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Factors and pathogenetic mechanisms of male infertility in the Jalal-Abad Region of the Kyrgyz Republic

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Abstract. Male infertility remains a significant medical and social challenge, while regional data on its aetiological structure and pathogenetic mechanisms are limited. In the Jalal-Abad region of the Kyrgyz Republic, there is an urgent need to strengthen the evidence base for targeted diagnostic and preventive strategies. The aim of this study was to describe the aetiological structure and probable pathogenetic mechanisms of primary male infertility based on comprehensive clinical, laboratory, and instrumental assessments. A total of 110 men aged 20-38 years were examined, including 60 patients with primary infertility (history ≥12 months) and 50 healthy men who formed the control group. Clinical examinations, laboratory and hormonal analyses, semen evaluation, microbiological and serological diagnostics of infections, and ultrasonographic assessment of the genitourinary system were performed. The leading causes were infectious and inflammatory disorders of the genitourinary tract: among the

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60 examined men, chronic urethritis was detected in a considerable proportion (non-specific – 23.3%; specific forms: trichomonal – 6.7%, chlamydial – 1.7%, ureaplasmal – 21.7%, mycoplasmal – 18.3%, and Gardnerella-associated – 26.7%), while ultrasound signs of chronic prostatitis were observed in 26.7% of participants. The microbiological profile was characterised by the predominance of staphylococci and Gram-negative bacilli, frequent mixed infections (46.7%), and urogenital candidiasis (8.3%). Seropositivity to cytomegalovirus reached 31.7%, and to herpes simplex virus types 1 and 2 – 25.0%. Among anatomical and functional factors, varicocele (6.7%), orchiepididymitis (5.0%), cryptorchidism (1.7%), and obstruction of the seminal ducts (3.3%) were identified; endocrine abnormalities included secondary hypogonadism (5.0%). Azoospermia was detected in 8.3% of cases, mainly associated with secondary hypogonadism or obstruction. Systemic inflammation was confirmed in some patients by leukocytosis and an elevated erythrocyte sedimentation rate, while leukocyturia was noted intermittently. The findings support the importance of early screening for sexually transmitted infections, chronic prostatitis, and hormonal disorders in the diagnostic pathways for primary male infertility in regional practice, enabling the personalisation of aetiotropic and pathogenetic therapy

Keywords: male fertility disorders; reproductive function; urogenital infections; *Chlamydia*; *Ureaplasma*; *Mycoplasma*

Introduction

Male fertility has become a significant public health issue globally and in Central Asian countries. Regions with uneven access to specialised care, including the Jalal-Abad region, continue to lack validated data on the structure of causes and the relative contribution of infectious-inflammatory, anatomical-functional, endocrine and behavioural factors. This necessitated a study with clinical and laboratory verification of key etiopathogenic mechanisms and clarification of diagnostic priorities.

According to the World Health Organisation [1], the burden of reproductive dysfunction in couples remains high, underscoring the need for accessible, quality care and data for decision-making. H. Levine *et al*. [2] in a global systematic review and meta-regression analysis showed that in the 21st century, the trend towards a decline in sperm parameters in men from different regions of the world continues, and the rate of change may have accelerated, requiring focused research into the causes and regional variability. Behaviour and lifestyle are consistently considered modifiable risk factors. S.W. Fan *et al.* [3] demonstrated that tobacco smoking in men seeking treatment for reproductive problems is associated with a statistically significant deterioration in standard ejaculate parameters, which highlights the importance of smoking cessation programmes in patient management. S.S. Bahreiny *et al*. [4] conducted a systematic review and meta-analysis involving 6,264 men on the impact of air pollution on sperm quality. The levels of PM2.5 and PM10 particulate matter in the environment were analysed. The results showed a significant decrease in ejaculate volume, concentration, total number, and motility of spermatozoa with an increase in particle concentration. Exposure to PM2.5 and PM10 was accompanied by statistically significant negative changes in sperm parameters, which the authors attributed to oxidative stress and damage to spermatogenic cells. The study emphasised the role of atmospheric pollution as an independent risk factor for male fertility disorders and the need to take environmental conditions into account when assessing reproductive health.

The evidence on endocrine disruptors remains inconsistent, but L. Mínguez-Alarcón *et al*. [5] note in their review for clinical practice the biological plausibility of the influence of bisphenols and phthalates on hormonal regulation and spermatogenesis and the need to standardise exposure assessment. Infectious and inflammatory processes of the urogenital tract are considered one of the leading and potentially eliminable risk factors. D.A. Paira *et al*. [6] in a large cross-sectional study (5,464 patients) linked positivity for *Chlamydia trachomatis*, *Ureaplasma* spp. and *Mycoplasma hominis* with infertility in couples, pointing to a lack of data on the magnitude of the effect and its clinical significance. K. Khalafalla *et al*. [7] conducted a systematic review of 70 studies to assess the association of sexually transmitted infections (STIs) with male subfertility. The analysis, performed according to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) protocol, showed conflicting data: most studies did not find a clear relationship between STIs and sperm parameters. The exceptions were *Ureaplasma* and *Mycoplasma*, for which the most reliable evidence of an effect on spermatogenesis and sperm motility was obtained. The authors Y.-Y. Wan *et al*. [8] compared sperm parameters in men with positive and negative *Ureaplasma urealyticum* status in seminal fluid and the pregnancy and birth outcomes of their partners. No statistically significant differences in ejaculate volume, concentration, motility, sperm morphology, leukocyte level, and antisperm antibodies were found between the groups ($P > 0.05$). The only difference was a higher sperm nuclear immaturity index in the presence of infection ($P = 0.04$), which may indicate the influence of *Ureaplasma* on

