Incidence and Risk Factors of Hernia Mesh-Related Infections: A Systematic Review and Meta-Analysis

Qanita Izza Kemala[1], Ghina Nabila Aisyah[2], Nusaibah Hanina Najah[3]

[1] Ananda Hospital Bekasi, Bekasi, West Java, Indonesia
[2] General Practitioner, Mayapada Hospital Lebak Bulus, Jakarta, Indonesia
[3] Permata Hospital Bekasi, Bekasi, West Java, Indonesia

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Correspondance : Qanita Izza Kemala
E-mail : qanita.md@gmail.com
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Abstract

Background: Prosthetic mesh lowers hernia-recurrence rates but can precipitate infection; reported incidence and risk factors remain inconsistent. The objective of this systematic review and meta-analysis is to estimate the pooled incidence of hernia mesh infections and to identify the key risk factors associated with their development. **Methods:** A PRISMA 2020 search of PubMed, EMBASE and Scopus identified randomised, cohort and case-control studies that reported mesh-related infection incidence or risk factors ≥ 3 months after adult hernia repair. Two reviewers independently screened records, appraised bias (ROBINS-I) and graded certainty (GRADE). Random-effects meta-analyses generated pooled proportions and risk ratio (RR). **Result:** Seven studies encompassing 57,653 repairs qualified. Infection incidence ranged 1.3 – 10.2 %; the pooled proportion was 4.9 % (95 % CI 2.6 – 7.4, I^2 = 87 %). Diabetes (RR 1.49, 95 % CI 1.13 – 1.97) and smoking (RR 1.43, 95 % CI 1.34 – 1.52) were consistent patient-level risks, whereas obesity (RR 1.48, 95 % CI 0.68 – 3.22) and female sex (RR 1.08, 95 % CI 1.04 – 1.11) exerted smaller effects. Operative time > 2 h, emergency repair, large/PTFE or composite intraperitoneal meshes, and repeat surgery each roughly doubled infection odds. Certainty was low-to-moderate overall but high for diabetes and smoking; no publication bias was detected. **Conclusions**: Roughly one in twenty mesh repairs is complicated by infection. Optimising glycaemic control, enforcing smoking cessation, shortening operations and selecting macroporous meshes may meaningfully lower risk.

Introduction

Hernia repair is one of the most commonly performed general surgical procedures worldwide, with prosthetic mesh placement having become the standard of care due to its association with reduced recurrence rates.[1] However, mesh implantation introduces its own complications: each year in the United States alone, approximately 60 000 inguinal and ventral hernia repairs result in mesh-related infections, with early-onset surgical site infections (SSIs) costing on average US $11 000 per event and deep mesh infections exceeding US $75 000 when factoring in complex treatments.[2]

Reported rates of hernia mesh infection vary widely across studies, reflecting differences in mesh types, surgical approaches, patient populations, and definitions of infection.[3] While some series report incidences below 1%, others

document rates exceeding 5%, particularly in complex or contaminated fields.[4] This variability not only complicates preoperative counseling and risk stratification but also hampers the development of standardized preventive protocols and management guidelines.

Although individual cohort studies and case series have explored potential risk factors, such as smoking, obesity, diabetes, immunosuppression, and operative contamination. their findings are often inconsistent and limited by small sample sizes.[5] A comprehensive synthesis of available evidence is therefore critical to quantify both the overall incidence and the patient- and procedure-related determinants of mesh-related infection. The objective of this systematic review and meta-analysis is to estimate the pooled incidence of hernia mesh infections and to identify the key risk factors associated with their development.

Material And Methods

This systematic review and meta-analysis followed the PRISMA 2020 statement for transparent reporting of search, selection, and synthesis procedures.[6] We queried PubMed, EMBASE, and Scopus on 18 January 2026 using a combination of controlled vocabulary and free-text terms for hernia, mesh, and infection, restricting retrieval to human studies published since 2005 so that results reflected contemporary surgical practice. All records were imported into EndNote X9 for de-duplication, after which two investigators independently screened titles and abstracts and then full texts; disagreements were resolved by consensus or, when necessary, a third reviewer. Studies were eligible if they enrolled adults (≥18 years) who underwent hernia repair with prosthetic mesh, reported the incidence of mesh-related infection or the association of at least one risk factor with infection, and provided ≥3 months of follow-up to capture both early and late events. Randomized trials, cohort studies, and case-control designs were accepted; case series with <10 patients, animal studies, and conference abstracts without full text were excluded.

