Ovarian cancer has high mortality and delayed diagnosis. Inflammation is considered as a risk for ovarian carcinoma, and it contributes to all grades of tumour progression. Inflammation involved in the development of cancer cell and can be caused by an increase in the formation of pro-inflammatory cytokines. This research was conducted to assess serum Interleukin, Gonadotropins hormone and Steroid hormone levels in ovarian cancer patients and to assess their impact in disease action. The present study is composed of 85 women (mean age = 62.03±12.4 yrs) with clinically and pathologically confirmed ovarian cancer and 65 healthy women as a control group (mean age = 61±12.1 yrs). The measured biochemical parameters included: the level of serum gonadotropins (LH, luteinizing hormone; FSH, follicle-stimulating hormone), steroid hormones (estrogen, progesterone, prolactin, testosterone) and Interleukin (IL-4, IL-6, and IL-8). The results demonstrated significantly high values of steroid hormones (p<0.01, p<0.05, p<0.002, p<0.01 (estrogen, progesterone, prolactin, testosterone) and significantly high (p<0.001, p<0.002, p<0.01) values in (IL-4, IL-6, and IL-8) in ovarian cancer in comparison with control group. There were also significantly high (p<0.0001, p<0.05 values of sera LH and FSH in women ovarian cancer in comparison with the control group. p<0.01, p<0.001) respectively in ovarian cancer in comparison with the control group. An elevation of serum steroid hormone, gonadotropins, and Interleukin levels in cases of ovarian carcinoma in our study is important as a marker of the disease.
tumours, particularly distant recurrence. However, for patients at a proceed grade and with a metastatic growth (Dubosq et al., 2012). Gametogenesis which including sexual steroid hormones such as usage of oral contraceptive drugs are correlated with the known risk factors in ovarian carcinomas. While the usage of hormonal replacement treatment. In contrast, increases risk of advanced ovarian carcinomas attributes to nulliparity and the utilization of hormonal replacement treatment. It has been confirmed that receptors of steroid hormones are expressed as cancerous tumours as well as ovarian carcinomas in its various microanatomical subtypes. Even though confirmations propose that the exposure to steroid hormones could be associated to the presence and advancement of ovarian carcinomas, current information regarding steroid hormones receptors till now is not enough to evaluate if the expression of steroid hormones receptors is linked to histological or molecular subtypes and grade of their malignancy or if it affects the specific clinical performance of each single cancer cell (Mendex et al., 2016). Disturbance in the menstrual cycle and hormonal alterations, have long been associated to ovarian tumorigenesis.

A large number of studies reveals that incidence and progression of ovarian carcinoma Due to abnormal expression of cytokines. Cytokines enhance or block the cancer cell growth via various secretory pathways, thereby influence the formation of new blood vessels and supplements food of the cancer cell, and control the immune responsibility of cancer cells to influence their clinical therapy. Ovarian carcinoma is correlated with several cytokines such as interleukin-8 (IL-8), interleukin-6(IL-6) interleukin-4 (IL-4)and IL-10, and their abnormal expression influence the process of incidence, progression and spread of tumour of ovarian carcinoma.IL-8 is a cytokine which can increase tumour cells metastasis. It has been reported that the expressions of IL-8 and IL-10 are accelerated in patients with ovarian carcinoma (Charbonneau et al., 2013). The goal of this study is to evaluate the Inflammatory response and hormonal alterations in ovarian cancer patients as a biochemical marker in prognostic ovarian carcinoma as well as indicators of cancer treatment response.

MATERIALS AND METHODS

Subjects
This study involved 85 females, aged 29–70 years, who have visited the Hiwa Hospital in Sulaymaniyah. These women were diagnosed to have ovarian cancer. A total of 65 healthy women from the outpa-tient department were involved in this study as a control group. Ethical approval and permission for the study were taken from the Ethical Committee of Sulaymaniyah University (Iraq). Informed consent was taken from all the study subjects purely for research purpose.

Samples
Collection of the blood
Blood was sampled before any treatment was given. Six millilitres of venous blood was taken without using tourniquet from each individual, collected in-plane polyethene tube, allowed to stand at room temperature for thirty minutes. Then the sample was centrifuged at (2000xg) for 10 minutes. The obtained serum transferred immediately to another test tube. These samples were estimated directly for enzymes activities or frozen at –20 C for subsequent analysis.

Methods
Measurement of serumInterleukin ( IL-4, IL-6, and IL-8)
The concentration of Interleukin (IL-4, IL-6, and IL-8) in serum samples were determined by sandwich enzyme-linked immunosorbent assay (ELISA) technique using the kit manufactured by BioVision company.

