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Prolactin beyond Lactation: Emerging Roles in Reproductive System Function, Infertility, and Hormonal Dysregulation

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ABSTRACT

Traditionally recognized for its role in lactogenesis, prolactin has emerged as a multifunctional hormone intricately involved in the regulation of reproductive physiology. This review explores the broader physiological roles of prolactin in the reproductive system, emphasizing its impact on ovarian and testicular function, gonadotropin release, and neuroendocrine modulation. Elevated or deficient prolactin levels collectively termed prolactin dysregulation have been increasingly implicated in infertility, luteal phase defects, and menstrual irregularities. In males, prolactin modulates Leydig cell function and libido, and its hypersecretion has been associated with hypogonadism and erectile dysfunction. Furthermore, prolactin interacts with other endocrine axes, including thyroid and adrenal systems, highlighting its pleiotropic influence. We also discuss the molecular mechanisms of prolactin signaling through the prolactin receptor (PRLR) and associated pathways, such as JAK-STAT, PI3K-Akt, and MAPK. A comprehensive understanding of prolactin's extramammary roles offers novel insights into diagnostic and therapeutic strategies for reproductive disorders linked to hormonal imbalance.

Keywords: Prolactin, Infertility, Hypogonadism, Hormonal dysregulation, Prolactin receptor

INTRODUCTION

Prolactin, a peptide hormone primarily secreted by lactotroph cells of the anterior pituitary, has been classically associated with the initiation and maintenance of lactation in mammals [1]. However, it is now well established that prolactin exerts diverse biological effects far beyond lactogenesis [1]. More than 300 functions of prolactin have been documented, reflecting its action in various physiological systems, including immunity, metabolism, and osmoregulation [2]. Among its emerging functions, the role of prolactin in reproductive health is particularly significant, affecting both female and male reproductive physiology [1]. The synthesis and secretion of prolactin are regulated by multiple factors, with dopamine acting as the principal inhibitory regulator via D2 dopamine receptors [3]. Estrogens are strong stimulators of prolactin production, particularly during pregnancy [1]. Prolactin exerts its biological effects through the prolactin receptor (PRLR), a member of the cytokine receptor family that activates multiple downstream pathways, including the Janus kinase-signal transducer and activator of transcription (JAK-STAT), mitogen-activated protein kinase (MAPK), and phosphatidylinositol 3-kinase (PI3K) signaling cascades [4]. Given its widespread expression and receptor distribution, prolactin plays critical roles in reproductive processes such as ovulation, implantation, corpus luteum function, and spermatogenesis [5]. Dysregulation of prolactin levels—whether hyperprolactinemia or hypoprolactinemia—can disrupt the delicate hormonal milieu necessary for fertility and reproductive health [6]. Hyperprolactinemia is a leading cause of anovulation and infertility in women and is also implicated in erectile dysfunction and testosterone suppression in men [7].

This review critically examines the physiological and pathological roles of prolactin in the reproductive system, the mechanisms by which it modulates hormonal balance, and its implications in infertility and endocrine disorders. Special attention is paid to the diagnostic and therapeutic relevance of prolactin in reproductive endocrinology.

Physiological Functions of Prolactin in Reproduction

Prolactin plays critical and multifaceted roles in the regulation of reproductive processes in both females and males [1]. It exerts these actions through binding to the prolactin receptor (PRLR), which is widely distributed in reproductive tissues including the hypothalamus, pituitary, gonads, and uterus [4]. In females, prolactin contributes to ovarian folliculogenesis, corpus luteum function, and preparation of the endometrium for implantation [5]. In males, it regulates testicular function and supports reproductive hormone signaling [8].

Female Reproduction:

Prolactin is essential in preparing the female reproductive system for conception and gestation [5]. It acts on ovarian granulosa and theca cells to regulate follicular maturation, steroidogenesis, and ovulation [5]. In the corpus luteum, prolactin enhances the production of progesterone, a hormone critical for the maintenance of early pregnancy [9]. Moreover, prolactin acts synergistically with luteinizing hormone (LH) to maintain the functional integrity of luteal cells [9]. It also modulates uterine receptivity by influencing endometrial differentiation and vascular remodeling during the window of implantation [10] In addition, prolactin has been shown to influence mammary gland development during pregnancy and contribute to the neuroendocrine control of maternal behavior [11]. Extra-pituitary prolactin production within the uterus, decidua, and immune cells further supports implantation and fetal tolerance, revealing a complex network of paracrine and autocrine actions within reproductive tissues [12].

