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Personalised Medicine in the Management of Endometriosis

Ananya Mondal
Amity University, Lucknow Campus, Lucknow, Uttar Pradesh, India

Abstract: *Endometriosis is a chronic gynaecological disorder defined by the occurrence of endometrial like tissue outside uterine cavity that affects around 42 million women in India alone and roughly 190 million women worldwide with pelvic pain affecting around 10% of reproductive age. It has different clinical manifestation like chronic pelvic pain, dysmenorrhea, dyschezia, dysapeunia and fatigue. Different theories are proposed such as retrograde menstruation, immune pro-inflammatory pathway, metastatic theory endometrial stem theory and genetic etiology for causing endometriosis. Based on location it is divided as superficial, cystic ovarian and deep infiltrating endometriosis. Based on severity of lesion it is divided into 4 categories minimal (stage 1), mild (stage 2), moderate (stage3) and severe (stage4). It is a complex disease which remains diagnosed for many years and remain undiagnosed for many years. It is diagnosed through detailed medical history and pelvic examination. Transvaginal ultrasound, MRI and laparoscopy are used for diagnosis. Traditional treatment approaches includes non-steroidal anti-inflammatory drugs, hormonal therapy (estrogen-progestin contraceptives or progestins only), GnRH agonists, GnRH antagonists and aromatase inhibitors. Personalised medicine optimizes diagnosis and treatment to each patient's unique traits based on molecular, genetic, epigenetic, immunological, and clinical data. It involves different biomarkers artificial intelligence and multi-omics technologies.*

Keywords: Endometriosis, dyspareunia, personalized medicine, pharmacogenomics, biomarker, multi-omics

I. INTRODUCTION

Endometriosis is defined as presence of abnormal endometrial glands and stroma outside uterine cavity associated with inflammation. It mostly affects pelvic regions such as ovaries, fallopian tubes, urinary bladder, intestinal tract or peritoneum (Saunders *et al*., 2021).It occurs mostly between 25 to 45 years of age (B.Smolarz *et al*, 2021). It has different clinical manifestations including persistent chronic pelvic pain, dysmenorrhea (painful menstrual cramps), deep dyspareunia, infertility, pain during pooping, abdominal pain, heavy bleeding or light spotting during periods and fatigue. It also occurs in asymptomatic women who have a risk of developing ovarian and breast cancers, melanoma, asthma, rheumatoid arthritis and cardiovascular disease (Kvaskoff *et al*., 2015). It is regarded as a systemic disease which affects more than one organ, having significant impact on social life, mental health, work activities and sexual relationship. Proposed mechanism for pathogenesis includes retrograde menstruation, coelomic metaplasia, altered immune dysfunction, chronic inflammation, angiogenesis, progesterone resistance and estrogen dominance. Other factors like never having children, short menstrual cycles, having long and heavy periods more than 8 days and epigenetic modifications contribute to endometriosis. Based upon position it has been divided into three types namely superficial, ovarian and deep infiltrating. Even though it is common, it is still poorly understood and often mismanaged and undiagnosed for average global diagnostic delay of 6-10 years (Horne, *et. al*, 2022). The severity of symptoms is not associated with the extent of disease as patients affecting severe disease can be asymptomatic. In about 30% of patients, problem of difficulty in conceiving occur (Presscott et al., 2016). Average global diagnostic delay of 6-10 years (Horne et al., 2022).Conventional therapy includes Non-steroidal Anti –inflammatory drugs, hormonal therapy (combined oral contraceptives), progestins, GnRH agonist and antagonist and surgical excise of lesions. Due to the

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severity of symptoms, different lesion types and hormones, traditional treatment approaches provides only temporary relief from the symptoms and causing severe side effects like weight gain, mood-swings, menopausal like symptoms, lifetime treatment. Surgical treatment carries a risk of long term complications and reoccurrence of disease in future. Choice of surgery and Assisted Reproductive Technologies needs to be individualized based on age, illness, phenotype and reproductive desires. Therefore there is a growing interest in personalised medicine for novel alternative treatment for heterogeneity of endometriosis. It tailors treatment to unique individuals based on lesion types, molecular characteristic and genetic profile using biomarker advanced data analysis, new diagnostic tools like microbiome analysis and multi-omics approaches (Sivahohan et al., 2022).

