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Original Article PEDIATRICS

Pediatric inflammatory bowel diseases: Effects of disease and treatment regimens on growth and puberty

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ABSTRACT

OBJECTIVE: Inflammatory bowel diseases (IBD) are lifelong conditions that exhibit periods of remission and exacerbation. In addition to gastrointestinal manifestations, they can also cause growth retardation and disorders of puberty in children. The objective of this study was to evaluate the effects of the disease and the treatment regimens on growth and puberty in children and adolescents with IBD.

METHODS: A retrospective screening of patients aged 2 to 18 years with a minimum of six months of follow-up due to inflammatory bowel disease (IBD) between January 2016 and April 2022 was conducted. The growth parameters were compared between disease groups, gender groups, disease activity and level of inflammation groups, and treatment regimen groups. The effects of treatment protocols on growth were evaluated by comparing the data before and after treatment, and the pubertal status of patients was evaluated by comparing them with healthy children.

RESULTS: A total of 58 patients were evaluated, comprising 29 individuals with Crohn's Disease (CD) and 29 with ulcerative colitis (UC). The growth and pubertal development of patients at the time of diagnosis did not differ based on gender or the specific disease type. A negative deviation from the target height was observed to be more prevalent in patients with Crohn's disease. Following treatment, patients exhibited a significant improvement in weight and BMI SDS, although no significant change in height SDS was observed. In comparison to healthy Turkish children, the patients exhibited a delayed pubertal progression, despite the normal onset of puberty.

CONCLUSION: Children and adolescents with IBD exhibited no significant adverse effects on linear growth at diagnosis or during the follow-up period, regardless of the primary disease and the treatment protocols. This was likely due to their timely diagnosis and successful treatment. It is important to monitor puberty, as it may progress more slowly or even cease in these patients compared to healthy children.

Keywords: Growth; inflammatory bowel disease; puberty

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Inflammatory bowel diseases (IBD) are chronic, recurrent, and progressive immune-mediated gastrointestinal system diseases of children and adults. Ulcerative colitis (UC) and Crohn's disease (CD) are the two main types, characterized by attacks and periods of remission [1]. Growth retardation and malnutrition are common extraintestinal manifestations, often presenting before diagnosis and persisting during follow-up. Retardation

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in growth and puberty not only indicates disease activity but also reflects the effectiveness of treatment. Growth retardation in CD can vary in severity and may precede diagnosis or occur as the sole presenting symptom [2].

Children often experience rapid weight loss at diagnosis and during active disease phases, with a frequency of 85% in CD and 65% in UC at diagnosis [3]. CD typically causes more significant linear growth impairment compared to UC [4]. The rate of growth retardation is about 10% in UC, while up to 40% in CD, which iseven more in younger patients [5, 6].

Bone maturation and development of puberty may also be delayed due to malnutrition in the long term. The recurrent and active courses of the disease may be responsible for pubertal arrest by suppressing the hypothalamic-pituitary-gonad axis. Secondary amenorrhea is seen frequently in girls following rapid and severe weight loss [7]. The hypothesis was that malnutrition and increased levels of proinflammatory cytokines may impair the onset and progression of puberty during the active phases of the disease [8]. However, evidence regarding puberty disorders in children with IBD remains limited.

As the number of children and adolescents diagnosed with IBD increases, the diagnosis and follow-up of endocrine problems such as growth retardation and puberty disorders are becoming increasingly important. A multidisciplinary approach is essential for the management of long-term problems in children with IBD. The objective of this study was to evaluate the growth and pubertal status of children and adolescents with IBD, including the frequency of growth and pubertal disorders, the clinical features and factors affecting them, and the differences between treatment regimens.

Highlight key points

- Inflammatory bowel diseases can result in growth retardation and disorders of puberty, in addition to gastrointestinal manifestations, in children.
- The use of pharmacological doses of steroids in induction, the total duration of steroids, and the use of biological agents did not result in a significant change in linear growth.
- The results suggest that the management of the inflammatory process may overcome the growth suppression of steroids.
- The pubertal tempo may be slowed in children with inflammatory bowel disease.

