Correcting Insulin Resistance Using Myo-inositol in Polycystic Ovarian Syndrome

Correção da Resistência à Insulina com o Uso do Mio-inositol na Síndrome do Ovário Policístico
Corregir la Resistencia Ala Insulina con el Uso de Mioinositol para el Síndrome de Ovario Poliquístico

RESUMO
Objetivo: Apresentar os principais aspectos endócrinos e metabólicos da Síndrome do Ovário Policístico, além de discutir alternativas terapêuticas para melhorar os desfechos clínicos e a qualidade de vida das pacientes. **Método:** Revisão de literatura sobre terapias tradicionais e emergentes, incluindo novas opções como Mio-inositol, diante da fisiopatologia da Síndrome do Ovário Policístico. **Resultado:** A Síndrome do Ovário Policístico está associada a diabetes tipo 2 e doença cardiovascular. A Metformina apresenta eficácia limitada na redução de peso e risco cardiovascular, enquanto miméticos da incretina e Mio-inositol, demonstraram benefícios na melhora dos níveis hormonais, desenvolvimento folicular e maturação do oócito. **Conclusão:** O tratamento da Síndrome do Ovário Policístico deve envolver estratégias ampliadas que abordem tanto os aspectos metabólicos quanto os reprodutivos. Novas terapias têm se mostrado promissoras na melhora dos resultados clínicos, reforçando a necessidade de uma abordagem mais abrangente no manejo da condição.
DESCRITORES: Síndrome do Ovário Policístico; Metformina; Inositol 1,4,5-Trifosfato.

ABSTRACT
Objective: To present the main endocrine and metabolic aspects of Polycystic Ovary Syndrome, as well as to discuss therapeutic alternatives to improve clinical outcomes and patients' quality of life. **Method:** Literature review on traditional and emerging therapeutic approaches, including new options such as Myo-inositol, considering the pathophysiology and consequences of Polycystic Ovary Syndrome. **Result:** Polycystic Ovary Syndrome is associated with type 2 diabetes and cardiovascular disease. Metformin has limited effectiveness in reducing weight and cardiovascular risk, while alternative therapies, such as incretin mimetics and Myo-inositol, have shown benefits in improving hormonal levels, follicular development, and oocyte maturation. **Conclusion:** The treatment of Polycystic Ovary Syndrome should involve expanded strategies addressing both metabolic and reproductive aspects. New therapies have shown promise in improving clinical outcomes, reinforcing the need for a more comprehensive approach to managing the condition.
DESCRIPTORS: Polycystic Ovary Syndrome; Metformin; Inositol 1,4,5-Trisphosphate.

RESUMEN
Objetivo: Presentar los principales aspectos endocrinos y metabólicos del Síndrome de Ovario Poliquístico, además de discutir alternativas terapéuticas para mejorar los resultados clínicos y la calidad de vida de las pacientes. **Método:** Revisión de literatura sobre terapias tradicionales y emergentes, incluyendo nuevas opciones como Mio-inositol, a partir de la fisiopatología del Síndrome de Ovario Poliquístico. **Resultado:** El Síndrome de Ovario Poliquístico está asociado con diabetes tipo 2 y enfermedades cardiovasculares. La Metformina presenta eficacia limitada en la reducción de peso y riesgo cardiovascular, mientras que los miméticos de la incretina y el Mio-inositol han demostrado beneficios en la mejora de los niveles hormonales, el desarrollo folicular y la maduración del oocito. **Conclusión:** El tratamiento del Síndrome de Ovario Poliquístico debe involucrar estrategias amplias que aborden tanto los aspectos metabólicos como reproductivos. Las nuevas terapias han mostrado ser prometedoras en la mejora de los resultados clínicos, destacando la necesidad de un enfoque más integral en el manejo de la condición.
DESCRIPTORES: Síndrome de Ovario Poliquístico; Metformina; Inositol 1,4,5-Trifosfato.

