УДК 33
JEL: H 52
DOI: 10.17238/issn1998-5320.2021.15.3.24

Ya. V. Kostroma[1]
[1]Family Medical Center, Siversky, Leningrad Region, Russian Federation

Cost management for various diagnostic schemes for cervicitis and chronic endometritis

Abstract. Financial management in health care assumes economic analysis as a management function comparing different diagnostic schemes for chronic diseases. In health care management, there are high diagnostic costs as the most important element in the correct diagnosis and prescription of the necessary therapy. Initial studies of the cervical canal and endometrial microbiome suggest an association with reproductive outcomes in assisted reproduction and various gynecologic pathologies such as chronic endometritis, endometriosis, and dysfunctional uterine bleeding. The high incidence of chronic cervicitis and endometritis increases interest in the study of this issue. In the pathogenesis of the development of these processes, the role of anaerobic-aerobic microbial associations is not excluded. The purpose of the scientific review was to analyze the costs of diagnosing diseases when changing the pre-existing paradigm of treatment and the importance of cervicitis in the development of chronic endometritis. The elements of an independent scientific contribution are the analysis of the role and contribution of cervicitis in the development of endometritis in order to optimize the timely and correct treatment of patients. New approaches have been proposed for calculating the economic feasibility of diagnosing chronic diseases.

Keywords: healthcare management, economic analysis, economic efficiency, cervicitis, chronic endometritis, microbiome.

Paper submitted: September 21, 2021.

For citation: Kostroma Ya. V. (2021) Cost management for various diagnostic schemes for cervicitis and chronic endometritis. The Science of Person: Humanitarian Researches, vol. 15, no. 3, pp. 207–215. DOI: 10.17238/issn1998-5320.2021.15.3.24.

Introduction. Economic growth or recession depends to a certain extent on the health status of the female population. Losses, both social and economic, will increase in the absence of effective measures to control the timely detection of chronic diseases. According to the forecasts of the Organization for Economic Cooperation and Development, the costs of health systems due to the growing burden of chronic diseases could double by 2050 and reach 13 % of the gross domestic product [1]. This will not only impose a significant burden on the health care system, but also pose a threat to the country's social and economic stability. Endometritis is a fairly common and difficult to diagnose disease that leads to serious economic losses, including a deterioration in a woman's quality of life and a high probability of reduced fertility.The microbial colonisation of the female genital tract has been extensively studied over the past few decades. The cervicovaginal microbiome plays an important role in women's reproductive health, influencing rates of preterm birth and neonatal mortality, the incidence, susceptibility and potential transmission of sexually transmitted infections, and other important clinical conditions, such as pelvic inflammatory disease [2, 3]. The difficulty in studying the upper parts of the genital tract and, in particular, the intrauterine and endometrial microbiome is complicated by the problem of sterile access without contamination by the flora of the cervical canal, as well as by the significantly lower biomass of the upper parts as compared to the lower part of the reproductive tract. Studies using next-generation sequencing of the bacterial 16S ribosomal RNA (16S rRNA) gene have provided convincing evidence for the existence of an upper female reproductive tract microbiome [4]. Materials and methods. A comparative analysis of the cost assessment of methods for diagnosing diseases using foreign data on the economic efficiency and diagnostic capabilities of methods for detecting cervicitis and chronic endometritis is carried out, and the lack of domestic research in this area is indicated. The analysis of literary data for the last years was carried out using the main electronic search databases. The analysis of microbiological and diagnostic characteristics as well as the frequency of occurrence of the described pathological conditions was carried out. Attention is focused on the emerging

difficulties in diagnosing and clarifying the diagnosis, as well as the imperfection of the diagnostic methods themselves.

Results. The structure of economic damage to diseases can be represented as a pyramid, in which the apparent direct costs represent a smaller part of the damage. The second link will be economic losses due to a decrease in labor efficiency due to temporary disability. The third link is indirect economic effects. In this regard, the most common method for determining the cost of illness, taking into account direct and indirect costs using the method of human capital [5, 6]. This method allows to take into account the direct costs of the health care system and indirect losses in the economy associated with temporary disability, disability and premature mortality. In the Russian Federation, the frequency of chronic endometritis ranges from 0.2 % to 60 %, mainly affecting a layer of women of reproductive age. The average cost of diagnosing this disease ranges from 10 to 20 thousand per woman, depending on the region, while the cost of diagnostics and timely treatment of inflammatory diseases of the cervix is much lower. Therefore, it is necessary to understand both the etiology and pathogenesis, and the optimal cost-effective ways to diagnose these conditions.

Over the past century, based on the work of Henry Tissier in 1900, it was unanimously believed that the uterine cavity was sterile, and for a long time this statement was not questioned [7, 8]. The new era has changed the existing axiom. Examining hysterectomized uterus, scientists discovered the presence of bacteria in the endometrium, but their number was much lower than in the vagina [9, 10]. Other researchers have also successfully confirmed that the uterine cavity is not sterilized [11, 12]. Currently, there is no unitary representation of the pathogenesis of the combination of chronic cervicitis and chronic endometritis, showing the biomechanisms of formation and interaction of the different aspects of the pathogenetic chain and explaining the basis of the chronic inflammatory process. Cervicitis is rarely isolated, forming a single biosystem with the vulva and vagina.

It is important to note that most cases of cervicitis are not a specific inflammatory agent but an association of several pathogens, which, according to Serov et al., is due to the low effectiveness of antibacterial drugs due to their improper use and development of resistance of microorganisms to modern anti-inflammatory drugs [13]. Endometrial condition plays an important role in the realization of reproductive potential. According to different authors, the prevalence of chronic endometritis ranges from 10 to 85 % [14], which is due to certain difficulties in diagnosis and clinical and morphological verification of this disease. To date, chronic endometritis is regarded as a clinical and morphological syndrome in which multiple secondary morphological and functional changes occur due to persistent endometrial damage by an infectious agent, impairing cyclic biotransformation and receptivity of the uterine corpus mucosa.

