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Review

Post-Hysterectomy Ovarian Consequences: Mechanisms, Risks, and Clinical Management Strategies—A Narrative Review

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Abstract

Objective(s): To examine the mechanisms underlying changes in ovarian function after total hysterectomy, identify relevant risk factors, and summarize clinical management strategies for such changes. **Mechanism**: The pathogenesis of impaired ovarian function post-total hysterectomy involves three key pathways: (1) reduced ovarian blood supply due to uterine artery ligation; (2) neuroendocrine imbalance caused by abnormal gonadotropin levels; (3) oxidative stress and fibrosis induced by chronic inflammation. **Findings in Brief**: Total hysterectomy is associated with diminished ovarian reserve, including a 20–30% decrease in anti-Müllerian hormone (AMH), elevated serum follicle-stimulating hormone (FSH) levels, and an approximate 3–4-year acceleration of menopause. Risk factors include the surgical approach (e.g., laparoscopic electrocoagulation decreases AMH by 40% vs. 20% with open surgery), unilateral ovarian preservation (increases the risk of menopause by 2.93-fold compared to bilateral preservation), and age <40 years (increases the risk of postoperative ovarian failure). **Conclusions**: Personalized clinical management, including preoperative assessment of AMH levels and ovarian blood flow, preference for ovarian and uterine artery-preserving techniques (e.g., STHMUV, uterine blood supply-preserving hysterectomy technique), and postoperative hormone/pelvic floor function monitoring may mitigate damage to ovarian function. To optimize long-term outcomes, future research should focus on vasoprotective strategies and precision interventions guided by biomarkers.

Keywords: hysterectomy; ovarian function; ovarian reserve; anti-Müllerian hormone (AMH); premature menopause; surgical approach

1. Introduction

1.1 Background

Total hysterectomy is one of the most important surgical procedures in many gynecologic conditions [1]. However, changes in ovarian function after surgery (such as early menopause and hormonal disorders) can have significant impacts on the reproductive and long-term health of women, including cardiovascular and cognitive issues [2,3]. There is currently a need for systematic integration of research on the mechanisms, risk stratification, and management strategies for total hysterectomy.

1.2 Objectives

The aims of this study were to elucidate the pathophysiological mechanisms underlying ovarian dysfunction following total hysterectomy, systematically evaluate the clinical evidence and epidemiological characteristics of postoperative ovarian dysfunction, identify key risk factors affecting postoperative ovarian function, and provide evidence-based management strategies for preserving ovarian function and improving long-term health outcomes after total hysterectomy.

2. Methods

Articles were identified via searches of Embase, PubMed, and Web of Science from each database's inception to June 2025, supplemented by manual screening of reference lists. The computerized search included only English-language articles, using the keyword combination: "Hysterectomy" paired with "Ovarian function" or "Ovarian Reserve", and supplemented by "premature menopause", "surgical approach", "AMH/anti-Müllerian hormone", etc.

This strategy initially yielded 383 records. After removing duplicates, we focused on screening for meta-analyses and clinical studies, while excluding abstracts (incomplete data) and review articles (non-original research); finally, 39 eligible studies were selected, categorized as follows: 7 on pathophysiologic mechanisms, 24 on clinical evidence and symptoms, and 8 on risk factors related to post-hysterectomy ovarian consequences.

3. Pathophysiologic Mechanisms of Ovarian Dysfunction After Total Hysterectomy

3.1 Decreased Blood Supply and Vascular Damage

The uterine artery contributes approximately 50–70% of the blood supply to the ovary. Multiple studies using Doppler ultrasound have reported elevated resistance in-

dex (RI) and pulsatility index (PI) post-hysterectomy, along with reduced peak flow velocity (PSV) in the ovarian arteries. These findings imply a diminished blood supply, possibly due to direct vessel injury or induction of thrombosis by the surgical procedures. Halmesmäki *et al*. (2007) [4] reported the results of a randomized controlled trial (n = 107) in which post-operative pelvic ultrasound revealed significant alterations in ovarian blood flow. Specifically, the PI showed a significant decrease (p = 0.01), potentially due to vascular dilation as a consequence of surgical tissue trauma. Lee *et al*. (2010) [5] conducted a prospective cohort study (evidence level II) comprising 32 patients who underwent hysterectomy and 21 control patients. Three months after hysterectomy with bilateral ovarian preservation, they found no significant changes in ovarian artery blood flow indices (PI, RI) using transvaginal Doppler ultrasound, and no change in anti-Müllerian hormone (AMH) level using the Enzyme-Linked Immunosorbent Assay (ELISA) method. Furthermore, no differences were found between the laparoscopically-assisted vaginal hysterectomy (LAVH) and total abdominal hysterectomy (TAH) groups. An animal model showed a 32% absolute reduction in endothelium-dependent vasodilatory function after ovariectomy, with impaired small-conductance Ca^{2+}-activated K^{+} (SK3) channels activity suggested to be the key mechanism [6].

