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Risk Factors of Adolescent Endometriosis Onset and Recurrence: A Retrospective Study

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Purpose: This study aimed to identify adolescent-specific risk factors for endometriosis onset and postoperative recurrence to facilitate early identification and risk stratification.

Patients and Methods: A retrospective study analyzed adolescents (10–19 years) undergoing diagnostic laparoscopy for suspected benign gynecological conditions at Zhejiang University Women's Hospital (2002–2024). Cases were 242 laparoscopy-confirmed EMs patients; controls were 242 age-matched laparoscopy-negative adolescents. Comprehensive clinical/laboratory variables were collected. Multivariable logistic regression identified onset risk factors. Cox regression analyzed recurrence predictors in followed EMs patients, with recurrence defined by symptomatic, imaging, or surgical confirmation.

Results: Dysmenorrhea (aOR 3.61; 95% CI 1.97–6.63), genital tract malformation (aOR 16.15; 95% CI 3.29–79.31), and elevated CA125 (aOR 1.02; 95% CI 1.01–1.04) were independent risk factors of the onset of adolescent endometriosis. Hyperfibrinogenemia (aOR 2.13; 95% CI 1.17–3.89) and dyslipidemia (hypotriglyceridemia, hypercholesterolemia, low LDL) showed significant associations. Among followed patients (median follow-up 56 months), recurrence rate was 25.3% (23/91). Significant predictors included larger ovarian cyst diameter (aHR 1.24; 95% CI 1.03–1.49), elevated CA125 (aHR 1.01; 95% CI 1.00–1.01), and shortened activated partial thromboplastin time (APTT) (aHR 0.77; 95% CI 0.60–0.99), indicating intrinsic hypercoagulation.

Conclusion: This study identifies dysmenorrhea, genital tract malformations, and elevated CA125 as independent risk factors for adolescent EMs onset, with hypercoagulability and metabolic disturbances as key associations. Beyond traditional recurrence markers (cyst size, CA125), shortened APTT emerges as a novel predictor of postoperative recurrence. These findings underscore the need for enhanced screening in high-risk adolescents and intensified postoperative monitoring, particularly for those with coagulation abnormalities.

Keywords: endometriosis, adolescent, recurrent endometriosis, dysmenorrhea, risk factors

Introduction

Endometriosis (EMs)[1] is a chronic gynecological disorder characterized by the presence of endometrial-like tissue outside the uterine cavity, leading to chronic pelvic pain and infertility. Globally, it affects 5–10% (approximately 176 million) of reproductive-aged women, imposing a substantial public health burden.[2] Critically, EMs impacts females across their entire lifespan—spanning premenarche, adolescence, adulthood, and even extending into post-menopause.

Adolescence (defined by WHO as ages 10–19 years) represents a critical period of reproductive maturation involving rapid physiological changes, particularly rising estrogen levels.[3] These hormonal shifts may potentiate EMs pathogenesis. Substantial evidence[4–7] indicates that symptoms in adult EMs patients—notably dysmenorrhea[8]—often originate during adolescence, suggesting disease roots traceable to this developmental stage. Compared to adult presentations, adolescent EMs exhibits greater clinical complexity and non-specific symptomatology, predominantly featuring chronic pelvic pain,

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dysmenorrhea, and gastrointestinal disturbances.[9] Alarmingly, diagnostic delays in adolescents exceed those in adults by >3-fold due to under-recognition of dysmenorrhea severity among patients and caregivers.[10] Such delays not only preclude timely intervention—risking ovarian dysfunction and compromised fertility—but also inflict profound psychosocial repercussions including academic impairment, social withdrawal, heightened anxiety/depression prevalence, and altered reproductive outlooks during this developmentally vulnerable period.[11]

Postoperative recurrence poses a significant challenge in adolescent endometriosis, with cumulative 5-year rates reaching approximately 20–30%[12,13]—comparable to the 20–40% observed in adults.[14,15] While the absolute risk is similar, the clinical stakes for adolescents are uniquely higher due to their extended reproductive window, which exposes them to decades of potential recurrence and repeated interventions. However, uncritically extrapolating adult-derived predictors to this population is clinically inappropriate due to distinct developmental biology. Adolescent disease typically presents as active, angiogenic "red lesions" that differ fundamentally from the fibrotic lesions common in adults.[16,17] Consequently, adult risk stratification models often show poor predictive value in this age group.[13] Furthermore, adolescents are systematically excluded from most clinical trials[18] leaving guidelines reliant on expert opinion rather than age-specific evidence. Critically, research dedicated to recurrence dynamics in adolescents remains profoundly inadequate, compromising the development of targeted management protocols.

To address these critical knowledge gaps, this large-scale retrospective study systematically investigates modifiable determinants of EMs onset in adolescents, longitudinally tracks post-interventional recurrence patterns, and establishes age-specific risk predictors, thereby informing evidence-based surveillance frameworks for this underserved population.

Materials and Methods

IRB Approval Statement

This study complied with the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Women's Hospital, Zhejiang University School of Medicine (IRB-20240034-R). All patients and their legal guardians provided written informed consent for the surgical procedures and the use of their clinical data for research purposes at the time of admission. Given the retrospective nature of the study, the committee waived the requirement for additional individual informed consent.

Patients

This study employed a two-stage hybrid design. First, a case-control analysis was conducted to identify risk factors for the onset of adolescent endometriosis by comparing confirmed cases with age-matched controls. Second, a retrospective cohort analysis was performed on the endometriosis case group to longitudinally evaluate predictors of postoperative recurrence. We enrolled adolescents aged 10–19 years who underwent diagnostic laparoscopy for suspected benign gynecological conditions (eg., ovarian cysts, endometriosis, reproductive tract anomalies) at Women's Hospital, Zhejiang University School of Medicine between 2002 and 2024. Adolescents with laparoscopically confirmed typical endometriotic lesions (including peritoneal, ovarian, and/or deep infiltrating endometriosis [DIE]) were classified as the EMs case cohort. Age-matched controls were randomly selected at a 1:1 ratio from laparoscopy-negative counterparts without clinical evidence of endometriosis.