Over the past few years, studies of endometrial and peritoneal samples obtained from healthy women and women with benign and non-infectious gynaecological diseases have shown a continuum of microbiota with decreasing biomass and increasing diversity from the lower to the upper parts of the reproductive tract. It has been suggested that upper genital tract infection results from pathological penetration of vaginal bacteria into the upper genital tract [8, 15]. Kunz et al. performed a study illustrating the reaching of the uterine cavity with radioactively labelled macrospheres within minutes of insertion into the external cervical pharynx, and documented a uterine peristaltic pump mechanism actively transporting vaginal contents into the uterus [16]. Zervomanolakis et al extended these results by demonstrating the lifting of particles through the cervix within minutes during the follicular and luteal phases of the cycle [17].

The pathogenesis of chronic endometritis is related to qualitative and quantitative changes in the endometrial microbiota, with abnormal proliferation of different types of micro-organisms, mainly Gram-negative and intracellular bacteria (e. g. Enterococcus faecalis, Mycoplasma, Ureaplasma, Chlamydia, Escherichia coli and Streptococcus spp.) [18]. As evidence of an infective etiology of chronic endometritis, several studies have found that certain courses of antibiotics can cure chronic endometritis in most patients [18, 19, 20]. It should be noted that chronic endometritis is characterised by plasma cell infiltrates, which are associated with almost all autoimmune conditions, including rare autoimmune diseases of the reproductive system (e.g. autoimmune oophoritis) [21]. To test the hypothesis of chronic endometritis as an autoimmune

condition, a recent study by Kushnir et al. compared different inflammatory and autoimmune markers between infertile women with chronic endometritis and infertile women without chronic endometritis. The authors failed to demonstrate different values of total immunoglobulins, antinuclear antibodies, thyroid antibodies and antiphospholipid antibodies between the two groups compared, concluding that chronic endometritis has no significant autoimmune component [22]. Although these results need further confirmation, the hypothesis of autoimmune chronic endometritis cannot be confirmed at present.

As early as 1985, a study analysing the prevalence and manifestations of endometritis among women with cervicitis was published. The results, which significantly correlated with the development of endometritis, included a history of intermenstrual bleeding, the presence of C. trachomatis, N. gonorrhoeae or S. agalactiae in the cervix, and the presence of serum antibodies against C. trachomatis or M. Hominis [23].

In 2018, Chinese researchers improved the method for recognizing chronic endometritis. The entire tissue sample was examined for the presence of CD138“+” plasma cells, the area of the examined tissue was determined, and the density of plasma cells per unit area was measured. To obtain a complete understanding of microorganisms in chronic endometritis, sequencing of the 16S rRNA gene amplicon was used. The results obtained confirmed the previously known data that Lactobacillus is the most widespread genus of the healthy endometrial microbiota In endometrial samples from patients with chronic endometritis, the content of Lactobacillus was 42.7 times lower than in the control group. Lactobacillus were more abundant in non-chronic endometritis microbiota [24]. There was a negative correlation between Lactobacillus and bacteria of the genus Anaerococcus, Finegoldia and Gardnerella, as well as between Staphylococcus and Lactobacillus. However, there was no statistically significant association of Streptococcus, Mycoplasma or Ureaplasma with chronic endometritis, although they were previously found in the uterine cavity in chronic endometritis in other studies. In other words, apart from Gardnerella and Staphylococcus, there has been no previously reported association of 16 genera other than Lactobacillus with chronic endometritis, as found by Yingyu Liu et al [24]. Fang et al., Studying the difference in the microbiome in a group of women with chronic endometritis and endometrial polyps and a group of healthy women, reported higher levels of Lactobacillus in women with endometrial pathology. Phylum Firmicutes in the study group were much higher, and Proteobacteria were much lower than in the control group. At the genus level, in the study group, a significant increase in Lactobacillus, Gardnerella, Bifidobacterium, Streptococcus and Alteromonas and a decrease in the amount of Pseudomona were found [25]. These results are supported by other studies based on the method of molecular biology. Mitchell et al [26]. demonstrated that the presence of bacteria in the uterine cavity is not associated with a significant inflammatory immune response, indicating that the existence of low-level commensal bacteria in the uterus is common and not pathological.However, the results of Fang et al. differed from the results obtained in the study by Ettore Cicinelli et al [27] who cultured various bacteria from endometrial samples from women with chronic endometritis. In their study, most of the samples tested only one species of bacteria due to the limitations of culture methods, and positive endometrial cultures showed a high correlation with the existence of chronic endometritis. The reason may be that most of the commensal intrauterine microbiota cannot be cultured, and intrauterine bacterial colonization is not always benign. The pathological properties of intrauterine bacteria can be associated with particularly virulent strains or species, high concentrations of bacteria, or polymicrobial dysbiosis on the surface of the endometrium.

