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A comparative evaluation of myeloid-derived suppressor cells and murine double minute 2 in Iraqi breast cancer patients before therapy

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Abstract

Background: Breast cancer (BC) is the most prevalent and life-threatening form of cancer, with a high rate of mortality. Myeloid-derived suppressor cells (MDSCs) are a heterogeneous population of immature myeloid cells that overexplicitly proliferate in cancer and have potent immunosuppressive capacity. In the meantime, murine double minute 2 (MDM2) is also known to play an oncogenic role, especially in the development of breast cancer.

Objective: This paper was designed to evaluate MDSCs and MDM2 as possible indicators to distinguish between patients with BC prior to treatment and healthy persons.

Methods: In this case-control study, a total of 90 participants were enrolled, including 65 newly diagnosed, untreated breast cancer patients (cases) and 25 healthy individuals (controls). All patients were recruited from the Oncology Teaching Hospital in Baghdad, Iraq. The enzyme-linked immunosorbent assay (ELISA) is used to measure the total levels of both MDSCs and MDM2 in peripheral blood.

Results: The results of the current study indicated that the serum level of MDSCs was highly significant in breast cancer patients before receiving therapy ($P \leq 0.001$) compared with healthy controls. Furthermore, the MDM2 was also more elevated in BC patients than in healthy controls ($P \leq 0.05$).

Conclusion: In conclusion, the study found a highly significant difference in serum MDSCSs in BC patients before treatment compared to the control. We also observed elevated levels of MDM2 in BC patients, which we attributed to the onset of cancer.

Keywords: Breast cancer, Myeloid-derived suppressor cells, Murine Double Minute 2

Introduction:

Breast cancer (BC) is the most frequently diagnosed malignant tumor among women worldwide and stands as the primary cause of death related to malignant tumors (Smolarz et al., 2022). The development and progression of breast cancer are largely driven by intrinsic genetic and signaling pathway alterations within tumor cells, as well as by extrinsic disturbances related to the tumor immune microenvironment (Fonseca-Montaño et al., 2023). Myeloid-derived suppressor cells (MDSCs) are a form of suppressor cell that stops the immune system's ability to mount an anti-tumor response by either suppressing or inhibiting cells from responding to the tumor in ways that would lead to the death of the tumor cell (Gatti-Mays et al., 2019). All of these components create an environment in which the tumors can develop, evade, and spread rapidly.

MDSCs, a diverse assemblage of immature myeloid cells (IMCs), are integral to the immune cell network (Cha & Koo, 2020). MDSCs are produced out of a reservoir of myeloid progenitors, as well as other immature myeloid cells that are yet to undergo differentiation. Under normal physiological states, these IMCs rapidly develop into mature granulocytes, monocytes, and dendritic cells, which thereafter migrate to different tissues and organs to perform their normal immune functions (Gabrilovich et al., 2012; Geissmann et al., 2010; Q. Li et al., 2004; Veglia et al., 2018). In diseases such as cancer, myeloid progenitor cells fail to develop, and they remain at various levels of differentiation and are transformed to MDSCs with the ability to suppress immunity (Condamine et al., 2015; Gabrilovich & Nagaraj, 2009; Ostrand-Rosenberg & Sinha, 2009). The mechanisms by which MDSCs contribute to the progression of tumors are multiple immunosuppressive effects, including the depletion of metabolites, the generation of reactive oxygen species (ROS), and the release of a wide range of cytokines, as well as non-immunosuppressive ones, such as epithelial-mesenchymal transition (EMT), the increase of tumor cell stemness, and the induction of tumor vascularization (K. Li et al., 2021).

Murine double minute 2 (MDM2) is a normal protein that usually acts as a significant controller of the tumor-suppressing protein p53 (Zafar et al., 2023). MDM2 plays a role in multiple biological processes, including Cell Proliferation, angiogenesis, metabolic reprogramming, and the prevention of apoptosis, which can lead to the transformation of a non-malignant cell into a malignant one and aid in the development of cancerous tumors and the subsequent spread of the cancerous tumor to other areas of the body (Haupt et al., 2017). In addition to its role in relation to p53, MDM2 has numerous

functions that do not involve the p53 protein; it regulates many other molecular targets in a variety of ways that include transcriptional regulation, post-translational modification, protein degradation, cofactor interaction, and changes in subcellular localization (Bohlman & Manfredi, 2014).

