Histopathological correlates of the napkin-ring sign plaque in coronary CT angiography

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A B S T R A C T

Objective: The purpose of this study was to identify histologic characteristics of advanced coronary atherosclerotic plaques that are related with the detection of the napkin-ring sign (NRS) in coronary CT angiography (CCTA).

Methods: CCTA was performed in 7 human donor hearts. Histological slicing and stainings were performed in 1 mm increments of each major coronary artery. Histology was co-registered with the CT-data and classified according to the modified AHA classification.

Results: Advanced plaques (types IV–VI) were present in 139 (23%) of 611 cross sections. Of these 33 (24%) demonstrated an NRS in CCTA. NRS plaques were associated with greater non-core plaque area (median 10.2 vs. 6.4 mm², p < 0.01) and larger vessel area (median 17.1 vs. 13.0 mm², p < 0.01). The area of the necrotic/lipid core was larger in plaques with NRS (median 1.1 vs. 0.5 mm², p = 0.05). Angiogenesis tended to be more frequent in plaques with NRS (48% vs. 30%) whereas micro-calciﬁcations tended to be more frequent in plaques without NRS (27% vs. 46%) (p = 0.06 and 0.07 respectively). In a multivariate analysis, necrotic/lipid core area (OR = 1.9), non-core plaque area (OR = 1.6), and total vessel area (OR = 0.9) independently predicted the appearance of the NRS in coronary CT angiography.

Conclusion: Delineation of NRS in CCTA is independently linked to the size of the necrotic/lipid core, the size of the non-core plaque and to the vessel area as measured in histology of advanced coronary atherosclerotic plaques.

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1. Introduction

Acute coronary syndromes frequently occur in patients with coronary atherosclerotic plaques exhibiting distinct morphological features such as increased plaque dimensions, a large lipid-rich core and a thin ﬁbrous cap [1,2]. Initially described in histopathology from patients who died of acute myocardial infarctions, atherosclerotic plaques with similar features are also present in patients who have not experienced an acute cardiovascular event [3]. To date invasive techniques are available to detect plaque features associated with acute coronary syndromes [4,5].

Coronary CT angiography (CCTA) has emerged as a non-invasive tool to detect coronary artery stenosis and a growing body of evidence suggests that it can also be used to discriminate ﬁbrotic and lipid-rich components of coronary plaques based on differences in attenuation [6,7]. However because of a substantial overlap of the CT numbers of the various plaque types, a reliable differentiation of plaques is not possible with sufﬁcient accuracy [8]. Recently, several small studies suggested that a certain morphological pattern in CT, a hyperdense ring-like structure surrounding a hypodense center, described as “napkin-ring” sign, is associated with advanced coronary plaque as deﬁned by the AHA classiﬁcation [9–11].

Previous studies have shown that the napkin ring sign in CCTA offers a high speciﬁcity and positive predictive value to detect lipid-rich plaques, yet the sensitivity of this sign to detect advanced plaque is rather low [9,12,13].

The purpose of this ex-vivo study was to determine histopathological features of advanced coronary atherosclerotic plaques that promote or interfere with the delineation of the napkin-ring sign in coronary CT angiography using co-registered data.

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2. Materials and methods

All procedures were approved by the institutional ethics committee and were performed according to local and federal regulations and the Declaration of Helsinki. For this study, the coronary arteries of seven donor-hearts (median age of the donors: 53, range 42–61 years) were imaged with coronary CT angiography and subsequently processed by histology.

The donor-hearts were provided by the International Institute for the Advancement of Medicine (IIAM, Jessup, PA). Inclusion criteria were as follows: donor age between 40 and 70 years and history of myocardial infarction or CAD proven by diagnostic tests. The cause of death of the donors was stroke in six cases and in one case the cause of death was non-natural (suicide). Donors, who underwent coronary artery bypass graft surgery were excluded from this study. The maximum allowed warm ischemia time was 6 h and the maximum cold ischemia time was 15 h. The fresh donor hearts were transported to our institution in Histidine-tryptophan-ketoglutarate (High-Dean) solution and packed in wet ice.