Exclusion criteria encompassed: (1) Confirmed pregnancy at admission; (2) Incomplete medical documentation; (3) Comorbid gynecological conditions potentially contributing to chronic pelvic pain (eg, adenomyosis, pelvic inflammatory disease); (4) Malignancy diagnosis; (5) Use of hormonal therapies within 3 months preceding admission.

Variables

Comprehensive clinical variables were systematically abstracted from medical records through standardized data collection protocols. Key parameters encompassed demographic parameters (age and body mass index), detailed menstrual history documentation (including menarche age, quantified menstrual flow volume categorized as light/moderate/heavy, cycle regularity patterns, prior hormonal medication exposure, and genital tract malformation), multidimensional dysmenorrhea characterization (frequency quantification, anatomical distribution mapping, and qualitative pain

descriptors), and comprehensive risk factor profiling (familial endometriosis history, sexual activity status, and substance exposure history including tobacco/alcohol consumption). All laboratory biomarkers—specifically coagulation profiles, serum cancer antigen 125 (CA125) concentrations, and metabolic parameters—were retrospectively retrieved via the institutional clinical data repository.

Surgery and Fellow-Up

This study employed standardized laparoscopic procedures tailored to intraoperative pathology: peritoneal endometriotic lesions underwent systematic electrocoagulation; ovarian endometriomas were universally resected via cystectomy, with histological confirmation of endometriotic cysts in all specimens; and deep infiltrating endometriosis (DIE) cases required adhesiolysis combined with targeted lesion electrocoagulation. Adolescents with surgically confirmed endometriosis entered a structured postoperative surveillance protocol. Comprehensive intraoperative parameters—including endometriosis phenotype classification, endometrioma dimensions (maximal diameter), laterality, multifocality, pouch of Douglas obliteration status, and revised American Society for Reproductive Medicine (rASRM) staging—were systematically abstracted from operative records. Postoperative monitoring incorporated longitudinal assessment through outpatient follow-up documentation and serial imaging to evaluate adjuvant medical therapy (specific agents and duration) and recurrence outcomes. Aligning with current clinical practice guidelines[19] and established international terminology[20,21] endometriosis recurrence encompasses various established categories: symptomatic recurrence, imaging-confirmed recurrence, and recurrence verified by laparoscopic visualization. For analytical purposes in our investigation, the detection of any single recurrence category constituted a recurrence event.

Statistical Analysis

All statistical analyses were performed using the R software (version 4.4.1) along with MSTATA software (www.mstata.com). Categorical variables are presented as frequencies and percentages, while continuous variables with non-normal distributions are expressed as medians with interquartile ranges (IQR).

Univariate logistic regression was initially employed to identify potential factors associated with primary endometriosis diagnosis in adolescents. Following assessment for multicollinearity among independent variables, significant predictors were entered into multivariate logistic regression modeling using backward stepwise selection. Similarly, Cox proportional hazards regression was implemented to evaluate predictors of endometrioma recurrence. After confirming proportionality assumptions, variables demonstrating univariate significance underwent backward stepwise selection in the final multivariable Cox model. Both regression equations incorporated covariates achieving statistical significance at $\alpha = 0.05$ in the final models.

Results

Demographic and Clinical Characteristics

A retrospective cohort of 1240 adolescents (aged 10–19 years) underwent diagnostic laparoscopy between 2002 and 2024. After applying exclusion criteria, 1119 patients were included in the base cohort (Figure 1). Systematic review of laparoscopic records identified 242 cases with macroscopically confirmed endometriosis lesions, classified as follows: 152 cases (62.8%) of isolated peritoneal endometriosis, 19 cases (7.9%) of ovarian endometriosis, 49 cases (20.2%) of concomitant peritoneal and ovarian lesions, 4 cases (1.7%) of ovarian endometriomas with deep infiltrating endometriosis (DIE), and 18 cases (7.4%) of multifocal disease involving peritoneal, ovarian, and DIE subtypes. These 242 patients constituted the case cohort.

Using age-matched (1:1) sampling from the remaining 877 endometriosis-negative adolescents without clinical symptoms of endometriosis, 242 controls were randomly selected. Baseline characteristics of both cohorts are presented in Table 1.

Comparative analysis demonstrated that adolescents with endometriosis exhibited significantly lower BMI (median 19.8 vs. 20.8 kg/m^2, $p < 0.001$). Patients with endometriosis also demonstrated a higher prevalence of menstrual cycle irregularities ($p < 0.001$), abnormal bleeding patterns ($p < 0.001$), and dysmenorrhea (48.8% vs. 24.0%, $p < 0.001$).

