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Reproductive Endocrine Mechanisms Regulating Ovarian Reserve in Polyendocrine Metabolic Ovarian Syndrome

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ABSTRACT

This paper investigates the reproductive endocrine mechanisms of the ovarian reserve in polyendocrinopathy, autoimmune polyglandular syndrome type 1 (APCOS), which is a new term for polycystic ovary syndrome (PCOS). Rather than viewing ovarian reserve as a single number, the paper believes that the concept of reserve in PMOs should be understood through the interplay of follicle number, follicular arrest, granulosa-cell signalling, gonadotropin imbalance, androgen excess and metabolic disturbances. Based on the major consensus statements and endocrine studies, this essay will explore how disturbed hypothalamic-pituitary-ovarian signalling, functional ovarian hyperandrogenism, insulin-related dysregulation and anti-Mullerian hormone overproduction affect folliculogenesis and complicate the interpretation of ovarian reserve markers. PMOS also has a problem: although it appears to be in an overstocked state, it may not be ready to reproduce normally. Therefore, Ovarian Reserve Markers in PMOS should be considered in the context of the whole-body endocrine environment and not regarded as fixed indicators of ovulation potential.

KEYWORDS

Polyendocrine metabolic ovarian syndrome; Ovarian reserve; Reproductive endocrinology; Anti-Mullerian hormone; Folliculogenesis; Hyperandrogenism

1. INTRODUCTION

Polyendemic metabolic ovarian syndrome is a relatively severe reproductive endocrine disorder in women of childbearing age. Disruptions in ovulation and other problems with the menstrual cycle and fertility, as well as long-term endocrine-metabolic diseases, are all related to this. Ovarian reserve in a clinical setting is generally considered a quantitative index and is often expressed as biomarkers or follicle counts. However, the concept of ovarian reserve is more complex in this syndrome due to the co-occurrence of follicle excess, impaired selection and endocrine disorders.

Therefore, the ovarian reserve in polyendocrine metabolic ovarian syndrome should not be expressed as a single laboratory index. There is a contradiction in the syndrome: although the size of the ovary and the quantity of anti-Müllerian hormone are large reserves, ovulatory dysfunction and impaired follicular development have reduced fertility. Therefore, a mechanism of action in the reproductive endocrine system will be proposed.

This paper will explore the endocrine mechanisms that regulate ovarian reserve in polyendocrine metabolic ovarian syndrome. It believes that ovarian reserve markers in this state should be considered in conjunction with impaired folliculogenesis due to abnormal gonadotropin, androgen, insulin-related and granulosa-cell signalling. Based on the above, this paper first introduces the concept of ovarian reserve in the syndrome, then explores the corresponding reproductive endocrine changes and discusses the clinical significance.

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2. OVARIAN RESERVE IN THE DIAGNOSTIC AND CONCEPTUAL CONTEXT OF PMOS

All talks about the ovarian reserve in polyendocrine metabolic ovarian syndrome should start with the diagnostic system for this disease. The Rotterdam consensus defined the syndrome as a combination of ovulatory dysfunction, hyperandrogenism and polycystic ovary morphology, and also pointed out that no single sign is sufficient [1]. It is necessary to consider ovarian reserve, and ovarian morphology has been included in the diagnosis for a long time without fully understanding its biological significance.

In 2019, the endocrine focus of the syndrome has been enhanced by the "Androgen Excess Society" guidelines, which now list hyperandrogenism as one of the main characteristics and direct attention towards endocrine disorders rather than morphology alone [2]. Therefore, the alteration of ovarian reserve in PMOS cannot be understood in isolation from androgen-related dysregulation. A follicle-rich ovary is not necessarily a normal-regulation ovary.

Recently, the international community has reached a consensus to rename the condition polyendocrine metabolic ovarian syndrome, and it has been determined that this disease encompasses multiple endocrine disorders and metabolic problems in the ovary [3]. The new names for these studies of the ovarian reserve are conceptually more convenient and less structurally interpretive. It is proposed that the ovarian reserve in PMOS represents a whole-organ endocrine system, and thus cannot be diagnosed solely based on ultrasound morphology.

Some data from this problem have also been provided in the previous clinical reports. Franks called this a disease of prolonged anovulation and androgen excess with characteristic ovarian changes, but he did not believe that the number of follicles directly determined ovulation [4]. The previous idea is still valid; now we know that 'ovarian reserve' should refer to follicular dynamics rather than the number of follicles.

Ehrmann's later review also noted that the syndrome is heterogeneous and clinically presents as reproductive and metabolic disorders rather than just ovarian changes [5]. This difference is what we mean by ovarian reserve. Although all these different phenotypic expressions may exhibit increased small-follicle activity, they differ in ovulatory regularity, endocrine severity and metabolic background; thus, reserve markers cannot be interpreted in isolation.