Principle
This ELISA kit uses Sandwich-ELISA as the method. The Microelisa strip plate provided in this kit has been pre-coated with an antibody specific to interleukin. Standards or samples are added to the appropriate Microelisa strip plate wells and combined to the specific antibody. Then a Horseradish Peroxidase (HRP)-conjugated antibody specific for interleukin is added to each Microelisa strip plate well and incubated. Free components (unbound conjugated antibodies) are washed away. The 3,3',5,5'-tetramethylbenzidine (TMB) substrate solution is added to each well. Only those wells that contain Interleukinant HRP conjugated Interleukinantibody will appear blue and then turn yellow after the addition of the stop solution (0.16M H2SO4). The optical density (OD) is measured spectrophotometrically at a wavelength of 450 nm. The OD value is directly proportional to the concentration of Interleukin standards and samples. We can calculate the concentration of interleukin the samples by comparing the OD of the samples to the standard curve.

Measurement of serum gonadotropins hormone
The concentration of gonadotropins hormone
(Estrogen, Progesterone, Prolactin, Testosterone) in serum samples was determined by sandwich enzyme-linked immunosorbent assay (ELISA) technique using the kit manufactured by BioVision company.

**Principle of the Assay**

This FSH enzyme-linked immunosorbent assay (ELISA) applies a technique called a quantitative sandwich immunoassay. The microtiter plate provided in this kit has been precoated with a monoclonal antibody specific for FSH. Standards or samples are then added to the microtiter plate wells and FSH if present, will bind to the antibody pre-coated on the wells, to quantitate the amount of FSH present in the sample, a standardized preparation of horseradish peroxidase (HRP) conjugated monoclonal antibody specific for FSH is added to each well to "sandwich" the FSH immobilized on the plate. The microtiter plate undergoes incubation, and then the wells are thoroughly washed to remove all unbound components. Next, a TMB (3,3’,5,5’ Tetramethyl-benzidine) substrate solution is added to each well. This enzyme (HRP) and substrate are allowed to react over a short incubation period. Only those wells that contain FSH and enzyme-conjugated antibody will exhibit a colour change. The enzyme-substrate reaction is terminated by the addition of a sulphuric acid solution, and the colour change is measured spectrophotometrically at a wavelength of 450 nm. To measure the concentration of FSH in the sample, this Human FSH ELISA Kit includes a set of calibration standards (6 standards). The calibration standards are assayed at the same time as the samples and allow the operator to produce a standard curve of Optical Density (OD) versus FSH concentration (mIU/mL). The concentration of FSH in the samples is then determined by comparing the OD of the samples to the standard curve.

**Measurement of serum steroid hormone**

The concentration of steroid hormone (LH and FSH) in serum samples were determined by sandwich enzyme-linked immunosorbent assay (ELISA) technique using the kit manufactured by BioVision company.

**Statistical analysis**

Statistical analysis was performed using the software Statistical Package for Social Sciences (SPSS) including data evaluation and tests for significance. Graph plots were done via windows excel version.

**RESULTS AND DISCUSSION**

The results in (Figure 1) showed a significant increase [p<0.001, p<0.002, p<0.01] in the level of interleukins (IL-4, IL-6, IL-8) in ovarian cancer patients compared to the healthy control group. These results were in agreement with those performed by others (Feng et al., 2018) which they found that serum interleukins increased in cancer patients compared to normal ones.

**Figure 1: Mean values of serum Interleukins (IL-6, IL-4, and IL-8) levels in control and Ovarian cancer patients**

**Figure 2: Mean values of serum Estrogen & progesteron levels in control and Ovarian cancer patients**

**Figure 3: Mean values of serum Testosterone & prolactin levels in control and Ovarian cancer patients**

The present study assessed possible interleukins that can be utilized as both diagnostic indicator and prognostic indicator in ovarian carcinoma. Various
types of cytokines (interleukins) are produced in response to immune responsivity that is involved in the induction of cell death and the regulation of spontaneous tumour remission.