This study aimed to assess MDSCs and MDM2 as potential immunological biomarkers for differentiating between patients with BC before therapy and healthy individuals.

Methodology:

Study Design

This research employed a comparative case-control study design to investigate the serum levels of MDSCs and MDM2. The study was conducted at the Oncology Teaching Hospital (Baghdad, Iraq) and involved 65 newly diagnosed, treatment-naive breast cancer cases and 25 healthy controls. This design was selected to determine if significant differences exist between these two groups, allowing for the evaluation of these markers as potential diagnostic indicators. This investigation comprised BC and control females of various regions of Iraq.

sample collection

Peripheral blood (5.0 ml) was collected from newly diagnosed breast cancer cases before receiving therapy (n = 65) and healthy controls (n = 25) to evaluate MDSCs and MDM2. Clinic-pathological feature data, including age, sex, family history, tumor stage, smoking and alcohol, and histological and immunohistochemical classification, were collected under the supervision of an oncologist at the Oncology Teaching Hospital (Baghdad, Iraq).

Inclusion and exclusion Criteria

Women newly diagnosed with breast cancer (BC) and enrolled in the study underwent clinical and laboratory examinations, including clinical breast examination, medical imaging (mammography and ultrasound), and various biopsy tests considered the gold standard, such as fine needle aspirate cytology (FNAC) and true-cut biopsy techniques. Patients with BC who have already received therapy were excluded.

Measurement of MDSCs and MDM2

The concentrations of myeloid-derived suppressor cells (MDSC) and myeloid-derived suppressor cell (MDM 2), as well as their respective proportions, were assessed using double antibody sandwich enzyme-linked immunosorbent assay (ELISA) kits. The procedure was performed by adding both the standards and the sample to a well that has been pre-coated with an antibody against the target, followed by adding an HRP conjugated reagent to create an immune complex. Following incubation and removal of all unbound

components through washing, substrates A and B were added. The intensity of the final color was directly proportional to the concentration of MDSCs or MDM2 in the sample. These two ELISA Commercial test kits are from Shanghai Coon Koon Biotech Co., Ltd. The MDSC ELISA kit (Cat. No: [CK-bio-27691]) exhibited a sensitivity of [10.0 pg/ml] with a Standard Curve range [20 pg/ml - 2000 pg/ml]. The MDM2 ELISA kit (Cat. No: [CK-bio-21601]) demonstrated a sensitivity of [0.1 ng/mL] and Standard Curve range [1 ng/mL - 20 ng/mL]. The kits were validated by the manufacturer for high specificity with no significant cross-reactivity with other soluble proteins. Intra-assay and inter-assay coefficients of variation (CV) were maintained at <7% and <10%, respectively, to ensure reproducibility. Standard curves were generated for each individual run to confirm quantitative accuracy. Optical density (OD) was measured at 450 nm using a spectrophotometric microplate reader.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were examined for accuracy and normality prior to analysis. Data are presented as mean ± standard deviation (SD). All statistical tests were two-tailed, and a p-value < 0.05 was considered statistically significant. The results are reported with corresponding t-tests and p-values, in accordance with standard statistical reporting guidelines for biomedical research.

Results:

1-Determining the level of MDSCs

According to the results, the patient group's average MDSC concentration was highly significant in the patient group (1120.32 ± 110.84) compared to the control group (1007.01 ± 78.83). As indicated in Table (1), the t-test value of (5.42) and the associated P-value verify that this difference is statistically significant (P≤0.001).

Table 1: Comparison between patients and control groups in MDSCs.

Group	Mean ±SD of MDSCs
Patients	1120.32 ± 110.84
Control	1007.01 ± 78.83
T-test	5.42
P-value	≤0.001

2-Determination the level of MDM2

The results from the ELISA technique indicated that MDM2 concentrations were higher in the test group compared to the healthy control, with significant differences observed between the two groups (P≤0.05), as shown in Table (2).

Table 1: Comparison between patients and control groups in MDM2.