3.2 Imbalance of Neuroendocrine Regulation

The uterus and ovaries are interconnected via the hypothalamic-pituitary-ovarian (HPO) axis and the autonomic nervous system (ANS). This regulatory balance between the HPO and ANS can be disrupted following hysterectomy. Postoperative changes include significant increases in the levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH), along with a decrease in estradiol (E2), indicating ovarian hypoplasia. These hormonal shifts are most notable 3–6 months after surgery and may result from reduced negative feedback by ovarian steroid hormones. Increased levels of sympathetic nerve activity trigger the toll-like receptor 4 (TLR4)/Nucleotide-binding oligomerization domain-like receptor protein 3 (NLRP3) inflammasome pathway, resulting in the release of Interleukin-1 beta (IL-1β),Interleukin-18 (IL-18), and other pro-inflammatory factors, thus accelerating follicular atresia. Concurrently, diminished parasympathetic inhibition intensifies the inflammatory cascade, while decreased expression of estrogen receptors (ERα/β) reduces follicular sensitivity to gonadotropins, impairing follicular development [7].

3.3 Inflammatory Response and Oxidative Stress

Surgical trauma can elicit sustained inflammatory responses, fostering a pro-aging milieu. Inflammaging, denoted by chronic low-grade inflammation, is a pivotal factor in ovarian senescence, influencing oxidative stress, fibrosis, and immune cell infiltration [8,9]. Elevated levels of ovarian oxidative stress driven by chronic inflammation can impede follicular maturation and development, thereby accelerating the depletion of ovarian reserve and egg quality [8–10]. Furthermore, chronic inflammation is associated with ovarian fibrosis, which disrupts the tissue architecture and compromises follicular growth. In addition, chronic inflammation exacerbates apoptosis and DNA damage during ovarian aging through molecular mechanisms such as activation of the NLRP3 inflammasome [7].

4. Clinical Evidence and Epidemiological Characteristics of Ovarian Dysfunction After Total Hysterectomy

4.1 Increased Risk of Early Menopause

Following a total hysterectomy, patients may enter menopause 3–4 years earlier. Siddle *et al*. (1987) [11] found that hysterectomy was associated with an earlier onset of ovarian failure compared to natural menopause (mean age: 45.4 $\pm$ 4.0 years vs. 49.5 $\pm$ 4.04 years, $p < 0.001$). Ahn *et al*. (2002) [12] also reported that hysterectomy was associated with a younger age at menopause (46.3 $\pm$ 3.0 vs. 48.1 $\pm$ 3.2 years, $p < 0.001$). Farquhar *et al*. (2005) [13] compared 257 women who underwent hysterectomy with ovarian preservation to 259 controls with intact uteri. These authors found that hysterectomy advances ovarian failure by 3.7 years, and women who had a hysterectomy entered menopause (FSH >40 IU/L) 3 years earlier than the controls (95% confidence interval [CI]: 1.5–6.0). By 5 years after surgery, 20.6% of hysterectomy patients had reached menopause (95% CI: 6.8%) compared to only 7.3% of controls ($p < 0.0001$). Women who had unilateral oophorectomy (n = 28) were more likely to have reached menopause (35.7%) within 5 years, and they also reached menopause 4.4 years earlier than women with bilateral ovarian preservation (95% CI: 0.6–7.9). In a prospective cohort study of 871 patients, Moorman *et al*. (2011) [14] demonstrated that the 4-year cumulative incidence of ovarian failure was 14.8% (60/406) in the hysterectomy group compared to 8.0% (46/465) in the control group, with an adjusted hazard ratio (HR) of 1.92 (95% CI: 1.29–2.86). These results suggest that hysterectomy is associated with an approximate 2-fold increase in the likelihood of ovarian failure.

