Photodynamic Therapy of Atherosclerotic Plaque Monitored by T1 and T2 Relaxation Times of Magnetic Resonance Imaging

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Abstract: Atherosclerosis, marked by plaque accumulation within arteries, results from lipid dysregulation, inflammation, and vascular remodeling. Plaque composition, including lipid-rich cores and fibrous caps, determines stability and vulnerability. Photodynamic therapy (PDT) has emerged as a promising treatment, leveraging photosensitizers to induce localized cytotoxicity upon light activation. PDT targets plaque components selectively, reducing burden and inflammation. Challenges remain in optimizing PDT parameters and translating preclinical success to clinical efficacy. Nonetheless, PDT offers a minimally invasive strategy for atherosclerosis management, promising personalized interventions for cardiovascular health. The objective of the current study was to present the findings from quantitative non-contrast MRI of atherosclerosis post-PDT by assessing relaxation times. The study aimed to utilize and optimize a 1.5T MRI system. Clinical scanners were used for MRI examinations. The research involved analyzing T1 and T2 relaxation times. Following treatment of the samples with Rose Bengal and exposure to pure oxygen, PDT irradiation was administered. The results indicated that the therapy impacted the crus, evidenced by a significant decrease in relaxation times in the MRI data.

Keywords: atherosclerosis; vulnerable plaque; TCFA; PDT; MRI

1. Introduction

Atherosclerosis and its consequences are still the main cause of mortality in developed societies [1], and the development of technologies and treatment methods will not reach a level that we could call a breakthrough in this field. Although the milestones were platelet drugs or statins [2,3], as well as various methods of revascularization of coronary [4,5], cervical [6], or peripheral vessels [7], the question remains whether it is possible to inhibit atherosclerosis globally around the main vascular beds distributing blood to the central nervous system, coronary arteries, kidney, or lower limb peripheral arteries. Treatment of atherosclerosis that significantly narrows the lumen of the vessels is scientifically established; the so-called borderline or insignificant lesions are a gray zone in the recommendations [8]. In the case of coronary artery disease, the cut-off values for atherosclerotic lesions significantly affecting stenosis and being the cause of ischemia
were quite clear and transparent. The cut-off value of invasive FFR (fractional flow reserve) of <0.75 [9] and the non-invasive [10] or cross-sectional area, for example of the left main coronary artery (LM) below 6 mm² [11], were confirmed to impact cardiovascular death in many trials. Their consequence are the guidelines of the European and American cardiac associations, which recommend treating or leaving untreated the mentioned coronary artery changes [12,13]. The management of atherosclerosis of the carotid arteries, responsible for ischemic strokes, or arteries of the vessels of the lower limbs, leading to intermittent claudication, is equally clear and defined [14,15]. The detection of TCFA or the assessment of the significance of stenosis is therefore precisely defined and possible in many vascular areas [16,17]. Magnetic Resonance Imaging (MRI) plays a crucial role in evaluating atherosclerosis, particularly in assessing changes in tissue characteristics following treatment. MRI relaxation times, specifically T1 and T2 relaxation times, provide detailed information about the tissue composition and structural integrity of atherosclerotic plaques. After treatment, such as lipid-lowering therapy or anti-inflammatory medication, changes in these relaxation times can indicate a reduction in plaque lipid content and inflammation, suggesting plaque stabilization or regression. Monitoring these parameters helps in evaluating the efficacy of the treatment and in making informed decisions about patient management. Changes in these relaxation times after PDT treatment can provide valuable insight into the efficacy of therapy and the underlying mechanisms of plaque stabilization and regression. Despite these advantages, the specific rationale for using PDT in the context of its effect on T1 and T2 relaxation times in atherosclerosis has not been clearly defined in previous studies. The inclusion of PDT in our study aims to fill this gap by highlighting its dual role as both a therapeutic and a diagnostic tool. By demonstrating the effects of PDT on T1 and T2 relaxation times, we aim to provide a comprehensive understanding of its potential to improve the treatment of atherosclerosis. This integrated approach not only underscores the innovative application of PDT but is also consistent with the broader goal of advancing cardiovascular imaging and therapy. The question remains open whether the current atherosclerotic lesions narrowing the lumen of the vessel to an insignificant degree, but meeting the criteria of vulnerable plaques, should only be treated conservatively. The goal of this article is to characterize atherosclerotic plaques, considering distinctive features useful for determining effectiveness of the application of photodynamic therapy (PDT).

