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ARTICLE

Associations between Body Mass Index and Breast Cancer Markers

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ABSTRACT

Body mass index (BMI) and breast cancer biomarkers (BCBs) such as resistin, leptin adiponectin, monocyte chemotactant protein-1 (MCP-1) and homeostasis model assessment of insulin resistance (HOMA-IR) are highly associated with each other. The report has focused the inter-relationship between BMI and BCBs based on probabilistic modeling. It has been shown that mean BMI is directly associated with leptin (P<0.0001) and MCP-1 (P=0.0002), while it is inversely associated with adiponectin (P=0.0003), HOMA-IR (P<0.0001), and it is higher for healthy women (P=0.0116) than breast cancer women. In addition, variance of BMI is inversely associated with resistin (P=0.1450). On the other hand, mean MCP-1 is directly associated with BMI (P<0.0001). Mean resistin is directly associated with the interaction effect of BMI and leptin (BMI*Leptin) (P=0.0415), while its variance is directly associated with BMI (P=0.0942), and it is inversely associated with BMI*Adiponectin (P=0.1518). Leptin is directly associated with BMI (P<0.0001). Also adiponectin is inversely associated with BMI (P<0.0001), BMI*Leptin (P=0.0415), while its variance is directly associated with BMI (P=0.0942), and it is inversely associated with BMI*Adiponectin (P=0.1518). Leptin is directly associated with BMI (P<0.0001). Also adiponectin is inversely associated with BMI (P<0.0001), BMI*Leptin (P=0.1729), while it is directly associated with Age*BMI (P=0.0017) and BMI*Resistin (P=0.0615). It can be concluded that BMI and BCBs are strongly associated with each other. Care should be taken on BMI for breast cancer women.

1. Introduction

BMI has been a fundamental psychosocial issue among human beings for millennia. It is a composite measure of height and weight, which is defined as BMI= Weight(kg) / Height(m²). An individual fatness index is measured by BMI. It is considered as the risk factor for the growth of many diseases such as breast cancer, diabetes, cardiovascular diseases, etc. [1-5]. In general, BMI less than or equal to 25 kg/m² is treated as the normal, otherwise it is considered as obesity. Excess weight has been associated with a variety of cancers such as postmenopausal breast, colon, renal, esophageal, endometrial etc. The International Research Agency on Cancer has predicted that BMI causes 9% of breast cancer, 25% of renal cancer, 11% of colon cancer, 39% of endometrial cancer, and 37% of esophageal cancer[6]. Calle et al. [7] pointed that BMI was associated with a greater risk of death from 14 cancers such as esophagus, liver, colon and rectum, gallbladder, kidney, pancreas, non-Hodgkin

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lymphoma, stomach, multiple myeloma, breast, prostate, cervix, uterus, and ovary, and it was predicted that BMI may account for 20% of all cancer deaths in women and 14% in men[7].

BMI is a well-known risk factor for postmenopausal breast cancer, whereas debatable outcomes have been presented in premenopausal women[8-10]. A large sample meta-analysis reported an inverse association between BMI and the chance of premenopausal breast cancer[11]. Recently, two large prevention data studies have shown that premenopausal women with higher BMI are at increased risk for growing breast cancer[12,13].

The relationships between BMI and BCBs are still contradictable[8,9,11,14-16]. These can be studied based on statistical modeling of BMI on the BCBs such as leptin, resistin, adiponectin and MCP-1 and other explanatory variables. Again, each BCB should be modeled on BMI and other explanatory variables. The current report focuses the associations between BMI and BCBs based on modeling of BMI, MCP-1, adiponection, resistin, and leptin. For a data set given in[17,18], these models have been studied in[19-23]. From these models, the relationships between BMI and BCBs are reported in the current article.

2. Materials and Methods

2.1 Materials

The data set can be found in the UCI Machine Learning Repository, and its detailed description is given in[17,18]. For immediate using of the covariates in the report, these are restated as BMI (kg/m²), Age, HOMA-IR, Insulin (μU/mL), Glucose (mg/dL), Adiponectin (μg/mL), Resistin (ng/mL), MCP-1, Leptin(ng/mL), Types of Patient (TYOP) (1=healthy controls; 2=patients).

2.2 Statistical Methods

The considered data set given in[17,18] is a multivariate data set. The interested responses are BMI, resistin, MCP-1, adiponection, leptin which are all positive continuous heterogeneous and non-normally distributed. These are required to be modeled herein. These can be appropriately modeled using joint generalized linear models (JGLMs) adopting both the Log-normal and Gamma distributions, which are clearly given in[24-26]. Both the JGLMs under the Log-normal and Gamma distributions are very shortly given in recent articles[22-23], which are not reproduced herein. For more discussions on JGLMs, readers can visit[24,25].

