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Prevalence of Helicobacter pylori and antisperm antibodies in Algerian men with isolated asthenozoospermia

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Abstract

Helicobacter pylori (HP) infection, which is endemic in several regions, is suspected to influence male fertility through autoimmune and inflammatory mechanisms. Little data is available in North Africa. To assess the frequency of HP infection and antisperm antibodies (ASA) in sterile Algerian men with asthenozoospermia and analyze their relationship with sperm parameters. A cross-sectional study conducted in Sidi Bel Abbes from 2017 to 2018 included 42 primary infertile men with isolated asthenozoospermia confirmed by two or more spermograms. HP status was evaluated using rapid IgG serology and ASA using semen ELISA. Comparisons were made using Fisher's exact test, the φ coefficient, and the Mann-Whitney and age-adjusted logistic regressions. Twenty-two patients (52.4%) were HP-seropositive, and 36 (85.7%) were ASA-positive, far above the usual % values of positive subjects (10-20%). The correlation between HP and hazardous accountability (HP-ASA) was weak and non-significant ($\varphi = 0.156$; $p = 0.41$). HP and ASA status were not associated with age. The regression models showed no robust associations. In this pilot cohort, HP and ASA were common but not significantly associated. HP does not appear to be a significant contributor to isolated male infertility in this context. Larger studies with fertile controls and strain typing are required.

Keywords:

Helicobacter pylori, Male infertility, Antisperm antibodies, Asthenozoospermia.

Introduction

Infertility, the inability of a couple to conceive after 12 months of regular unprotected intercourse, affects 17% of couples in their reproductive years [1-4]. A male factor is implicated in 20-50% of cases [5-8]. According to the MENA-CCNIN, sexual health among men in North Africa and the Middle East, including Algeria, is affected by several epidemiological determinants [9]. Despite these advancements, the etiology of 20-30% of cases of male infertility is idiopathic [10]; thus, studies have targeted infectious and autoimmune mechanisms [11, 12].

Helicobacter pylori (HP) is traditionally thought to affect male fertility beyond digestion [10, 13, 14]. Antigenic cross-reactivity of its proteins (flagellin, VacA, and CagA) with sperm β-tubulin may be reflected in the generation of antisperm antibodies (ASA), which are related to fertility failure, as well as changes in sperm motility and concentration [11 − 23]. Moreover, systemic inflammation under HP stimulation can induce impaired spermatogenesis and deteriorated sperm quality [21, 24-27] through the function of pro-inflammatory cytokines (TNF-α, IL-6, IL-1β, and IL-8). In addition, the relationship between HP and numerous autoimmune diseases [13, 23] also confirms this immunopathogenic potential. However, the results are still inconclusive, as other studies have not observed similar impacts on semen quality [28]. In Algeria, particularly in Sidi Bel Abbes, the high prevalence of HP has led researchers to focus on digestive pathologies and antibiotic resistance [29-33]. The effects of male infertility have not yet been established [34-36]. This study aimed to address this gap by investigating the association between Helicobacter pylori, antisperm antibodies, and sperm parameters, particularly asthenozoospermia, in primarily infertile men.

Materials and Methods

A cross-sectional observational study was performed in the province of Sidi Bel Abbes (Algeria) from August 2017 to March 2018, according to the guidelines for observational studies following STROBE [37]. This study aimed to evaluate the correlation between HP infection, ASAs, and semen quality in infertile males. The inclusion criteria were as follows: primary male infertility (defined as the absence of conception for over 12 months), isolated asthenozoospermia on at least two spermograms, adherence to other sperm parameters based on the WHO 2010 criteria [38], testicular volume within average limits, a BMI ranging from 18 to 29 kg/m², and no use of antibiotics or proton pump inhibitors in the three months prior to the study. Subjects with active urogenital infections, severe varicocele, uncontrolled endocrine disorders, recent exposure to high temperatures, a history of gonadotoxic treatments, and diagnosed female factor infertility were excluded.