Atherosclerosis Characteristics

The key pathophysiological element, which is the common nomenclature of the above-mentioned destinations, is the concept of forming or already present atherosclerotic plaque [18]. The concept of inflammation as a pivotal reason for the pathogenesis of atherosclerosis and its complications has achieved considerable attention but has not yet entered routine clinical practice [19]. Atherosclerosis is a lipoprotein-driven disease that over time leads to plaque formation at specific sites of the arterial tree due to a continuous process of intimal inflammation, necrosis, fibrosis, and calcification [20]. After decades of silent progression, such plaques may suddenly become more severe and cause life-threatening coronary thrombosis, presenting as an acute coronary syndrome. Most often, the culprit morphology is plaque rupture or local erosion with exposure of highly thrombogenic, red cell-rich necrotic core material [21]. The prognosis is more favorable for onset of the disease in stable coronary artery disease, when, over time, the atherosclerotic plaque builds up, hardens, and consequently narrows the lumen of the arteries, in this way reducing blood flow [22]. The artery walls also become thickened and stiff. This obstructive stable plaque can be detected and treated; however, the artery walls have never been brought back to their state before the onset of atherosclerosis [23]. Soft atherosclerotic plaques are a much bigger problem, leading in the case of coronary artery disease to acute coronary syndromes, including myocardial infarction or even in the worst scenario to sudden cardiac death, because the plaque can burst, leading to blood clot creation, thus starting a cascade resulting in the activation of fibrinogen platelets and the formation of the
so-called primary plug [24]. These types of cases affect a section of the younger population and are associated with the presence of vulnerable atherosclerotic plaques referred to as TCFA [25]. A high content of lipid tissues and a poor connective tissue cap are the essence of this type of most vulnerable plaque [26].

2. Materials and Methods

2.1. Carotid Artery Samples

The study design is the first phase of a prospective population-based study observing the composition of atherosclerotic plaque lesions and, at a later stage, the effect of PDT therapy. The study aimed to apply an MRI method to evaluate in vitro sections of atherosclerotic plaques after PDT therapy with Rose Bengal, focusing on changes in T1 and T2 relaxation times. The research received approval (No. 17/02/2019) from the Bioethics Committee at the University of Rzeszów. A patient with clinically diagnosed atherosclerotic plaque in a specified artery was included. The study presents a sample from a male patient carotid artery with clinical vascular changes and diagnosed atherosclerosis at the time of vessel collection. Carotid artery tissue samples were obtained by endarterectomy. The study protocol was approved, and the participant gave informed consent for the use of sections from his body. A sample of a vessel with atherosclerosis lesions was taken under surgical conditions by a vascular surgeon. The sample was frozen at −80 degrees Celsius until it was removed. The sample took about 5 h to thaw at room temperature, and no thermal procedures were used (Figure 1). From the obtained specimen we prepared 5 samples with atherosclerosis with RB and 1 control sample only with RB (n = 6).

![Figure 1. View of the retrieved vessel after defrosting.](image-url)

2.2. Rose Bengal Concentration

Rose Bengal disodium salt (95%) was used at concentrations of 0.01 mM, 0.02 mM, 0.03 mM, 0.04 mM, and 0.05 mM, with each concentration applied to each sample. Oxygen gas (99%) was obtained from STP & DIN Chemicals, Bielsko-Biała, Poland. The water for preparing the RB stock solutions was purified using the AquaB Duo reverse osmosis system from Fresenius Medical Care, Singapore (Figure 2). The concentration of RB applied to the tissue ranged from 0.01 mM to 0.05 mM. Lower concentrations, such as 0.01 mM, were rapidly adsorbed. Higher concentrations were able to penetrate and diffuse into the tissue.
2.3. PDT Procedure

Samples were injected with 0.1 mL of various RB concentrations and irradiated at 532 nm for 15 min. The radiant power of the 532 nm light was measured using a Newport power meter, model 1918-C. To optimize the distance between the laser light source and the samples, studies were conducted to measure tissue temperature and laser spot size by adjusting the light at distances of 5 cm, 10 cm, and 15 cm from the tissue. A solid-state laser (LD Pumped All-Solid-State Green Laser, MGL-III-532 nm/300 mW) provided 532 nm light, which is near the maximum absorption wavelength of RB. The laser was connected to a fiber optic cable. The distance between the light source and the tissue surface was chosen to avoid excessive heating, with sample temperatures not exceeding 30 °C after 15 min of exposure. The light source was positioned at a distance from the tissue surface to create an illumination area of 2.5 × 2.5 cm². Laser fluence was determined using the formulas: energy [J] = power [W] × time [s], and fluence = energy [J]/area [cm²]. For instance, 15 min (900 s) of irradiation over an area of 6.25 cm² with a 300 mW laser results in a fluence of 43.2 J/cm².