4.2 Ovarian Reserve and Blood Supply Damage

Since the uterine artery provides 50–70% of the blood supply to the ovaries, surgical severance results in ischemic damage to these organs. Postoperative Doppler ultrasound showed elevated ovarian artery RI and PI, and decreased PSV, suggesting decreased blood supply [15]. Tapisiz *et al*. (2008) [16] examined histopathological changes in ovarian tissues after hysterectomy in a rat model. These authors observed a 50% reduction in primordial follicle number (p = 0.01), and a 300% increase in atretic follicle number (p = 0.02). Hu *et al*. (2006) [17] found that hysterectomy af-

fected the ovarian vasculature and gland function in women aged 32–45 years. A transient increase in Vmax was observed 5 days after surgery (26.47 vs. 22.00 cm/s, $p < 0.01$), followed by a decline to 17.20–17.60 cm/s at 1–3 months ($p < 0.001$). A gradual increase in PI from 1.45 to 1.77 ($p < 0.05$) was also observed, suggesting a long-term decrease in blood flow.

4.3 Changes in Serum Hormone Markers

4.3.1 Serum Anti-Müllerian Hormone

AMH is produced by small antral follicles and is the most reliable circulating marker of ovarian reserve. Following total hysterectomy, the concentration of AMH may decline by 20–30%, and even more in patients with low reserve. Atabekoğlu *et al*. (2012) [18] reported that total hysterectomy resulted in a larger decrease in the ovarian reserve, as measured by AMH level, at 4 months after surgery. Specifically, the AMH level decreased by 30% more than in the controls. A prospective cohort study by Trabuco *et al*. (2016) [19] showed that hysterectomy resulted in a significant decrease of almost 20% in the AMH level at 1 year compared with the control group. The hysterectomy group also exhibited a significantly greater fall in AMH (–40.7% vs. 20.9%, $p < 0.001$), and a higher rate of undetectability (12.8% vs. 4.7%, $p < 0.02$). The postoperative decrease in AMH was 0.77 times greater than in the control group ($p < 0.001$). The low reserve group (AMH $\leq$1.2 ng/mL) showed a greater decline in AMH in the hysterectomy group than controls (58.3% vs. 19.1%, $p < 0.003$), with 24.6% having undetectable postoperative AMH. In the high reserve group (AMH >1.2 ng/mL), the decline in AMH was not significantly different to controls (34.4% vs. –21.2%, $p = 0.06$), but AMH was still lower (0.81-fold, $p < 0.003$). In a meta-analysis of 14 studies with a total of 1457 women, Huang *et al*. (2023) [20] found significantly lower AMH levels in the hysterectomy group than the control group, with a weighted mean difference (WMD) of –0.56 (95% CI: –0.72 to –0.39, $p < 0.0001$).

4.3.2 Serum Follicle Stimulating Hormone

Serum FSH levels are significantly elevated in patients after hysterectomy. Huang *et al*. (2023) [20] reported significantly elevated FSH levels in the hysterectomy group compared to the control group (WMD = 2.96, 95% CI: 1.47–4.44, $p < 0.001$). Maiti *et al*. (2018) [21] found that 1 year after hysterectomy, patients had significantly increased serum FSH from baseline (7.5 IU/L to 12.3 IU/L), as well as a significant ($p < 0.05$) decrease in ovarian volume and PI, suggesting decreased blood flow with atrophy. After 1 year, 20% had FSH >20 IU/L, indicating perimenopausal transition, while 5% had FSH >40 IU/L, indicating menopausal transition. Cooper and Thorp (1999) [22] reported a strong association between hysterectomy and elevated serum FSH level (>20 IU/L). Patients with unilateral oophorectomy had 2.4-fold higher odds of elevated FSH (>20 IU/L) compared to those with bilateral ovarian preservation (OR = 2.4, 95% CI: 1.3–4.6).