A standardized questionnaire was used to collect clinical and sociodemographic information (age, BMI, medical history, tobacco smoking status, occupational exposure, and family history of disease). Sperm analyses were performed in a single laboratory by the same senior biologist using samples collected through masturbation (2-7 days of sexual abstinence) following the WHO procedure. The measured parameters were volume, pH, concentration, motility (progressive and total), morphology, and vitality. Progressive motility

less than 32% or total motility less than 40% was considered asthenozoospermia. Infectious conditions were determined using a rapid serological test (IgG anti-HP, Cypress Diagnostics, Belgium). ASAs were analyzed using ELISA (ALPCO Diagnostics, USA) with a cutoff value of ≥60 U/mL; in some cases, their presence was confirmed using an immunobead test.

SPSS v23.0 was adopted to perform the statistical analyses. Quantitative data are presented as mean ± SD (or n, %). The relationship between HP infection and age was analyzed using Fisher's exact test and comparisons using the Mann-Whitney U. test. The significance level was set at $p < 0.05$.

Ethics approval

Our research protocol was approved by the Ethics Committee of the Djillali Liabes University.

Consent to participate

Each participant signed a written consent form in accordance with the Declaration of Helsinki.

Results

They described the age and demographics of the patient population and the prevalence of HP infection and antisperm antibodies (ASA) in their center with respect to their association with semen parameters.

The study included 42 infertile men with isolated asthenozoospermia. The basic demographic data and clinical characteristics are shown in Table 1. Most of them (except in one case, aged 65) were in the age range between 30 and 45 years old. In total, 22 patients tested positive for Helicobacter pylori antibodies (52.4%). Both the HP-positive and HP-negative groups showed similar distributions of clinical findings, with comparable numbers of subjects exhibiting them at varying levels, especially in the digestive system.

Table 1: Descriptive statistics of the 42 asthenozoospermic infertile patients.

Variable	Main values	Comments
Age (years)	Mean ± SD: 37.8 ± 6.9 (Median: 38, IQR: 33.0-39.8)	Symmetrical distribution (30-65 years)
HP serology	22 positive (52.4%); 20 negative (47.6%)	Groups of comparable size
ASA (semen, ELISA)	36 positive (85.7%); 6 negative (14.3%)	ASA+ very predominant.
Asthenozoospermia	100% (isolated)	Confirmed on ≥2 spermograms
Progressive motility	Mean 22.5% (SD 6.8%)	WHO 2010 standard ≥ 32% [39]
Total motility (PR+NP)	Mean 30.1% (SD 7.4%)	WHO 2010 standard ≥ 40%
Sperm concentration	Mean 38 M/mL (SD 12)	WHO 2010 standard ≥ 15 M/mL
Normal forms	Mean 5.1% (SD 1.7%)	WHO 2010 standard ≥ 4%
Sperm leukocytes (peroxidase)	Median: 0.2 x 10^6/mL (IQR: 0-0.5)	Threshold leukocytospermia> 10^6 [39]

IQR: interquartile range; M: million; PR: progressive mobility.

Semen analysis confirmed asthenozoospermia: progressive motility averaged 22.5% ± 6.8, while overall motility was 30.1% ± 7.4, both falling short of the WHO 2010 reference criteria. However, the values of sperm concentration (38 million/mL) and morphology (5.1% normal) remained within the normal range. Elevated white blood cell counts in the semen were rare (median 0.2 × 10^6/mL).

When analyzing the association between HP status and ASA positivity (Table 1), 91% of the HP-positive group was also positive for ASA, whereas only 80% of the ASA-negative group was positive. This difference was not statistically significant (Fisher's exact test p = 0.412; Φ = 0.156). In patients with ASA, the higher rate made discrimination between these two groups impossible.