A large number of studies propose that factors linked to the inflammation of the ovarian surface epithelium are correlated with an elevated risk for epithelial ovarian cancer (EOC). IL-6 is a cytokine with multiple activities performing various biological functions, and its circulating concentrations are increased in different types of cancer such as lung cancer, renal cell carcinoma and ovarian cancer. Previous studies on IL-6 have demonstrated its biological functions in the mechanism and development of cancer cell; IL-6 can act as Autocrine growth factors and paracrine growth factors, and it may enhance metastasis of tumour cell. IL-6 belongs to an immunoregulatory class of cytokines that are found in the ovarian carcinoma microenvironment. Both ovarian carcinoma cells and related phagocytic cells synthesize IL-6 and increased circulating concentration of IL-6 are known to be associated with critical alterations in the immune system and metabolic pathways that consequently causes ovarian carcinoma. It has been reported that IL-6 is complicated in the pathogenesis of ovarian carcinoma via autocrine signalling by increasing their ability to release matrix metallopeptidase 9 (Piura et al., 2013), thus enhancing angiogenesis. High IL-6 concentrations are linked with the progression of tumour and therapy outcome (Lippitz and Harris, 2016). Furthermore, several studies have demonstrated that IL-6 level is a possible prognostic marker in ovarian cancer; (Dalal et al., 2018). IL-8 plays a crucial role in several pathological conditions like inflammation, the responsibility of the immune system and inhibition of tumour metastasis. As a significant inflammatory agent, remarkably increased concentration of IL-8 are seen at sites of inflammation and bloodstream in the existence of contamination and various autoimmune disor-

der (Russo et al., 2014).

IL-8 also functions in the growth of normal cells and cancer cells. Enormous studies have demonstrated that IL-8 can enhance the progression of several kinds of cancers, such as pancreatic cancer, head and neck cancer; colon cancer, and lung cancer; (Chen et al., 2015). Mainly, it acts as a diagnostic marker in various cancers in agreement with previous research, the current study revealed that increased concentration of serum IL-8 was an indicator of the diminished rate of survival times and poor diagnosis in ovarian carcinoma.

High circulating levels of IL-8 were correlated with the shape of the tumour, depth of penetration, or elevation stage of disease in ovarian carcinoma. Investigated a cohort of ovarian carcinoma patients and observed that raised concentration of IL-8 was associated with an elevated death rate of diseases. In ovarian carcinoma increased concentration of IL-8 may cause the formation of new blood vessels of a developed nodule of a malignant tumour. These finding support the principle for selecting IL-8 as a curative approach in ovarian carcinoma. Wang et al. (2012) suggested that IL-8 released by ovarian carcinoma cells may involve in malignant action of these cells through enhancing binding signalling molecules to intracellular receptors in the cytoplasm or nucleus. IL-8-stimulated cell growth may be linked with changes of development of cell cycle and phosphorylation some signalling pathway such as PI3K/Akt and Raf/MEK/ERK pathway, whereas IL-8- enhance ovarian carcinoma cell occupation may be linked with both, elevated activity and expression of MMP-2 and MMP-9. Consequently, modification of IL-8 expression and its signalling pathway may be a good approach for regulating the development and spread of cancer cells in ovarian carcinoma.

Serum levels of estrogen

There was a significant increase (p < 0.01) in the level of estrogen hormones in ovarian cancer patients compared to the healthy control group as shown in Figure 2.

Increasing confirmation proposes that the hormonal environment is an essential impact of cancer progression, especially in ovarian carcinoma (Zhang et al., 2009). These hormones, comprising hormones of gonadotropin and steroid class of hormone, often play various roles; they commonly control cell proliferation, cell migration and cell death. Furthermore, in gynaecological cancers, gonadotropin causes ontogenesis, which is the foremost step in cancer progression, growth.
Estrogens have long doubted as causative agents of ovarian carcinoma. Even though the risk of ovarian carcinoma decreased with the utilization of oral contraceptive that contains estrogen, its effective role of estrogen is principally due to a decrease in the prevalence of the ovulation process. Concentrations of estrogen in ovarian tissue about a hundredfold higher than their concentrations in the blood and the follicular fluid, high circulating concentrations of estrogen in the ovarian stoma responsible to the damage that occurs in the cells of ovarian stoma epithelium which covering the ovulating follicles.

Critical causes and development of human gynecologic cancers are broadly attributed to the mitogenic properties of estrogen critical. The primarily biological actions of estrogens are to effects the activity of reproductive tissues, growth, and differentiation.

The risk of ovarian carcinoma is likely linked with the interaction of estrogens with their receptors which involved several signalling pathways (Zhao et al., 2010). Estrogen hormone has two types of receptors estrogen receptor alpha (ERα) and estrogen receptor beta (ERβ), which is the principal estrogen receptor in the ovary. Even though the exact effect of in ovarian carcinogenesis remains to be estimated, recent in vivo and in vitro studies propose that estrogen receptor beta induces the control of cellular differentiation, motility and cell death in ovarian carcinoma; and lack of estrogen receptor beta expression is linked with tumour development.