2.3 Statistical and Graphical Analysis

For ready reference, first we examine BMI model on age, insulin, glucose, and BCBs. The detailed analysis is given by Das et al.[19]. It is mentioned herein that BMI and BCBs such as MCP-1, resistin, leptin and adiponection can be modeled adopting JGLMs under both the Log-normal & Gamma distributions[24-26]. Log-normal JGLMs fit of BMI is better than the Gamma fit, which is presented in Table 1, and its fitting diagnostic is revealed in Figure 1. Figure 1(a) displays the absolute residuals plot against the predicted BMI values, which is closely a flat straight line, implying that variance is constant with the running means. Figure 1(b) represents the normal probability plot of the fitted BMI mean Log-normal model in Table 1. No lack of fit is identified in both the figures. So, Log-normal fitted BMI model (Table 1) is an approximate form of its true model. Fitted BMI mean & dispersion models are as follows:

Fitted Log-normal BMI mean (\(Z\)) model (from Table 1) is

\[ \hat{Z} = \log(\text{BMI}) = 3.0370 - 0.0421 \text{HOMA-IR} + 0.0015 \text{Glucose} + 0.0123 \text{Insulin} - 0.0068 \text{Adiponectin} + 0.0001 \text{MCP-1} + 0.0053 \text{Leptin} - 0.0708 \text{TYOP}, \]

and the BMI fitted Log-normal variance (\(\hat{\sigma}^2\)) model is

\[ \hat{\sigma}^2 = \exp(-4.445 - 0.018 \text{Insulin} - 0.019 \text{Resistin} + 0.015 \text{Age}). \]

Figure 1. For the joint Log-normal BMI fitted models (Table 1), the (a) absolute residuals plot against the fitted values, and (b) the normal probability plot for the mean BMI model.

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Biomarker MCP-1 analysis is given by Kim et al. [20], and for ready reference it is reproduced in Table 2. Fitted MCP-1 mean & dispersion models are as follows:

MCP-1 Gamma fitted mean ($\hat{\mu}$) model (from Table 2) is

$$\hat{\mu} = \exp(5.1791 - 0.0265 \text{Insulin} + 0.0455 \text{BMI} - 0.0192 \text{Leptin} + 0.0220 \text{Resistin} + 0.0009 \text{Insulin}\ast \text{Leptin})$$

and MCP-1 Gamma fitted dispersion ($\hat{\sigma}^2$) model (from Table 2) is

$$\hat{\sigma}^2 = \exp(0.7374 - 0.0868 \text{Insulin} - 0.0293 \text{Age} + 0.0051 \text{Age}\ast \text{Insulin} + 0.0053 \text{Glucose} - 0.8286 \text{HOMA-IR} - 0.0997 \text{Leptin} - 0.0405 \text{Resistin} + 0.0007 \text{Glucose}\ast \text{Leptin} + 0.0010 \text{Leptin}\ast \text{Resistin})$$

Biomarker adiponectin analysis is given by Das and Lee [21], and for ready reference it is reproduced in Table 3. Fitted adiponectin mean & dispersion models are as follows.

Adiponectin Gamma fitted mean ($\hat{\mu}$) model (from Table 3) is

$$\hat{\mu} = \exp(6.7778 - 0.1475 \text{BMI} - 0.0617 \text{Age} + 0.0020 \text{Age}\ast \text{BMI} - 0.0662 \text{Resistin} + 0.0282 \text{Leptin} + 0.0018 \text{BMI}\ast \text{Resistin} - 0.0008 \text{BMI}\ast \text{Leptin})$$

and Adiponectin Gamma fitted variance ($\hat{\sigma}^2$) model (from Table 3) is

$$\hat{\sigma}^2 = \exp(-2.318 + 0.017 \text{Age})$$
Table 3. Results for mean and dispersion models for Adiponectin from Log-Normal and Gamma fit

<table>
<thead>
<tr>
<th>Model</th>
<th>Covariate</th>
<th>Log-normal fit</th>
<th>Gamma fit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Estimate</td>
<td>S.E.</td>
</tr>
<tr>
<td>Mean</td>
<td>Constant</td>
<td>6.8667</td>
<td>1.0012</td>
</tr>
<tr>
<td></td>
<td>Age (x1)</td>
<td>-0.0627</td>
<td>0.0178</td>
</tr>
<tr>
<td></td>
<td>BMI (x2)</td>
<td>-0.1512</td>
<td>0.0367</td>
</tr>
<tr>
<td></td>
<td>AGE*BMI</td>
<td>0.0020</td>
<td>0.0006</td>
</tr>
<tr>
<td></td>
<td>Leptin (x6)</td>
<td>0.0217</td>
<td>0.0184</td>
</tr>
<tr>
<td></td>
<td>Resistin (x8)</td>
<td>-0.0701</td>
<td>0.0296</td>
</tr>
<tr>
<td></td>
<td>BMI*Resistin</td>
<td>0.0020</td>
<td>0.0010</td>
</tr>
<tr>
<td></td>
<td>BMI*Leptin</td>
<td>-0.0006</td>
<td>0.0006</td>
</tr>
<tr>
<td>Dispersion</td>
<td>Constant</td>
<td>-2.325</td>
<td>0.6674</td>
</tr>
<tr>
<td></td>
<td>Age (x1)</td>
<td>0.019</td>
<td>0.0114</td>
</tr>
</tbody>
</table>

Biomarker resistin analysis is given by Das and Lee\[22\], and for ready reference it is reproduced in Table 4. Fitted resistin mean & dispersion models are as follows:

Resistin Gamma fitted mean ( $\hat{\mu}$ ) model (from Table 4) is

$$\hat{\mu} = \exp(1.6651 - 0.0306 \text{Leptin} - 0.0052 \text{Age} + 0.0888 \text{Adiponectin} + 0.5421 \text{TYOP} - 0.1087 \text{HOMA-IR} + 0.0007 \text{MCP-1} + 0.0015 \text{Age}\text{HOMA-IR} - 0.0028 \text{BMI} + 0.0068 \text{Glucose} + 0.0014 \text{BMI}\text{Leptin} - 0.0010 \text{Glucose}\text{Adiponectin} - 0.0009 \text{Leptin}\text{Adiponectin})$$

and Resistin Gamma fitted variance ($\hat{\sigma}^2$) model (from Table 4) is

$$\hat{\sigma}^2 = \exp(- 4.8464 + 0.7971 \text{TYOP} + 0.1090 \text{BMI} + 0.0129 \text{Leptin} + 0.1885 \text{Adiponectin} - 0.0083 \text{BMI}\text{Adiponectin})$$

Table 4. Results for mean & dispersion models for Resistin from Log-Normal and Gamma fit

<table>
<thead>
<tr>
<th>Model</th>
<th>Covariate</th>
<th>Gamma fit</th>
<th>Log-normal fit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Estimate</td>
<td>s.e.</td>
</tr>
<tr>
<td>Mean</td>
<td>Constant</td>
<td>1.6651</td>
<td>0.7909</td>
</tr>
<tr>
<td></td>
<td>Age (x1)</td>
<td>-0.0052</td>
<td>0.0033</td>
</tr>
<tr>
<td></td>
<td>Leptin (x6)</td>
<td>-0.0306</td>
<td>0.0226</td>
</tr>
<tr>
<td></td>
<td>Adiponectin (x8)</td>
<td>0.0007</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>MCP-1 (x9)</td>
<td>0.5421</td>
<td>0.1084</td>
</tr>
<tr>
<td></td>
<td>Patient’s typ (Fx10)</td>
<td>-0.1087</td>
<td>0.0593</td>
</tr>
<tr>
<td></td>
<td>HOMA-IR (x5)</td>
<td>0.0015</td>
<td>0.0009</td>
</tr>
<tr>
<td></td>
<td>Adiponectin (x7)</td>
<td>-0.0028</td>
<td>0.0175</td>
</tr>
<tr>
<td></td>
<td>BMI (x2)</td>
<td>0.1090</td>
<td>0.0645</td>
</tr>
<tr>
<td></td>
<td>Leptin*BMI</td>
<td>0.0014</td>
<td>0.0007</td>
</tr>
<tr>
<td></td>
<td>Glucose (x3)</td>
<td>0.0068</td>
<td>0.0062</td>
</tr>
<tr>
<td></td>
<td>Adiponectin*Glucose</td>
<td>-0.0010</td>
<td>0.0006</td>
</tr>
<tr>
<td></td>
<td>Leptin*Adiponectin</td>
<td>-0.0009</td>
<td>0.0005</td>
</tr>
<tr>
<td>Dispersion</td>
<td>Constant</td>
<td>-4.8464</td>
<td>1.7259</td>
</tr>
<tr>
<td></td>
<td>Leptin (x6)</td>
<td>0.0129</td>
<td>0.0091</td>
</tr>
<tr>
<td></td>
<td>Patient’s typ (Fx10)</td>
<td>0.7971</td>
<td>0.3097</td>
</tr>
<tr>
<td></td>
<td>BMI (x2)</td>
<td>0.1090</td>
<td>0.0645</td>
</tr>
<tr>
<td></td>
<td>Adiponectin (x7)</td>
<td>0.1885</td>
<td>0.1468</td>
</tr>
<tr>
<td></td>
<td>BMI*Adiponectin</td>
<td>-0.0083</td>
<td>0.0058</td>
</tr>
</tbody>
</table>

AIC

729.369

731.2
Biomarker leptin analysis is given by Das and Lee [23], and for ready reference it is reproduced in Table 5. Fitted leptin mean & dispersion models are as follows:

Leptin Gamma fitted mean ( \( \hat{\mu} \) ) model (from Table 5) is

\[
\hat{\mu} = \exp(-0.227 + 0.092 \text{BMI} + 0.006 \text{Glucose} + 0.010 \text{Insulin} + 0.025 \text{Adiponectin} + 0.016 \text{Resistin} - 0.001 \text{MCP.1} - 0.001 \text{Adiponectin*Resistin}),
\]

and Leptin Gamma fitted variance ( \( \hat{\sigma}^2 \) ) model (from Table 5) is

\[
\hat{\sigma}^2 = \exp(-3.809 + 0.042 \text{Age} + 0.153 \text{Resistin} - 0.003 \text{Age*Resistin}).
\]

Table 5. Associations of leptin with BMI, diabetes, age and breast cancer biomarkers