(a) **(b)**

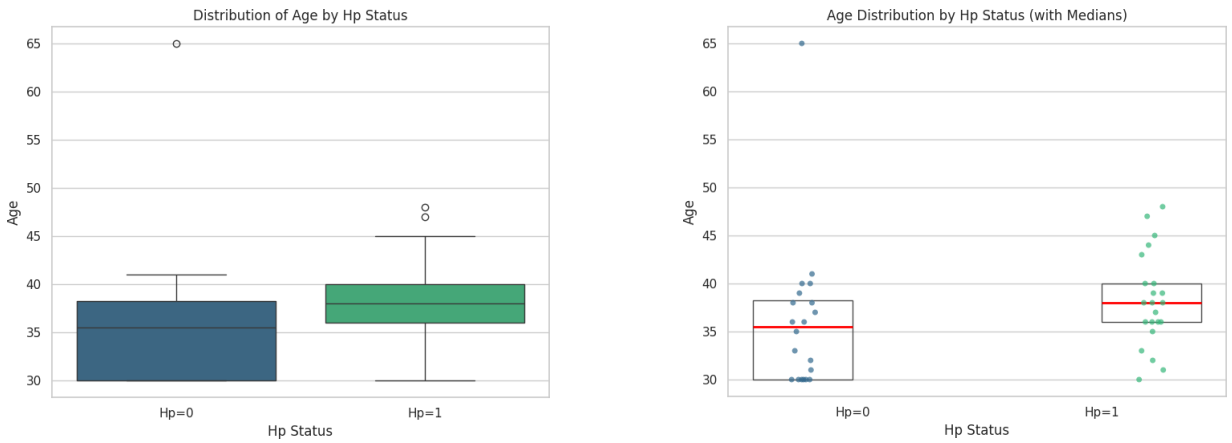

Figure 1: Distribution of age according to HP serological status

Age distribution of HP-negative (HP=0) and HP-positive (HP=1) individuals. The median age is slightly higher for HP-positive (38-39 years) than HP-negative (35 years), and the distribution overlaps to a large extent. Outliers can be considered 65-year-old subjects in the HP-negative group.

(a) **(b)**

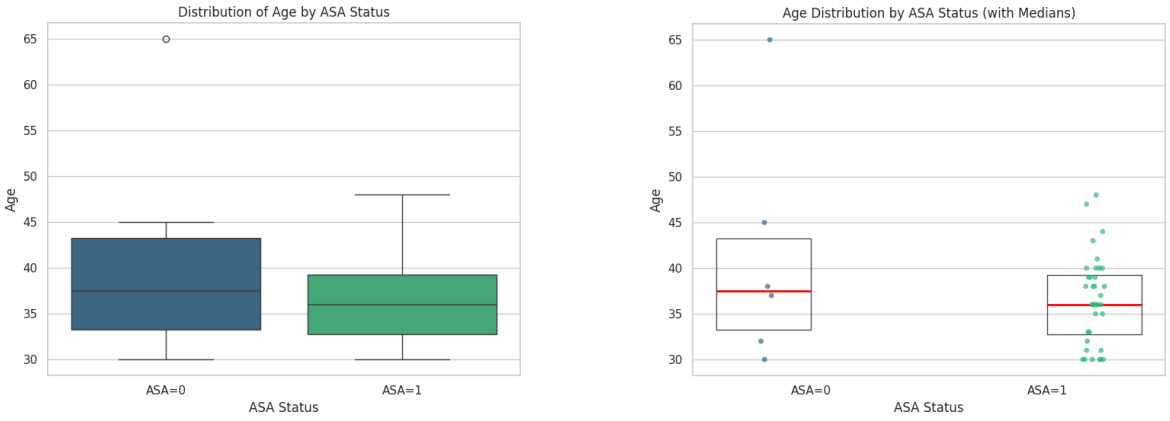

Figure 2: Age distribution according to the presence of antisperm antibodies (ASA)

Age distribution of patients according to ASA status (ASA=0: negative, ASA=1: positive) (a) Box plots with median and interquartile range (IQR), whiskers, and extremes. (b) The same datasets with ages superimposed (one point = one subject); medians are in red. The median age is similar in both groups, with a mild outlier of 65 years in the ASA-negative group.

Therefore, we examined whether age served as a confounding variable. The median age did not significantly differ between the HP-positive and HP-negative groups (38.0 and 35.5 years, p=0.251, Figure 1) or ASA-positive and ASA-negative groups (36.0 and 37.5 years, p=0.704, Figure 2). Moreover, logistic regression analyses (Table 2) revealed no substantive associations; neither age nor ASA status predicted HP status, and the same was true for the reverse relationship. The confidence intervals were much wider, indicating a large amount of uncertain data. Results: The findings of this review are as follows. We are confident that there is no major relationship between HP infection and sperm antibodies in the study population.

Table 2: Binary logistic models exploring the links between HP, ASA, and age.