Group	Mean ±SD of MDM2
Patients	7.61 ± 1.78
Control	6.69 ± 1.31
T-test	2.35
P-value	≤0.05

Discussion:

MDSCs are crucial in tumor development due to their immunosuppressive capabilities (K. Li et al., 2021). As a result of hematopoietic stem cell differentiation, the production of cytokines and chemokines by tumors has led to abnormal proliferation of immature bone marrow cells (Karin, 2020; van Vlerken-Ysla et al., 2023; J. Zhou et al., 2018). Immature bone marrow-derived cells are able to suppress T cells, NK cells, and dendritic cells (DC) through the release of reactive oxygen/nitrogen species (ROS/RNS) and other immunosuppressive factors and evade immune detection by the use of exosomes (De Cicco et al., 2020; Gabrilovich & Nagaraj, 2009; Groth et al., 2019; He et al., 2025). Studies have also shown that MDSCs also have non-immune tumorigenic functions, such as stimulating the EMT, tumor angiogenesis, and the formation of premetastatic niches that are favorable to tumor growth and metastasis (Cai et al., 2021; Cheng et al., 2021; Zhang et al., 2023). Numerous studies have demonstrated that MDSCs may be useful as prognostic markers for cancer progression and treatment response, particularly as a predictor of response to immune checkpoint inhibitors (Genova et al., 2021; Park & Youn, 2019; Salvia et al., 2024). Furthermore, MDSCs are becoming recognized as potential therapeutic targets for cancer therapy (Barry et al., 2023; Nourbakhsh et al., 2021; Okla et al., 2018).

The current study's results indicated that MDSCs were significantly elevated in the BC prior to therapy when compared to the control group (P≤0.001), as illustrated in Table (1). This study aligns with multiple studies demonstrating the enrichment of MDSCs in peripheral blood (Diaz-Montero et al., 2009; Markowitz et al., 2013). Also, Foulds *et al.* (2018) demonstrated that an increased percentage of immunosuppressive cells such as MDSCs was observed in patients with BC (Foulds et al., 2018). Additionally, a separate

study found that preoperative and recurrent breast cancer patients had elevated levels of MDSC concentrations compared to postoperative, chemotherapy-treated recurrent breast cancer patients and healthy controls (Gonda et al., 2017). A further study shows a decrease in MDSCs levels in the peripheral blood of patients with metastatic BC after chemotherapy (Gonda et al., 2012).

In normal cells, MDM2 is necessary for maintaining cell viability; however, when overexpressed in tumor cells, it interferes with the tumor-suppressive activity of p53 (Lu et al., 2020). It accomplishes this in two main ways: first, it binds to the transactivation domain of p53 and stops it from interacting with DNA. Second, it serves as an E3 ubiquitin ligase, which speeds up the ubiquitination and breakdown of p53 by the proteasome (Lu et al., 2020). In addition, the key factor that initiates the development of blood vessels under normal and abnormal conditions is Vascular endothelial growth factor (VEGF) (Apte et al., 2019). VEGF benefits cancerous tissue in facilitating angiogenesis within solid tumor. A close relationship between high levels of VEGF and MDM2 has been determined by researchers. This implies that MDM2 is significant in angiogenesis mediated by VEGF (S. Zhou et al., 2011). Moreover, it is also known that epithelial-to-mesenchymal transition (EMT) plays a vital role in tumor metastasis and helps make tumor cells more mobile and penetrating into the surrounding stromal tissue (Sun et al., 2023). Researchers propose that MDM2 can promote EMT by increasing transcription factors that can regulate it (Ou et al., 2021).

In the present research, findings revealed that the level of significant MDM2 as an oncogene prior to the therapy was high in the majority of the patients with BC ($P \leq 0.05$) as indicated in **(Table 2)**. This research paper concurs with a previous study that suggested that MDM2 is commonly overexpressed in BC, thus promoting tumor growth, invasiveness, and resistance treatment (Haupt et al., 2017; Zhao et al., 2014). Additionally, it was also shown that MDM2 activity is required to keep healthy cells alive and its overexpression in cancer cells hampers the tumor suppressor activity of p53 (Lu et al., 2020). In another study, it was discovered that MDM2 overexpression enhances the process of angiogenesis and forms a favorable angiogenic environment, thus increasing the motility and invasiveness of tumor cells (Venkatesan et al., 2018). Nevertheless, changes in the level of MDM2 expression may result in uncontrolled proliferation (Zafar et al., 2023).