Together, these conceptual developments show why ovarian reserve in PMOS cannot be equated with the ordinary model of reproductive ageing. Many patients have preserved or even increased numbers of reserve markers but still show disrupted dominant follicle selection. Therefore, the follicles have not been lost; rather, there is a problem with the regulation of their development by the endocrine system.

3. REPRODUCTIVE ENDOCRINE MECHANISMS REGULATING OVARIAN RESERVE

The first is a mechanism of disorganised hypothalamic-pituitary-ovarian signalling. Ehrmann believes that this syndrome is often associated with abnormal gonadotropin fluctuations, particularly a relatively high ratio of luteinising hormone to follicle-stimulating hormone; thus, there is chronic anovulation and altered follicular development [5]. The asymmetry of follicular development is difficult to interpret in terms of ovarian reserve, and these follicles may stall at the small antral stage without reaching ovulation. Therefore, there is a relative abundance of apparent reserves and defective selection.

The second is a mechanism of inhibition by anti-Müllerian hormone (AMH). La Marca and Volpe have been using anti-Mullerian hormone to evaluate the size of the ovarian follicle pool and the

presence of small growing follicles in a clinical setting [6]. It is difficult to determine whether the increased concentration of this marker in PMOS is due to an increase in small follicle numbers, altered granulosa-cell function, or both, from this marker alone. Therefore, anti-Müllerian hormone only indicates the extent of the reserve and does not distinguish between preserved physiological function and arrested folliculogenesis.

The third way is granulosa cell overproduction of anti-Müllerian hormone (AMH). Pellatt and others have shown that granulosa cells from polycystic ovaries produce increased anti-Müllerian hormone (AMH), and thus the high-hormone state of the syndrome may not be due to an excess of follicles [7-8]. This may be due to the state of ovarian reserve, and thus the endocrine microenvironment of the follicles may have been altered. Amplification of the reserve signal occurs due to abnormal follicular function.

A fourth way is the changes in the physiology of anti-Müllerian hormone during follicular development. Dewailly and his colleagues have indicated that anti-Mullerian hormone is related to preantral and small antral follicle development, has some applications in practice, but should be used cautiously in cases of disrupted follicle recruitment and selection [8]. PMOS are used to indicate that a high amount of anti-Mullerian hormone (AMH) may be present in a large number of recruitable follicles; however, it can also indicate continued developmental arrest. The Ovarian Reserve is therefore quantitatively abundant but functionally restricted.

A fifth mechanism is the connection among altered metabolism and ovarian function. Dumesic and his colleagues have described this syndrome as a group of disorders in reproduction, metabolism and endocrinology that occur at the same time [9]. The above expanded view is required to address the issue that insulin-related disorders may increase ovarian androgen secretion and change follicular regulation. Therefore, ovarian reserve in PMOs should be viewed in light of the metabolic-endocrine network and not solely within the ovary.

The sixth is functional ovarian hyperandrogenism. Rosenfield and Ehrmann have been studying again whether intrinsic ovarian androgen excess causes diseases and impairs follicular development and other disorders of the ovary [10]. Excess androgens are required for the initial development of early follicles; otherwise, if there is a prolonged excess of androgens, no maturation will occur and a dominant follicle may fail to develop. It can help explain the PMO paradox: although there are a relatively large number of small follicles in the ovary, they are not being efficiently developed into mature ovulatory follicles.

A seventh way is the disruption of feedback between follicle development and endocrine signals. When small follicles accumulate and continue to produce anti-Mullerian hormone and steroid precursors, the ovary sends out a regulatory signal that is not in line with normal cyclical maturation [6, 8]. Therefore, the endocrine markers of reserve are partially decoupled from the endocrine markers of successful selection and ovulation. A reserve pool exists, but it has not shown any development.

The eighth mechanism is a change in granulosa-theca cooperation. Gonadotropin support generally promotes androgen synthesis by the theca cells and aromatisation and maturation of granulosa cells. Coordination is disrupted in PMOs by excessive androgenic drive and impaired follicular responsiveness; therefore, the endocrine support for growth favours the persistence of small follicles over organised maturation [5, 10]. Thus, the apparent reserves are not directly linked to reproductive function.

These mechanisms should not be considered independent paths. Gonadotropin deficiency, anti-Mullerian hormone excess, granulosa-cell dysfunction, insulin-related disturbances and ovarian hyperandrogenism all occur together. Together, they have altered the biological concept of ovarian reserve from a simple number of follicles to a problem of endocrine regulation, developmental timing and follicular competence.