4.3.3 Serum Inhibin B

Inhibin B is secreted by growing follicles and negatively regulates pituitary FSH secretion, with its decline indicating a diminished follicular pool. Studies have consistently shown that inhibin B levels are significantly reduced after hysterectomy. A meta-analysis conducted by Huang *et al*. (2023) [20] revealed the test group showed a decrease of 14.34 pg/mL (95% CI: –24.69 to –3.99, $p < 0.001$) in the level of inhibin B, aligning with changes in AMH and indicating a decline in follicular reserve. Tapisiz *et al*. (2008) [16] also found that inhibin B was significantly lower in the hysterectomized group than in the control group ($p = 0.007$), reflecting diminished ovarian feedback inhibition, as demonstrated by experiments in rats. A randomized controlled trial by Halmesmäki *et al*. (2007) [4] involving 107 participants found that serum inhibin B levels were significantly decreased ($p < 0.05$), with hysterectomy emerging as an independent predictor ($p = 0.05$) in multivariate regression analysis. Nahás *et al*. (2003) [23] conducted a prospective case-control study with 61 participants in the hysterectomy group and 30 in the control group. They found a significant decline in serum inhibin B levels post-TAH, with median inhibin B levels decreasing notably at 6 and 12 months ($p < 0.05$). Some patients experienced biochemical failure, defined as FSH >40 mIU/mL and E2 <20 pg/mL. Post-surgery, a percentage of patients exhibited FSH >40 mIU/mL, E2 <20 pg/mL, and inhibin B <5 pg/mL at the 12-month mark.

5. Clinical Symptoms and Long-Term Health Risks of Ovarian Dysfunction After Total Hysterectomy

5.1 Vasomotor Symptoms

Women have a higher persistence of hot flashes and night sweats following hysterectomy. A prospective observational study by Maiti *et al*. (2018) [21] found that 34% of patients developed menopausal symptoms within one year following hysterectomy. The observed symptoms were somatic (30% of cases), psychological (19%), and genitourinary (12%) in nature. A longitudinal study of 6106 women over 17 years found that those with a history of hysterectomy had higher incidences of persistent hot flashes (30% vs. 15%) and night sweats (19% vs. 9%) than women without hysterectomy. Moreover, women with a history of hysterectomy had higher rates of persistent hot flashes (1.97%, 95% CI: 1.64–2.35) and persistent night sweats (2.09%, 95% CI: 1.70–2.55) compared to those without hysterectomy [24].

5.2 Genitourinary Symptoms

Several studies have found that about one-third of post-hysterectomy patients subsequently develop genitouri-

nary and vaginal prolapse problems. Following hysterectomy, many patients experience lower urinary tract dysfunction (LUTD) caused by synergistic dysfunction of the bladder detrusor muscle with the urethral sphincter due to pelvic autonomic nerve injury. One study found that about a third of post-hysterectomy patients develop genitourinary issues and vaginal prolapse [25]. Patients may experience urinary frequency, dysuria (56–64%), urgency and/or stress urinary incontinence (37–60%), and incomplete bladder emptying (36.7%) [26]. Compared to standard hysterectomy, radical hysterectomy has a higher incidence of urinary complications (odds ratio [OR] = 15.63, p = 0.001), residual urine sensation (OR = 10.37, $p < 0.001$), and urinary tract infections (OR = 6.22, $p < 0.001$). Radical hysterectomy is also associated with a higher incidence of urodynamic problems (max flow rate: 19.76 mL/s with standard hysterectomy vs. 12.35 mL/s with radical hysterectomy), increased residual urine volume (57.69 mL vs. 221.28 mL), and abnormal urinary sensation (presence of sensation: 93.8% vs. 43.9%) [27]. In a prospective cohort study by Proshchenko and Ventskivska (2022) [28] involving 160 women aged 40–49 years, the incidence of stress urinary incontinence increased from 13.75% before surgery to 41.02% at 5 years post-surgery ($p < 0.05$). Abdominal hysterectomy resulted in less favorable urodynamic outcomes, including a larger reduction in bladder capacity (1.5-fold decrease), higher residual urine volume (+32%), and increased rates of voiding dysfunction compared with vaginal or laparoscopic approaches [28].

