Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Association Between GLP-1 Receptor Agonist Use and Postpartum Pelvic Complications: A Global Matched Cohort Study

Muhammed A. M. Hammad

mahammad@hs.uci.edu

University of California

Sara Perelmuter

Weill Cornell Medicine

Vada Furlan

University of California

Hana Nakamura

University of California

Rachel S. Rubin

Georgetown University

Jessica Yih

University of California

Research Article

Keywords:

Posted Date: August 20th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10580243/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Postpartum women experience high rates of urinary tract infections (UTIs), inflammatory genital conditions, and pelvic pain syndromes, which negatively affect quality of life and sexual health. Obesity and systemic inflammation may contribute to these complications. Glucagon-like peptide-1 receptor agonists (GLP-1RAs), increasingly prescribed for weight management in non-diabetic individuals, have anti-inflammatory and immunomodulatory effects that may influence pelvic health; however, their role in postpartum pelvic morbidity has not been studied. The objective of this study was to evaluate the association between GLP-1RA use and postpartum pelvic complications including UTIs, inflammatory genital infections, and pelvic pain syndromes, in a large, global cohort of non-diabetic women.

Methods

We conducted a retrospective cohort study using the TriNetX Global Collaborative Network. Postpartum women prescribed GLP-1RAs were identified and matched 1:1 by propensity scores to non-users based on age, body mass index (BMI), hypertension, and race/ethnicity. Primary outcomes included incident UTIs, inflammatory genital conditions (e.g., vaginitis, vulvitis), and pelvic pain syndromes (including dyspareunia and vulvodynia). Outcomes were assessed using cumulative incidence following propensity score matching. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to compare risk between cohorts.

Results

After matching, 5,387 women remained in each cohort. GLP-1RA use was associated with significantly lower odds of UTIs (4.09% vs 6.94%; OR 0.571, 95% CI 0.461–0.707, $p < 0.0001$), inflammatory genital infections (12.19% vs 18.65%; OR 0.605, 95% CI 0.504–0.727, $p < 0.0001$), and pelvic pain syndromes (7.03% vs 11.98%; OR 0.555, 95% CI 0.465–0.663, $p < 0.0001$).

Conclusions

In this large, propensity-matched cohort of non-diabetic postpartum women, GLP-1RA use was associated with significantly reduced risks of infectious and pain-related pelvic complications after adjustment for baseline BMI. These findings suggest a potential protective association between GLP-1RA therapy and postpartum pelvic health and warrant prospective validation.

Introduction

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are increasingly prescribed for weight management in non-diabetic individuals. As their use expands among reproductive-aged women, understanding their systemic effects beyond glycemic control has become clinically important. The postpartum period represents a biologically vulnerable window characterized by pelvic floor trauma, hormonal shifts, immune modulation, and metabolic stress. During this time, women face increased risks of urinary tract infections (UTIs), inflammatory genital conditions (e.g., vaginitis, vulvitis), and pelvic pain syndromes including dyspareunia, vulvodynia, and chronic pelvic pain. Many of these conditions persist beyond the traditional 6-week recovery period and are associated with impaired sexual function, depressive symptoms, reduced quality of life, and increased healthcare utilization[1–4].

Postpartum weight retention and obesity are increasingly prevalent and may exacerbate pelvic morbidity. Adiposity is now recognized as a chronic pro-inflammatory state characterized by elevated tumor necrosis factor-α, interleukin-6, and C-reactive protein, alongside endothelial dysfunction and altered immune signaling[5–7]. These inflammatory pathways overlap mechanistically with those implicated in recurrent genitourinary infections and chronic pain sensitization. Obesity has been associated with higher rates of UTIs and lower urinary tract symptoms, as well as dysregulated mucosal immunity and microbiome alterations[8–10]. Moreover, systemic inflammation may enhance peripheral afferent nerve excitability and central pain amplification, contributing to persistent pelvic pain syndromes[11,12]. Traditional postpartum interventions, including antibiotics, symptomatic analgesia, and lifestyle counselling, address discrete manifestations but may not target underlying inflammatory drivers.

GLP-1 receptor agonists exert pleiotropic effects extending beyond weight loss. In addition to reducing adiposity, GLP-1 signaling improves endothelial function, modulates immune responses, and reduces circulating inflammatory cytokines in both diabetic and non-diabetic populations[13–16]. Experimental and clinical data suggest GLP-1RAs attenuate systemic inflammation and may influence autonomic and central regulatory pathways, including the arcuate nucleus of the hypothalamus, which integrates metabolic, inflammatory, and autonomic signaling[17,18]. Emerging real-world evidence suggests GLP-1RA use may favorably influence lower urinary tract outcomes independent of glycemic status[19]. However, their potential role in postpartum pelvic health has not been examined.

