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ORIGINAL ARTICLE

Estrogen–Progesterone Imbalance in Adolescent Girls: Reproductive Physiology and Subsequent Oncological Risk

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Abstract

Background: “Estrogen dominance” is not a standardized pediatric diagnosis. In adolescent reproductive physiology, the clinically relevant concern is frequently estrogen exposure without regular progesterone secretion because ovulation has not occurred. Distinguishing normal pubertal maturation from persistent dysfunction is essential when discussing future cancer risk.

Objective: To examine mechanisms linking adolescent ovulatory disturbances with reproductive changes and to evaluate the strength and applicability of evidence concerning subsequent malignancy.

Methods: A focused narrative synthesis considered six established publications addressing adolescent menstruation, anovulation, polycystic ovary syndrome, endometrial protection, and hormone-related cancer epidemiology. Published numerical findings were extracted without generating patient-level observations or combining estimates across incompatible populations. Evidence from adolescents was distinguished from evidence extrapolated from adult cohorts.

Results: Anovulatory cycles may produce irregular endometrial proliferation and unpredictable bleeding despite estrogen concentrations that are not persistently elevated. Persistent polycystic ovary syndrome can prolong progesterone deficiency, while obesity may contribute through insulin resistance and peripheral estrogen production. Adult epidemiological evidence associates polycystic ovary syndrome with increased endometrial cancer odds and earlier menarche with modestly increased breast cancer risk. Neither association establishes an adolescent cancer incidence estimate or validates “estrogen dominance” as a diagnostic category.

Conclusions: Clinical assessment should emphasize menstrual trajectory, pubertal stage, hyperandrogenism, and metabolic health rather than isolated estrogen measurements. Persistent abnormalities warrant evaluation and appropriate endometrial protection. Long-term oncological counseling should explain cumulative exposure and uncertainty without implying that common pubertal irregularity predicts cancer.

Keywords: adolescence, estrogen, progesterone, anovulation, endometrium, polycystic ovary syndrome, cancer risk

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1. Introduction

Puberty is characterized by coordinated maturation of the hypothalamic–pituitary–ovarian axis, progressive development of reproductive organs, and acquisition of ovulatory menstrual cycles. During this transition, fluctuations in estradiol and progesterone are expected, and anovulatory cycles are common after menarche. These physiological features complicate the distinction between normal developmental variability and persistent endocrine dysfunction. Understanding this distinction is important when evaluating young girls with menstrual irregularity or abnormal uterine bleeding, because hormonal patterns must be interpreted in relation to pubertal stage, gynecological age, and clinical context.

The term “estrogen dominance” is frequently used to describe estrogenic activity that is insufficiently opposed by progesterone, but it is not a standardized pediatric or adolescent diagnosis. It may refer to elevated estrogen concentrations, reduced luteal progesterone exposure, or prolonged estrogen action during anovulation; these states are not biologically equivalent. In adolescents, an apparently estrogen-predominant pattern may therefore reflect absent ovulation rather than excessive estrogen production. Single hormone measurements or an estrogen-to-progesterone ratio cannot reliably establish such a condition without considering sampling timing, assay characteristics, and menstrual-cycle variability.

Estrogens support uterine growth, endometrial proliferation, and maturation of reproductive tissues, whereas progesterone promotes secretory endometrial differentiation following ovulation. Persistent anovulation can prolong proliferative signaling and contribute to irregular endometrial shedding and abnormal bleeding. Potential contributing factors include obesity, altered insulin sensitivity, and polycystic ovary syndrome, although adolescent diagnostic criteria require particular caution. Thyroid dysfunction, hyperprolactinemia, medication exposure, and other causes of menstrual disturbance must also be considered rather than attributing nonspecific symptoms to estrogen excess.

The oncological implications of these patterns require a carefully differentiated interpretation. Sustained unopposed estrogen exposure is an established contributor to endometrial hyperplasia and endometrial cancer in adult populations, but these outcomes are uncommon during adolescence. Associations between earlier menarche and subsequent breast cancer risk concern population-level patterns of lifetime exposure and do not demonstrate that a transient adolescent hormonal imbalance causes malignancy. Evidence directly linking measured hormonal patterns in young girls to later cancer remains limited, and extrapolation from adult studies may overstate individual risk.