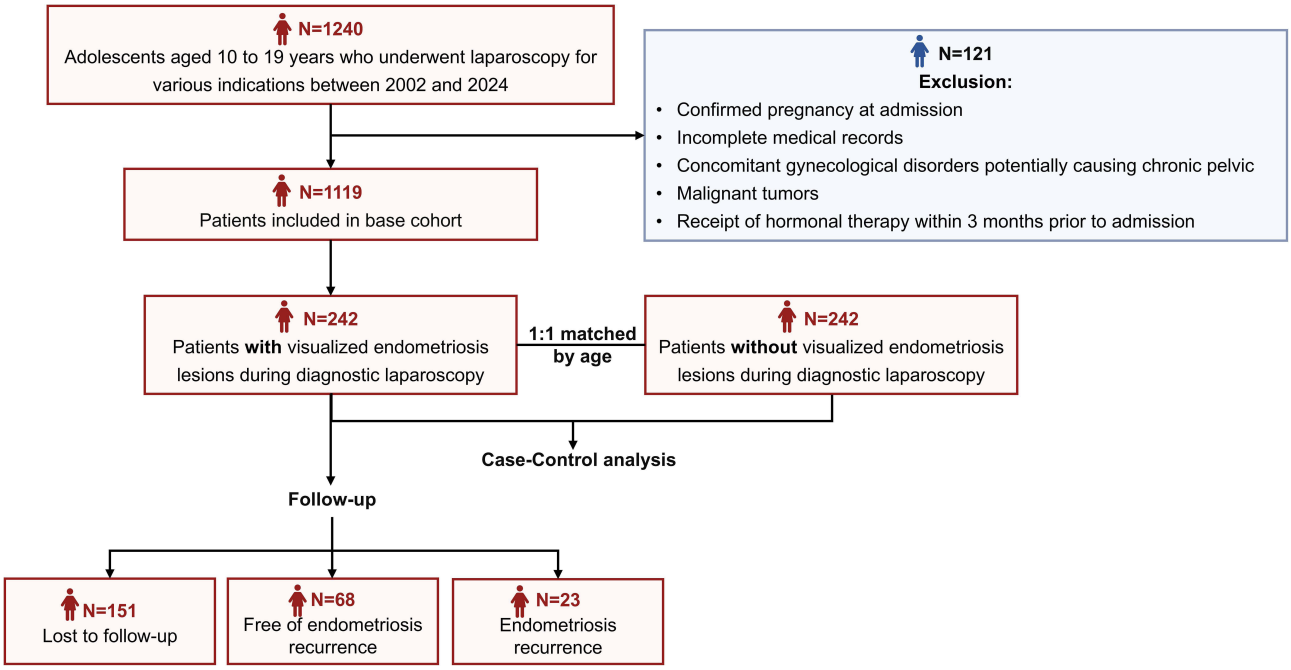

Figure 1 Flowchart of study population selection. The diagram illustrates the recruitment process, exclusion criteria, case-control matching, and follow-up outcomes for the adolescent endometriosis cohort (2002–2024).

Additionally, they exhibited more pronounced atypical pelvic pain manifestations and menstruation-associated gastrointestinal symptoms (eg, pain with nausea and vomiting).

Table 1 Patient Demographics and Clinical Characteristics

Characteristic	Endometriosis		*p*-value[b]
	No N = 242[a]	Yes N = 242[a]	
Age (years)	17.00 (15.00, 19.00)	17.00 (15.00, 19.00)	>0.999
BMI (kg/m^2)	20.8 (18.7, 22.8)	19.8 (18.2, 21.3)	<0.001
Age of menarche (years)	13.00 (12.00, 14.00)	13.00 (12.00, 14.00)	0.202
Menstrual cycles (days)			<0.001
<21	2 (0.8%)	6 (2.5%)	
21~35	171 (70.7%)	180 (74.4%)	
>35	68 (28.1%)	22 (9.1%)	
Absence of Menarche	1 (0.4%)	34 (14.0%)	
Duration of menstruation (days)			<0.001
<3	0 (0.0%)	1 (0.4%)	
3~7	218 (90.1%)	184 (76.0%)	
>7	23 (9.5%)	23 (9.5%)	
Absence of Menarche	1 (0.4%)	34 (14.0%)	
Dysmenorrhea			<0.001
Yes	58 (24.0%)	118 (48.8%)	
No	183 (75.6%)	90 (37.2%)	
Absence of Menarche	1 (0.4%)	34 (14.0%)	

(*Continued*)

Table 1 (Continued).

Characteristic	Endometriosis		*p*-value[b]
	No N = 242[a]	Yes N = 242[a]	
Pain symptoms			<0.001
None	183 (75.6%)	90 (37.2%)	
Dysmenorrhea	47 (19.4%)	87 (36.0%)	
Chronic pelvic pain	5 (2.1%)	17 (7.0%)	
Pain with nausea and vomiting	6 (2.5%)	14 (5.8%)	
Absence of Menarche	1 (0.4%)	34 (14.0%)	
Sexual life			0.586
Yes	29 (12.0%)	33 (13.6%)	
No	213 (88.0%)	209 (86.4%)	

Notes: [a]Median (Q1, Q3); n (%). [b]Wilcoxon rank sum test; Fisher's exact test; Pearson's Chi-squared test.

Risk Factor Profiles in Adolescent Endometriosis: Case-Control Comparison

To identify potential determinants of endometriosis onset in adolescents, we performed binary logistic regression analysis on all clinically collected variables with putative relevance to endometriosis pathogenesis (Table 2), including baseline characteristics (age, BMI), clinical features (dysmenorrhea patterns, menarche age, menstrual cycle length, bleeding duration, sexual history, genital tract malformation), and laboratory parameters (CA125, coagulation profile, metabolic markers). Comprehensive assessment of personal and family histories revealed no participants with positive EMS family

Table 2 Univariate and Multivariate Analysis of Factors Associated with Adolescent EMs Onset