spermatogenesis processes. At the same time, pregnancy and birth outcomes did not differ. The authors concluded that *Ureaplasma urealyticum* infection does not significantly affect the effectiveness of intrauterine insemination, but may affect sperm quality at the subcellular level.

Along with infections, the contribution of anatomical, functional, and inflammatory conditions of the genitourinary system is confirmed by clinical data. A. Agarwal *et al.* [9] showed in a systematic review and meta-analysis that correction of varicocele in infertile men leads to a statistically significant improvement in sperm concentration, motility, and morphology compared to the untreated control group, with no differences in ejaculate volume. The authors emphasised the high level of evidence for these data and confirmed the role of venous stasis as a pathophysiological mechanism of spermatogenesis impairment. J. Rosellen *et al*. [10] reported a decrease in ejaculate quality in patients with chronic prostatitis, discussing possible mechanisms through inflammation, reactive oxygen species, and changes in prostate secretory function. These results emphasise the need for a comprehensive assessment and combination of etiotropic and pathogenetic approaches.

The aim of this study was to systematise the spectrum and relative contribution of infectious-inflammatory, anatomical and functional, endocrine, and behavioural factors in men of reproductive age with long-term fertility disorders in the Jalal-Abad region, as well as to identify priorities for optimising the diagnostic pathway.

Materials and Methods

The study was conducted in 2022-2024 at the Research Institute of Medical and Biological Problems of the Southern Branch of the National Academy of Sciences of the Kyrgyz Republic and the Regional Centre for Reproductive Health in Jalal-Abad. Ethical approval was obtained from the local bioethics committee, and the study was conducted in accordance with the principles of The Declaration of Helsinki [11]. All participants signed an informed consent form for participation in the study and processing of personal data. The examinations were performed using approved national and international protocols of the World Health Organisation (WHO), European Association of Urology (EAU), International Council for Standardisation in Haematology (ICSH), Centers for Disease Control and Prevention (CDC), and Clinical and Laboratory Standards Institute (CLSI) [12-18].

The study included 110 men aged 20-38 years, permanently residing in the Jalal-Abad region. The main group consisted of 60 patients with clinically confirmed primary infertility (absence of pregnancy in a partner with regular sexual intercourse without contraception for at least 12 months). The control group consisted of 50 men with preserved fertility and at least one child. Selection was carried out by continuous inclusion among patients who sought treatment for reproductive dysfunction. The exclusion criteria were surgery on the scrotum or prostate, severe chronic diseases, active inflammatory processes, alcoholism, and smoking for more than 10 years. The complex of clinical, laboratory, and instrumental methods used is presented in Table 1.

Table 1. Main research methods and their characteristics

Results	Characteristics/purpose	Main parameters assessed	Standards used
Clinical examination	History taking, physical examination, palpation of scrotal organs	Varicocele, epididymo-orchitis, cryptorchidism, anatomical anomalies	[14]
General clinical analyses (GCA, ESR, and GUA)	Assessment of general condition and inflammatory changes	Leucocytes, ESR (by Westergren), leucocyturia, bacteriuria	[15,16]
Bacterioscopy of cultures (Urethra, prostate secretion, ejaculate)	Identification of microflora and microbial susceptibility to antibiotics	Presence of cocci, Gram-negative flora, fungi; interpretation of MIC as per CLSI	[18]
PCR-Diagnostics (NAAT)	Identification of STI-causing agents	Chlamydia trachomatis, Ureaplasma spp., Mycoplasma spp., Trichomonas vaginalis (as indicated)	[13,17]
EIA (Serology)	Determination of antibodies to viruses	IgG to CMV and HSV 1/2	
Spermogram	Ejaculate analysis based on WHO standards (6th ed.)	Volume ≥ 1.4 ml; concentration ≥ 16 million/ml; total motility ≥ 42%; progressive motility ≥ 30%; normal morphology ≥ 4% (5th percentile in fertile men, not a diagnostic threshold)	[12]
Hormonal profile (as indicated)	Assessment of endocrine disturbances	Testosterone, FSH, LH, PRL; detection of secondary hypogonadism (↓T with ↑ FSH/LH)	[14]
Ultrasound scan of the genitourinary system	Examination of scrotal organs, prostate, kidneys, and bladder (B-mode, CDM)	Varicocele, chronic prostatitis, cysts, dilatation of the pampiniform plexus veins	

