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The Role of Hormone Imbalance in the Pathogenesis of Endometriosis

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Abstract: *Endometriosis (EMS) is a common gynecological disease that affects the reproductive health and quality of life of millions of women worldwide. This study aims to explore the specific role of hormone imbalance in the pathogenesis of EMS, particularly the regulatory roles of estrogen and progesterone in endometrial tissue growth and inflammatory response. In terms of experimental methodology, a classical mouse endometriosis model was firstly established, in which endometrial tissues were transplanted into the abdominal cavity of mice by surgical methods to simulate the pathological state of human EMS. An environment of hormonal imbalance was artificially created by administering different doses of estrogen and progesterone. This step critically mimics the pathological changes that can result from hormonal imbalance. During the treatment period, blood and tissue samples were collected regularly to evaluate the CRP (C-reactive protein) level and the expression of related inflammatory factor (IF) by ELISA (Enzyme Linked Immunosorbent Assay) and immunohistochemistry, and the endothelial tissue hyperplasia was evaluated by microscopic observation and histological analysis to evaluate the proliferation of endothelial tissue. The data on the growth rate of endometriotic tissue showed a gradual decrease in the rate of tissue growth over time after the start of treatment. The gradual decrease from the initial 100% to about 10% indicates that the treatment has a significant inhibitory effect. By clarifying how hormones affect the development of EMS, it helps the medical community to design more targeted interventions to improve treatment outcomes and patients' quality of life, and also provides a scientific basis for developing more effective treatment strategies in the future.*

Keywords: *Hormonal Imbalance, Endometriosis, Pathogenesis, Endometrial Tissue Proliferation*

1. Introduction

EMS is a chronic disease that generally affects the health of women of childbearing age. It is characterized by the abnormal position of endometrial tissue outside the uterine cavity, which leads to pain and infertility. Although researchers have made some progress, the exact pathogenesis of EMS is still not completely clear, which limits the development of effective treatment methods to some extent. Previous studies mainly focused on inflammatory reaction and its contribution to disease progression, but these studies often did not completely solve the role of hormone imbalance in pathological development. Hormone imbalance, especially the abnormal levels of estrogen and progesterone, is considered to be the key factor to promote the development of EMS. In recent years, some studies have pointed out that estrogen not only promotes the growth of intima tissue, but also increases inflammatory reaction, thus aggravating the disease. However, current treatments, such as hormone therapy, do not fully address the problem of recurrence and are often associated with side effects. Therefore, elucidating the role and mechanism of hormone dysregulation in the development of EMS is of great significance for the development of new EMS prevention and treatment strategies.

This paper intends to use an animal model to simulate the hormonal imbalance state in human body, and to explore the role of hormonal imbalance in the development of EMS in depth from both in vivo and in vitro levels. This paper takes estrogen and progesterone as research objects to explore the role of estrogen and progesterone in the disease process from the aspects of cell proliferation, migration, and inflammatory pathways. In addition, this paper will investigate the role of hormone modulation therapy on the development of the disease and explore its palliative effect on the disease. This paper will provide new ideas for the development of novel anti-tumour drugs.

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This paper intends to take mice as the research object and observe its effect on mouse endometrium by regulating hormone levels in vivo. On this basis, this paper proposes to establish an animal model of endometrial injury in rats by using high-throughput sequencing technology to detect the growth rate of endometrium, inflammatory markers and the expression of IF. The main contents are as follows: firstly, we introduce the construction method and steps of EMS animal model in detail. Secondly, we further study the effects of estrogen and progesterone on endometrium, especially on inflammatory markers and cytokines (CK). Ultimately, we elucidate the mechanism of action of hormone modulation therapy on the above indicators and provide a theoretical basis for its clinical application. This study will provide new ideas for the pathogenesis and prevention of EMS.

2. Related Work

The study of EMS has always been an important topic in the field of gynaecological diseases, as it is directly related to the quality of life and fertility of women in general. Past studies have focused on pathophysiology and hormone therapy, attempting to control the development of the disease by regulating hormone levels. He Fangzi conducted a study on EMS moxibustion therapy [1]. Kang Wenwen discussed a case of unilateral bloody pleural effusion complicated by EMS [2]. Cao Gaijing discussed the clinical staging of EMS [3]. Desai S studied the atypical manifestations of hemorrhagic ascites disease [4]. Li M studied the effect of acupuncture on the expression of gene regulatory protein in endometrium of endometriosis rats [5]. However, the current research often ignores the interaction between hormone balance and inflammatory reaction in the body and its influence on the disease process. However, although the understanding of the disease is not deep enough at present.