Characteristic	Control N = 242	EMs N = 242	Univariable			Multivariable		
			OR	95% CI	*p*-value	OR	95% CI	*p*-value
Age (years)	17 (15, 19)	17 (15, 19)	1.00	0.93, 1.08	>0.999			
BMI (kg/m^2)	20.8 (18.7, 22.8)	19.8 (18.2, 21.3)	0.91	0.86, 0.96	0.001			
Dysmenorrhea								
No	183 (75.9%)	90 (43.3%)	–	–				
Yes	58 (24.1%)	118 (56.7%)	4.14	2.76, 6.19	<0.001			
Pain symptoms								
None	183 (75.9%)	90 (43.3%)	–	–		–	–	
Chronic pelvic pain	5 (2.1%)	17 (8.2%)	6.91	2.47, 19.34	<0.001	5.21	0.53, 50.82	0.156
Dysmenorrhea	47 (19.5%)	87 (41.8%)	3.76	2.44, 5.82	<0.001	3.61	1.97, 6.63	<0.001
Pain with nausea and vomiting	6 (2.5%)	14 (6.7%)	4.74	1.76, 12.76	0.002	4.34	0.89, 21.09	0.069
Age of menarche (years)	13 (12, 14)	13 (12, 14)	1.10	0.96, 1.28	0.176			
Menstrual cycles (days)								
21~35	171 (71.0%)	180 (86.5%)	–	–				
<21	2 (0.8%)	6 (2.9%)	2.85	0.57, 14.31	0.203			
>35	68 (28.2%)	22 (10.6%)	0.31	0.18, 0.52	<0.001			
Duration of menstruation (days)								
3~7	218 (90.5%)	184 (88.5%)	–	–				
<3	0 (0.0%)	1 (0.5%)	9.23×10^5	0.00, Inf	0.980			
>7	23 (9.5%)	23 (11.1%)	1.18	0.64, 2.18	0.586			
Sexual life								
No	213 (88.0%)	209 (86.4%)	–	–				
Yes	29 (12.0%)	33 (13.6%)	1.16	0.68, 1.98	0.587			
Genital tract malformation								
No	236 (97.5%)	171 (70.7%)	–	–		–	–	
Yes	6 (2.5%)	71 (29.3%)	16.33	6.94, 38.45	<0.001	16.15	3.29, 79.31	<0.001

(*Continued*)

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Table 2 (Continued).

Characteristic	Control N = 242	EMs N = 242	Univariable			Multivariable		
			OR	95% CI	*p*-value	OR	95% CI	*p*-value
Coagulation function								
PT (s)	13.10 (12.70, 13.50)	13.20 (12.80, 13.70)	1.39	1.06, 1.83	0.018	1.34	0.81, 2.21	0.251
APTT (s)	36.4 (34.5, 39.1)	37.4 (35.0, 39.8)	1.05	1.00, 1.10	0.065			
TT (s)	16.20 (15.60, 16.80)	15.70 (15.20, 16.40)	0.54	0.43, 0.67	<0.001			
FIB (g/L)	2.85 (2.55, 3.24)	3.04 (2.67, 3.48)	1.60	1.22, 2.10	<0.001	2.13	1.17, 3.89	0.013
PLT (*10^9/L)	271 (235, 316)	247 (206, 287)	0.99	0.99, 1.00	<0.001			
CA125 (U/mL)	18 (13, 25)	39 (21, 98)	1.02	1.01, 1.03	<0.001	1.02	1.01, 1.04	<0.001
Metabolic indicators								
TG (mmol/L)	0.88 (0.63, 1.26)	0.78 (0.62, 1.10)	0.56	0.38, 0.82	0.003	0.40	0.23, 0.69	0.001
TC (mmol/L)	4.20 (3.70, 4.79)	4.14 (3.72, 4.71)	0.95	0.75, 1.22	0.699	7.17	3.19, 16.12	<0.001
LDL (mmol/L)	2.50 (2.08, 2.86)	2.27 (1.92, 2.70)	0.71	0.53, 0.96	0.028	0.10	0.04, 0.27	<0.001
HDL (mmol/L)	1.28 (1.12, 1.49)	1.40 (1.19, 1.61)	0.98	0.93, 1.03	0.493			
Glu (mmol/L)	5.03 (4.66, 5.38)	5.08 (4.72, 5.46)	1.14	0.89, 1.46	0.302			
Transferrin (g/L)	2.85 (2.57, 3.17)	2.73 (2.50, 3.12)	0.76	0.48, 1.20	0.238			
Cysteine (μmol/L)	11.1 (9.1, 13.2)	10.3 (8.5, 12.3)	0.99	0.94, 1.05	0.722			

history or tobacco/alcohol use, consequently excluding these variables from final analysis. Following univariate screening, backward stepwise elimination selected dysmenorrhea, genital tract malformation, specific coagulation indices (prothrombin time, fibrinogen), CA125, and metabolic markers (triglycerides, total cholesterol, LDL cholesterol) for inclusion in the multivariable model. Final regression results established dysmenorrhea symptomatology (aOR 3.61; 95% CI 1.97–6.63), genital tract malformation (aOR 16.15; 95% CI 3.29–79.31), and elevated CA125 (aOR 1.02; 95% CI 1.01–1.04) as independent risk factors. Coagulation dysfunction - particularly hyperfibrinogenemia (aOR 2.13; 95% CI 1.17, 3.89) - and dyslipidemia patterns featuring hypotriglyceridemia, hypercholesterolemia, and low LDL levels demonstrated significant associations with adolescent EMS onset (Figure 2).