2.1. Ex vivo heart preparation and computed tomography imaging

The method of donor heart preparation and ex-vivo coronary CT imaging was described previously in detail [14]. Briefly, a balloon was placed in the left ventricle and inflated with saline to maintain the physiological shape of the heart. The right and the left coronary arteries were selectively cannulated and flushed to remove air bubbles and superficial thrombi. The coronary arteries were then filled with a contrast medium (approximately 5–10 ml) consisting of low viscosity methylcellulose (Methocel, DOW Chemical Company, Midland, Michigan) and 3% of a non-ionic contrast agent (Isovue 370, Bracco Diagnostics, Milan, Italy) to achieve an intra-luminal contrast enhancement similar to in-vivo coronary CT angiography (\(=250\) HU). The organ was positioned in the center of a canola oil tank to simulate the pericardial adipose tissue. CT data acquisition was performed with a 64-detector row CT scanner (High-Definition, GE Discovery, CT 750HD) using a standard coronary CT acquisition mode and the following scan parameters: 64 \(\times\) 0.625 mm collimation; 0.35 s rotation time; tube voltage of 120 kV; tube current time product of 500 mAs. Images were reconstructed with a slice thickness of 0.625 mm. The dataset was reconstructed using an adaptive statistical iterative reconstruction technique (ASIR, GE Healthcare, Milwaukee, US). Iterative reconstruction techniques have been shown to markedly reduce the image noise and enhance plaque visualization compared to conventional image reconstruction using filtered back-projection [14]. CT images were sent to an offline workstation for further analysis. Subsequent to the CT imaging, the coronary arteries were excised along with the surrounding tissue and the side-branches were ligated. The coronaries were pressure-perfused with 10% buffered neutral formalin solution in order to achieve tissue fixation. The preparation and the CT imaging were completed within 4 h to avoid potential post-mortem changes in plaque morphology.

2.2. Histological preparation and classification

The histological analysis was performed by experts specialized in cardiovascular pathology (CVPath Laboratory, Maryland, MD). From the coronary arteries, 6 \(\mu\)m thick paraffin sections were obtained in 1-mm and 5-mm increments (where minimal disease was present), resulting in a total of 611 sections. These were stained using Movat’s pentachrome. Each cross-section was classified according to the modified AHA scheme [15] into the following categories: adaptive intimal thickening (AIT), pathological intimal thickening (PIT), fibrous plaque (Fib), early fibroatheromas (EFA), late fibroatheroma (LFA), thin cap fibroatheroma (TCFA). According to a report on atherosclerotic lesion classification from the American Heart Association, AIT, Fib, and PIT were considered as early atherosclerotic lesions, and EFA, LFA, and TCFA as advanced lesions [16].

2.3. Co-registration of histology and computed tomography

A multiplanar reconstruction technique was used to generate CT images perpendicular to the vessel centerline to match the position of the histological cuts. The cross-sectional and rotational position of the slices were identified by distance measurements from the distal end of the plastic cannulas and verified by using fiducial markers such as side branches, bifurcations and features of vessel wall morphology (e.g.: plaque shape, calcification pattern, orientations of the myocardium and the pericardial adipose tissue layer).

2.4. CT data analysis

As the purpose of this study was to define features of advanced atherosclerotic plaques that promote or interfere with the delineation of the NRS in coronary CT angiography, only cross-sections containing advanced plaques as classified by histology were used in this study. The corresponding CT images were reviewed in consensus by two radiologists with 6 and 10 years of experience in coronary CT angiography. All reading was performed using a fixed window setting (700 HU width, 200 HU level). Based on the CT appearance, it was specified whether a napkin-ring sign could be identified within the plaque. The NRS was defined by a low-attenuation plaque core surrounded by a circumferential area of higher attenuation [11]. Additionally, we measured the attenuation of the non-calciﬁed plaque portion of all plaques using an in-house program developed in Matlab. For this purpose, the outer circumference of the vessel and the lumen were manually traced and the median density of the pixels within the plaque was calculated. In plaques with a positive NRS, we also measured the median density of the central hypodense area and the peripheral hyperdense ring.