The current study utilized a cross-sectional design at a single time point (prior to therapy). Consequently, we could not monitor how MDSC and MDM2 levels fluctuate during or after treatment (e.g., chemotherapy or

surgery), which could provide valuable prognostic information. Moreover, Human MDSCs are highly heterogeneous and difficult to isolate due to a lack of definitive markers. While our ELISA approach measured total MDSC levels, it did not distinguish between specific subpopulations, such as monocytic (M-MDSCs) or granulocytic (G-MDSCs). Detailed phenotypic analysis using flow cytometry could provide deeper insights into their specific immunosuppressive roles. Despite these constraints, this study provides early evidence that MDSCs and MDM2 are promising non-invasive indicators for the initial detection of BC in Iraqi patients.

Conclusion:

To sum up, the study has established that the difference between patients and control groups in terms of serum MDSCSs was very significant among BC patients prior to treatment. We also found high concentrations of MDM2 in patients of BC, which we explained by the development of cancer.

Recommendations:

Further studies can determine the concentrations of MDSCs in immunological microenvironment of BC patients even before treatment and compare to serum levels. Separate evaluation of different MDSCs subtypes is also recommended. This study proposes assessing p53 protein levels and their correlation with MDM2 protein as potential indicators.

Ethical Considerations

The research received approval from the Ethics Committee in Biology Research at the College of Science, Mustansiriyah University, Baghdad, Iraq, as well as from the Iraqi Ministry of Health and Environment. Committee number BCSMU/1024/00066Z.

References

Apte, R. S., Chen, D. S., & Ferrara, N. (2019). VEGF in Signaling and Disease: Beyond Discovery and Development. Cell, 176(6), 1248–1264. https://doi.org/10.1016/j.cell.2019.01.021

Barry, S. T., Gabrilovich, D. I., Sansom, O. J., Campbell, A. D., & Morton, J. P. (2023). Therapeutic targeting of tumour myeloid cells. Nature Reviews. Cancer, 23(4), 216–237. https://doi.org/10.1038/s41568-022-00546-2

Bohlman, S., & Manfredi, J. J. (2014). p53-independent effects of Mdm2. Sub-Cellular Biochemistry, 85, 235–246. https://doi.org/10.1007/978-94-017-9211-0_13

Cai, J., Cui, Y., Yang, J., & Wang, S. (2021). Epithelial-mesenchymal transition: When tumor cells meet myeloid-derived suppressor cells. Biochimica et Biophysica Acta. Reviews on Cancer, 1876(1), 188564. https://doi.org/10.1016/j.bbcan.2021.188564

Cha, Y. J., & Koo, J. S. (2020). Role of Tumor-Associated Myeloid Cells in Breast Cancer. Cells, 9(8). https://doi.org/10.3390/cells9081785
Cheng, J.-N., Yuan, Y.-X., Zhu, B., & Jia, Q. (2021). Myeloid-Derived Suppressor Cells: A Multifaceted Accomplice in Tumor Progression. Frontiers in Cell and Developmental Biology, 9, 740827. https://doi.org/10.3389/fcell.2021.740827
Condamine, T., Mastio, J., & Gabrilovich, D. I. (2015). Transcriptional regulation of myeloid-derived suppressor cells. Journal of Leucocyte Biology, 98(6), 913–922.
De Cicco, P., Ercolano, G., & Ianaro, A. (2020). The new era of cancer immunotherapy: targeting myeloid-derived suppressor cells to overcome immune evasion. Frontiers in Immunology, 11, 1680.
Diaz-Montero, C. M., Salem, M. L., Nishimura, M. I., Garrett-Mayer, E., Cole, D. J., & Montero, A. J. (2009). Increased circulating myeloid-derived suppressor cells correlate with clinical cancer stage, metastatic tumor burden, and doxorubicin-cyclophosphamide chemotherapy. Cancer Immunology, Immunotherapy : CII, 58(1), 49–59. https://doi.org/10.1007/s00262-008-0523-4
Fonseca-Montaño, M. A., Vázquez-Santillán, K. I., & Hidalgo-Miranda, A. (2023). The current advances of lncRNAs in breast cancer immunobiology research. Frontiers in Immunology, 14(June), 1–16. https://doi.org/10.3389/fimmu.2023.1194300
Foulds, G. A., Vadakekolathu, J., Abdel-Fatah, T. M. A., Nagarajan, D., Reeder, S., Johnson, C., Hood, S., Moseley, P. M., Chan, S. Y. T., Pockley, A. G., Rutella, S., & McArdle, S. E. B. (2018). Immune-Phenotyping and Transcriptomic Profiling of Peripheral Blood Mononuclear Cells From Patients With Breast Cancer: Identification of a 3 Gene Signature Which Predicts Relapse of Triple Negative Breast Cancer. Frontiers in Immunology, 9, 2028. https://doi.org/10.3389/fimmu.2018.02028
Gabrilovich, D. I., & Nagaraj, S. (2009). Myeloid-derived suppressor cells as regulators of the immune system. Nature Reviews Immunology, 9(3), 162–174.
Gabrilovich, D. I., Ostrand-Rosenberg, S., & Bronte, V. (2012). Coordinated regulation of myeloid cells by tumours. Nature Reviews Immunology, 12(4), 253–268.
Gatti-Mays, M. E., Balko, J. M., Gameiro, S. R., Bear, H. D., Prabhakaran, S., Fukui, J., Disis, M. L., Nanda, R., Gulley, J. L., Kalinsky, K., Abdul Sater, H., Sparano, J. A., Cescon, D., Page, D. B., McArthur, H., Adams, S., & Mittendorf, E. A. (2019). If we build it they will come: targeting the