Cicinelli at al. conducted a prospective diagnostic study investigating the reliability of vaginal and endocervical cultures to assess endometrial microbiology in women with chronic endometritis. Microorganisms commonly found in chronic endometritis are the common bacteria – Streptococcus species, Escherichia coli species, Enterococcus faecalis and Staphylococcus species, Mycoplasma / ureaplasma species (Mycoplasma genitalium, Mycoplasma hominis and Ureaplasma proteugiosaus), Pneumococcus species, Gardnerella vaginalis, Corynebacterium and yeast (Saccharomyces cerevisiae and Candida species) [25, 28]. Endometrial, vaginal and cervical canal cultures from women with chronic endometritis were compared to examine the percentage ratio of the etiological agent. There was a statisti-

cally significant difference for streptococci, staphylococci, Escherichia coli and Ureaplasma urealyticum (which were found to be more prevalent in the vagina than in the endometrial samples). The match rate between endocervical and endometrial samples for simple bacteria was 48 %; 100 % for C. trachomatis and 58 % for U. Urealyticum. Both vaginal and cervical cultures have a low concordance with endometrial cultures. In particular, positive endometrial cultures for Staphylococcus aureus did not show positive vaginal results. On the other hand, chlamydia has 100 % concordance between endometrial and cervical cultures.

Changes in the ratio of lactobacilli, [9], may be another characteristic feature of chronic endometritis, although research results are conflicting. While analysis of studies using classical cultures showed a decrease in the number of lactobacilli in the endometrium in women with chronic endometritis and suffering from infertility than in women without chronic endometritis, other studies using barcode sequencing have found an increase in the level of lactobacilli in chronic endometritis [25, 29].

It is important to emphasise that micro-organisms detected in endometrial tissue are often incompatible with those found in cervical tissue or vaginal discharge [18], and it is likely that microbiological studies using lower genital tract samples cannot predict pathogens of chronic endometritis [25]. A study by C. Mitchell et al, based on a qualitative endometrial polymerase chain reaction after hysterectomy, showed that 95 % of samples showed colonisation by at least one bacterial species. In addition, evidence of non-identicality of the vaginal and endometrial microbiota is presented.

Moreno and colleagues reported similar results. In particular, the most abundant bacteria detected in his study were Streptococci (47 %), followed by Enterococci (15 %), E. coli (12 %), K. pneumoniae (5 %), Staphylococci (3 %) and M. hominis (2 %) [30]. These results are very similar to previous findings, and in particular that streptococci are the most common micro-organism in the endometrium in chronic endometritis. G. vaginalis was detected in 7 % of the samples analysed and this is in agreement with Liu [24].

To address the problem of contamination of cervical samples by vaginal microbiota, Chen at al. collected endometrial samples without passing through the cervix. However, the specificity of the sampling route was tested by collecting endometrial samples through the uterus as well as through the cervical pharynx and vagina. The distribution of bacteria in samples collected via the cervical pharynx showed a high similarity to that in samples obtained by opening the uterus during surgery, suggesting that the uterine and cervical microbiota may be easily accessible and analysed in women without surgical intervention [9].

Bacterial vaginosis is known to be characterised by a lower prevalence of lactobacilli and a high activity of obligate-anaerobic bacteria and, if cervicitis is also present, by facultative-anaerobic flora in titres exceeding the clinically acceptable threshold. Although there is a large number of studies confirming the etiological role of bacterial vaginosis-associated micro-organisms in the causation of pelvic inflammatory processes, their role in the initiation and maintenance of chronic uterine inflammatory processes is controversial [31]. A study by Korn et. al. showed that plasmatic endometritis was more common in women with bacterial vaginosis, without other cervico-vaginal infections, suggesting a possible association between bacterial vaginosis and non-chlamydial, non-celiac upper tract infections [32]. Contrary results were obtained by W.W. Andrews et al. who found an association between asymptomatic bacterial vaginosis and the likelihood of endometrial microbial colonisation and bacterial colonisation associated with bacterial vaginosis, but found no association of bacterial vaginosis with plasmatic endometritis [33].

A recent study conducted at the D.O. Ott Research Institute found no association between chronic endometritis and bacteria associated with bacterial vaginosis, as detected by the Femoflor-16, an expanded real-time PCR assay of the urogenital tract microflora profile of women. In the study groups of women with and without chronic endometritis, there were no statistically significant differences in the frequency of micro-organisms characteristic of bacterial vaginosis. The only microorganism associated with the development of bacterial vaginosis found much more frequently in patients with severe chronic endometritis than in those without signs of inflammation in the endometrium was Atopobium vaginae. However, the difference was not statistically significant [34]. In contrast, other studies by foreign colleagues have identified an important role of Atopobium vaginae in the genesis of chronic en-

dometritis. In a study by Haggerty et al. A. vaginae was found in 83 % of patients with pelvic inflammatory disease [35].

An earlier study by another author also showed an association with histological endometritis of A. vaginae and G. vaginalis . Other anaerobic bacteria associated with bacterial vaginosis, including Prevotellatimonensis, P. amnii and Peptoniphilusharei were also more frequently found in the endometrium in women with endometritis, but no statistical significance was found [36].

Haggerty and colleagues investigated the relationship between bacteria associated with bacterial vaginosis, pelvic inflammatory disease and its long-term sequelae. The researchers performed an endometrial biopsy and polymerase chain reaction of both the endometrium and cervical discharge. S. sanguinegens (54 %), S. amnionii (66 %), A. vaginae (83 %), Bacteria associated with bacterial vaginosis (65 %), U. urealyticum (30 %) and U. parvum (58 %) were commonly isolated from the cervix and endometrium. S. sanguinegens, S. amnionii, A. vaginae and bacteria associated with the development of bacterial vaginosis were significantly associated with bacterial vaginosis. Women who tested positive for each of the bacteria in the cervix were more likely to test positive for each of the other bacteria, except for U. parvum, which was not associated with any other bacteria. S. sanguinegens and S. amnionii, were found simultaneously in the endometrium in 97 % of samples positive for S. sanguinegens and in 52 % of samples positive for S. amnionii. It was also observed that women who tested positive for all four bacteria associated with bacterial vaginosis in the cervix and endometrium were almost four times more likely to suffer from infertility [35].

