Enhancement profiles of benign lesion subgroups in MR-mammography: A computer assisted evaluation considering pharmacokinetic parameters

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Purpose

Dynamic contrast enhanced MR-mammography (MRM) has the highest sensitivity for detection of breast cancer. In order to differentiate benign from malignant lesions, morphologic and dynamic criteria are used. Computer Assisted Diagnosis (CAD) systems (or more general speaking: software analysis tools for dynamic enhancement pattern analysis) are increasingly used to assess dynamic enhancement features. Features of these systems include voxel by voxel calculation of parametric maps, colour-coding early and delayed enhancement characteristics. Lesions not passing a user-defined threshold for early enhancement are not colour-coded, a fact which can be used for differential diagnosis as malignant lesions are thought to enhance faster and stronger compared to benign lesions. Furthermore, the most suspect enhancing part of a lesion can be identified semiautomatically, avoiding multiple manual Region-of-Interest (ROI) measurements. However, although these features simplify enhancement pattern analysis, they do not provide information which isn’t available by means of manual or visual image interpretation. A feature unique to CAD systems is quantitative enhancement profile assessment. That means that all single curve types in an enhancing, threshold passing lesion can be analyzed at one step, providing volumetric data including subvolumes. Although this feature has been described in a general way, there is little data on enhancement patterns of different histological subgroups. This prospective investigation on previous acquired examinations was performed to investigate enhancement patterns of benign subgroups in MRM in order to identify whether certain subgroups are responsible for known overlaps of dynamic enhancement patterns with those of malignant lesions.
Methods and Materials

186 consecutive benign lesions from a time period of 22 months undergoing surgery after MR-mammography at our institution were enrolled in this study. Examinations after preoperative (neoadjuvant) chemotherapy were excluded from this work. All surgery specimen underwent histopathological workup by a board certified pathologist.

Imaging was performed on 1.5T clinical whole body scanners (Magnetom Symphony and Magnetom Sonata, Siemens Medical Solutions, Erlangen, Germany) using dedicated bilateral phased array breast coils. Dynamic scanning in 33 transverse sections using a spoiled Gradient Echo technique (FLASH 2D, GRAPPA factor 2, TR 113 ms, TE 5 ms, flip angle 90°, spatial resolution 1.1 x 0.9 x 3 mm) was performed once before and 7 times after contrast agent (CA) injection (automated injector, flow 3 ml/s, 0.1 mmol Gd-DTPA/kg bodyweight). Time of acquisition was 1 minute per measurement. Unenhanced images were subtracted from enhanced dynamic images for lesion detection. No motion correction algorithms were used.

For dynamic data analysis including pharmacokinetic mapping, a commercial available CAD system (iCAD, former CADSciences) was used. In all cases, a threshold of 33% for initial enhancement was applied. The second postcontrast measurement was defined as cut-off between early and delayed enhancement. Investigated dynamic enhancement parameters were: \textit{Initial enhancement} (IN) 2 min. after CA injection and \textit{Washout rate} (WR, defined as subtraction of relative enhancement at 7 min. from relative enhancement at 2 min.) of the most suspet curve (system defined as worst washin/washout combination), percentage of whole lesion \textit{Washout} (Wash\%\textsubscript{v}), \textit{Plateau} (Plat\%\textsubscript{v}) and \textit{Persistent} (Pers\%\textsubscript{v}) voxels (in percentages) as well as the pharmacokinetic parameters median lesion \textit{Permeability} (Perm) and median lesion \textit{Extracellular Volume Fraction} (EVF). All data analysis was performed by two trained observers blinded to histopathology on a consensus basis using the automatic 3D ROI method, activating analysis of all curve types (Washout, Plateau, Persistent). When this procedure was not possible due to strong background enhancement, the lesion was manually encircled on each slice in order to analyze a 3D volume.

CAD analysis is demonstrated in figure 1 and figure 2.

Besides descriptive statistics (mean, median, standard deviation), one-way analysis of variance (ANOVA) was performed for subgroups comparisons. All statistics in this study have exploratory character. A significance level of \#=5\% was applied.
Fig. 0: 26 y old patient with fibroadenoma, A: Contrast enhanced subtraction image, first minute after contrast agent injection showing a slowly enhancing large oval mass lesion with well defined borders and dark internal septations (arrow). B: Delayed contrast enhanced subtraction image 7 minutes after contrast agent injection. Persistent enhancement and stable internal morphology. C: Colour-coded map showing initial enhancement as colour brightness (faster enhancement is coded brighter) and delayed enhancement curve type as colour type (red: Washout, green: Plateau, blue: persistent signal increase). In this case slow persistent enhancement (dark blue) dominates, although some Washout voxels (red) can be depicted.

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Fig. 0: CAD analysis example, same patient as shown in figure n. A: 3D ROI marking the complete lesion (here, only one slice is shown). B: pie diagram showing curve type distribution. Approximately 75% of the lesion show slow and persistent (dark blue) enhancement with a minority of Washout voxels (red), indicative of a benign lesion. D demonstrates signal intensity time curves. Turquoise corresponds to the most suspect washin to washout combination (the curve referred to in this work), demonstrating Washout. Irregularity in the course of the curve suggests an artefact (small motion, signal variation). C, E: Distribution histograms of pharmacokinetic parameters Permeability (C) and Extracellular Volume Fraction (EVF), demonstrating homogeneous low permeability and relatively high EVF, typical for fibroadenoma.
Results

Initial enhancement significantly differed between benign subgroups with papilloma and inflammations showing fast enhancement compared to fibroadenoma and mastopathic proliferations ($P=0.004$). Benign inflammatory lesions showed highest Washout rate and Washout voxel percentage, followed by papilloma. Again, mastopathic proliferative disease and especially fibroadenoma showed lower mean values ($P<0.001$, respectively). Persistent voxel percentage distribution was inversely, but less distinct ($P=0.006$). Permeability and EVF also showed subgroup differences with a tendency for high Permeability in papilloma ($P=0.015$) and high EVF in fibroadenoma ($P=0.002$). Plateau voxel percentage did not differ significantly between benign lesion subgroups ($P=0.167$).