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INTRODUCTION

In Polycystic Ovary Syndrome (PCOS), insulin resistance (IR) causes deterioration of the beta cells of the pancreas, causing glucose intolerance and a phenomenon called "compensatory hyperinsulinemia", resulting in a vicious cycle that predisposes the patient to other pathologies.[1] Furthermore, insulin can also stimulate, when induced by luteinizing hormone (LH), the secretion of androgens and decrease the hepatic production of sex hormone binding globulin (SHBG).[1]

The topic presented was selected because most of the available studies on the treatment of Polycystic Ovary Syndrome (PCOS) have little statistical power, great heterogeneity and scarcity, making them inconclusive.[1] Thus, the need to search for new therapeutic options was observed, given the frequency of this pathology in the lives of women of childbearing age.[1]

Metformin has been used in patients with Polycystic Ovary Syndrome (PCOS) for years to stimulate ovulation in women. Furthermore, studies indicate that this medication can also be an effective agent in combating other symptoms associated with hyperandrogenism, including hirsutism and acne.[1] However, as it is not a specific drug for this disorder, being originally used to treat type 2 diabetes mellitus (DM2), other alternatives should be considered, such as Myo-inositol (MI).[1]

Research indicates that Myo-inositol (MI) has not only been shown to be capable of reducing hormonal, metabolic and oxidative changes in patients with Polycystic Ovary Syndrome (PCOS), but also of regulating ovulatory function, attenuating serum androgen and plasma triglyceride concentrations.[2] Thus improving insulin resistance (IR) given its mimetic effect on this hor-

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mone. [2]

Furthermore, considering the adverse gastrointestinal effects caused by Metformin, Myo-inositol (MI) currently appears as a safe therapeutic option as it promotes minimal adverse effects and successful results in metabolic abnormalities, hyperandrogenism and menstrual ovulation. [2]

Recent analyses have investigated the implications of Myo-inositol (MI) and concluded that supplements in this class have improved hormonal and reproductive problems in women with the syndrome, in addition to increasing follicular development and oocyte maturation. [3] From this perspective, it can be seen that, given the specific groups of patients with Polycystic Ovary Syndrome (PCOS), there are those who are not covered by Metformin, such as in cases of intolerance to this drug or in non-obese women, therefore Myo-inositol (MI) becomes a substantial choice. [3]

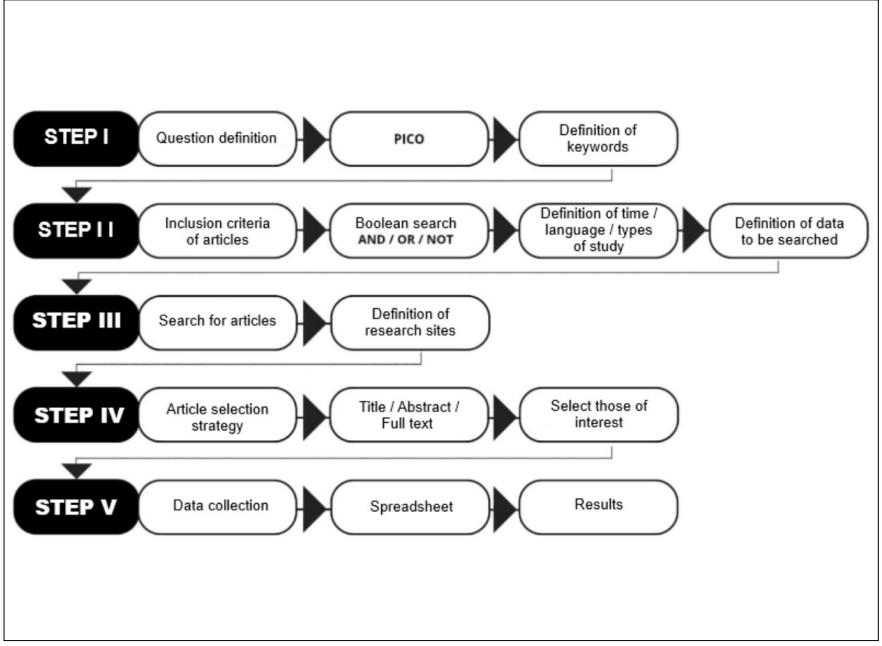
Still in this context, it was observed that the association of Myo-inositol (MI) with Metformin provides lower doses of Metformin in patients who face problems in relation to this drug, and in relation to fasting glycemic levels and insulin, no prominent difference was observed. [4]

In this article, we hope to better understand the mechanisms of insulin resistance (IR) in Polycystic Ovary Syndrome (PCOS), the combined treatment of these two dysfunctions, as well as the therapeutic limitations of the use of Metformin in view of the benefits proposed by the use of Myo-inositol (MI). Thus, we will address, as a prosperous method in pharmacological advancement, the duality of action of Myo-inositol (MI) in improving the symptoms of the syndrome and inducing spontaneous ovulation, a fact that allows us to exclude other pharmaceutical approaches that contain more adverse effects, such as Metformin. [5]

METHOD

The review research was carried out in 5 stages, following the methodological rigor that guaranteed the reproducibility of the information found. The stages are listed in Figure 1.