Male Reproduction:

Though historically underappreciated, prolactin plays a supportive role in male reproductive physiology [8]. It enhances the sensitivity of Leydig cells to luteinizing hormone, promoting testosterone production [13]. Prolactin receptors are expressed in testicular tissues, including Sertoli and Leydig cells, indicating its role in spermatogenesis and testicular development [14]. Low circulating levels of prolactin appear necessary for maintaining optimal sperm production and libido. Animal studies demonstrate that prolactin modulates the secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus, thereby affecting the entire hypothalamic-pituitary-gonadal (HPG) axis [15].

Prolactin Dysregulation and Infertility

Prolactin dysregulation, particularly hyperprolactinemia, is a common endocrine disorder that can severely impair reproductive function [7]. Abnormally elevated levels of prolactin are associated with a broad range of clinical manifestations including infertility, sexual dysfunction, and menstrual irregularities [1]. Although hypoprolactinemia is rarer, it also presents notable reproductive challenges.

Hyperprolactinemia:

The most common causes of hyperprolactinemia include prolactin-secreting pituitary adenomas (microprolactinomas or macroprolactinomas), hypothyroidism, polycystic ovary syndrome (PCOS), chronic stress, and the use of dopamine antagonist medications such as antipsychotics and antidepressants [16]. In women, prolactin inhibits the pulsatile secretion of GnRH, leading to decreased LH and FSH release [17]. This disrupts the menstrual cycle and results in anovulation, amenorrhea, galactorrhea, and infertility. In men, hyperprolactinemia suppresses gonadotropin release and reduces testosterone synthesis, resulting in hypogonadism [18]. Clinical symptoms may include reduced libido, erectile dysfunction, gynecomastia, and infertility. Prolonged elevation of prolactin can also lead to decreased testicular volume and impaired spermatogenesis [19].

Hypoprolactinemia:

While less frequently reported, low levels of prolactin may also impair reproductive function, particularly in females. Hypoprolactinemia can be caused by pituitary insufficiency, Sheehan's syndrome, or pharmacologic suppression with dopamine agonists [20]. It has been associated with luteal phase defects, reduced progesterone secretion, and implantation failure. Women with low prolactin levels may experience shortened luteal phases and inadequate endometrial preparation for embryo implantation [21]. In men, the implications of hypoprolactinemia remain underexplored, but limited data suggest it may affect testicular function, possibly through reduced GnRH activity. Both hyper- and hypoprolactinemia can therefore exert detrimental effects on fertility, underscoring the importance of maintaining prolactin levels within a physiological range.

Mechanisms of Action and Signal Transduction

The physiological and pathological actions of prolactin are mediated through its receptor, a member of the class I cytokine receptor superfamily [1]. The prolactin receptor exists in multiple isoforms (long, intermediate, and short), with the long isoform being the most functionally active in reproductive tissues [22]. Ligand binding induces

receptor dimerization and activation of downstream signaling cascades, which vary depending on the tissue and isoform expressed [22].

JAK-STAT Pathway:

Upon prolactin binding, the long form of PRLR activates Janus kinase 2 (JAK2), which in turn phosphorylates signal transducers and activators of transcription (STAT) proteins, predominantly STAT5 [3]. This pathway regulates the transcription of genes involved in cell proliferation, differentiation, survival, and hormone synthesis. In reproductive tissues, JAK-STAT signaling supports follicle maturation, luteal function, and endometrial receptivity.

PI3K-Akt Pathway:

The phosphoinositide 3-kinase (PI3K)-Akt pathway is another critical signaling route activated by prolactin [24]. It promotes cell survival, inhibits apoptosis, and enhances protein synthesis [24]. In the ovary, activation of this pathway by prolactin protects luteal cells from apoptosis and sustains progesterone production [25]. In the testes, the pathway may support Leydig cell steroidogenesis and maintain spermatogenic activity.