Several confirmations propose that steroid hormones, such as estrogen and progesterone, are involved in ovarian carcinoma. Estrogens prefer transformation of neoplastic cells in epithelium ovarian carcinoma, while progesterone provide defence against progression of ovarian carcinoma. Estrogens, especially that existence in ovulatory follicles, are potentially mutagenic or carcinogenic to ovarian surface epithelium cells. In contrast, the concentration of progesterone that is corresponding to pregnancy is a powerful hormone that causes cell death for both ovarian carcinoma cells and ovarian surface epithelium. In this consideration high-dosage of progestin may induce removing dead skin cells from the surface of the skin and clear elderly ovarian surface epithelium cells. It has been reported that ovarian carcinoma cells under estrogen regulation containing a complex that is formed by binding of estrogen to its specific receptors which are attached to regions of promoter regions of genes that initiate transcription of a single RNA from the DNA downstream of it (Ho, 2003).

Furthermore, ovarian carcinoma has been associated with an elevated level of estrogen in the body. High levels of fatty cells can induce the body to keep estrogen. They may transform other steroid hormones into a configuration of estrogen, elevating the possibility incidence of ovarian carcinoma (Wolin et al., 2010).

About eighty-six per cent of ovarian cancer samples smeared positive for receptors estrogen, and fifty per cent smeared positive for receptors of progesterone. Many studies propose that the usage of hormone replacement treatment of menopausal is linked with the elevation of rate of ovarian carcinoma especially in users under treatment with estrogen-progesterone for an extended period time (Anderson, 2003).

Serum levels of progesterone

The Mean±SD values of progesterone in sera samples are presented in Figure. The results in Figure 2 showed a significant increase (p < 0.01) in the level of estrogen and progesterone hormones in ovarian cancer patients compared to the healthy control group. The obtained results were in agreement with other investigations which suggest progesterone is implicated in ovarian carcinogenesis (Ibrahim, 2013).

The relationship between Progesterone hormone and ovarian carcinoma has been confirmed by various studies that illustrating a preventative role from the utilization of some medication such as oral contraceptives which contain progestins (Schüler et al., 2013).

Similarly, an equivalence is regarded as a preventative factor on the progression of ovarian carcinoma. Numerous studies attempted to establish a link between Progesterone Receptors and Estrogen Receptors, and the overall rate of viability in patients with ovarian carcinoma one of the study revealed that the existence of Progesterone Receptors is correlated with ameliorating development free viability in high rate in serum carcinoma and ovarian carcinoma (Sieh et al., 2013).

Previous studies have shown that the risk of ovarian epithelial carcinoma increases with decreasing in the response of progesterone hormone to its receptor because of a discontinuous genetic variation in the G intron of the progesterone receptor which is called PROGINS. It has been recorded that females with ovarian epithelial carcinoma with expressing both progesterone receptors and androgen receptors have elevated progression-free viability overall rate of viability (Jönsson et al., 2015). Several studies propose that progesterone hormone causes cell death via pro-apoptotic genes such as P53, Bax and the declination of Bcl-2 expression in ovarian car-
cinoma cells. Progesterone hormone has a controlling impact in the ADAMS family proteins (disintegrins and metalloproteinases with thrombospondin motifs), which are released by several kinds of cells and involved in the separation of proteoglycans in the matrix as well as in ontogenesis. It has been recorded that progesterone acts via its specific receptor, which induces the movement and invasion processes of ovarian carcinoma cell lines (Lima et al., 2016).

**Serum levels of testosterone**

The concentration of testosterone was measured in sera samples of healthy individuals and ovarian cancer patient groups. Mean±SD values of testosterone concentration in sera samples are presented in Figure 3. The results showed that there was a significant increase (P < 0.01) in serum concentration level in ovarian cancer group when compared with the control group.

