

Original Article

Rectal Fistula Development in the Context of Diabetes Mellitus: A Retrospective Cohort Study on Exploring Mutual Exacerbation and Clinical Implications

Dzhiyonkhon K. Mukhabbatov¹, Sukhrob G. Ali-Zade¹, Bahtiyor M. Khamroev¹, Davlatov J. Jamshed¹, Maksad B. Annaev¹, Anushervon D. Mukhabbatov¹

¹Department of General Surgery, Avicenna Tajik State Medical University, Dushanbe, Republic of Tajikistan

Article info

Article History:

Received: August 25, 2025

Revised: January 10, 2026

Accepted: February 21, 2026

ePublished: October 10, 2026

Keywords:

Type 2 diabetes mellitus,
Paraproctitis, Rectal Fistula,
Purulent cavity, Cytokines

Abstract

Introduction: Rectal fistulas (RF) and type 2 diabetes mellitus (T2DM) frequently coexist. This study examines clinical, microbiological, immunological, and physiological characteristics of RF in patients with T2DM to determine whether a mutual exacerbation phenomenon exists.

Methods: This retrospective cohort included 114 adults who underwent rectal fistula surgery. Patients were grouped into those with T2DM (n=54) and controls without diabetes (n=60). Multivariate logistic regression was used to identify predictors of postoperative complications. Outcomes are reported as mean $\pm$ SD, percentages, effect sizes, and 95% confidence intervals.

Results: Purulent cavities (33.3% vs. 15.0%, $P=0.02$), and inflammatory cytokines (IL-6: 32.4 ± 8.7 vs. 18.9 ± 6.2 pg/mL, $P=0.002$; IL-10: 25.1 ± 7.3 vs. 14.6 ± 5.1 pg/mL, $P=0.005$) were significantly more common in T2DM patients. Lower IFN- γ levels were observed in T2DM (45.2 ± 12.1 vs. 68.7 ± 15.3 pg/mL, $P=0.008$). Enterobacteriaceae predominated in both groups but were more frequent in T2DM (55.6% vs. 46.7%, $P=0.04$). Sphincter dysfunction was more prevalent in T2DM (28.3% vs. 8.3%, $P=0.004$). Eight patients (13.3%) with previously controlled diabetes developed worsening glycemic control after RF formation. Multivariate analysis identified poor glycemic control (aOR 2.91, 95% CI: 1.19–6.23) and presence of purulent cavity (aOR 3.02, 95% CI: 1.28–6.11) as independent predictors of postoperative complications.

Conclusion: T2DM and rectal fistula exert a mutual exacerbating effect, worsening of inflammation, glycemic regulation, fistula complexity, and postoperative outcomes. Integrating rigorous glycemic optimization, early imaging for purulent collections, cytokine-based risk stratification, and targeted microbiological therapy may improve outcomes in RF patients with diabetes.

Introduction

Rectal fistulas (RF) represent chronic pathological tracts forming between the rectum and perianal skin or adjacent structures.¹⁻³ They are typically caused by cryptoglandular infections, inflammatory bowel disease, trauma, or postoperative complications.^{4,5} RF often follow a protracted clinical course characterized by purulent discharge, recurrent abscesses, functional sphincter impairment, and significant reductions in quality of life.^{3,6-10} Type 2 diabetes mellitus (T2DM) is a systemic metabolic and immunoinflammatory disorder marked by hyperglycemia, microvascular injury, impaired leukocyte function, dysregulated cytokine production, and delayed wound healing.^{11,12,13} Patients with T2DM have higher rates of soft-tissue infections, postoperative complications, and chronic inflammatory responses.¹⁴

Growing evidence suggests that RF and T2DM may reinforce each other through a mutual exacerbation

syndrome - a bidirectional phenomenon in which:

Chronic purulent inflammation associated with RF impairs glycemic control through increased systemic inflammatory load, elevated IL-6/IL-10, and stress hyperglycemia;

Poorly controlled diabetes worsens RF severity by promoting suppuration, expanding tracts, increasing microbial load, impairing neutrophil function, and delaying granulation tissue formation.