To date, no large, propensity-matched real-world cohort studies have evaluated the association between GLP-1RA therapy and postpartum pelvic complications, including UTIs, inflammatory genital infections, and pelvic pain syndromes. It remains unclear whether any observed benefits are independent of body mass index (BMI) and weight loss alone. The primary objective of this study was to evaluate the association between GLP-1RA use and postpartum pelvic morbidity in a large global cohort of non-diabetic women. We hypothesized that GLP-1RA use would be associated with reduced rates of postpartum UTIs, inflammatory genital conditions, and pelvic pain syndromes after matching for BMI.

Methods

We conducted a retrospective cohort study using the TriNetX Global Collaborative Network, a federated electronic health record database that aggregates de-identified clinical data from healthcare organizations worldwide. We identified adult postpartum females (≥ 18 years) and constructed exposure and control cohorts based on GLP-1RA use. Postpartum status was defined by the presence of Current Procedural Terminology (CPT) code 59430 ("postpartum care only"), which captures routine postpartum follow-up after delivery. The exposure cohort included patients with documented GLP-1RA use, identified using both medication-specific and class-level coding, including RxNorm code 1991302 (semaglutide) and Anatomical Therapeutic Chemical (ATC) code A10BJ (glucagon-like peptide-1 analogues), in conjunction with postpartum status. The control cohort consisted of postpartum females without any prior or subsequent exposure to GLP-1RAs. To minimize confounding from metabolic disease, patients with any diagnosis of diabetes mellitus were excluded from both cohorts using ICD-10 codes E08–E13, including E11 (type 2 diabetes mellitus).

To ensure appropriate temporal sequencing, the index event for the GLP-1RA cohort was defined as the first recorded GLP-1RA prescription (RxNorm 1991302 or ATC A10BJ) occurring on or after documentation of postpartum care (CPT 59430), whereas for the control cohort, the index event was defined as the date of postpartum care (CPT 59430). Outcomes were assessed after the index event and followed longitudinally, thereby capturing incident events and minimizing inclusion of pre-existing conditions or events recorded concurrently with cohort entry.

The study population initially included 6,118 postpartum women receiving GLP-1RA therapy and 283,497 postpartum women without GLP-1RA exposure.

To reduce confounding and balance baseline characteristics between groups, 1:1 propensity score matching was performed using a nearest-neighbor algorithm. Matching variables included age, body mass index (BMI), hypertension, and race/ethnicity. After matching, 5,387 patients remained in each cohort.

The primary outcomes evaluated were UTIs, pelvic pain syndromes including dyspareunia and vulvodynia, and inflammatory genital infections such as vaginitis and vulvitis. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to compare risk between cohorts.

Results

After 1:1 propensity score matching, 5,387 patients remained in each cohort. The mean follow-up duration was 644.8 days for the GLP-1 receptor agonist (GLP-1RA) cohort and 1,564.4 days for the control cohort (Fig. 1).

Urinary tract infections (UTIs) occurred in 4.09% of patients receiving GLP-1RA therapy compared with 6.94% of controls, corresponding to a significantly reduced risk among GLP-1RA users (OR 0.571, 95% CI 0.461–0.707, $p < 0.0001$) (Fig. 2).

Inflammatory genital infections were diagnosed in 12.19% of GLP-1RA users compared with 18.65% of controls, indicating a significantly lower risk among GLP-1RA users (OR 0.605, 95% CI 0.504–0.727, $p < 0.0001$) (Fig. 2).

Pelvic pain syndromes, including dyspareunia and vulvodynia, occurred in 7.03% of patients in the GLP-1RA group and 11.98% of patients in the control group, representing a significant reduction in risk associated with GLP-1RA exposure (OR 0.555, 95% CI 0.465–0.663, $p < 0.0001$) (Fig. 2).

Overall, GLP-1RA therapy was consistently associated with lower incidence of postpartum pelvic complications across all evaluated outcomes.

Discussion

In this large, global, propensity-matched cohort of non-diabetic postpartum women, GLP-1RA use was associated with lower incidence of UTIs, inflammatory genital infections, and pelvic pain syndromes. Compared with matched controls, GLP-1RA users had lower odds of UTI, inflammatory genital conditions, and pelvic pain outcomes, with a consistent direction and magnitude of association across all prespecified endpoints. Importantly, these associations were consistent across all evaluated outcomes using incidence-based comparisons following propensity score matching.