Note: GCA – general blood analysis; ESR – erythrocyte sedimentation rate (Westergren method); GUA – general urine analysis; PCR/NAAT – nucleic acid amplification tests; EIA – enzyme-linked immunoassay; IgG – immunoglobulin G; MIC – minimum inhibitory concentration; CMV – cytomegalovirus; HSV – herpes simplex virus; FSH – follicle-stimulating hormone; LH – luteinising hormone; PRL – prolactin; T – testosterone; B-mode – grey-scale imaging; CDM – colour Doppler mapping

Source: compiled by the authors

Internal quality controls and certified reagents were used for all laboratory procedures. Ultrasound examinations were performed using Philips HD11XE (Netherlands) and Mindray DP-50 (China) devices with linear and convex probes in B-mode and CDK mode. All examinations were performed by an ultrasound physician with > 10 years of experience. Statistical processing was performed using IBM SPSS Statistics v.26. Categorical variables were compared using Fisher's exact test; Pearson's χ^2 was used for expected frequencies ≥ 5. The effect was presented as the odds ratio (OR) with a 95% confidence interval (CI); for zero cells, the Haldane-Anscombe correction was used. For single proportions, 95% confidence intervals were calculated using the Wilson method. All tests were two-tailed; differences were considered statistically significant at $p < 0.05$. The Benjamini-Hochberg procedure (False Discovery Rate – FDR) was applied to control for multiple comparisons. A study of fructose levels in ejaculate to assess seminal vesicle function was planned but not performed due to the lack of a spectrophotometer, which is considered one of the limitations of the study.

Results

General clinical analyses. General blood analysis was within reference limits in 46 of 60 men, 76.7%. Signs of a systemic inflammatory response, including moderate leukocytosis and accelerated erythrocyte sedimentation rate, were observed in 14 out of 60 (23.3%). For this proportion, the 95% confidence interval was 14.4-35.4%. According to the general urine analysis, normal values were found in 48 out of 60 (80.0%). Leukocyturia was recorded in 2 out of 60 (3.3%) (95% confidence interval 0.9-11.4%). Elevated salt content was noted in 10 out of 60, 16.7% (95% confidence interval 9.3-28.0%). The data are consistent with the predominance of chronic rather than acute inflammatory conditions.

Urogenital infection: microbiology, PCR, serology. Microscopy of urethral smears revealed a pronounced inflammatory response and polymicrobial associations in 28 out of 60 (46.7%; 95% confidence interval 34.8-59.0%). A predominance of Gram-positive cocci and Gram-negative rods was noted. *Gardnerella* spp. was found in 16 out of 60 (26.7%); yeast-like fungi were found in 5 out of 60 (8.3%). The results of urine, prostate secretion, and ejaculate cultures confirmed the predominance of staphylococci and Gram-negative rods, with a significant proportion of mixed growth. The picture corresponds to chronic inflammation and probable colonisation of several sections of the urogenital tract.

Trichomonas vaginalis was verified in 4 out of 60 (6.7%). *Chlamydia trachomatis* was found in 1 out of 60 (1.7%) (95% confidence interval 0.3-8.9%). *Ureaplasma* spp. was detected in 13 out of 60 (21.7%, 95% confidence interval 13.1-33.7%). *Mycoplasma* spp. was detected in 11 out of 60 (18.3%, 95% confidence interval 10.6-30.1%). A profile with a predominance of ureaplasma and mycoplasma over chlamydia and trichomonas is typical for outpatient cohorts and reflects the polymicrobial nature of chronic processes. CMV IgG was detected in 19 out of 60, 31.7% (95% confidence interval 20.8-45.0%). HSV-1/2 IgG was detected in 15 out of 60, 25.0% (95% confidence interval 15.4-38.0%). Serostatus reflects past or latent infection and provides a background for interpreting chronic inflammation of the prostate gland and appendages.

Ultrasound and instrumental examinations. Ultrasound signs of chronic prostatitis were recorded in 16 out of 60, 26.7% (95% confidence interval 17.0-39.1%). Signs of chronic pyelonephritis were detected in 12 out of 60, 20.0% (95% confidence interval 11.6-32.0%). Cystic formation of the prostate gland was found in 1 out of 60, 1.7%. Clinically and based on the data, chronic urethritis was established in 14 out of 60, 23.3%. A comparison of instrumental and microbiological results indicates a high proportion of chronic urogenital inflammation in the cohort.

Hormonal indicators. Secondary hypogonadism was diagnosed in 3 out of 60 (5.0%). Despite the small proportion, this phenotype demonstrates a close connection with extreme spermatogenesis disorders. No other hormonal disorders of independent diagnostic significance were identified.

Spermogram. Azoospermia was detected in 5 out of 60 (8.3%, 95% confidence interval 3.6-18.1%). In 3 cases, azoospermia was combined with secondary hypogonadism, and in 2 cases with obstruction of the seminal ducts. In the remaining 55 observations, azoospermia was not recorded. The lack of complete distributions by concentration, progressive motility, and morphology limits the quantitative analysis of all parameters, but the association of extreme phenotypes with endocrine and obstructive causes appears to be consistent.