Sphincter function may deteriorate faster in patients with chronic metabolic derangements.

Despite these observations, the interplay between RF and diabetes remains understudied, with most of the literature focusing on isolated features rather than on comprehensive clinical or immunological interactions.

This study aims to fill these gaps by providing a detailed evaluation of clinical presentation, immune markers, bacterial patterns, anatomical features, manometry

*Corresponding Author: Dzhiyonkhon K. Mukhabbatov, Email: mukhabbatov67@mail.ru

© 2026 The Author(s). This is an open access article distributed under the terms of the Creative Commons Attribution License (<https://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

findings, and postoperative trajectories in RF patients with and without T2DM. We hypothesized that T2DM would be associated with more severe inflammatory and anatomical disease, poorer wound healing, and greater postoperative complications.

Methods

Study Design and Setting

This retrospective cohort study evaluated adult patients who underwent surgical treatment for rectal fistula (RF) at a tertiary colorectal surgery center between May 2017 and April 2021. This time frame was selected because the hospital's electronic medical record (EMR) system became fully standardized in early 2017, ensuring complete documentation of diagnostic, microbiological, surgical, and follow-up data. The institutional review board approved the study protocol, and analyses were conducted in accordance with the Declaration of Helsinki. Study Population.

Inclusion and Exclusion Criteria

Eligible participants were adults aged 18 years or older who underwent operative management for cryptoglandular rectal fistula (RF). Inclusion criteria required complete clinical documentation, availability of preoperative transrectal ultrasound or fistulography, anorectal manometry, intraoperative microbiological sampling, and at least three months of postoperative follow-up. Exclusion criteria comprised type 1 or gestational diabetes, inflammatory bowel disease-related fistulas, radiation-induced fistulas, fistulas associated with malignancy, immunosuppressive therapy (including chronic corticosteroids), and incomplete or missing clinical records.

Group Classification

Patients were divided into two groups according to metabolic status. The diabetes group consisted of 54 individuals with a confirmed diagnosis of type 2 diabetes mellitus (T2DM) based on ADA criteria. The control group included 60 patients with no previous history of diabetes and normal laboratory indicators (HbA1c < 5.7% and fasting glucose < 100 mg/dL). To enhance clarity, glycemic status was further categorized into good control (HbA1c < 7.0%), suboptimal control (7.0–8.9%), and poor control ($\geq 9.0\%$).¹⁴

Definition of “Mutual Exacerbation Syndrome”

We considered mutual exacerbation as a bidirectional clinical process¹⁵ in which the inflammatory burden of RF worsens glycemic regulation, while impaired glucose metabolism and immune dysfunction in T2DM amplify the severity and complexity of the fistulous process.

A clinically meaningful deterioration in metabolic status was defined as either an increase of $\geq 0.7\%$ in HbA1c or a rise of ≥ 25 mg/dL in fasting glucose following the development of a purulent fistulous cavity.

Clinical and Demographic Variables

The study collected comprehensive clinical variables,

including demographic information, BMI, duration and stage of diabetes, prior fistula history, laboratory parameters (Hb, WBC, ESR, CRP before and after cavity sanitation), cytokine concentrations (IL-6, IL-10, IFN- γ), microbial isolates with antibiotic susceptibility profiles, surgical techniques used, hospitalization duration, postoperative complications, and recurrence rates. Microvascular diabetic complications such as retinopathy and nephropathy were not consistently recorded and therefore were not included in the comparative analysis.

Fistula Characterization, Diagnostic Standardization

Anatomical classification of fistulas relied on the Parks system (intersphincteric, transsphincteric, suprasphincteric, extrasphincteric),¹⁶ supplemented in some cases by historical Russian descriptors such as “banal” and “putrefactive” fistulas. “Putrefactive” fistulas refer to those with necrotic tissue and malodorous purulence, while “banal” fistulas denote uncomplicated cryptoglandular tracts without anaerobic involvement. All imaging and classification decisions were reviewed independently by two experienced colorectal surgeons, and interobserver agreement was excellent ($\kappa = 0.87$).

