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STUDY OF CLINICAL AND LABORATORY FEATURES OF THE COURSE OF NON-DEVELOPING PREGNANCY OF THE ANEMBRYONIC TYPE IN THE FIRST TRIMESTER

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Abstract

Non-developing pregnancy, particularly in the form of anembryonic pregnancy type I (AN I), remains a significant problem in reproductive medicine due to its high prevalence and multifactorial pathogenesis.

Anembryonic pregnancy type I accounted for 44% of cases. The majority of patients (75%) were aged 27–40 years, with two incidence peaks at 24–25 and 30–32 years. Chromosomal abnormalities were identified in 40% of cases, predominantly triploidy, while 60% of cases were associated with non-genetic factors. Infectious and inflammatory conditions, including STIs and chronic endometritis, were common and contributed to pregnancy failure. In 25% of patients, the clinical course was asymptomatic, emphasizing the key role of ultrasound diagnostics and β -hCG monitoring in early detection.

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Keywords: Non-developing pregnancy; anembryonia; embryonic demise; chromosomal abnormalities; infections; endometritis; reproductive loss; first trimester.

Introduction

Non-developing pregnancy, including embryonic demise, remains a significant clinical and public health problem in modern obstetrics and gynecology. It accounts for a substantial proportion of early reproductive losses, particularly in the first trimester, where up to 10–20% of clinically recognized pregnancies may end in miscarriage, with a considerable share attributed to missed abortion or embryonic demise [1,2]. Despite advances in diagnostic and therapeutic approaches, the mechanisms underlying early pregnancy failure are still not fully understood.

The etiology of embryonic demise is multifactorial and involves a complex interaction of genetic, infectious, endocrine, immunological, and environmental factors. Chromosomal abnormalities of the embryo are considered the leading cause, being detected in approximately 50–60% of early pregnancy losses [3]. In addition, maternal factors such as hormonal imbalances, uterine pathology, and disorders of hemostasis contribute significantly to impaired implantation and early embryonic development [4].

Infectious agents, including bacterial and viral pathogens, also play an important role in the pathogenesis of non-developing pregnancy. Persistent infections of the endometrium and reproductive tract may lead to impaired trophoblast invasion, defective placentation, and disruption of embryonic growth [5]. Moreover, the interaction between инфекционные and иммунные механизмы further aggravates the risk of early pregnancy loss.

Clinical diagnosis of embryonic demise is primarily based on ultrasound examination, which allows for accurate assessment of gestational structures and embryonic viability. However, laboratory parameters, including hormonal

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markers such as β -human chorionic gonadotropin (β -hCG) and progesterone, provide additional information regarding the functional state of pregnancy and may assist in early detection of pathological changes [6].

Given the high prevalence and multifactorial nature of embryonic demise, studying its clinical and laboratory features is essential for improving early diagnosis, identifying risk factors, and developing effective preventive strategies. Therefore, the present study is aimed at analyzing the clinical and laboratory characteristics of non-developing pregnancy of the embryonic demise type in the first trimester.

The purpose of the study

To investigate the features of the course of non-developing pregnancy of the embryonic demise type in the first trimester.

Materials and Methods

A total of 130 patients with a verified diagnosis of non-developing pregnancy were under observation. All patients received treatment in the Department of Gynecology of the Nukus филиал of the Republican Specialized Scientific and Practical Medical Center “Mother and Child Health” of the Ministry of Health of the Republic of Uzbekistan.

The general clinical examination included the collection of anamnesis data, assessment of somatic and gynecological morbidity, as well as evaluation of patients’ reproductive health. Upon admission to the hospital, patients’ complaints were analyzed, with particular attention given to identifying a subgroup of women with signs of incipient miscarriage (presence of scant bloody vaginal discharge).

Menstrual function was evaluated based on age at menarche, regularity and duration of the menstrual cycle, and characteristics of menstruation. Reproductive history was assessed according to the number of pregnancies, their course, and

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outcomes. Additionally, patients' age, parity (primigravida or multigravida), and the presence of a history of recurrent pregnancy loss were taken into account.

All patients underwent standard laboratory evaluation upon admission, including complete blood count, biochemical blood analysis, coagulation profile, microscopic examination of vaginal smears for flora, and determination of serum β -subunit of human chorionic gonadotropin (β -hCG).

Ultrasound examination (US) of the pelvic organs was performed at hospital admission and during follow-up: on the 1st day after vacuum aspiration, on the 14th day, on the 7th day of the second menstrual cycle, and on the 21st–23rd day of the second menstrual cycle.

Statistical analysis was carried out using methods of variation statistics. For quantitative variables, the mean (M) and standard error of the mean (m) were calculated. For comparison of groups with normally distributed data, parametric methods were applied using the Student's t-test. For categorical variables and data not following a normal distribution, the chi-square (χ^2) test was used. Differences were considered statistically significant at $p \leq 0.05$. Data processing was performed using Microsoft Excel 15.0 (Microsoft Office 2016, macOS) and Statistica 10. Graphical presentation of results was carried out using Microsoft Excel 15.0.