2.4. MRI Analysis

Spin–lattice (T1) and spin–spin (T2) relaxation times were measured using a Tesla Optima MR360 MRI device from General Electric Healthcare (Milwaukee, WI, USA) (Figures 3 and 4), with SV23 software. The tests were performed using fast spin echo (FSE) sequences with axial projection and a small flex coil. DICOM figures were analyzed, and ROI measurements were taken from a series of figures for each test, allowing T1 and T2 relaxation times to be determined based on the collected data. The samples were examined at two different stages of the study. MRI technical parameters remained consistent throughout, with a scanning matrix of 320 × 224, section thickness of 4 mm, and NEX = 1.0. T1 measurements were conducted in 12 steps with a repetition time (TR) ranging from 200 to 12,000 ms and an echo time (TE) of 27 ms. For T2, 12 steps were performed with a fixed repetition time of 10,000 ms and an echo time ranging from 21.0 to 240.0 ms. The temperature during the test before laser irradiation was 22 degrees and after it was 20 degrees. A difference of 2 degrees is insignificant if the results of the final relaxation times are evaluated. The duration from after the PDT slides were prepared and irradiated to the time of glamorous resonance imaging was used to determine T1 and T2 relaxation times. The scanning parameters used in this stage of the study were unchanged from the first round.
Figure 3. Pre and post samples irradiated at 532 nm for 15 min. Samples with Rose Bengal disodium salt at concentrations of 0.01 mM (No. 1), 0.02 mM (No. 2), 0.03 mM (No. 3), 0.04 mM (No. 4), and 0.05 mM (No. 5).

Figure 4. Tesla Optima MR360 MRI used to determine T1 and T2 relaxation times.

3. Results

The signal intensity measure was used to determine relaxation times. The measured signal intensity of the atherosclerotic sample within the region of interest (ROI) was used to calculate the T1 and T2 relaxation times. These values were calculated in the program based on the increase in intensity of the magnetic resonance signal for T1 relaxation and the decay of the signal for T2 relaxation. Figures 5 and 6 show T1 and T2 times. The figures show “Ependorf” tubes in which the tested preparations were placed. The tests were performed in the sagittal plane and not, and it was considered pointless to correct the image rotation by 90° as this action does not contribute anything to the methodology. T1 time values were determined using the SE (saturation recovery) method. This method involves recording a series of T1-weighted figures with a constant TE time (e.g., 27 ms) and a variable repetition time (TR). Then, after generating the figures, the signal intensity (IS) in the ROI is measured...
as a digital value saved in DICOM files. Only such an approach to the issue provides appropriate data for further analysis. The read IS data combined with the TR times at which the figures were recorded are normalized and approximated with an exponential function. Time T1 is calculated as the time at which the approximation function reaches 63% of its maximum value. The T2 time is determined in a similar way to the longitudinal relaxation time, with the difference being that the TE time changes, while the repetition time remains unchanged. Performing this operation for all image pixels allows you to draw a map of the distribution of T1 and T2 times for the examined area.

![Figure 5](image1.png)

**Figure 5.** Determined T1 and T2 relaxation times of atherosclerotic samples before PDT. The yellow box is the area of the Voxel.

![Figure 6](image2.png)

**Figure 6.** Determined T1 and T2 relaxation times of atherosclerotic samples after PDT. The yellow box is the area of the Voxel.

Table 1 shows the T1 and T2 values of the relaxation times of the tested samples before and after PDT.
Table 1. Comparison of relaxation times before and after irradiation.