The magnitude of effect across multiple pelvic outcomes was clinically meaningful. For example, the absolute risk of UTI was reduced from 6.94% to 4.09%, and inflammatory genital infections from 18.65% to 12.19%, corresponding to relative risk reductions of approximately 40–45%. These conditions are highly prevalent in the postpartum period and represent important contributors to impaired sexual function, diminished quality of life, and increased healthcare utilization[1,2,4]. Postpartum urinary tract infections and vulvovaginal inflammatory conditions are well-recognized complications following delivery, while persistent pelvic pain syndromes including dyspareunia and vulvodynia, can substantially affect long-term sexual and psychosocial well-being[20–22]. The consistency of findings across both infectious and pain-mediated endpoints strengthens the overall signal and reduces the likelihood that these associations are isolated or spurious.

The observed associations are biologically plausible given the known anti-inflammatory and immunomodulatory properties of GLP-1 signaling. Obesity is characterized by chronic low-grade inflammation driven by adipokine dysregulation and elevated cytokines such as TNF-α and IL-6[5,6]. GLP-1RAs have been shown to reduce circulating inflammatory markers, including high-sensitivity C-reactive protein and pro-inflammatory cytokines, in both diabetic and non-diabetic populations[14,16,23]. Attenuation of systemic inflammation may improve mucosal immune regulation and barrier integrity within the genitourinary tract, potentially reducing susceptibility to recurrent infections.

Beyond infection risk, inflammatory mediators are central drivers of peripheral and central pain sensitization[11]. Cytokine-mediated amplification of nociceptive signaling has been implicated in chronic

pelvic pain syndromes, including vulvodynia and dyspareunia. By modulating inflammatory pathways, GLP-1RAs may reduce nociceptor excitability and central pain amplification. Additionally, GLP-1 receptors are expressed in central regulatory regions, including the arcuate nucleus of the hypothalamus, which integrates metabolic, inflammatory, and autonomic signaling[17,18]. Neuroendocrine modulation at this level could plausibly influence autonomic pelvic organ function and pain processing.

In the postpartum period, GLP-1RAs may regulate hypersensitivity states after peripheral nerve injury[24]. During difficult childbirth, damage may occur to the branches of the inferior hypogastric plexus. Subsequently, nerve fibers proliferate along the damaged nerve trunk leading to disorganized reinnervation of the pelvic viscera, contributing to chronic pelvic pain[25]. Animal studies have shown that the GLP-1 receptors on microglia are upregulated in the dorsal horn of the spinal cord after peripheral nerve injury, and administering GLP-1RA decreases peripheral nerve injury hypersensitivity states[24].

Pelvic venous congestion, specifically elevated isovolumetric venous pressure, is another proposed mechanism underlying chronic pelvic pain[26]. During pregnancy, pelvic veins dilate to accommodate the increased volume of venous return. After birth, veins lack the elastic recoil of arteries, so the veins remain dilated, and the valvular leaflets do not touch, even with decreased demand[27]. Data from RCTs have found that locally delivered GLP-1RAs enhance dermal microvascular perfusion, hypothesized to occur via a nitric oxide-dependent mechanism[28]. However, other RCTs found that GLP-1RA administration did not influence systemic microvascular perfusion[29].

Improved glycemic-independent immune regulation may contribute to reduced UTI risk. While diabetes is a known risk factor for recurrent urinary infection[8], our BMI-matched design suggests that the observed protective association is not solely attributable to differences in adiposity. The persistence of effect after matching for BMI supports a potential weight-independent component of GLP-1–mediated benefit. Emerging evidence also suggests that GLP-1RAs may influence gut microbial composition and intestinal immune signaling[30,31]. Preclinical and clinical studies demonstrate that GLP-1RAs can alter microbial diversity and increase the relative abundance of beneficial taxa such as *Akkermansia*, *Bacteroides*, and *Lactobacillus*, while reducing dysbiosis associated with metabolic disease and systemic inflammation[30–32]. These microbiome shifts may enhance gut barrier integrity and modulate host immune responses, mechanisms that could indirectly affect genitourinary microbial ecology and infection susceptibility[31,32]. Although causal evidence linking GLP-1RA-induced microbiome changes directly to urinary tract infection risk is currently limited, these findings suggest a potential microbiome-mediated pathway contributing to glycemic-independent protection.

Most prior GLP-1RA studies have focused on glycemic control, cardiovascular risk reduction, renal outcomes, and weight loss[14,15,33]. Emerging literature has explored lower urinary tract outcomes in diabetic populations and overactive bladder cohorts, but postpartum pelvic health has not been systematically evaluated. To our knowledge, this represents the first large real-world propensity-matched cohort study evaluating the association between GLP-1RA therapy and postpartum pelvic morbidity in a

non-diabetic population. These findings should be interpreted as associative rather than causal; however, the consistency across multiple pelvic outcomes and survival analyses suggests a signal worthy of prospective validation. In this context, our results are hypothesis-generating yet clinically compelling, particularly given the high prevalence of postpartum pelvic complications and limited preventive options.