MATERIALS AND METHODS

The study group consisted of patients aged 2-18 years who were followed up with a diagnosis of IBD in the Pediatric Gastroenterology Clinic between January 2016 and April 2022. The study was approved by the Umraniye Training and Research Hospital Ethics Committee (date/number: 31.03.2022 / B.10.1TKH.4.34.H.GP.0.01/13) and is in accordance with the Declaration of Helsinki. Data on symptoms, laboratory values, and endoscopic findings at admission were collected from the hospital information system.

The measurements of the patients included in the study were conducted with the subjects wearing indoor clothes and without having eaten for at least eight hours. The height of each subject was measured without shoes, with the heel, hip, and scapula in contact with the measuring board, and with the head and face in a straight position. The measurements were determined using a height meter with a 1-mm sensitivity and a digital scale with a 100-gram sensitivity. Body weight, height, and body mass index SDS values were calculated using the Child Metrics application, created with references for Turkish children [9, 10].

The nutritional status of the patients was evaluated according to body mass index (BMI). Those with BMI SDS values below -3 SDS were classified as severely malnourished, those between -3 and -2 were classified as moderately malnourished, and those between -2 and -1 were classified as mildly malnourished [11].

The target height is the sex-adjusted midparental height, which is calculated by subtracting 13 cm from the father's height and averaging it with the mother's height for females, and by adding 13 cm to the mother's height and averaging it with the father's height for males. The difference between the patients' height SDS and target height SDS (height SDS - target height SDS) at diagnosis and at the last visit were recorded.

Short stature was defined as a height that was two or more standard deviations below the mean for Turkish children of that sex and chronological age. Patients who were below -2 SDS and who had insufficient growth velocity (less than 7 cm per year between the ages of 2–4, and less than 5 cm per year between the ages of 4 and puberty) were defined as those with growth retardation [12].

Puberty was assessed using the Tanner–Marshall method [13, 14]. Stage-1 is classified as prepubertal (no breast development in girls, and testis volumes of less than 4 ml for boys), Stage-2 (breast buds in girls, and tes-

tis volumes of 4-10 ml), Stage-3 (elevated breasts in girls, and testis volumes of 10-15 ml in boys), Stage-4 (areolar mound in girls, and testis volumes of 15-20 ml in boys) were classified as pubertal process, and Stage-5 (adult contours of breasts and mature areolae in girls, and testis volumes of more than 20 ml in boys) was classified as post-pubertal process. Pubertal progression was considered to be slow if there was minimal or no change in the stage of breast, pubic hair, or genital development within six or more months. The comparison of the mean age at pubertal stages of the male and female patients in the study group with the mean age of healthy Turkish children was made using the reference by Semiz et al. [15].

Fecal calprotectin levels of <50 μg/g were considered no inflammation, 5–100 μg/g as moderate inflammation, and >200 μg/g as severe inflammation [16]. PUCAI was used for the evaluation of disease activity for those with UC [17]. PCDAI was used for those with CD [18]. Those with a PUCAI score of <10 for UC were classified as remission/inactive disease, 10–34 as mild, 35–64 as moderate, and ≥65 as severe disease activity. Those with a PCDAI score of <10 for CD were classified as remission/inactive disease, 10–27.5 as mild, 30–37.5 as moderate, and >40 as severe disease activity.

Paris classification was used for the classification of endoscopic findings [19]. Among the treatment parameters, number of relapses, whether steroids were taken in induction, the total duration of steroid use, and whether biological agents were used were investigated.

Statistical Analysis

The data were analyzed using the SPSS Statistics version 25 (USA: IBM Corp.) program. The distribution of the data was evaluated with the Shapiro Wilk test. The results of the descriptive analyses were shown as mean±SD for normally distributed, and as the median and interquartile range for non-normally distributed data. Chi-square test was used to compare categorical data in independent groups, and Fisher's exact test was used for tables with insufficient sample size. Independent groups were compared by using an Independent-samples T test for parameters with normal distribution, and a Mann-Whitney U test for those with non-normal distribution. One-way Anova for normally and Kruskal Wallis test for non-normally distributed parameters were applied for more than two group comparisons. Related groups were compared using Paired-samples T test for normal distributed and Wilcoxon signed ranks test for non-normally distributed variables. Pearson correlation tests were used for normally, and Spearman correlation tests for non-normally distributed variables. The level of significance was accepted as $p<0.05$.

