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Personalized treatment algorithm for acute anal fissures: comparison with traditional symptomatic therapy

L. Hrytsak

Bogomolets National Medical University, Kyiv

✉ Liliia Hrytsak: lylychka5@gmail.com

L. Hrytsak, https://orcid.org/0000-0003-1779-8912

Objective – to evaluate the clinical efficacy of a personalized treatment algorithm for acute anal fissures based on a fissure chronicity risk scale compared with standard symptomatic therapy.

Materials and methods. This prospective non-randomized comparative study included 175 patients with acute anal fissure treated at the proctology department of Kyiv City Clinical Hospital № 18 between 2021 and 2024. The mean patient age was 39.29 ± 12.98 years. The study group received individualized treatment based on chronicity risk assessment, while the control group received standard symptomatic treatment. Efficacy was assessed by the rate of complete healing at week 4, pain dynamics on the VAS scale, and the incidence of chronicity.

Results. The personalized treatment protocol resulted in a higher rate of complete healing compared to standard therapy (81.63 % versus 41.56 %, $p < 0.001$). The incidence of chronicity of anal fissures was lower in the study group (4.08 %) than in the control group (23.38 %). Patients in the study group achieved pain reduction of more than 50 % faster than those in the control group (6.1 ± 2.3 versus 12.8 ± 3.5 days). Side effects were observed in both groups, including local redness (11 % and 14 %), temporary incontinence (7 % and 0 %), and headache (0 % and 3 %).

Conclusions. The personalized protocol for managing acute anal fissures shows higher efficacy and significantly improved clinical outcomes compared to standard conservative therapy. Implementation of this approach in clinical practice accelerates healing, reduces chronicity rates, and lowers early recurrence. Therefore, adoption of the proposed protocol is recommended.

Keywords
minimally invasive proctology, combined pathology, anal fissure, hemorrhoids, scar deformities, perianal scar.

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Anal fissure represents the second most prevalent disease of the anorectal region, accounting for 10 % to 15 % of visits to a proctologist [9, 12, 14]. An acute anal fissure is defined as a linear defect of the anoderm, typically accompanied by severe pain. This condition often leads to anal sphincter spasm due to irritation of its fibers, resulting in ischemia. This pathological cycle constitutes the key pathogenetic mechanism. Prolonged ischemia usually causes the development of a chronic anal fissure [5, 9, 12, 14].

Although this pathology is quite common, optimal management tactics remain limited. Current recommendations for acute anal fissure management generally endorse the same methods, but the application process, specific indications, effectiveness evaluation, and criteria for therapy escalation are insufficiently defined. The primary therapeutic goals are to alleviate anal sphincter spasm and restore adequate blood supply. Delayed therapy escalation or inadequate therapy are associated with an increased risk of chronicity [2, 8, 13, 17]. It should be noted that predictors of an unfavourable course of acute anal fissure include pain intensity, symptom duration for more than 4 weeks, severe spasm, and concomitant functional disorders. At present, no guidelines support individualized treatment approaches.

According to the recommendations, standard conservative therapy includes stool regulation through a high-fiber diet, maintenance of water balance, sitz baths, and application of local anesthetic ointments. These measures promote healing in approximately 60 % of patients with acute anal fissures [2, 13, 17].

Early administration of topical calcium channel blockers or nitrates significantly accelerates healing and reduces the risk of chronic fissure development. However, these methods may be insufficient for patients at high risk of poor outcomes.

Therefore, there is a need to develop a personalized treatment protocol that incorporates clinical predictors of unfavourable outcomes and facilitates the rational selection of therapeutic strategies.

Objective – to evaluate the clinical efficacy of a personalized algorithm for the treatment of acute anal fissures based on a fissure chronicity risk scale compared to standard symptomatic therapy.