If validated prospectively, GLP-1RAs may represent a novel adjunctive strategy for high-risk postpartum women, particularly those with obesity, recurrent UTIs, or inflammatory pelvic pain syndromes. The potential dual benefit of weight reduction and systemic anti-inflammatory modulation may offer advantages beyond traditional symptom-targeted therapies. These findings are relevant to multidisciplinary postpartum care involving urology, urogynecology, obstetrics, and women's sexual health specialists. Careful patient selection, counseling regarding known gastrointestinal side effects, and individualized risk-benefit discussions remain essential. While GLP-1RAs are not currently indicated for pelvic disorders, the present data suggest a potential role in integrated postpartum metabolic and pelvic health strategies.

Strengths and Limitations

This study has several notable strengths. First, it leverages a large, globally representative real-world cohort derived from the TriNetX Global Collaborative Network, enhancing generalizability across healthcare systems and geographic regions. The inclusion of over 5,000 matched postpartum women per group provides substantial statistical power to detect clinically meaningful differences in relatively common pelvic outcomes. Second, the use of 1:1 propensity score matching on age, BMI, hypertension, and race/ethnicity strengthens internal validity and reduces measurable confounding. By matching on BMI, we sought to partially disentangle GLP-1RA effects from weight-related differences in pelvic floor risk, allowing for a more focused assessment of potential weight-independent associations. Third, multiple clinically relevant pelvic outcomes were assessed, including urinary tract infections, inflammatory genital conditions, and pelvic pain syndromes. The consistency of associations across infectious and pain-mediated endpoints supports robustness of findings and reduces the likelihood of isolated outcome bias. Finally, this study uniquely focuses on a non-diabetic postpartum population, a group that has been underrepresented in GLP-1RA research, which has historically centered on diabetes, cardiovascular risk, and obesity outcomes. This emphasis enhances the novelty and clinical relevance of our findings.

Several limitations warrant consideration. Most importantly, this was a retrospective observational study. Although propensity matching mitigates measured confounding, causality cannot be established. Unmeasured variables may have influenced the associations observed. The analysis relied on diagnostic coding within electronic health records. Misclassification bias is possible, and coding does not capture symptom severity, duration, or patient-reported burden. Furthermore, postpartum pelvic complications are frequently underdiagnosed or undercoded, which may attenuate true incidence estimates. Residual confounding remains a concern. Although GLP-1RAs produce significant weight reduction, the persistence of associations after matching for BMI suggests that weight-independent mechanisms may

also contribute. However, longitudinal weight trajectories were not available in this dataset, and weight change could partially mediate the observed associations. Variables such as breastfeeding status, parity, mode of delivery (vaginal versus cesarean), degree of perineal trauma, smoking status, hormonal contraception use, socioeconomic status, and access to care were not directly controlled. These factors are known to influence postpartum pelvic morbidity[1,2] and may partially account for observed differences. Follow-up duration differed between cohorts, with shorter mean follow-up in the GLP-1RA group. Finally, we were unable to directly measure inflammatory biomarkers, microbiome composition, hormonal status, or weight change trajectories. Differential healthcare utilization between groups may also have influenced diagnosis rates, as patients receiving GLP-1RA therapy may have had different patterns of clinical follow-up. As such, mechanistic inferences remain speculative.

Future Directions

The findings of this study are hypothesis-generating and support several avenues for future investigation. Prospective clinical trials in postpartum women are needed to validate the observed associations and assess causality. Randomized or pragmatic comparative effectiveness trials could determine whether GLP-1RA therapy reduces incident UTIs, inflammatory genital conditions, or chronic pelvic pain in high-risk postpartum populations. Mechanistic studies are also warranted to explore the anti-inflammatory and immunomodulatory effects of GLP-1 signaling in the postpartum genitourinary tract. Investigation of cytokine profiles, vaginal and urinary microbiome composition, and mucosal immune markers could clarify biological pathways underlying the associations observed[5,11].

Future research should incorporate validated patient-reported outcome measures to assess symptom severity, sexual function, and quality of life. Objective pelvic floor assessments and imaging may further elucidate whether GLP-1RA therapy influences pelvic floor structure or neuromuscular function. Finally, comparative effectiveness studies evaluating GLP-1RAs alongside other postpartum interventions, such as pelvic floor physical therapy, weight management programs, and antimicrobial prophylaxis for recurrent UTIs, would help define optimal multidisciplinary care pathways.

Conclusions

In this large, matched cohort of non-diabetic postpartum women, GLP-1RA use was associated with significantly lower risks of UTIs, pelvic pain syndromes, and genital inflammatory infections. These findings suggest a protective effect of GLP-1RAs on pelvic health in postpartum women beyond weight loss.