<table>
<thead>
<tr>
<th>Response</th>
<th>Associated with</th>
<th>Types of association</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin mean</td>
<td>BMI (x2)</td>
<td>Directly</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>Glucose (x3)</td>
<td>Directly</td>
<td>0.0135</td>
</tr>
<tr>
<td></td>
<td>Insulin (x4)</td>
<td>Directly</td>
<td>0.0557</td>
</tr>
<tr>
<td></td>
<td>Adiponectin (x7)</td>
<td>Directly</td>
<td>0.0110</td>
</tr>
<tr>
<td></td>
<td>Resistin (x8)</td>
<td>Directly</td>
<td>0.0073</td>
</tr>
<tr>
<td></td>
<td>MCP-1 (x9)</td>
<td>Inversely</td>
<td>0.0330</td>
</tr>
<tr>
<td></td>
<td>Adiponectin*Resistin</td>
<td>Inversely</td>
<td>0.0966</td>
</tr>
<tr>
<td>Leptin variance</td>
<td>Age (x1)</td>
<td>Directly</td>
<td>0.0034</td>
</tr>
<tr>
<td></td>
<td>Resistin (x8)</td>
<td>Directly</td>
<td>0.0028</td>
</tr>
<tr>
<td></td>
<td>Age*Resistin</td>
<td>Inversely</td>
<td>0.0009</td>
</tr>
</tbody>
</table>

3. Results

In Table 1, it is shown that mean BMI is directly associated with leptin (P<0.0001) and MCP-1 (P=0.0002), while it is inversely associated with adiponectin (P=0.0003), HOMA-IR (P<0.0001), and it is higher for healthy women (P=0.0116) than breast cancer women. In addition, variance of BMI is inversely associated with resistin (P=0.1450). On the other hand, from Table 2, mean MCP-1 is directly associated with BMI (P<0.0001). In Table 3, it is shown that mean adiponectin is inversely associated with BMI (P<0.0001), BMI*Leptin (P=0.1729), while it is directly associated with Age*BMI (P=0.0017) and BMI*Resistin (P=0.0615). From Table 4, it is noted that mean resistin is directly associated with BMI*Leptin (P=0.0415), while its variance is directly associated with BMI (P=0.0942), and it is inversely associated with BMI*Adiponectin (P=0.1518). In Table 5, it is shown that mean leptin is directly associated with BMI (P<0.0001).

4. Discussion

The summarized analyses of BMI, MCP-1, adiponectin, resistin and lepin are given in Tables (1-5). From Table 1, mean BMI is directly associated with leptin (P=0.0001), or MCP-1 (P=0.0002), concluding that it increases as leptin, or MCP-1 rises. In addition, it is inversely associated with adiponectin (P=0.0003), or HOMA-IR (P=0.0001), interpreting that it increases as adiponectin, or HOMA-IR decreases. Mean BMI is inversely associated with patient types (P=0.0116), indicating that BMI is higher for healthy women than breast cancer women. Variance of BMI is partially inversely associated with resistin (P=0.1450), interpreting that BMI variance rises as resistin level decreases. It is noted that partially significant effect is treated as confounder in epidemiology.

From Table 2, it is observed that MCP-1 is directly associated with BMI (P<0.0001), indicating that it increases as BMI increases. This is also observed from the BMI model as stated above. From Table 3, it is noted that mean adiponectin is inversely associated with BMI (P<0.0001), indicating that it decreases as BMI rises. This is also observed from BMI model. Mean adiponectin is directly associated with BMI*Resistin (P=0.0615), concluding that it rises as the interaction effect BMI*Resistin increases. In addition, mean adiponectin is inversely associated with BMI*Leptin (P=0.1729), indicating that it decreases as BMI*Leptin rises. Moreover, mean adiponectin is directly associated with Age*BMI (P=0.0017), concluding that it rises as the interaction effect Age*BMI increases. This implies that overweight women at older ages have higher level of adiponectin. From Table 4, mean resistin is directly associated with the interaction effect of BMI*Leptin (P=0.0415), concluding that it rises as interaction effect of BMI*Leptin increases. Variance of resistin is directly associated with BMI (P=0.0942), interpreting that it in-

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creases as BMI increases. Variance of resistin is inversely associated with BMI*Adiponectin (P=0.1518), indicating that it rises as BMI*Adiponectin decreases. From Table 5, mean leptin is directly associated with BMI (P<0.0001), indicating that it rises as BMI rises. This is also observed in BMI model. All the above summarized associations between BMI and BCBs are displayed in Table 6.

**Table 6. Associations between BMI & breast cancer biomarkers**

<table>
<thead>
<tr>
<th>Model</th>
<th>Response</th>
<th>Associated with</th>
<th>Association types</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>BMI (x2)</td>
<td>Leptin (x6)</td>
<td>Directly</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adiponectin (x7)</td>
<td>Negative</td>
<td>0.0003</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MAC-1 (x9)</td>
<td>Directly</td>
<td>0.0002</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HOMA-IR (x5)</td>
<td>Inversely</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient’s type</td>
<td>Inversely</td>
<td>0.0116</td>
</tr>
<tr>
<td>Dispersion</td>
<td>Resistin (x8)</td>
<td>BMI (x2)</td>
<td>Directly</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mean</td>
<td>MCP-1 (x9)</td>
<td>BMI (x2)</td>
<td>Directly</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mean</td>
<td>Resistin (x8)</td>
<td>BMI (x2)</td>
<td>Directly</td>
<td>0.0415</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BMI (x2)</td>
<td>Inversely</td>
<td>0.0942</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BMI*Adiponectin</td>
<td>Inversely</td>
<td>0.1518</td>
</tr>
<tr>
<td>Mean</td>
<td>Leptin (x6)</td>
<td>BMI (x2)</td>
<td>Directly</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age*BMI</td>
<td>Directly</td>
<td>0.0017</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BMI*Resistin</td>
<td>Directly</td>
<td>0.0615</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BMI*Leptin</td>
<td>Inversely</td>
<td>0.1729</td>
</tr>
</tbody>
</table>