Analysis on Predictors of Postoperative Recurrence in Adolescent Endometriosis

Among the 242 adolescent endometriosis patients in the case cohort, postoperative follow-up was conducted to investigate disease recurrence. Ultimately, 91 patients were successfully followed up, with 151 lost to follow-up

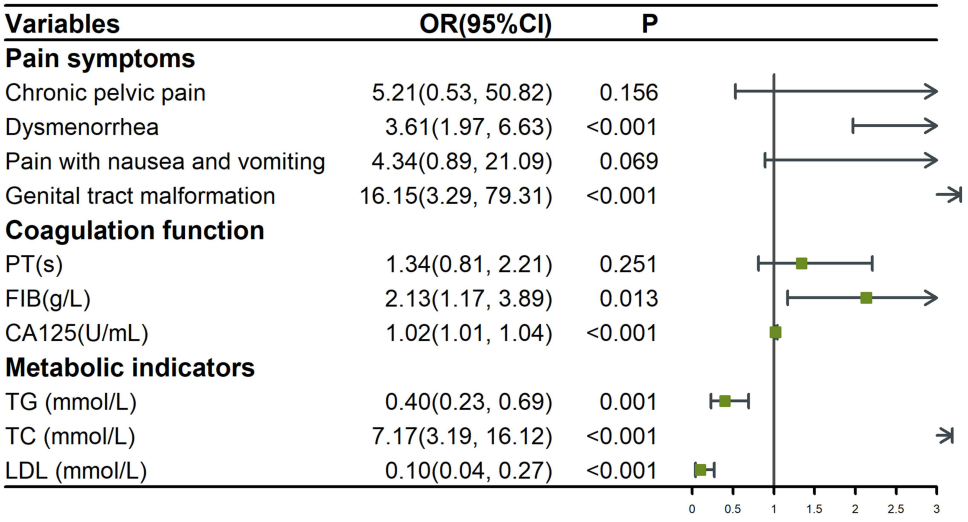

Figure 2 Forest plot of risk factors associated with the onset of adolescent endometriosis. Multivariable logistic regression analysis displaying adjusted odds ratios (OR) and 95% confidence intervals (CI) for independent predictors of disease onset.
Abbreviations: PT, prothrombin time; FIB, fibrinogen; CA125, cancer antigen 125; TG, triglycerides; TC, total cholesterol; LDL, low-density lipoprotein.

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(Table 3). The highest loss-to-follow-up rate occurred in peritoneal endometriosis cases (approximately 80%). Similarly, patients with lower rASRM stages demonstrated elevated attrition rates. Among the 91 successfully followed adolescents (mean follow-up duration: 56 months), lesion distribution included peritoneal endometriosis (n=31, 34.1%), ovarian endometriosis (n=10, 11.0%), combined peritoneal-ovarian lesions (n=35, 38.5%), ovarian-deep infiltrating endometriosis (DIE) comorbidity (n=3, 3.3%), and multifocal peritoneal-ovarian-DIE involvement (n=12, 13.2%). Overall recurrence occurred in 23 patients (25.3%), with recurrence patterns comprising isolated radiologic confirmation (n=14), concurrent symptomatic-radiologic recurrence (n=6), and radiologic-surgical confirmation (n=3)-no isolated symptomatic recurrence was documented.

Of the 91 followed patients, 47 (51.6%) received postoperative hormonal suppression, for example, gonadotropin-releasing hormone agonist (GnRH-a), combined oral contraceptives, or dienogest, while 44 (48.4%) did not. Univariate analysis indicated a higher recurrence rate in the treated group (38.3%) compared to the untreated group (11.4%) (HR 0.38 for non-users; 95% CI 0.14–1.03; p=0.057). This association likely reflects clinical selection bias, where high-risk patients (eg, advanced stage) were preferentially targeted for aggressive medical management.

Regarding recurrence dynamics, the median time to recurrence was 36 months (IQR: 12–50 months; range: 5–178 months). Stratified by lesion phenotype, recurrence was exclusively observed in patients with ovarian or deep infiltrating involvement. Notably, no patients with isolated peritoneal endometriosis experienced recurrence (0/31), whereas those with ovarian endometriomas (alone or with peritoneal/DIE lesions) exhibited significantly higher recurrence rates (ranging from 30.0% to 50.0%). The cumulative recurrence dynamics are further illustrated in Supplementary Figure 1.

Comprehensive baseline profiling was performed for all followed patients (Table 4), encompassing demographic parameters (age, BMI), clinical characteristics (menarche age, sexual history, genital tract malformation), initial endometriosis presentation (dysmenorrhea characteristics, EMs lesion types, CA125 levels), and primary surgical records (interval from menarche to surgery, cyst diameter, multifocality, rectouterine pouch obliteration, rASRM staging). Additional metrics included coagulation profiles and metabolic markers at initial diagnosis, along with postoperative management (adjuvant pharmacotherapy regimens). Subsequent Cox regression analysis incorporated all collected variables: univariate screening followed by backward stepwise selection identified age, ovarian cyst diameter, multifocality, CA125 levels, rASRM stage, specific coagulation indices, and metabolic markers for the multivariate model.

Table 3 Baseline Characteristics of Initial Endometriosis Cohort and Follow-up Cohort

Characteristic	Initial EMs Cohort N = 242[a]	Follow-up EMs Cohort N = 91[a]	*p*-value[b]
Age (years)	17.00 (15.00, 19.00)	17.00 (16.00, 19.00)	0.350
BMI (kg/m^2)	19.8 (18.2, 21.3)	20.1 (18.5, 22.2)	0.082
Age of menarche (years)	13.00 (12.00, 14.00)	13.00 (12.00, 13.00)	0.091
Dysmenorrhea			<0.001
Absence of Menarche	34 (14.0%)	0 (0.0%)	
No	90 (37.2%)	36 (39.6%)	
Yes	118 (48.8%)	55 (60.4%)	
r-ASRM Stage			<0.001
I	152 (62.8%)	34 (37.4%)	
II	6 (2.5%)	2 (2.2%)	
III	50 (20.7%)	33 (36.3%)	
IV	34 (14.0%)	22 (24.2%)	
EMs Phenotype			<0.001
Peritoneal	152 (62.8%)	31 (34.1%)	
Peritoneal and ovarian	49 (20.2%)	35 (38.5%)	
Peritoneal and ovarian and DIE	18 (7.4%)	12 (13.2%)	
Ovarian	19 (7.9%)	10 (11.0%)	
Ovarian and DIE	4 (1.7%)	3 (3.3%)	

Notes: [a]Median (Q1, Q3); n (%). [b]Wilcoxon rank sum test; Pearson's Chi-squared test; Fisher's exact test.