5.3 Cardiovascular Disease

Hysterectomy has been associated with a 27% reduction in carotid artery compliance (p = 0.004), independent of traditional cardiovascular risk factors [29]. A matched case-control study (n = 246; 123 matched pairs) conducted by Punnonen *et al.* (1987) [8] found that premenopausal hysterectomy tripled the risk of cardiovascular disease relative risk [RR] = 3.0, significant in McNemar test) compared to myomectomy controls. Notably, hypertension was more prevalent among hysterectomy cases (6/20) than controls (1/6). A nationwide cohort study by Lai *et al.* (2018) [9] on 4986 women, including 1083 bilateral salpingo-oophorectomy (BSO) cases, found that undergoing BSO during hysterectomy did not significantly increase the overall risk of stroke during a 13-year follow-up (HR = 0.84, 95% CI: 0.63–1.13). However, BSO decreased the risk of stroke by 64% (HR = 0.36, 95% CI: 0.16–0.79) in women aged 50 years or older who were using estrogen therapy. This protective effect in older women receiving estrogen therapy suggests that hormonal compensation may attenuate the cardiovascular risks following BSO, offsetting the effects of surgical menopause. No increase in risk was observed for either ischemic stroke (HR = 0.85, 95% CI: 0.61–1.18) or hemorrhagic stroke (HR = 0.82, 95% CI: 0.42–1.60). Analysis of the Nurses' Health Study data by Parker *et al.* (2009) [30] revealed a 17% increase in the risk of coronary heart disease among women who had undergone hysterectomy (HR = 1.17, 95% CI: 1.02–1.35), irrespective of ovarian status. Gavin *et al.* (2012) [29] examined the relationship between hysterectomy (with or without bilateral oophorectomy) and large artery stiffness. Both hysterectomy alone and hysterectomy with bilateral oophorectomy were found to be associated with increased arterial stiffness, as indicated by reduced carotid compliance, and independently of traditional cardiovascular risk factors.

5.4 Dementia

Phung *et al.* (2010) [10] conducted a nationwide historical cohort study (n = 2,313,388) to investigate the association between hysterectomy (with or without oophorectomy) and the risk of dementia. Their analysis revealed that hysterectomy was associated with an elevated risk of early-onset dementia (diagnosed before age 50), with the risk increasing progressively according to the extent of surgery: hysterectomy alone (RR = 1.38, 95% CI: 1.07–1.78), hysterectomy with unilateral oophorectomy (RR = 2.10, 95% CI: 1.28–3.45), and hysterectomy with bilateral oophorectomy (RR = 2.33, 95% CI: 1.44–3.77). Notably, the magnitude of risk exhibited a strong inverse relationship with age at surgery, with younger patients having a disproportionately higher risk.

6. Risk Factors for Ovarian Dysfunction After Total Hysterectomy

6.1 Impact of the Surgical Procedure

6.1.1 Opportunistic Bilateral Salpingectomy vs. Standard Hysterectomy

Most studies have shown that simultaneous removal of the fallopian tubes during hysterectomy (opportunistic salpingectomy (OS) or prophylactic bilateral salpingectomy (PBS)) does not cause significant acute damage to ovarian reserve markers (e.g., AMH, FSH, LH). Behnamfar and Jabbari (2017) [31] compared combined BSO in hysterectomy with tubal preservation group, finding significantly higher FSH and LH ($p < 0.001$), but no difference between groups (FSH: p = 0.17; LH: p = 0.16). A retrospective cohort study (n = 79) by Chen *et al.* (2022) [32] found that hysterectomy with OS significantly reduced the time to menopause (1.84 vs. 2.93 years, p = 0.031; p = 0.029 after adjusting for covariates). The OS group also had higher body mass index (BMI) (25.27 vs. 22.97 kg/m^2, p = 0.01) and sleep disturbances (41% vs. 12%, p = 0.01). Tehranian *et al.* (2017) [33] conducted a randomized controlled trial on the effect of simultaneous salpingo-oophorectomy during hysterectomy on ovarian reserve (n = 30). All patients showed a significant postoperative decrease in AMH (1.32 $\pm$ 0.91 to 1.05 $\pm$ 0.88 ng/mL, $p < 0.001$), but no significant difference was found in the rate of decrease in AMH between the two groups (25% in salpingo-oophorectomy group vs. 26% in the control group, p = 0.23). A systematic