Concomitant conditions. Varicocele was detected in 4 out of 60 (6.7%). Orchiepididymitis – in 3 out of 60 (5.0%). Cryptorchidism was found in 1 out of 60 (1.7%). Obstruction of the seminal ducts was found in 2 out of 60 (3.3%). These conditions are potentially modifiable and often coexist with infectious and inflammatory components, enhancing the cumulative effect on spermatogenesis.

Associative analysis of extreme phenotypes. The link between azoospermia and the endocrine mechanism is confirmed as follows. Among patients with secondary hypogonadism, azoospermia was observed in 3 out of 3 (100.0%); among patients without hypogonadism – in 2 out of 57 (3.5%). According to Fisher's exact test, $p = 0.00029$. This indicates a strong association between pronounced hormonal dysregulation and the complete absence of sperm in the ejaculate.

The obstructive mechanism also demonstrated the expected relationship. In the presence of obstruction, azoospermia was observed in 2 out of 2 (100.0%); in its absence, it was observed in 3 out of 58 (5.2%).

According to Fisher's exact test, p = 0.0056. The results are consistent with the clinical logic of obstructive azoospermia. The power of the comparisons is limited by the small numbers, and the conclusions should be interpreted as research findings. The total distribution of the identified causes is presented in Table 2. The summary structure includes 58 out of 60 patients, as the data volume was insufficient for aetiological attribution in two cases. One patient may have had several simultaneous factors, including infectious-inflammatory and anatomical-functional ones, so the sum of the shares may exceed 100%.

Table 2. Range of identified causes of infertility

Identified causes of infertility	Number of patients (n = 58)	
	Abs.	%
Chronic prostatitis, exacerbation stage	9	15.0
Chronic prostatitis, remission stage	7	11.67
Chronic pyelonephritis, exacerbation stage	5	8.33
Chronic pyelonephritis, remission stage	7	11.67
Chronic urethritis, non-specific	14	23.33
Chronic urethritis, specific, including:	28	46.67
└ Trichomonal	4	6.67
└ Chlamydial	1	1.67
└ Ureaplasmal	13	21.67
└ Mycoplasmal	11	18.33
└ Gardnerella	16	26.67
Candidiasis of the genitourinary system	5	8.33
Prostatic cyst	1	1.67
Orchoepididymitis	3	5.0
Varicocele	4	6.67
Secondary hypogonadism	3	5.0
Obstruction of the deferent ducts	2	3.33
Azoospermia	5	8.33
Cryptorchidism	1	1.67

Source: compiled by the authors as a result of research

The data obtained indicate that the proportion of urogenital inflammation confirmed microbiologically and instrumentally remains high. PCR more often detects *Ureaplasma* spp. and *Mycoplasma* spp. than *Chlamydia trachomatis* and *Trichomonas vaginalis*. The serostatus for CMV and HSV corresponds to the population background and requires careful interpretation when assessing causal relationships. The most severe phenotype, azoospermia, is almost entirely associated with secondary hypogonadism and obstruction of the seminal ducts within the cohort. The contribution of anatomical and functional conditions (varicocele, orchiepididymitis) appears to be moderate, but when combined with infectious and inflammatory stress, it may increase the risk of adverse changes in sperm parameters. Chronic prostatitis causes not only a delay in spermatogenesis, but also a significant deterioration in its quality. These changes are particularly pronounced in patients with a disease duration of more than five years, which negatively affects the functional state of the male reproductive system [19,20]. Based on the summary structure (Table 2), the results were compared with a control group of fertile men (n = 50) whose indicators corresponded to those expected for a healthy population. Quantitative differences and statistical criteria are summarised in Table 3.

Table 3. Comparison of the main and control groups by aetiological positions

Indicator	Main group (n=58) Abs., %	Control group (n=50) Abs., %	OR (95% CI)	p
Chronic prostatitis, exacerbation stage	9 (15.5%)	0 (0.0%)	19.38 (1.10-342.13)	0.00336
Chronic prostatitis, remission stage	7 (12.1%)	3 (6.0%)	2.15 (0.53-8.80)	0.33445
Chronic pyelonephritis, exacerbation stage	5 (8.6%)	0 (0.0%)	10.38 (0.56-192.62)	0.06011
Chronic pyelonephritis, remission stage	7 (12.1%)	1 (2.0%)	6.73 (0.80-56.69)	0.06613
Chronic urethritis, non-specific	14 (24.1%)	1 (2.0%)	15.59 (1.97-123.46)	0.00068
Chronic urethritis, specific, including:	28 (48.3%)	4 (8.0%)	10.73 (3.42-33.70)	<0.00001
└ Trichomonal	4 (6.9%)	0 (0.0%)	8.34 (0.44-158.82)	0.12214
└ Chlamydial	1 (1.7%)	0 (0.0%)	2.63 (0.10-66.14)	1.00000
└ Ureaplasmal	13 (22.4%)	2 (4.0%)	6.93 (1.48-32.45)	0.01004