High serum levels of testosterone, reaching levels above 200 ng/dL, could propose the existence of ovarian carcinoma. The biological role of androgens based on their interaction with their specific receptors (androgens receptors), which belong to the member of receptors that called nuclear receptors which acting as a protein that controls the rate of transcription of genetic information from DNA to messenger RNA, by binding to a specific DNA sequence. The existence of androgens receptors is linked to several kintypes of malignant tumours, like those found in the prostate, breast and bladder, making its participant in tumourigenesis processes apparent, either solely or in participation with other transcription coregulators and growth factor, androgen receptors and estrogen (ER) receptors are expressed in ovarian surface epithelial cells which is the normal ovary cell (Gibson et al., 2014). It has been reported that ovarian carcinoma progresses in a hormonal milieu enriched with androgens such as testosterone hormone (Blanco et al., 2017). The high circulating levels of androgens in ovarian carcinoma is identified by functional ovarian linked with high levels of androgens, with an increase in the rate of testosterone formed in the ovarian thecal cells. Many studies linked the circulating concentrations of androgens to the increased susceptibility of suffering from ovarian carcinoma patients, classifying the cancer cells either by their, grade and clinical stage (Ose et al., 2015). The concentration of mRNA in the androgen receptors are high in endometriosis, contributing in the elevation the rate for progression of endometriodi and clear-cell carcinomas, for which cause the potentiality that both the androgen receptors and androgens themselves could be involved in the appearance and advancement of these carcinomas.

**Serum levels of prolactin (PRL)**

Prolactin (PRL) concentration in sera samples of ovarian cancer and control group was measured. Mean±SD values of PRL in sera samples are presented in Figure. The result illustrating that there is a significant increase (p<0.0001) in serum PRL concentration in ovarian cancer patient group compared to those of the control group. These results were intimately comparable with the studies of Levin et al. (2009) who they reported significant-high serum PRL level in patients with ovarian cancer.

High levels of prolactin found in a remarkable number of patients with ovarian carcinoma may indicate a more severe defect in hypothalamic dopamine, besides characterized by increasing of main concentrations of prolactin.

Several confirmations that prolactin hormone plays a crucial role in several kinds of cancer, including prostate, breast and colon cancers through local synthesis. Increased concentrations of circulating prolactin in ovarian carcinoma have been recorded, stating that prolactin has a possible impact in ovarian carcinogenesis. Circulating concentration of prolactin are remarkably increased in females with ovarian carcinoma; this fact makes prolactin good indicator for ovarian carcinoma (Yurkovetsky et al., 2007). It can be proposing that increased serum prolactin may indicate an elevate in the formation of the hormones of anterior pituitary due to stress linked to cancer diagnosis, several confirmations declare versus it. It has been reported that serum prolactin is increased to a various extent in several types of cancer. Many studies of stress-enhanced elevate in serum prolactin in healthy individuals suggested that prolactin reactions to weakly emotional stress are seldom seen.

Although prolactin solely was not Expressed excessively or exaggeratedly in ovarian carcinoma, the high circulating concentration of prolactin should be enough for activating prolactin signalling in malignancy. These elevated concentrations of serum prolactin must generate either from anterior pituitary or arising outside the pituitary gland such as white blood cells. Remarkable increasing the response to a stimulus specifically of prolactin could be responsible for elevated prolactin signalling in ovarian carcinoma, causing to a high growth of cancer cells and cell survival rate. Additionally, prolactin may play various other effective functions in tumour progression via stimulating the growth of cancer tissue and phosphorylation pathways complicated in cellular sticking.
Serum levels of Follicle-Stimulating Hormone (FSH)

The results of FSH levels in serum of patients women with ovarian cancer and compared with control are presented in Figure. The results showed a significant increase (p < 0.05) in the level of FSH hormone in ovarian cancer patients compared to the healthy control group. The obtained results were in agreement with other investigations which suggest that FSH is implicated in ovarian carcinogenesis (Arslan et al., 2003).

The exact mechanism for the development of ovarian carcinoma remains unknown. To date, various theories have been suggested to describe the cause of ovarian carcinoma. Factors that increase the risk of ovarian carcinoma are early menarche, and late menopause led to the theory that inhibition of ovulation may be an essential factor in the progression of ovarian carcinoma. Another general research theory is the ‘gonadotrophin hypothesis’, which suggests that high circulating concentrations of gonadotrophin hormones after Primary ovarian insufficiency may play a vital role in the progression of ovarian carcinoma. Most women with ovarian carcinoma having high levels of FSH reaching (50–100 mIU/ml) (Howlader et al., 2011). Continuity of patients with ovarian carcinoma has been associated with expression the levels of Follicle-Stimulating Hormone. High rise in the levels of gonadotrophin hormones is found in the cyst fluid from Cancerous ovarian tumours. These findings propose that Follicle-Stimulating Hormone may play a key role in tumorigenesis of ovarian carcinoma. Elevated levels of serum Follicle-Stimulating Hormone diminished the risk of g ovarian carcinoma progression. The correlation between Follicle-Stimulating Hormone and ovarian carcinoma remains indecisive hormones of Gonadotrophin after binding to their specific receptor followed by and activation the signalling pathways including various cascades like PKA, PI3K/Akt and MAPK, this binding result in the controlling, cell death, cell growth, apoptosis and enlargement of tumour size in ovarian carcinoma. It has been reported that Follicle-Stimulating Hormone involved in the stimulation cell growth of ovarian carcinoma, blocks cell death, increases in the number of new blood vessels and facilitates the expression of VEGF (Huang et al., 2010).