2.5. Histologic data analysis

All plaques that were previously classified as advanced were analyzed by experienced pathologists (M.N., F.O.) regarding the presence or absence of a necrotic and/or lipid core, intralipoprotein hemorrhage, intralipid microvessels, macrophage inﬁltration and calcifications utilizing H&E and Movat’s pentachrome stains. The latter characteristics were stratified as being related or unrelated to the lipid/necrotic core. Calcifications were subdivided into microcalcifications (punctate calcification of smooth muscle cells, macrophages, or extracellular matrix, maximum diameter <50 \(\mu\)m), spotty (coalescent calcification, maximum diameter 50–1000 \(\mu\)m) and sheet calcifications (plate calcification, maximum diameter >1000 \(\mu\)m) [17]. All cuts were subsequently scanned and converted into high-resolution TIFF images and further analyzed using dedicated software (ImageJ 1.44o, National Institutes of Health, MD, USA).

Vessel area (VA) was deﬁned as the area inside the external elastic lamina. Furthermore, the lumen area (LA) and the area of the lipid/necrotic core (CA) were measured. From this data, the total plaque area (TPA) was calculated as TPA = VA − LA. The area of non-core plaque was calculated as NCA = TPA − CA. The plaque burden (PB) was calculated using the following formula: PB = (TPA/VA) * 100. We also assessed the thickness of the fibrous cap, which was measured at its thinnest part.

Histologic features representing the atherosclerotic plaque such as size and area of the necrotic core and area of non-core plaque as...
well as the presence of calcification, hemorrhage and macrophages are further referred to as histologic plaque features.

Variables associated with the plaque such as the vessel area, the lumen area, plaque burden and distance of the plaque from the ostium are further referred to as associated plaque features.

2.6. Statistics

Continuous variables are reported as mean (standard deviation) or median [interquartile range] and categorical by percentage (counts).

In univariate analysis, we tested which parameters derived from histology were significantly different between sections with and without NRS in CT. Wilcoxon signed-rank test and Fisher's exact test were used to assess for differences within continuous and categorical variables. All variables which showed a difference at a significance level of \( p < 0.1 \) were included in multivariate analysis. We fitted two different logistic regression models, one for associated variables and one for histologic variables. Non-significant variables were removed stepwise from the initial models. The remaining significant variables from both models (associated and histologic) were combined in a final multivariate logistic regression model. We derived from these models the c-statistic, which is equal to the AUC.

To estimate the probability of NRS appearance in dependency of the necrotic core size, we fitted a univariate logistic regression model where the necrotic size served as the predictor.

To assess the inter-observer variability of the measurements in the histology sections, the measurements were repeated by a second reader in a subset of 30 randomly chosen histology slices. The agreement between the two readers was evaluated using Pearson correlation coefficients and calculation of relative differences. A paired t-test was performed to assess whether the difference varied significantly from zero.

To determine inter-observer variability for the detection of the napkin ring sign, an independent reader assessed a random subset of 100 co-registered CCTA images for the presence of the napkin ring sign. The inter-observer agreement was evaluated using Cohen's kappa statistics that was interpreted as follows: A \( \kappa \) value greater than 0.80 corresponded to an excellent agreement and a \( \kappa \) value of 0.61–0.80 corresponded to a good inter-observer agreement.

3. Results

Histopathology identified advanced lesions (EFA, LFA and TCFA) in 139 out of the total 611 slices (22.7%). Of these, 59 (9.6%) were classified as EFA, 60 (9.8%) as LFA, and 20 (3.3%) were classified as TCFA. Early lesions (AIT, PIT and Fib) were found in 472 slices (77.3%). In CT angiography, the napkin-ring sign was observed in 33 (24%) advanced lesions, whereas no NRS was found in 106 slices (see Table 1 and Figs. 1 and 2). However, an NRS was also found in 5 of the 472 (1.1%) slices classified as early lesions. Of these five lesions, 4 were classified by histology as PIT and one as Fib.

The napkin-ring sign was most commonly observed in the RCA (55%), followed by the LCX (30%) and the LAD (15%). In contrast, plaques without NRS were evenly spread among the 3 arteries.

The median density of the non-calcified plaque portion was 61.8 HU [48.4–70.1 HU] for plaques with an NRS and 65.9 HU [49.3–87.7 HU] for plaques without NRS (\( p = 0.10 \)). In plaques with a positive NRS, the median HU of the central hypodense area was 48.1 HU [33.4–61.6 HU], whereas the rim showed a median density of 68.2 HU [52.3–76.5 HU]. The difference between the density values was significant (\( p < 0.001 \)).

### 3.1. Histologic features corresponding to the NRS

The frequency of histologic plaque features and their relation to the presence or absence of NRS on CT is summarized in Table 1.