Studies published to date suggest that the upper and lower genital tracts share common microbial species. Additional possible routes of microbial contamination of the uterine cavity include haematogenous spread of the gut and oral microbiota [27]. Although Lactobacillus species dominate the vaginal microbiome in most women, it is unclear whether this feature is common to healthy and inflammatory conditions of the upper genital tract, including the endocervix and uterine cavity. While most studies of intrauterine specimens obtained transcervically revealed a predominance of Lactobacillus presence and the relative amount of Lactobacillus in endometrial specimens obtained by hysterectomy was more variable.

Several studies comparing the microbiome of the reproductive system in one woman have suggested a decrease in Lactobacillus abundance from the lower to the upper parts of the genital tract [19]. This is consistent with Winters at al, who examined the molecular microbial profiles of mid-endometrial samples obtained after hysterectomy and compared them with those of the cervix, vagina, rectum and oral cavity, and also performed controls for background DNA contamination. The bacterial profiles of the endometrium differed from those of the oral cavity, rectum, vagina and background DNA, but not the cervix. Lactobacillus and Gardnerella predominated in the profiles of the vaginal samples. The most prominent taxa in rectal samples were Finegoldia, Prevotella, Peptoniphilus, Streptococcus and Bacteroides. In oral samples, the most prominent taxa were Streptococcus, Veillonella, Haemophilus, Neisseria, Prevotella, Fusobacterium, Actinomyces, Rothia and Gemella. In contrast, Acinetobacter predominated in the bacterial profiles of cervical and endometrial samples. The major taxonomic units in both cervix and endometrium included Acinetobacter, Pseudomonas, Comamonadaceae and Cloacibacterium [37].

Kobaidze E.G. and Padrul M.M. compared the microbiome of the cervical canal and uterine cavity in chronic endometritis and in healthy women. The findings showed a positive microflora growth in cervical and uterine samples obtained from both groups of women. In healthy women as well as in patients with chronic endometritis, lactobacilli were detected in both cervical and uterine specimens, but a higher growth of the different microflora was recorded in specimens from patients with chronic endometritis. One in eight patients with chronic endometritis was found to have Escherichia coli producing extended-spectrum β-lactamase in cervical specimens. The microbiome of the endometrium and cervical canal were similar but not identical [38].

Conclusions. Cervicitis and chronic endometritis are associated with negative socio-economic consequences not only for the woman herself, but also for the state, as the chances of temporary disability and the emergence of a demographic crisis increase [39]. The cost-effectiveness of prevention interventions should include the full range of health promotion benefits, not just the impact on the costs of health care. In the aspect of health economics, population-based measures, primarily those recom-

mended by the World Health Organization, are advisable. Microflora is an integral part of the human body, which has an undeniable effect on the regulation and stabilization of the most important metabolic and energy processes at an optimal level. It is safe to say that further determination is needed as to whether a "nucleus" or resident microbiota of the uterus exists, as well as its contribution to health and homeostasis. In addition, complementary studies are needed to elucidate the functional influence of the microbiota of the uterus or specific types of bacteria, as well as the influence of these microbes on the physiological state of local homeostasis. Modulating the microbiota of the uterus to restore and maintain microbial composition is a promising area of research with high clinical relevance. Further study of the cervical canal and uterine cavity microbiome is therefore important in understanding and predicting the development of chronic pelvic inflammatory processes and reproductive outcomes.