Figure 1: Research method used and its 5 investigation stages



The stages of this research are described below:

Stage I, which includes defining the research question "What is the effectiveness of correcting insulin resistance with the use of Myo-inositol in relation to the use of Metformin in women with Polycystic Ovary Syndrome" obtained via the PICO method. Once the research question was defined, the key words that would compose the research were then defined.

Stage II consisted of defining the Boolean scheme that would meet the resolution of the research problem, as well as definitions of eligibility of articles, such as [a] Availability in Portuguese, English and Spanish, [b] Direct relationship with the object of study and its guiding question, [c] No conflicts of interest, [d] Having a maximum of 5 years of publication. At this stage, the characteristics of the treatment and the effect of insulin resistance in patients with PCOS were also established; Myo-inositol and its adverse effects compared to Metformin; efficacy of Myo-inositol in insulin resistance; and Myo-inositol as an alternative in the treatment of PCOS. The Boolean scheme used was Polycystic Ovary Syndrome AND Insulin Resistance, Insulin Resistance AND Inositol 1,4,5-Triphosphate, Inositol 1,4,5-Triphosphate OR Metformin, Inositol 1,4,5-Triphosphate AND Polycystic Ovary Syndrome.

Stage III corresponded to the activity of defining search sites, using the PMC, Pubmed and Dynamed portals.

Stage IV It was the phase of selecting the articles found in the portals, in which the analysis was initially based on the title and abstract, and those of interest were separated for analysis, aiming to answer the research problem.

Stage V was the analysis of the results using spreadsheets and other instruments, aiming to generate the results and discussion of the article.

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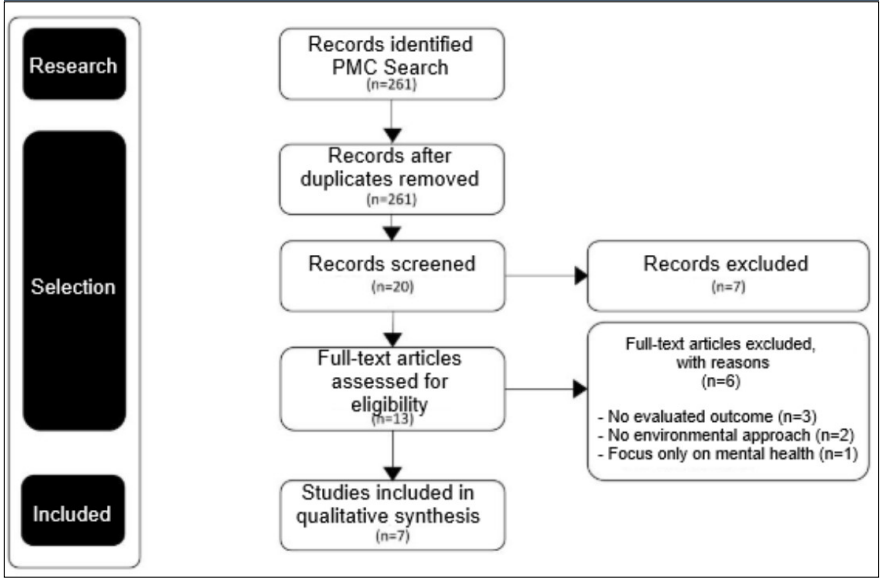
RESULTS

Overview of the works found in the research

A total of 261 articles were found, of which 13 met the research selection criteria to find propositions that addressed the research question of the work, as shown in Figure 2.

Figure 2: Article screening results.



The issue of insulin resistance in PCOS cases

The pathophysiology of PCOS includes IR as one of its main implications and hyperinsulinemia, which together with the LH-inducing effect of theca cells, suppress the hepatic production of SHBG and increase the levels of free androgens, contributing to the interruption of the maturation of ovarian follicles and the consequent polycystic morphological condition of the ovaries. [1-2-3]

In addition to its central role in PCOS, IR has long-term detrimental effects, such as T2DM and cardiovascular disease (CVD), and is prevalent in 60 to 80% of women with the syndrome, being aggravated by obesity. [3-4] Such consequences are also due to another characteristic associated with IR, the high levels of free fatty acids, caused by the high synthesis and mobilization of hepatic and adipose tissues. [5] Despite being endogenous, IR tends to be selective, affecting only muscle, adipose and liver tissue, and even without reaching ovarian tissue, its cells remain sensitive to insulin, causing hyperandrogenemia induced by hyperinsulinemia. [1]