Serum levels of Luteinizing hormone (LH)

Luteinizing hormone (LH) levels were determined in sera of control and ovarian cancer patient groups. Mean±SD values of sera LH levels are presented in Figure 4. The result uncovers the presence of statistically highly significant increase (P<0.0001) in the serum LH level in ovarian cancer group in comparison to that of the control group. These results were found to be compatible with the results obtained by Ran et al. (2017), who found that serum level of LH was higher than the usual physiologic levels in ovarian cancer patients.

The regulation of ovarian steroid synthesis is attributed to Luteinizing hormone that belongs to the gonadotropins class of hormone that secreted by the pituitary gland. The hypothesis for gonadotropin hormone of ovarian carcinoma suggested that progression of ovarian carcinoma due to accumulation of circulating both gonadotropins (FSH and LH) hormones. Several confirmations have described the impact of Luteinizing hormone in the causation of ovarian carcinoma. Previous researches report that Luteinizing hormone blocks Fas-induced -induced cell death in ovarian carcinoma. It has been reported that Luteinizing hormone could augment the occupation and moving of ovarian carcinoma through a signalling pathway called PI3K/AKT pathway. Several confirmations propose that hormones of gonadotropins enhance ovarian carcinoma OV207 cell migration and multiplication via phosphorylation of ERK1/2 signalling in a calcium (Mertens-Walker et al., 2010). Nevertheless, a few are known regarding the impact of Luteinizing hormone in the process of cancer cell growth.

The cause behind high levels of Luteinizing hormone in the current study remains not clear. The previous review attributes the high serum Luteinizing hormone level to amenorrhea that linked with the level of Luteinizing hormone with and low level of estradiol hormone. Some theories are proposed in previous studies.

The gonadotropin profile prompted a review of some unusual etiologies of amenorrhea associated with high LH, normal FSH and low estradiol. Some hypotheses are suggested based on a literature review. The formation of Luteinizing hormone by the ovarian cancer was eliminated via immunostaining technique.

Some studies described high serum Luteinizing hormone level to secretion a factor by cancer cells of Ovarian granulosa that could enhance the secretion of Luteinizing hormone from the pituitary glands. One potential prospect factor is a gonadotropin-releasing hormone-like substance, which has been found in the normal cells of Ovarian granulosa. The Ovarian granulosa cell cancer may have a role in the formation of the gonadotropin-releasing hormone during the production of tumour cell in Ovarian car-
cinoma patient. Another probability for the increasing of Luteinizing hormone in Ovarian granulosa cancer cells is linked to the mutation of Luteinizing hormone receptor, which is uncommon aetiology of amenorrhea linked with increasing levels of Luteinizing hormone (Donovan et al., 2010). On the one hand, indirectly, the cancer cell may have released some compound that causes an elevation in the circulating levels of Luteinizing hormone.

CONCLUSION

The findings of the present study suggest that an elevation of serum steroid hormone, gonadotropins, and Interleukin levels in cases of Ovarian carcinoma in our study is significant as a marker of the disease. Those biochemical parameters could be useful markers for prediction of prognosis in patients with Ovarian cancer and alteration of serum interleukin levels are strongly associated with ovarian cancer.

ACKNOWLEDGEMENT

The authors wish to thank Hiwa Hospital Staffs for their support in conducting the study.

Conflict of Interest

The authors declare that they have no conflict of interest for this study.

Funding Support

The authors declare that they have no funding support for this study.

REFERENCES

Anderson, G. L. 2003. Effects of Estrogen Plus Progestin on Gynecologic Cancers and Associated Diagnostic Procedures. The Women’s Health Initiative Randomized Trial. JAMA, 290(13):1739–1748.