Declarations

Ethics approval and consent to participate: This study utilized only de-identified patient data from the TriNetX Global Collaborative Network. As the data are de-identified and aggregated at the point of access, this study was exempt from Institutional Review Board (IRB) review. It is IRB exempt as the data

is publicly available with access/wording of the trinetx searches found in methods section is this manuscript. No individual patient consent was required given the retrospective, de-identified nature of the data.

Consent for publication: Not applicable.

Competing Interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Funding: The authors received no financial support for the research, authorship, and/or publication of this article.

Availability of data and materials: The data that support the findings of this study are available from TriNetX, LLC, but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are, however, available from the authors upon reasonable request and with permission of TriNetX, LLC.

Author Contribution

SP: Conceptualization, Writing – Original Draft, Writing – Review & Editing, Project Administration.MH: Conceptualization, Methodology, Formal Analysis, Data Curation, Writing – Review & Editing, Supervision.VF and HN: Writing – Original Draft, Writing – Review & Editing, prepared the Figures and Tables. JY: Formal Analysis, Writing – Review & Editing, Supervision.RR: Writing – Review & Editing, Supervision.

References

1. Thompson JF, Roberts CL, Currie M, et al. Prevalence and Persistence of Health Problems After Childbirth: Associations with Parity and Method of Birth. Birth. 2002;29:83–94.
2. McDonald EA, Gartland D, Small R, et al. Dyspareunia and childbirth: a prospective cohort study. BJOG Int J Obstet Gynaecol. 2015;122:672–9.
3. Barrett G, Pendry E, Peacock J, et al. Women's sexual health after childbirth. BJOG Int J Obstet Gynaecol. 2000;107:186–95.
4. Woolhouse H, Gartland D, Perlen S, et al. Physical health after childbirth and maternal depression in the first 12 months post partum: results of an Australian nulliparous pregnancy cohort study. Midwifery. 2014;30:378–84.
5. Hotamisligil GS. Inflammation and metabolic disorders. Nature. 2006;444:860–7.
6. Lumeng CN, Saltiel AR. Inflammatory links between obesity and metabolic disease. J Clin Invest. 2011;121:2111–7.

7. Ouchi N, Parker JL, Lugus JJ, et al. Adipokines in inflammation and metabolic disease. Nat Rev Immunol. 2011;11:85–97.
8. Gupta K, Grigoryan L, Trautner B. Urinary Tract Infection. Ann Intern Med. 2017;167:ITC49–64.
9. Semins MJ, Shore AD, Makary MA, et al. The impact of obesity on urinary tract infection risk. Urology. 2012;79:266–9.
10. Nitzan O, Elias M, Chazan B, et al. Urinary tract infections in patients with type 2 diabetes mellitus: review of prevalence, diagnosis, and management. Diabetes Metab Syndr Obes Targets Ther. 2015;8:129–36.
11. Ji R-R, Chamessian A, Zhang Y-Q. Pain regulation by non-neuronal cells and inflammation. Science. 2016;354:572–7.
12. Sluka KA, O'Donnell JM, Danielson J, et al. Regular physical activity prevents development of chronic pain and activation of central neurons. J Appl Physiol. 2013;114:725–33.
13. Brock C, Hansen CS, Karmisholt J, et al. Liraglutide treatment reduced interleukin-6 in adults with type 1 diabetes but did not improve established autonomic or polyneuropathy. Br J Clin Pharmacol. 2019;85:2512–23.
14. Mosenzon O, Capehorn MS, De Remigis A, et al. Impact of semaglutide on high-sensitivity C-reactive protein: exploratory patient-level analyses of SUSTAIN and PIONEER randomized clinical trials. Cardiovasc Diabetol. 2022;21:172.
15. Heerspink HJL, Apperloo E, Davies M, et al. Effects of Semaglutide on Albuminuria and Kidney Function in People With Overweight or Obesity With or Without Type 2 Diabetes: Exploratory Analysis From the STEP 1, 2, and 3 Trials. Diabetes Care. 2023;46:801–10.
16. Xu D, Nair A, Sigston C et al. Potential Roles of Glucagon-Like Peptide 1 Receptor Agonists (GLP-1 RAs) in Nondiabetic Populations. *Cardiovasc Ther* 2022; 2022: 6820377.
17. Secher A, Jelsing J, Baquero AF, et al. The arcuate nucleus mediates GLP-1 receptor agonist liraglutide-dependent weight loss. J Clin Invest. 2014;124:4473–88.
18. Timper K, Brüning JC. Hypothalamic circuits regulating appetite and energy homeostasis: pathways to obesity. Dis Model Mech. 2017;10:679–89.
19. Hammad MAM, Quesada SG, Belczyk AL, et al. Beyond Glycemic Control: Concurrent GLP-1 Receptor Agonist Use Is Associated with Reduced Urinary Adverse Events Following OnabotulinumtoxinA Treatment in Non-Diabetic Adults with Overactive Bladder. Toxins. 2025;17:542.
20. Foxman B. Urinary tract infection syndromes: occurrence, recurrence, bacteriology, risk factors, and disease burden. Infect Dis Clin North Am. 2014;28:1–13.
21. Reed BD, Haefner HK, Sen A, et al. Vulvodynia incidence and remission rates among adult women: a 2-year follow-up study. Obstet Gynecol. 2008;112:231–7.
22. Hagenbeck C, Kössendrup J, Soff J, et al. Pelvic floor-related sexual functioning in the first 24 months postpartum: Findings of a large cross-sectional study. Acta Obstet Gynecol Scand. 2025;104:203–14.