Model (dependent variable)	**Predictor**	**Adjusted OR (95% CI)**	**p (Wald)**
1. HP status (0/1)	Age (per year)	1.04 (0.96-1.12)	0.35
	ASA positive (vs. 0)	2.44 (0.40-14.8)	0.33
2. ASA status (0/1)	Age (per year)	0.98 (0.89-1.08)	0.71
	HP positive (vs. 0)	2.44 (0.40-14.8)	0.33

OR: odds ratio. 95% CI: 95% confidence interval. (Method: logistic regression, n=42.)

Discussion

This study aimed to evaluate whether there is an association between HP infection and male infertility factors affected by antisperm antibodies, the main cause of asthenozoospermia. The number of patients with ASA (86%) was high, as the literature reports the prevalence of 10-20% [40-42]. This estimate should be interpreted in the context of a clinically selected cohort with isolated asthenozoospermia and may not be generalizable to unselected infertile men or the general population due to the small sample size. While a sensitive ELISA test or the study of a homogenous population defined by the idiopathic nature of asthenozoospermia, often immunological, may partly explain this result, ASA is known to have deleterious effects on motility, promote sperm agglutination, and hamper fertilization [20, 21, 39-45].

The seroprevalence of HP in our series was 52.4%, comparable to that in the general Algerian male population, which is approximately 60% [30, 46]. Other authors have suggested the usefulness of wide screening for HP in infertile men; our results are in line with those of Feng et al. and Andreeva-Gateva et al., who found a slight, non-significant increased risk [11, 28, 42]. Overall, HP infection is frequent but sporadic in this population group. We found no significant correlation between HP seropositivity and positive ASA, and the trend of a higher proportion of ASA in HP+ patients was insignificant [16]. This negative relationship is in opposition to certain studies conducted on gastroduodenal diseases; however, only a few of the most virulent strains of HP (CagA+) may negatively impact male fertility [24, 25]. Our methods did not enable strain definition; therefore, this question remains unknown. Our study is underpowered due to the small sample size, and its pilot nature suggests we cannot definitively claim superiority in the comparison of changes between groups with respect to other therapies or clinical impact. The definite limitations are the relatively low number of ASA patients, non-fertile control groups, and unsystematic testing for other urogenital diseases; therefore, our prevalence data should be considered with caution, calling for future studies in fertile controls.

IgG serology only shows past exposure, not active infection, which could weaken the links to semen quality or ASA status; future research should screen for active infections. Overall, HP is probably of minor importance for idiopathic male infertility, and screening for HP does not seem to justify this population of infertile men. We suggest larger studies with comparison groups and strain-specific CagA assignments.

Conclusion

By analyzing data from 42 Algerian asthenozoospermic men, ongoing HP seroprevalence (52%) was found to be in accordance with local endemicity. The presence of antisperm antibodies and the grade of sperm changes were not correlated. ASA was present in over 85% of patients, indicating an immunological trait distinct from that of HP. Our findings suggest that the involvement of this infection in causing isolated male infertility is unlikely, which supports some recent studies. However, further studies, including fertile controls and strain-typing analysis, are required to investigate the possible involvement of virulent species.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflict of interest.

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Author Contributions

Study design, data acquisition and analysis, writing, and final approval: All authors.

References

[1] Kazemijaliseh H, Ramezani Tehrani F, Behboudi-Gandevani S, Hosseinpanah F, Khalili D, Azizi F. The Prevalence and Causes of Primary Infertility in Iran: A Population-Based Study. Glob J Health Sci 2015;7:226-32. https://doi.org/10.5539/gjhs.v7n6p226.

[2] Organization WH. Infertility prevalence estimates, 1990-2021. World Health Organization; 2023.

[3] EAU Guidelines on Sexual and Reproductive Health-Uroweb n.d. https://uroweb.org/guidelines/sexual-and-reproductive-health (accessed on August 13, 2025).

[4] O'Flynn N. Assessment and treatment for people with fertility problems: NICE guideline. Br J Gen Pract 2014;64:50-1. https://doi.org/10.3399/bjgp14X676609.

