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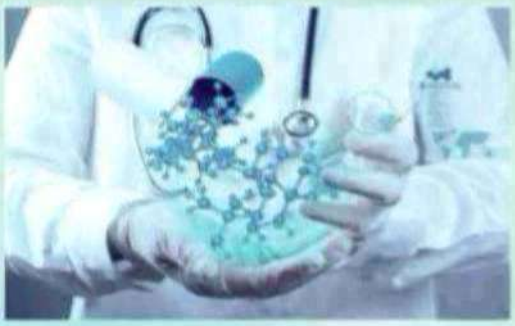

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ABNORMAL UTERINE BLEEDING IN WOMEN WITH CONNECTIVE TISSUE DYSPLASIA: NEW CLINICAL EVIDENCE

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✓ ***Resume***

Abnormal uterine bleeding (AUB) is a common concern for women of reproductive age. Among the many causes behind AUB, ovulatory dysfunction stands out as a significant factor. This condition disrupts the normal menstrual cycle, leading to irregular or heavy bleeding.At the same time, some women have connective tissue dysplasia (CTD), a group of inherited disorders affecting the body's connective tissues. They may experience joint hypermobility, skin laxity, and fragile blood vessels. Interestingly, recent studies suggest a link between CTD and various gynecological issues, including abnormal bleeding patterns.Understanding how ovulatory dysfunction and connective tissue dysplasia intersect is key for accurate diagnosis and effective treatment. This review aims to explore the current literature, highlighting the relationship between these conditions in women of reproductive age.

Keywords: ovulation, uterine bleeding, menstrual irregularity.

BOG'LOVCHI TO'QIMA DISPLAZIYASI BO'LGAN AYOLLARDA ANOMAL BACHADON QON KETISHLARI: YANGI KLINIK MA'LUMOTLAR

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✓ ***Rezyume***

Anomal bachadon qon ketishi (ABQ) reproduktiv yoshdagi ayollar uchun keng tarqalgan tashvishdir. AUBning ko'plab sabablari orasida ovulyatsiya disfunktsiyasi muhim omil sifatida ajralib turadi. Bu holat odatdagi hayz davrini buzadi, bu esa tartibsiz yoki og'ir qon ketishiga olib keladi.Shu bilan birga, ba'zi ayollarda biriktiruvchi to'qimalar displazi (BTD), tananing biriktiruvchi to'qimalariga ta'sir qiluvchi irsiy kasalliklar guruhi mavjud. Ularda bo'g'imlarning gipermobilligi, terining egiluvchanligi va mo'rt qon tomirlari paydo bo'lishi mumkin. Qizig'i shundaki, yaqinda o'tkazilgan tadqiqotlar BTD va turli ginekologik muammolar, jumladan, g'ayritabiiy qon ketish shakllari o'rtasidagi bog'liqlikni ko'rsatadi. Ovulyatsiya disfunktsiyasi va biriktiruvchi to'qima displazi qanday kesishishini tushunish to'g'ri tashxis qo'yish va samarali davolash uchun kalit hisoblanadi. Ushbu sharh reproduktiv yoshdagi ayollarda ushbu shartlar o'rtasidagi munosabatlarni ta'kidlab, joriy adabiyotlarni o'rganishga qaratilgan.

Kalit so'zlar: ovulyatsiya, bachadondan qon ketish, hayz davrining buzilishi.

АНОМАЛЬНЫЕ МАТОЧНЫЕ КРОВОТЕЧЕНИЯ У ЖЕНЩИН С ДИСПЛАЗИЕЙ СОЕДИНИТЕЛЬНОЙ ТКАНИ: НОВЫЕ КЛИНИЧЕСКИЕ ДАННЫЕ

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✓ ***Резюме***

Аномальное маточное кровотечение (АМК) является распространенной проблемой для женщин репродуктивного возраста. Среди многих причин, вызывающих ДСТ, овуляторная дисфункция выделяется как существенный фактор. Это состояние нарушает нормальный менструальный цикл, что приводит к нерегулярным или обильным кровотечениям. В то же время у некоторых женщин есть дисплазия соединительной ткани (ДСТ), группа наследственных заболеваний, поражающих соединительные ткани организма. У них может наблюдаться гипермобильность суставов, дряблость кожи и хрупкие кровеносные сосуды. Интересно, что недавние исследования предполагают связь между ДСТ и различными гинекологическими проблемами, включая аномальные кровотечения. Понимание того, как пересекаются овуляторная дисфункция и дисплазия соединительной ткани, является ключом к точной диагностике и эффективному лечению. Цель этого обзора — изучить современную литературу, подчеркнув взаимосвязь между этими состояниями у женщин репродуктивного возраста.