Materials and methods

This prospective non-randomized comparative study was conducted from 2021 to 2024 and included 175 patients with acute anal fissures. Depending on the management approach, patients were divided into two groups. The study group (n = 98) received treatment according to the chronicity risk scale (Table 1), and the control group (n = 77) received standard conservative therapy.

The fissure chronicity risk (FCR) scale was used to stratify the risk of chronicity of acute anal fissures. This scale includes five independent clinical factors with established pathogenetic significance:

Table 1. **Personalized fissure chronicity risk scale**

Criterion	Points
Symptoms lasting > 4 weeks	1
Pain on the VAS > 7	1
Pronounced anal sphincter spasm	1
Nocturnal pain	1
History of fissure recurrence	1

Interpretation:
0 – standard therapy,
1–2 – topical calcium channel blockers,
≥ 3 – botulinum toxin injections (30–50 U).

Table 2. **Internal validation of fissure chronicity risk scale in our sample**

Score	Chronic cases after treatment
0 (n = 14)	0
1 (n = 23)	1 (4.3 %)
2 (n = 31)	1 (3.2 %)
≥ 3 (n = 30)	2 (6.7 %)

symptoms persisting for more than four weeks, intense pain syndrome (> 7 on the Visual Analogue Scale (VAS)), severe sphincter spasm, presence of nocturnal pain, and a history of anal fissure recurrence. These indicators present the principal mechanisms underlying chronicity: anoderm ischemia, neuromuscular regulation disorders, and reduced reparative potential. Each criterion was assigned one point, as all indicators have comparable independent prognostic value and reflect distinct aspects of the pathophysiology. The total FCR score was used to stratify patients and guide therapeutic escalation decisions. To confirm the predictive utility of the FCR scale, an internal validation analysis was conducted. Patients were grouped according to their initial FCR score, and the frequency of chronicity after treatment was recorded (Table 2). The observed distribution demonstrates a progressive increase in chronicity among patients with higher FCR scores, thereby supporting the scale's internal validity and justifying treatment escalation in the subgroup with scores ≥ 3.

Symptoms lasting more than four weeks were interpreted as evidence of fibrotic changes at the fissure margins and persistent ischemia. A pain intensity VAS > 7 was used to assess the severity of neurogenic spasm in the internal anal sphincter. Severe sphincter spasm was identified as the primary mechanism sustaining ischemia and hindering tissue repair. Nocturnal pain was considered indicative of damage extending beyond defecation and was associated with more pronounced ischemia. A history of previous relapses suggested structural vulnerability of the anoderm and an increased risk of defect recurrence. Each of these five factors was assessed as equally influential in determining the likelihood of chronicity and was therefore assigned a weight of one point. The total FCR score was used to evaluate the overall risk and to guide decisions regarding therapeutic escalation.

Patients with 0 points received standard symptomatic therapy, while those with 1–2 points were additionally prescribed topical calcium channel blockers to reduce internal sphincter tone. Patients with a total score of ≥ 3 were considered a high-risk group in whom the pathogenetic cycle of «pain-spasm-ischemia» had already formed and required a more intensive effect on the sphincter apparatus. In such cases, botulinum toxin injection was selected as the preferred method, providing reversible chemical sphincterotomy, significantly improving blood supply to the anoderm, and facilitating effective fissure healing.

Topical calcium channel blockers were used in patients with an FCR score of 1–2. Nifedipine 0.2 % ointment was applied 2–3 times a day in

a thin layer along the anal canal (≈1 cm from the anal ring) for 14 days. The choice between nifedipine and diltiazem was based on the tolerability of previous treatments and individual patient sensitivity. Both drugs are regarded as equally effective in reducing intrasphincteric pressure.

Patients with an FCR score of ≥ 3 received botulinum toxin type A (Botox®, Allergan) injections. The drug was administered at a dose of 20–30 units, evenly distributed in the internal anal sphincter. Injections were performed at two sites, corresponding to the 3 and 9 o'clock positions on the conventional clock face, to a depth of approximately 3–4 mm. This approach ensured a uniform reduction in sphincter pressure and minimized the risk of local excessive relaxation. The injection was performed under sterile conditions, without anesthesia or with minimal local anesthesia if necessary.