II. DISEASE HETEROGENEITY AND PATHOPHYSIOLOGY

Based on histopathology and location it is mainly classified into three types: superficial peritoneal endometriosis, endometriotic ovarian cysts and deep infiltrating endometriosis. Peritoneal endometriosis is the most common type, lesions forming on the surface of peritoneum. These lesions do not cause any clinical symptoms. Ovarian endometriotic cysts form cyst-like structures on the ovaries called “endometriomas or chocolate cysts” which contain old blood, vary in size and are associated with infertility and ovarian cancer. Deep infiltrating endometriosis invades visceral organs like ureter, urinary bladder and rectosigmoid by a depth of 5mm or more within or outside peritoneum. Lesions are nodular or fibrotic and cause severe clinical symptoms so surgery is commonly required. Extra pelvic endometriosis is less common and occurs at sites like diaphragm, surgical scars, lungs and thoracic activity (Y.Wang et al, 2020). Revised American Society for Reproductive Medicine classifies endometriosis into 4 stages; stage 1(minimal) consists of small implants with little or no scarring. Stage 2 (mild) includes light lesions and shallow implants on ovary. Stage 3 (moderate) is composed of deeper implants, small cyst and scarring on ovary and pelvic lining. Stage 4 (severe) involves multiple large endometriomas and deep implants leading to adhesions between different organs together like ovary, uterus, bladder or bowel. It has certain limitations that it excludes extra pelvic lesion or deep infiltrating endometriosis and correlates poorly with symptoms intensity (Athan et al., 2025).

It is a complex multifactorial estrogen dependent disease also called “estradiol dependent” caused by irregular regulation of hormone including estrogen dominance and progesterone resistance. It is also affected by sedentary lifestyle, lack of physical activity and increased consumption of alcohol, caffeine and hormone like Oestradiol. Different theories have been proposed to explain the cause of endometriosis. Widely accepted theory of retrograde menstruation, John A. Sampson hypothesize that menstrual blood flows backwards into the pelvic cavity through fallopian tubes where fragment of menstrual tissue adhere to peritoneal organs and form ulcers or lesions. It has high chances of short menstrual cycle, longer menstrual flow and uterine outflow obstruction (Signorile at al, 2022). Coelomic metaplasia theory proposed by Robert Mayer, states that cells originating from coelomic epithelium metaplastically convert into stroma and glands. It explains ovarian endometrioma and cases in premenarchal girls (Konrad et al., 2019). Embryogenetic theory suggests that endometrial tissue develops from embryogenic remnants of mullerian or wolffian ducts. It is found in rare cases, mostly in men.

In immune dysregulation, proinflamamtory pathways prevent apoptotic process and harmful cells stick to distant sites. Ectopic endometrial cells can survive due to decreased natural killer cells activity, altered macrophage function and elevated inflammatory cytokines like macrophages, NK cells, dendritic cell and T cells causing chronic inflammation and development of endometrial lesions. Number of macrophages in peritoneal fluid and ectopic endometrium is elevated (Kapoor et al., 2021). According to metastatic theory during menstruation, tiny fragments of endometrial tissue spread through blood vessels or uterine lymphatics and implant at ectopic sites. It explains endometriosis at distant and uncommon sites like lungs, thighs and intestine, **pleura, brain, umbilicus, and surgical scars** which cannot be explained by retrograde menstruation theory. Angiogenesis and immunological tolerance contribute to proliferation of these cells (Jerman et al., 2015). Endometrial stem cell recruitment theory proposes that endometriosis develop from stem or progenitor cells originating from basal layer of the endometrium or bone marrow (Lamceva et al., 2023).