The present derived associations between BMI and BCBs are little compared with the previous findings as the earlier research articles have not considered all these BCBs along with BMI. In addition, the earlier articles have not considered probabilistic joint modeling to derive these associations. All these reported results herein are only based on the articles [19-23].

**5. Conclusions**

The report examines the associations between BMI and BCBs such as MCP-1, leptin, adiponectin and resistin. These associations are reported herein considering the models of BMI and each BCB. From these models, it can be concluded that BMI and BCBs are associated in both mean and variance models. BMI increases as leptin, or MCP-1 increases, or adiponectin, or resistin, or HOMA-IR decreases. Many interaction effects such as BMI*Leptin and BMI*Adiponectin are associated with resistin, while BMI*Resistin, BMI*Leptin and Age*BMI are associated with adiponectin. The report gives clear associations between BMI and BCBs which are very helpful to the researchers and medical practitioners. Medical practitioners and women should care on BMI along with breast cancer biomarkers.

**Conflict of Interest**

The authors confirm that this article content has no conflict of interest.

**References**


ARTICLE

Application of AI Technology in Tumor medical Imaging

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2. Beijing Anxun Dasheng Technology Co., Ltd., Beijing, 100000, China

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Oncology
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1. Introduction

The term “artificial intelligence” was first introduced and adopted at the Dartmouth College Symposium in 1956, and since the beginning of the 21st century, the research and application of AI in medical imaging has developed rapidly, especially in tumor imaging, and there have been more clinical research and applications in tumor detection, qualitative diagnosis, automatic structured reporting, tumor extraction, and tumor radiation target organ sketching.

2. The Classic AI Algorithm and Its Application in Medical Imaging

The current classical AI algorithm can be summarized as a collection theory classification, based on probability statistical classification, graph-based classification, spatial geometric classification, based on cross-disciplinary classification, and so on, each classification contains some commonly used algorithms, such as the artificial intelligence algorithm based on probability statistics has simple Bayes, logic regression, maximum entropy, etc. The deep learning algorithm which simulates human neural network is one of the algorithms belonging to the interdisciplinary classification.

3. Application of AI Technology in Medical Imaging

According to statistics, more than 90% of the information stored in hospitals comes from medical imaging equipment; AI in medical imaging equipment can make low-dose CT, PET images by reconstructing high-dose images, reduce radiation risk; AI has also made some substantial...
developments in the diagnosis of ultrasound imaging and the analysis of pathological slicing. In terms of intelligent services, Medical Imaging AI provides intelligent services ranging from assisted detection, assisted diagnostics, accurate diagnosis, quantitative follow-up to precision therapy. Image classification, target detection, object segmentation and image generation are key issues in AI image processing, which are particularly important in medical imaging analysis and processing. Deep learning is a very wide range of algorithms, common types include regional convolutional neural network algorithms, spatial pyramid pooling algorithms, fast R-CNN algorithms, Faster R-CNN algorithms, YOLO algorithms, etc. In addition, more and more target detection algorithms are now abandoning the design of anchorbox. One of the most innovative target detection algorithms is called detr. This algorithm extracts features from the input picture, adds position coding, puts it into the transformer, and directly gets the target location, size, and category. This novel design completely abandons post-processing, reduces the need for calculation of target detection, and improves the efficiency of target detection.

4. The Application of AI Technology in Tumor Imaging

4.1 Application of Artificial Intelligence in Central Nervous System Tumors

The purpose of brain tumor segmentation is to identify the location of the tumor and the area where the tumor is immersed outward, i.e. to identify tumor tissue, necrotic tissue, and surrounding edema. Soltaninejad et al. [3], using machine learning algorithms based on 3D Super voxel, analyzed MR images from 2 clinical databases (11 samples and 30 cases, respectively) with Dice coefficient of 0.84, 0.89, which have high accuracy. On the basis of identifying and dividing brain tumors, artificial intelligence algorithms can also be used to grade gliomas, identify glioma recurrence and post-treatment necrosis, prognosis prediction of gliomas, and predict the prognosis of gliomas.

4.2 Applications of Human Intelligence in Lung Cancer

Artificial intelligence has shown good clinical application value in lung cancer detection and staging. Masood et al. used deep learning algorithm to analyze chest CT images from multiple databases, and the average accuracy of diagnosis reached 84.58%, and the accuracy of lung cancer t1-T4 staging reached 77.89% ~ 90.14%. Sun et al. conducted a comparative study on deep learning algorithm and traditional shallow learning algorithm. The data came from Lung Image Database Consortium (LIDC), which contained 13,668 chest CT images. The results showed that the AUC of deep learning was 0.899±0.018 and that of shallow learning was 0.848±0.026 [4]. Artificial intelligence identification of pulmonary nodules is also a major research direction for domestic companies.