References

1. Ade la Maisonneuve C., Martins J. O. (2015) The future of health and long-term care spending. OECD J Econ Stud. 2015, 2014, pp. 61–96.
2. Hillier S. L., Kiviat N. B., Hawes S. E. et al. (1996) Role of bacterial vaginosis-associated microorganisms in endometritis. American Journal of Obstetrics And Gynecology, 1996, Aug, no. 175 (2), pp. 435–441. DOI: 10.1016/s0002-9378(96)70158-8.
3. Taylor B. D., Darville T., Haggerty C. L. (2013) Does bacterial vaginosis cause pelvic inflammatory disease? Sexually transmitted diseases, 2013 Feb, no. 40 (2), pp. 117–122. DOI: 10.1097/OLQ.0b013e31827c5a5b.
4. Zhou X., Brotman R. M., Gajer P., Abdo Z. (2010) Recent advances in understanding the microbiology of the female reproductive tract and the causes of premature birth. Infect. Dis. Obstet. Gynecol., 2010. DOI: 10.1155 / 2010/737425.
5. Stuckler D., Reeves A., McKee M. (2017) Social and economic multipliers: what they are and why they are important for health policy in Europe. Scandinavian Journal of Public Health, 2017, no. 45 (Suppl 18), pp. 17–21.
6. Sanders G. D., Neumann P. J., Basu A., et al. (2016) Recommendations for conduct, methodological practices, and reporting of cost-effectiveness analyses second panel on cost-effectiveness in health and medicine. JAMA, 2016, no. 316 (10), pp. 1093–1103. DOI: https: //doi.org/10.1001/jama.2016.12195.
7. Ansbacher R., Boyson W. A., Morris J. A. (1967) Sterility of the uterine cavity. American Journal of Obstetrics & Gynecology, 1967, no. 99 (3), pp. 394–396. DOI: 10.1016/s0002-9378(16)34549-5.
8. Takebayashi A., Kimura F., Kishi Y. et al. (2014) The association between endometriosis and chronic endometritis. PLos One, 2014, no. 9, pp. e88354. DOI: 10.1371/journal.pone.0088354 DOI: 10.1371/journal.pone.0088354.
9. Mitchell C. M., Haick A., Nkwopara E. et al. (2015) Colonization of the upper genital trac t by vaginal bacterial species in nonpregnant women. American Journal of Obstetrics & Gynecology, 2015, no. 212 (5), 611. pp. 1–9. DOI: 10.1016/j.ajog.2014.11.043.
10. Chen C., Song, X., Wei, W. Et al. (2017) The microbiota continuum along the female reproductive tract and its relation to uterine-related diseases. Nature Communications, 2017, no. 8, art. no. 875. DOI: https: //doi.org/10.1038/s41467-017-00901-0.
11. Khan K. N., Fujishita A., Masumoto H. et al. (2016) Molecular detection of intrauterine microbial colonization in women with endometriosis. Science of The Total Environment, 2016, no. 199, pp. 69–75. DOI: 10.1016 / j.ejogrb.2016.01.040.
12. Khan K. N., Fujishita A., Kitajima M. et al. (2014) Intra-uterine microbial colonization and occurrence of endometritis in women with endometriosis. Human Reproduction, 2014, no. 29, pp. 2446–2456. DOI: 10.1093/humrep/deu222.
13. Serov V. N. (2005) Modern problems of diagnostics and treatment of reproductive health disorders in women. Rostov on Don, 2005, pp. 35–38.
14. Sukhikh G. T., Shurshalina A. V. (2010) Chronic endometritis. Manual. Moscow, GEOTAR-Media, 2010, 64 p.
15. Rizzo A. E., Gordon J. C., Berard A. R. (2021) The female reproductive tract microbiome – implications for gynecologic cancers and personalized medicine. Journal of Personalized Medicine, 2021 June, no. 11 (6), 546. DOI: 10.3390/jpm11060546.
16. Kunz G., Beil D., Deiniger H., Einspanier A., Mall G., Leyendecker G. (1997) The uterine peristaltic pump – normal and impeded sperm transport within the female genital tract. Advances in experimental medicine and biology, 1997, no. 424, pp. 267–277.
17. Zervomanolakis I. et al. (2007) Physiology of upward transport in the human female genital tract. Annals of the New York Academy of Sciences, 2007, vol. 1101, pp. 1–20. DOI: 10.1196/annals.1389.032.
18. Liu Y., Phil M., Chen X., Huang J. et al. (2018) Comparison of the prevalence of chronic endometritis as determined by means of different diagnostic methods in women with and without reproductive failure. Fertil Steril, 2018, no. 109, pp. 832–839.
19. Kitaya K., Takeuchi T., Mizuta S., Matsubayashi H., Ishikawa T. (2018) Endometritis: new time, new concepts. Views and Review, vol. 110, august 01, 2018, issue 3, pp. 344–350. DOI: https: //doi.org/10.1016/j.fertnstert.2018.04.012.
20. Cicinelli E., Matteo M., Trojano G., Mitola P. C., Tinelli R., Vitagliano A. et al. (2017) Chronic endometritis in patients with unexplained infertility: prevalence and effects of antibiotic treatment on spontaneous conception. American Journal of Reproductive Immunology, 2017, no. 79 (1). DOI: 10.1111/aji.12782.
21. Buzzaccarini G., Vitagliano A., Andrisani A. et al. (2020) Chronic endometritis and altered embryo implantation: a unified pathophysiological theory from a literature systematic review. Journal of Assisted Reproduction And Genetics, 2020, vol. 37, pp. 2897–2911. DOI: 10.1007 / s10815-020-019558.