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Table 4 Univariate and Multivariate Analysis of Influencing Factors Associated with Endometriosis Recurrence (Cox Regression)

Characteristic	Recurrence		Univariable			Multivariable		
	No N=68	Yes N=23	HR	95% CI	*p*-value	HR	95% CI	*p*-value
Age (years)	17.00 (16.00, 19.00)	18.00 (17.00, 19.00)	1.10	0.87, 1.39	0.422	1.11	0.73, 1.71	0.622
BMI (kg/m^2)	20.1 (18.6, 22.2)	20.1 (18.4, 21.4)	1.00	0.86, 1.16	0.984			
Age of menarche (years)	13.00 (12.00, 13.00)	12.00 (12.00, 13.00)	0.87	0.62, 1.22	0.418			
Dysmenorrhea								
No	28 (41.2%)	8 (34.8%)	–	–				
Yes	40 (58.8%)	15 (65.2%)	1.13	0.47, 2.72	0.792			
Pain symptoms								
None	27 (39.7%)	7 (30.4%)	–	–				
Chronic pelvic pain	4 (5.9%)	5 (21.7%)	1.84	0.55, 6.23	0.325			
Dysmenorrhea	35 (51.5%)	9 (39.1%)	1.03	0.38, 2.78	0.956			
Pain with nausea and vomiting	2 (2.9%)	2 (8.7%)	1.78	0.35, 9.20	0.490			
Interval from menarche to surgery (years)	4.00 (3.00, 6.00)	5.00 (4.00, 6.00)	1.10	0.89, 1.37	0.366			
Genital tract malformation								
No	59 (86.8%)	19 (82.6%)	–	–				
Yes	9 (13.2%)	4 (17.4%)	0.93	0.30, 2.88	0.896			
Sexual life								
No	62 (91.2%)	17 (73.9%)	–	–				
Yes	6 (8.8%)	6 (26.1%)	2.36	0.92, 6.05	0.073			
EMs Phenotype								
Peritoneal and ovarian and DIE	6 (8.8%)	6 (26.1%)	–	–				
Ovarian	7 (10.3%)	3 (13.0%)	0.64	0.16, 2.61	0.538			
Peritoneal	31 (45.6%)	0 (0.0%)	0.00	0.00, Inf	0.998			
Peritoneal and ovarian	24 (35.3%)	14 (60.9%)	0.80	0.31, 2.09	0.650			
Maximum ovarian cyst diameter (cm)	5.50 (4.25, 8.00)	6.00 (4.00, 8.00)	1.05	0.92, 1.19	0.464	1.24	1.03, 1.49	0.024
Multiple ovarian cysts								
No	31 (83.8%)	16 (69.6%)	–	–		–	–	
Yes	6 (16.2%)	7 (30.4%)	1.65	0.68, 4.04	0.271	2.72	0.62, 11.98	0.186
CA125 (U/mL)	33 (19, 85)	123 (45, 304)	1.00	1.00, 1.01	0.003	1.01	1.00, 1.01	<0.001
Rectouterine pouch								
Non-obliterated	55 (80.9%)	12 (52.2%)	–	–				
Partially obliterated	7 (10.3%)	4 (17.4%)	1.18	0.37, 3.75	0.776			
Completely obliterated	6 (8.8%)	7 (30.4%)	2.21	0.86, 5.66	0.099			
r-ASRM stage								
1	33 (48.5%)	1 (4.3%)	–	–		–	–	
2	0 (0.0%)	2 (8.7%)	24.88	2.24, 276.82	0.009	0.16	0.00, 6.49	0.333
3	24 (35.3%)	9 (39.1%)	7.12	0.90, 56.55	0.064	1.09	0.06, 18.90	0.951
4	11 (16.2%)	11 (47.8%)	11.40	1.46, 88.90	0.020	0.87	0.05, 14.58	0.924
Postoperative pharmacotherapy								
Administered	29 (42.6%)	18 (78.3%)	–	–				
Not administered	39 (57.4%)	5 (21.7%)	0.38	0.14, 1.03	0.057			
Type of postoperative pharmacotherapy								
GnRH-a monotherapy	15 (51.7%)	7 (38.9%)	–	–				
GnRH-a with combined oral contraceptives	9 (31.0%)	7 (38.9%)	2.91	0.95, 8.90	0.062			
Combined oral contraceptives	4 (13.8%)	1 (5.6%)	0.91	0.11, 7.60	0.928			
GnRH-a with dienogest	1 (3.4%)	3 (16.7%)	7.99	1.79, 35.61	0.006			
PT (s)	13.30 (12.90, 13.70)	13.20 (13.00, 13.80)	0.79	0.43, 1.44	0.437	0.52	0.11, 2.38	0.396
APTT (s)	37.3 (34.5, 40.5)	35.8 (33.1, 38.7)	0.94	0.87, 1.01	0.101	0.77	0.60, 0.99	0.042
TT (s)	15.82 ± 0.82	15.72 ± 0.77	0.94	0.56, 1.60	0.830			
FIB (g/L)	3.08 (2.71, 3.48)	3.06 (2.69, 3.86)	1.22	0.72, 2.06	0.462	0.64	0.23, 1.78	0.390
PLT (*10^9/L)	249 (215, 293)	260 (238, 312)	1.00	1.00, 1.01	0.528	1.00	0.99, 1.01	0.957
TG (mmol/L)	0.95 (0.66, 1.18)	0.74 (0.68, 0.81)	0.52	0.15, 1.80	0.300	0.21	0.02, 1.81	0.154
TC (mmol/L)	4.09 ± 0.78	4.16 ± 0.76	1.21	0.66, 2.23	0.542	18.48	0.60, 570.51	0.096
LDL (mmol/L)	2.29 ± 0.59	2.30 ± 0.72	1.23	0.55, 2.73	0.610	0.06	0.00, 2.85	0.153
HDL (mmol/L)	1.25 (1.12, 1.50)	1.33 (1.16, 1.54)	0.91	0.38, 2.19	0.830	0.03	0.00, 2.35	0.118
Glu (mmol/L)	5.09 (4.71, 5.52)	5.08 (4.70, 5.44)	1.28	0.70, 2.34	0.419	2.23	0.73, 6.79	0.157