MAPK/ERK Pathway:

The mitogen-activated protein kinase (MAPK) pathway is involved in cellular proliferation and differentiation [26]. In endometrial and ovarian tissues, prolactin-induced MAPK signaling contributes to tissue remodeling, follicular development, and decidualization [27]. This pathway also mediates the crosstalk between prolactin and other sex steroid hormones, particularly estrogen and progesterone [28].

Cross-Talk with Other Hormonal Pathways:

Prolactin signaling does not function in isolation. It interacts with estrogen, progesterone, insulin, and glucocorticoid pathways, amplifying or modulating their downstream effects. For instance, prolactin upregulates estrogen receptor expression in mammary and uterine tissues, while estrogen enhances PRLR transcription, forming a regulatory feedback loop [29]. Additionally, prolactin modulates insulin sensitivity and adrenal steroid output, revealing a broader systemic role in endocrine balance [1] These signal transduction pathways collectively enable prolactin to exert precise control over reproductive physiology. Aberrations in receptor expression or signaling fidelity contribute to disease states, including infertility, PCOS, and hormone-dependent tumors.

Prolactin and Hormonal Crosstalk

Prolactin functions within a broader hormonal milieu, engaging in complex cross-talk with other endocrine systems that regulate reproduction, metabolism, and stress responses [1]. This inter-hormonal interaction significantly influences reproductive function, either enhancing or inhibiting other hormonal signals depending on physiological context.

Thyroid Axis Interaction:

One of the most clinically significant interactions is between prolactin and the hypothalamic-pituitary-thyroid (HPT) axis [30]. Thyrotropin-releasing hormone (TRH), produced by the hypothalamus, stimulates the anterior pituitary to release both thyroid-stimulating hormone (TSH) and prolactin [30]. In hypothyroidism, elevated TRH levels may lead to secondary hyperprolactinemia, contributing to menstrual irregularities and infertility [31]. Conversely, correcting hypothyroidism often results in normalization of prolactin levels and restoration of reproductive function [32].

Adrenal Axis and Glucocorticoids:

Prolactin also modulates adrenal function. It stimulates adrenal androgen production and enhances adrenal responsiveness to adrenocorticotropic hormone (ACTH) [33]. In women, this may contribute to hyperandrogenism, which is particularly relevant in conditions like polycystic ovary syndrome (PCOS). In men, the effect on adrenal androgens is less clear but may influence libido and mood. Moreover, prolactin modulates glucocorticoid receptors in immune and reproductive tissues, potentially influencing the stress response and its reproductive consequences [34].

Gonadal Hormones and Estrogen Feedback:

Prolactin and estrogen engage in a bidirectional regulatory relationship. Estrogen stimulates prolactin gene transcription and promotes the proliferation of pituitary lactotrophs, especially during pregnancy [18]. In turn, prolactin enhances estrogen receptor expression in target tissues such as the uterus and mammary glands [1]. Disruption of this balance, as seen in estrogen-dominant conditions, may contribute to pathologies such as fibroids, endometriosis, and hormone-sensitive tumors [35]. In males, elevated prolactin can inhibit the hypothalamic release of GnRH, reducing testosterone levels and affecting spermatogenesis [19].

Insulin and Metabolic Hormones:

Emerging evidence suggests a role for prolactin in metabolic regulation. Physiological levels of prolactin are associated with enhanced insulin sensitivity, whereas chronic hyperprolactinemia may contribute to insulin resistance and metabolic syndrome [36]. This is of particular relevance in reproductive endocrinology, as insulin

resistance is a common feature in both PCOS and male infertility syndromes [37,38]. The interaction between prolactin and metabolic hormones reinforces the concept that reproductive and metabolic health are intricately linked.

Therapeutic Implications

Given prolactin's pivotal role in reproductive and endocrine regulation, targeted therapeutic interventions have been developed to manage prolactin-related disorders, particularly hyperprolactinemia. The most widely used pharmacologic agents are dopamine agonists, which inhibit prolactin release through D2 receptor stimulation in the anterior pituitary [39].