<table>
<thead>
<tr>
<th>Sample</th>
<th>Before</th>
<th>After</th>
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<tr>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>T1 = 1030.1 ms ± 154.52 T2 = 136.33 ms ± 20.45</td>
<td>T1 = 1209.8 ms ± 181.47 T2 = 97.011 ms ± 14.55</td>
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<tr>
<td>4</td>
<td>T1 = 977.63 ms ± 146.64 T2 = 128.83 ms ± 19.32</td>
<td>T1 = 1201.1 ms ± 180.17 T2 = 105.06 ms ± 15.76</td>
</tr>
<tr>
<td>5</td>
<td>T1 = 1737 ms ± 260.55 T2 = 198.24 ms ± 29.74</td>
<td>T1 = 1944.3 ms ± 291.60 T2 = 127.65 ms ± 19.15</td>
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Figures 7 and 8 show images from histological analysis and T1 and T2 maps. A region of interest (ROI) was selected in the imaged tissues to calculate the T1 and T2 values. The region of interest measurements were selected very close to the tissues.

**Figure 7.** (a,d) contain the results of mapping the longitudinal relaxation times. They present maps of the distribution of T1 times. The sharp demarcation between the fluid and the examined structures is clearly visible. (b,e) present the distribution of the R² coefficient—it is a measure of the fit of the approximating curve describing the measurement data. It is clearly seen that this coefficient is close to “1”. This proves a very good fit. These figures also show a decrease in the R² value for regions more distant from the coil plane. This is a characteristic phenomenon because the coil used has the characteristics of a flat-loop coil which produces very good figures in its plane, but when moving away from it, the signal quality decreases and noise increases. This type of coil was chosen due to the geometric characteristics of the tested objects. The aim of the study was to image the structures lying in the plane of the urethra as well as possible. The figures presented in (c,f) are histograms, allowing determination of the quantitative distribution of pixels in the examined figures. The histogram plot is the number of pixels in the image (vertical axis) with a particular brightness value (horizontal axis). The histogram plot is the distribution of the number of pixels according to their intensities, corresponding to the time value that is calculated.
Figure 8. (a,d) contain the results of mapping the transverse relaxation times. It should be said that in this case the noise is significantly increased. Very short relaxation times resulted in poorer image quality—the system used for research has limitations regarding the parameters that can be set for TE and TR times. This is most noticeable in the parts of the image showing fluids. The regions imaging the structures being examined are mapped with greater accuracy—their times are significantly longer than the T2 of fluid areas. In the context of the decrease in the quality of fitting the curves to the measurement data, the figures for $R^2$ (b,e) are very telling, where the fluid areas are dark blue and therefore $R^2$ is close to the value “0”. (c,f) are histograms showing the distribution of the number of pixels in the examined region. The histogram plot is the number of pixels in the image (vertical axis) with a particular brightness value (horizontal axis). The histogram plot is the distribution of the number of pixels according to their intensities, corresponding to the time value that is calculated.
4. Discussion