4.3 Application of Artificial Intelligence in Breast Cancer

In 2016, Quellec et al. proposed a new computer-aided detection and diagnosis system for mammography, which relies on the MIL (a weak supervised learning method) paradigm and uses only whole-image level labels. They first divide the breast adaptively into multiple regions, then extract and combine features of detected lesions from each region to classify mammograms as normal or abnormal. Becker et al. applied deep learning algorithm to classify 632 patients (550 benign and 82 malignant). The total time of computer classification of benign and malignant was 3.7s and the AUC could reach 0.84. The three doctors with high to low experience spent 28 minutes, 22 minutes and 25 minutes respectively, with an AUC of 0.79 ~ 0.88, indicating that artificial intelligence can achieve similar accuracy in the classification of benign and malignant breast cancer as imaging doctors, and can significantly improve work efficiency.

4.4 Application of Artificial Intelligence in Prostate Cancer

In western countries Prostate cancer is the first incidence of malignant tumors in male, in recent years, the incidence of China has also shown a clear upward trend. Wang et al. compared the diagnostic accuracy of deep learning algorithms and non-deep learning algorithms in prostate cancer and benign hyperplasia based on MR images, the AUC of Deep Learning AUC was 0.83 and the AUC of non-deep learning algorithm was only 0.70, that means it is better to use depth study to identify the performance.

4.5 Application of Artificial Intelligence in Bone Tumors

The quality of life of patients with spinal metastases is significantly reduced, so early detection has great significance. Wang et al. used deep learning algorithm to analyze the sagittal MR images of the spine of 26 patients with spinal metastasis. After combining the analysis results of the twin neuron network, the diagnostic sensitivity reached 90%.

In addition to the above-mentioned tumors, AI has also
been used in other tumors such as liver tumors.

5. Industrial Status of Medical Imaging Artificial Intelligence in China

In July 2017, China released the Development Plan for The Next Generation of Artificial Intelligence, which proposes the development of intelligent disease prediction, intelligent diagnosis, intelligent treatment mode and intelligent medical system. The establishment of medical imaging AI model often needs a large amount of data support, at this stage, each research team is prone to form data silos, data labeling consistency is low, gold standard is missing, and quality control, data security and privacy protection are also problems, there are significant differences in the understanding and implementation ability of various participants in medical imaging AI. From ethical point of view, the current research on medical imaging AI is still insufficient to pay attention to the questions of the patient’s right to know, privacy protection and other medical ethics issues, data security issues and the safety of patients' treatment.

Among the physicians and researchers in China, young and middle-aged physicians, senior doctors and radiologists are generally concerned about AI technology, and the researchers involved in AI research are mostly young students, preferring to be younger, which brings broad prospects for the development of the AI industry. In addition, China’s hospitals need to improve the current system of talent introduction, attract high-end talent in China’s medical imaging in the field of artificial intelligence innovation and development, is conducive to China’s medical imaging AI industry towards the direction of standardization and normalization.

References

REVIEW

Progress in Diagnosis and Treatment of Basal Cell Carcinoma

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ABSTRACT

Basal cell carcinoma is a common skin carcinogenesis that occurs in the epidermis and the basal layer of the skin. In general, basal cell carcinoma grows slowly, rarely metastasizes, but is locally invasive and destructive. The diagnosis is based on clinical manifestations, but the clinicopathological manifestations are different, and sometimes it is difficult to differentiate from pigmented nevus, malignant melanoma, etc. Therefore, skin biopsy is essential for the diagnosis and assessment of the risk of recurrence. There are many ways to treat basal cell carcinoma. This article reviews the diagnosis and treatment.

1. Introduction

Basal cell carcinoma is a common skin cancer, often occurs on sun-exposed skin, common parts such as face, neck, hand, etc. Most basal cell carcinoma grow slowly, they are locally invasive and can destroy surrounding tissues [1]. The main causes of the disease are ultraviolet radiation, prolonged exposure to arsenic-contaminated water, food and drugs, or increased risk factors for human immunodeficiency virus infection and immunosuppression [2].

2. Diagnosis

The diagnosis of basal cell carcinoma depends on clinical manifestation, dermoscopy and histology. Dermoscopy will improve the diagnostic accuracy of skin lesions [3]. However, skin biopsy is essential for the diagnosis and assessment of the risk of recurrence. Histopathologically, basal cell carcinoma is usually composed of uniformly proliferating basal cell-like cells with dense nuclei, relatively few cytoplasm and unclear boundaries [4]. In histopathology, nodular lesions are formed by the proliferation of large basal cells, forming huge tumor nests, surrounded by palisades, with irregular central arrangement [5]. Histopathologically, a sclerosing collagen stroma encapsulates a narrow cell chain and a small island of tumor cells. Fibroepithelial type usually occurs in the lower back, presenting as skin color or erythema soft papules or pedicle papular nodular lesions similar to fibroma or papilloma [6].