The area of the necrotic core was more than twice as large in plaques with NRS as compared to those without NRS (median 1.10 mm\(^2\) vs. 0.46 mm\(^2\), \( p = 0.05 \) — see Fig. 3). Similarly, the area of the non-core plaque was significantly larger in plaques with NRS as compared to those without (median 10.15 mm\(^2\) vs. 6.37 mm\(^2\), \( p < 0.001 \) — see Fig. 3). The thickness of the fibrous cap was not different for plaques with and without NRS (median 0.39 mm vs. 0.30 mm, \( p = 0.15 \)). Microvessels within the plaque, indicative of angiogenesis were more common in plaques exhibiting the NRS sign (48% vs. 30%, \( p = 0.06 \)), while microcalcifications were more common in plaques without an NRS (27% vs. 46%, \( p = 0.07 \)). In contrast, the presence of macrophages, hemorrhage and calcifications (independent of whether they were spotty or sheet-like) in close proximity to the lipid core was not associated with the NRS. Additional lipid pools, smaller than the main core, were more common in plaques without an NRS (35% vs. 12%, \( p = 0.02 \)). Histologic plaque features distant from the core were generally not associated with the NRS except for core-unrelated spotty and sheet calcifications, which were more commonly detected in the absence of an NRS (3% vs. 17%, \( p = 0.04 \)).

In multivariate adjusted analysis the area of the necrotic core (OR: 1.70 (95% CI: 1.12–2.57) per increase by one mm\(^2\), \( p = 0.01 \)) and the area of the non-core plaque (OR: 1.24 (95% CI: 1.11–1.40) per increase by one mm\(^2\), \( p < 0.01 \)) remained as independent predictors of the NRS. In contrast, the presence of additional smaller lipid pools independently reduced the probability of the appearance of the NRS (OR: 0.22 (95% CI: 0.07–0.75)).

### 3.2. Other features associated with the NRS

Plaques exhibiting an NRS in CT were located more proximally as compared to those without NRS (median distance from ostium 19.0 mm vs. 31.0 mm, respectively; \( p = 0.02 \)). Also, the vessel area was larger at the site of the plaque (median 17.06 mm\(^2\) vs. 12.95 mm\(^2\), \( p = 0.02 \)) and plaque area itself was larger (median 11.49 mm\(^2\) vs. 7.49 mm\(^2\), \( p < 0.01 \)) in plaques with NRS than in plaques without NRS. Interestingly, the lumen area was not different between plaques with and without NRS (median 12.95 mm\(^2\) vs. 12.85 mm\(^2\), \( p = 0.58 \)). As a result, plaque burden was significantly larger in plaques with NRS as compared to those without NRS (79.8% vs. 68.5%, \( p < 0.001 \)).

In multivariate adjusted analysis both vessel area (OR: 0.84 (95% CI: 0.75–0.94) per 1 mm\(^2\) increase, \( p < 0.01 \)) and plaque area

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**Table 1**

<table>
<thead>
<tr>
<th>NRS 33</th>
<th>No-NRS 106</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Components related to the lipid/necrotic core</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Macrophages</td>
<td>18 (55%)</td>
<td>48 (45%)</td>
</tr>
<tr>
<td>Angiogenesis</td>
<td>16 (48%)</td>
<td>32 (30%)</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>11 (33%)</td>
<td>25 (24%)</td>
</tr>
<tr>
<td>Micro-calcification</td>
<td>9 (27%)</td>
<td>49 (46%)</td>
</tr>
<tr>
<td>Spotty or sheet calcification</td>
<td>14 (42%)</td>
<td>31 (29%)</td>
</tr>
<tr>
<td><strong>Components unrelated to the lipid/necrotic core</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Macrophages</td>
<td>4 (12%)</td>
<td>7 (7%)</td>
</tr>
<tr>
<td>Angiogenesis</td>
<td>2 (6%)</td>
<td>7 (7%)</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>2 (6%)</td>
<td>10 (9%)</td>
</tr>
<tr>
<td>Micro-calcification</td>
<td>2 (6%)</td>
<td>10 (9%)</td>
</tr>
</tbody>
</table>

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(OR: 1.74 (95% CI: 1.36–2.21) per 1 mm² increase, \( p < 0.0001 \)) remained independent predictors for the delineation of the NRS in CT. Plaque burden was not included in this analysis as it was co-linear and inferior to the combination of vessel area and plaque area. When adjusting for both histologic and associated features the area of the necrotic core area, the area of the non-core plaque, and the vessel area remained independent predictors for the appearance of the NRS with the area of the necrotic core being the strongest predictor (OR 1.91; 95% CI: 1.23–2.98). The combined model reached a c-statistic of 0.816 (Table 2).