22. Kushnir V. A., Solouki S., Sarig M. T., Vega M. G., Albertini D. F., Darmon S. K. et al. (2016) Systemic inflammation and autoimmunity in women with chronic endometritis. American Journal of Reproductive Immunology, 2016, 75, pp. 672–677. DOI: 10.1111/aji.12508.
23. Paavonen J., Kiviat N., Brunham R. C., Stevens C. E. (1985) Prevalence and manifestations of endometritis among women with cervicitis American Journal of Obstetrics and Gynecology Volume 152, Issue 3, 1 June 1985, pp. 280–286 DOI: 10.1016/s0002-9378 (85) 80210-6.
24. Liu Y., Ko E. Y., Wong K. K., Chen X. et al. (2019) Endometrial microbiota in infertile women with and without chronic endometritis as diagnosed using a quantitative and reference range-based method. Fertil Steril, 2019 Oct, no. 112(4), pp. 707–717.e1. DOI: 10.1016/j.fertnstert.2019.05.015.
25. Fang R. L., Chen L. X., Shu W. S., Yao S. Z. et al. (2016) Barcoded sequencing reveals diverse intrauterine microbiomes in patients suffering with endometrial polyps. American Journal of Translational Research, 2016, vol. 8, 3 pp. 1581–1592.
26. Mitchell C., Marrazzo J. (2014) Review. Bacterial vaginosis and the cervicovaginal immune response. American Journal of Reproductive Immunology, 2014 Jun, no. 71 (6) pp. 555–63. DOI: 10.1111 / aji.12264.
27. Cicinelli E., De Ziegler D., Nicoletti R. et al. (2008) Chronic endometritis: correlation among hysteroscopic, histologic, and bacteriologic findings in a prospective trial with 2190 consecutive office hysteroscopies. Fertil Steril, 2008, no. 89 (3), pp. 677–684. DOI: 10.1016/j.fertnstert.2007.03.074.
28. Cicinelli E., Trojano G., Mastromauro M. et al. (2017) Higher prevalence of chronic Endometritis in women with endometriosis: a possible etiopathogenetic link. Fertil Steril, 2017, no. 108 (2), pp. 289–295.e1. DOI: 10.1016/j.fertnstert.2017.05.016.
29. Kitaya K., Takaya Y., Nishiyama R., Yamaguchi K., Takeuchi T., Matsubayashi H. (2017) Live birth rate following oral antibiotic treatment for chronic endometritis in infertile women with repeated implantation failure. American Journal of Reproductive Immunology, 2017 Nov, no. 78(5). DOI: 10.1111/aji.12719.
30. Moreno I., Cicinelli E. et al. (2018) The diagnosis of chronic endometritis in infertile asymptomatic women: a comparative study of histology, microbial cultures, hysteroscopy, and molecular microbiology. American Journal of Obstetrics & Gynecology, 2018 Jun, pp. 602.e1–602.e16.
31. Ravel J., Moreno I., Simón C. (2021) Bacterial vaginosis and its association with infertility, endometritis, and pelvic inflammatory disease. Expert Review, march 01, 2021, vol. 224, issue 3, pp. 251–257. DOI: https: //doi.org/10.1016/j.ajog.2020.10.0.
32. Korn A., Bolan G., Padian N. et al. (1995) Plasma cell endometritis in women with symptomatic bacterial vaginosis. Obstetrics & Gynecology, 1995, vol. 85, no. 3 , pp. 387–390. DOI: 10.1016/0029-7844(94)00400-8.
33. Andrews W. W., Hauth J. C., Cliver S. P. et al. (2006) Association of a symptomatic bacterial vaginosis with endometrial microbial colonization and plasma cell endometritis in non pregnant women. American Journal of Obstetrics &Gynecology, 2006, no. 195(6), pp. 1611–1616. DOI: 10.1016/j.ajog.2006.04.010.
34. Tapilskaya N. I., Budilovskaya O. V., Krysanova A. A., Tolibova G. Kh. et al. (2020) Endometrial microbiota of women with chronic endometritis and idiopathic infertility. Obstetrics and gynecology, 2020, no. 4, pp. 72–81. DOI: https://dx.doi.org/10.18565/aig.2020.4.72-81.
35. Haggerty C. L., Totten P. A., Tang G. et al. (2016) Identification of novel microbes associated with pelvic inflammatory disease and infertility. Sexually Transmitted Infections, 2016, vol. 92, no. 6, pp. 441–446. DOI: 10.1136/sextrans-2015-052285
36. Hillier S. L, Rabe L. K, Meyn L. et al. (2013) Endometrial gardnerella vaginalis and atopobium vaginea are associated with histologic endometritis among women with clinically diagnosed pelvic inflammatory disease (PID). Sexually Transmitted Infections, 2013, no. 89 (Suppl 1), pp. A36–A36. DOI: 10.1136/sextrans-2013-051184.0112.
37. Winters A. D., Romero R., Gervasi M. T. et al. (2019) Does the endometrial cavity have a molecular microbial signature? Scientific Reports, 2019, no. 9, pp. 1–17. DOI: 10.1038/s41598-019-46173-0.
38. Kobaidze E. G., Padrul M. M. (2017) Some microbiological characteristics of women with chronic endometritis and its absence. Obstetrics, gynecology and reproduction. 2017, 11 (4), pp. 14–22. DOI: 10.17749 / 2313-7347.2017.11.4.014-022.
39. Stolyarov S. A. Management in health care: a textbook for universities. 2nd ed., Rev. and add. Moscow: Yurayt Publishing House, 2019, 764 p.

Information about the author

Yana V. Kostroma

Family Medical Center (4 Vokzalnaya St., Siversky, 188330, Leningrad Region, Russian Federation). ORCID ID: orcid.org/0000-0001-7648-5196. SPIN-code: 1332-2695. E-mail: kostromayana@gmail.com

Я. В. Кострома[1]

[1]ООО «Семейный медицинский центр», гп. Сиверский, Ленинградская область, Российская Федерация

Управление затратами при различных схемах диагностики цервицита и хронического эндометрита

Аннотация. Финансовый менеджмент в здравоохранении предполагает экономический анализ как функцию сравнения управления различными схемами диагностики хронических заболеваний. В менеджменте здравоохранения существуют высокие затраты на диагностику, как наиболее важный элемент в правильной постановке

диагноза и назначении необходимой терапии. Первоначальные исследования микробиома цервикального канала и эндометрия предполагают их связь с репродуктивными исходами при вспомогательной репродукции и различными гинекологическими патологиями, такими как хронический эндометрит, эндометриоз и дисфункциональное маточное кровотечение. Высокая частота хронических цервицитов и эндометритов повышает интерес к изучению этого вопроса. В патогенезе развития этих процессов не исключена роль анаэробно-аэробных микробных ассоциаций. Целью научного обзора является анализ издержек на диагностику заболеваний при изменении ранее существовавшей парадигмы лечения и значение цервицита в развитии хронического эндометрита. Элементом самостоятельного научного вклада является анализ роли цервицита в развитии эндометрита для целей оптимизации своевременного и правильного лечение пациенток. Предложены новые подходы в расчётах экономической обоснованности диагностики хронических заболеваний.

Ключевые слова: менеджмент в здравоохранении, экономический анализ, экономическая эффективность, цервицит, хронический эндометрит, микробиом.

Дата поступления статьи: 21 сентября 2021 г.

Для цитирования: Кострома Я. В. (2021) Управление затратами при различных схемах диагностики цервицита и хронического эндометрита // Наука о человеке: гуманитарные исследования. Том 15. № 3. С. 207–215. DOI: 10.17238/issn1998-5320.2021.15.3.24.