review and meta-analysis of 9 studies by Gelderblom *et al.* (2022) [34] found that OS at the end of pregnancy did not affect ovarian reserve markers. In particular, there was no significant decrease in AMH at 3 months after salpingectomy ($p = 0.21$). Van Lieshout *et al.* (2018) [35] conducted a Cochrane systematic review and found that combined OS during hysterectomy did not significantly increase the surgical risk or impair ovarian function. A prospective cohort study (n = 60) by Naaman *et al.* (2017) [36] found that hysterectomy with bilateral salpingectomy or fimbriectomy did not significantly affect ovarian reserve, as evidenced by changes in AMH (+0.53 vs. –0.02 ng/mL, $p = 0.25$), FSH (–2.53 vs. –7.20 IU/L, $p = 0.30$), and Doppler ultrasound parameters (all $p > 0.05$). A recent multicenter randomized controlled trial (n = 104) by Van Lieshout *et al.* (2019) [37] found that hysterectomy with opportunistic bilateral salpingectomy did not significantly impact ovarian reserve compared to standard hysterectomy. The study reported a non-significant difference in AMH levels between the two groups (change in AMH: –0.14 vs. 0.00 pmol/L, $p = 0.49$). A prospective observational study (n = 71) conducted by Venturella *et al.* (2017) [38] found that PBS in total laparoscopic hysterectomy (TLH) did not affect the long-term ovarian reserve 3 years postoperatively (OvAge vs. chronological age: 49.22 $\pm$ 2.57 vs. 49.61 $\pm$ 2.15 years, $p = 0.900$). The OvAge® model for AMH (0.12 $\pm$ 0.20 ng/mL), FSH (44.30 $\pm$ 219.92 mU/mL) and 3D-AFC (1.91 $\pm$ 1.28) showed equivalent slopes in the PBS and control groups (r = 1.0008, $p = 0.001$).

6.1.2 Total Laparoscopic Hysterectomy (TLH) vs Laparoscopic Supracervical Hysterectomy (LSH)

The prospective cohort study (n = 67 patients) by Yuan *et al.* (2015) [39] revealed that TLH caused a greater decline in serum AMH levels than LSH at 4 months postsurgery ($p = 0.017$).

6.1.3 Bilateral Ovarian Preservation vs. Unilateral Ovarian Preservation

Women with bilateral ovarian preservation show a significantly higher 5-year rate of normal ovarian function than those with unilateral preservation (89% vs. 66%). Intraoperative preservation of both ovaries is therefore recommended as a priority. For women requiring unilateral ovarian removal, postoperative monitoring of AMH should be performed every 6 months for early detection of functional decline. A prospective randomized study by Bukovsky *et al.* (1995) [40] found that abdominal hysterectomy with unilateral oophorectomy (USO) resulted in a higher dysfunction rate (35% vs. 10%, $p = 0.02$) at the 6-month follow-up assessment compared to ovarian conservation. A prospective cohort study by Farquhar *et al.* (2005) [13] involving 257 women in the hysterectomy group and 259 controls found the 5-year menopausal rate was significantly higher in women who retained one ovary (35.7%, 10/28) compared to those retaining both ovaries (16.9%, $p < 0.01$). A prospective cohort study by Moorman *et al.* (2011) [14] involving 406 individuals in the hysterectomy group and 465 controls found that USO was associated with a higher risk (HR = 2.93) compared to women who retained bilateral ovaries (HR = 1.74).

6.1.4 Laparoscopic vs. Non-Laparoscopic Surgery

Laparoscopic hysterectomy has been associated with substantial short-term impacts on ovarian reserve function, possibly due to the thermal effects of electrocoagulation during the procedure. In contrast, open surgical approaches or techniques that preserve the ovarian blood supply may offer superior protection of ovarian function. A prospective cohort study by Chun and Ji (2020) [41] examined the impact of hysterectomy with ovarian preservation on ovarian reserve in 86 premenopausal women aged 31–48 years. The results showed differential effects during the early postoperative period depending on the surgical approach. While the laparoscopic group experienced a greater reduction in AMH level (0.42 ng/mL) compared to the open group (0.01 ng/mL), this did not reach statistical significance ($p = 0.053$). Cho *et al.* (2017) [42] prospectively monitored the AMH level in 91 individuals and found no significant difference in the rate of decline between TLH and non-TLH groups at 6 months postoperatively (TLH 42.1% vs. non-TLH 33.3%, $p = 0.545$). However, the TLH group exhibited a sustained decrease in the mean AMH value (3.5 to 1.6 ng/mL), whereas the AMH level remained relatively stable in the non-TLH group (2.4 to 2.6 ng/mL). The systematic review and meta-analysis of 9 studies by Gelderblom *et al.* (2022) [34] found a significant decline of more than 40% in the AMH level at 2 months postoperatively in the TLH group ($p = 0.042$), compared to a 20% decline in the non-TLH group. This difference may be attributable to thermal damage from the electrocoagulation equipment used in laparoscopic procedures, which can adversely impact ovarian tissues or blood vessels through heat diffusion. A randomized controlled trial (n = 100) conducted by Cai *et al.* (2017) [43] compared traditional hysterectomy with a novel hysterectomy technique that preserves the uterine blood supply (STHMUV, uterine blood supply-preserving hysterectomy technique). Superior ovarian protection was observed with the STHMUV technique, which maintained stable postoperative estradiol (E2) levels (346.12 pg/mL to 298.34 pg/mL) over 2 years ($p > 0.05$). In contrast, traditional hysterectomy exhibited a significant decline in E2 (343.24 pg/mL to 203.17 pg/mL, $p < 0.05$), and significantly lower E2 levels compared to STHMUV. Furthermore, the STHMUV group showed a significantly smaller increase in FSH (17.65 U/L to 20.17 U/L) compared to the traditional hysterectomy group (16.32 U/L to 89.01 U/L, $p < 0.05$).