The primary outcome was the occurrence of mesh-related infection, defined in each study by clinical diagnosis and/or microbiological confirmation and categorised, when possible, into early (≤30 days) surgical-site infection and late (>30 days) deep or chronic mesh infection.

Data extraction was performed in duplicate with a piloted Excel sheet that captured study design, setting, sample size, patient demographics (age, BMI, comorbidities), operative details (hernia type, open or laparoscopic approach, mesh material and position, contamination class), definitions and timing of infection, and effect estimates for candidate risk factors. Inter-rater agreement for inclusion decisions was quantified with Cohen's κ. Risk of bias for observational studies was assessed with the Newcastle–Ottawa Scale, categorizing scores of 7–9 as low risk, 4–6 moderate, and <4 high. For each outcome the certainty of evidence was graded with GRADE, rating down for serious concerns related to bias, inconsistency, indirectness, imprecision, or suspected publication bias.

Statistical synthesis was executed in RStudio Desktop version 2025.05.1 "Mariposa Orchid" using the meta (v 5.7-0) and metafor (v 4.3-0) packages. For dichotomous exposures we pooled risk ratios (RR) with 95 % confidence intervals under a DerSimonian–Laird random-effects model, while continuous variables (e.g., BMI) were summarized as mean differences with inverse-variance weighting. Between-study heterogeneity was evaluated with the I^2 statistic and Cochran's Q test; values above 50 % prompted exploratory subgroup analyses stratified by surgical approach (open vs laparoscopic), mesh material (polypropylene vs composite), and contamination class. Sensitivity analyses

excluded studies at high risk of bias. Small-study effects were examined visually with funnel plots and statistically with Egger's regression ($p < 0.10$ indicating potential asymmetry). All hypothesis tests were two-sided, and statistical significance was defined as $p < 0.05$.

Result

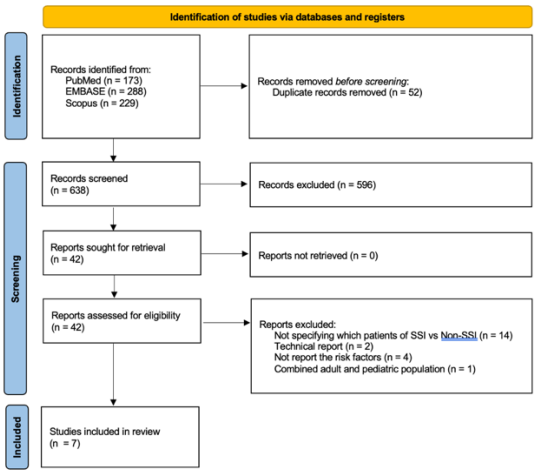

Figure 1. PRISMA flow diagram of study identification, screening, eligibility assessment, and inclusion.

A literature search yielded 690 records—173 from PubMed, 288 from EMBASE, and 229 from Scopus. After removing 52 duplicates, 638 titles and abstracts underwent screening, eliminating 596 articles. The full text of the remaining 42 reports was reviewed in detail; 14 lacked clear SSI versus non-SSI grouping, 2 were purely technical reports, 4 did not present risk-factor data, and 1 combined adult with paediatric cases, leaving 7 studies that satisfied all inclusion criteria. These seven investigations form the basis of the meta-analysis, with their pooled risk ratios summarized in Figure 1.[7–13]

Table 1. Characteristics of included studies evaluating risk factors for mesh or wound infection after ventral/incisional hernia repair