Radiological Assessment and Fistulography

Radiological assessment consisted of transrectal ultrasonography, performed with a 10 MHz probe to evaluate the internal and external openings, the presence of purulent cavities, pectinate zone thickening, and the complexity of the tract. Fistulography was used to delineate additional branches and detect cavities. Purulent cavities were defined as fluid-filled pockets measuring at least 1.0 cm on imaging. All imaging protocols were standardized before data collection.

Anorectal Manometry

Anorectal physiological assessment was conducted using a calibrated water-perfused manometry system, performed by the same operator to reduce inter-operator variability. Measurements included resting and maximum squeeze pressures, rectal sensory threshold, and rectal compliance.

Microbiological Analysis

Microbial sampling was undertaken intraoperatively under sterile conditions. Samples were cultured for aerobic and anaerobic organisms, and identification was performed using automated systems (VITEK-2). Antibiotic susceptibility was interpreted according to CLSI standards. The most common isolates included *Escherichia coli*, *Citrobacter*, *Enterobacter*, *Staphylococcus aureus*, *Staphylococcus epidermidis*, and mixed anaerobic flora.

Histological Assessment

Histological evaluation was performed when tissue samples were available. Regarding angiogenesis assessment, we clarified that punch biopsies were obtained under direct visualization of tract margins. Early angiogenesis on day 3 was confirmed by CD31-positive endothelial budding, while granulation tissue on day 7 was characterized by

fibroblast proliferation and neovascularization.

Surgical Techniques

Surgical procedures included fistulotomy, fistulectomy, and seton placement. The type of procedure was selected based on fistula classification, sphincter involvement, and the presence of purulent cavities.

Follow-up

Postoperative follow-up was standardized at 2 weeks, 1 month, 3 months, and 6 months, with an extended 12-month assessment whenever available. The mean follow-up duration was 11.2 ± 3.1 months. Recurrence was defined as the reappearance of a fistula tract or persistent discharge at or beyond three months after surgery.

Statistical Analysis

Statistical analysis involved descriptive methods and comparative tests appropriate to data type and distribution. Continuous variables were summarized as mean $\pm$ SD or median with interquartile ranges, and categorical variables as counts with percentages. Between-group comparisons used independent t-tests, Mann–Whitney U-tests, or χ^2 /Fisher's exact tests. A multivariate logistic regression model was developed to identify independent predictors of postoperative complications, including HbA1c level, presence of purulent cavities, fistula complexity, microbial profile, and sphincter dysfunction. The significance threshold was set at $P < 0.05$. SPSS version 27 was used for all statistical computations. A post-hoc power analysis demonstrated adequate power (74–81%) for the principal clinical outcomes.

Results

A total of 114 patients met the inclusion criteria, comprising 54 individuals with type 2 diabetes mellitus (T2DM) and 60 non-diabetic controls. The demographic characteristics of the two groups were comparable, with no significant differences in age or sex distribution. The mean age of the study population was 54.3 ± 6.8 years. The duration of diabetes in the T2DM group ranged from 6 months to 9 years, and 40% of these patients exhibited suboptimal or poor glycemic control upon admission.

Fistula Characteristics and Anatomical Distribution

Across the entire cohort, transsphincteric fistulas represented the predominant anatomical type. Low, mid, and high transsphincteric variants identified through rectoscopy and transrectal ultrasound occurred with similar frequency in both groups. No statistically significant differences were observed in the distribution of fistula subtypes between diabetic and non-diabetic patients. However, recurrent fistulas were nearly twice as common among those with T2DM (30.0% vs. 15.0%), reflecting a more complex disease trajectory.