Ключевые слова: овуляция, маточные кровотечения, нарушение менструального цикла.

Relevance

The relationship between abnormal uterine bleeding becomes particularly complex in women with connective tissue disorders (CTD). While the PALM-COEIN classification system helps categorize AUB into structural and nonstructural causes, its application in CTD patients presents unique challenges. Furthermore, recent clinical studies indicate that women with CTDs may experience different patterns and mechanisms of bleeding compared to the general population. Despite the high prevalence of AUB—affecting up to one-third of women in their lifetime—a recent national audit in England and Wales revealed that only one-third of women were satisfied with their AUB management one year after referral.

In this article, we will explore new evidence from clinical studies examining the intersection of abnormal uterine bleeding and connective tissue disorders, highlighting the pathophysiological mechanisms, diagnostic challenges, and emerging treatment approaches for this underserved patient population.

Revisiting the FIGO PALM-COEIN Framework in the Context of CTD

The FIGO PALM-COEIN [1,2] classification system, established to standardize the diagnosis and treatment of abnormal uterine bleeding, requires careful reconsideration when applied to patients with connective tissue disorders. Initially designed to create a universal language for AUB assessment, this framework categorizes causes into structural (PALM) and non-structural (COEIN) groups. However, in CTD patients, the boundaries between these categories frequently blur, necessitating a more nuanced approach.

Structural vs Non-Structural Causes in CTD Patients

The PALM-COEIN system divides AUB etiologies into two major groups:

- Structural causes (PALM) [1,3,17,18]. : Polyps, Adenomyosis, Leiomyoma, and Malignancy/hyperplasia – disorders that can be visually identified through imaging or histopathology
- Non-structural causes (COEIN): Coagulopathy, Ovulatory dysfunction, Endometrial disorders, Iatrogenic factors, and disorders Not otherwise classified – conditions not typically visible on imaging [3,5,6,9,11].

In CTD patients, this clean division becomes complicated. The FIGO classification encourages clinicians to consider the entire range of etiologies, looking beyond preconceptions or biases during diagnosis. This approach proves particularly valuable for CTD patients, whose underlying connective tissue abnormalities may influence multiple categories simultaneously.

For instance, coagulopathy (AUB-C) appears in up to 13% of women with heavy menstrual bleeding and up to 20% of women may have an underlying bleeding disorder. These percentages likely increase in CTD populations, where bleeding tendencies may stem from both structural vascular fragility and functional coagulation issues. Moreover, the tissue fragility characteristic of many CTDs can affect the structural integrity of the endometrium itself, creating a situation where AUB presents with features of both structural and non-structural causes.

According to FIGO data, clinicians initially categorize approximately 50% of AUB cases as having structural causes, but histopathological examination often reveals this number to be closer to 64%. This discrepancy highlights the challenge of accurate classification, especially in CTD patients where subtle structural abnormalities may be overlooked during initial assessment.

Overlap of AUB-L and AUB-E in Connective Tissue Disorders

Perhaps nowhere is the classification complexity more evident than in the overlap between leiomyoma-associated bleeding (AUB-L) and primary endometrial dysfunction (AUB-E) in connective tissue disorders. Leiomyomas (fibroids) represent the most common structural cause of AUB, with one study identifying them in 41.1% of AUB cases. Nevertheless, the relationship between fibroids and abnormal uterine bleeding remains incompletely understood.