6.2 Patient Characteristics

6.2.1 Age

Younger patients (<40 years) have a higher risk of postoperative ovarian failure and a stronger association with surgery. Older patients (≥40 years) also have a significantly higher risk of ovarian failure (HR = 1.79) after hysterectomy. However, their risk is lower than for the younger group, probably because their ovarian reserve is already in decline approaching the age of natural menopause, and hence the 'extra blow' of surgery is relatively limited. Huang *et al*. (2023) [20] conducted a systematic review and meta-analysis of 14 studies with 1457 premenopausal women. Their analysis revealed that women aged >40 years exhibited greater increases in FSH and LH levels, and more significant decreases in E2 concentrations, compared to their younger counterparts. However, the reduction in AMH did not show any significant age-related effects, whereas some studies have suggested the decline in AMH levels following hysterectomy is more pronounced in younger patients. For instance, a study by Yuan *et al*. (2019) [44] involving 84 participants found a stronger negative correlation between hysterectomy and AMH levels in patients aged <40 years (r = –0.48 at 6 weeks, $p < 0.001$). Additionally, a prospective cohort study by Moorman *et al*. (2011) [14] involving 406 hysterectomy patients and 465 controls reported a stronger association between hysterectomy and ovarian failure in women aged <40 years (HR = 4.29, 95% CI: 0.83–22.3). While the risk of ovarian failure was also significantly elevated in the ≥40 years group, the magnitude was lower (HR = 1.79, 95% CI: 1.18–2.71), likely due to the wider confidence intervals resulting from a limited sample size.

6.2.2 Smoking

Cooper and Thorp (1999) [22] reported the impact of hysterectomy on FSH levels (OR = 1.5) was less pronounced than that of smoking (OR = 2.0), but significantly greater than the natural aging process. This finding underscores the importance of incorporating the effect of hysterectomy on the FSH level into postoperative management strategies.

7. Clinical Management Strategies for Ovarian Dysfunction After Total Hysterectomy

The management of post-hysterectomy ovarian hypoplasia and associated complications requires a full-cycle approach, incorporating detailed preoperative evaluation, optimized surgical techniques, and extended postoperative monitoring. The following evidence-based strategy is recommended:

7.1 Preoperative Assessment of Ovarian Function

In patients of childbearing age or those concerned about endocrine function, preoperative testing of AMH, FSH, and E2 should be conducted to evaluate the risk of postoperative ovarian failure. The factors of age, BMI, and smoking history should also be considered. Age >40 years and AMH levels below 1.2 ng/mL were identified as risk factors for significant postoperative ovarian function decline [45]. When necessary, three-dimensional Doppler ultrasound should be used to assess ovarian blood flow (PI, RI), with increased risk of functional decline observed in patients with abnormal blood flow (PI >1.77, RI >0.8).

7.2 Surgical Options

7.2.1 Ovary Preservation

In the absence of clear ovarian pathology, bilateral preservation is favored (89% vs. 66% for unilateral preservation 5 years postoperatively) [13]. Patients undergoing unilateral oophorectomy should be informed of the 2–3-fold increased risk of postoperative POI (HR = 2.93) [14].