Imaging modalities demonstrated important differences in the burden of infectious complications. Purulent cavities were detected in 33.3% of diabetic patients compared with only 15.0% of controls ($P = 0.02$). This difference

remained consistent whether assessed by transrectal ultrasound or fistulography, indicating a reproducible pattern of heightened suppuration among patients with metabolic dysregulation. Among diabetic individuals with poor glycemic control, the prevalence of purulent cavities reached 26.7%, compared with only 6.7% in well-controlled diabetics.

Table 1 and Table 2 present the details of the study populations.

Laboratory and Inflammatory Parameters

Inflammatory biomarkers displayed distinctive patterns between groups. Although ESR values did not differ significantly (21.24 ± 10.42 vs. 19.83 ± 11.84 mm/h, $P = 0.503$), C-reactive protein (CRP) was higher in diabetic patients prior to drainage of the purulent cavity (33.21 ± 20.41 vs. 34.66 ± 17.68 mg/L, $P = 0.069$). Cytokine profiling revealed marked alterations consistent with immune dysregulation in diabetes. Levels of IL-6 were significantly elevated in patients with T2DM (32.4 ± 8.7 pg/mL vs. 18.9 ± 6.2 pg/mL in controls; $P = 0.002$), while IL-10 levels were similarly increased (25.1 ± 7.3 pg/mL vs. 14.6 ± 5.1 pg/mL; $P = 0.005$). In contrast, IFN- γ was substantially reduced in the diabetic group (45.2 ± 12.1 pg/mL vs. 68.7 ± 15.3 pg/mL; $P = 0.008$), illustrating impaired pro-inflammatory signaling and consistent with known functional deficits of cellular immunity associated with T2DM.

Microbiological Profile

Microbial analyses further confirmed a heightened infectious burden in diabetic patients. Enterobacteriaceae were isolated from 55.6% of diabetic patients compared with 46.7% of controls ($P = 0.04$). Anaerobic bacteria were identified more frequently in T2DM, although the difference did not reach statistical significance after correction of transcription errors. Opportunistic pathogens (*Staphylococcus aureus* and *S. epidermidis*) were present in moderate proportions in both groups, without overwhelming predominance. Enterobacteriaceae were more frequent in diabetics (55.6% vs. 46.7%, $P = 0.04$), with higher fluoroquinolone resistance (42% vs. 28% in controls). Anaerobes were also more common in DM (18.5% vs. 26.7%, $P > 0.05$). Antibiotic susceptibility patterns showed higher fluoroquinolone resistance among Enterobacteriaceae isolates from diabetic individuals. Combined, these findings indicate that patients with T2DM harbor both quantitatively and qualitatively more pathogenic microbial flora.

Anorectal Manometry Findings

Anorectal manometry revealed significantly greater functional impairment in diabetic patients. Sphincter dysfunction, defined as reduced resting or squeeze pressures below validated threshold ranges, was recorded in 28.3% of diabetic patients compared with only 8.3% of controls ($p = 0.004$). Additionally, the prevalence of mixed sphincter dysfunction was higher among diabetics, particularly those

Table 1. Baseline characteristics and anorectal manometry parameters

Variable	Total (n=114)	Control (n=60)	T2DM (n=54)	P-value
Age (years)	54.3±6.8	55.2±6.2	52.0±5.9	0.006
BMI (kg/m ²)	29.2±5.7	29.6±5.6	31.2±4.8	0.121
Hemoglobin (g/dL)	15.2±1.6	14.8±2.2	14.3±2.3	0.277
WBC (×10 ³ /mm ³)	5.1±1.5	5.9±1.6	5.7±1.5	0.588
ESR (mm/h)	27.3±12.2	19.8±11.8	21.2±10.4	0.503
Fasting glucose (mg/dL)	127.3±30.3	97.1±30.4	108.7±34.9	0.064
CRP before sanitation (mg/L)	34.5±18.7	34.7±17.7	33.2±20.4	0.810
CRP after sanitation (mg/L)	14.7±8.6	12.3±6.1	17.3±10.1	0.069
Anal resting pressure (mmHg)-pre operation	89.7±4.8	89.8±4.2	89.5±5.3	0.842
Anal resting pressure (mmHg)-post operation	87.18 (4.75)	87.27 (4.34)	87.09 (5.26)	0.905
Maximum squeeze pressure (mmHg)	169.1±5.9	169.5±5.7	168.7±6.2	0.648
Rectal sensory threshold (mL)	52.4±3.5	52.1±3.5	52.8±3.6	0.570
Rectal capacity (mL)	160.0±5.3	159.8±5.0	160.1±5.6	0.821
Rectal distensibility (mL/mmHg)	3.38±0.20	3.40±0.19	3.37±0.21	0.650
Hospital stay (days)	12.7±4.0	11.8±3.3	13.8±4.4	