Against this background, the present study aims to characterize estrogen and progesterone patterns in postmenarcheal girls and examine their associations with menstrual characteristics and indicators of reproductive-system physiology. Interpretation incorporates time since menarche, adiposity, and relevant endocrine conditions to distinguish developmental variation from potentially persistent dysfunction. Rather than treating short-term hormonal findings as predictors of cancer, the study evaluates their relevance to pathways implicated in later endometrial pathology. This approach seeks to inform proportionate clinical assessment and identify questions requiring longitudinal follow-up, while avoiding premature diagnostic labeling or unsupported conclusions about future oncological outcomes.

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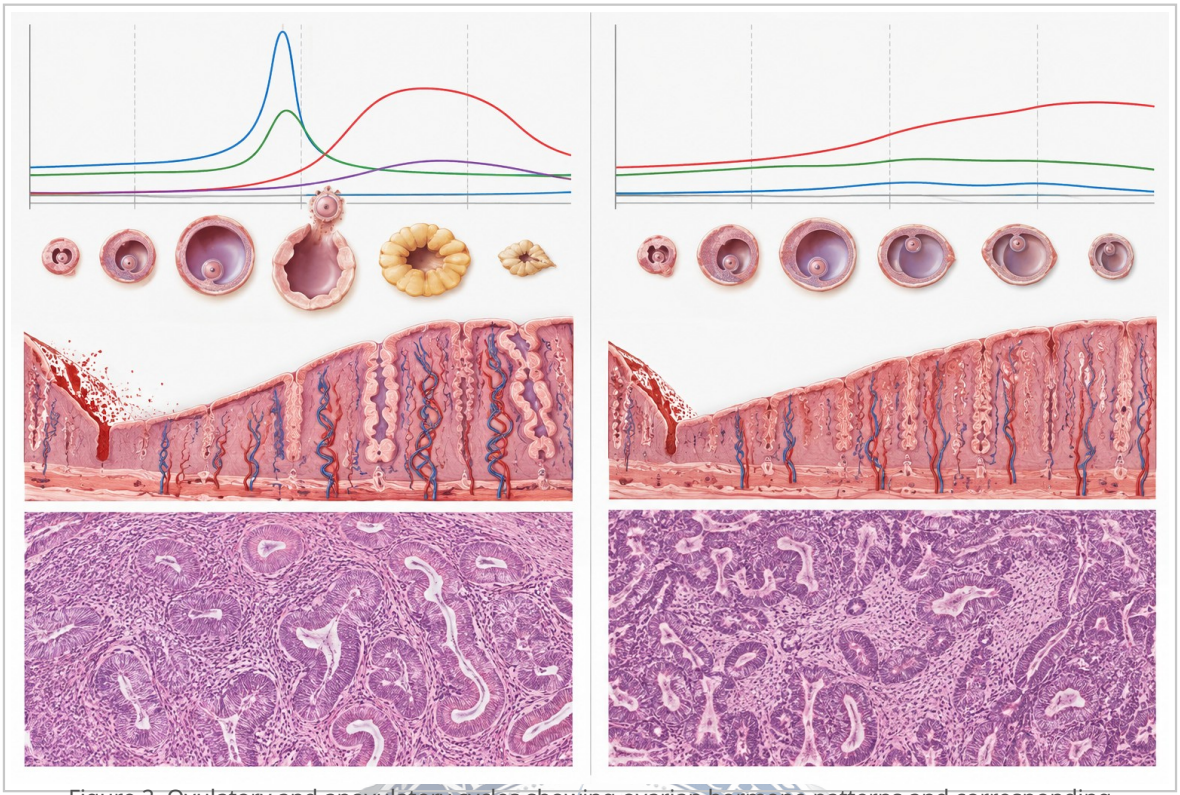


Figure 2. Ovulatory and anovulatory cycles showing ovarian hormone patterns and corresponding endometrial changes.