Although statin-based pharmacotherapy has proven effective in numerous clinical trials, most acute coronary syndromes arise from non-critical or borderline plaques destabilized by various factors [27]. Understanding the differences in the structure and pathomechanism of atherosclerotic plaque formation may aid in developing new therapeutic methods for patients with atherosclerosis, which is not yet considered significant in terms of narrowing of the vessel lumen [28]. Carotid plaque characteristics were previously associated with restenosis at the site of carotid endarterectomy after one year [29]. Endarterectomy of lipid-rich, inflammatory plaques reduces restenosis compared to fibrous plaques, regardless of clinical characteristics [29]. Hellings et al. concluded that carotid plaque composition also predicts future cardiovascular events elsewhere in the vascular system, independently of specific risk factors and medication use. Other histological parameters, such as macrophage infiltration, large lipid core, calcifications, collagen, and smooth muscle cell infiltration, were not linked to the risk of secondary events [29]. One promising, though still underexplored, treatment method is PDT [30]. PDT generates ROS that interfere with cell survival and the remodeling process. ROS-induced cell death depends on the type of cell, photosensitizer, its cellular localization, and the light dose [31]. When light dose and target tissue are constant, PDT-mediated cell death depends on the photosensitizer and its subcellular localization [32]. Different photosensitizers can localize in multiple organelles, activating more than one cell death pathway, including programmed (apoptotic and autophagy) and nonprogrammed (necrosis) pathways. Generally, apoptosis occurs at low light intensity, while necrosis is activated at higher light doses. PDT may also stimulate autophagy, delivering cytoplasmic constituents to the lysosome [33]. In this study, PDT was evaluated using MRI, an innovative method to analyze its efficacy. PDT-treated atherosclerotic plaque showed higher mean T1 and T2 values compared to those before PDT. The mean T1 value increased from 1248.24 ms ± 187.24 before PDT to 1451.73 ms ± 217.75 after PDT. The mean T2 value decreased from 154.47 ms ± 23.17 before PDT to 109.91 ms ± 16.49 after PDT. Studies by Wang et al. and Fei et al. reported different T1 and T2 values post-PDT, consistent with the conclusion that PDT with Rose Bengal affects cell death [34,35]. This reduction in T2 relaxation time indicates altered plaque properties, demonstrating PDT’s potential as a minimally invasive treatment for atherosclerosis. The significant changes in T1 and T2 values before and after PDT therapy make MRI a non-invasive method for monitoring PDT-induced changes. In the last decade, porphyrins, chlorines, and dye-based photosensitizers have been tested for treating atherosclerotic plaques. Porphyrin-based photosensitizers selectively accumulate within plaques. In one study, a benzoporphyrin derivative selectively accumulated in rabbit atherosclerotic plaques when preassociated with low-density lipoprotein [36]. Hematoporphyrin derivative-associated PDT inhibits smooth muscle cell growth and decreases the intima/media ratio of rabbit atheroma 7–14 days post-PDT compared to controls [36,37]. Verteporfin, a second-generation photosensitizer, binds with low-density lipoproteins and induces apoptosis upon light activation by increasing mitochondrial cytochrome c and apoptosis-inducing factor levels [36]. Benzoporphyrin derivative monoacid ring A was taken up by plaques in hyperlipidemic rabbits, humans, and miniswine [36–39]. Motexafin lutetium, another photosensitizer, is distributed with the LDL-cholesterol fraction after intravascular injection in diseased animal models and shows a clear tropism to atheromatous plaques [40,41]. Protoporphyrin-IX-based PDT has been effective in preventing and treating atherosclerotic plaque in rabbits and pigs [42,43]. An endovascular light diffuser significantly reduced plaque without visible damage to the artery wall’s middle layer [44]. Equally important is the inhibition of restenosis resulting from neointima formation, caused by endothelial or medial injury followed by inflammatory cell infiltration, vascular smooth muscle cell proliferation, and migration [45]. Despite advances in stents and drug balloons, restenosis remains an issue [46,47]. The total length of atherosclerotic areas that can be treated with stents or drug balloons is limited, especially in patients with advanced kidney disease or diabetes [48,49]. PDT could fill the treatment gap by inhibiting smooth muscle
cell proliferation and promoting plaque stabilization, paradoxically causing injury to almost all plaque cell types while reducing macrophage and foam cell content [50,51]. The challenge is to selectively target the plaque without harming the normal vessel wall. No photosensitizer currently meets all ideal criteria, such as no dark toxicity, high selectivity for plaque macrophages, long activation wavelengths, and targeted activation. Balloon angioplasty with photosensitizers is an attractive prospect. Light sources must be minimized and adapted to vessel size, access, and sterility. Laser sources provide powerful and bright light beams that can be coupled in optical fibers, emitting light in a narrow spectral domain, reducing illumination time for a given dose [52,53]. Despite promising animal studies, PDT has not yet fully realized its potential in treating atherosclerosis. Potential applications include treating vulnerable plaques confirmed by intravascular ultrasound assessment (IVUS) or optic coherence tomography (OCT). These methods help detect TCFA in human coronary arteries, targeting vulnerable changes for therapy, including PDT. Approximately 14% of patients experience plaque ruptures post-stenting, with PDT potentially stabilizing these plaques and reducing rupture risk. Another application is preventing neoatherosclerosis leading to restenosis within treated segments. New techniques, such as drug balloons with paclitaxel or sirolimus, do not completely protect against plaque destabilization [54,55]. PDT’s capability to provoke plaque stabilization qualifies it as a complementary tool for treating plaque rupture and neoatherosclerotic lesions. Although new stent generations and drug-covered balloons have reduced in-stent restenosis risk, neither is entirely resistant [56–58]. Combining PDT with percutaneous coronary intervention may prevent stent-induced restenosis recurrence or eliminate the need for stents in borderline lesions within vulnerable plaques. Miniaturizing PDT emitters and selecting the right photosensitizer for local delivery could revive clinical work on PDT for human atherosclerosis treatment. The known physicochemical properties of atherosclerotic plaques, presented in this article, are steps toward this goal. MRI’s utility in plaque characterization relies on T1 and T2 relaxation times, providing insights into tissue properties. T1 reflects the tissue’s fat content and fibrosis, while T2 indicates inflammation, edema, and necrosis. By analyzing these times, MRI differentiates between lipid-rich, fibrous, and calcified plaques, aiding in risk stratification and treatment planning [59,60]. MRI with T1 and T2 mapping could complement PDT by identifying plaques suitable for treatment and monitoring response over time [61,62]. Studies have significantly advanced understanding of atherosclerotic plaque characteristics using MRI. Yuan et al. (2001) used multiparametric MRI to identify lipid-rich necrotic cores and intraplaque hemorrhage [63]. Zhao et al. (2001) showed intensive lipid-lowering therapy’s effects on plaque characteristics [64]. Cai et al. (2005) compared contrast-enhanced MRI with histology to quantify fibrous cap and lipid-rich necrotic core sizes [65,66]. Cappendijk et al. (2004) focused on MRI detection of plaque hemorrhage [67]. Trivedi et al. (2004) investigated ultrasmall superparamagnetic iron oxide-enhanced MRI to detect macrophages [68]. Fayad et al. (1998) enabled high-resolution MRI of atherosclerotic lesions in mice [69]. Edelman et al. developed fast selective black blood MRI techniques for visualizing plaque morphology [70,71]. Tang et al. (2009) evaluated atorvastatin therapy’s effects on macrophage activity using ultrasmall superparamagnetic iron oxide-enhanced MRI [72]. MRI has become crucial for understanding plaque composition, vulnerability, and therapeutic intervention effects in carotid disease.