Abbreviations: CI, Confidence Interval; HR, Hazard Ratio.

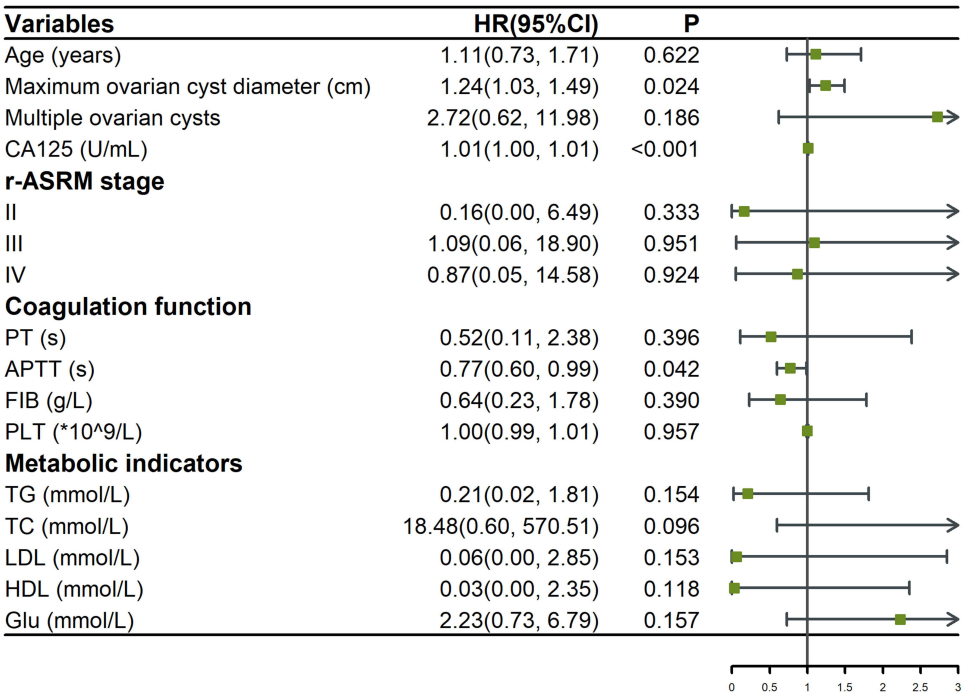

Figure 3 Forest plot of predictors for postoperative recurrence in adolescent endometriosis. Multivariable Cox proportional hazards regression analysis displaying adjusted hazard ratios (HR) and 95% confidence intervals (CI) for factors predicting disease recurrence.
Abbreviations: rASRM, revised American Society for Reproductive Medicine; PT, prothrombin time; APTT, activated partial thromboplastin time; FIB, fibrinogen; PLT, platelet count; TG, triglycerides; TC, total cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein; Glu, glucose.

Final multivariate Cox regression demonstrated significant associations between postoperative recurrence in adolescent endometriosis and larger ovarian cyst size (aHR 1.24; 95% CI 1.03–1.49), elevated CA125 (aHR 1.01, 95% CI 1.00–1.01), and coagulation abnormalities—specifically shortened activated partial thromboplastin time (APTT) (aHR 0.77, 95% CI 0.60–0.99) (Figure 3).

Discussion

Adolescent EMs, representing a distinct developmental phase of the disorder, has garnered increasing clinical attention. Leveraging a two-stage hybrid design, the case-control component of our study identified dysmenorrhea severity, genital tract malformation, and elevated CA125 as independent risk factors for the onset of adolescent EMs. Concurrently, coagulation disorders—particularly hyperfibrinogenemia—and dyslipidemia demonstrated significant associations with disease onset. Furthermore, our longitudinal cohort analysis established ovarian cyst diameter, persistent CA125 elevation, and acquired coagulopathies—notably shortened APTT—as robust predictors of postoperative recurrence in this population.

Current evidence consistently demonstrates that adolescents with EMs exhibit more complex pain phenotypes[22] and higher rates of concomitant Müllerian anomalies[23,24] - findings concordant with our data. Mechanistically, reproductive tract malformations may promote ectopic endometrial implantation through retrograde menstruation and disrupted uterine peristalsis. Epidemiological studies report 11–40% of adolescent EMs patients present with congenital anomalies, predominantly vaginal atresia.[25,26] Our findings reveal that while Müllerian anomalies are potent drivers of initial disease development due to increased retrograde menstruation, they do not independently increase the risk of postoperative recurrence once surgically corrected. This aligns with the "anatomical restoration" hypothesis proposed by Acién,[27] which suggests that resolving the outflow obstruction eliminates the primary source of peritoneal seeding. Recent longitudinal data in adolescents by Kapczuk et al[24] further support this, demonstrating that early surgical correction of anomalies favors the resolution of existing lesions and significantly reduces the risk of subsequent recurrence. Consequently, in adolescents with obstructive anomalies, timely anatomical restoration represents a critical preventive strategy to mitigate long-term recurrence risk.[28]