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III. GENETIC BASIS OF ENDOMETRIOSIS

When endometriosis is detected in a family, the risk of developing the disease increases, indicating inheritance pattern . Twin studies show a higher concordance among monozygotic twins compared to dizygotic twins indicating significant heredity. According to twins studies, heritability of endometriosis is 0.47-0.51, while heritability based on SNPs is about 0.26. Risk factors for endometriosis include shorter menstrual cycles, increased level of estrogen (due to early menarche to late menopause), shorter menstruation period less than 25 days, heavy periods for more than 8 days and never having children.

Numerous susceptibility genes have been identified by genome-wide association studies (GWAS), including those related to developmental pathways (WNT4), cell adhesion and tissue remodeling (VEZT, FN1), immune and inflammatory responses (IL-1A, IL-6, TNF-α), and estrogen biosynthesis and signaling (ESR1, ESR2, CYP19A1). Epigenetic mechanisms such as DNA methylation, histone modification and microRNAs production affecting gene expression, leading to progesterone resistance and estrogen dominance. Gene involved are HOX gene clusters, ESR1, PGR, GATA transcription factor family, 144 WNT signaling and cadherin signaling (E- Cadherin), CYP19A1, CDKNA1. Abnormal ESR2 gene expression is linked to various types and locations ofendometriotic lesions. Hypermethylation of Let-7 microRNA leads to increased expression of Let-7 and loss of inhibition of KRAS and other genes responsible for the growth of endometriosis (Meixell et al., 2022). Hypomethylation of histone and decreased level of H3 and H4 histones are found in ectopic lesions aand eutopic endometrium as compared to healthy women (La Ferlita et al., 2018). Human leukocyte antigen (HLA –G) interacts with KIR2DL4, LILRB1 and LILRB2 receptors on NK cells, antigen presenting cells and T cells. The expression of HLA-G molecules on the ectopic endometrium has been observed (Osinski M. et al., 2018). Three Single Nucleotide Polymorphism (SNPs) are identified as risk factor for causing endometriosis: LINCOO339-WNT4 at locus 1p36.12 (rs2235529) and RND3-RBM43 at locus 2q23.3 (rs1519761 and rs6757804). Meta-analysis identified two additional loci RNF144B-ID4 at 6p22.3 (rs6907340) and HNRNPA3P1- LOC100130539 at 10q11.21 (rs10508881) (Kwapis et al.2017).

Diagnosis

Diagnostic process begins with detailed medical history and pelvic examination, focusing on symptoms like painful menstruation, infertility, pain during intercourse. Transvaginal ultrasound is commonly used as the first line imaging tool for patients suspected of having endometriosis to identify ovarian cyst and deep infiltrating lesions (Kutnezov *et.al*., 2017). Advanced ultrasonography can be used to analyse nodules and deep infiltrating endometriosis. MRI is used to diagnose deep endometriosis which has same sensitivity and specificity to sophistic transvaginal ultrasound. It provides comprehensive detailed information about locations and histological characteristics of endometriosis, but it is quite expensive and time consuming. Laparoscopy is recommended for patients with suspected endometriosis in which imaging does not reveal any clear findings or when previous treatment has been unsuccessful. It remains a standard method for detection of endometriotic lesions with histopathological characteristic. A laparoscope with camera is inserted by the surgeon through a small abdominal incision to visualize and analyse the pelvic organs (Watkins et al., 2021).

Conventional Therapy

First line treatment consists of non -steroidal anti-inflammmatory drugs such as ibuprofen, naproxen which minimize menstrual pain, prostaglandin synthesis and inflammation by blocking cyclooxygenase enzymes (COX-1 and COX-2). Neuro modulators like tricyclic antidepressants (amitryptine) selective serotonin uptake inhibitors and anticonvulsant (gabapentin and pregabal mainly affect the central system regulation. It can also cause side effects like insomnia, sedation and weight gain. Hormonal therapy suppresses ovulation by directly influencing enzymes and steroid receptors on lesions and endometrium. This makes it unsuitable making it unsuitable for individuals seeking pregnancy .It includes estrogen-progestin contraceptives (cyclic or continuous) or progestin only medications which include oral pills, injectables or subcutaneous implants (Grandi *et.al*, 2019). Combined Oral contraceptive contains estrogen-