Table 3. Continued

Indicator	Main group (n=58) Abs., %	Control group (n=50) Abs., %	OR (95% CI)	p
└ Mycoplasmal	11 (19.0%)	1 (2.0%)	11.47 (1.42-92.34)	0.00522
└ Gardnerella	16 (27.6%)	1 (2.0%)	18.67 (2.37-146.74)	0.00030
Candidiasis of the genitourinary system	5 (8.6%)	1 (2.0%)	4.62 (0.52-40.97)	0.21340
Prostatic cyst	1 (1.7%)	0 (0.0%)	2.63 (0.10-66.14)	1.00000
Orchoepididymitis	3 (5.2%)	1 (2.0%)	2.67 (0.27-26.54)	0.62215
Varicocele	4 (6.9%)	2 (4.0%)	1.78 (0.31-10.14)	0.68395
Secondary hypogonadism	3 (5.2%)	0 (0.0%)	6.37 (0.32-126.36)	0.24714
Obstruction of the deferent ducts	2 (3.4%)	0 (0.0%)	4.47 (0.21-95.32)	0.49810
Azoospermia	5 (8.6%)	0 (0.0%)	10.38 (0.56-192.62)	0.06011
Cryptorchidism	1 (1.7%)	0 (0.0%)	2.63 (0.10-66.14)	1.00000

Note: p – level of statistical significance of differences between groups (p < 0.05 indicates a significant difference, p ≥ 0.05 – the difference is statistically insignificant and may be random)
Source: compiled by the authors as a result of research

Intergroup comparison based on aetiological positions shows that differences between the cohort of men with primary infertility and fertile controls are primarily due to the infectious-inflammatory domain of the lower urogenital tract. Statistically significant associations with infertility were found for: chronic urethritis (nonspecific) – OR 15.59; 95% CI 1.97-123.46; p = 0.00068; the total category of "specific urethritis" – OR 10.73; 3.42-33.70; p < 0.00001; as well as its subtypes of ureaplasma – OR 6.93; 1.48-32.45; p = 0.01004, mycoplasma – OR 11.47; 1.42-92.34; p = 0.00522, and gardnerella urethritis – OR 18.67; 2.37-146.74; p = 0.00030. These estimates are consistent with the microbiological profile of the main group (increased proportion of polymicrobial associations and mixed growth) and indicate a clinically relevant association between urethral inflammation, including that involving opportunistic microorganisms, and the infertility phenotype. The position "chronic prostatitis, acute stage" also demonstrates a pronounced association (OR 19.38; 1.10-342.13; p = 0.00336), while the "remission stag" is statistically indistinguishable from the control (p = 0.33445). This indirectly supports the thesis of greater clinical significance of active/exacerbated forms of prostate inflammation compared to latent progression, when ultrasound signs may persist without a concomitant clinical equivalent.

For chronic pyelonephritis, the patterns are unidirectional (increased OR during exacerbation and remission), but due to small numbers and wide confidence intervals, statistical significance is not achieved at the level of p < 0.05 (p = 0.06011 and p = 0.06613, respectively). Such lines should be interpreted as hypothesis-generating and requiring an increase in the sample size. Anatomical and functional factors (varicocele, orchiepididymitis, cryptorchidism), as well as rare nosologies (prostate cyst), did not show statistically confirmed differences. The lack of significance in these positions may reflect either a real moderate association or insufficient power to detect small/medium-sized effects.

The endocrine contribution (secondary hypogonadism) and obstructive mechanism (obstruction of the seminal ducts) in Table 3 did not reach statistical significance according to the intergroup criterion, which is explained by zero events in the control group and small absolute numbers. At the same time, intragroup analysis of extreme phenotypes showed their close association with azoospermia (p = 0.00029 and p = 0.0056, respectively). This highlights the different roles of intergroup and intragroup comparisons: the former record the contribution of a factor to the risk of infertility at the population level, while the latter record its association with the severity of spermatogenic disorders within the infertility cohort. Interpretation of effects with zero events in the control requires caution: OR in these rows are known to be high, and confidence intervals are wide due to small numbers. The study used the Haldane-Anscombe correction, which is correct for calculating OR, but it is advisable to confirm the final conclusions on such positions on an expanded sample and, if possible, in multivariate models taking into account age, duration of infertility, smoking, and other accompanying variables.

Taken together, the data in Table 3 indicate that the most reproducible and statistically confirmed area of difference is inflammatory and infectious processes in the distal parts of the urogenital tract (urethritis of various aetiologies) and active inflammation of the prostate gland. These domains are biologically consistent with the expected mechanisms of influence on spermatogenesis (inflammation, local oxidative stress, disorders of the secretory function of the prostate and seminal vesicles), while the proportion of "classical" STIs (chlamydia, trichomoniasis) remains low and does not form significant intergroup differences. The CMV/HSV serostatus corresponds to the population background and, based on the study design, does not provide grounds for causal attribution. The combination of results indicates the leading role of infectious and inflammatory processes in the distal parts of the

urogenital tract and active inflammation of the prostate gland in the structure of associations with male infertility at the population comparison level; severe spermatogenic phenotypes within the cohort are predominantly associated with endocrine and obstructive mechanisms.