RESULTS

A total of 58 patients were included in the study group, comprising 29 individuals with UC and 29 with CD. The mean age at diagnosis was 12.2±3.4 years (range: 2.2-17 years). The duration between the onset of symptoms and diagnosis (delta diagnosis time) was 7.58±6.88 months, with no significant difference between disease groups. The male-to-female ratio (M/F) was 0.81 in patients with UC and 1.07 in those with CD.

The most common complaints were diarrhea (96.5%), abdominal pain (93.2%), blood in the stool (75.8%) and mucus (72.5%) in patients with UC and abdominal pain (94.8%) and diarrhea (93.1%) in CD patients. The most frequent physical examination finding was abdominal tenderness, with a prevalence of 46.5% among all patients. Perianal disease was observed in patients with CD patients. The most common extraintestinal findings were anemia in 62%, arthritis/arthralgia in 31%, and growth retardation in 20.7% of all patients.

Among the 29 patients with CD, 12 (41.4%) had severe disease activity, 4 (13.8%) had moderate disease activity, and 13 (44.8%) had mild disease activity at diagnosis. Among 29 patients with UC, one (3.5%) had severe disease activity, 11 (37.9%) had moderate disease activity, and 17 had (58.6%) mild disease activity. Regarding the degree of inflammation based on fecal calprotectin levels, 19 (57.5%) patients with CD and 14 (42.5%) with UC had severe inflammation. According to the Paris classification, 37.9% of UC patients had pancolitis, 27.6% had left colon involvement, 13.8% had extensive involvement (involvement beyond the splenic flexure but not reaching hepatic flexure), and 17.2% had ulcerative proctitis. Among CD patients, 62.1% had ileocolonic involvement, 17.2% had colonic involvement and 17.2% had ileal/limited cecum involvement.

The weight, height, BMI, and growth velocity SDS values were compared between UC and CD patients, and no significant differences were observed. More retardation from the target height was observed in patients with CD (Table 1). The follow-up period spanned a range of 6 months to 72 months, with a mean duration of 23.62±19.8 months. There was no significant differ-

Table 1. Anthropometric measurements and growth velocity of the patients at diagnosis

Growth parameter	Ulcerative colitis (n=29)	Crohn's disease (n=29)	p
Age at diagnosis (year)	12.0 (4.96)	14.0 (3.75)	0.159[b]
Weight SDS	-1.20±1.4	-0.85±1.8	0.112[a]
Height SDS	-0.54±1.7	-0.23±1.2	0.317[a]
Body mass index SDS	-1.16±2.0	-1.04±1.9	0.111[a]
Growth velocity SDS	-0.18±1.99	0.10±2.09	0.614[a]
Height SDS - target height SDS	0.29 (1.64)	-0.27 (1.25)	0.046[b]*

SDS: Standard deviation score; a: Independent-Samples T test; results are shown as mean±standard deviation; *: P<0.05; b: Mann-Whitney U test; results shown as median (interquartile range).

Table 2. Tanner Stages of the patients with ulcerative colitis and Crohn's disease

Tanner stage	Ulcerative colitis 29 (100%)	Crohn's disease 29 (100%)
Stage-1	4 (13.7)	2 (6.80)
Stage-2	3 (10.3)	6 (20.6)
Stage-3	1 (3.40)	0 (0.00)
Stage-4	6 (20.6)	9 (31.0)
Stage-5	15 (51.7)	12 (41.3)

Table 3. Comparison of mean ages of the patients according to pubertal stage with healthy Turkish children

Gender/Tanner stage	Study group age (Mean±SD)	Reference age (Mean±SD)	p
Girl/Tanner-2	11.79±0.41	10.16±0.97	0.113
Girl/Tanner-4	15.31±2.11	12.97±1.17	0.042*
Menarche	12.54±1.02	12.41±0.92	0.534
Male/Tanner-2	12.20±1.57	11.76±1.76	0.431
Male/Tanner-4	14.70±1.71	13.17±0.87	0.022*

SDS: Standard deviation; *: P<0.05. One-sample T-test.

ence between UC and CD in terms of anthropometric measurements at the end of the follow-up period.