23. Tan SA, Tan L, LIRAGLUTIDE AND SEMAGLUTIDE ATTENUATE INFLAMMATORY CYTOKINES INTERFERON-GAMMA, TUMOR NECROSIS FACTOR-ALPHA. AND INTERLEUKIN-6: POSSIBLE MECHANISM OF DECREASING CARDIOVASCULAR RISK IN DIABETES MELLITUS. J Am Coll Cardiol. 2019;73:1866.
24. Gong N, Xiao Q, Zhu B, et al. Activation of spinal glucagon-like peptide-1 receptors specifically suppresses pain hypersensitivity. J Neurosci Off J Soc Neurosci. 2014;34:5322–34.
25. Quinn M. Obstetric denervation-gynaecological reinnervation: disruption of the inferior hypogastric plexus in childbirth as a source of gynaecological symptoms. Med Hypotheses. 2004;63:390–3.
26. Foong LC, Gamble J, Sutherland IA, et al. Microvascular changes in the peripheral microcirculation of women with chronic pelvic pain due to congestion. BJOG Int J Obstet Gynaecol. 2002;109:867–73.
27. Riding DM, Hansrani V, McCollum C. Pelvic vein incompetence: clinical perspectives. Vasc Health Risk Manag. 2017;13:439–47.
28. Aung MM, Slade K, Freeman LAR, et al. Locally delivered GLP-1 analogues liraglutide and exenatide enhance microvascular perfusion in individuals with and without type 2 diabetes. Diabetologia. 2019;62:1701–11.
29. Smits MM, Tonneijck L, Muskiet MHA, et al. GLP-1-Based Therapies Have No Microvascular Effects in Type 2 Diabetes Mellitus: An Acute and 12-Week Randomized, Double-Blind, Placebo-Controlled Trial. Arterioscler Thromb Vasc Biol. 2016;36:2125–32.
30. Gofron KK, Wasilewski A, Małgorzewicz S. Effects of GLP-1 Analogues and Agonists on the Gut Microbiota: A Systematic Review. Nutrients. 2025;17:1303.
31. Kanbay M, Al-Shiab R, Shah E, et al. Gut microbiota modulation in GLP-1RA and SGLT-2i therapy: clinical implications and mechanistic insights in type 2 diabetes. Clin Kidney J. 2025;18:sfaf351.
32. Singh K, Aulakh SK, Nijjar GS et al. Rebalancing the Gut: Glucagon-Like Peptide-1 Agonists as a Strategy for Obesity and Metabolic Health. *Cureus*; 16: e64738.
33. Drucker DJ. Mechanisms of Action and Therapeutic Application of Glucagon-like Peptide-1. Cell Metab. 2018;27:740–56.

Tables

A- Table. ICD-10 Codes Used to Define Inflammation / Infection Outcomes

ICD-10 Code	Diagnosis Description	Category	Approx. Count
N76.0	Acute vaginitis	Vaginal infection	2,456,593
R10.2	Pelvic and perineal pain	Pelvic inflammatory symptoms	3,706,733
N94.8	Other specified conditions associated with female genital organs and menstrual cycle	Genital inflammatory condition	1,068,541
N94.89	Other specified conditions associated with female genital organs and menstrual cycle	Genital inflammatory condition	1,009,361
N76.1	Subacute and chronic vaginitis	Vaginal infection	693,805
N76.2	Acute vulvitis	Vulvar infection	658,161
N76.3	Subacute and chronic vulvitis	Vulvar infection	585,576
N75.1	Abscess of Bartholin’s gland	Vulvar abscess	52,911
A06.89	Other amebic infections	Parasitic infection	338
A06.82	Other amebic genitourinary infections	Parasitic GU infection	71
A59.01	Trichomonal vulvovaginitis	STI	139,070
N77.1	Vaginitis, vulvitis and vulvovaginitis in diseases classified elsewhere	Vulvovaginal infection	93,775
A60.04	Herpesviral vulvovaginitis	STI (viral)	68,618
N94.818	Other vulvodynia	Vulvar pain/inflammatory overlap	9,561
A56.02	Chlamydial vulvovaginitis	STI (bacterial)	7,685
A54.02	Gonococcal vulvovaginitis, unspecified	STI (bacterial)	3,436
N70–N77	Inflammatory diseases of female pelvic organs	Pelvic inflammatory disease	3,855,558
B37.3	Candidiasis of vulva and vagina	Fungal infection	1,387,804
B95.5	Unspecified streptococcus as cause of diseases classified elsewhere	Bacterial infection	89,366
A54.00	Gonococcal infection of lower genitourinary tract, unspecified	STI (bacterial)	35,348
A56.00	Chlamydial infection of lower genitourinary tract, unspecified	STI (bacterial)	28,634