7.2.2 Salpingectomy Decision

Opportunistic Salpingectomy (OS) does not cause significant acute damage to ovarian function, but may shorten the time to menopause (1.84 years in the OS group vs. 2.93 years in the preserved group) [32,34], and should thus be considered in the context of the patient's age and reproductive needs. When tubal resection is required, fine dissection should be used to avoid damage to the ovarian mesosalpinx vessels. AMH is monitored postoperatively until it stabilizes, usually after 3–6 months.

7.2.3 Surgical Extent

In benign conditions, extrafascial subtotal resection is favored over radical resection to minimize the decrease in AMH level ($p = 0.001$) and to maintain the ovarian blood supply provided by the uterine artery, which normally contributes 50–70% of the total supply [34].

7.2.4 Laparoscopic Surgery

Careful use of electrocoagulation equipment is required, with preference given to cold knife separation or low-power modes of energy instrumentation to minimize ovarian damage from heat spread [34,43].

7.2.5 Vascular-Preserving Techniques

Surgical techniques that preserve the uterine vasculature, such as the STHMUV procedure, are recommended to maintain postoperative estrogen homeostasis. These modified approaches result in a less pronounced decrease in estradiol levels (<15%) compared to conventional methods (>40%), which is a desirable outcome [43].

7.3 Postoperative Monitoring and Management

In line with the 2022 European Society of Human Reproduction and Embryology (ESHRE) Guidelines on the management of premature ovarian insufficiency [46], as well as the expert consensus [20], the levels of AMH, FSH and E2 should be monitored postoperatively. Intervals should be shortened for patients who undergo laparoscopic hysterectomy, or who retained only one ovary. Annual evaluation should focus on FSH levels that exceed 40 IU/L (indicative of menopausal status), as well as the presence of perimenopausal symptoms such as hot flashes and vaginal dryness. Particular consideration should be given to interventions for individuals who experience early-onset menopause (<40 years of age).

Postoperative screening of pelvic floor function, including urodynamics, is recommended from 6 months onward in patients undergoing transvaginal surgery or radical resection. These should have careful monitoring for vaginal prolapse (37.8% incidence) and urethral dysfunction such as stress urinary incontinence (41% incidence at five years postoperatively).

8. Conclusions

Post-hysterectomy ovarian function undergoes significant changes, including diminished ovarian reserve, menstrual alterations, and early menopausal symptoms. The impact of surgical techniques and adjunct procedures on ovarian function is varied, and can influence the patients' quality of life and psychological well-being. Clinicians should consider factors such as age, fertility desires, and disease status when selecting surgical methods and devising treatment plans. Surgical benefits and drawbacks must be balanced with ovarian function to tailor patient-specific strategies. Careful monitoring and management of postoperative ovarian function are crucial to promptly address issues, thereby improving the quality of life and health of patients. Future research should focus on personalized surgical designs such as vascular refinement protection, novel biomarkers including inflammatory factor profiles, and targeted interventions such as antifibrotic drugs. Advances in these areas should help to refine clinical management and improve long-term patient outcomes.

Abbreviations

AMH, anti-Müllerian hormone; FSH, follicle-stimulating hormone; STHMUV, uterine blood supply-preserving hysterectomy technique; HPO, hypothalamic-pituitary-ovarian; ANS, autonomic nervous system; PI, pulsatility index; RI, resistance index; PSV, peak flow velocity; LAVH, laparoscopically-assisted vaginal hysterectomy; TAH, total abdominal hysterectomy; LUTD, lower urinary tract dysfunction; OS, opportunistic salpingectomy; PBS, prophylactic bilateral salpingectomy; BSO, bilateral salpingo-oophorectomy; LH, luteinizing hormone; E2, estradiol; USO, unilateral oophorectomy; TLH, total laparoscopic hysterectomy; LSH, laparoscopic supracervical hysterectomy.

Author Contributions

YC conceptualized and designed the review, conducted comprehensive literature search and selection, and compiled and synthesized the relevant data. LM not only provided critical advice on data collation and interpretation of the review findings, but also took the lead in formulating literature inclusion and exclusion criteria, participated in the quality assessment of included literature, and constructed the core argumentation. Both authors contributed to editorial changes in the manuscript. Both authors read and approved the final manuscript. Both authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

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