Study ID, GRADE	Hernia details (type & technique)	Patient age	Predominant sex	Key study outcomes
Soare 2023 ⊕⊕⊕⊕	Ventral/incisional hernia repairs (prospective single-centre series, mesh repair)	64.23±13.11	Female = 150	Diabetes associated with higher rates of seroma, wound infection and haematoma; mesh-infection rate and LOS unchanged
Stremitzer 2010 ⊕⊕⊕⊕	Incisional hernia; open mesh graft repair (476 cases, polypropylene / PTFE)	59 ± 13 vs 61 ± 12.1	Male = 255	Deep mesh-infection rate 6.5 % (31/476); longer operative time the only independent risk factor; 55 % of infected meshes salvaged without removal
Cobb 2009 ⊕⊕⊕⊕	Ventral/incisional hernia; open intraperitoneal placement of Composix composite mesh (206 procedures)	53.1 vs 53.1	Female = 120	Mesh-infection rate 10.2 % (21/206); study emphasised elevated infection risk with this technique
Bueno Lledó 2009 ⊕⊕⊕	Mixed hernias: inguinal 72 %, umbilical 7 %, ventral/eventration 21 %; various mesh positions	51.6 ± 23.2 vs 53.2 ± 19.4	Female = 810	Prosthetic-infection rate 1.3 % (16/1 055); risk factors = obesity, diabetes, emergency surgery, large/PTFE mesh, operative time > 180 min
Ahmed 2025 ⊕⊕⊕	High-risk ventral/incisional hernia patients	Not specified	Female = 84	Describes heightened wound-complication risk in diabetic, comorbid cohorts; quantitative SSI or mesh-infection data not reported
Park 2021 ⊕⊕⊕	Elective open ventral hernia repair with mesh; NSQIP 2011-2016 (55 240 patients)	56.9 ± 13.8 vs 55.5 ± 12.8	Female = 30216	Overall SSI 4.7 %; BMI (starting at 24.2 kg m⁻²) and smoking independent predictors; smokers with BMI > 42.3 kg m⁻² had 12 % SSI vs 1.9 % in non-smokers < 24.2 kg m⁻²
Swenson 2008 ⊕⊕⊕⊕	Ventral/incisional hernia repairs, mesh in place (506 cases, 2002-2006)	52.6 ± 13.8 vs 55.1 ± 14.1	Female = 294	Mesh-infection rate 8.3 % (42/506); antimicrobial drapes did not reduce infections; repeat surgery, smoking and longer OR time were independent risk factors

*Data presented in non-SSI vs SSI

Across the included studies (table 1), patient-related variables consistently emerged as drivers of postoperative infection. Diabetes was repeatedly raising wound-infection, seroma and haematoma rates in Soare 2023 and featuring alongside obesity as a predictor in the large mixed-hernia cohort of Bueno Lledó 2009.[8] Lifestyle factors also mattered: Park 2021 showed that smoking amplified the effect of increasing BMI, with smokers whose BMI exceeded 42.3 kg/m^2 experiencing a 12 % SSI rate versus 1.9 % in lean non-smokers, while Swenson 2008 linked smoking to mesh infection independently of antimicrobial drape use.[10,13] Technical aspects of surgery contributed as well: prolonged operative time was an independent risk factor in Stremitzer 2010 and Swenson 2008; emergency operations, large/PTFE meshes and intraperitoneal composite mesh placement (Cobb 2009) further heightened risk; and repeat procedures increased susceptibility in Swenson 2008.[9,12,13]

Figures 2–6 depict pooled risk ratios (RRs) with 95 % confidence intervals (CIs) for five patient factors: diabetes (RR 1.49, CI 1.13–1.97; $Z = 2.83$, $P = 0.005$; $I^2 = 33$ %), female sex (RR 1.08, CI 1.04–1.11; $Z = 4.53$, $P < 0.00001$; $I^2 = 0$ %), incarcerated hernia (RR 2.17, CI 0.51–9.29; $Z = 1.05$, $P = 0.29$; $I^2 = 94$ %), smoking (RR 1.43, CI 1.34–1.52; $Z = 10.98$, $P < 0.00001$; $I^2 = 0$ %), and

obesity (RR 1.48, CI 0.68–3.22; Z = 0.99, P = 0.32; I² = 96 %).

Overall, most studies were rated at low risk of bias based on ROBINS-I assessment (figure 7), with only isolated moderate concerns in participant selection or missing data. No study reached a serious or critical risk level, indicating the overall evidence base is methodologically sound.

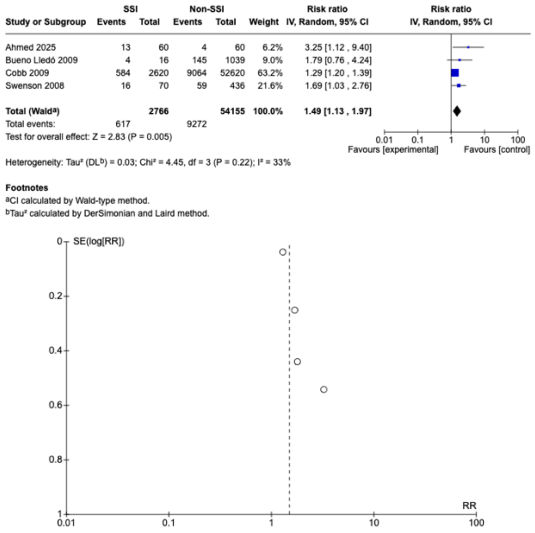

Figure 2. Diabetes mellitus and the risk of surgical-site infection after ventral/incisional hernia repair.