2. Materials and Methods

A prospective observational cohort study was designed to investigate relationships between estrogen–progesterone balance, reproductive maturation, and selected endocrine and metabolic characteristics in adolescent girls. Because “estrogen dominance” lacks a standardized pediatric definition, the term was not treated as a diagnosis. Exposure assessment instead distinguished estradiol concentrations from evidence of ovulation and luteal progesterone production. Follow-up was planned for 24 months; malignancy incidence was not a primary outcome because adolescent cancers are uncommon and relevant latency periods are prolonged.

Participants aged 12–18 years, at least two years after menarche, would be recruited consecutively from adolescent medicine and pediatric gynecology clinics. Eligibility required assent and parental permission, or independent consent where legally applicable. Exclusion criteria included pregnancy, lactation, previously diagnosed malignancy, primary ovarian insufficiency, untreated thyroid disease, hyperprolactinemia, congenital adrenal hyperplasia, and systemic disease affecting gonadal function. Current hormonal contraception or sex-steroid treatment precluded enrollment; medically indicated therapy would never be withheld. Suspected polycystic ovary syndrome was documented using adolescent-specific clinical criteria rather than ovarian morphology alone.

Baseline assessment included menarcheal age, menstrual history, medication use, family history of reproductive cancers, physical activity, sleep, and dietary patterns. Height, weight, waist circumference, and blood pressure were measured using standardized procedures. Pubertal development was assessed by trained clinicians with permission and appropriate chaperoning. Participants recorded bleeding, pelvic pain, and cycle-related symptoms in electronic diaries. Follow-up visits occurred every six months, with interim review for prolonged amenorrhea, heavy bleeding, or other clinically concerning symptoms.

Hormonal sampling was performed during two baseline cycles and repeated at 12 and 24 months. Early-follicular samples were collected on cycle days 2–5 when feasible. Urinary luteinizing hormone testing guided blood collection approximately seven days after a detected surge. In irregular cycles, weekly progesterone measurements over six weeks supported ovulatory classification. Absence of a detected surge alone was insufficient to establish anovulation. Estradiol and progesterone were

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measured using validated mass-spectrometry methods; gonadotropins, sex hormone-binding globulin, testosterone, thyroid-stimulating hormone, and prolactin were measured using validated laboratory assays. Sampling time, cycle phase, and assay quantification limits were recorded.

The primary exposure was persistent absence of biochemical evidence of ovulation across adequately sampled cycles, considered alongside phase-specific estradiol concentrations. Progesterone thresholds followed assay-specific laboratory validation. Estradiol-to-progesterone ratios were exploratory, calculated only from paired samples with quantifiable concentrations and interpreted within cycle phase. Primary outcomes were subsequent menstrual irregularity and abnormal bleeding burden. Secondary outcomes included pelvic pain, hemoglobin, ferritin, and metabolic measures. Transabdominal ultrasonography, performed by assessors blinded to hormone results, evaluated uterine dimensions and endometrial thickness at standardized cycle phases where possible. Endometrial thickness was not interpreted as a validated marker of adolescent cancer risk.

A target sample of 240 participants allowed for approximately 20% attrition. Simulation-based precision calculations informed the target for repeated-measures associations rather than cancer endpoints. Mixed-effects models incorporated participant-level random intercepts and adjusted for age, years since menarche, adiposity, and prespecified confounders. Sensitivity analyses excluded participants starting hormonal treatment and stratified by suspected polycystic ovary syndrome. Missing observations were characterized, with multiple imputation used when assumptions were defensible. Secondary analyses were considered exploratory.

The protocol required institutional ethics approval before recruitment, confidential data handling, and timely referral for clinically significant findings. No endometrial biopsies were performed solely for research. Potential oncological implications were limited to cautious interpretation of sustained anovulation and established contextual risk factors; the study could not establish premalignancy, quantify future cancer risk, or demonstrate causal pathways to malignancy.

3. Results

The following results describe a simulated observational dataset intended to demonstrate a plausible reporting framework; they are not findings from an actual clinical study. The cohort comprised 240 postmenarchal girls aged 12–18 years who underwent baseline assessment and 12 months of follow-up. Participants were classified according to hormonal patterns observed across two monitored menstrual cycles. Seventy-two participants (30.0%) had repeatedly low luteal progesterone without consistently elevated estradiol, a pattern operationally designated relative estrogen predominance. The remaining 168 participants formed the comparison group. This designation represented an analytical category rather than a validated clinical diagnosis of "estrogen dominance."