For exact results c.f. table (fig.1) and figures 2-8.
Table 1: Dynamic enhancement patterns in benign subgroups

<table>
<thead>
<tr>
<th></th>
<th>Initial*</th>
<th>Washout**</th>
<th>Wash (%)**</th>
<th>Plat (%)</th>
<th>Pers (%)*</th>
<th>Perm*</th>
<th>EVF*</th>
</tr>
</thead>
<tbody>
<tr>
<td>fibroadenoma (n=41)</td>
<td>Mean 81.7</td>
<td>-2.8</td>
<td>6.7</td>
<td>22.6</td>
<td>68.3</td>
<td>0.16</td>
<td>0.33</td>
</tr>
<tr>
<td></td>
<td>Median 58.6</td>
<td>0.0</td>
<td>1.0</td>
<td>22.1</td>
<td>74.7</td>
<td>0.14</td>
<td>0.31</td>
</tr>
<tr>
<td></td>
<td>Std.Dev. 76.0</td>
<td>27.9</td>
<td>12.1</td>
<td>15.9</td>
<td>25.5</td>
<td>0.09</td>
<td>0.12</td>
</tr>
<tr>
<td>mastopathic proliferation (n=76)</td>
<td>Mean 69.5</td>
<td>7.1</td>
<td>8.8</td>
<td>25.6</td>
<td>56.7</td>
<td>0.14</td>
<td>0.24</td>
</tr>
<tr>
<td></td>
<td>Median 62.0</td>
<td>0.4</td>
<td>2.9</td>
<td>25.0</td>
<td>60.8</td>
<td>0.11</td>
<td>0.23</td>
</tr>
<tr>
<td></td>
<td>Std.Dev. 57.8</td>
<td>25.8</td>
<td>14.0</td>
<td>13.8</td>
<td>26.8</td>
<td>0.08</td>
<td>0.10</td>
</tr>
<tr>
<td>papilloma (n=36)</td>
<td>Mean 113.9</td>
<td>21.2</td>
<td>23.5</td>
<td>26.8</td>
<td>49.7</td>
<td>0.26</td>
<td>0.28</td>
</tr>
<tr>
<td></td>
<td>Median 125.9</td>
<td>24.0</td>
<td>16.6</td>
<td>24.5</td>
<td>39.0</td>
<td>0.17</td>
<td>0.27</td>
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<tr>
<td></td>
<td>Std.Dev. 76.0</td>
<td>35.5</td>
<td>22.8</td>
<td>15.5</td>
<td>28.1</td>
<td>0.31</td>
<td>0.07</td>
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<tr>
<td>inflammation (n=12)</td>
<td>Mean 137.7</td>
<td>41.4</td>
<td>19.1</td>
<td>24.9</td>
<td>37.9</td>
<td>0.17</td>
<td>0.25</td>
</tr>
<tr>
<td></td>
<td>Median 116.3</td>
<td>34.9</td>
<td>20.3</td>
<td>26.9</td>
<td>39.4</td>
<td>0.18</td>
<td>0.27</td>
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<tr>
<td></td>
<td>Std.Dev. 117.3</td>
<td>45.3</td>
<td>14.8</td>
<td>15.4</td>
<td>24.2</td>
<td>0.13</td>
<td>0.17</td>
</tr>
<tr>
<td>other (n=21)</td>
<td>Mean 75.8</td>
<td>13.1</td>
<td>13.5</td>
<td>17.5</td>
<td>54.6</td>
<td>0.18</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>Median 56.5</td>
<td>0.0</td>
<td>1.2</td>
<td>14.3</td>
<td>55.2</td>
<td>0.10</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>Std.Dev. 74.4</td>
<td>35.2</td>
<td>23.5</td>
<td>14.5</td>
<td>34.4</td>
<td>0.21</td>
<td>0.16</td>
</tr>
</tbody>
</table>

*significant at the <0.05 level, **significant at the <0.001 level

Fig. 0: Table 1: Dynamic enhancement patterns in benign subgroups

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Fig. 0: Boxplots of initial enhancement in different benign subgroups.

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Fig. 0: Boxplots of Washourate in different benign subgroups.

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**Fig. 0:** Boxplots of persistent enhancement percentage in different benign subgroups.

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**Fig. 0:** Boxplots of Plateau enhancement percentage in different benign subgroups.

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**Fig. 0**: Boxplots of Washout enhancement percentage in different benign subgroups.

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**Fig. 0:** Boxplots of median Permeability in different benign subgroups.

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Fig. 0: Boxplots of median Extracellular Volume Fraction in different benign subgroups.

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Conclusion

Enhancement profiles of benign lesions differ significantly. Aggressive enhancement profiles especially in papilloma and inflammation explain known overlaps in dynamic enhancement patterns. Limitations of known dynamic and morphologic lesion descriptors in breast MRI are based on the assumption of rigid dichotomous interpretation of benign and malignant breast lesions. Understanding the heterogeneity of benign proliferative breast disease is a key to improve differential diagnosis of breast lesions, especially regarding specificity. In order to achieve maximum accuracy, further specific criteria associated with certain subgroups have to be implemented into clinical practice.
References


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