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The role of tight junction proteins in oncogenesis

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Claudins are tight junction proteins which along with adherens junctions and desmosomes form cellular sheets. Tight junctions (TJs) are critical for sealing the cellular sheets thereby controlling paracellular ion flux. The high degree of cellular organization typically observed in normally differentiated tissues is often lost in cancer. Loss of epithelial integrity with changing claudins levels and resultant increased para-cellular leakage plays a critical role in providing a space for tumor cell mobility and increased nutrients' supply for tumor cells underneath. Several studies analyzing the roles of tight junctions in oncogenesis have revealed either downregulation or upregulations of claudins expression. Claudins, whether upregulated or down regulated are proving to be a vital piece of the puzzle of oncogenesis. A prime example of the aforementioned role of claudins in carcinogenesis and metastases is claudin-1. It is one of the genes strongly regulated by B-catenin. The latter gene is known for its role in maintaining cell-to-cell adhesion and most importantly in mediating the oncogenic Wnt/B-catenin transduction pathway. Claudin-1 along with claudin-3 and claudin-5 are shown to promote pro-MMP2 whereby claudins recruit MMPs on the cell surface to achieve elevated focal concentrations and eventual activations of pro-MMP2. Claudin-1 expression is frequently altered in several cancers including upregulation and downregulation. In Colorectal cancer, claudin-1 plays an important role in oncogenesis and has been proven to have a prognostic value. In this talk, we present the role of claudin-1 as a biomarker of oncogenesis invasion and metastases in colorectal cancer. We also address some of the controversies surrounding the use of claudin-1 as a biomarker of G.I. tumors. Lastly, we summarize our recommendations for the type of cases where claudins should be utilized as biomarkers of oncogenesis and invasion.

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Resistin is not an appropriate biochemical marker to predict severity of acute pancreatitis: A case-controlled study

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Aim: To assess levels of serum resistin upon hospital admission as a predictor of acute pancreatitis (AP) severity.

Methods: AP is both a common and serious disease, with severe cases resulting in a high mortality rate. Several predictive inflammatory markers have been used clinically to assess severity. This prospective study collected data from 102 patients who were diagnosed with an initial acute biliary pancreatitis between March 2010 and February 2013. Measurements of body mass index (BMI) and waist circumference (WC) were obtained and serum resistin levels were analyzed at the time of hospital admission using enzyme-linked immunosorbent assay. Additionally, resistin levels were measured from a control group after matching gender, BMI and age.

Results: A total of 102 patients (60 females and 42 males) were diagnosed with acute gallstone-induced pancreatitis. The mean age was 45 years, and mean BMI value was 30.5 kg/m2 (Obese, class I). Twenty-two patients (21.6%) had severe AP, while eighty eight patients had mild pancreatitis (78.4%). Our results showed that BMI significantly correlated with pancreatitis severity (P=0.007). Serum resistin did not correlate with BMI, weight or WC. Furthermore, serum resistin was significantly higher in patients with AP compared to control subjects (P<0.0001). The mean resistin values upon admission were 17.5 ng/mL in the severe acute biliary pancreatitis group and 16.82 ng/mL in the mild AP group (P=0.188), indicating that resistin is not an appropriate predictive marker of clinical severity.

Conclusion: We demonstrate that obesity is a risk factor for developing severe AP. Further, although there is a correlation between serum resistin levels and AP at the time of hospital admission, resistin does not adequately serve as a predictive marker of clinical severity.

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