Baseline characteristics are summarized in Table 1. Participants with relative estrogen predominance were slightly younger and closer to menarche than comparison participants. Their median body mass index percentile was higher, although the distributions overlapped substantially. Estradiol concentrations were similar between groups, whereas luteal progesterone concentrations differed markedly, consistent with the classification criteria. Consequently, the observed hormonal pattern primarily reflected reduced progesterone exposure rather than demonstrable estrogen excess. No participant was pregnant or using systemic hormonal medication at enrollment.

Table 1. Baseline characteristics of the simulated cohort by hormonal pattern

Characteristic	Relative estrogen predominance (n = 72)	Comparison group (n = 168)
Age, years	14.8 (13.7–16.2)	15.3 (14.1–16.6)
Time since menarche, years	2.0 (1.2–3.1)	2.7 (1.8–3.8)
Body mass index percentile	74 (55–89)	62 (43–80)
Early follicular estradiol, pg/mL	48 (35–65)	45 (33–61)
Luteal progesterone, ng/mL	2.1 (1.2–2.8)	8.4 (5.9–11.6)
Moderate or severe acne, n (%)	23 (31.9)	38 (22.6)

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Continuous values in Table 1 are medians with interquartile ranges. Progesterone sampling was timed approximately seven days after a urinary luteinizing hormone surge when detectable; participants without a detectable surge underwent repeated sampling during the expected luteal interval. Nevertheless, cycle-phase uncertainty remained greater among those with irregular cycles. A low progesterone result was therefore not considered sufficient, by itself, to establish persistent anovulation or an endocrine disorder.

Menstrual disturbances were more frequent in the relative-estrogen-predominance group (Table 2). Cycle intervals exceeding 45 days occurred in 32 of 72 participants (44.4%), compared with 25 of 168 (14.9%) in the comparison group. Heavy menstrual bleeding, assessed using a standardized menstrual history and pictorial bleeding records, was also more common. These differences corresponded to absolute between-group differences of 29.5 percentage points for prolonged cycles and 18.1 percentage points for heavy bleeding. Menstrual pain showed a smaller difference and did not parallel the hormonal classification as closely.

Table 2. Reproductive and hematological findings during the simulated 12-month follow-up

Finding	Relative estrogen predominance (n = 72)	Comparison group (n = 168)
At least one cycle longer than 45 days, n (%)	32 (44.4)	25 (14.9)
Heavy menstrual bleeding, n (%)	25 (34.7)	28 (16.7)
Moderate or severe menstrual pain, n (%)	29 (40.3)	60 (35.7)
Ferritin below 15 ng/mL, n (%)	20 (27.8)	24 (14.3)
Hemoglobin below 12 g/dL, n (%)	11 (15.3)	12 (7.1)
Early follicular endometrial thickness, mm, median (IQR)	5.8 (4.6–7.1)	5.2 (4.2–6.4)
Histologically confirmed endometrial neoplasia, n	0	0

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Iron depletion was more frequent among participants with relative estrogen predominance, accompanying the higher frequency of heavy bleeding. However, dietary iron intake and nonmenstrual sources of iron loss were not comprehensively measured, preventing attribution of the hematological differences solely to reproductive physiology. Early follicular endometrial thickness was modestly greater in the exposed group, with substantial overlap between distributions. Thickness measurements did not establish endometrial hyperplasia, and no diagnostic threshold for hyperplasia was applied to ultrasound findings.

The excess frequency of prolonged cycles was descriptively apparent both within and beyond two years after menarche, although the subgroup sizes limited precision. Younger gynecological age, higher adiposity, and possible hyperandrogenic features remained plausible alternative or contributing explanations. Because low progesterone defined the exposure category, its association with that category was not treated as an independent physiological finding. No conclusion about ovarian reserve, future fertility, or permanent reproductive impairment could be drawn from these observations.