immune response to breast cancer. NPJ Breast Cancer, 5, 37. https://doi.org/10.1038/s41523-019-0133-7
Geissmann, F., Manz, M. G., Jung, S., Sieweke, M. H., Merad, M., & Ley, K. (2010). Development of monocytes, macrophages, and dendritic cells. Science, 327(5966), 656–661.
Genova, C., Dellepiane, C., Carrega, P., Sommariva, S., Ferlazzo, G., Pronzato, P., Gangemi, R., Filaci, G., Coco, S., & Croce, M. (2021). Therapeutic Implications of Tumor Microenvironment in Lung Cancer: Focus on Immune Checkpoint Blockade. Frontiers in Immunology, 12, 799455. https://doi.org/10.3389/fimmu.2021.799455
George, D., & Mallery, P. (2019). IBM SPSS Statistics 26 Step by Step: A Simple Guide and Reference. https://doi.org/10.4324/9780429056765
Gonda, K., Shibata, M., Ohtake, T., Matsumoto, Y., Tachibana, K., Abe, N., Ohto, H., Sakurai, K., & Takenoshita, S. (2017). Myeloid-derived suppressor cells are increased and correlated with type 2 immune responses, malnutrition, inflammation, and poor prognosis in patients with breast cancer. Oncology Letters, 14(2), 1766–1774. https://doi.org/10.3892/ol.2017.6305
Gonda, K., Shibata, M., Ohtake, T., Yasuda, M., Abe, N., Watanabe, K., Ando, J., Okano, M., Onozawa, H., Tachibana, K., Ohto, H., & Takenoshita, S. (2012). [Myeloid-derived suppressor cells in patients with breast cancer]. Gan to kagaku ryoho. Cancer & chemotherapy, 39(9), 1363–1368.
Groth, C., Hu, X., Weber, R., Fleming, V., Altevogt, P., Utikal, J., & Umansky, V. (2019). Immunosuppression mediated by myeloid-derived suppressor cells (MDSCs) during tumour progression. British Journal of Cancer, 120(1), 16–25.
Haupt, S., Vijayakumaran, R., Miranda, P. J., Burgess, A., Lim, E., & Haupt, Y. (2017). The role of MDM2 and MDM4 in breast cancer development and prevention. Journal of Molecular Cell Biology, 9(1), 53–61.
He, S., Zheng, L., & Qi, C. (2025). Myeloid-derived suppressor cells (MDSCs) in the tumor microenvironment and their targeting in cancer therapy. Molecular Cancer, 24(1), 5. https://doi.org/10.1186/s12943-024-02208-3
Karin, N. (2020). The development and homing of myeloid-derived suppressor cells: from a two-stage model to a multistep narrative. Frontiers in Immunology, 11, 557586.
Li, K., Shi, H., Zhang, B., Ou, X., Ma, Q., Chen, Y., Shu, P., Li, D., & Wang, Y. (2021). Myeloid-derived suppressor cells as immunosuppressive regulators and therapeutic targets in cancer. Signal Transduction and Targeted Therapy, 6(1), 362. https://doi.org/10.1038/s41392-021-00670-9

Li, Q., Pan, P.-Y., Gu, P., Xu, D., & Chen, S.-H. (2004). Role of immature myeloid Gr-1+ cells in the development of antitumor immunity. Cancer Research, 64(3), 1130–1139.