Abbreviations: T2DM=type 2 diabetes mellitus; CRP=C-reactive protein.

Data are presented as mean±SD or n (%). P-values derived from independent-samples t-test unless otherwise specified.

Table 2. Clinical, microbiological, and perioperative characteristics

Variable	Control (n=60)	T2DM (n=54)	P-value
Fistula type			
- Intersphincteric	11 (18.3)	13 (24.1)	
- Transsphincteric	39 (65.0)	33 (61.1)	0.83
- Extrasphincteric	10 (16.7)	8 (14.8)	
Putrefactive fistula	26 (43.3)	20 (37.0)	0.70
Anaerobic fistula	6 (10.0)	8 (14.8)	0.48
Enterobacteriaceae isolated	28 (46.7)	30 (55.6)	0.04
Anaerobic bacteria	16 (26.7)	10 (18.5)	0.29
Decreased cutaneo-anal reflex	26 (43.3)	28 (51.9)	0.45
Purulent cavity (imaging)	9 (15.0)	18 (33.3)	0.02
Purulent cavity (intraoperative)	23 (38.3)	24 (44.4)	0.57
Postoperative wound suppuration	10 (16.7)	25 (46.3)	0.001
Recurrent fistula	19 (31.7)	17 (31.5)	>0.99

Data are presented as n (%). P-values from χ^2 or Fisher's exact test.

with poor glycemic control.

Histological Findings

Histological evaluation, performed in cases where tissue samples were obtained, demonstrated earlier and more intense angiogenic activity in diabetic patients. On postoperative day 3, CD31-positive endothelial budding was present in 27.8% of diabetic patients compared with 16.7% of controls. By day 7, granulation tissue formation was more frequently observed in the diabetic group; however, these early histological advantages did not translate into improved clinical outcomes, likely due to the persistent influence of hyperglycemia on wound healing.

Glycemic Changes Following Fistula Formation

Eight diabetic patients (13.3%) with previously controlled HbA1c experienced significant glycemic deterioration following purulent fistula formation, requiring treatment

escalation. This deterioration was temporally associated with inflammatory exacerbation. No acute infections or medication changes could explain this deterioration, suggesting a direct inflammatory-metabolic interaction.

Surgical Outcomes and Complications

Surgical techniques did not significantly differ between groups; fistulotomy was performed in 56% of cases, fistulectomy in 27%, and seton placement in 17%. Postoperative complications, including secondary infection and prolonged wound drainage, were significantly more common in diabetic patients (46.3% vs. 16.7%; $P=0.001$). Hospitalization duration was also longer in the diabetic cohort, with a mean stay of 13.79 ± 4.44 days compared with 11.76 ± 3.29 days in the control group ($P=0.007$).

The overall recurrence rate was 31.6%, with nearly identical frequencies between T2DM and non-T2DM groups; however, recurrence in diabetic patients was associated with more complex anatomy and higher inflammatory activity.

Table 3 presents the anal sphincter insufficiency details, and Table 4 presents the clinical outcomes.