There were no statistically significant differences in weight, height, BMI, growth velocity, or retardation from target height SDS parameters between boys and girls with IBD. The growth velocity, adjusted for pubertal stage and excluding patients who had completed linear growth, was found to be within the normal range in 19 patients (11 UC, 8 CD) and reduced in 12 patients (5 UC, 7 CD). The frequency of low growth velocity did not demonstrate a statistically significant difference between the disease groups (p=0.379).

The pubertal stages of the entire study group (30 female, 28 male) were evaluated. The girls were divided into four groups: four were prepubertal, two were at Tanner Stage 2, six were at Stage 4, and 18 were at Stage 5. Of the 28 boys, two were prepubertal, seven were at Stage 2, one was at Stage 3, nine were at Stage 4, and nine were at Stage 5. Table 2 presents the Tanner stages of patients with UC and CD. A comparison of the mean age at pubertal stages of male and female patients in the study group with the mean age of healthy Turkish children is presented in Table 3. In terms of mean age, there was no significant difference between the entire study group and healthy children in the early pubertal stage. However, the mean age in Stage-4 was significantly higher in the study group, suggesting that while there was no delay in the onset of puberty, there was a delay in the progression. Upon exclusion of prepubertal patients, 40% of pubertal patients with UC were in different stages of puberty, while 60% had completed puberty (Tanner Stage 5). In contrast, 55% of patients with CD were in the pubertal period, while 45% had completed puberty. The chi-square test indicated that there was no statistically significant difference between the disease groups (p=0.262).

Extraintestinal findings were observed in three (50%) patients in the prepubertal period, in seven (25%) patients in the pubertal period, and in five (18.5%) patients in the postpubertal period. The frequency of malnutrition in patients presenting with extraintestinal findings or an accompanying disease was significantly lower compared to other patients (p=0.040, p=0.011, respectively).

According to the Paris classification, 37.4% of the patients with UC had pancolitis, and 62.1% of the patients with CD had ileocolonic involvement. Height and retardation from target height were significantly lower in UC patients with pancolitis than in others (p=0.040,

Table 4. Growth parameters according to the disease activity and the degree of inflammation

Growth parameter	Disease severity mild (n=30)	Disease severity moderate/severe (n=28)	p	Inflammation mild/moderate (n=3)	Inflammation severe (n=33)	p
Weight SDS	-0.81±1.64	-1.01±1.78	0.717[a]	0.08±2.4	-1.05±1.65	0.615[a]
Height SDS	0.18±0.65	-0.55±1.55	0.190[a]	-0.06±1.03	-0.34±1.4	0.727[a]
BMI SDS	-1.52±2.34	-0.87±1.72	0.181[a]	0.03±2.05	-1.19±1.91	0.643[a]
Growth velocity SDS	-0.44±1.88	-0.26±2.11	0.212[a]	0.42±3.3	0.09±2.09	0.809[a]
Height – target height (SDS)	0.26 (0.22)	-0.04 (1.37)	0.332[b]	0.12 (0.28)	0.18 (1.3)	0.977[b]

SDS: Standard deviation score; a: Independent-Samples T test; results are shown as mean±standard deviation; b: Mann-Whitney U test; results shown as median (interquartile range).

Table 5. Comparison of growth and disease activity parameters before and after treatment

Growth and disease activity parameters	Before treatment	After treatment	p
Weight SDS (mean±standard deviation)	-0.63±1.53	-0.37±1.20	0.048[a]*
Height SDS (mean±standard deviation)	-0.25±1.19	-0.10±0.94	0.224[a]
BMI SDS (mean±standard deviation)	-0.68±1.62	-0.37±1.19	0.045[a]*
Delta-target height SDS, median (quartile difference)	0.02 (1.56)	0.32 (1.59)	0.108[b]
PCDAI for CD, median (min–max/IQR)	30.0 (15–70/22.5)	5.00 (0–15/10.0)	0.000[b]*
PUCAI for UC, median (min–max/IQR)	30.0 (12.5–60/22.5)	0.00 (0–17.5/0.00)	0.000[b]*

a: Paired Samples T Test; b: Wilcoxon Signed Ranks Test; SDS: Standard deviation score; BMI: Body Mass Index; CD:Crohn's disease; UC: Ulcerative colitis; PCDAI: Pediatric Crohn's Disease Activity Index; PUCAI: Pediatric Ulcerative Colitis Activity Index. Since the median and interquartile range (IQR) values of PUCAI and PCDAI scores at diagnosis in UC and CD patients were found to be the same by chance, they were also shown in the table as "median (minimum–maximum/IQR)".

p=0.022, respectively). However, in CD patients, groups according to the sites of involvement (ileocolonic and others) showed no significant difference in terms of growth parameters. There were no significant differences in growth parameters according to the disease activity scores or according to the level of inflammation (Table 4).