Discussion

The observed association of infertility with polymicrobial urethral processes and an increased proportion of *Ureaplasma/Mycoplasma* is consistent with the data of M. Frølund *et al*. [21], which showed strong positive correlations between bacteria associated with bacterial vaginosis and ureaplasma load, which was interpreted as a pH-mediated effect of urease activity. At the same time, the absence of differences in the prevalence of BV taxa between idiopathic non-gonococcal urethritis and the control group reflected a different endpoint (urethritis versus reproductive outcome), different biomaterial (first-void urine versus comprehensive assessment), and the location of the process (urethra versus prostate/seminal vesicle involvement). Consequently, polymicrobial ecology is necessary but not sufficient; the clinical effect depends on the activity of inflammation and proximal involvement.

Key observations by E.L. Plummer *et al.* [22] were consistent with the polymicrobial concept of the lower tract: the composition of the urethral microbiota differed in idiopathic non-gonococcal urethritis and varied depending on the sex of the sexual partner. Taxa associated with idiopathic urethritis in men who have sex with men (MSM) were noted, with an increased relative abundance of *Haemophilus influenzae*, while in men who have sex with women (MSW), Corynebacterium was noted, as well as the dominance of a number of bacteria in cases but not in controls, including *Ureaplasma*, *Staphylococcus haemolyticus*, *Streptococcus pyogenes*, *Escherichia*, and *S. pneumoniae/pseudopneumoniae*. These data supported a polymicrobial profile and the involvement of *Ureaplasma*, which was consistent with the increased proportion of *Ureaplasma/Mycoplasma* in the infertility cohort, although the endpoints (urethritis versus reproductive outcome), matrix (first-void urine versus comprehensive examination), and methodology (16S versus targeted tests) differed.

A systematic review and meta-analysis by C. Cheng *et al*. [23] confirmed a statistically significant association between male infertility and genital mycoplasmas: *Mycoplasma genitalium* (pooled odds ratio, OR 3.44; 95% confidence interval, CI 1.78-6.64), *Mycoplasma hominis* (OR 1.84; 95% CI 1.01-3.34) and *Ureaplasma urealyticum* (OR 3.28; 95% CI 2.08-5.18), while Ureaplasma parvum showed no association (OR 1.67; 95% CI 0.95-2.95). The quality of the studies was assessed using the Newcastle-Ottawa scale; regional heterogeneity was noted (stronger associations in samples from China), indicating the influence of design, diagnosis and epidemiology. These results were consistent with the increased prevalence of *Ureaplasma/Mycoplasma* in men with infertility in the present study and explained the marked intergroup differences in "specific urethritis". A limitation of the comparison was the inability to differentiate between *U. urealyticum* and *U. parvum* in the current data set, which prevented direct validation of species-specific effects.

F.L. Nassan *et al*. [24] examined the impact of diet and individual food components on male fertility – from dietary patterns and antioxidant supplements to types of fats and consumption of dairy/meat products – in relation to sperm parameters, hormonal profile, and clinical reproductive outcomes. The authors demonstrated that adherence to a "pro-fertility" diet (fish/seafood, nuts, whole grains, fruits and vegetables, poultry) and adequate intake of omega-3 fatty acids were associated with better sperm parameters and more favourable reproductive outcomes, while excess trans and saturated fats were associated with poorer parameters. Antioxidant supplements were considered potentially beneficial, especially in couples undergoing ART, although there was no consensus on optimal compositions and doses. Data on dairy/meat products remained inconsistent; high-quality randomised studies are needed to develop clear clinical recommendations for men.

A comparison of the results of this study with the review by O. Batiha *et al*. [25] showed methodological consistency at the mechanism level: in 2020, a hypothesis was outlined about the possible effect of SARS-CoV-2 on male fertility through the expression of ACE2/TMPRSS2 in the testicle, fever, and endocrine shifts, while simultaneously reminding of the reproductive relevance of other viruses (HPV, HSV, HIV) in the absence of direct data on COVID-19 at that time. Later reviews and meta-analyses by Y. Xie *et al*. [26] clarified the picture: SARS-CoV-2 infection was associated with a transient deterioration in basic sperm parameters (volume, concentration, motility, morphology), especially in the short term after the disease. At the same time, no evidence of an adverse effect of vaccination on spermogram was obtained – data on inactivated and mRNA vaccines, as well as observations in sperm donors, showed no significant changes; this position is confirmed by a recent clinical review by Y.C. Ma *et al.* [27] on reproduction. In the cohort analysed, CMV/HSV serostatus was interpreted as a population background, and causal links between reproductive outcomes and COVID-19 were not assessed; nevertheless, the identified leading role of urogenital inflammation is consistent with the pathophysiological contour proposed for COVID-19 (inflammation/oxidative stress and subsequent damage to spermatozoa), described in mechanistic reviews.