Источники

1. Ade la Maisonneuve C., Martins J. O. (2015) The future of health and long-term care spending. OECD J Econ Stud. 2015, 2014, pp. 61–96.
2. Hillier S. L., Kiviat N. B., Hawes S. E. et al. (1996) Role of bacterial vaginosis-associated microorganisms in endometritis. American Journal of Obstetrics And Gynecology, 1996, Aug, no. 175 (2), pp. 435–441. DOI: 10.1016/s0002-9378(96)70158-8.
3. Taylor B. D., Darville T., Haggerty C. L. (2013) Does bacterial vaginosis cause pelvic inflammatory disease? Sexually transmitted diseases, 2013 Feb, no. 40 (2), pp. 117–122. DOI: 10.1097/OLQ.0b013e31827c5a5b.
4. Zhou X., Brotman R. M., Gajer P., Abdo Z. (2010) Recent advances in understanding the microbiology of the female reproductive tract and the causes of premature birth. Infect. Dis. Obstet. Gynecol., 2010. DOI: 10.1155/2010/737425.
5. Stuckler D., Reeves A., McKee M. (2017) Social and economic multipliers: what they are and why they are important for health policy in Europe. Scandinavian Journal of Public Health, 2017, no. 45 (Suppl 18), pp. 17–21.
6. Sanders G. D., Neumann P. J., Basu A., et al. (2016) Recommendations for conduct, methodological practices, and reporting of cost-effectiveness analyses second panel on cost-effectiveness in health and medicine. JAMA, 2016, no. 316 (10), pp. 1093–1103. DOI: https: //doi.org/10.1001/jama.2016.12195.
7. Ansbacher R., Boyson W. A., Morris J. A. (1967) Sterility of the uterine cavity. American Journal of Obstetrics & Gynecology, 1967, no. 99(3), pp. 394–396. DOI: 10.1016/s0002-9378(16)34549-5.
8. Takebayashi A., Kimura F., Kishi Y. et al. (2014) The association between endometriosis and chronic endometritis. PLos One, 2014, no. 9, pp. e88354. DOI: 10.1371/journal.pone.0088354 DOI: 10.1371/journal.pone.0088354.
9. Mitchell C. M., Haick A., Nkwopara E. et al. (2015) Colonization of the upper genital trac t by vaginal bacterial species in nonpregnant women. American Journal of Obstetrics & Gynecology, 2015, no. 212 (5), 611. pp. 1–9. DOI: 10.1016/j.ajog.2014.11.043.
10. Chen C., Song, X., Wei, W. Et al. (2017) The microbiota continuum along the female reproductive tract and its relation to uterine-related diseases. Nature Communications, 2017, no. 8, art. no. 875. DOI: https://doi.org/10.1038/s41467-017-00901-0.
11. Khan K. N., Fujishita A., Masumoto H. et al. (2016) Molecular detection of intrauterine microbial colonization in women with endometriosis. Science of The Total Environment, 2016, no. 199, pp. 69–75. DOI: 10.1016 / j.ejogrb.2016.01.040.
12. Khan K. N., Fujishita A., Kitajima M. et al. (2014) Intra-uterine microbial colonization and occurrence of endometritis in women with endometriosis. Human Reproduction, 2014, no. 29, pp. 2446–2456. DOI: 10.1093/humrep/deu222.
13. Серов В. Н. Современные проблемы диагностики и лечения расстройств женского репродуктивного здоровья. Ростов н / Д, 2005. С. 35–38.
14. Сухих Г. Т., Шуршалина А. В. Хронический эндометрит. М. : Геотар-Медиа, 2010. 64 с.
15. Rizzo A. E., Gordon J. C., Berard A. R. (2021) The female reproductive tract microbiome – implications for gynecologic cancers and personalized medicine. Journal of Personalized Medicine, 2021 June, no. 11 (6), 546. DOI: 10.3390/jpm11060546.
16. Kunz G., Beil D., Deiniger H., Einspanier A., Mall G., Leyendecker G. (1997) The uterine peristaltic pump – normal and impeded sperm transport within the female genital tract. Advances in experimental medicine and biology, 1997, no. 424, pp. 267–277.
17. Zervomanolakis I. et al. (2007) Physiology of upward transport in the human female genital tract. Annals of the New York Academy of Sciences, 2007, vol. 1101, pp. 1–20. DOI: 10.1196/annals.1389.032.
18. Liu Y., Phil M., Chen X., Huang J. et al. (2018) Comparison of the prevalence of chronic endometritis as determined by means of different diagnostic methods in women with and without reproductive failure. Fertil Steril, 2018, no. 109, pp. 832–839.