Arslan, A. A., Zeleniuch-Jacquotte, A., Lundin, E., Micheli, A., Lukanova, A., Afanasyeva, Y., Lenner, P., Krogh, V., Muti, P., Rinaldi, S., Kaaks, R., Berrino, F., Hallmans, G., Toniolo, P. 2003. Serum follicle-stimulating hormone and risk of epithelial ovarian cancer in postmenopausal women. Biomarkers & Prevention: A Publication of the American Association for Cancer Research, 12(12):1531–1535.

Blanco, L. Z., Kuhn, E., Morrison, J. C., Bahadirli-Talbott, A., Smith-Sehdev, A., Kurman, R. J. 2017. Steroid hormone synthesis by the ovarian stroma surrounding epithelial ovarian tumors: a potential mechanism in ovarian tumorigenesis. Modern Pathology, 30(4):563–576.

Charbonneau, B., Goode, E. L., Kalli, K. R., Knutson, K. L., DeRycke, M. S. 2013. The Immune System in the Pathogenesis of Ovarian Cancer. Critical Reviews in Immunology, 33(2):137–164.

Chen, L., Fan, J., Chen, H., Meng, Z., Chen, Z., Wang, P., Liu, L. 2015. The IL-8/CXCR1 axis is associated with cancer stem cell-like properties and correlates with clinical prognosis in human pancreatic cancer cases. Scientific Reports, 4(1):1–7.

Dalal, V., Kumar, R., Kumar, S., Sharma, A., Kumar, L., Sharma, J. B., Roy, K. K., Singh, N., Vanamail, P. 2018. Biomarker potential of IL-6 and VEGF-A in ascitic fluid of epithelial ovarian cancer patients. Clinica Chimica Acta, 482:27–32.

Donovan, L. E., Brain, P. H., Duggan, M. A. 2010. Isolated luteinizing hormone (LH) elevation in a woman with secondary amenorrhea: a clue to the diagnosis of an inhibin B–producing thecoma and insights into the influence of inhibin B on LH. Fertility and Sterility, 94(3):1097.e9–1097.e12.

Dubosq, F., Ploussard, G., Soliman, H., Turpin, E., Latil, A., Desgrandchamps, F., de The, H., Mongiat-Artus, P. 2012. Identification of a three-gene expression signature of early recurrence in non-muscle-invasive urothelial cell carcinoma of the bladder. Urologic Oncology: Seminars and Original Investigations, 30(6):5228–5236.

Feng, L., Qi, Q., Wang, P., Chen, H., Chen, Z., Meng, Z., Liu, L. 2018. Serum levels of IL-6, IL-8, and IL-10 are indicators of prognosis in pancreatic cancer. Journal of International Medical Research, 46(12):5228–5236.

Gibson, D. A., Simitisidellis, I., Collins, F., Saunders, P. T. K. 2014. Evidence of androgen action in endometrial and ovarian cancers. Endocrine-Related Cancer, 21(4):T203–T218.

Ho, S. M. 2003. Estrogen, progesterone and epithelial ovarian cancer. Reproductive Biology and Endocrinology, 1(1):1–8.

Howlader, N., Noone, A. M., Krapcho, M., Neyman, N., Aminou, R., Waldron, W., Altekruse, S. F., Kosary, C. L., Ruhl, J., Tatalovich, Z. 2011. Cancer of the ovary. SEER Cancer Statistics Review, 1975–2008. National Cancer Institute.

Huang, Y., Jin, H., Liu, Y., Zhou, J., Ding, J., Cheng, K. W., Yu, Y., Feng, Y. 2010. FSH inhibits ovarian cancer cell apoptosis by up-regulating survivin and down-regulating PDCD6 and DR5. Endocrine Related Cancer, 18(1):13–26.

Ibrahim, R. T. 2013. Study of Estrogen, Progesterone, Copper, Zinc and some Antioxidants in Sera of Ovarian Cancer Patients. Rafidain journal of science, 24(5E):64–71.

Jönsson, J.-M., Arildsen, N. S., Malander, S., Måsbäck,
Parween Abdulsamad Ismail and Lana Muhammad Ali, Int. J. Res. Pharm. Sci., 2020, 11(4), 7166-7174

A., Hartman, L., Nilbert, M., Hedenfalk, I. 2015. Sex Steroid Hormone Receptor Expression Affects Ovarian Cancer Survival. Translational Oncology, 8(5):424–433.

Levina, V. V., Nolen, B., Su, Y., Godwin, A. K., Fishman, D., Liu, J., Mor, G., Maxwell, L. G., Herberman, R. B., Szczepanski, M. J., Szajnik, M. E., Gorelik, E., Lokshin, A. E. 2009. Biological Significance of Prolactin in Gynecologic Cancers. Cancer Research, 69(12):5226–5233.