[5] Abdalla RAH. The Experience of Infertility: A Review of Recent Literature on Male Infertility Due to Post Testicular Disorders. Journal of Advances in Medicine and Medical Research 2023;35:23-30. https://doi.org/10.9734/jammr/2023/v35i105015.

[6] Gabrielsen JS, Lamb DJ. 11 - Disorders that impact reproduction. In: Dhar SU, Nagamani SCS, Eble TN, editors. Handbook of Clinical Adult Genetics and Genomics, Academic Press; 2020, p. 147-62. https://doi.org/10.1016/B978-0-12-817344-2.00011-3.

[7] Kim ED, Benton O. Best Practice Guidelines for Male Infertility Diagnosis and Management. In: Parekattil SJ, Esteves SC, Agarwal A, editors. Male Infertility: Contemporary Clinical Approaches, Andrology, ART and Antioxidants, Cham: Springer International Publishing; 2020, p. 779-91. https://doi.org/10.1007/978-3-030-32300-4_62.

[8] Maamar MB, K. Skinner M. Genomics, Epigenetics, and Male Reproduction. In: Niederberger CS, Lamb DJ, Lipshultz LI, Howards SS, editors. Infertility in the Male. 5th ed., Cambridge: Cambridge University Press; 2023, p. 94-106. https://doi.org/10.1017/9781108937054.008.

[9] Abangah G, Rashidian T, Parizad Nasirkandy M, Azami M. A Meta-Analysis of The Prevalence and Etiology of Infertility in Iran. International Journal of Fertility and Sterility 2023;17:160-73. https://doi.org/10.22074/ijfs.2023.541991.1215.

[10] Moretti E, Figura N, Collodel G, Ponzetto A. Can Helicobacter pylori infection influence human reproduction? World J Gastroenterol 2014;20:5567-74. https://doi.org/10.3748/wjg.v20.i19.5567.

[11] Figura N, Piomboni P, Ponzetto A, Gambera L, Lenzi C, Vaira D, et al. Helicobacter pylori infection and infertility. European Journal of Gastroenterology & Hepatology 2002;14:663. https://doi.org/10.1097/00042737-200206000-00012.

[12] El-Garem Y, El-Sawy M, Mostafa T. Seminal Helicobacter pylori Treatment Improves Sperm Motility in Infertile Asthenozoospermic Men. Urology 2014;84:1347-50. https://doi.org/10.1016/j.urology.2014.09.004.

[13] He J, Liu Y, Ouyang Q, Li R, Li J, Chen W, et al. Helicobacter pylori and unignorable extragastric diseases: Mechanism and implications. Front Microbiol 2022;13. https://doi.org/10.3389/fmicb.2022.972777.

[14] Moretti E, Collodel G, Mazzi L, Campagna MS, Figura N. CagA-positive Helicobacter pylori infection and reduced sperm motility, vitality, and normal morphology. Dis Markers 2013;35:229-34. https://doi.org/10.1155/2013/919174.

[15] Chereshnev VA, Pichugova SV, Beikin YB, Chereshneva MV, Iukhta AI, Stroev YI, et al. Pathogenesis of Autoimmune Male Infertility: Juxtacrine, Paracrine, and Endocrine Dysregulation. Pathophysiology 2021;28:471-88. https://doi.org/10.3390/pathophysiology28040030.

[16] Dimitrova-Dikanarova D, Lazarov V, Tafradjiiska-Hadjiolova R, Dimova I, Petkova NU, Krastev Z. Association between Helicobacter pylori infection and the presence of anti-sperm antibodies. Biotechnology & Biotechnological Equipment 2017;31. https://doi.org/10.1080/13102818.2016.1258330.

[17] Kurotsuchi S, Ando H, Iwase A, Ishida Y, Hamajima N, Kikkawa F. The plausibility of Helicobacter pylori-related infertility in Japan. Fertility and Sterility 2008;90:866-8. https://doi.org/10.1016/j.fertnstert.2007.06.097.

[18] Li Y, Choi H, Leung K, Jiang F, Graham DY, Leung WK. Global prevalence of Helicobacter pylori infection between 1980 and 2022: a systematic review and meta-analysis. Lancet Gastroenterol Hepatol 2023;8:553-64. https://doi.org/10.1016/S2468-1253(23)00070-5.