3. Treatment

Surgical resection is the standard treatment for basal cell carcinoma. The study of surgical margin may realize complete resection of tumor at different anatomical locations. It is reported that the 5-year cure rate of 4-5 mm surgical margin is 9.95% [7]. Electro-drying and curettage is the removal of the surface of the tumor with a blade or a curettage device, and the burning of the remaining substrate with an electroacupuncture to control bleeding and destroy residual tumor cells. This method is simple but can not fully demonstrate that the tumor has been removed.

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and is more suitable for basal cell carcinoma located in low-risk areas without invasive histopathological features. Cryosurgery liquid nitrogen acts directly on tumors, can form extracellular and intracellular ice crystals, destroy cell phospholipid membranes, and destroy peripheral tumor-associated blood vessels for therapeutic purposes, but can not be histologically confirmed. Cryosurgery can not be used as a first-line treatment for basal cell carcinoma, especially for tumors with a high risk of recurrence. Topical imiquimod cream is an immune response regulator that induces cytokine production, stimulates cellular immunity, and promotes apoptosis by bypassing anti-apoptotic mechanisms of tumor cells. The curative effect is not as good as the local operation, the systemic side effect is many, usually uses in the low-risk position superficial basal cell carcinoma treatment. Local fluorouracil by inhibiting thymidine synthase interference DNA synthesis, local application of 5% FU cream has been proved to be generally effective, local fluorouracil is usually used only for superficial basal cell carcinoma in non-critical anatomical area, while for nodular or invasive basal cell carcinoma, it is generally forbidden. Light energy therapy, because reactive oxygen species produced by photosensitizers selectively absorb light induces cytotoxicity in tumor cells and local inflammatory responses that may lead to tumor destruction, complete tumor resection depends on the concentration of photosensitizers in tumor cells and the ability of light focusing in related regions. The effect of radiotherapy is positive, but recurrent basal cell carcinoma after radiotherapy may exhibit greater aggressiveness, including second recurrence and distant metastasis. In addition, the evidence of long-term efficacy of radiotherapy is limited. Common side effects include chronic radiation dermatitis, permanent alopecia, dermal and subcutaneous fibrosis, necrosis, and secondary cutaneous malignancies. Therefore, radiotherapy is generally applicable to patients not suitable for surgery, especially in high-risk areas.

4. Conclusion

To sum up, preclinical and pathological assessment of recurrence risk is necessary to select the best treatment strategy. According to the location of the tumor, the pathological subtype, the patient’s condition, the beauty effect and the cost of treatment, different treatment methods can be used.

References

REVIEW
The Clinical Application Value of Susceptibility Weighted Imaging in the Central Nervous System

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1. Introduction

Susceptibility weighted imaging (SWI) is a non-invasive magnetic resonance imaging technique that takes advantage of the difference in magnetic properties of different tissues. It is based on a new long echo time, complete flow compensation, and three-dimensional gradient imaging sequence. Compared with the traditional T2* weighted sequence, it has the characteristics of three-dimensionality, high resolution, and high signal-to-noise ratio. The original SWI image is the Magnitude image and the Phase image obtained by scanning the T2* weighted echo sequence. The two can be analyzed separately or processed for image fusion. The Magnitude image contains most of the tissue contrast information, and the Phase image reflects the tissue contrast from the perspective of susceptibility, especially the tissues with large differences in magnetic susceptibility. These two images are obtained at the same time during the scanning process, always appearing in pairs, and the anatomical position corresponding to each pair of images is exactly the same.

SWI is very sensitive to display venous structure, blood metabolites (such as deoxyhemoglobin), iron deposition, calcification, etc. Even without the use of contrast agents, it can clearly show the vascular system and hemorrhagic components, so it is used in cerebrovascular diseases, brain Tumors, brain trauma, neurodegenerative diseases and other central nervous system diseases have high clinical application prospects and value. SWI has now become a key magnetic resonance imaging sequence for the diagnosis of central nervous system diseases, and is an effective supplement to traditional spin echo magnetic resonance imaging sequences. In recent years, quantitative magnetic susceptibility mapping (QSM) that can quantitatively analyze tissue magnetic susceptibility have emerged based on SWI [1]. This article will
analyze the clinical application value of SWI in the central nervous system from the following points.

2. Cerebrovascular Disease

Biological tissue generates a specific induced magnetic field under the action of an external magnetic field, which depends on the strength of the external magnetic field and the magnetic sensitivity of tissue molecules. Magnetic susceptibility can be measured by the magnetic susceptibility. The degree of magnetization of the reaction material under the action of an external magnetic field is a variable reflecting the organization. The oxygenation and deoxygenation conversion of hemoglobin is the basis of blood oxygen level-dependent imaging. Oxyhemoglobin is diamagnetic, deoxyhemoglobin has 4 unpaired electrons that are paramagnetic, and hemoglobin is a strong paramagnetic substance but has weak magnetic sensitivity, hemosiderin is a paramagnetic substance, deoxyhemoglobin and hemosiderin have strong magnetic sensitivity. Non-heme iron (often in the form of ferritin) is a paramagnetic substance and has a strong magnetic sensitivity effect. The magnetic sensitivity effect of calcification is weaker than that of iron. It is usually diamagnetic. On the SWI corrected phase image, the right-hand magnetic resonance imaging system (United States GE, Philips) showed calcification as a high signal, while the signal in the left-hand MRI system (Siemens, Germany) was opposite. Studies have shown that SWI also has significant advantages in the detection of calcium, and it is the only imaging method that can distinguish bleeding and calcification at the same time [5].