B-Table. ICD-10 Codes Used to Define Pain Outcomes

ICD-10 Code	Diagnosis Description	Category	Approx. Count
N94.81	Vulvodynia	Vulvar pain	62,286
N94.819	Vulvodynia, unspecified	Vulvar pain	42,421
R10.2	Pelvic and perineal pain	Pelvic pain	3,706,733
N94.1	Dyspareunia	Sexual pain	482,750
F52.6	Dyspareunia not due to substance or known physiological condition	Sexual pain (functional)	7,445
N94.10	Unspecified dyspareunia	Sexual pain	345,489
N94.19	Other specified dyspareunia	Sexual pain	34,840
N94.12	Deep dyspareunia	Sexual pain	19,110
N94.11	Superficial (introital) dyspareunia	Sexual pain	10,779

C-Table. ICD-10 Codes Used to Define Urinary Tract Infection (UTI)

ICD-10 Code	Diagnosis Description	Category	Approx. Count
N39.0	Urinary tract infection, site not specified	UTI	8,811,652

Figures

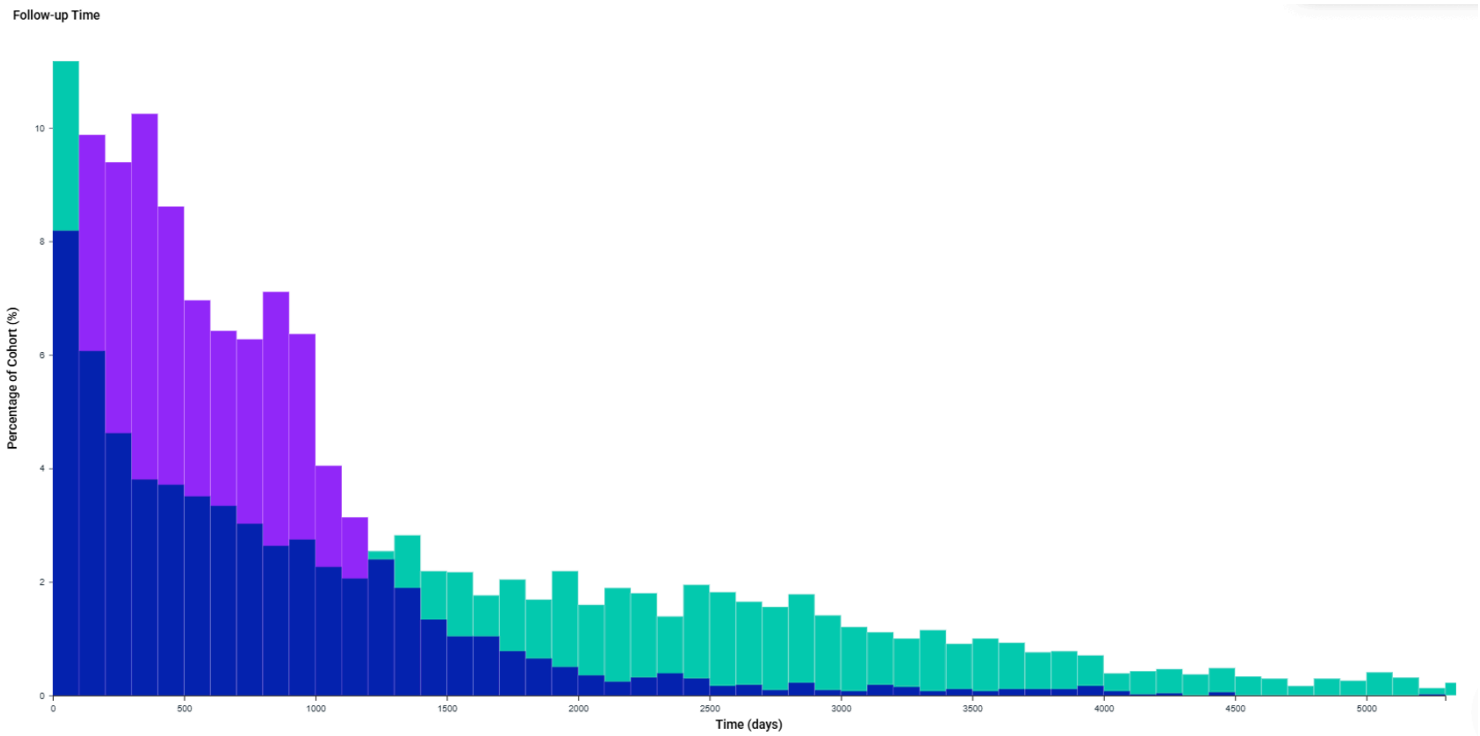


Figure 1

Mean follow up time for postpartum non-diabetic women on GLP-1RAs (purple) versus Not on GLP-1RAs (turquoise).