[19] Gravina AG, Zagari RM, De Musis C, Romano L, Loguercio C, Romano M. Helicobacter pylori and extragastric diseases: A review. World J Gastroenterol 2018;24:3204-21. https://doi.org/10.3748/wjg.v24.i29.3204.

[20] Ahmed Berwary NJ. Effect of H.pylori and Cag-A on the infertility among males. IJB 2017;6:5292. https://doi.org/10.21746/ijbio.2017.03.001.

[21] Cui D, Han G, Shang Y, Liu C, Xia L, Li L, et al. Antisperm antibodies in infertile men and their effect on semen parameters: a systematic review and meta-analysis. Clin Chim Acta 2015;444:29-36. https://doi.org/10.1016/j.cca.2015.01.033.

[22] El-Kady HM. The Plausibility of Helicobacter Pylori and CagA Strains Related Infertility Among Males in Alexandria, Egypt. Journal of High Institute of Public Health 2019;49:56-63. https://doi.org/10.21608/jhiph.2019.29467.

[23] Chmiela M, Gonciarz W. Molecular mimicry in Helicobacter pylori infections. World J Gastroenterol 2017;23:3964-77. https://doi.org/10.3748/wjg.v23.i22.3964.

[24] Collodel G, Moretti E, Campagna MS, Capitani S, Lenzi C, Figura N. Infection by CagA-Positive Helicobacter pylori Strains may Contribute to Alter the Sperm Quality of Men with Fertility Disorders and Increase the Systemic Levels of TNF-α. Dig Dis Sci 2010;55:94-100. https://doi.org/10.1007/s10620-008-0704-1.

[25] Moretti E, Figura N, Campagna MS, Gonnelli S, Iacoponi F, Collodel G. Sperm Parameters and Semen Levels of Inflammatory Cytokines in Helicobacter pylori-infected Men. Urology 2015;86:41-7. https://doi.org/10.1016/j.urology.2015.02.068.

[26] Wang S, Zhang K, Yao Y, Li J, Deng S. Bacterial Infections Affect Male Fertility: A Focus on the Oxidative Stress-Autophagy Axis. Front Cell Dev Biol 2021;9:727812. https://doi.org/10.3389/fcell.2021.727812.

[27] Mohammadi F, Karimi-Dehkordi M, Pouriayevali F. Evaluation of the sperm parameters, oxidative stress, and histopathological effects of vitamin B12 in preventing Helicobacter pylori-induced testicular toxicity: An experimental study. Int J Reprod Biomed 2024;22:383-94. https://doi.org/10.18502/ijrm.v22i5.16439.

[28] Feng C, Lv P-P, Huang C-C, Yang S-Q, Yao Q-P, Shen J-M, et al. Sperm parameters and anti-Müllerian hormone remain stable with Helicobacter pylori infection: a cross-sectional study. BMC Urology 2020;20:188. https://doi.org/10.1186/s12894-020-00725-z.

[29] Chama Z, Titsaoui D, Benabbou A, Hakem R, Djellouli B. Effect of Thymus vulgaris oil on the growth of Helicobacter pylori. South Asian Journal of Experimental Biology 2020;10:374-82. https://doi.org/10.38150/sajeb.10 (6). p374-382.

[30] Kasmi H, Doukani K, Ali A, Tabak S, Bouhenni H. Epidemiological Profile of Helicobacter pylori Infection in Patients with Digestive Symptoms in Algeria. Journal of Epidemiology and Global Health 2020;10:293-7. https://doi.org/10.2991/jegh.k.200527.001.

[31] Chen Y-C, Malfertheiner P, Yu H-T, Kuo C-L, Chang Y-Y, Meng F-T, et al. Global Prevalence of Helicobacter pylori Infection and Incidence of Gastric Cancer Between 1980 and 2022. Gastroenterology 2024;166:605-19. https://doi.org/10.1053/j.gastro.2023.12.022.

[32] Bachir M, Allem R, Benejat L, Tifrit A, Medjekane M, Drici AE-M, et al. Molecular detection of mutations involved in Helicobacter pylori antibiotic resistance in Algeria. J Antimicrob Chemother 2018;73:2034-8. https://doi.org/10.1093/jac/dky167.