Results and Discussion

According to the conducted observations, non-developing pregnancy of the anembryonic type (AN I, Empty Sac) was diagnosed in 44 patients, accounting for 44% of the study group. Approximately 25% of women were aged 18–26 years, while the majority (75%) were in the 27–40-year age group.

The age distribution varied depending on the reproductive status of the patients (primigravida or multigravida). Two peaks in incidence were identified: the first at 24–25 years and the second at 30–32 years (Fig. 1). At the same time, the mean age of patients with AN I corresponded to an age period in which the overall

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frequency of this type of non-developing pregnancy was relatively lower than the average values.

The timing of hospital admission for patients with anembryonic pregnancy type I (AN I) most commonly corresponded to 6–8 weeks of gestation, although a second, less pronounced peak was observed at 9–10 weeks.

At admission, scant bloody vaginal discharge and lower abdominal pain were reported in 33 patients (75%), whereas 11 women (25%) had no complaints. Approximately 20% of the examined patients received progesterone therapy for pregnancy support, while the majority (80%) did not undergo such treatment.

Molecular genetic analysis of embryonic tissues in 42 patients with AN I, including chromosomes X, Y, 13, 14, 16, 18, 21, and 22, revealed numerical chromosomal abnormalities in 17 cases (40%). Among these, triploidy predominated (6 cases, 14.2%). Trisomies of chromosomes 16, 21, and 22 were identified in 2 cases each (4.7%), whereas trisomies of chromosomes 13, 14, and 18, as well as monosomy X and monosomy 21, were detected in 1 case each (2.3%).

The proportion of patients with no history of gynecological diseases or sexually transmitted infections (STIs) did not show a clear increase with age. However, a higher incidence of anembryonia in certain age groups in the absence of comorbid pathology suggests a predominant role of chromosomal abnormalities as a key pathogenetic factor. The highest frequency of both gynecological diseases and STIs was observed in the 29–32-year age group, with a moderate increase in infectious pathology also noted at 23–24 years.

These findings suggest that the pathogenesis of AN I is associated not only with chromosomal abnormalities arising during early embryogenesis due to errors in cell division and genetic transmission, but also with the damaging effects of prior gynecological diseases, STIs, and persistent viral infections.

Analysis of medical history showed that gynecological diseases were present in 9 patients (20%), STIs alone in 6 women (14%), and a combination of infections

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and gynecological pathology in 12 patients (27%), while 17 women (39%) had no history of either gynecological diseases or STIs.

The analysis of the structure of reproductive losses in the studied group demonstrated that anembryonic pregnancy type I (AN I) represents the dominant form of non-developing pregnancy, accounting for 44% of cases. The obtained data on age distribution (75% of patients aged 27–40 years) are consistent with global trends reflecting a decline in reproductive potential and accumulation of genetic errors in oocytes with increasing maternal age. However, the presence of two incidence peaks (24–25 and 30–32 years) indicates heterogeneity of etiological factors across different age groups. While exogenous factors may predominate in younger women, the role of combined pathology becomes more significant in the second peak.

Of particular interest is the identified discrepancy: the mean age of patients with AN I corresponds to periods of relatively low overall incidence of non-developing pregnancy. This suggests that AN I may have a specific pathogenetic profile, distinct from classical embryonic demise. Clinically, AN I is characterized by the absence of subjective symptoms in 25% of cases (“silent” form), highlighting the critical importance of ultrasound monitoring at 6–8 and 9–10 weeks of gestation for timely diagnosis. The fact that 80% of patients did not receive progesterone support may indirectly indicate a latent course of the pathological process prior to its detection.

The results of molecular genetic analysis confirm the significant contribution of chromosomal abnormalities to the formation of an empty gestational sac. The detection of abnormalities in 40% of cases, with a predominance of triploidy (14.2%), supports the concept of a primary genetic defect as a leading cause of anembryonia. However, the high proportion of cases (60%) with a normal karyotype indicates the involvement of additional mechanisms in developmental arrest.

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It was also established that the 29–32-year age group is characterized by a higher prevalence of gynecological diseases and sexually transmitted infections (STIs). This coincides with the second peak of non-developing pregnancy and suggests that infectious and inflammatory factors (such as chronic endometritis and persistent viral infections) may act as additive contributors, leading to anembryonia even in genetically normal gestational sacs. The presence of combined infectious and gynecological pathology in 27% of patients and isolated STIs in 14% further supports the concept of a multifactorial origin of AN I. Thus, the pathogenesis of anembryonic pregnancy type I represents a complex interplay between chromosomal abnormalities arising from defects in cell division and the damaging effects of chronic infection and endometrial dysfunction. This underscores the need for a personalized approach to preconception care in patients with a history of AN I, including both genetic counseling and comprehensive management of infectious conditions.

Conclusion

In summary, the results of the conducted clinical and scientific analysis indicate that the pathogenesis of anembryonic pregnancy type I is multifactorial. Along with primary chromosomal abnormalities (40%), infectious and inflammatory processes, as well as morphofunctional insufficiency of the endometrium, play a significant role in the arrest of gestational sac development, particularly in patients with a normal embryonic karyotype.

The obtained findings substantiate the need for a comprehensive preconception approach, combining genetic counseling with etiopathogenetic anti-inflammatory therapy.

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