3.3. Interobserver-agreement

We found a very good correlation between the measurements for the vessel area, lumen area and the area of the necrotic core (\( r = 0.998, 0.994 \) and 0.997 respectively) in histology. Measurements for necrotic core area and vessel area were significantly different between the two readers \( (p < 0.05) \), however, the mean difference between the measurements was 1.9% \( (0.34 \pm 0.52 \text{ mm}^2) \) for the vessel area and 2.9% \( (0.04 \pm 0.08 \text{ mm}^2) \) for the area of the necrotic core. No difference was found between the two readers for the lumen area \( (p = 0.61) \).

The interobserver agreement between the two CT readers to detect the napkin ring sign was excellent (Cohen’s kappa = 0.86; 95%CI 0.76–0.96).

4. Discussion

In the search to find a pattern of coronary atherosclerotic plaque in coronary CT angiography beyond CT attenuation that is associated with advanced coronary atherosclerotic plaque several authors have described the napkin-ring sign \([9,10,12]\). However, the factors that influence the delineation of this sign remain unclear. In this ex-vivo study we used the gold standard histopathology to define the equivalent of the napkin-ring sign and the components of atherosclerotic plaque that correspond to this sign in coronary CT angiography.

Our results demonstrate that the histopathological equivalent of the NRS is a large and advanced atherosclerotic lesion with a large necrotic core.

4.1. The NRS in CT and the corresponding histopathology

Plaques with low density values (typically below 30HU) in coronary CT angiography correlate with lipid rich plaques as
angiography also had a significantly larger in plaques with positive NRS. Note that the vast majority of cores in plaques without the NRS had an area below 1 mm², which is deemed a crucial size often found in rupture-prone lesions. Plaques with a positive NRS in coronary CT angiography also had a significantly higher non-core plaque area as compared to plaques without the NRS.

Fig. 3. Areas of the lipid/necrotic core and the non-core plaque as measured in histology in plaques with presence and absence of the napkin-ring sign in coronary CT angiography. Although there is a substantial overlap, the mean size of the core is significantly larger in plaques with positive NRS. The second most important feature in plaques with an NRS in CT was the size of the plaque surrounding the necrotic core, which was also nearly twice as large compared to plaques without the NRS (10.15 mm² vs. 6.37 mm², \( p < 0.001 \)). The plaque component surrounding the necrotic core mainly consists of fibrous tissue and smooth muscle cells and represents the equivalent of the rim of the napkin-ring sign as seen in CT.

The association between plaque size and NRS is explained in that the limited spatial resolution of CT requires a certain number of pixels representing a specific plaque component in order to be able to differentiate them from each other, i.e. the hypodense core from the hyperdense rim. While a large plaque area is required for the delineation of the napkin-ring sign, it is also a feature of advanced atherosclerotic plaques. Several CT and IVUS studies have reported that unstable lesions associated with ACS show a larger plaque area with positive remodeling compared to stable lesions in patients with stable angina [6,22,23]. The association of a large plaque area with the NRS is also mirrored in the association of NRS and plaque burden and the fact that the napkin-ring sign was more commonly detected in proximal segments of the coronary artery tree.

Initially it was speculated that the ring like sign was caused by deep calcifications within the plaque. Indeed, core associated, larger calcifications such as spotty calcifications were slightly more common in plaques where the NRS was present (42% vs. 29%). However, the formation of these calcifications did not explain the appearance of the NRS. In addition, several authors demonstrated the absence of macrocalcifications around the hyperdense central part of the napkin-ring sign in non-enhanced CT scans [11].