Dopamine Agonists:

Bromocriptine and cabergoline are the mainstays of treatment for hyperprolactinemia [40]. Cabergoline, owing to its longer half-life and superior tolerability, is preferred in many clinical settings. These agents not only normalize serum prolactin levels but also restore ovulatory cycles in women and improve testosterone levels and libido in men. In patients with prolactinomas, dopamine agonists can induce tumor shrinkage and reduce compressive symptoms such as visual disturbances [41].

Addressing Underlying Causes:

In cases where hyperprolactinemia is secondary to hypothyroidism, antipsychotic use, or stress, addressing the primary condition may eliminate the need for direct prolactin-lowering agents. For example, levothyroxine therapy in hypothyroid patients often restores normal prolactin levels and reproductive function without further intervention [42].

Prolactin Monitoring in Fertility Clinics:

Measuring serum prolactin is a routine component of infertility assessments. In women with amenorrhea or irregular cycles, and in men with sexual dysfunction or low testosterone, prolactin testing can guide clinical management. Timely diagnosis allows for early intervention, which can prevent the long-term reproductive and metabolic sequelae of prolactin dysregulation.

Emerging Therapies:

Research is ongoing into prolactin receptor antagonists, which may provide more selective modulation of prolactin signaling without suppressing systemic prolactin levels. These therapies hold promise for patients who are resistant to dopamine agonists or who experience adverse effects. Additionally, lifestyle interventions such as stress reduction, weight management, and exercise may indirectly modulate prolactin levels through their effects on dopamine and cortisol [1].

CONCLUSION

Prolactin has evolved from its classical role in lactation to emerge as a central regulator of reproductive physiology and endocrine homeostasis. Its effects span ovulation, implantation, corpus luteum maintenance, spermatogenesis, and sexual function. Prolactin also interacts with major hormonal systems, including thyroid, adrenal, and metabolic axes, amplifying its systemic significance. Both hyperprolactinemia and hypoprolactinemia are associated with infertility, highlighting the importance of maintaining prolactin within an optimal physiological range. Advances in pharmacologic therapy—particularly dopamine agonists—have dramatically improved outcomes for patients with prolactin-related disorders. However, diagnostic and therapeutic challenges remain, especially in subtle cases where prolactin imbalance presents with nonspecific symptoms or overlaps with other endocrine pathologies. As research continues to uncover the diverse roles of prolactin, future directions will likely include the development of receptor-specific therapies, deeper understanding of tissue-specific signaling, and integration of prolactin evaluation into personalized reproductive medicine. Recognizing prolactin's contributions beyond lactation will enhance our capacity to diagnose, treat, and prevent a wide array of reproductive and hormonal disorders.

REFERENCES

1. Al-Chalabi M, Bass AN, Alsalman I. Physiology, prolactin. StatPearls – NCBI Bookshelf. 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK507829/
2. Kolnikaj TS, Muşat M, Salehidoost R, Korbonits M. Pharmacological causes of hyperprolactinemia. Endotext – NCBI Bookshelf. 2024. Available from: https://www.ncbi.nlm.nih.gov/books/NBK599196/
3. Mizutani K, Takai Y. Prolactin. In: Elsevier eBooks. 2018. Available from: doi:10.1016/B978-0-12-801238-3.98018-8
4. Gorvin CM. The prolactin receptor: Diverse and emerging roles in pathophysiology. Journal of Clinical & Translational Endocrinology. 2015;2(3):85–91. doi:10.1016/j.jcte.2015.05.001
5. Szukiewicz D. Current insights in prolactin signaling and ovulatory function. International Journal of Molecular Sciences. 2024;25(4):1976. doi:10.3390/ijms25041976