Inclusion criteria were as follows: diagnosis of acute anal fissure (symptom duration ≤ 6 weeks), patient age 18–65 years, a follow-up period of at least 6 months, and provision of signed informed consent.

Exclusion criteria were as follows: diagnosis of chronic anal fissure (symptom duration ≥ 6 weeks), pregnancy, inflammatory bowel diseases, HIV, specific proctitis, previous calcium channel blocker or botulinum toxin therapy, and atypical anal fissures.

The clinical characteristics of the patients included in the study are presented in Table 3. Both groups were statistically homogeneous at the baseline with respect to age, sex, duration of symptoms, initial pain syndrome, severity of spasm, and distribution of high-risk patients according to the FCR scale (all $p > 0.2$).

Table 3. **Clinical characteristics of patients**

Indicator	Study group (n = 98)	Control group (n = 77)
Age, years	37.83 ± 11.62	41.16 ± 14.39
Men	43 (43.9 %)	35 (45.5 %)
Duration of symptoms, days	8.7 ± 2.4	9.1 ± 2.6
VAS (baseline)	7.1 ± 1.2	7.0 ± 1.3
Severe spasm	37,8 %	33,8 %
FCR ≥ 3	30,6 %	28,6 %
Concomitant anorectal diseases, %	65.3 %	72.7 %
History of constipation or diarrhea	43.9 %	45.5 %
History of acute anal fissure	23.3 %	24.3 %

All $p > 0.05$.

Additionally, possible confounding factors such as concomitant anorectal diseases, defecation disorders (constipation or diarrhea), and previous episodes of acute anal fissures did not differ significantly between groups ($p = 0.28$; $p = 0.83$; $p = 0.85$, respectively). These findings confirm the clinical homogeneity of the study samples at the time of inclusion.

Treatment efficacy was assessed based on the following criteria: complete epithelialization at week 4, pain syndrome dynamics, frequency of progression to chronic fissure, and early recurrence of fissure. However, a preliminary assessment was performed on day 14 to determine the need for therapy escalation. In cases where clinical improvement was ≥ 30 %, patients who received standard therapy alone were additionally prescribed calcium channel blockers; those receiving calcium channel blockers were escalated to Botox therapy; and those on Botox were considered for lateral internal sphincterotomy (LIS), although this intervention was not included in the present study.

The follow-up period lasted at least 6 months. The median follow-up was 7 months (range: 6–12 months), allowing assessment of both early and delayed chronicity.

Statistical analysis was performed using MedStat software. Quantitative variables are presented as mean ± standard deviation (M ± SD), and categorical variables are presented as frequencies and their percentage distributions (n, %). Normality of quantitative data was assessed both graphically and using descriptive statistics.

Student's t-test was used to compare the means of two independent groups. Categorical variables were analyzed using Pearson's χ^2-test, and Fisher's exact test was employed when expected frequencies in the cells were < 5. Differences in proportions of complete epithelialization were assessed using the χ^2 test, with relative risk (RR) and its 95 % confidence interval (CI) also calculated.

The dynamics of pain syndrome were analyzed using Student's t-test for independent samples. The frequency of progression from acute to chronic fissures was assessed by comparing proportions (χ^2), with calculation of RR and odds ratio (OR). Statistical significance was defined as $p < 0.05$. No correction for multiple comparisons was applied, as the study had a predefined primary endpoint.

Results

Baseline characteristics of both groups were homogeneous with respect to age, sex, initial pain syndrome intensity, duration of clinical manifestations, and severity of internal anal sphincter spasm (see Table 3).