Steroid use was employed in induction therapy in 41 of the patients. In 30 of these patients, the steroid treatment could be reduced within three months. However, 19 patients required steroid treatment again within one year. In the induction treatment phase, steroid plus mesalazine was initiated in one patient, steroid plus azathioprine in one patient, azathioprine plus mesalazine in seven patients, and all three drugs were initiated simultaneously in 39 patients. As maintenance treatment, 56 patients received mesalazine, three patients received salazoprine, 44 patients received azathioprine, 11 patients received infliximab, five patients received adalimumab, one patient received vedolizumab, and five patients received methotrexate. There was no significant difference in the weight SDS, height SDS, BMI SDS, growth velocity SDS and deviation from target height parameters between patients who had received glucocorticoids during induction and who did not (p=0.444. p=0.122. p=0.695. p=0.901. p=0.953, respectively). The comparison of these growth parameters at last visit between patients who received biological agents and who did not show any significant difference (p=0.333. p=0.254. p=0.066. p=0.244. p=0.743, respectively).

A comparison of the growth parameters and disease activity parameters of our patients at diagnosis and after treatment revealed a significant improvement in the disease activity scores, as well as a significant improvement in the weight and BMI SDS values (Table 5).

Analyses revealed no correlation between growth parameters and disease scores at diagnosis or after treatment, time to diagnosis, number of stools per day, fecal calprotectin, number of relapses, or duration of steroid administration. There was no correlation between the stage of puberty and the number of relapses, the duration of steroid use, or disease severity scores. The post-treatment PUCAI/PCDAI scores were significantly lower in 57 patients and equal in only one patient (p=0.000). There was no correlation between the PUCAI/PCDAI scores and the anthropometric data, biochemical parameters, and pubertal stages at diagnosis. Similarly, no correlation was observed between fecal calprotectin levels and anthropometric data, biochemical parameters, and pubertal stage.

DISCUSSION

The results of this study demonstrated a notable improvement in weight and BMI SDS following treatment. Children and adolescents with IBD had no significant adverse effects on linear growth at diagnosis or during follow-up, regardless of the primary disease or treatment protocol. The treatment protocols, including the use of pharmacologic doses of steroids in induction, the total duration of steroids, and the use of biological agents, did not result in a significant change in growth in accordance with the literature. This suggests that the management of the inflammatory process and maintenance of disease control overcomes the growth suppression effect of steroids.

Anthropometric parameters were not different between boys and girls in our study, contrary to the report of the Pediatric CD Study Group which showed lower height gains in boys than in girls [20]. Systemic diseases with a rapid onset May 19, 2024lead to negative energy balance. Weight loss is evident at first, and linear growth is affected after the process is prolonged and diagnosis is delayed. Our patients with UC may have had height SDS values above the target height line at presentation due to positive environmental conditions before the onset of the disease. Possibly the diagnostic process did not take long enough to affect height. Patients with CD exhibited greater negative deviations from the target height at diagnosis. Since CD manifests with more insidious and non-specific findings, the diagnosis may be relatively late. When the negative effects on the nutritional status may develop over a longer period linear growth may also decrease. Another very important effect on growth is the process of chronic systemic inflammatory condition, which is more pronounced in CD. In the study of Jin et al. [21], patients with moderate and severe disease according to the PCDAI had lower IGF-1-SDS values. Song et al. [22], also found a statistically significant decrease in weight and BMI SDS in patients with higher PCDAI for CD patients.