A critical insight from recent research is that many women with fibroids have entirely normal bleeding patterns, suggesting that the mere presence of fibroids does not guarantee bleeding abnormalities. In contrast, submucous leiomyomas that distort the endometrial cavity (classified as AUB-LSM) demonstrate a more convincing connection to abnormal uterine bleeding than other types.

For CTD patients, this relationship becomes even more complex. The high prevalence of potential endometrial dysfunction means that those with AUB-L frequently have an element of AUB-E contributing to increased or aberrant menstrual blood loss. This overlap has important implications for therapy selection and effectiveness.

Additionally, in CTD patients who exhibit both fibroids and ovulatory dysfunction, the co-existing conditions may exacerbate menstrual loss. The endometrial environment in these patients often shows signs of inflammation and altered vascular function. Studies have identified increased inflammatory markers in both the eutopic endometrium and adenomyotic lesions of women with adenomyosis compared to healthy controls, suggesting inflammatory dysregulation that may be further amplified in CTD patients.

At the molecular level, women with AUB-E show decreased menstrual endometrial HIF1A protein compared to those with normal menstrual bleeding, along with a reduction in HIF1 downstream targets. Given that HIF1A deficiency delays endometrial repair in experimental models, the altered connective tissue environment in CTD patients likely compounds these healing deficiencies.

In accordance with FIGO recommendations, the PALM-COEIN system undergoes revision every three years to incorporate new evidence. As our understanding of CTD-related abnormal uterine bleeding and dysfunctional uterine bleeding evolves, future iterations may include specific considerations for this patient population, potentially creating subcategories that better reflect the unique pathophysiology of AUB in connective tissue disorders.

DISCUSSION

Pathophysiological Links Between CTD and Abnormal Uterine Bleeding

The underlying mechanisms that connect connective tissue disorders to abnormal uterine bleeding stem from fundamental alterations in tissue structure, immune function, and hormonal regulation. Understanding these pathophysiological links provides essential insights into why conventional approaches to abnormal uterine bleeding often fall short in CTD patients.

Vascular Fragility in Ehlers-Danlos Syndrome

Ehlers-Danlos Syndrome (EDS) presents a clear example of how structural connective tissue abnormalities directly contribute to abnormal uterine bleeding. At the core of this relationship lies the compromised vascular integrity characteristic of EDS, particularly in the vascular subtype. This subtype results from a mutation in the COL3A1 gene, which encodes type III procollagen—an essential component of blood vessel walls and hollow organs.

The vascular fragility in EDS manifests as a bleeding tendency that affects virtually all patients, albeit with varying severity. Almost all EDS patients display some degree of vascular fragility, which commonly presents as easy bruisability after minor trauma. Beyond superficial bruising, this vascular weakness extends to internal structures, including the uterine lining.

Women with EDS frequently experience menometrorrhagia (abnormal uterine bleeding) [4,5,6,19]. as part of their broader bleeding diathesis. This bleeding pattern stems from the inherent fragility of blood vessel walls and surrounding perivascular connective tissues. Consequently, the endometrial vasculature in EDS patients lacks the structural integrity necessary for proper hemostasis during menstruation.

Pregnancy poses substantial risks for women with vascular EDS specifically, with potential for severe complications including uterine rupture and life-threatening hemorrhage. The excessive risk of arterial tear or dissection makes conventional diagnostic approaches like angiography hazardous, necessitating non-invasive imaging techniques.

Autoimmune Endometrial Disruption in Systemic Lupus Erythematosus

Systemic Lupus Erythematosus (SLE) represents a fundamentally different mechanism through which abnormal uterine bleeding occurs in CTD patients. Rather than primary structural defects, SLE creates an autoimmune environment that directly impacts endometrial function and integrity.

The physiological inflammatory state of normal endometrium relies on a delicate balance of pro-inflammatory and pro-resolution mechanisms. In SLE, this balance is disrupted by systemic autoimmune processes. Notably, more than three-fourths of females with SLE experience menstrual changes, making this a significant clinical concern.