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progesterone formulations, reducing FSH levels stabilize the endometrium and reduce pain. A number of therapies may be tried to get effective therapy with manageable side effects. A first line treatment can be used for many years after it has been shown to be effective. Second line therapy includes GnRH agonists and antagonist, which decreases the estrogen levels and stop menstrual cycle creating a synthetic menopause leading to the shrinkage of endometriotic tissue. It is often prescribed to adolescents only after surgical confirmation and lesion excision (Shim *et.al*, 2024). Add-back hormone replacement treatment is necessary alongside GnRH antagonist to prevent severe hypoestrogenism and menopausal side effects. Aromatase inhibitors are used for such patients with pain from rectovaginal endometriosis that is unresponsive to other medical treatment or surgery; these should be administered in combination with oral contraceptives, GnRH analogs and progestagens.

If medication therapy is not tolerated by patients or fails to provide adequate relief then surgery becomes an option removing Deep infiltrating endometriosis (DIE), large endometriomas, chronic or severe pain, organ involvement like ureter, bowel and infertility (Becker *et.al*,2022). Laparoscopic excision is usually preferred as gold standard because it enables the complete elimination of visible lesions, restoration of anatomy of pelvic region and adhesiolysis with shorter recovery time and has lower morbidity compared to open surgery. Excision is favoured over ablation especially in case of Deep Infiltrating endometriosis as it provides better pain relief and lower recurrence rates. In complicated cases involving the bowel, bladder or ureter multidisciplinary surgery include procedures like disc resection, segmental bowel resection and shaving. Laparoscopy hysterectomy with or without removal of one or both ovaries are considered for patients such as having dysmennorhea, heavy bleeding, adenomyosis and those who have no desire for pregnancy after proper counseling of risks and benefits. Hysterectomy is more effective treatment than conservative surgery but it is not a complete cure for endometriosis. Removal of both ovaries only slightly reduces pain and leading early surgical menopause with side effects on the heart and bones (Long AJ et al., 2023).

Therapy	Drug	Mechanism of action	Clinical role	Side effects
NSAIDs (Non-steroidal anti-inflammatory drugs)	Ibuprofen,naproxen, mefenamic acid	Decrease prostaglandin production, anti-inflammatory effects	First line treatment for pelvic pain	Gastrointestinal ulcers, edema
Combined oral contraceptives	Ethinyl estradiol + progestin derivatives	Inhibits FSH and LH, increases endometrial apoptosis	First line treatment for pain control	Nausea, vomiting, fatigue, thromboebolism
Progestins	Dienogest,Norethindrone, Medroxyprogesterone	Induces atrophy of ectopic lesions, anti-inflammatory effects	Long term use for chronic pelvic pain	Irregualr bleeding, weight gain
GnRH agonists	Leuprolide, Goserelin	GnRH down regulation in pituitary block lesions	Second line treatment for moderate or severe endometriosis	Vaginal atrophy,bone loss, venous thromboembolism
GnRH anatagonists	Elagolix,relugolix, linzagolix	Inhibit growth and proliferation of endometrial tissue	Used for moderate –severe endometriosis	Low bone density, limited long term use
Aromatase inhibitor	Letrozole,Anastrozole	Inhibit estrogen secretion in ectopic endometriosis	Severe or refractory endometriosis	Ovarian cyst, hypoestrogen symptoms

Table shows Conventional therapy of endometriosis (Mikus et.al, 2023)

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IV. LIMITATIONS

Conventional treatment including the use of medications such as NSAIDs, hormonal therapy and surgery which provides only temporary relief from symptoms. Hormonal therapy cause adverse effects like bloating, weight gain, osteoporosis, bone fractures, gastrointestinal ulcers and cardiovascular issues. These methods cannot be used for women who are wishing to conceive as they suppress ovulation. Symptoms reappear after discontinuing the treatment even after surgery. It carry a risk of complication like organ damage, infection, bleeding and postoperative adhesions which can lead to pelvic pain and infertility. Recurrence rates are high and many women may require multiple surgeries. Overall, these treatments do not provide a complete cure for the endometriosis (Prajapati et.al, 2023).