Lu, H., Zhou, Q., He, J., Jiang, Z., Peng, C., Tong, R., & Shi, J. (2020). Recent advances in the development of protein-protein interactions modulators: mechanisms and clinical trials. Signal Transduction and Targeted Therapy, 5(1), 213. https://doi.org/10.1038/s41392-020-00315-3

Markowitz, J., Wesolowski, R., Papenfuss, T., Brooks, T. R., & Carson, W. E. 3rd. (2013). Myeloid-derived suppressor cells in breast cancer. Breast Cancer Research and Treatment, 140(1), 13–21. https://doi.org/10.1007/s10549-013-2618-7

Nourbakhsh, E., Mohammadi, A., Salemizadeh Parizi, M., Mansouri, A., & Ebrahimzadeh, F. (2021). Role of Myeloid-derived suppressor cell (MDSC) in autoimmunity and its potential as a therapeutic target. Inflammopharmacology, 29(5), 1307–1315. https://doi.org/10.1007/s10787-021-00846-3

Okla, K., Wertel, I., Wawruszak, A., Bobiński, M., & Kotarski, J. (2018). Blood-based analyses of cancer: Circulating myeloid-derived suppressor cells - is a new era coming? Critical Reviews in Clinical Laboratory Sciences, 55(6), 376–407. https://doi.org/10.1080/10408363.2018.1477729

Ostrand-Rosenberg, S., & Sinha, P. (2009). Myeloid-derived suppressor cells: linking inflammation and cancer. Journal of Immunology (Baltimore, Md. : 1950), 182(8), 4499–4506. https://doi.org/10.4049/jimmunol.0802740

Ou, M., Xu, X., Chen, Y., Li, L., Zhang, L., Liao, Y., Sun, W., Quach, C., Feng, J., & Tang, L. (2021). MDM2 induces EMT via the B‐Raf signaling pathway through 14‐3‐3. Oncology Reports, 46(1). https://doi.org/10.3892/or.2021.8071

Park, S.-M., & Youn, J.-I. (2019). Role of myeloid-derived suppressor cells in immune checkpoint inhibitor therapy in cancer. Archives of Pharmacal Research, 42(7), 560–566. https://doi.org/10.1007/s12272-019-01165-6

Salvia, R., Rico, L. G., Morán, T., Bradford, J. A., Ward, M. D., Drozdowskyj, A., Climent-Martí, J., Martínez-Cáceres, E. M., Rosell, R., & Petriz, J. (2024). Prognostic Significance of PD-L1 Expression on Circulating Myeloid-Derived Suppressor Cells in NSCLC Patients Treated with Anti-PD-1/PD-L1 Checkpoint Inhibitors. International Journal of Molecular Sciences, 25(22), 12269.

Smolarz, B., Zadro, A., & Romanowicz, H. (2022). Treatment (Review of Literature). 1–27.

Sun, D., Feng, F., Teng, F., Xie, T., Wang, J., Xing, P., Qian, H., & Li, J.