At present, hormone therapy is widely used in EMS, but its mechanism is still unclear. At present, people pay more and more attention to finding new targets to develop more effective anti-tumor drugs. Ito V explored women's experience of chronic illness and used a mixed analysis of EMS narratives [6]. Ito S used laparoscopic surgery to treat hepatic EMS [7]. Gerasimov A M examined the prevalence and structure of external genital EMS in hospitals [8]. Kojima N reported on cases of abdominal wall EMS in the scarred area after cesarean section [9]. Ceccaroni M used laparoscopic segmental proctocolectomy for the treatment of deep EMS, and retrospectively analyzed the outcome and postoperative complications of 3,050 patients at a referral centre over a period of 17 years [10]. However, most of the current treatment strategies lack targeting and are often accompanied by adverse effects and a tendency for the disease to recur. This suggests that more in-depth research is needed to explore safer and more effective treatments, especially intervention strategies in the early stages of the disease.

3. Methods

3.1 IF and EMS

(1) Association of IF with EMS pathogenesis

EMS is a chronic disease involving several biological factors [11]. Studies have shown that inflammatory factors are important factors in the development of EMS. Our previous study found that there were a large number of inflammatory factors in the tumour microenvironment, which can both promote inflammatory responses and regulate the activities of immune cells, thus playing a role in the development of the disease. Therefore, this project intends to explore the molecular mechanisms of EMS development at the molecular, cellular and molecular levels, and provide new ideas for the prevention and treatment of EMS [12-13].

(2) Mechanisms of action of IF

Inflammatory factors play an important role in the pathogenesis of EMS. Among them, CK, represented by interleukin -1, plays an important role in inflammatory reaction [14]. Our previous research found that there were a large number of inflammatory factors in endometrial tissue, which can induce the aggregation and activation of inflammatory cells and up-regulate cell adhesion molecules, thus affecting abnormal implantation and proliferation of endometrium. Therefore, the study of its regulation mechanism is of great significance to reveal the pathogenesis of the disease.

It is assumed that changes in hormone levels can be described by a first-order linear differential equation, where $H(t)$ represents the hormone concentration, k is the elimination rate constant, and S

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is the hormone synthesis rate.

$$\frac{dH}{dt} = S - kH \tag{1}$$

This equation shows that the change in hormone concentration over time consists of both synthesis and elimination components, reflecting the basic mechanism of dynamic hormone balance in the body.

(3) IF regulates EMS pathological processes

Inflammatory factor is an important factor in regulating the development of EMS. It can not only regulate the inflammatory microenvironment, but also affect the processes of apoptosis, proliferation, and neovascularisation, thus influencing the development of the disease. Inflammatory factors can affect the biological behaviour of diseases through the above pathways, and elucidating their mechanisms of action will help to develop anti-inflammatory therapies with higher targeting.

(4) Interaction between IF and hormonal imbalance

The development of EMS is closely related to endocrine disruption, and the process is further complicated by the interaction between inflammatory factors and endocrine imbalance. Inflammatory factors can affect the normal transmission of hormones by influencing the expression and function of hormone receptors. In addition, some hormones can also regulate the production and secretion of inflammatory factors and constitute a complex interaction with the organism. Elucidation of such interactions is the key to EMS prevention and treatment.

By delving deeper into the role of IF in EMS, we can not only better understand the pathological mechanisms of this complex disease, but also provide possible new avenues for clinical treatment [15].

The organizational growth of EMS can be simulated using the following logistic growth model, where represents the size of the organization, represents the growth rate, and represents the environmental carrying capacity (i.e. the upper limit of growth).

$$\frac{dG}{dt} = rG\left(1 - \frac{G}{K}\right) \tag{2}$$

This model suggests that the growth of endothelial tissue initially grows exponentially and slows down as it approaches the carrying limit.