Several pathophysiological processes contribute to abnormal uterine bleeding in SLE:

1. Thrombocytopenia: Between 20% and 40% of people with SLE develop low platelet levels (thrombocytopenia) when the immune system mistakenly attacks platelets, leading to heavy menstrual bleeding.
2. Kidney involvement: Up to 40% of SLE patients develop lupus nephritis, causing increased production of prolactin, which may lead to spotting between periods.
3. Antiphospholipid syndrome: Research indicates that 40% of people with SLE have antiphospholipid antibodies, which can cause both serious blood clots and heavy periods.

Importantly, the endometrium itself becomes a target for autoimmune inflammation. The chronic inflammatory state characteristic of SLE affects the endometrium's repair capabilities, essentially creating what has been termed "autoimmune endometritis". This inflammation alters the normal cyclical injury-healing process of the endometrium, impairing tissue repair and contributing to abnormal uterine bleeding [12-14,16].

Hormonal Dysregulation in Mixed Connective Tissue Disease

Mixed Connective Tissue Disease (MCTD) presents a complex interplay between hormonal regulation and connective tissue dysfunction. This rare autoimmune disease is characterized by the presence of anti-U1-ribonucleoprotein antibodies and features of multiple connective tissue diseases, including SLE, systemic sclerosis, inflammatory myositis, and rheumatoid arthritis.

The etiology of MCTD remains unclear but involves interaction between genetic predisposition and environmental factors. Primarily, two pathogenic mechanisms have been proposed for anti-U1-RNP antibodies: direct binding to endothelial cells and immune complex formation. These mechanisms likely contribute to menstrual irregularities through vascular damage and tissue inflammation.

Hormonal imbalances play a critical role in MCTD-related abnormal uterine bleeding. Women with connective tissue diseases often demonstrate abnormal levels of reproductive hormones including androgens, estrogen, progesterone, and prolactin. These hormonal aberrations lead to irregular and unpredictable menstrual cycles, as well as breakthrough bleeding between periods.

Furthermore, sex hormones, particularly estrogens, actively modulate immune responses and affect the risk of developing or exacerbating autoimmune diseases. Supporting this relationship, women diagnosed with SLE after menopause typically experience less severe organ involvement and disease flares. This hormonal-immune interplay creates a complex environment where abnormal uterine bleeding emerges as a symptom of underlying hormonal dysregulation [19,20].

In the broader context of menstrual dysfunction in CTD patients, up to 90% of women with connective tissue diseases experience some form of menstrual irregularity. This high prevalence underscores the fundamental connection between sex hormones and immune function in CTD pathophysiology.

Clinical Presentation and Diagnostic Challenges in CTD-Associated AUB [4,5].

Women with connective tissue disorders face distinct challenges in the diagnosis and management of abnormal uterine bleeding that extend beyond standard clinical approaches. The intersection between tissue fragility, vascular dysfunction, and hormonal imbalances creates unique presentations that require specialized assessment.

Menstrual Irregularities in CTD vs Non-CTD Populations

The pattern and prevalence of menstrual disturbances differ substantially between women with connective tissue disorders and general populations. Studies of women with Ehlers-Danlos Syndrome (EDS) reveal that 33-76% report heavy menstrual bleeding (HMB) and 72-93% experience dysmenorrhea. These rates far exceed the general population prevalence of abnormal uterine bleeding, estimated at 14-25%.

Beyond prevalence differences, the clinical presentation often varies in severity and duration. Among CTD patients, 57.7% report dysmenorrhea, 50% complain of heavy menstrual bleeding, and 38.5% experience irregular menses. This contrasts with typical presentations in non-CTD populations, where heavy menstrual bleeding with normal duration is the predominant symptom, occurring in approximately 64% of cases [6,7].

Many women with hypermobility disorders also notice worsening joint laxity preceding their menstrual cycle. This phenomenon, linked to progesterone peaks during the luteal phase, illustrates how hormonal fluctuations directly influence connective tissue throughout the body, creating a cyclical pattern of symptom exacerbation unique to CTD patients.

Limitations of Standard Imaging in Hypermobile Patients

Standard diagnostic imaging for abnormal uterine bleeding faces significant challenges in patients with hypermobility disorders. Although transvaginal ultrasonography (TVUS) remains the first-line imaging modality for evaluating structural causes of AUB, its effectiveness diminishes in hypermobile patients due to anatomical variations and positioning difficulties.