At week 4, a significantly higher frequency of complete epithelialization was observed in patients treated according to a personalized protocol, with 80 patients (81.63 %) achieving this outcome. In contrast, only 32 patients (41.56 %) receiving standard therapy achieved healing at the same time point, representing nearly half the rate noted in the personalized protocol group. These findings indicate that a personalized management protocol based on risk assessment for anal fissure chronicity improves healing effectiveness within a month, which is statistically significant ($p < 0.001$).

Given that the primary complaint is severe pain during defecation, it is essential to assess treatment effectiveness using an analog pain scale. Patients recorded pain intensity daily. To standardize the assessment, the number of days with a pain reduction exceeding 50 % was calculated. In the study group, this indicator was 6.1 ± 2.3 days, compared to 12.8 ± 3.5 days in the control group ($p < 0.001$). This statistically significant difference suggests that addressing anal sphincter spasm and ischemia is more effective than solely providing local analgesia. Early pain relief also prevents the development of defecation-related fear and reduces the risk of early recurrence of anal fissures.

During the first week of treatment, a reduction in anal sphincter spasm was observed in 71.43 % (70 patients) of the study group and 28.57 % (22 patients) of the control group ($p < 0.001$). These results indicate that the absence of persistent tonic spasm facilitates faster healing and epithelialization.

In both groups, some patients required therapy escalation. Therefore, we conducted the initial assessment of its necessity on day 14. In the personalized protocol group, 9 patients (9.18 %) required escalation, with 5 receiving calcium channel blockers and 4 receiving botulinum toxin injections. In the control group, 21 patients (27.27 %) required escalation. It should be noted that surgical intervention was indicated for 1 patient (1.09 %) in the study group and 9 patients (11.69 %) in the control group. These indicators are critical because they increase treatment costs, risk of side effects and complications, and prolong the patient's incapacity.

Assessment of chronicity risk is essential in our study. A reduction in its score of ≥ 1 point on day 14 was observed in 68 (69.39 %) patients in the study group and 21 (27.27 %) patients in the control group. At the same time, only 4.08 % (4 patients) of the study group developed chronic anal fissures, compared with 23.38 % (18 patients) in the control group $p < 0.001$. These findings confirm that timely intervention in the pathogenetic cycle is crucial for preventing chronicity. Chronic fissures require longer, more complex treatment and can significantly impair quality of life.

A comparative analysis demonstrated significant advantages of the personalized treatment algorithm over standard therapy for most clinical endpoints (Table 4). The probability of complete healing was almost twice as high with the personalized algorithm (RR 1.96), and the chances of treatment success were more than 6 times higher (OR 6.25). The incidence of chronicity was markedly lower in the personalized group (4.08 % vs. 23.38 %), with an RR of 0.17 (95 % CI 0.06–0.49). A trend toward reduced risk of early recurrence was observed (RR 0.22), although the 95 % CI partially overlapped 1. Improvements in pathogenetic markers, specifically reduction in sphincter spasm and a decrease in FCR ≥ 1 point, occurred significantly more often in the personalized group (RR 2.50 and RR 2.54, respectively). The personalized approach also reduced the need for therapy escalation (RR 0.34 (95 % CI 0.16–0.69)) and surgical intervention (RR 0.09 (95 % CI 0.01–0.67)).

