notably active disease and patient characteristics are consistent with previous research [2, 3] looking at weight gain in patients with IBD treated with IFX.

In patients with other immune-mediated inflammatory diseases (IMID) such as psoriasis and psoriatic arthritis, anti-TNF-α treatment was associated with an increase in fat and lean mass as demonstrated by Di Renzo et al. and Briot et al. [4, 5]. They suggest that weight gain is induced by a reversal of the systemic TNF- α effect on body weight hemostasis such as increased lipolysis, increased muscle cell catabolism, and general proteolysis. Other studies have suggested that TNF-α blockers might increase visceral adiposity [7], have a central orexigenic effect [6], and suppress inflammatory myopenia [13].

In our study, the magnitude of weight gain achieved in IFX-treated patients appears to be beyond what might be expected with control of inflammation and raises the hypothesis of a class-dominant effect, however, this is difficult to confirm due to the retrospective nature of our work.

Interestingly, patients treated with ADA showed a trend towards weight gain from induction to 24 months but not further. This might be explained by drug-specific characteristics such as gastrointestinal side effects (nausea, vomiting, and abdominal pain), pharmacokinetics, patients-specific factors, or simply by the smaller ADA subgroup size.

Similarly, UST-treated patients showed a trend for weight gain that did not reach significance.

Vedolizumab treated patients acquired significant weight gain. It was noted that the mean age of this subgroup (51, SD13.9) is the highest in the cohort. Being a gut-specific biological, we suggest that the significant weight gain seen in the VDZ treated patients, is due to improved intestinal mucosal health, nutritional status, and decreased protein wasting. Age might be an important factor in this subgroup.

This aligns with the results of Borren et al.'s study [23] showing that all patients with IBD treated with biologicals acquire a degree of weight gain, however, in their study, none of the disease activity parameters showed any statistical association with weight gain and no significant differences between any of the biologic therapies for weight gain was seen.

Disease activity as assessed by endoscopic remission did not seem to affect weight gain. This might reflect a lack of precision in the available data, a smaller size of cohort with follow-up colonoscopy at 18 months, or a potential systemic, rather than gut-specific mechanism of weight gain in patients treated with those drugs. This has been previously suggested for IFX in other studies [2, 6–8].

To date, the largest analysis of weight gain on TNF-α inhibitors for IBD patients is a post-hoc analysis where a total of 1273 patients from ACCENT I, ACCENT II, ACT 1, and SONIC trials were compared to a subgroup of 170 patients on Azathioprine monotherapy from the sonic trial [2]. The study concluded that patients on AZA gained less weight than IFX treated patients and within the latter group, patients with markers of severe disease, CD patients, and male patients were likely to gain more weight.

Our findings are similar; however, we did not have an adequate sample size ratio to examine differences in weight patterns between CD and UC. The authors did not consider CD distribution in their calculations and did not comment on changes in BMI. Additionally, it is potentially more valid to compare patients on a certain biologic therapy to patients on another biologic, as patients who qualify for this treatment are likely to have a more severe disease phenotype.

IBD as most of the immune-mediated inflammatory diseases is a systemic disease associated with cardiovascular and metabolic comorbidities, hence it is important to determine whether weight gain was due to an increase in fat or muscle mass; this can only be done with prospective assessment of body composition, nutritional status, and basal metabolic rate.

Due to the limitations of our study, it is hard to confidently comment on whether where it occurred, weight gain reflects a desirable increase in lean body mass due to reduced systemic inflammation, or an undesirable drug-specific side effect.

The lower baseline weight and high indices of systemic inflammation in the weight gain subgroups suggest it may be the former. Significant weight gain was observed in only 14 patients (< 5% of the whole cohort), 6 of whom were initially underweight which suggests that, unlike steroids, weight gain on biologics is likely to represent a desired drug-related effect.

Another interesting observation in this study is that around half of our biological treated patients with IBD are obese or overweight with only 10% being underweight.

This is likely a reflection of the international "obesity pandemic". "Obesity prophylaxis" with appropriate diet and physical activity should be addressed at every clinical consultation, given the proven IBD and non-IBD-related complications of obesity.

Our study has multiple limitations, particularly its retrospective nature, hence nutrition and calorie intake, physical activity, accurate remission status, and body composition were not well examined. Trough levels of biological at regular treatment intervals would have been helpful to accurately measure the effect of the drug on weight gain.

Conclusion

Just over half of our biological-treated IBD patients lost a small amount of weight during treatment. That said, around 50% of patients were overweight/obese at induction of therapy.

Weight gain, when acquired was associated with therapy-specific factors (IFX and VDZ), patient's clinical characteristics (male gender, low baseline BMI), and disease-specific factors (active systemic inflammation).

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10620-022-07488-7.

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Author's contribution PK: study design, data collection sheets design, coordinating the work between team members, data collection at two sites, data analysis, manuscript writing (first and last drafts). ZT—Data collection at one site. M and M: data collection at one site. SE: statistical analysis and review. M: Draft review and help with study design. A: Multiple study draft reviews and senior study design. All authors approved the final version of the manuscript.

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Declarations

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