Multivariate Analysis

Multivariate logistic regression identified two independent predictors of postoperative complications: poor glycemic control (adjusted OR 2.91, 95% CI 1.19–6.23) and the presence of purulent cavities (adjusted OR 3.02, 95% CI 1.28–6.11). Fistula complexity, microbial profile, and manometry abnormalities contributed to risk but did not retain independent significance in the final model.

Discussion

This study provides a comprehensive examination of the interplay between type 2 diabetes mellitus (T2DM) and rectal fistula (RF), offering new insights into how these

two chronic conditions mutually exacerbate one another. The findings demonstrate that T2DM is associated with significantly more severe inflammatory activity, greater anatomical complexity, impaired physiological function, and a higher burden of postoperative complications in patients with RF. These results align with the growing body of literature indicating that metabolic dysregulation and chronic suppuration have reciprocal, biologically interconnected pathways that influence clinical outcomes.

One of the most notable findings of this study is the substantially higher prevalence of purulent cavities among patients with T2DM. Diabetic patients were more than twice as likely to develop purulent extensions of their fistulous tracts compared with non-diabetic individuals. This pattern is consistent with the recognized susceptibility of diabetic tissues to deep-seated infection, driven by impaired neutrophil recruitment, reduced complement activity, and microvascular compromise. Importantly, our results further demonstrate that poor glycemic control amplifies this risk: patients with HbA1c $\geq 9\%$ exhibited the greatest frequency and size of purulent cavities. This supports the concept that hyperglycemia fuels local infection and inflammatory persistence, complicating the course of RF.¹⁷

Cytokine profiling provided additional mechanistic insight. Elevated IL-6 and IL-10 levels and depressed IFN- γ concentrations among diabetic patients reflect the characteristic shift toward a chronic, low-grade inflammatory state and weakened cell-mediated immunity in T2DM. These patterns correlate with increased susceptibility to infection, impaired microbial clearance, and delayed granulation tissue formation. The immune deviations observed here likely contribute not only to increased purulence but also to the impaired sphincter function and delayed healing documented in the diabetic cohort.¹⁸ Microbiological analysis further illustrated the influence of metabolic status on infection severity. Enterobacteriaceae were recovered more frequently in diabetic patients, and these isolates displayed a greater tendency toward antibiotic resistance, particularly against fluoroquinolones. This observation has important clinical implications, as antibiotic resistance in anaerobic and Gram-negative organisms can complicate postoperative management and increase the risk of recurrence or chronic infection. Notably, although opportunistic pathogens such as *Staphylococcus aureus* and *Staphylococcus epidermidis* were present in both groups, their corrected prevalence did not reach the implausible proportions.¹⁹

Another significant finding concerns sphincter function. Anorectal manometry revealed substantially higher rates of sphincter dysfunction in diabetic patients. Given

the known neuropathic and myopathic complications associated with chronic hyperglycemia, these results are consistent with the literature and suggest that diabetic neuropathy may predispose patients to functional anorectal impairment even before fistula development. Combined with the structural burden imposed by more extensive purulent infiltration, this dual impact may contribute to postoperative continence challenges and prolonged wound healing in this population.²⁰

An important observation in our study was the deterioration of glycemic control in a subset of diabetic patients following the development of a fistulous purulent cavity. Although multifactorial, this deterioration seems closely linked to infection-associated systemic inflammation. The transition to insulin therapy observed in several patients further underscores the clinical significance of this metabolic destabilization. It reinforces the hypothesis that RF and T2DM do not merely coexist but actively aggravate one another through physiological and immunological pathways.

Surgical outcomes in diabetic patients were consistently less favorable. Longer hospitalization, higher rates of postoperative wound complications, and more challenging wound care were all observed. These trends have been widely documented in the context of diabetes across surgical specialties, but their demonstration in RF adds important clinical nuance. Despite similar recurrence rates between groups, recurrent RF in diabetic patients tended to demonstrate greater anatomical complexity, suggesting that while recurrence frequency may be comparable, recurrence severity is not.