Table 4. **Therapy effectiveness indicators**

Indicator	Study group (n = 98)	Control group (n = 77)	p	RR (95 % CI)	OR (95 % CI)
Complete healing at week 4	80 (81.6 %)	32 (41.6 %)	< 0.001	1.96 (1.48–2.60)	6.24 (3.16–12.38)
Time to ≥ 50 % pain reduction, days	6.1 ± 2.3	12.8 ± 3.5	< 0.001	–	–
Chronicity	4 (4.1 %)	18 (23.4 %)	< 0.001	0.17 (0.06–0.49)	0.14 (0.04–0.43)
Early recurrence of fissure	2 (2.0 %)	7 (9.1 %)	0.036	0.22 (0.05–1.05)	0.21 (0.04–1.03)
Reduction of sphincter spasm on day 7	70 (71.4 %)	22 (28.6 %)	< 0.001	2.50 (1.72–3.64)	6.25 (3.23–12.10)
Reduction of FCR ≥ 1 point by day 14	68 (69.4 %)	21 (27.3 %)	< 0.001	2.54 (1.73–3.75)	6.04 (3.12–11.70)
Need for therapy escalation (Botox/medication)	9 (9.2 %)	21 (27.3 %)	0.002	0.34 (0.16–0.69)	0.27 (0.12–0.63)
Need for surgical intervention (LIS)	1 (1.0 %)	9 (11.7 %)	0.004	0.09 (0.01–0.67)	0.08 (0.01–0.63)

During treatment, patients reported side effects in the form of local reactions to topical agents and headaches (Figure). However, the most notable, yet anticipated and manageable, adverse event was temporary anal gas incontinence, which occurred in 7 patients (7.14 %) after botulinum toxin injections in the study group. This mild, transient anal incontinence, primarily manifested by gas incontinence, was reversible and disappeared within 1–3 weeks without treatment. This effect is consistent with the expected temporary decrease in internal sphincter tone, which underlies the therapeutic action of botulinum toxin.

In summary, the data indicate that the personalized protocol offers a clear advantage over conservative therapy. This superiority is reflected in greater reductions in spasm, pain, and chronicity, thereby supporting the pathogenetic validity of the algorithm.

Discussion

Most associations, including the American Society of Colon and Rectal Surgeons (ASCRS) and Association of Coloproctology of Great Britain and Ireland (ACPGBI), recommend conservative therapy (stool regulation, sitz baths, topical nitrates, calcium channel blockers, botulinum toxin), but do not offer a clear quantitative stratification of patients or an escalation algorithm [4, 5]. Our approach seeks to address this gap by systematizing actions and creating a precise algorithm for acute fissure treatment. Although international recommendations and meta-analyses provide a list of remedies, they lack specific guidance on the sequence and techniques for their use [1—3, 6, 8, 9, 15]. We offer an easy-to-use scale to assess fissure chronicity, enabling individualized treatment strategies. Importantly, the study groups did not have statistically significant differences in any of the key clinical or potentially confounding variables, including concomitant anorectal pathology, defecation disorders, and a history of acute anal fissures. Therefore, the observed treatment effects are unlikely to be attributable to differences in baseline patient characteristics.