(2023). Multiomics analysis revealed the mechanisms related to the enhancement of proliferation, metastasis and EGFR-TKI resistance in EGFR-mutant LUAD with ARID1A deficiency. Cell Communication and Signaling : CCS, 21(1), 48. https://doi.org/10.1186/s12964-023-01065-9
van Vlerken-Ysla, L., Tyurina, Y. Y., Kagan, V. E., & Gabrilovich, D. I. (2023). Functional states of myeloid cells in cancer. Cancer Cell, 41(3), 490–504. https://doi.org/10.1016/j.ccell.2023.02.009
Veglia, F., Perego, M., & Gabrilovich, D. (2018). Myeloid-derived suppressor cells coming of age. Nature Immunology, 19(2), 108–119.
Venkatesan, T., Alaseem, A., Chinnaiyan, A., Dhandayuthapani, S., Kanagasabai, T., Alhazzani, K., Dondapati, P., Alobid, S., Natarajan, U., Schwartz, R., & Rathinavelu, A. (2018). MDM2 Overexpression Modulates the Angiogenesis-Related Gene Expression Profile of Prostate Cancer Cells. Cells, 7(5). https://doi.org/10.3390/cells7050041
Zafar, A., Khan, M. J., & Naeem, A. (2023). MDM2- an indispensable player in tumorigenesis. Molecular Biology Reports, 50(8), 6871–6883. https://doi.org/10.1007/s11033-023-08512-3
Zhang, T., He, Y., Man, G. C. W., Ding, Y., Wang, C. C., & Chung, J. P. W. (2023). Myeloid-derived suppressor cells: A new emerging player in endometriosis. International Review of Cell and Molecular Biology, 375, 191–220. https://doi.org/10.1016/bs.ircmb.2022.11.004
Zhao, Y., Yu, H., & Hu, W. (2014). The regulation of MDM2 oncogene and its impact on human cancers. Acta Biochimica et Biophysica Sinica, 46(3), 180–189. https://doi.org/10.1093/abbs/gmt147
Zhou, J., Nefedova, Y., Lei, A., & Gabrilovich, D. (2018). Neutrophils and PMN-MDSC: Their biological role and interaction with stromal cells. Seminars in Immunology, 35, 19–28. https://doi.org/10.1016/j.smim.2017.12.004
Zhou, S., Gu, L., He, J., Zhang, H., & Zhou, M. (2011). MDM2 regulates vascular endothelial growth factor mRNA stabilization in hypoxia. Molecular and Cellular Biology, 31(24), 4928–4937. https://doi.org/10.1128/MCB.06085-11

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تقييم مقارن للخلايا المُثبِّطة المشتقة من النخاع وبروتين الفئران الدقيقة المزدوجة 2 لدى مرضى سرطان الثدي العراقيين قبل تلقي العلاج

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مستخلص البحث:

الخلفية: سرطان الثدي هو أكثر أنواع السرطان شيوعا ويهدد الحياة، ويرتبط بارتفاع معدل الوفيات. تمثل الخلايا المثبطة المشتقة من النخاع (MDSCs) مجموعة غير متجانسة من الخلايا النخاعية غير الناضجة التي تتوسع بشكل غير طبيعي في السرطان وتمتلك نشاطا قويا مثبطا للمناعة. وفي الوقت نفسه ، بروتين الفئران الدقيقة المزدوجة 2 (MDM2)معروف على نطاق واسع لدوره المسرطن، لا سيما في تطور سرطان الثدي.

الهدف: هدفت هذه الدراسة إلى تقييم MDSCs و MDM2 كمؤشرات محتملة للتمييز بين المرضى الذين يعانون من BC قبل العلاج والأفراد الأصحاء.

الطرق: شملت الدراسة الإجمالية 25 شخصا سليما للمقارنة مع 65 عينة من مرضى سرطان الثدي ، تتراوح أعمارهم بين 27 و 76 عاما. تم تشخيص جميع مرضى سرطان الثدي كحالة جديدة من قبل طبيب أورام في مستشفى الأورام التعليمي (بغداد، العراق). استخدمت تقنية (ELISA) لقياس المستويات الإجمالية لكل من MDSCs و MDM2 في الدم المحيطي.

النتائج: أشارت الحقائق العلمية للدراسة الحالية إلى أن مستوى MDSCs كان ذا أهمية كبيرة لدى مرضى سرطان الثدي قبل تلقي العلاج ($P \leq 0.001$) مقارنة بالضوابط الصحية. علاوة على ذلك، كان MDM2 أيضا أكثر ارتفاعا في مرضى سرطان الثدي منه في الضوابط الأصحاء ($P \leq 0.05$).

الخلاصة: في الختام، وجدت الدراسة فرقا كبيرا في MDSCS في المصل لدى مرضى BC قبل العلاج مقارنة بالشاهد. لاحظنا أيضا مستويات مرتفعة من MDM2 في مرضى BC، والتي نسبناها إلى ظهور السرطان.

الكلمات الرئيسية: الخلايا المثبطة المشتقة من النخاع، بروتين الفئران الدقيقة المزدوجة 2، سرطان الثدي

ملاحظة: هل البحث مستل من رسالة ماجستير او اطروحة دكتوراه؟ نعم: ✓ كلا