From a phenotypic perspective, ovarian endometrioma constitutes the most common subtype in adults, while peritoneal EMs has a higher proportion in adolescents.[29,30] Our study also found that peritoneal EMs is the highest proportion type (62.8%), which indicates the critical need for early detection and precision diagnosis in young populations. However, diagnosing adolescent EMs remains a persistent clinical challenge. Beyond symptom assessment, ancillary modalities including imaging and laboratory testing are essential diagnostic adjuncts.[6] The technical limitation of transabdominal ultrasound significantly impedes early lesion detection. Mounting evidence now positions EMs as a systemic disorder characterized by chronic inflammation and metabolic dysregulation, driving research into novel molecular biomarkers.[31] Among these, CA125 - the most extensively studied diagnostic indicator – demonstrated significant elevation in our adolescent cohort, aligning with established adult data.[32]

Furthermore, emerging evidence suggests that circulating proinflammatory cytokines in adult EMs may contribute to a hypercoagulable state.[33,34] Our study also demonstrated significantly elevated fibrinogen (FIB) levels in adolescents with EMs, indicating potential coagulation abnormalities in this population. However, key metabolic discordances were observed: unlike adults where hypertriglyceridemia is common,[35,36] our adolescent cohort exhibited a distinct "lean phenotype" with lower BMI and lipid levels. This aligns with the inverse association between BMI and EMs frequently reported in large cohorts[37,38] potentially reflecting diagnostic bias where lean individuals are more likely to undergo surgery. Mechanistically, this suggests drivers distinct from obesity-associated leptin-JAK-STAT signaling[39] supporting our finding of a dominant coagulation-inflammation crosstalk (mediated by the TF-Thrombin-PAR pathway). Furthermore, pubertal physiology—characterized by transient insulin resistance and elevated GH/IGF-1 for somatic growth[40]—inherently prioritizes lean mass accretion. Consequently, the "lean phenotype" in adolescents likely represents a specific high-risk subpopulation driven by coagulation-inflammatory mechanisms rather than metabolic dysfunction, suggesting that adult metabolic risk models should be applied with caution.

EMs, as a hormone-dependent disorder, presents significant challenges in postoperative recurrence management. This is particularly critical for adolescent patients, where stringent recurrence control is essential to safeguard their life-course reproductive health and minimize repeat surgeries over decades.[41] In reproductive-aged cohorts, intraoperative rAFS score, maximum cyst diameter, and lesion multiplicity constitute established independent risk factors for recurrent endometrioma.[15,42] Our study corroborates two key predictors in adolescents with ovarian EMs: maximum cyst diameter and preoperative CA125. This pathophysiological convergence likely reflects shared recurrence mechanisms: larger cysts increase technical difficulty in achieving complete excision, elevating risks of residual disease;[43] concurrently, elevated CA125 signifies greater disease burden and inflammatory activity.[44] Consequently, surgical management of large endometriomas demands exceptional technical proficiency-balancing complete cyst enucleation against optimal ovarian tissue preservation. Furthermore, structured postoperative surveillance combined with extended pharmacotherapy should be prioritized in this high-risk adolescent subgroup.

Similarly, a hypercoagulable state of the blood is not only associated with the initial onset of EMs, but also with its postoperative recurrence.[29] We found that shortened APTT—indicative of intrinsic coagulation overactivation—was significantly correlated with recurrence in adolescents. This likely reflects the activation of the Tissue Factor (TF)-Thrombin-PAR pathway.[45,46] Ectopic lesions overexpress TF, triggering thrombin generation, which acts not only in hemostasis but also as a signaling molecule via PAR-1/2 receptors. This activation stimulates the release of VEGF and IL-8,[45] creating a "coagulation-inflammation" positive feedback loop that promotes neuroangiogenesis and lesion survival. Consequently, the systemic hypercoagulable state observed in our cohort represents an active pathogenic process facilitating recurrence, suggesting that targeting this crosstalk could offer novel therapeutic strategies.

However, our study has limitations inherent to its retrospective design. First, the selection of surgical controls (adolescents with benign ovarian cysts) rather than healthy asymptomatic individuals may attenuate observed associations, as these controls likely have a higher baseline of gynecological symptoms. Second, confounding by indication influenced our recurrence analysis. We observed a paradoxical trend where hormonally treated patients had higher recurrence rates; however, this reflects clinical practice where adjuvant therapy was selectively targeted at severe phenotypes (Stage III/IV), masking potential protective effects. Third, a significant limitation is the high loss-to-follow-up rate (62.4%), attributable to the high mobility and lower compliance characteristic of adolescence. Comparative analysis revealed that lost patients predominantly had mild (Stage I) and asymptomatic disease. Consequently, our

reported recurrence rate (25.3%) likely overestimates the population risk by reflecting a symptomatic, high-risk subpopulation. Sensitivity analysis adjusting for disease stage suggests a more conservative population-level recurrence rate of approximately 17.0% (Supplementary Table 1). Finally, the single-center setting may restrict external validity.

Conclusion

Based on a large-sample retrospective analysis, this study identified dysmenorrhea, reproductive tract anomalies, and elevated CA125 levels as independent risk factors for the development of endometriosis in adolescents. Additionally, a hypercoagulable state and adolescence-specific metabolic disturbances were significantly associated with EMs. Beyond traditional predictors of recurrence such as ovarian cyst size and CA125 levels, shortened APTT (indicating hyperactive intrinsic coagulation) was also identified as a predictor of endometriosis recurrence in adolescents. Therefore, our findings suggest the need to enhance screening and postoperative monitoring for high-risk adolescents in clinical management.

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Disclosure

The authors declare no conflicts of interest in this work.

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