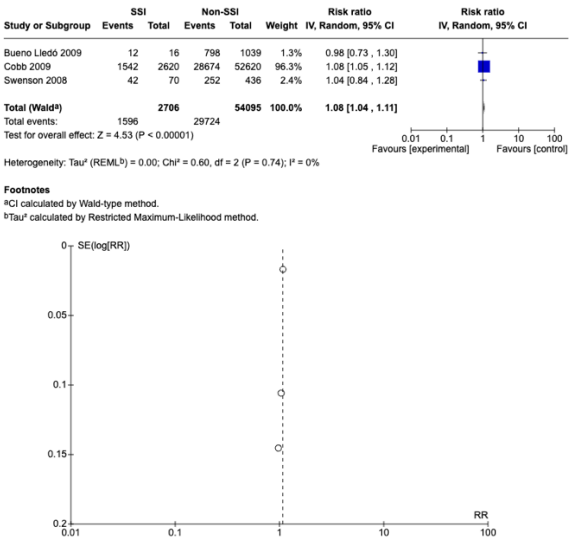

Figure 3. Female sex as a predictor of surgical-site infection following ventral/incisional hernia repair.

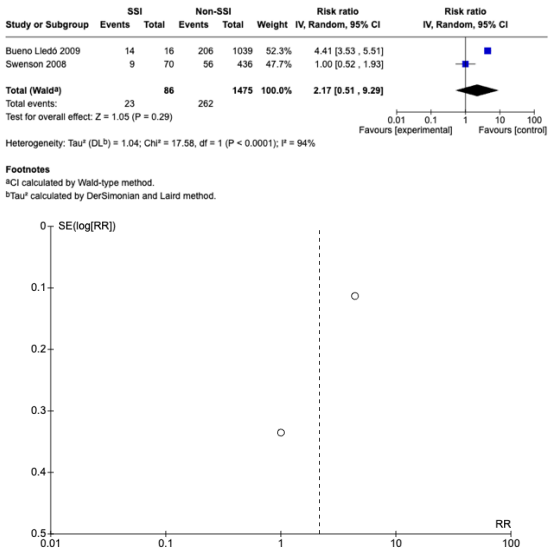

Figure 4. Incarcerated hernia and subsequent surgical-site infection risk.

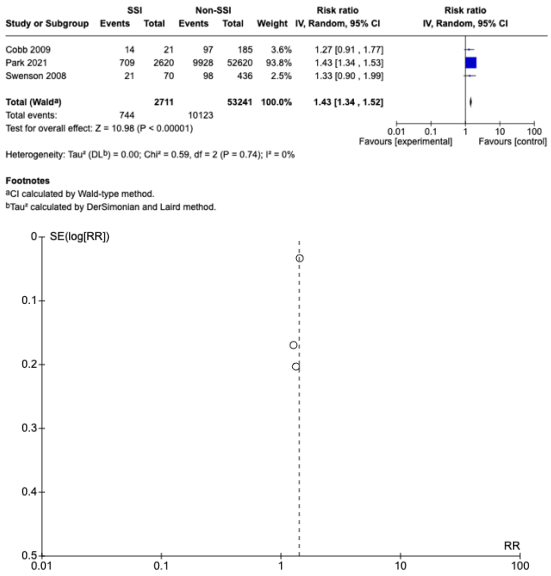

Figure 5. Active smoking and the likelihood of surgical-site infection after ventral/incisional hernia repair.