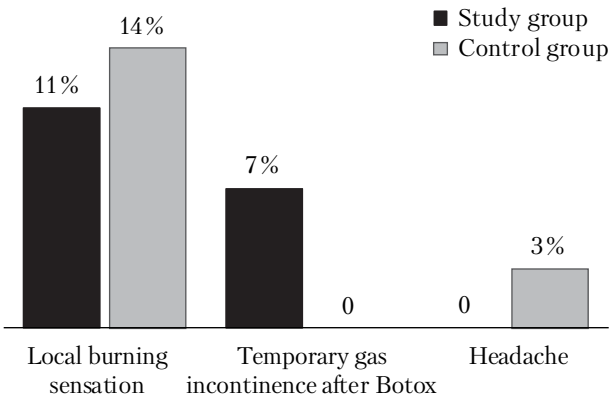

Figure. **Side effects observed in the study and control groups**

Our findings are consistent with existing literature regarding the effectiveness of topical calcium channel blockers. Several studies and clinical guidelines support the administration of topical calcium channel blockers and nitrates for the treatment of acute anal fissures. A randomized study by Momayez Sanat et al. demonstrated that nifedipine achieved higher remission rates and faster pain relief than diltiazem in patients with acute anal fissures [11]. Another randomized study found that 0.5 % nifedipine was more effective and better tolerated than 0.2 % glyceryl trinitrate in the treatment of acute fissures [1]. Although debate persists regarding the relative effectiveness of nitrates versus calcium channel blockers, meta-analyses by Sahebally et al. confirm that calcium channel blockers are both more effective and better tolerated [1, 8, 12, 15]. In our study, only calcium channel blockers were administered to patients with moderate risk (1–2 points on the FCR scale), while topical nitrate-based agents were not used. It can be assumed that, in this subgroup, the use of calcium channel blockers contributed to the high healing rate due to the rapid relief of anal sphincter spasm. These findings suggest that calcium channel blockers may be the first-line escalation for patients with moderate risk.

There is scientific debate regarding the use of botulinum toxin in the treatment of anal fissures, including questions about its effectiveness, optimal injection site, and appropriate dosage [12, 16, 17]. Most randomized controlled trials investigating botulinum toxin have focused on chronic anal fissures, where the drug serves as an alternative or adjunct to lateral internal sphincterotomy. The current ASCRS and ACPGBI guidelines do not consider botulinum toxin as a standard treatment for acute anal fissures, a position shaped by the historical context of the available evidence. Most of the existing randomized studies and meta-analyses on which these recommendations are based addressed the treatment of chronic fissures, as this was the main clinical problem requiring an alternative to surgical intervention [4, 5]. Botulinum toxin was practically excluded from studies of the acute phase, limiting its representation in the recommendations [7, 14]. The rationale for using botulinum toxin in acute anal fissures is its capacity to directly target the principal pathogenetic mechanism: pronounced tonic spasm of the internal anal sphincter, which influences anoderm ischemia and tissue repair rates.

Unlike chronic fissures, in which pathomorphological changes are established, reducing sphincter tone during the acute phase can rapidly restore blood supply at a critical time, when complete healing without surgery remains possible. Notably, the use of botulinum toxin in the acute phase, specifically for high-risk patients, has been minimally explored in the literature, making this approach a novel contribution to the field. The present findings demonstrate that targeted administration of botulinum toxin during the acute phase can substantially decrease the incidence of chronicity and reduce the need for surgical intervention.

In our study, botulinum therapy was administered exclusively in high-risk patients (FCR ≥ 3) or those unresponsive to previous therapeutic stages. The findings confirm that an FCR score of ≥ 3 identifies patients with a high probability of acute anal fissures progressing to chronicity. The presence of prolonged symptoms, severe pain, sphincter spasm, nocturnal pain, and recurrent episodes indicates a pathological cycle of «pain-spasm-ischemia» that does not respond to standard conservative therapy. In these cases, topical calcium channel blockers provide insufficient antispasmodic effects, failing to restore blood supply to the anoderm and stop disease progression. Thus, the use of botulinum toxin in high-risk patients is not an additional option, but an essential component of a treatment strategy aimed at preventing chronic fissure development. These results are particularly significant given the low chronicity rate observed in the study group. Early administration of botulinum toxin in a clearly defined high-risk category may help avoid the need for lateral internal sphincterotomy. The use of botulinum toxin during the acute phase in patients at elevated risk of chronicity aligns with and enhances current guidelines, thereby improving management for this clinically vulnerable population.

Under standard therapy, the rate of progression from acute to chronic anal fissures was consistent with international statistics and amounted to 23.38 %. This therapy includes dietary modifications, combination ointments, sitz baths, and symptomatic laxatives [1–3, 6, 8, 10]. Typically, pain reduction and partial remission occur as part of the natural disease course, without interfering with the chronicity risk assessment and the development of a structured treatment algorithm. In contrast to conventional practice, which is often chaotic or overly conservative in the early stages, the proposed algorithm enables clinicians to select the most effective method for each patient immediately. The assessment on day 14 plays a crucial role in preventing the prolongation of ineffective therapy, a primary contributor to chronicity in clinical practice. This approach advances the management of acute fissures toward personalized medicine by basing decisions on a formalized patient risk profile. The obtained RR and OR values demonstrate that the personalized algorithm not only accelerates healing but also modulates key pathogenetic mechanisms, including spasm, ischemia, and the risk of chronicity. The FCR and sphincter spasm indicators are particularly significant, as OR values exceeding 6 suggest a strong effect of the algorithm in disrupting the pathological cycle of «pain-spasm-ischemia-chronicity». Low RRs for chronicity and the need for LIS confirm that early risk stratification can prevent progression to chronic forms and reduce the need for surgical intervention. Thus, the personalized approach demonstrates both symptomatic and substantial pathogenetic efficacy, distinguishing it from standard therapy.