3.2 CK and EMS

(1) Expression and role of CK in EMS

CK plays a key role in the development of EMS. CK, such as interleukins and growth factors, are generally found in high levels in ectopic endometrial tissues. CK can regulate the immune response, promote inflammatory responses, and facilitate cell proliferation and migration, thus promoting the invasion and survival of ectopic endometrium.

(2) Relevance of CK to the pathogenesis of EMS

CK is the central link in the development of EMS. Analyzing the special function played by CK in the process of EMS occurrence and development as well as the inter-regulatory relationship between them is the key to study the pathogenesis of EMS.

The direct effect of hormones on the growth of endometrial tissue can be described by the following equation, where αrepresents the intensity of the hormone's impact on endometrial tissue growth:

$$\frac{dG}{dt} = rG\left(1 - \frac{G}{K}\right) + \alpha H(t) \tag{3}$$

This equation adds a term $\alpha H(t)$, which represents the positive stimulatory effect of hormone levels on endothelial tissue growth.

(3) The role of 3)CK in regulating the pathological process of EMS

It is found that many kinds of CK participate in the pathogenesis of EMS. It can not only regulate the inflammatory reaction, but also affect the functions of cell migration and infiltration, thus affecting the development of diseases. It is reported in literature that IF can regulate cell cycle and apoptosis by regulating immune cell function and intracellular signaling pathway.

(4) Interaction between 4)CK and hormone imbalance and IF.

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EMS is the pathogenesis of EMS with CK, hormone balance and inflammatory factors as the core. For example, estrogen can regulate some CK, and then affect hormone receptors. In addition, inflammatory factors such as interleukin -1 and tumor necrosis factor α can participate in the pathogenesis of EMS by regulating hormone synthesis and metabolism. Understanding the specific process of their interaction is very important to reveal the overall picture of the disease.

The production of IF$I(t)$may be related to hormone levels, and let β denote the coefficient of hormonal influence on IF production:

$$\frac{dI}{dt} = \beta H(t) - \gamma I(t) \tag{4}$$

Here, γ is the natural rate of decay of IF, and the equation indicates that IF production is directly stimulated by hormone levels and has a natural process of abatement.

The hormone levels of different groups are shown in Table 1. The levels of estrogen, luteinizing hormone, follicle stimulating hormone, and cortisol in patients with endometriosis were significantly higher than those in the healthy control group, while the levels of progesterone and gonadotropin-releasing hormone were significantly reduced. These hormonal imbalances may promote the onset and expansion of endometriosis.

Table 1: Hormone levels in different groups

Hormone	Average level of healthy control group	Average level of patients with endometriosis	P value
Estrogen	50 pg/mL	75 pg/mL	< 0.01
Progesterone	10 ng/mL	5 ng/mL	< 0.05
Androgen	70 ng/dL	60 ng/dL	< 0.05
Gonadotropin Releasing hormone	3.0 ng/mL	2.5 ng/mL	< 0.01
Luteinizing hormone	5.5 IU/L	7.0 IU/L	< 0.01
Follicle stimulating hormone	6.0 IU/L	8.0 IU/L	< 0.05
Cortisol	10 μg/dL	15 μg/dL	< 0.05
Antidiuretic hormone	3.0 pg/mL	4.5 pg/mL	< 0.01

4. Results and Discussion

4.1 Diagnosis and Treatment

(1) Clinical manifestations and diagnostic methods of EMS

The clinical manifestations of EMS are diverse, with common symptoms including menstrual pain, chronic pelvic pain, and infertility. These symptoms tend to vary from person to person and are sometimes related to the location and size of the lesion. In terms of diagnostic methods, ultrasound is the preferred non-invasive method to detect ectopic endometrial cysts in the uterus and ovaries. In addition, magnetic resonance imaging can be used to assess the extent and depth of the lesion. Confirmation of the diagnosis usually requires laparoscopic surgery, where tissue samples are obtained under direct vision for pathological examination.

(2) Methods and effects of hormonal modulation in the treatment of EMS

Hormone therapy is one of the commonly used treatments for EMS, mainly by inhibiting ovarian function and reducing estrogen levels, thus inhibiting the activity and growth of endometriotic foci. Commonly used hormone therapy drugs include progestins, oral contraceptives and luteinising hormone-releasing hormone analogues. These treatments are effective in relieving pain symptoms and controlling the progression of the disease, but the disease may recur when the drugs are discontinued. The effects of hormone therapy on EMS are shown in Figure 1.