5. Conclusions

While pharmacotherapy, particularly statins, has proven effective in clinical trials, most acute coronary syndromes arise from non-essential or borderline plaques destabilized by various factors. Understanding the structural and pathomechanical differences in atherosclerotic plaque formation is crucial for developing new therapeutic approaches, especially for patients with early-stage atherosclerosis. This study focuses on carotid plaque characteristics post-surgery, highlighting their relevance for restenosis prediction and cardiovascular event prognosis. PDT emerges as a promising but still underexplored treatment method for atherosclerosis. PDT selectively targets plaque components, inducing cell death and promot-
ing plaque stabilization. Various photosensitizers, such as porphyrins and chlorines, have shown promise in preclinical studies by demonstrating selective plaque accumulation and inhibition of smooth muscle cell proliferation. PDT’s potential lies in its ability to inhibit neointimal hyperplasia post-revascularization, addressing a critical challenge in preventing restenosis. Despite remaining challenges in photosensitizer selectivity and delivery methods, PDT holds promise as a complementary tool in atherosclerosis management, offering potential for personalized, targeted interventions to mitigate cardiovascular risk. Continued research and technological advancements in PDT are essential for translating its therapeutic potential into clinical practice effectively. MRI has established itself as a crucial modality in the non-invasive assessment of atherosclerotic plaques, providing detailed information on plaque composition, morphology, and activity. Recent advances in MRI technology, such as 3T and 7T MRI systems, have significantly improved spatial resolution and signal-to-noise ratio, enabling more detailed plaque characterization. The development of novel contrast agents, including those targeting specific molecular markers of plaque instability, holds promise for more precise identification of high-risk plaques. Additionally, the integration of MRI with other imaging modalities, such as PET/MRI, provides complementary information on plaque metabolism and inflammation, offering a more comprehensive assessment of plaque pathophysiology. Despite its advantages, MRI in atherosclerosis plaque imaging faces several challenges. Motion artifacts from cardiac and respiratory movements can degrade image quality, particularly in coronary artery imaging. Moreover, MRI is contraindicated in patients with certain implants or devices, and the use of gadolinium-based contrast agents carries a risk of nephrogenic systemic fibrosis in patients with severe renal impairment. This study represents a first step in assessing the impact of PDT therapy through T1 and T2 relaxation times. MRI provides information on relaxation time values and their differences in healthy and atherosclerotic plaques, allowing for the assessment of physicochemical differences in plaques. The results indicate the utility of MRI relaxation times in differentiating myoablative plaques. In vitro PDT therapy, combined with MRI, enabled imaging of changes in atherosclerotic plaques. This experiment may lead to further studies on monitoring PDT in vitro as well as in vivo by MRI.


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Data Availability Statement: All data are included in the manuscript.

Conflicts of Interest: The authors declare no conflicts of interest.

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