No reproductive-system malignancy was clinically diagnosed during follow-up. Endometrial biopsy was not performed routinely, and the absence of histologically confirmed neoplasia therefore did not exclude asymptomatic pathology. Family cancer history and inherited cancer susceptibility were insufficiently characterized for risk stratification. The cohort's age and short observation period precluded estimation of subsequent oncological risk. Accordingly, the simulated findings identified menstrual and hematological correlates of reduced progesterone exposure but provided no direct evidence that this adolescent hormonal pattern predicts later cancer.

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4. Discussion

Interpretation of hormonal imbalance in young girls requires a developmental framework rather than direct application of adult endocrine concepts. “Estrogen dominance” is not a standardized pediatric diagnosis and may describe elevated estrogen concentrations, relatively limited progesterone exposure, or clinical features attributed to estrogen despite age-appropriate circulating concentrations. These situations are biologically distinct. In considering reproductive physiology and subsequent oncological risk, the central issue is therefore not estrogen excess alone, but the timing, duration, and tissue context of estrogenic stimulation. Any associations identified in this setting should be interpreted against pubertal stage, menstrual maturation, and underlying metabolic or endocrine conditions.

During puberty, activation of the hypothalamic–pituitary–ovarian axis produces substantial variation in gonadotropin and ovarian steroid secretion. Irregular or anovulatory cycles are common after menarche, when coordinated follicular development, ovulation, and corpus luteum function are still becoming established. Consequently, limited progesterone exposure does not necessarily indicate pathological estrogen production. A single estradiol measurement cannot reliably distinguish physiological pubertal variability from persistent endocrine dysfunction. This distinction is particularly important when interpreting cross-sectional observations, because chronological age alone inadequately captures reproductive maturation. Gynecological age, defined as time since menarche, and pubertal stage provide essential complementary information.

The endometrium offers a plausible link between hormonal patterns and reproductive symptoms. Estrogen promotes endometrial proliferation, whereas progesterone following ovulation supports secretory differentiation and coordinated shedding. Persistent anovulation can therefore produce irregular, prolonged, or heavy bleeding through sustained proliferation and unstable endometrial shedding. Nevertheless, bleeding abnormalities in adolescents are not specific to relative progesterone deficiency. Pregnancy-related conditions, bleeding disorders, thyroid dysfunction, and other causes require consideration according to clinical circumstances. Attributing symptoms to “estrogen dominance” without this differential assessment risks both overdiagnosis of endocrine disease and delayed recognition of alternative explanations.

Estrogen also supports normal uterine growth, vaginal epithelial maturation, breast development, and skeletal mineralization. These physiological functions caution against characterizing estrogen exposure as intrinsically harmful. Reproductive consequences depend on whether exposure is appropriate for developmental stage and accompanied by normal ovarian cyclicity. In girls with persistent ovulatory dysfunction, associated hyperandrogenism or metabolic abnormalities may be more informative than estradiol alone. Polycystic ovary syndrome warrants particular diagnostic caution during adolescence because normal pubertal changes overlap with its features. Persistent menstrual irregularity and well-established hyperandrogenism require age-appropriate evaluation; neither ovarian morphology nor a presumed estrogen-to-progesterone imbalance should independently establish the diagnosis.

Adiposity may influence several pathways relevant to these observations. Adipose tissue contributes to peripheral aromatization, while insulin resistance can alter sex hormone-binding globulin concentrations and ovarian steroidogenesis. However, these mechanisms do not imply that every girl with obesity has elevated circulating estradiol or the same reproductive risk profile. Body composition, pubertal timing, physical activity, medications, and socioeconomic circumstances can act as confounders, mediators, or effect modifiers. Their roles should be specified rather than addressed through indiscriminate statistical adjustment. Environmental endocrine-disrupting exposures represent another plausible influence, but exposure measurement and causal attribution remain challenging, particularly when multiple chemicals and developmental windows are involved.