Consequently, most authors support the hypothesis that the napkin-ring sign is caused by vasa vasorum enhancing the outer curvature of the plaque. Our results demonstrate that angiogenesis is associated with the NRS (48%) but it is also quite common in plaques not characterized by an NRS (30%). In our ex-vivo study design, contrast material was viscous due to the use of methylcel-lulose and probably did not enter the vasa vasorum. Thus, the contrast agent did not enhance the delineation of the hyperdense rim. This is in line with previous reports demonstrating the NRS in non-contrast enhanced images [11]. However, it is conceivable that neovascularization contributes to the delineation of the napkin-ring sign in vivo by increasing the attenuation of the tissue around the core after the administration of contrast media. Independent of this, the fact that neovessels are often found in plaques with an NRS is important as neovascularization arising from adventitial vasa vasorum is common in advanced coronary plaques without the NRS.

Table 2
The model combining both histologic and plaque associated features reached a c-statistic of 0.816.

<table>
<thead>
<tr>
<th>Independent predictors</th>
<th>OR (95%CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vessel area [per mm²]</td>
<td>0.88 (0.78—0.99)</td>
<td>0.07</td>
</tr>
<tr>
<td>Area of non-core plaque [mm²]</td>
<td>1.60 (1.24—2.05)</td>
<td>0.0002</td>
</tr>
<tr>
<td>Area of lipid and/or necrotic core [mm²]</td>
<td>1.91 (1.23—2.98)</td>
<td>0.004</td>
</tr>
<tr>
<td>Presence of additional lipid cores</td>
<td>0.36 (0.10—1.25)</td>
<td>0.11</td>
</tr>
</tbody>
</table>

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atherosclerotic lesions and has been associated with intraplaque hemorrhage and subsequent plaque destabilization [24,25]. Somewhat surprisingly, we found microcalcifications in the rim surrounding the core only in a quarter of NRS plaques (27%). Microcalcifications have also been suggested as a possible cause for an increase in CT attenuation as in some cases the brighter rim of the NRS has been observed in non-contrast scans [11]. Indeed, data from atherosclerosis models shows that microcalcifications often develop around the necrotic core [26]. In the present study, microcalcifications were more commonly found in plaques without an NRS (46%), suggesting that microcalcifications can elevate the Hounsfield values of the tissue around necrotic core and thus lead to volume averaging which prevents the hypodense core from being identified in coronary CT angiography. The presence of microcalcifications around the core might also explain why density values in advanced atherosclerotic plaques vary, making a reliable differentiation of plaques types impossible [8].

4.2. Limitations

Although we injected a contrast agent into the coronary arteries, we presume that this did not reach the small neovessels within the plaques because we used a viscous contrast agent and a low pressure to push the contrast agent into the coronary arteries. The second reason is that residual blood clots in the vasa vasorum prevent the contrast agent from entering the neovessels. In patients, the contrast agent will reach the neovessels during the first pass leading to an enhancement of the fibrous tissue around the necrotic core in advanced coronary atherosclerotic plaques. Thus, the number of plaques with a positive napkin-ring sign might be higher than in the present study. However Pfleiderer et al. report a similar percentage of plaques with contrast enhanced rims detected in culprit lesions in patients with ACS [1].

The current study was performed in an ex-vivo setting offering ideal conditions for the imaging of the coronary vessels, especially with regard to motion artifacts. However coronary CT angiography can be performed with little or no motion artifacts using modern scanners careful planning of the exam [27,28]. For the present study, we used a scan protocol with a tube voltage of 120 kVp. While this has been the standard setting for many years, scan protocols using 100 kVp are now standard in smaller patients [29]. The choice of a scan protocol with lower kVp values alters the Hounsfield units of all structures containing iodine and calcium whereas the density values of all other structures are not altered significantly [30]. Thus, the selection of an imaging protocol with 100 kVp might have slightly enhanced the detection of calcium. As the contrast material in the present study did not enter the plaque itself, the delineation of the necrotic core would not have been affected by such a protocol. While these factors might limit the generalizability of our findings our approach offered the unique possibility to compare the CT morphology of advanced atherosclerotic plaques to the gold standard of histopathology.

5. Conclusion

Coronary atherosclerotic plaques with a napkin-ring sign in CT have a distinct histopathological appearance primarily characterized by a large necrotic core with a large fibrous component often associated with neovascularization. Because these features have been associated with advanced atherosclerotic and rupture-prone lesions in histology, the napkin-ring sign can possibly serve as a marker for advanced lesions in coronary CT angiography.

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Disclosures

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