

البحث الخامس

الأهمية الإكلينيكية الباثولوجية للخلايا الجذعية بالسرطان سي دي ٤٤ و سي دي ١٣٣ (CD44 و CD133) والبيئة الإلتهابية المستصغرة للورم (الخلايا الضامة المصاحبة للورم والخلايا البدنية) لحالات سرطان القولون والمستقيم: دراسة مناعية هيستوكيميائية

Clinicopathological significance of Cancer stem cell (CD 44 and CD133) and inflammatory microenvironment (TAMs and mast cells) in colorectal cancer: Immunohistochemical study

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بحث جماعى

Abstract

Background: many studies have investigated the role of CSCs and also some studies referred to a role of the inflammatory microenvironment in CRC especially their relation to prognosis. We aimed by this work to examine the possible clinicopathological significance of CSCs and the inflammatory microenvironment in CRC using immunohistochemistry. So we selected 2 cancer stem cell markers; CD44 and CD133 and 2 inflammatory microenvironment members; TAMs and mast cells.

Materials and Methods: 60 cases of radical resection specimens of CRC collected as fresh samples and paraffin blocks from archives used for CD133, CD44, CD68 and CD117 immunohistochemistry.

Results: There was a statistically significant relation for CD133 expression and the (T) status of the tumor ($P=0.012$), while CD44 expression was significantly related to both tumor size ($P=0.006$) and Dukes classification ($P=0.019$). The positivity of both markers was significantly related to metastasis (M) ($P=0.023$). The expression of CD68 was statistically significant in relation to the tumor size ($P=0.018$), the T ($P=0.007$) and the N ($P=0.049$) status of the tumor. The expression of CD117 was also significantly related to the T status of the tumor ($P=0.011$) and it was also significantly related to M status ($P=0.003$) and Dukes classification of the tumor ($P=0.001$). The CRC cases with infiltration of moderate and strong intensity for both markers had a strongly significant relation to the T status ($P=0.027$), the metastasis (M) ($P=0.034$) and the presence of lymphovascular invasion ($P=0.011$). The expression of CD133 was significantly related to that of CD44 ($P=0.013$) and to that of CD117 ($P=0.013$) but was not related to the expression of CD68 ($P=0.92$). CD44 expression was significantly related to CD68 expression ($P=0.013$) but not related to the expression of CD117 ($P=0.137$).

Conclusion: The significant relations between our studied markers and the clinicopathological features of CRC may refer to a role played by both CSCs (CD133 and CD44) and inflammatory microenvironment (TAMs and mast cells) in CRC progression, particularly distant metastasis, also the significant relation we found between different studied markers may also refer to a possible cross-talk between them which needs further studies for confirmation.