The study by K. Gill *et al*. [20] assessed the impact of sedentary employment on the integrity of sperm nuclear DNA by comparing men who spent ≥ 50% of their working time sitting (n = 152) with participants

with less sedentary work (< 50%, n = 102). No statistical differences were found in standard ejaculate parameters, whereas the proportion of spermatozoa with DNA fragmentation (SDF, Halosperm) was higher in sedentary work (median 21.00% vs. 16.50%); low SDF levels were less common (0-15%: 27.63% vs. 45.10%) and high levels were more common (> 30%: 30.92% vs. 16.67%), with more than a twofold increase in the risk of high SDF (OR ≈ 2.24). The authors concluded that sedentary work (≥ 50% of working time) is associated with impaired sperm nuclear DNA integrity in the absence of differences in standard semen analysis; the proposed mechanism is testicular heat stress and disruption of chromatin remodelling in spermatogenesis.

The combination of the sources reviewed forms a consistent picture: the infectious-inflammatory domain of the distal sections and active inflammation of the prostate are statistically and biologically associated with reproductive disorders; severe phenotypes of spermatogenesis are more often associated with endocrine and obstructive causes requiring targeted additional examination and correction; Modifiable lifestyle factors – diet, past infections, nature of employment – affect fertility mainly through oxidative stress and damage to sperm DNA, which justifies expanding the laboratory panel beyond the standard semen analysis (e.g., SDF/ORP assessment). These findings justify a clinical and diagnostic focus on a multi-level approach: a unified PCR panel and microbiology, assessment of prostate inflammation, screening for endocrine and obstructive causes, and measurement of oxidative stress markers and sperm DNA integrity; in parallel – targeted lifestyle modification and nutritional support.

Conclusions

The results of the study confirm the complex and multifactorial nature of male infertility, in which infectious and inflammatory processes of the genitourinary system play a key role. The data obtained indicate that the most common clinical forms associated with fertility disorders are chronic urethritis and prostatitis, often occurring as combined or mixed infections. The high prevalence of ureaplasma, mycoplasma and gardnerella infections highlights their significant role in the development of pathological changes in spermatogenesis.

An important pathogenetic mechanism has been identified as the effect of a prolonged inflammatory process on the condition of the prostate and epididymis, leading to impaired secretion and deterioration in sperm quality. In combination with possible anatomical and functional disorders, such as varicocele or the consequences of orchiepididymitis, this forms the basis for the development of persistent forms of infertility. The results also demonstrate that endocrine disorders and functional disorders of spermatogenesis occur predominantly in cases of pronounced changes in the spermogram, including cases of azoospermia and severe oligozoospermia. The study substantiated the need for a comprehensive diagnostic approach, including microbiological examination of ejaculate, serological tests, hormonal assessment, and ultrasound examination of the scrotum and prostate. This approach ensured early detection of aetiological factors and allowed for the development of individualised treatment and prevention programmes for male infertility.

The data obtained from clinical, instrumental, and laboratory examinations are not exhaustive and require further in-depth study, given the growing medical and social significance of the problem of declining male reproductive capacity. The current understanding of the pathogenesis of male infertility requires a comprehensive approach that takes into account both identified factors and existing gaps in knowledge. In particular, it is necessary to continue fundamental research aimed at assessing the impact of generally toxic, carcinogenic and teratogenic agents, including chemical pollutants and radioactive substances, on the male reproductive system.

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Conflict of Interest

None.

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Кыргыз Республикасынын Жалал-Абад облусундагы эркектердин тукумсуздугунун факторлору жана патогенетикалык механизмдери