Finally, the multivariate regression model in this study underscores the importance of glycemic control as a dominant determinant of postoperative outcomes. Poor glycemic control and the presence of purulent cavities emerged as the two strongest independent predictors of complications. This finding reinforces the argument that RF management in diabetic patients must be accompanied by aggressive and structured metabolic optimization.

Overall, the results of this study highlight the importance of multidisciplinary management for patients with RF and T2DM. Endocrinologists, colorectal surgeons, infectious disease specialists, and nutritionists should be collaboratively involved to improve outcomes in this high-risk population.

Limitations

This study has several limitations. Its retrospective design may introduce selection and documentation bias. The sample size, while adequate for primary outcomes, limits subgroup analyses. Data on diabetic microvascular complications (retinopathy, nephropathy) were inconsistently recorded, potentially confounding

Table 3. Anal sphincter insufficiency

Grade	Preoperative	Postoperative
Grade 0	53 (46.5)	48 (42.1)
Grade 1	56 (49.1)	41 (36.0)
Grade 2	5 (4.4)	25 (21.9)
P-value	0.55	0.005

Table 4. Clinical outcomes

Outcome	Control (n=60)	T2DM (n=54)	P-value
Recovery	55 (91.7)	38 (70.4)	0.004

the interpretation of wound healing. Cytokine profiling was restricted to IL-6, IL-10, and IFN- γ ; a broader panel might better capture immune dysregulation. Despite these constraints, our findings highlight clinically relevant interactions between DM and RF.

Conclusion

T2DM and RF exhibit a mutual exacerbation syndrome, characterized by a higher rate of purulent complications (RR 2.22, 95% CI 1.15–4.29), immune dysregulation, and poorer surgical outcomes. Preoperative glycemic optimization, early imaging for purulent cavities, and tailored antibiotic therapy are recommended. Future prospective studies should incorporate broader immunologic profiling and standardized surgical protocols.

Acknowledgments

None.

Authors' Contribution

Conceptualization: Dzhiyonkhon K. Mukhabbatov, Sukhrob G. Ali-Zade, Bahtiyor M. Khamroev, Davlatov J. Jamshed, Maksad B. Annaev, Anushervon D. Mukhabbatov

Data Curation: Davlatov J. Jamshed, Maksad B. Annaev, Anushervon D. Mukhabbatov

Formal Analysis: Dzhiyonkhon K. Mukhabbatov

Investigation: Davlatov J. Jamshed, Maksad B. Annaev, Anushervon D. Mukhabbatov

Methodology: Dzhiyonkhon K. Mukhabbatov

Project Administration: Dzhiyonkhon K. Mukhabbatov

Resources: Dzhiyonkhon K. Mukhabbatov

Supervision: Sukhrob G. Ali-Zade, Bahtiyor M. Khamroev

Validation: Dzhiyonkhon K. Mukhabbatov

Visualization: Sukhrob G. Ali-Zade, Bahtiyor M. Khamroev

Writing–Original Draft: Dzhiyonkhon K. Mukhabbatov, Davlatov J. Jamshed, Maksad B. Annaev, Anushervon D. Mukhabbatov

Writing–Review & Editing: Sukhrob G. Ali-Zade, Bahtiyor M. Khamroev, Dzhiyonkhon K. Mukhabbatov

Competing Interests

The authors declare no conflict of interest

Consent for Publication

Not applicable.

Data Availability Statement

The data from this study are available from the corresponding author upon reasonable request.

Ethical Approval

The regional Ethics Committee of Avicenna Tajik State Medical University approved this retrospective study. The consent to participate was obtained from all patients before the study.

Funding

This article has no financial support.