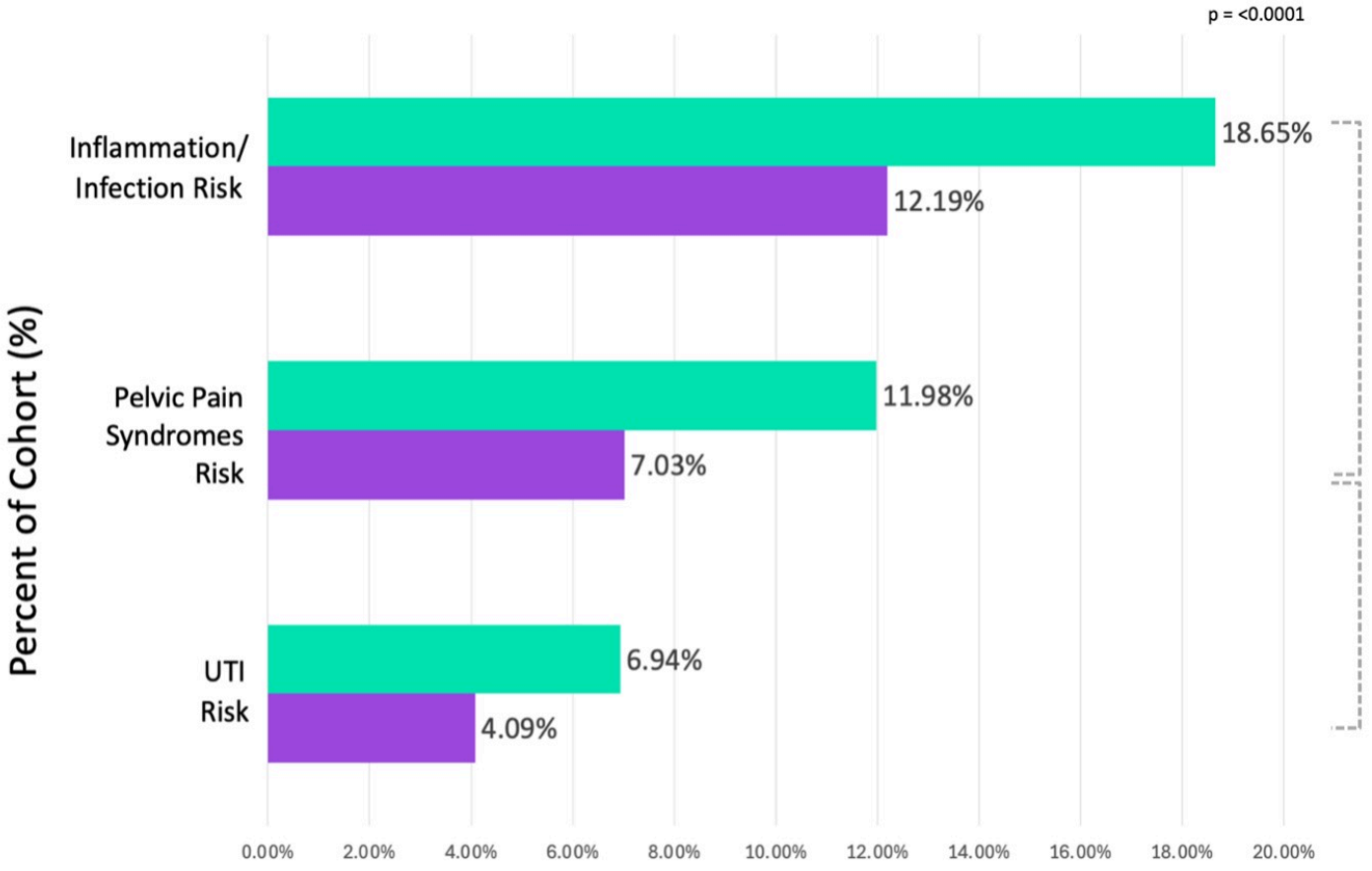


Figure 2

Risk outcomes of postpartum non-diabetic women on GLP-1RAs (purple) versus Not on GLP-1RAs (turquoise).