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Аннотация. Эркектердин тукумсуздугу маанилүү медико-социалдык көйгөй бойдон калууда, ал эми анын себептик түзүлүшү жана патогенетикалык механизмдери боюнча аймактык маалыматтар жетишсиз. Кыргыз Республикасынын Жалал-Абад облусунда максаттуу диагностика жана алдын алуу боюнча далилдүү негизди жаңылоо зарыл. Бул изилдөөнүн максаты – комплекстүү клиникалык, лабораториялык жана инструменталдык текшерүүнүн негизинде эркектердеги баштапкы фертилдүүлүктүн бузулушунун этиологиялык түзүлүшүн жана ыктымал патогенетикалык механизмдерин аныктоо болгон. Изилдөөгө 20–38 жаштагы 110 эркек катышкан, алардын ичинен 60ы баштапкы тукумсуздук (анамнези ≥12 ай) менен, ал эми 50ү саламат эркектер болуп, көзөмөл тобун түзгөн. Клиникалык кароо, лабораториялык жана гормоналдык анализдер, спермограмма, микробиологиялык жана серологиялык инфекцияларды аныктоо, ошондой эле заара-жыныс системасынын ультраүндүү изилдөөсү жүргүзүлгөн. Башкы себептердин көпчүлүгүн заара-жыныс системасынын инфекциялык-сезгенүү оорулары түздү: 60 текшерилген эркектин арасында өнөкөт уретрит көпчүлүк бөлүгүндө аныкталган (тубаса эмес түрү – 23,3 %; спецификалык түрлөрү: трихомоноздук – 6,7 %, хламидиялык – 1,7 %, уреаплазмалык – 21,7 %, микоплазмалык – 18,3 %, гарднереллездук – 26,7 %); ультраүндүү изилдөө боюнча өнөкөт простатиттин белгилери 26,7 % учурларда байкалган. Микробиологиялык профилде стафилококтор менен грамтерс бактериялардын үстөмдүгү, аралаш флоранын көп кездешүүсү (46,7 %) жана заара-жыныс системасынын кандидозу (8,3 %) аныкталды. Цитомегаловируска серопозитивдүүлүк 31,7 %, ал эми жөнөкөй герпес вирусунун 1- жана 2-типтерине – 25,0 % түздү. Анатомиялык-функционалдык факторлор арасында варикоцеле (6,7 %), орхоэпидидимит (5,0 %), крипторхизм (1,7 %), уруктук жолдордун тосулушу (3,3 %) катталды; эндокриндик бузулуулар экинчи даражадагы гипогонадизм (5,0 %) менен көрсөтүлдү. Спермограммаларда азооспермия 8,3 % учурларда кездешип, негизинен экинчи даражадагы гипогонадизм

же обструкция менен байланыштуу болгон. Айрым бейтаптарда системалык сезгенүү лейкоцитоз жана эритроциттердин чөгүү ылдамдыгынын жогорулашы аркылуу лабораториялык жактан тастыкталган, ал эми лейкоцитурия мезгил-мезгили менен байкалган. Алынган маалыматтар жыныстык жол менен жугуучу инфекцияларды, өнөкөт простатитти жана гормоналдык бузулууларды эрте аныктоо зарылдыгын көрсөтүп, аймактык деңгээлдеги баштапкы эркек фертилдүүлүгүнүн бузулушун диагностикалоо жолдорун жакшыртууга жана этиотроптук-патогенетикалык дарылоону жеке ыкма менен жүргүзүүгө мүмкүнчүлүк берет

Негизги сөздөр: эркек фертилдүүлүгүнүн бузулуусу; репродуктивдик функция; заара-жыныс инфекциялары; хламидия; уреаплазма; микоплазма

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Аннотация. Мужское бесплодие остается значимой медико-социальной проблемой, а региональные данные по структуре причин и патогенетическим механизмам ограничены. В Жалал-Абадской области Кыргызской Республики необходима актуализация доказательной базы для выработки таргетированной диагностики и профилактики. Целью исследования было описать этиологическую структуру и вероятные патогенетические механизмы первичного мужского бесплодия на основании комплексного клинико-лабораторного и инструментального обследования. Обследованы 110 мужчин в возрасте 20-38 лет, включая 60 пациентов с первичным бесплодием (анамнез ≥12 мес) и 50 здоровых мужчин, составивших контрольную группу. Проведены клиническое обследование, лабораторные и гормональные исследования, оценка спермограммы, микробиологическая и серологическая диагностика инфекций, а также ультразвуковая визуализация органов мочеполовой системы. Ведущую долю составляла инфекционно-воспалительная

патология мочеполовой системы: среди 60 обследованных мужчин хронический уретрит выявлен у значительной части пациентов (неспецифический – 23,3 %; специфические формы: трихомонадная – 6,7 %, хламидийная – 1,7 %, уреаплазменная – 21,7 %, микоплазменная – 18,3 %, гарднереллёз – 26,7 %); признаки хронического простатита по данным ультразвукового исследования определялись у 26,7 % обследованных. Микробиологический профиль характеризовался преобладанием стафилококков и грамотрицательных палочек, частой смешанной флорой (46,7 %) и кандидозом мочеполовой системы (8,3 %). Серопозитивность к цитомегаловирусу составила 31,7 %, к вирус простого герпеса 1-го и 2-го типов – 25,0 %. Среди анатомо-функциональных причин выявлены варикоцеле (6,7 %), орхоэпидидимит (5,0 %), крипторхизм (1,7 %), обтурация семявыводящих путей (3,3 %); эндокринные нарушения представлены вторичным гипогонадизмом (5,0 %). В спермограммах азооспермия встречалась у 8,3 % (ассоциировалась со вторичным гипогонадизмом или обструкцией). Системное воспаление подтверждалось у части пациентов лабораторно (лейкоцитоз, ускоренная скорость оседания эритроцитов), а лейкоцитурия фиксировалась эпизодически. Полученные данные обосновывают приоритет раннего скрининга инфекций, передаваемых половым путём, хронического простатита и гормональных нарушений в региональных маршрутах диагностики первичного мужского бесплодия и позволяют персонализировать этиотропную и патогенетическую терапию

Ключевые слова: нарушение фертильности у мужчин; репродуктивная функция; урогенитальные инфекции; хламидия: уреаплазма; микоплазм