Cerebral microbleeds (CMBs), as a marker of small blood vessel diseases in the brain, is more sensitive than other sequences in displaying microbleeds in the brain, and it is currently the best imaging method for the diagnosis of CMBs [6]. Studies have shown that the prevalence of CMBs in healthy people is 5%, the prevalence of ischemic stroke patients is 22.9% -43.6%, and the incidence of hemorrhagic stroke patients and mixed stroke patients are respectively As high as 51.8% -82.5% and 41.2% -70.2% [5]. Cerebral microhemorrhage is mainly caused by the deposition of hemosiderin and mononuclear macrophages that have swallowed hemosiderin, which leads to local arteriole hyaloid degeneration and amyloid deposition [6]. Therefore, CMBs appear on SWI as clear edges and uniform properties. The diameter is between 2-5mm (the highest is no more than 10mm), and the shape is a round or oval low signal or lack of signal focus, without edema around it [7].

3. Brain Tumors

Conventional MRI examination sequence is difficult for the preoperative grading diagnosis of brain tumors, and the use of SWI to evaluate the angiogenesis and intratumor hemorrhage within the tumor is an effective supplement to the conventional MRI examination. In addition to distinguishing bleeding and calcification in brain tumors, SWI can clearly show the neovascular structure in the tumor and the blood oxygen status inside the tumor, which is of great significance for the staging and grading of the tumor [8]. At the same time, SWI is proposed to be used to evaluate the effects and differences of different anti-vascular gene drugs, as well as the diagnosis of curative effects after radiotherapy and chemotherapy, the progression of tumors can be studied longitudinally. Studies have shown that high-grade tumors generally have rapid growth and a large number of new pathological blood vessels and are prone to bleeding and necrosis; on the contrary, low-grade tumors often appear calcification due to slow growth and malnutrition [9]. Grading and scoring using intratumoral susceptibility signal intensity (ITSS) is considered to be related to its pathological grading and is an important basis for distinguishing benign and malignant tumors [10].

4. Brain Injury

Diffuse axonal injuries (DAI) is the most common type of brain trauma. It is caused by the shear force formed during the acceleration or deceleration of the head. Its common occurrence areas include the junction of the gray matter of the brain, The pressure part of the corpus callosum, the basal segment area and the dorsal side of the brain stem, etc., can detect punctate microhemorrhages in the deep white matter that cannot be displayed by CT or conventional MRI sequences. A study specifically for patients with mild traumatic brain injury compared the results of CT and SWI. The conclusion showed that the detection rate of SWI was as high as 97.83%, especially for brain contusion and laceration [11]. The detection rate was significantly higher than that of CT. It is more sensitive to traumatic brain injury and can accurately assess the patient’s prognosis.

5. Central Nervous System Degenerative Disease

Central nervous system degenerative disease is a chronic progressive neurodegenerative disease. Studies have shown that the increase of iron in the brain is related to the increase of age [12]. The abnormal iron metabolism is a significant feature of central nervous system degenerative diseases, including Parkinson’s disease (PD), Alzheimer’s disease (AD) and multiple sclerosis (MS), etc. SWI can accurately analyze the distribution range and amount of iron...
deposits in the brain. The detection provides a safe, simple and non-invasive inspection method, and is clearer than conventional sequences. The etiology of PD is due to abnormal iron metabolism resulting in increased iron deposits in the substantia nigra and striatum, leading to progressive loss of dopaminergic neurons, resulting in increased muscle tone, resting tremor, retardation of movement, postural balance disorders, and a series of clinical symptoms. Studies have shown that [13] possible signal changes related to iron deposition on SWI can accurately distinguish PD from various other forms of Parkinson’s disease. At the same time, the FA value of the substantia nigra and the size of the substantia nigra compact zone are helpful for the early diagnosis of PD. By measuring the signal intensity of related brain nuclei and calculating the phase value, it can evaluate brain iron deposition, which is the treatment and treatment of PD patients. Provide help for the subsequent effect evaluation. For AD, studies have shown that [14] the vast majority of AD patients are transformed from mild cognitive impairment (MCI). SWI can not only intervene in the conversion process from MCI to AD, but also monitor iron deposition during the conversion process. In order to guide clinical diagnosis and treatment in the early stage of dementia.

SWI is a relatively new imaging method, which has great development prospects in the diagnosis and treatment of central nervous system diseases. However, SWI still has some shortcomings. For example, SWI has a long scanning time and requires a long inspection site. The time limit is temporarily not suitable for tissues and organs with greater mobility. At the same time, parts with extremely large differences in magnetic susceptibility (air-tissue plane) such as the level of the skull base and sinuses will cause heavier artifacts and have “magnification effects”, which affect the observation and evaluation of lesions. These challenges require further research and improvement by technicians and scientific researchers. It is believed that with the development and progress of science and technology, SWI’s diagnosis of diseases will become deeper and deeper, and it will no longer be limited to the central nervous system.

References

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XI. Glossary of Publication Type

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