6. Iancu ME, Albu AI, Albu DN. Prolactin relationship with fertility and in vitro fertilization outcomes—a review of the literature. Pharmaceuticals. 2023;16(1):122. doi:10.3390/ph16010122
7. Haidenberg-David F, Sidauy-Adissi J, Moscona-Nissan A, Jonguitud-Zumaya E, Fugarolas-Morinelli M, Martinez-Mendoza F, et al. Overview of hyperprolactinemia: General approach and reproductive health implications. Archives of Medical Research. 2024;55(8):103102. doi:10.1016/j.arcmed.2024.103102
8. Gill-Sharma MK. Prolactin and male fertility: The long and short feedback regulation. International Journal of Endocrinology. 2009;2009:687259. doi:10.1155/2009/687259
9. Magon N, Kumar P. Hormones in pregnancy. Nigerian Medical Journal. 2012;53(4):179. doi:10.4103/0300-1652.107549
10. Zhang S, Lin H, Kong S, Wang S, Wang H, Wang H, et al. Physiological and molecular determinants of embryo implantation. Molecular Aspects of Medicine. 2013;34(5):939–80. doi:10.1016/j.mam.2012.12.011
11. Grattan DR, Steyn FJ, Kokay IC, Anderson GM, Bunn SJ. Pregnancy-induced adaptation in the neuroendocrine control of prolactin secretion. Journal of Neuroendocrinology. 2008;20(4):497–507. doi:10.1111/j.1365-2826.2008.01661.x
12. Flores-Espinosa P, Méndez I, Irles C, Olmos-Ortiz A, Helguera-Repetto C, Mancilla-Herrera I, et al. Immunomodulatory role of decidual prolactin on the human fetal membranes and placenta. Frontiers in Immunology. 2023;14. doi:10.3389/fimmu.2023.1212736
13. Williams VL, DeGuzman A, Dang H, Kawaminami M, Ho TWC, Carter DG, et al. Common and specific effects of the two major forms of prolactin in the rat testis. AJP Endocrinology and Metabolism. 2007;293(6):E1795–803. doi:10.1152/ajpendo.00541.2007
14. O'Donnell L, Stanton P, De Kretser DM. Endocrinology of the male reproductive system and spermatogenesis. Endotext – NCBI Bookshelf. 2017. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279031/
15. Acevedo-Rodriguez A, Kauffman AS, Cherrington BD, Borges CS, Roepke TA, Laconi M. Emerging insights into hypothalamic-pituitary-gonadal axis regulation and interaction with stress signalling. Journal of Neuroendocrinology. 2018;30(10). doi:10.1111/jne.12590
16. Glezer A, Bronstein MD. Hyperprolactinemia. Endotext – NCBI Bookshelf. 2022. Available from: https://www.ncbi.nlm.nih.gov/books/NBK278984/
17. Kaiser UB. Hyperprolactinemia and infertility: New insights. Journal of Clinical Investigation. 2012;122(10):3467–8. doi:10.1172/JCI64455
18. Thapa S, Bhusal K. Hyperprolactinemia. StatPearls – NCBI Bookshelf. 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK537331/
19. Dabbous Z, Atkin SL. Hyperprolactinaemia in male infertility: Clinical case scenarios. Arab Journal of Urology. 2017;16(1):44–52. doi:10.1016/j.aju.2017.10.002
20. Uzun I, Karaca Z, Hacioğlu A, Unluhizarci K, Kelestimur F. The diagnosis and prevalence of hypoprolactinemia in patients with panhypopituitarism and the effects on depression and sexual functions. Pituitary. 2024;27(3):277–86. doi:10.1007/s11102-024-01393-0
21. Terranova P. Luteal phase defects. In: Elsevier eBooks. 2014. Available from: doi:10.1016/B978-0-12-801238-3.05125-4
22. Binart N, Bachelot A, Bouilly J. Impact of prolactin receptor isoforms on reproduction. Trends in Endocrinology and Metabolism. 2010;21(6):362–8. doi:10.1016/j.tem.2010.01.008
23. Guzeloglu-Kayisli O, Kayisli U, Taylor H. The role of growth factors and cytokines during implantation: Endocrine and paracrine interactions. Seminars in Reproductive Medicine. 2009;27(1):62–79. doi:10.1055/s-0028-1108011
24. Neri LM, Borgatti P, Capitani S, Martelli AM. The nuclear phosphoinositide 3-kinase/AKT pathway: A new second messenger system. Biochimica et Biophysica Acta – Molecular and Cell Biology of Lipids. 2002;1584(2–3):73–80. doi:10.1016/S1388-1981(02)00300-1
25. Giaccari C, Antonouli S, Anifandis G, Cecconi S, Di Nisio V. An update on physiopathological roles of AKT in the reproductive mammalian ovary. Life. 2024;14(6):722. doi:10.3390/life14060722
26. Munshi A, Ramesh R. Mitogen-activated protein kinases and their role in radiation response. Genes & Cancer. 2013;4(9–10):401–8. doi:10.1177/1947601913485414
27. Makieva S, Giacomini E, Ottolina J, Sanchez AM, Papaleo E, Viganò P. Inside the endometrial cell signaling subway: Mind the gap(s). International Journal of Molecular Sciences. 2018;19(9):2477. doi:10.3390/ijms19092477

