Polycystic ovarian syndrome (PCOS) is the most common neuro-endocrine disorder of women of reproductive age, characterized by excess androgen, ovulatory dysfunction and polycystic ovaries. PCOS is also linked with several metabolic dysfunctions including type 2 diabetes mellitus, obesity, cardiovascular disorders and psychological co-morbidities, viz., anxiety, depression and mood disorders. Although the prevalence of and the discomfort caused by PCOS is very high, very little is known about its clear patho-etiologie. Thereby, the current study was aimed at understanding the status of various regulatory molecules to decipher the neuro-endocrine pathology of PCOS, using rodent model. Letrozole, an aromatase inhibitor, was used for PCOS induction. Results of the present study demonstrate that letrozole is able to mimic reproductive, metabolic and neuro-endocrine characteristics similar to the human PCOS condition. Studies suggest that increased GnRH pulsatility and concurrently elevated LH/FSH ratio may underpin the pathology. Moreover, the pulsatile release of GnRH/LH results from the coordinated actions of steroids, neuro-peptides and neurotransmitters in discrete areas of brain. In this context, the current study clearly demonstrates the involvement of neuropeptides kisspeptin, Neurokinin B, Dynorpin and RFRP3 in steroid-mediated feedback regulation that is hampered in PCOS condition. Furthermore, the current study, for the first time, depicts that along with ovary and adrenal, steroidogenesis is also altered in several areas of the brain, suggesting a putative role of local steroids (neurosteroids) in PCOS pathology. We also aptly demonstrate that increased adrenal androgen, a key feature of PCOS, is due to increased responsiveness of adrenal gland that results into activation of a signalling cascade, leading to overproduction of androgens as well as corticosterone from PCOS adrenals. Further, a neurotransmitter evaluation revealed that the GnRH-stimulatory neurotransmitters are elevated whereas GnRH-inhibitory neurotransmitters are decreased in PCOS condition, which is clearly the cause of increased GnRH/LH release. Additionally, our results indicate that the disease causes a pro-inflamed state of endocrine and neuronal tissues that is linked with altered neuronal signalling and behaviour modulations. Present study concludes that PCOS is associated with an altered brain microenvironment, resulting into neuro-endocrine and psychological complications. This is the first study which holistically demonstrates that PCOS is a reproductive disease having clear associations with all other organ systems, thus addressing the different targets which can be explored for a detailed understanding.
Abstract: Objective: To establish rat models for the study of polycystic ovarian syndrome due to Kidney Deficiency-Blood Stagnation by using letrozole and endotoxin (LPS), and evaluated them comprehensively. Methods: Sixty SD female rats aged six weeks were divided into two groups, including experimental group of fifty rats and control group of ten rats. Rats were administered letrozole combined with LPS. The ovary was red and the surface was smooth. Conclusion: Letrozole and endotoxin (LPS)-induced model of polycystic ovarian syndrome due to Kidney Deficiency-Blood Stagnation in rats is successful. Keywords: PCOS, Letrozole, Endotoxin, Animal model References: [1] Wang Wenlai, Liu Meijie, Zhao Hongxia, Wang Shaojun, Wang Zhen, Zhang Fangzhen. Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in women of reproductive age. PCOS is a heterogeneous clinical. To examine the serum visfatin, adiponectin and leptin levels in patients with polycystic ovarian syndrome (PCOS), and to explore the basis of the pathogenesis of PCOS. A PCOS group (n=73) and a healthy control group (n=75) were included in the study, which were matched in age and body mass index (BMI). Polycystic ovarian syndrome (PCOS), recently referred also as hyperandrogenic anovulation, is a chronic anovulation syndrome associated with androgen excess. The diagnosis of PCOS generally requires any two of the following three criteria for the syndrome: 1- Insulin resistance and obesity are also common components of the syndrome. 1,2 PCOS is one of the most prevalent endocrine disorders in women, affecting an estimated 4% to 10% of females of reproductive age. 1,2 Anovulation associated with PCOS is a leading cause of infertility; however, complications of PCOS extend beyond fertility concerns. Polycystic Ovary Syndrome (PCOS), Nerve Growth Factor (NGF), Sympathetic Nervous System (SNS), Interleukin 1α, 1β, 17A, TNFα. *Corresponding author. In fact NGF is involved in the modulation of the neuro-endocrine-immune (NEI) system and has an important role in maintaining homeostasis of these systems. NGF is a strong marker for sympathetic nerve activity. 460. Evidence from studies on women with PCO and on an experimental rat PCO model suggests that the sympathetic regulatory drive to the ovary may be unbalanced. Most reports support the theory that increased sympathetic activity contributes to the development and maintenance of PCOS.