[33] Raaf N, Amhis W, Saoula H, Abid A, Nakmouche M, Balamane A, et al. Prevalence, antibiotic resistance, and MLST typing of Helicobacter pylori in Algiers, Algeria. Helicobacter 2017;22. https://doi.org/10.1111/hel.12446.

[34] Khalloua ZC, Chebli I, Meziani S, Ferrag D, Itatahine A, Mai H, et al. Does BMI Really Alter the Hormonal Profile in Infertile Women? Retrospective Study in the Region of Sidi Bel Abbes (West Algeria): THE HORMONAL PROFIL IN INFERTILE WOMEN? RETROSPECTIVE STUDY IN THE REGION OF SIDI BEL ABBES (WEST ALGERIA). Journal of Drug Delivery and Therapeutics 2020;10:142-7. https://doi.org/10.22270/jddt.v10i4-s.4320.

[35] Maï HA, Demmouche A. A Case-Control study of Body Mass Index and Infertility in Algerian Women (Sidi Bel Abbes, West of Algeria). International Journal of Infertility & Fetal Medicine 2012;6:103-7. https://doi.org/10.5005/jp-journals-10016-1110.

[36] Motlagh Asghari K, Nejadghaderi SA, Alizadeh M, Sanaie S, Sullman MJM, Kolahi A-A, et al. Burden of polycystic ovary syndrome in the Middle East and North Africa region, 1990-2019. Sci Rep 2022;12:7039. https://doi.org/10.1038/s41598-022-11006-0.

[37] STROBE. STROBE n.d. https://www.strobe-statement.org/ (accessed August 30, 2025).

[38] Cooper TG, Noonan E, von Eckardstein S, Auger J, Baker HWG, Behre HM, et al. World Health Organization reference values for human semen characteristics. Hum Reprod Update 2010;16:231-45. https://doi.org/10.1093/humupd/dmp048.

[39] Semen Analysis: Technique and Normal Values n.d. https://www.urology-textbook.com/semen-analysis.html (accessed September 8, 2025).

[40] Piroozmand A, Mousavi Nasab SD, Erami M, Hashemi SMA, Khodabakhsh E, Ahmadi N, et al. Distribution of Human Papillomavirus and Antisperm Antibody in Semen and Its Association with Semen Parameters Among Infertile Men. J Reprod Infertil 2020;21:183-8.

[41] Bozhedomov VA, Nikolaeva MA, Ushakova IV, Lipatova NA, Bozhedomova GE, Sukhikh GT. Functional deficit of sperm and fertility impairment in men with antisperm antibodies. Journal of Reproductive Immunology 2015;112:95-101. https://doi.org/10.1016/j.jri.2015.08.002.

[42] Andreeva-Gateva P, Masseva A. Similarly High Seroprevalence of Anti-Helicobacter pylori Antibodies in Men and Women with and without Idiopathic Infertility. J of IMAB 2021;27:3497-501. https://doi.org/10.5272/jimab.2021271.3497.

[43] Kiani AH, Asadbeik E, Bibalan MH, Sedighi M, Eshaghi M, Gholami M, et al. Serological Diagnosis of Helicobacter pylori Infection in Patients With a Polycystic Ovary Syndrome. Archives of Clinical Infectious Diseases; 2015.

[44] Yavaşoglu I, Küçük M. Anti-Helicobacter pylori antibodies and polycystic ovary syndrome. European Journal of Obstetrics and Gynecology and Reproductive Biology 2012;163:243. https://doi.org/10.1016/j.ejogrb.2012.04.006.

[45] Gupta S, Sharma R, Agarwal A, Boitrelle F, Finelli R, Farkouh A, et al. Antisperm Antibody Testing: A Comprehensive Review of Its Role in the Management of Immunological Male Infertility and Results of a Global Survey of Clinical Practices. World J Mens Health 2022;40:380-98. https://doi.org/10.5534/wjmh.210164.

[46] Alsulaimany FAS, Awan ZA, Almohamady AM, Koumu MI, Yaghmoor BE, Elhady SS, et al. Prevalence of Helicobacter pylori Infection and Diagnostic Methods in the Middle East and North Africa Region. Medicina 2020;56:169. https://doi.org/10.3390/medicina56040169.