The connections among the above mechanisms can be shown at three levels of regulation. Changes in the neuroendocrine axis alter the balance of gonadal stimulation at the level of the ovary. Increased androgen exposure and excessive anti-Mullerian hormone activity at the intraovarian level affect granulosa-theca coordination, follicle sensitivity and dominant-follicle selection. Insulin-related disorders can enhance androgen production and alter the ovarian response to gonadotropins at the metabolic level [5, 9, 10]. The above levels are interdependent, not in sequence. Hyperinsulinemia increases the production of androgens by the ovaries, androgen excess promotes the survival of small antral follicles, and the expansion of the small-follicle cohort may be responsible for elevated anti-Müllerian hormone (AMH) levels. The resulting endocrine pattern may preserve or increase the amount of common reserve factors and reduce the efficiency of follicular maturation simultaneously. The above integrated view can avoid attributing all the reasons for the PMOS abnormality to a single cause. It can be seen from this that the clinical paradox is due to a combination of factors affecting follicle number and regulation in the ovary; although there may be many small follicles, the hormonal environment is not conducive to their organisation for selection and ovulation.

4. CLINICAL INTERPRETATION OF OVARIAN RESERVE IN PMOS

Therefore, the clinical assessment of ovarian reserve in polyendocrine metabolic ovarian syndrome should move away from single-marker analysis. In 2023, the international evidence-based guideline proposed a specific diagnosis and management interpretation for this syndrome, and did not attribute the disease features or clinical decisions to isolated findings [11]. This rule directly refers to ovarian reserve assessment. Anti-Mullerian hormone (AMH), antral follicle count, ovulation history, androgen status and metabolic environment should all be considered simultaneously, not in isolation.

Endocrinologically, the indices of ovarian reserve in PMOS need to be interpreted in the context of dynamic endocrinology and folliculogenesis. The Rotterdam framework and the following endocrine guidelines already indicate that ovarian structure, androgen excess and ovulatory dysfunction are interconnected but not identical regions of disease manifestation [1, 2]. A patient has a high reserve marker but still exhibits follicular arrest or irregular ovulation. In other words, the reserved amount is not the same as the breeding efficiency.

Distinguish between the two in the context of fertility counselling when using anti-Müllerian hormone (Anti-MCH). La Marca and Volpe's studies, as well as those by Dewailly and others, have supported the value of anti-Mullerian hormone as a biomarker and also pointed out the need for physiological interpretation in disorders of abnormal follicle dynamics [6, 8]. Elevated anti-Mullerian hormone in PMOs is associated with a large small-follicle pool, but it does not necessarily indicate that oocyte development will proceed normally. A syndrome changes the meaning of a biomarker by modifying it.

Thus, in practice, it should be clear whether an index relates to ovarian reserve or fertility. Anti-Mullerian hormone and antral follicle count are generally related to the quantity or activity of the small-follicle pool; they do not directly indicate oocyte quality, the chance of spontaneous ovulation, or the risk of pregnancy for an individual [6, 8]. Differential operation of PMOS transistors is required because, for instance, the same endocrine disorder may increase the small-follicle area while inhibiting dominant-follicle selection. Therefore, the raised reserve markers should be regarded as indicators of an enlarged or highly active small-follicle compartment, and not as evidence of unrestrained reproductive capacity. Age, menstrual and ovulation history, androgen levels, metabolic disorders, ultrasound results, the reasons for reserve tests, etc., should all be considered before reaching a conclusion [11]. For instance, a marker can predict changes in the ovary after fertility treatment but not whether a person is naturally fertile. Separate the above questions to make the arguments more specific and avoid combining ovarian reserve, follicular function and general fertility. It would also be consistent with the standard reproductive-endocrine nomenclature of the paper:

PMOS alters the meaning of reserve markers, but it does not equate ovarian reserve and ovulatory competence.

Knowingly identify androgen excess in the context of reduced ovarian function. Broader pathophysiological syntheses by Dumesic and colleagues, as well as Rosenfield and Ehrmann, suggest that ovarian dysfunction in the syndrome results from continuous endocrine cross-talk rather than isolated ovarian morphology [9, 10]. Therefore, the study of reproductive hormones will focus on how they regulate follicular recruitment, survival, arrest and ovulation. Thus, it can be seen why the preserved reserve does not indicate infertility.

Another is a concept problem. Franks's first clinical paper and Ehrmann's later review have shown that for a long time, the syndrome has been difficult to associate with normal fertility [4, 5]. The new framework of PMOS further refines this interpretation by placing endocrine and metabolic diseases at the centre of research on diseases [3]. Ovarian Reserve in PMOS should thus be considered a regulated functional reserve, rather than just an anatomical store.

The same reason will cause the doctor to diagnose the disease differently. Reserve markers may be reassuring according to the general infertility model, but the endocrine pattern of PMOS can still indicate irregular maturation and complex treatment [8, 11]. Thus, a good interpretation should not be based on a single figure but on the syndrome-specific physiology that yields that figure.