Furthermore, insulin acts on glucose transport proteins (GLUT), sensitizing the uptake of glucose into the intracellular environment for energy production and anabolic processes. [2] When insulin stimulation is inadequate, as in PCOS, glucose transport by GLUT type 4 to skeletal muscle and adipocytes results in IR. [2] According to studies, the large expression of the Syntaxin 4 protein can increase the translocation of GLUT4, amplifying insulin sensitivity, being promising in the creation of new drugs used in IR. [2] In this way, insulin sensitizers act to improve ovulatory function, reduce LH and free testosterone levels, and consequently minimize hyperandrogenism. [2]

In this context, chronic inflammatory processes are commonly found in women with PCOS and are correlated with high androgen levels, IR, atherosclerosis and obesity. [2] Furthermore, patients with PCOS demonstrate excess C-reactive protein, interleukins and tumor necrosis factor α, while SHBG is low, suggesting that inflammatory cytokines may regulate their expression. [2] It is also noted that oxidative stress is elevated in women with PCOS due to the increased production of free radicals related to the impairment of plasma total antioxidant capacity (TAC). [3]

Alternatives for treating PCOS and insulin resistance

Due to impaired glucose tolerance and insulin sensitivity, more than 50% of women with PCOS are overweight or obese. [3] Thus, lifestyle modification - such as changes in diet and physical exercise - is among the first and most important therapeutic options for PCOS, given that weight loss reduces androgen levels, IR, CVD risks, and improves ovulatory function and fertility. [6]

Among the alternatives for treating IR in PCOS are Thiazolidinediones (TZDs), which stimulate the uptake and storage of fatty acids, increase the transcription of insulin-sensitizing genes and increase the secretion of the hormone adiponectin in order to increase insulin susceptibility; however, they are associated with weight gain. [7,4] Also, there is Alpha-Lipoic Acid (ALA), a biological antioxidant used in patients with DM2, which is related to improving peripheral insulin sensitivity and glucagon-like peptide-1 (GLP-1), which has been shown to be reduced in PCOS according to recent research. [7,3]

Therefore, therapy with GLP-1 agonists is advantageous, since they act indirectly on insulin sensitivity through weight reduction. [3] However, when analyzing individuals intolerant

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to Metformin, dipeptidyl peptidase-4 (DPP-4) inhibitors may be a choice due to their performance in β cells (HOMA-B), promoting greater insulin sensitivity. [3] In this context, there is also growing evidence that Statins are beneficial in intervening in the syndrome, as they can significantly reduce IR and hyperandrogenia. [3]

Still regarding pharmacotherapy linked to PCOS, it was found that Vitamin D supplementation is important, since its deficiency is associated with IR, since it acts directly on pancreatic β cells, facilitating the secretion of this hormone. [9] In addition, there is the use of Myo-inositol and Metformin, which have a positive effect on BMI, insulin sensitivity and the menstrual cycle in patients with PCOS. [3]

Metformin treatment and its limitations

Recently, insulin-sensitizing drugs are being recommended as a long-term therapeutic alternative in the treatment of PCOS, given the relevance of hyperinsulinemia in the development of hyperandrogenism and disruption of folliculogenesis. [10] Among these drugs, the most studied has been Metformin, an oral hypoglycemic agent from the biguanide class that has been used for a long time to control DM2, which acts by inhibiting hepatic glucose production and increasing glucose uptake and insulin sensitivity in peripheral tissues. [3] Furthermore, common adverse effects associated with Metformin are nausea, vomiting, diarrhea, abdominal distension and cobalamin deficiency, with these having variable prevalence and severity depending on dose titration or modified release preparations. [3]

In a study comparing Metformin and lifestyle intervention in women with PCOS, a significant reduction in BMI was found in both groups, however, the decrease in androgen levels was only observed with the use of Metformin. [10] However, although this medication presents an improvement in dyslipidemia due to direct effects on the hepatic metabolism of free fatty acids (FFA) or indirect effects on the reduction of hyperinsulinemia, its impact is limited, as such effects are associated with higher doses and the obese female population, in addition to not showing any benefit in terms of total cholesterol levels. [10]

Furthermore, in this regard, Metformin, compared to other drugs, presents lower results in relation to the ovulation rate, however, as it is a cheap drug, it is more accessible. [12] From the same perspective, the analysis also demonstrates a reduction in the risk of Ovarian Hyperstimulation Syndrome (OHSS) due to the aforementioned medication, an adverse effect of infertility treatments. [12]

It is also noted that the effectiveness of Metformin in the treatment of hyperandrogenism in patients with the syndrome is more evident in the group of non-obese patients, restricting, in this case, its effectiveness to a specific group. [12] Therefore, despite its applicability, due to intolerance to Metformin and its various limitations, it is important to also consider other therapeutic possibilities for the treatment of PCOS. [10]