Personalised medicine in endometriosis

Endometriosis is a heterogeneous disease, meaning that symptoms, pathogenesis, lesion types and treatment response differs among the patients. Conventional treatment follows a standarised strategy, which does not provide optimal outcomes for all patients. Personalised medicine in endometriosis involves tailoring treatment to the patient's specific subtype, lesion location and genetic profile utilizing molecular biomarkers, advanced data analysis and new diagnostic tools such as microbiome analysis. Various biomarkers such as genetic or epigenetic, metabolomic profiles, microRNAs and inflammatory cytokines have been studied to improve treatment. Emerging technologies like artificial intelligence, digital health applications and patient- derived organoid models are being discovered to better understand of variety of lesion and to predict therapeutic response. It also includes different omics technologies like genomics, transcriptomics, proteomics and metabolomics to examine biological process at different molecular levels and to develop predictive models for disease progression (Brulport et al., 2024).

Biomarkers in endometriosis

Biomarkers are measurable biological indicators found in blood, urine, tissue samples and peritoneal fluid. It helps in tracking of disease development and reoccurrence, analyse the treatment response and reduces the time of diagnosis enabling early detection of disease. Various types of biomarkers have been identified, including serum markers, genetic markers, epigenetic signatures, proteomic biomarkers and metabolomic alterations. The discovery of novel biomarkers related to immune dysfunction, inflammation, hormone imbalance and genetic susceptibility in endometriosis has been made possible by advances in omics technologies and molecular biology.

1. **Serum biomarker** – These biomarker are most widely used. Among them, CA-125(Carcinoma Antigen 125) is most common. Its levels are elevated in moderate to severe endometriosis, gynecological condition like pelvic inflammatory disease and ovarian cancer. However its sensitivity is limited to the early stage of the disease, so it is often used in combination with other biomarkers to improve diagnostic accuracy (Feduniaw et al., 2024).Cytokines play a significant role in the implantation of endometriotic foci by promoting angiogenesis, detection and removal of endometrial cells. Women with endometriosis have higher level of cytokines like Interleukin-6 (IL-6), interleukin -8 (IL-8) in serum and peritoneal fluid. Macrophages and other immune cells in the peritoneal environment are activated by IL-6, which enhances the inflammatory response (Zondervan *et.al,*2020). Glycodelin A is a glycoprotein that stimulates cell proliferation and neovascularization. Studies have shown that intracellular adhesion molecule like ICAM-2, IL-6 and glycodelin A are increased in serum of women with endometriosis aged 21 to 48. Glycodelin and IL-6 have sensitivities and specificities of 91.7%and 75.0% respectively for serum detection (Mosbah *et.al,* 2016). Tumor Necrosis Factor-alpha stimulates cell adhesion, invasion and differentiation of ectopic endometrial cells simultaneously promoting angiogenesis and the secretion of other inflammatory mediators (Chapron *et.al*, 2019).Vascular endothelial growth Factor (VEGF) promotes the formation of blood vessel in ectopic endometrial tissue leading to growth of lesions and disease progression. Because of its important role in angiogenesis, it is considered a potential target and diagnostic biomarker (Becker et al., 2022).

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2. Oxidative stress marker – It reflects imbalance between antioxidant defenses and Reactive oxygen Species(ROS). Increased level of oxidative products like malondialdehyde (MDA), asymmetric dimethylarginine(ADMA) and other lipid peroxidation biomarkers cause cellular and membrane damage. Changes in antioxidant enzymes like superoxide dismutase (SOD) and glutathione peroxidase (GP_x) results in impaired defensive mechanism promoting angiogenesis and proliferation of tissues.