The diagnostic accuracy of ultrasonography in identifying structural causes varies considerably between conditions. Research demonstrates that USG sensitivity reaches 100% for leiomyoma and malignancy but drops to 66% for polyps and 77.78% for adenomyosis. This variable performance becomes more problematic in hypermobile patients, where tissue laxity can alter anatomical relationships.

MRI offers superior diagnostic performance, with 100% sensitivity across all structural causes (polyps, adenomyosis, leiomyoma, and malignancy). Accordingly, MRI should be considered earlier in the diagnostic algorithm for CTD patients, particularly when USG findings conflict with clinical presentation. As one study concluded, "MRI is the best tool for determining the location, number, and characterization of lesions," providing valuable preoperative mapping benefits.

Endometrial Sampling Considerations in Coagulopathic CTD Cases

Endometrial sampling presents particular risks in CTD patients with coagulopathic tendencies. Standard sampling techniques include Pipelle biopsy (an outpatient procedure) and dilation and curettage (D&C), each with specific considerations in the CTD population.

First, technical challenges arise more frequently in these patients. Studies report technical failure rates of 1.3% with Pipelle biopsy and inadequate sampling in 4.6% of cases. These rates likely increase in CTD patients due to anatomical variations and tissue fragility. Furthermore, failed endometrial sampling is usually associated with pain or cervical stenosis, complications that may be more prevalent in CTD populations.

Patient positioning presents another challenge, as hypermobile patients often struggle to maintain standard examination positions. Additionally, uterine perforation, though rare in general populations (0.002-1.7%), may occur more frequently in patients with tissue fragility. One case report documented a perforation during D&C that required conservative management.

For these reasons, clinicians should consider alternatives like hysteroscopy-guided biopsies for CTD patients with high bleeding risk. Although more invasive, this approach offers direct visualization and potentially reduces perforation risk through precise targeting.

Methods:

Evidence from Recent Clinical Studies on AUB in CTD

Recent clinical studies examining abnormal uterine bleeding in connective tissue disorders (CTD) reveal significant gaps in both research methodology and patient management. First and foremost, a striking finding from real-world CTD cohorts shows that 52% of patients would not qualify for recruitment into phase-III clinical trials. This exclusion stems primarily from stringent eligibility criteria, with 90% of trials requiring patients to meet classification criteria for their respective diagnosis.

In young women with CTDs, dysmenorrhea and abnormal uterine bleeding represent the most common menstrual dysfunctions, arising from immune dysregulation, autoimmune changes, and inflammation. Yet management of these patients remains complex, requiring an interdisciplinary approach involving rheumatology, gynecology, and other specialties.

Study 1: AUB Prevalence and Assessment Challenges

Patient-reported outcome measures (PBOMs) form a crucial component in evaluating abnormal uterine bleeding, with a comprehensive review identifying 50 different instruments used to assess bleeding amount, bleeding-related symptoms, and menstrual bleeding-specific quality of life. Most notably, the majority of these instruments lack documentation of reliability, precision, or feasibility. Indeed, researchers found no satisfactory evidence that any single instrument completely addresses all eight important measurement properties needed for comprehensive assessment.

Current treatment approaches for CTD patients with abnormal uterine bleeding involve both hormonal options (synthetic and natural estrogens, progesterone, gonadotropin-releasing hormone analogs) and non-hormonal interventions (NSAIDs, tranexamic acid). Still, undifferentiated connective tissue dysplasia (UCTD) remains understudied due to clinical heterogeneity and lack of uniform diagnostic criteria [2,4,6].

Conclusion

The relationship between abnormal uterine bleeding and fibroids—often comorbid in CTD patients—continues to puzzle researchers. Obviously, the paradox that many women with fibroids maintain entirely normal bleeding patterns suggests complex underlying mechanisms beyond mere structural presence. As such, future studies must address the specific needs of CTD populations, whose unique pathophysiology may require tailored assessment tools and treatment approaches.

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