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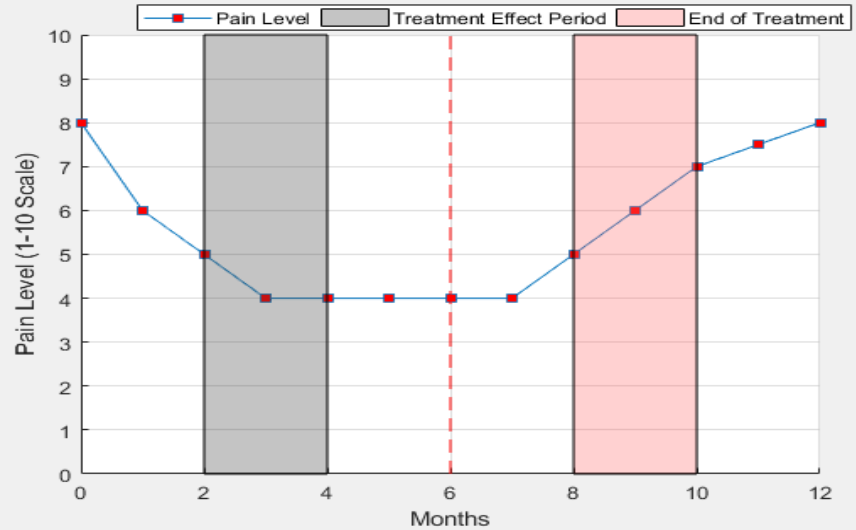

Figure 1: The effect of hormone therapy on EMS

In this figure, we show the effect of hormone therapy on EMS pain ratings. It is clear from the graph that there is a significant decrease in pain rating at the beginning of the treatment period (2 to 4 months), which suggests that the hormone therapy is effective in relieving the symptoms in the short term. The pain rating decreased from 5 to 4 during this period.

In the middle of the treatment period (May to July), pain grades remained low, indicating a sustained effect of hormone therapy. However, once the treatment was over, especially between July and October, we observed a renewed rise in pain levels.

The red dotted line in the figure marks the point at which treatment ended, providing us with a clear visual demarcation of the change in pain level after treatment cessation. Overall, this figure effectively demonstrates the short-term effects of hormone therapy in controlling EMS pain and its persistent limitations, emphasizing that patients may require further management and follow-up after treatment to sustain disease control and improve quality of life.

(3) Targeted therapy and immunomodulatory therapy

Targeted therapy and immunomodulatory therapy are relatively new approaches in EMS treatment. Targeted drugs, such as aromatase inhibitors, are treatments that target specific biomarkers in an attempt to reduce estrogen synthesis and inhibit lesion growth.

The effects of targeted and immunomodulatory therapies on physiological and symptomatic changes in EMS patients are shown in Figure 2.

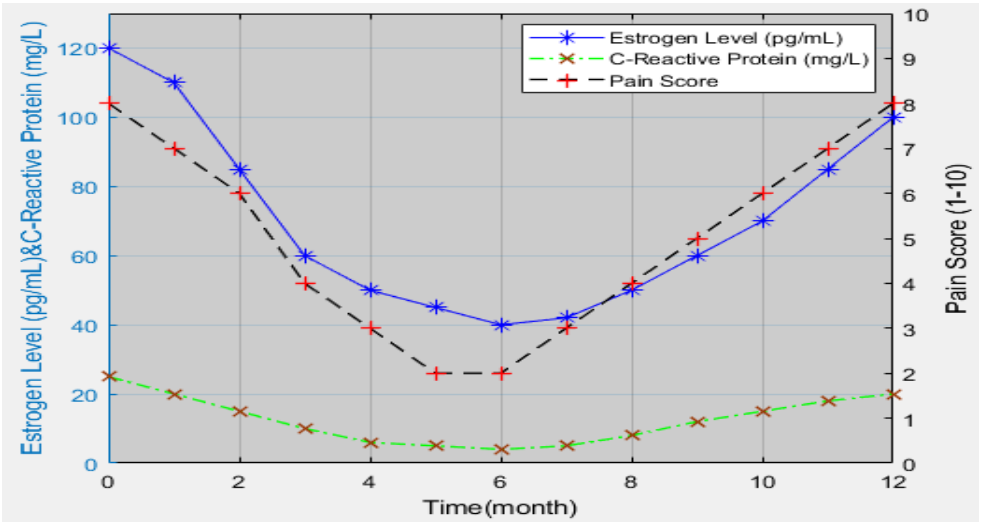

Figure 2: Effects of targeted therapy and immunomodulatory therapy on physiological and symptom changes in EMS patients

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Figure 2 presents three main data series: estrogen levels, CRP levels (an inflammatory marker), and patient pain scores.