This way is also suitable for academic terms. When authors describe ovarian reserve only as preserved or high in PMOS, they may be transferring assumptions from normal ovarian ageing that do not fit the syndrome. A more precise description is that the reserve index often indicates an increased small-follicle area under dysregulated endocrine conditions, and such a state requires functional interpretation rather than simple ranking.

Thus, it will be possible to avoid the common interpretation errors in academic writing and clinical discussion. When the reserve markers are presented without mention of endocrine regulation, the syndrome may appear to have only an increased capacity of the ovaries. The current problem is how to organise the endocrine environment for follicle recruitment, selection and maturation and ovulation. Therefore, the assessment of the Ovarian Reserve in PMOS should be in a mechanistic way rather than descriptively.

5. CONCLUSION

Ovarian reserve markers in polyendocrine metabolic ovarian syndrome cannot be reliably determined from follicle count or a single biochemical test. The syndrome is a reproductive endocrine paradox; that is, there is an apparent excess of reserves but abnormal follicular development, ovulatory dysfunction and impaired reproductive function. The conflict will be more pronounced if we treat the ovarian reserve as an organised process rather than a fixed amount.

This paper has proposed that the main reproductive endocrine mechanisms regulating the ovarian reserve in PMOS include hypothalamic-pituitary-ovarian dysregulation, anti-Mullerian hormone overexpression, granulosa-cell dysfunction, metabolic-endocrine interaction, and functional ovarian hyperandrogenism. Together, these mechanisms will change both the number of visible follicles and the behaviour of those follicles in the ovary.

Therefore, the following research on the ovarian reserve of PMOs should focus on endocrine regulation and follicular function. The first two problems are not a larger number of follicles in the ovary, but rather whether the endocrine environment can promote the healthy growth and release of these follicles.

REFERENCES

[1] Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. (2004). Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Human Reproduction, 19(1), 41–47.

[2] Azziz, R., Carmina, E., Dewailly, D., Diamanti-Kandarakis, E., Escobar-Morreale, H. F., Futterweit, W., Janssen, O. E., Legro, R. S., Norman, R. J., Taylor, A. E., & Witchel, S. F. (2006). Criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: An Androgen Excess Society guideline. The Journal of Clinical Endocrinology & Metabolism, 91(11), 4237–4245.

[3] Teede, H. J., Khomami, M. B., Morman, R., Laven, J. S. E., Joham, A. E., Costello, M. F., Patil, M., Rees, D. A., Berry, L., Cree, M. G., Zhao, H., Norman, R. J., Dokras, A., Piltonen, T., & Global Name Change Consortium. (2026). Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: A multistep global consensus process. The Lancet, 407(10545), 2329–2339.

[4] Franks, S. (1995). Polycystic ovary syndrome. The New England Journal of Medicine, 333(13), 853–861.

[5] Ehrmann, D. A. (2005). Polycystic ovary syndrome. The New England Journal of Medicine, 352(12), 1223–1236.

[6] La Marca, A., & Volpe, A. (2006). Anti-Müllerian hormone (AMH) in female reproduction: Is measurement of circulating AMH a useful tool?. Clinical Endocrinology, 64(6), 603–610.

[7] Pellatt, L., Hanna, L., Brincat, M., Galea, R., Brain, H., Whitehead, S., & Mason, H. (2007). Granulosa cell production of anti-Müllerian hormone is increased in polycystic ovaries. The Journal of Clinical Endocrinology & Metabolism, 92(1), 240–245.

[8] Dewailly, D., Andersen, C. Y., Balen, A., Broekmans, F., Dilaver, N., Fanchin, R., Griesinger, G., Kelsey, T. W., La Marca, A., Lambalk, C., Mason, H., Nelson, S. M., Visser, J. A., Wallace, W. H. B., & Anderson, R. A. (2014). The physiology and clinical utility of anti-Müllerian hormone in women. Human Reproduction Update, 20(3), 370–385.

[9] Dumesic, D. A., Oberfield, S. E., Stener-Victorin, E., Marshall, J. C., Laven, J. S. E., & Legro, R. S. (2015). Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and molecular genetics of polycystic ovary syndrome. Endocrine Reviews, 36(5), 487–525.

[10] Rosenfield, R. L., & Ehrmann, D. A. (2016). The pathogenesis of polycystic ovary syndrome (PCOS): The hypothesis of PCOS as functional ovarian hyperandrogenism revisited. Endocrine Reviews, 37(5), 467–520.

[11] Teede, H. J., Tay, C. T., Laven, J. J. E., Dokras, A., Moran, L. J., Piltonen, T. T., Costello, M. F., Boivin, J., Redman, L. M., Boyle, J. A., Norman, R. J., Mousa, A., Joham, A. E., & International PCOS Network. (2023). Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. European Journal of Endocrinology, 189(2), G43–G64.