19. Kitaya K., Takeuchi T., Mizuta S., Matsubayashi H., Ishikawa T. (2018) Endometritis: new time, new concepts. Views and Review, vol. 110, august 01, 2018, issue 3, pp. 344–350. DOI: https: //doi.org/10.1016/j. fertnstert.2018.04.012.
20. Cicinelli E., Matteo M., Trojano G., Mitola P. C., Tinelli R., Vitagliano A. et al. (2017) Chronic endometritis in patients with unexplained infertility: prevalence and effects of antibiotic treatment on spontaneous conception. American Journal of Reproductive Immunology, 2017, no. 79 (1) DOI: 10.1111/aji.12782.
21. Buzzaccarini G., Vitagliano A., Andrisani A. et al. (2020) Chronic endometritis and altered embryo implantation: a unified pathophysiological theory from a literature systematic review. Journal of Assisted Reproduction And Genetics, 2020, vol. 37, pp. 2897–2911. DOI: 10.1007 / s10815-020-019558.
22. Kushnir V. A., Solouki S., Sarig M. T., Vega M. G., Albertini D. F., Darmon S. K. et al. (2016) Systemic inflammation and autoimmunity in women with chronic endometritis. American Journal of Reproductive Immunology, 2016, 75, pp. 672–677. DOI: 10.1111/aji.12508.
23. Paavonen J., Kiviat N., Brunham R. C., Stevens C. E. (1985) Prevalence and manifestations of endometritis among women with cervicitis American Journal of Obstetrics and Gynecology Volume 152, Issue 3, 1 June 1985, pp. 280–286 DOI: 10.1016/s0002-9378 (85) 80210-6.
24. Liu Y., Ko E. Y., Wong K. K., Chen X. et al. (2019) Endometrial microbiota in infertile women with and without chronic endometritis as diagnosed using a quantitative and reference range-based method. Fertil Steril, 2019 Oct, no. 112(4), pp. 707–717.e1. DOI: 10.1016/j.fertnstert.2019.05.015.
25. Fang R. L., Chen L. X., Shu W. S., Yao S. Z. et al. (2016) Barcoded sequencing reveals diverse intrauterine microbiomes in patients suffering with endometrial polyps. American Journal of Translational Research, 2016, vol. 8, 3 pp. 1581–1592.
26. Mitchell C., Marrazzo J. (2014) Review. Bacterial vaginosis and the cervicovaginal immune response. American Journal of Reproductive Immunology, 2014 Jun, no. 71 (6) pp. 555–63. DOI: 10.1111 / aji.12264.
27. Cicinelli E., De Ziegler D., Nicoletti R. et al. (2008) Chronic endometritis: correlation among hysteroscopic, histologic, and bacteriologic findings in a prospective trial with 2190 consecutive office hysteroscopies. Fertil Steril, 2008, no. 89 (3), pp. 677–684. DOI: 10.1016/j.fertnstert.2007.03.074.
28. Cicinelli E., Trojano G., Mastromauro M. et al. (2017) Higher prevalence of chronic Endometritis in women with endometriosis: a possible etiopathogenetic link. Fertil Steril, 2017, no. 108 (2), pp. 289–295.e1. DOI: 10.1016 / j.fertnstert.2017.05.016.
29. Kitaya K., Takaya Y., Nishiyama R., Yamaguchi K., Takeuchi T., Matsubayashi H. (2017) Live birth rate following oral antibiotic treatment for chronic endometritis in infertile women with repeated implantation failure. American Journal of Reproductive Immunology, 2017 Nov, no. 78(5). DOI: 10.1111/aji.12719.
30. Moreno I., Cicinelli E. et al. (2018) The diagnosis of chronic endometritis in infertile asymptomatic women: a comparative study of histology, microbial cultures, hysteroscopy, and molecular microbiology. American Journal of Obstetrics & Gynecology, 2018 Jun, pp. 602.e1–602.e16.
31. Ravel J., Moreno I., Simón C. (2021) Bacterial vaginosis and its association with infertility, endometritis, and pelvic inflammatory disease. Expert Review, march 01, 2021, vol. 224, issue 3, pp. 251–257. DOI: https://doi.org/10.1016/j.ajog.2020.10.0.
32. Korn A., Bolan G., Padian N. et al. (1995) Plasma cell endometritis in women with symptomatic bacterial vaginosis. Obstetrics & Gynecology, 1995, vol. 85, no. 3 , pp. 387–390. DOI: 10.1016/0029-7844(94)00400-8.
33. Andrews W. W., Hauth J. C., Cliver S. P. et al. (2006) Association of a symptomatic bacterial vaginosis with endometrial microbial colonization and plasma cell endometritis in non pregnant women. American Journal of Obstetrics &Gynecology, 2006, no. 195(6), pp. 1611–1616. DOI: 10.1016/j.ajog.2006.04.010.
34. Tapilskaya N. I., Budilovskaya O. V., Krysanova A. A., Tolibova G. Kh. et al. (2020) Endometrial microbiota of women with chronic endometritis and idiopathic infertility. Obstetrics and gynecology, 2020, no. 4, pp. 72–81. DOI: https://dx.doi.org/10.18565/aig.2020.4.72-81.
35. Haggerty C. L., Totten P. A., Tang G. et al. (2016) Identification of novel microbes associated with pelvic inflammatory disease and infertility. Sexually Transmitted Infections, 2016, vol. 92, no. 6, pp. 441–446. DOI: 10.1136/sextrans-2015-052285.
36. Hillier S. L, Rabe L. K, Meyn L. et al. (2013) Endometrial gardnerella vaginalis and atopobium vaginea are associated with histologic endometritis among women with clinically diagnosed pelvic inflammatory disease (PID). Sexually Transmitted Infections, 2013, no. 89 (Suppl 1), pp. A36–A36. DOI: 10.1136/sextrans-2013-051184.0112.
37. Winters A. D., Romero R., Gervasi M. T. et al. (2019) Does the endometrial cavity have a molecular microbial signature? Scientific Reports, 2019, no. 9, pp. 1–17. DOI: 10.1038/s41598-019-46173-0.
38. Kobaidze E. G., Padrul M. M. (2017) Some microbiological characteristics of women with chronic endometritis and its absence. Obstetrics, gynecology and reproduction. 2017, 11 (4), pp. 14–22. DOI: 10.17749/2313-7347.2017.11.4.014-022.
39. Столяров С. А. Менеджмент в здравоохранении : учебник для вузов. 2-е изд., перераб. и доп. М. : Юрайт, 2019. 764 с.

Информация об авторе

Кострома Яна Владимировна

ООО «Семейный медицинский центр» (188330, РФ, Ленинградская обл., гп. Сиверский, Вокзальная ул., 4).

ORCID ID: orcid.org/0000-0001-7648- 5196. SPIN-код: 1332-2695. E-mail: kostromayana@gmail.com