At the beginning, the concentration of estrogen has decreased, from 120 pg/ml to 40 pg/ml.

Subsequently, the C-reactive protein concentration showed a similar change, decreasing from 25 mg/L to 4 mg/L, indicating that the inflammatory reaction had been effectively controlled. After completion of the treatment, this measurement increased slightly, suggesting a possible rebound of the inflammatory reaction or recurrence of the disease.

Pain scores dropped from a high score of 8 before treatment to 2 and then slowly returned to 8 after treatment was completed. This curve visualizes the immediate effect of the treatment modality on reducing symptoms and the limitations of the duration.

Accordingly, we have clarified the near-term efficacy of targeted and immunomodulatory therapies in reducing oestrogens, inflammatory markers, and pain relief, but also the risk of recurrence after discontinuation.

(4) Surgical treatment and assisted reproductive technology

Surgery can remove lesions, relieve symptoms and improve fertility. However, the risks of surgery include possible recurrence and surgery-related complications. For patients who wish to preserve their fertility, assisted reproductive technologies (e.g., in vitro fertilisation) offer another option. These techniques can help patients achieve pregnancy, although they do not cure the condition itself.

This provides an understanding of the diagnostic and therapeutic approach to EMS, which is critical to improving the quality of life of patients and managing disease-related complications.

4.2 Prevention and Rehabilitation

(1)Preventive strategies for EMS

EMS can reduce the occurrence of some risk factors by changing patients' living habits and early intervention. The prevention method can be to maintain a normal weight, because overweight will cause endocrine disorders, which will make the situation worse. Another point is that moderate physical exercise can help reduce the chance of getting sick. In addition, some hormonal contraceptives can also help reduce the risk of endometriosis.

(2) Rehabilitation nursing and lifestyle adjustment

For EMS patients, treatment and living habits are an important part of the treatment of the disease. Patients should eat more whole grains, fruits and unsaturated fatty acids to reduce inflammation and pain. Regular exercise helps to promote blood circulation and relieve pain. In addition, adequate sleep and avoiding excessive tension can also help alleviate these symptoms.

(3) Psychological counseling and social support

EMS will not only bring physical harm to women, but also bring mental and emotional troubles to women. Therefore, psychological counseling has become an important part of rehabilitation work. With professional counseling, patients can learn to cope with the emotional tension caused by illness. In addition, participating in support groups and communicating with patients with similar experiences can provide them with necessary social support, so that they can better face the disease.

(4) Evaluation and follow-up of rehabilitation effect

The assessment usually includes regular medical check-ups, recording of symptoms, and questionnaires to assess the quality of life. Follow-up visits are extremely important for monitoring changes in the condition, making timely adjustments to treatment strategies, and providing ongoing support. Regular follow-up visits can help the healthcare team catch potential relapses or problems in treatment and ensure that patients are able to achieve the best possible outcome.

The results of the rehabilitation outcome assessment and follow-up visits are shown in Figure 3.

