Commentary

Immunology Advancements

Abdhul Rehman Asif*

Department of Central Laboratory, Georg-August-University, Gottingen, Germany

ABSTRACT

Van Wyk Grumbach syndrome (VWG) is a rare syndrome characterized by precocious puberty, hypothyroidism, delayed skeletal maturation and bilateral ovarian cysts. Majority of these features are with thyroxine supplementation including ovarian cyst. This case report illustrates a case of 6 yr old girl who presented with vaginal bleeding and pain abdomen. She had hypothyroidism. The ultrasound abdomen reported multicystic ovarian cysts in bilateral ovaries. Timely done thyroid function tests and prompt diagnosis of VWG syndrome alleviated the need of a major surgery. **Keywords:** Van Wyk Grumbach syndrome; Microglia; Alzheimer's disease

COMMENTARY

Unexplained recurrent implantation failure has become a public health problem especially in the context of a high cost, demand for assisted reproductive therapy and multiple therapy failures [1,2]. It is attributed to the failure of immunological tolerance between the transferred embryo and the endometrium during assisted reproductive therapy cycles. Cytokines are surrogate mediators of immunological tolerance mechanisms [3]. Since the synergistic interactions between individual cytokines are dynamic, perturbations in the cytokine crosstalk during embryo implantation is considered a major etiology for unexplained recurrent implantation failures. Genome Wide Association Studies suggests that most cytokines initiate their actions through receptor interactions that activate the JAK-STAT signaling pathways. An aberration in the JAK-STAT signaling pathways has as well been shown to cause perturbations in cytokine crosstalk those results in diseases. We therefore propose the JAK-STAT signaling pathway a potential therapeutic target for unexplained recurrent implantation failures [4-6]. Currently, many studies have recorded potential therapies in IL-6/JAK/ STAT 3 pathway to treat many diseases but limited attention has been paid to unexplained recurrent implantation failures [7].

The examination of morphological features of gestational products accounts as a core diagnostic process, especially for the distinction of complete hydatidiform mole (CM) from partial mole (PM) cases. Nevertheless, subjective evaluation of the based-criteria might occur with substantial inter-observer variability [8-11]. Objective: To assess the utility of p57

immunohistochemical expression in distinguishing the CM from PM. Materials and methods: This study was a cross-sectional analysis conducted entirely among 34 patients, including those cases with molar pregnancies and product of conception after uterine evacuation [12,13]. All cases were recruited between January-July 2018 into a Gynecology and Obstetrics Department. Together with the histomorphological assessment, we performed p57KIP2 immunohisto-chemical staining in all the specimens. Results: the histological diagnostic categories were as follows: complete mole (n=12), partial mole (n=8), and placenta and nonmolar product of conception group (n=14), based on previously reported criteria and IHC [14-19]. Accordingly, morphological CM diagnosis was consistent with p57 IHC, displaying cytotrophoblast and villous stromal cells with a negative stain in 9 out of 12 observed CM specimens. However, one case had aberrant p57 expression and two others were morphologically concerned, having a mild degree of villous edema and greater scalloping morphology. The later cases confirmed as PMs based on IHC p57 positive staining [20]. For PM, almost all cases histologically had consistent IHC findings with positive p57 immunostaining in cytotrophoblast and villous stromal cells. Two cases were lacked p57 marker positivity and considered as CM albeit with a milder degree of trophoblast hyperplasia [21]. All products of conception and hydropic abortion showed fewer villi formation and positive p57KIP2 immunoreactivity [22,23]. Conclusions: This study further confirms the importance of p57KIP2 immuno-staining as an ancillary test with the traditional histopathological criteria to distinguish complete mole from other mimic cases.

Correspondence to: Abdul Rahman Asif, Department of Central Laboratory, Georg-August-University, Gottingen, Germany, E-mail: abdhulasif@gmail.com

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Endorphins are endogenous morphine, neuropeptides, produced in the pituitary gland in response to stress and pain. There are three types of endorphins beta-endorphins, enkephalins, and dynorphins binds to mu,kappa, and delta receptors situated on nervous system and immune cells. Cancer is a major threat to mankind killing millions of people around the world annually. There has been recent advancements in the field of surgery, chemotherapy, and radiotherapy, still the prognosis of cancer patients not improved much with increasing morbidity. We cannot kill cancer cells without killing normal cells. Cancer cells and normal cells work alike. The aim of the review was to determine the anticancer activities of betaendorphins. Materials and methods-; Articles regarding about endorphins and its therapeutic application in cancer were searched on pubmed and google scholar. This review includes studies, reviews, clinical trials and key findings of my research were included in the manuscript. Results-; Beta- endorphin is an abundant endorphin, potent than morphine, synthesized and secreted in the anterior pituitary gland, it is a precursor of POMC (proopiomelanocortin). It has got various mechanisms of action such as analgesic activity, anti-inflammatory activity, immune stimulatory activity, stress buster activity, and euphoric activity. Conclusion-; Beta endorphin is an abundant endogenous morphine used for natural holistic preventive, therapeutic, promotive, and palliative treatment of cancer without adverse effects and inexpensive.

The pathogenesis of inflammatory complication after chest trauma and pulmonary injury is incompletely understood. Injury can trigger a systemic inflammatory response, which leads to pre-activation of neutrophils in blood. The aim of this study was to determin the specific expression profiles of neutrophil receptors in relation to the systemic inflammatory response after chest trauma. Blood samples from fifty patients with isolated thoracic injury were analysed for changes in the neutrophil phenotype within 3, 6 and 24 hours after injury. Study patients were assessed for any inflammatory complications during the first 24 hours. L-Selectin expression remained decreased until 24 hrs while CXCR1, CXCR2 and C5aR levels gradually increased. Expression of Fcl3RII and expression of the active form were lower in trauma patients; no patients developed ARDS [24]. Thoracic trauma leads to activation of the circulating neutrophils which is transient accompanied by mobilization of young neutrophils into the circulation which leads to systemic inflammatory reactions which need a second stimulus to cause inflammatory complications like ARDS.

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