3. Extracellular vesicles including apoptotic bodies, microvesicles (100-1000nm) and microscopic vesicles have been identified .as potential biomarker. It is important for IVF related endpoints and helps in detection of endometriosis. For example, Uterine fluid (UF-EV) concentration is associated with mid secretory endometrial condition regulates implantation, Serum SEV-miRNA shows efficacy (AUC>0.8) in predicting clinical pregnancy and delivery in ART populations (Muraka et al., 2025). Extracellular vesicles- derived miRNAs and circular / long non –coding RNAs function as post- transcriptional regulator of different pathways involved in folliculogenesis (KITL/KItl/BMP15), angiogenesis(VEGF/TIE2) stromal decidualization (PR/HOXA10/IGFBP1) and trophoblast adhesion(TROPHININ)

4. Epigenetic and Genetic biomarkers- In genetic biomarker numerous genes are associated with a high risk of disease have been identified though Genome Wide Association Studies (GWAS) (Zondervan et al., 2020). For example genes like WNT4 helps in development of reproductive tract, GREB1 is linked to estrogen-regulated pathway, VEZT is important for cell adhesion and ESR1 encodes the estrogen receptor (Bongiovanni et al.,2021). Epigenetic biomarker involves heritable change in gene expression which occurs without alterations in DNA sequence. For instance, hypermethylation of certain genes like HOXA 10 and progesterone receptor (PR-B) results in reduced gene expression. Hypomethylation of genes like ESR2 leads to increased estrogen activity. Increased histone deacetylase activity altered the acetylation and methylation of H3 and H4 histones which involved in inflammation and apoptosis (Marquardlt et al., 2023).

5. Molecular marker- MicroRNA (miRNAs) is non-invasive biomarker that binds to mRNA, inhibiting translation and promoting degradation. Altered expression of circulating miRNAs in blood, plasma and endometrial tissues has been observed in endometriosis. Deregulation of mi-RNA contributes to inflammation, angiogenesis, proliferation and tissue remodeling. A widely studied group is miR200 family (miR-200a, miR-200b, and miR-200c) controlling epithelial-mesenchymal process. Increased expression of miR-135a/b during the proliferative phase leads to repression of transcription factor HOXA-10. Over expression of miR-194-3pand miR-29 and upregulation of miR-196 or activation of MEK/ERK signaling pathway leads to progesterone resistance and impaired decidualization in endometrial stromal cells (Pei et al., 2018).

Pharmacogenomics aims to optimize treatment based on individual's genetic variation, which affects drug response and side effects. Polymorphism in CYP450 enzymes like CYP3A4 and CYP2C19 change the side effects of oral contraceptives and GnRH analogs. Patients are benefitted from novel therapies like immune-modulators, anti-angiogenic drugs or epigenetic modulators as identified by their genetic profiles.

V. OMICS TECHNOLOGY USED IN ENDOMETRIOSIS

It includes genomics, transcriptomics, proteomics, metabolomics. In genomics Genome Wide Association Studies have identified many susceptibility loci for endometriosis including genes involved in cell proliferation, hormone regulation and immune response. For example genes like ESR1, FSHB and CDC42 regulate hormone related pathways (Zondervan et al.,2020). In transcriptomics advancement in RNA sequencing especially single cell RNA sequencing (scRNA-seq) have shown significant cellular heterogeneity within endometriotic lesions. Changes in inflammatory pathways including cytokines like TNF- α and IL-1β, as well as NFKB pathway contributing progesterone resistance. Advanced techniques like mass spectrometry and protein microarray have enabled the identification of differentially

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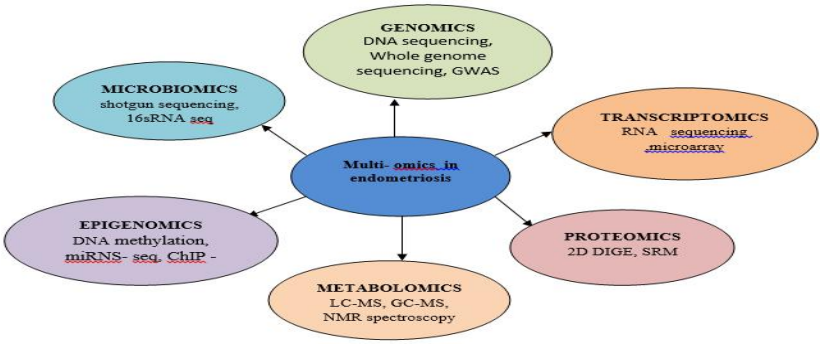
expressed proteins in biological fluids like plasma, serum, peritoneal fluid and ectopic lesions. There is dysregulation of angiogenic factors, adhesion molecules, heat shock proteins and components involved in immunological regulation.