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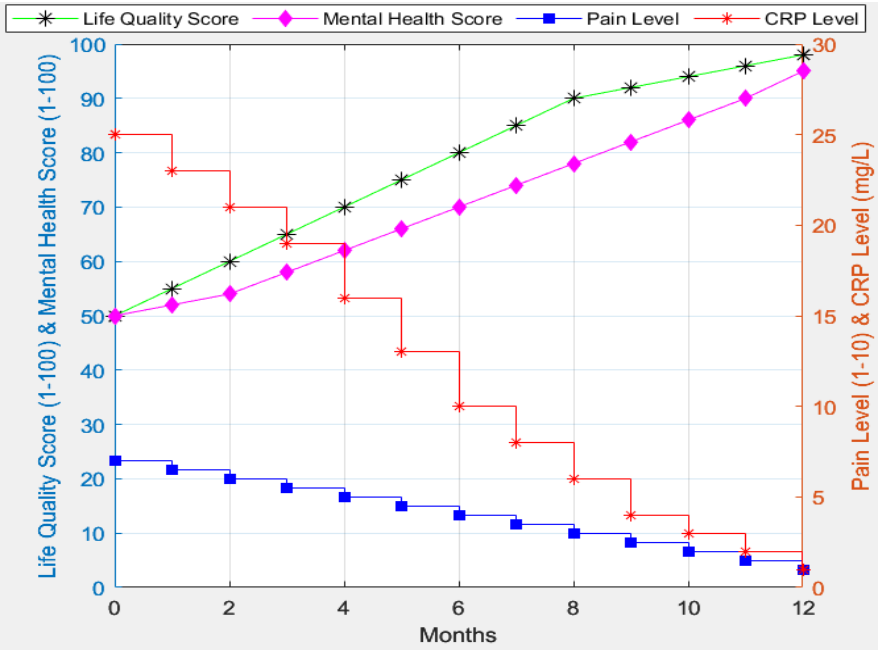

Figure 3: Assessment of rehabilitation effects and follow-up results

Through Figure 3, we can see the changes in various physical functions of EMS patients after rehab and lifestyle habits. Taking 12 months as a cycle, the information shows that such changes have a great impact on the patients.

Firstly, on the left, the Y-axis data is progressively increasing. Rehabilitation and lifestyle modifications through improving diet, increasing exercise, controlling stress, ensuring adequate rest play a significant role in improving the overall well-being of patients.

The right Y-axis, on the other hand, indicates the level of pain and the level of CRP. The pain level decreased from a score of 7 at the beginning to a score of 1, and the concentration of CRP also decreased from 25 mg/l to between 0-5 mg/l, indicating that both inflammation and pain were effectively controlled. The improvement of all the above physiological indicators was mutually reinforcing with the improvement of the patient's survival and mental state.

Overall, this figure shows the positive impact of rehabilitative care and lifestyle improvements in improving patients' quality of life and managing their illnesses.

4.3 Example Exploration and Result Analysis

(1) Animal model construction

In the study of the pathogenesis and treatment strategy of EMS, the construction of animal model is a crucial step. Usually, we use rats or mice as experimental animals, and build an endometriosis model by transplanting the endometrial tissue of animals into abdominal cavity or subcutaneous tissue. This model can simulate the pathological process of human EMS, including ectopic growth of endometrial tissue and related inflammatory reactions. In the experiment, we strictly monitor the health status of animals, and analyze the growth and pathological changes of intima tissue through regular sampling.

(2) The effect of hormone imbalance on the pathogenesis of EMS

Through animal experiments, we further discussed the influence of hormone imbalance on the pathogenesis of EMS. Generally speaking, it is observed in the experiment that the growth of endometriosis tissue in animal model is more active in the environment of hormone imbalance. Especially when the level of estrogen is increased, the proliferation of ectopic endometrium is significantly accelerated, accompanied by the intensification of inflammatory reaction. This shows that estrogen plays a key role in promoting the development of EMS, which intensifies the pathological process of the disease by promoting the proliferation and migration of endometrial cells.

Through quantitative PCR and histological analysis, we can also find that hormone imbalance significantly increases the expression mechanism of inflammatory CK. These factors include

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interleukin -6 and tumor necrosis factor α, which form an inflammatory microenvironment at the lesion site and further promote the pathological process. These results emphasize the importance of regulating hormone levels in the treatment of EMS.

(3) Experimental verification of hormone control therapy.

In view of the influence of hormone imbalance on the pathogenesis of EMS, we further verified the effect and mechanism of hormone regulation therapy in animal models. Progesterone and estrogen receptor antagonists were used in the experiment to observe their effects on the growth of endometriosis tissue. The results show that these hormone-regulating drugs can effectively inhibit the proliferation of ectopic endometrium and alleviate the inflammatory situation.

The effects of hormone modulation therapy on EMS are shown in Figure 4.