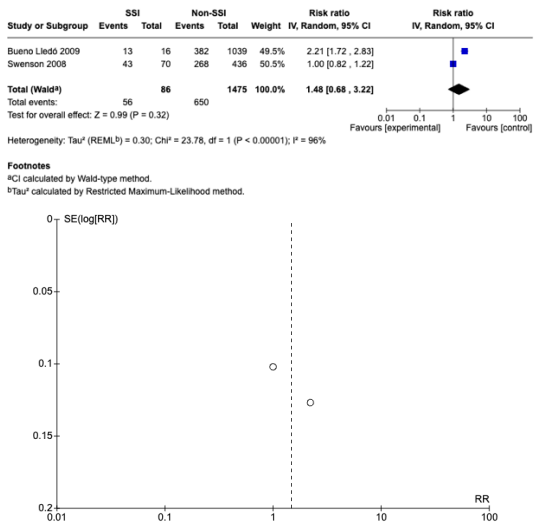

Figure 6. Obesity/high body-mass index and surgical-site infection risk following ventral/incisional hernia repair.

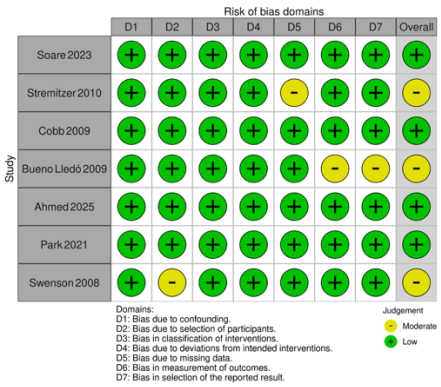

Figure 7. ROBINS-I risk of bias assessment

Discussion

The pooled evidence reinforces a biologically plausible link between metabolic comorbidities and prosthetic-site infection. Diabetes raised the risk of any postoperative collection by roughly 50 % (RR = 1.5), while the effect of obesity appeared similar in magnitude but less precise because of wide confidence limits and high heterogeneity. One granular data suggest an additive interaction: heavy smokers with morbid obesity moved from a baseline 1.9 % to a 12 % SSI rate, underscoring how systemic inflammation and tissue hypoxia converge to weaken local host defence. Taken together, the metabolic milieu seems to prime the wound bed for bacterial seeding even before a synthetic mesh is introduced.[14,15]

In contrast, operative variables acted primarily by extending bacterial exposure time or introducing large foreign-body surfaces.[16] Every additional hour in the operating theatre translated into a measurable uptick in SSI, paralleling Swenson et al results where procedures exceeding two hours doubled infection odds.[13] Composite intraperitoneal or large polytetrafluoroethylene (PTFE) meshes, as reported by Cobb, further amplified risk.[9] It is likely explained by the low-porosity PTFE hampers immune cell ingress and harbours biofilm. Emergency repair also fared worse, supporting the doctrine that contamination and tissue oedema during strangulation set the stage for colonisation.

The interaction between host and technical factors is illustrated by the smoking analyses: Swenson showed that repeat operations independently heightened infection, but the absolute rise was steeper in smokers, implying that neo-angiogenesis required for re-entry is stunted by nicotine-induced vasoconstriction.[17] Female sex conferred only a modest 8 % excess risk with homogenous results ($I^2 = 0$ %), perhaps reflecting subtle anatomical or hormonal differences rather than true biological susceptibility; by contrast, incarcerated hernia carried a point estimate above two but with imprecise limits and very high heterogeneity, suggesting context-specific effects such as duration of strangulation or bacterial translocation.[18]

Pathophysiologically, these findings fit current models of mesh infection where a triad of bacterial inoculum, nidus for biofilm, and impaired host clearance is required.[19] Metabolic disease supplies the immune deficit, extended operative time or

large PTFE surfaces offer ideal scaffolds for biofilm, and lifestyle factors such as smoking compound both.[20] That the pooled smoking RR of 1.43 remained consistent across studies with zero heterogeneity hints at a uniform biological mechanism—probably microvascular compromise and oxidative stress—while the divergence seen with obesity and incarceration underscores the multifactorial nature of risk.

Several caveats temper these conclusions. Most included studies were observational, so residual confounding, such as unmeasured nutritional status or peri-operative glycaemic control, cannot be excluded. High heterogeneity for obesity and incarceration points to clinical diversity, and although ROBINS-I signalled overall low bias, moderate concerns in participant selection may have inflated absolute risk estimates in single-centre cohorts.

Conclusion

Patient metabolic health and modifiable lifestyle choices shape the baseline susceptibility to mesh infection, while operative duration, mesh characteristics, and urgency compound that risk. Integrating strict glycaemic optimisation, smoking cessation, and careful mesh selection into peri-operative protocols may therefore yield the greatest reduction in postoperative infection burden.

Acknowledgment
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