Metabolomics and lipidomics have become powerful approaches endometriosis. A woman with endometriosis shows significant changes in their metabolomic profiles particularly in lipids, amino acids and energy metabolism pathways. These changes lead to the process of oxidative stress, chronic inflammation and cellular proliferation. Metabolites like lactate, succinate, pyruvate and few phospholipids and sphingolipids are elevated indicating problems related to mitochondrial activity. Lipid peroxidation occurs in oxidative stress which promotes tissue damage. MicroRNA and circular RNAs (cir-RNA) are important for inflammation, cell proliferation and epenchymal epithelial transition (Plunk et al., 2022).

In microbiome, patients shows altered microbial communities in the gut, vagina, peritoneal cavity, marked by reduced diversity and increased levels of pro-inflammatory bacteria. It influences estrogen metabolism, immune system and chronic inflammation (Ata et al., 2019). Disruption of the gut microbiota leads to pelvic inflammatory adhesions causing infertility. Patients with deep infiltrating and ovarian endometriosis show increased levels of Ruminococcus and decrease in Firmicutes and Clostridium (Weber et al., 2023).In the vaginal microbiome, beneficial Lactobacillus species decrease, while harmful bacteria like Gardnerella and Atopobium increases. In the uterine microbiota, number of Lactobacillae decreases whereas the ratios of Streptomycetaceae, Staphylococcaceae and Enteriobacteriaceae are increased (Khan et al., 2016).

Recent development in Artificial Intelligence and machine learning are being used to improve the treatment of endometriosis. AI analyses clinical features, genetic data and imaging patterns more precisely. It is also used for prediction of disease and multi-modal data integration to facilitate workflow between algorithms and doctors (Zhang et al., 2025).



Multi- omics approaches in endometriosis

Challenges in personalised medicine

The biological heterogeneity of endometriotic lesions which differ in subtypes and exhibit different responses to treatment makes the development of reliable biomarker challenging. Many biomarkers lack proper validation, standardization and insufficient research studies. Advancement in genomics, proteomics and transcriptomics have produced numerous databases but clinical application of these findings remain slow (Zhan &Cao, 2024). There is a lack of standardized Bioinformatics pipelines and clinicians often lack proper training to interpret complex result generated by omics. Additionally methods like multi-omics profiling and pharmacogenomics are very expensive. Concerns about data security, privacy and informed consent have also been raised (Rosendo et al., 2025).

Future prospects

Personalised medicine is highly promising for future of endometriosis with recent molecular diagnostic tools, advancement in multi-omics technology artificial intelligence and biomarker discovery. The introduction of genomics, proteomics and transcriptomics enables researchers to develop targeted therapy rather than one-size-fits all approach.

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These advancements will allow for more specific predictions of response to hormonal, surgical or immmunomodulatory therapy. Advancement in pharmacogenomics is also expected to help in reducing treatment-related side effects.

VI. CONCLUSION

Personalised medicine is a major shift in the management of endometriosis offering potential to move beyond the traditional "one-size-fits-all" treatment strategy. It takes account of individual's genetic, molecular, environmental factors and lifestyle .Advancement in genomics, transcriptomics, proteomics and biomarker research have improved the understanding of heterogeneous nature of endometriosis and have identified potential targets for precision based interventions. Use of molecular profiling, non-invasive biomarker and patient-specific features is ised for the early detection of disease, predict its progression, maximize treatment effectiveness and minimize the side effects. Artificial intelligence and machine learning helps in better stratification of patients. Despite these advances, significant challenges remain including the need for validation, standardization, conducting large clinical trials and implementing these approaches in a clinical and affordable way. Personalised approaches have great potential to improve the well-being of women suffering from this condition by controlling symptoms, improving fertility , increasing quality of life and effectively managing endometriosis (Stojko ,R,2024).

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