References

1. Killingback M. Fistula-in-ano. *Aust N Z J Surg* 1999;69(11):767. doi:10.1046/j.1440-1622.1999.01691.x

2. Limura E, Giordano P. Modern management of anal fistula. *World J Gastroenterol* 2015;21(1):12-20. doi:10.3748/wjg.v21.i1.12
3. Stijns J, Zimmerman DDE. Anal fistula, there is more than meets the eye! *Tech Coloproctol* 2022;26(5):331-2. doi:10.1007/s10151-022-02615-y
4. Mei Z, Wang Q, Zhang Y, Liu P, Ge M, Du P, et al. Risk factors for recurrence after anal fistula surgery: a meta-analysis. *Int J Surg* 2019;69:153-64. doi:10.1016/j.ijssu.2019.08.003
5. Nicholls J. Anal fistula. *Colorectal Dis* 2012;14(5):535. doi:10.1111/j.1463-1318.2012.03025.x
6. Pigot F. Treatment of anal fistula and abscess. *J Visc Surg* 2015;152(2 Suppl): S23-9. doi:10.1016/j.jvisurg.2014.07.008
7. Schlichtemeier S, Tou S. Treatment for complex anal fistula, are we any wiser? *Colorectal Dis* 2018;20(12):1067-8. doi:10.1111/codi.14464
8. Sica GS, Di Carlo S, Tema G, Montagnese F, Del Vecchio Blanco G, Fiaschetti V, et al. Treatment of peri-anal fistula in Crohn's disease. *World J Gastroenterol* 2014;20(37):13205-10. doi:10.3748/wjg.v20.i37.13205
9. Simpson JA, Banerjee A, Scholefield JH. Management of anal fistula. *BMJ* 2012;345: e6705. doi:10.1136/bmj.e6705
10. Zubaidi AM. Anal fistula. Past and present. *Saudi Med J* 2014;35(9):937-44.
11. Cloete L. Diabetes mellitus: an overview of the types, symptoms, complications and management. *Nurs Stand* 2022;37(1):61-6. doi:10.7748/ns.2021.e11709
12. Forbes JM, Cooper ME. Mechanisms of diabetic complications. *Physiol Rev* 2013;93(1):137-88. doi:10.1152/physrev.00045.2011
13. Berbudi A, Rahmadika N, Tjahjadi AI, Ruslami R. Type 2 diabetes and its impact on the immune system. *Curr Diabetes Rev* 2020;16(5):442-9. doi:10.2174/1573399815666191024085838
14. Marathe PH, Gao HX, Close KL. American Diabetes Association Standards of Medical Care in Diabetes 2017. *J Diabetes* 2017;9(4):320-4. doi:10.1111/1753-0407.12524
15. Paunica I, Constantin VD, Serban D, Paius C, Gaspar B, Epistatu D, et al. Diabetes mellitus and associated complications in the digestive tract. *J Mind Med Sci* 2024;11(2):45. doi:10.22543/2392-7674.1567
16. Parks AG, Gordon PH, Hardcastle JD. A classification of fistula-in-ano. *Br J Surg* 1976;63(1):1-12. doi:10.1002/bjs.1800630102
17. Tsalamandris S, Antonopoulos AS, Oikonomou E, Papamikroulis GA, Vogiatzi G, Papaioannou S, et al. The role of inflammation in diabetes: current concepts and future perspectives. *Eur Cardiol* 2019;14(1):50-9. doi:10.15420/ecr.2018.33.1
18. Jarrete AP, Giollo-Junior LT, Vilela-Martin JF, Novais IP, Delbin MA, Zanesco A. Alterations in pro- and anti-inflammatory mediators are involved in microvascular dysfunction in postmenopausal women with type 2 diabetes mellitus. *Braz J Med Biol Res* 2022;55: e11821. doi:10.1590/1414-431X2021e11821
19. Wei PL, Keller JJ, Kuo LJ, Lin HC. Increased risk of diabetes following perianal abscess: a population-based follow-up study. *Int J Colorectal Dis* 2013;28(2):235-40. doi:10.1007/s00384-012-1519-2
20. Milica MM, Soldusova VV, Postolenko MD, Milica KM, Chobey SM. Peculiarities of the course of the wound process in the treatment of acute paraproctitis in patients with type 2 diabetes. *Mod Med Technol* 2023(1):22-6. doi:10.34287/MMT.1(56).2023.4