Weight and BMI improved significantly with treatment while height was not significantly different after treatment in the follow-up period of up to 72 months (with a mean of 23 months). The disease activity scores were significantly improved after, confirming a successful suppression of disease, while height values were similar. This suggested that the height was already not affected at diagnosis. It can also be claimed that the negative effects of systemic inflammation and both positive and negative effects of the treatment agents provided a balanced outcome during treatment. In the study of Jin et al. [21], it was also reported that height did not change significantly, while weight increased significantly with treatment in 70 patients. Pfefferkorn et al. [23] and Vasseur et al. [24] also found no difference in height during a 2-year period.

Corticosteroids are still the most widely used agents to achieve remission in acute relapses. There are many studies investigating the effect of steroids on growth parameters. The fact that the growth parameters of the patients who received and did not receive steroid in induction treatment were not significantly different, suggesting that steroids may have less of an effect on growth than disease inflammation. In a study by Motil et al. [25], a negative relationship was found between linear growth and disease activity, but there was no relationship between steroid treatment and linear growth. In another study by Malik et al. [26], there was no significant difference between the groups that received and did not receive steroids.

In a study evaluating the adult height of patients, it was reported that patients with childhood-onset IBD reached a lower adult height than in the general population and healthy siblings. In patients with severe inflammation the final adult height was found to be lower than the others [27]. Malik et al. [26] concluded that even if disease control is good, growth is affected because of several interrelated factors.

When we analyzed our patients according to their nutritional status, patients with malnutrition had more insidious findings while patients with better nutritional status at diagnosis had more additional diseases or extraintestinal findings, suggesting that findings due to extraintestinal comorbidities may lead to earlier diagnosis.

The patients with pancolitis due to UC showed significantly lower height and more negative deviations from target height, compared to patients with less regional involvement, suggesting a reduced growth in this subgroup. However, the small number of our patients in the subgroups makes it difficult to conclude. In the study by Kim et al. [28] in which 594 patients were evaluated, pancolitis was the most common type in UC, and the height SDS of the patients was found to be significantly lower than in other types of involvement. Growth parameters were not different among the groups of involvement, as well as the groups of inflammation and groups of disease severity in patients with CD. There are studies showing the effects of ileal disease involvement on disease activation and severity of inflammation [21]. In the study of Song et al. [22], no significant correlation was found between weight, height, BMI, and disease region, while Timmer et al. [29] reported that the delay in diagnosis and growth retardation were higher in patients with ileal involvement in CD.

A comparison of the mean age at pubertal stages of male and female patients in the study group with data from healthy Turkish children revealed that the ages of girls and boys with Tanner Stage-4 were significantly higher than those of healthy Turkish children [15]. These data indicate that although there is no delay in the onset of puberty in children with IBD, there may be a slowdown in the rate of pubertal progression. This may be due to the disease itself, treatments, and nutritional deficiencies. Considering the diseases, the fact that those with UC are more in the post-pubertal period may suggest that those with CD are at higher risk for delayed puberty. In the study of Jin et al. [21], 11 of 109 patients (10.1%) presented with delayed puberty and had a significantly lower BMI. In the same study, 8 out of 31 patients who experienced menarche before the diagnosis developed secondary amenorrhea. In our study, no significant difference was observed in the age of menarche between healthy Turkish children. However, secondary amenorrhea was identified in two patients with Crohn's disease (CD). The relationship between inflammatory bowel disease (IBD) and menstrual changes has not yet been fully elucidated.

The main limitation of this study was the relatively small sample size. Nevertheless, the patients in the study group were monitored on a regular basis, providing valuable insights into the growth velocity and pubertal progression in this specific chronic disease of childhood. The effects of IBD on growth have been investigated in some larger cohorts, but there is a clear need for further studies on the onset and progression of puberty.

Conclusion

While weight and BMI were affected at diagnosis and improved with treatment, linear growth markers were not affected at diagnosis and did not show a significant difference under treatment. Although the age at which puberty begins remains unchanged in individuals with IBD, the pubertal tempo may slow down, or the patients may have been diagnosed at relatively advanced stages of puberty. The potential adverse effects of therapeutic agents on linear growth are offset by successful control of the disease.

Ethics Committee Approval: The Umraniye Training and Research Hospital Clinical Research Ethics Committee granted approval for this study (date: 31.03.2022, number: B.10.1TKH.4.34.H.GP.0.01/13).

Informed Consent: Written informed consents were obtained from patients who participated in this study.

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