28. Yamakawa K, Arita J. Cross-talk between the estrogen receptor-, protein kinase A-, and mitogen-activated protein kinase-mediated signaling pathways in the regulation of lactotroph proliferation in primary culture. The Journal of Steroid Biochemistry and Molecular Biology. 2004;88(2):123–30. doi:10.1016/j.jsbmb.2003.11.003
29. Kavarthapu R, Dufau ML. Prolactin receptor gene transcriptional control, regulatory modalities relevant to breast cancer resistance and invasiveness. Frontiers in Endocrinology. 2022;13. doi:10.3389/fendo.2022.949396
30. Shahid MA, Ashraf MA, Sharma S. Physiology, thyroid hormone. StatPearls – NCBI Bookshelf. 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK500006/
31. Bahar A, Akha O, Kashi Z, Vesgari Z. Hyperprolactinemia in association with subclinical hypothyroidism. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3766941/
32. Goswami B, Patel S, Chatterjee M, Koner B, Saxena A. Correlation of prolactin and thyroid hormone concentration with menstrual patterns in infertile women. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3719326/
33. Lalli E, Figueiredo BC. Prolactin as an adrenocorticotropic hormone: Prolactin signalling is a conserved key regulator of sexually dimorphic adrenal gland function in health and disease. BioEssays. 2022;44(10). doi:10.1002/bies.202200109
34. Rasmi Y, Jalali L, Khalid S, Shokati A, Tyagi P, Ozturk A, et al. The effects of prolactin on the immune system, its relationship with the severity of COVID-19, and its potential immunomodulatory therapeutic effect. Cytokine. 2023;169:156253. doi:10.1016/j.cyto.2023.156253
35. Borahay MA, Asoglu MR, Mas A, Adam S, Kilic GS, Al-Hendy A. Estrogen receptors and signaling in fibroids: Role in pathobiology and therapeutic implications. Reproductive Sciences. 2016;24(9):1235–44. doi:10.1177/1933719116678686
36. Yang H, Lin J, Li H, Liu Z, Chen X, Chen Q. Prolactin is associated with insulin resistance and beta-cell dysfunction in infertile women with polycystic ovary syndrome. Frontiers in Endocrinology. 2021;12. doi:10.3389/fendo.2021.571229
37. Amisi CA. Markers of insulin resistance in polycystic ovary syndrome women: An update. World Journal of Diabetes. 2022;13(3):129–49. doi:10.4239/wjd.v13.i3.129
38. Mansour R, El-Faissal Y, Kamel A, Kamal O, Aboulserour G, Aboulghar M, et al. Increased insulin resistance in men with unexplained infertility. Reproductive BioMedicine Online. 2017;35(5):571–5. doi:10.1016/j.rbmo.2017.08.020
39. Choi J, Horner KA. Dopamine agonists. StatPearls – NCBI Bookshelf. 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK551686/
40. Nunes VDS, Dib RE, Boguszewski C, Nogueira C. Cabergoline versus bromocriptine in the treatment of hyperprolactinemia: A systematic review of randomized controlled trials and meta-analysis. DARE: Quality-assessed Reviews – NCBI Bookshelf. 2011. Available from: https://www.ncbi.nlm.nih.gov/books/NBK85990/
41. Drummond JB, Molitch ME, Korbonits M. Prolactinoma management. Endotext – NCBI Bookshelf. 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279174/
42. Aziz K, Shahbaz A, Umair M, Sharifzadeh M, Sachmechi I. Hyperprolactinemia with galactorrhea due to subclinical hypothyroidism: A case report and review of literature. Cureus. 2018. doi:10.7759/cureus.2723

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