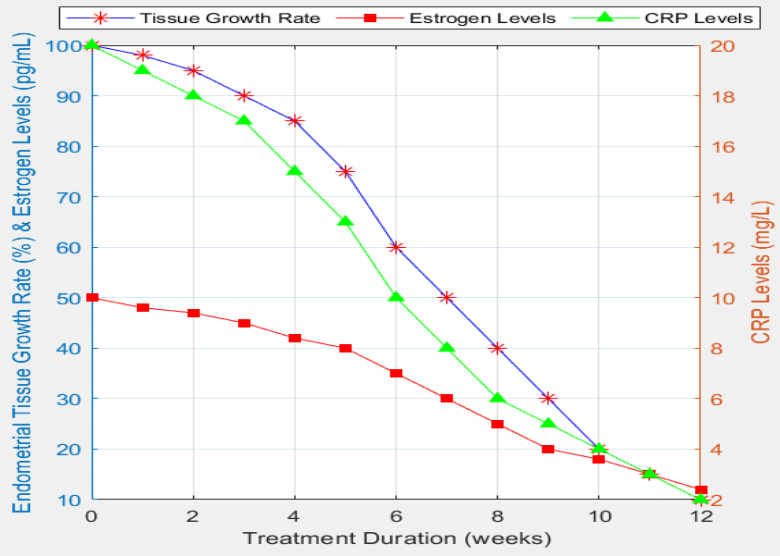

Figure 4: The effect of hormone regulated therapy on EMS

Here, we can clearly observe the effect of hormone modulation therapy on EMS. Three main data series are shown: endometriotic tissue growth rate, CRP levels and oestrogen levels, which reflect physiological changes during treatment.

Firstly, the data on endothelial tissue growth rates show a gradual decrease in tissue growth rates over time after the start of treatment. This suggests that hormone modulation therapy is effective in inhibiting endothelial tissue overgrowth, a key goal in EMS therapy. The gradual decrease from the initial 100 per cent to approximately 10 per cent suggests that the treatment has a significant inhibitory effect.

Secondly, the data on CRP levels showed a similar downward trend. The decrease in CRP levels precisely reflects the reduction of the inflammatory state in the body. This is consistent with the mechanism of action of hormone therapy in reducing inflammatory CK release, further demonstrating the effectiveness of hormone modulated therapy in reducing inflammation.

Ultimately, the estrogen data also showed a significant decrease. Estrogen is an important hormone that regulates female reproductive function, and its imbalance plays an important role in the development of EMS. The decrease in oestrogen levels during the course of treatment indicates that the hormonal balance in the body is effectively regulated in favour of further progression of the disease.

This study will provide new ideas for the prevention and treatment of EMS. This result will be of great help in understanding and evaluating the efficacy of tumours.

5. Conclusion

In this paper, we propose to conduct a systematic study on the changes of hormone levels during the development of EMS from both in vivo and in vitro levels. In our previous study, we found that high estrogen can significantly promote endometrial hyperplasia, which is in line with the hypothesis of "hormonal imbalance leads to ectopic endometrial lesions". Hormone modulation therapy can

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significantly reduce the expression of CRP and slow down disease progression. This study will provide new ideas for the prevention and treatment of EMS.

The shortcomings of this paper are: firstly, this study takes animals as research subjects, whose physiological state is quite different from that of human body, and their human adaptive ability needs to be further evaluated. Secondly, we only explored other factors of EMS occurrence and development from the perspective of hormonal imbalance, such as genes, immunity, etc., while ignoring other factors of EMS occurrence and development. In addition, the number of samples in the efficacy study was small, and further research is needed.

In response to the limitations of the current study and the future direction of EMS research, firstly, a multi-model and multi-sample approach should be considered in future studies to verify the generalisability of the effects of hormonal imbalance on EMS and its mechanisms. Second, exploring multifactorial interactions, including genetic markers, may provide new perspectives for understanding the complexity of the disease. In addition, the development of new therapeutic approaches, such as targeted drugs or gene editing techniques, is an important direction for future research. Finally, increasing collaboration in interdisciplinary research, such as the integration of biology, immunology and clinical medicine, will contribute to a comprehensive understanding and treatment of EMS. In conclusion, through in-depth studies on hormonal imbalance and EMS, we can not only better understand the pathological mechanisms of the disease, but also promote the development of more effective treatment strategies. Future studies should build on the existing foundation and explore further with the aim of achieving better disease management and therapeutic outcomes.

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