However, this study has several limitations. First, the non-randomized design does not eliminate the possibility of systematic errors, despite comparable baseline characteristics across groups. Second, the findings are limited to data from a single center. Third, the study group included several treatment methods, depending on risk level, which complicates direct comparison. Consequently, further evaluation through additional randomized studies is warranted.

Conclusions

This study demonstrates that a personalized approach based on risk assessment for anal fissure chronicity can significantly improve treatment effectiveness compared to standard practice regimens. The proposed algorithm facilitates rational therapy selection and reduces the need for surgical intervention. These findings provide a foundation for further randomized studies and multicenter validation of the chronicity risk assessment scale, which has the potential to change management strategies for acute anal fissures.

Declaration of interests

The author declares that there is no conflict of interest.

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Ethics approval and written informed consent statements

The study was conducted in accordance with the Declaration of Helsinki.

All patients provided informed consent.

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Персоналізований алгоритм лікування гострої анальної тріщини: порівняння з традиційною симптоматичною терапією

Л. Грицак

Національний медичний університет імені О. О. Богомольця, Київ

Мета — оцінити клінічну ефективність персоналізованого алгоритму лікування гострої анальної тріщини з використанням шкали ризику хронізації тріщини порівняно із стандартною симптоматичною терапією.

Матеріали та методи. Проведено проспективне нерандомізоване порівняльне дослідження за участю 175 пацієнтів із діагнозом «гостра анальна тріщина», які проходили лікування в проктологічному відділенні КНП «Київська міська клінічна лікарня №18» протягом 2021—2024 рр. Середній вік пацієнтів становив (39,29 ± 12,98) року. Пацієнти дослідної групи отримували індивідуальне лікування відповідно до оцінки рівня ризику хронізації, пацієнти групи контролю — рутинне симптоматичне лікування. Оцінку ефективності проводили за частотою повного загоєння на 4-й тиждень, динамікою болю за візуальною аналоговою шкалою та частотою хронізації.

Результати. У пацієнтів, які отримували лікування згідно з персоналізованим протоколом, частота повного загоєння була вищою порівняно з тими, хто отримував стандартну терапію (81,63 і 41,56% відповідно, $p < 0,001$). Частота хронізації анальної тріщини у дослідній групі становила 4,08%, у контрольній групі — 23,38%. Зменшення болю більше ніж на 50% швидше досягалося в пацієнтів дослідної групи (через (6,1 ± 2,3) та (12,8 ± 3,5) дня). Побічні дії зареєстровано в дослідній та контрольній групах: місцеве почервоніння (11 і 14% відповідно), тимчасову інконтиненцію (7 та 0%), головний біль (0 і 3%).

Висновки. Запропонований персоналізований протокол ведення пацієнтів із гострою анальною тріщиною продемонстрував вищу ефективність і значно кращі клінічні результати порівняно зі стандартною консервативною терапією. Його використання в клінічній практиці прискорює загоєння гострої тріщини, зменшує частоту хронізації та раннього рецидиву. Тому запропонований підхід доцільно впровадити в клінічну практику.

Ключові слова: малоінвазивна проктологія, поєднана патологія, анальна тріщина, геморой, рубцеві деформації, перианальний рубець.

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