The use of myo-inositol and its benefits

The inositol group is characterized by being chemically identified as a cyclic polyalcohol, comprising nine stereoisomers, being considered part of the B vitamin complex and involved in metabolism. [1] However, MI is the most widely distributed isomer and has a diversified biosynthesis. [1]

That said, the action of MI targets cellular glucose uptake, inhibition of the enzyme adenylate cyclase and reduction of FFA release in adipose tissue. [1] Thus, several benefits are evident in the treatment of PCOS, endocrinally and metabolically, whether used alone or in combination. [1,13]

Intervention with MI is considered safe, as it presents minimal adverse effects when compared to other pharmacological approaches to ovulatory induction. [13] In fact, it is very present in the ovaries and follicular fluid, acting not only on insulin signaling, but also on follicular development by stimulating FSH signaling as a second carrier. [13] Consequently, positive effects were noted in the maintenance and restoration of the normal menstrual cycle, as well as improvement of ovarian function and fertility, with the use of MI, so that it became a requested option to improve both spontaneous ovulation and to induce it. [1,15, 16]

Regarding hyperandrogenism in PCOS, MI management led to the attenuation of mild-moderate hirsutism and reduction of total androgen, FSH, LH and LDL cholesterol levels. [1] Furthermore, the decrease in plasma levels of LH, prolactin, testosterone, insulin and FSH, together with the repair of insulin sensitivity, were evidenced in other studies after 12 weeks of MI administration, and, in 24 weeks, there was a significant increase in SHBG levels. [1,14]

Regarding metabolic abnormalities in the pathophysiology of PCOS in patients treated with MI, there is some controversy. Some studies indicate a significant reduction in BMI while others demonstrate no significant change in BMI. [6,1] Yet another study shows greater efficiency of MI in obese patients, who have high fasting insulin levels. [1]

Similarly, considering the possible gestational complications that may be favored by PCOS, such as a greater susceptibility to developing an increase and severity of GDM, arterial hypertension and pre-eclampsia, studies suggest MI as a treatment to attenuate the risk of GDM in women with family genetics favorable to DM2, who are overweight and obese. [1,18] Finally, it is reported that there was some improvement in glucose homeo-

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stasis resulting from the management of MI in patients already diagnosed with GDM. [1]

DISCUSSION

Among the drug options used in the treatment of PCOS, Metformin and MI are among the most promising, due to the action of both in sensitizing insulin. [3] However, although most studies are favorable to the use of Metformin, systematic reviews on the subject have little statistical knowledge and wide heterogeneity, which makes them inconclusive, thus requiring caution with its use, especially prolonged use or during pregnancy. [10] Furthermore, current studies have shown greater adverse effects in those who use only Metformin compared to patients who use MI or MI combined with Metformin. [1]

In this context, it was shown that both Metformin and MI restore the reduced levels of GLUT1 protein and glucose uptake through the sodium-myoinositol co-transporter-1 (SMIT-1) and the p-AMP-dependent mechanism (AMPK - Adenosine Monophosphate Activated Protein Kinase). [1] Regarding treatment with MI, used for more than 3 months, a significant improvement was observed not only in IR, but also in glycosylated hemoglobin, blood pressure and reduction in cholesterol and triglyceride levels, in relation to therapy with Metformin. [1] Furthermore, the use of Metformin is restricted to patients with kidney or liver disease, hypoxic lung disease or in a state of shock, since lactic acidosis is among the adverse effects resulting from the drug, a potentially fatal condition. [17]

Therefore, MI in combination with Metformin may act synergistically, allowing lower doses of Metformin in patients with intolerance to this drug. [1] According to this hypothesis, the joint action of MI and Metformin presented a significant attenuation of HOMA-IR (IR homeostasis model assessment index), however, regarding fasting glycemic levels and insulin levels, no notable difference was observed. [1] Furthermore, the aforementioned therapeutic combination demonstrated benefits in terms of menstrual cycles, BMI, modified Ferriman Gallwey score, acne, and hormone levels. [1]

CONCLUSION

After analysis and discussion, it is concluded that the option of using MI medication is promising when compared to the most commonly used alternatives currently for the treatment of IR in women with PCOS, such as Metformin. Thus, after reviewing the aforementioned article and the wide range of inconclusive treatments, it is suggested that MI is the best practical alternative for medical qualification, given that it expresses fewer adverse effects and demonstrates good performance in IR. Therefore, the problem raised demands possible future studies, in order to ensure greater veracity regarding the use of MI in the treatment of PCOS, in order to protect women's health.

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