Lima, M. A., da Silva, S. V., Freitas, V. M. 2016. Progesterone acts via the progesterone receptor to induce adams proteases in ovarian cancer cells. Journal of Ovarian Research, 9(1):1–11.

Lippitz, B. E., Harris, R. A. 2016. Cytokine patterns in cancer patients: A review of the correlation between interleukin 6 and prognosis. Oncoimmunology, 5(5):1–12.

Mendez, Morales-Vasquez, Gómora, M. J., Calvillo-Robledo, Lopez, Basave, P. 2016. Sexual Steroids in Epithelial Ovarian Cancer. Ovarian Cancer, 311:1321–1312.

Mertens-Walker, I., Bolitho, C., Baxter, R. C., Marsh, D. J. 2010. Gonadotropin-induced ovarian cancer cell migration and proliferation require extracellular signal-regulated kinase 1/2 activation regulated by calcium and protein kinase C8. Endocrine-Related Cancer, 17(2):335–349.

Ose, J., Fortner, R. T., Rinaldi, S., Schock, H., Overvad, K., Tjonneland, A., Hansen, L., Dossus, L., Fournier, A., Baglietto, L., et al. 2015. Endogenous androgens and risk of epithelial invasive ovarian cancer by tumor characteristics in the European Prospective Investigation into Cancer and Nutrition. International Journal of Cancer, 136(2):399–410.

Piura, B., Medina, L., Rabinovich, A., Dyomin, V., Huleihel, M. 2013. Thalidomide distinctly affected TNF-α, IL-6 and MMP secretion by an ovarian cancer cell line (SKOV-3) and primary ovarian cancer cells. European Cytokine Network, 24(3):122–129.

Ran, S., Yu, Q., Deng, S., Xu, L. 2017. Luteinizing hormone elevation in ovarian granulosa cell tumor: a case report and review of the literature. Journal of Ovarian Research, 10(1):1–5.

Russo, R. C., Garcia, C. C., Teixeira, M. M., Amaral, F. A. 2014. The CXCL8/IL-8 chemokine family and its receptors in inflammatory diseases. Expert Review of Clinical Immunology, 10(5):593–619.

Schüler, S., Ponnath, M., Engel, J., Ortmann, O. 2013. Ovarian epithelial tumors and reproductive factors: a systematic review. Archives of Gynecology and Obstetrics, 287(6):1187–1204.

Sieh, W., Köbel, M., Longacre, T. A., Bowtell, D. D., Defazio, A., Goodman, M. T., Høgdall, E., Deen, S., Wentzensen, N., Moysich, K. B., Brenton, J. D., Clarke, B. A., Menon, U., Gilks, C. B., Kim, A., Madore, J., Fereday, S., George, J., Galletta, L., Ramus, S. J. 2013. Hormone-receptor expression and ovarian cancer survival: an Ovarian Tumor Tissue Analysis consortium study. The Lancet Oncology, 14(9):70253–70258.

Wang, Y., Xu, R. C., Zhang, X. L., Niu, X. L., Qu, Y., Li, L. Z., Meng, X. Y. 2012. Interleukin-8 secretion by ovarian cancer cells increases anchorage-independent growth, proliferation, angiogenic potential, adhesion and invasion. Cytokine, 59(1):145–155.

Wolin, K. Y., Carson, K., Colditz, G. A. 2010. Obesity and Cancer: The Oncologist, 15:556–565.

Yurkovetsky, Z., Taasan, S., Skates, S., Rand, A., Lomakin, A., Linkov, F., Marrangoni, A., Velikokhatnaya, L., Winans, M., Gorelik, E. 2007. Development of multimarker panel for early detection of endometrial cancer. High diagnostic power of prolactin. Gynecologic Oncology, 107(1):58–65.

Zhang, Z., Jia, L., Feng, Y., Zheng, W. 2009. Overexpression of follicle-stimulating hormone receptor facilitates the development of ovarian epithelial cancer. Cancer Letters, 278(1):56–64.

Zhao, B. B., Yang, Z. J., Wang, Q., Pan, Z. M., Zhang, W., Li, L. 2017. Clinical validation of multiple biomarkers suspension array technology for ovarian cancer. Zhonghua fu Chan ke za zhi, 52(1):11–19.

Zhao, C., Dahlman-Wright, K., Gustafsson, J.-Å. 2010. Estrogen Signaling via Estrogen Receptor β. Journal of Biological Chemistry, 285(51):39575–39579.