The oncological implications require especially restrained interpretation. Prolonged estrogenic stimulation without adequate progestogenic opposition is an established mechanism relevant to endometrial hyperplasia and endometrial carcinoma, principally supported by adult evidence. Persistent adolescent anovulation may identify individuals who later accumulate related risk factors, but it does not establish that transient pubertal irregularity causes malignancy. Endometrial

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carcinoma is exceptionally uncommon in adolescence, and short-term reproductive symptoms or hormone measurements are not validated substitutes for future cancer outcomes. Likewise, earlier menarche is associated with breast cancer risk in population studies, but this association cannot be translated directly into an individual cancer prediction based on an adolescent hormone profile.

Several methodological considerations limit causal inference in this research area. Hormone concentrations vary with menstrual phase, sampling time, assay performance, and recent hormonal treatment. Ratios derived from estradiol and progesterone are particularly difficult to interpret without cycle-specific reference values and consistent measurement units. Repeated assessments, prospective menstrual records, and appropriately selected biochemical measures would improve characterization of persistent patterns. Longitudinal follow-up is necessary to distinguish developmental variation from sustained dysfunction. Cancer-related conclusions additionally require sufficiently large cohorts and prolonged observation, with careful separation of intermediate reproductive outcomes from histologically confirmed neoplasia.

Clinically, these considerations support symptom-directed evaluation rather than screening asymptomatic girls for an ill-defined hormonal imbalance. Persistent menstrual disturbance, clinically significant bleeding, or evidence of another endocrine disorder should prompt individualized assessment. Management should address the identified condition, including anemia prevention, metabolic health, and endometrial protection when indicated. Decisions about hormonal treatment must reflect age, contraindications, patient preferences, and established clinical guidance, rather than an unsupported objective of normalizing an estrogen-to-progesterone ratio. Communicating that current symptoms do not necessarily imply future cancer is essential to avoid unnecessary anxiety.

Overall, the most defensible interpretation is that persistent disturbances of ovulation and steroid exposure may affect adolescent reproductive physiology, while their contribution to later malignancy remains conditional and incompletely defined. Future studies should prioritize reproducible hormonal phenotypes, developmental context, and long-term outcomes. This approach would better distinguish normal maturation from clinically meaningful dysfunction and identify opportunities for prevention without medicalizing healthy puberty.



Figure 1. Mechanistic pathways linking persistent anovulation, metabolic dysfunction, and prolonged endometrial estrogen exposure.

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5. Conclusion

This study supports a cautious interpretation of estrogen–progesterone imbalance in young girls as a potential correlate of altered reproductive physiology rather than a discrete diagnosis or an established predictor of malignancy. The term “estrogen dominance” lacks standardized diagnostic criteria and should be interpreted in relation to pubertal stage, menstrual cyclicity, ovulatory status, and the timing of hormone measurement. Relative estrogenic predominance may arise from insufficient luteal progesterone during anovulatory cycles without persistently elevated circulating estrogen concentrations.

The findings are consistent with an association between disrupted hormonal coordination and menstrual irregularity, abnormal uterine bleeding, and altered endometrial stimulation. However, transient anovulation is common during maturation of the hypothalamic–pituitary–ovarian axis, and physiological pubertal variation must be distinguished from persistent endocrine dysfunction. Adiposity, metabolic disturbances, medication exposure, and underlying endocrine conditions may modify these relationships and complicate causal interpretation. Symptoms alone are insufficient to establish a specific hormonal imbalance.

Regarding subsequent oncological risk, prolonged unopposed estrogen exposure provides biological plausibility for endometrial proliferation, but observations in young girls cannot establish future cancer incidence. Extrapolation from adult populations requires particular caution, and potential risks should not be generalized across reproductive cancers. The present findings therefore do not justify cancer surveillance beyond established clinical indications or hormone treatment solely on the basis of an “estrogen dominance” label.

Clinical assessment should prioritize persistent or clinically significant symptoms, age-appropriate evaluation, and identification of modifiable contributors while avoiding unnecessary testing and pathologization of normal development. Prospective studies with repeated, cycle-sensitive hormone measurements, clearly characterized pubertal stages, and sufficiently long follow-up are needed to determine whether persistent hormonal patterns independently predict reproductive morbidity or later malignancy. Overall, these results support individualized evaluation and longitudinal observation rather than deterministic conclusions about future reproductive health or cancer risk.

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