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INTRAVASCULAR IMAGING OF ATHEROSCLEROTIC PLAQUES IN PATIENTS WITH CARDIORENAL SYNDROME: POTENTIAL USE OF OPTICAL COHERENCE TOMOGRAPHY

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Currently, kidney transplantation and hemodialysis are the primary therapies for end-stage renal disease. High mortality, mostly caused by cardiovascular disease, remains the main challenge in the treatment of this category of patients. It has been shown that in patients with end-stage chronic kidney disease undergoing hemodialysis, the risk of mortality due to cardiovascular disease is up to 20 times higher than in the sex- and age-matched general population. The indicated data determined the appropriateness of isolating cardiorenal relationships into a single cardiorenal syndrome (CRS). Due to the facts mentioned above, intravascular imaging methods, notably optical coherence tomography (OCT), are particularly important in diagnosing coronary artery lesions. This review analyses the data published to date on the features and capabilities of OCT in CRS patients.

Keywords: cardiorenal syndrome, optical coherence tomography, intravascular imaging.

Cardiovascular disease remains a leading cause of morbidity and mortality in patients with chronic kidney disease (CKD). In this case, the risk of mortality increases as estimated glomerular filtration rate (eGFR) falls: in patients with end-stage CKD undergoing hemodialysis, the risk of mortality from cardiovascular diseases is up to 20 times higher than in the sex- and age-matched general population [1]. Symbiosis of the mechanisms of regulation and functioning of the heart and kidney, and dysfunctions of both organs served as the basis for distinguishing cardiorenal relationships into a single syndrome. As defined by Ronco et al. (2008), cardiorenal syndrome is a simultaneous dysfunction of both the heart and the kidney, in which acute or chronic injury to one organ can cause acute or chronic injury to the other [2].

Coronary heart disease (CHD) is a leading cause of morbidity and mortality in CRS patients. CKD is a risk factor for acute coronary syndrome (ACS) [3].

Postmortem and life-time computed tomography imaging and intravascular ultrasound (IVUS) showed a reliable correlation between CKD and severity of CHD and atherosclerotic plaque calcification in coronary arteries.

Optical coherence tomography (OCT) permits visualization of atherosclerotic plaques in coronary arteries with higher resolution; better than with IVUS, calcium penetration is determined. However, the use of OCT in CKD patients is limited due to the need for additional use of contrast agents required to create an optically clear medium.

This review presents an analysis of reports published to date on the features and capabilities of OCT in CRS patients. Our own examples of visualization of atherosclerotic lesions in coronary arteries are given as illustrations [4].

OCT is an intravascular imaging method based on reflection of infrared rays from vessel wall structures [5, 6]. OCT was developed in the late 1980s and early 1990s [7]. In the early 2000s, there were studies showing that OCT is a safe diagnostic method and is not inferior in efficiency to IVUS [8]. This led to more studies with OCT and increased the need to unify image analysis techniques. The first part of a review document on the methodology, terminology and clinical use of OCT, prepared by an international team of experts, was published in 2010 [9]. The document covered the physical principles of OCT, method for obtaining OCT images, and safety and effectiveness of OCT. Data on normal morphology of coronary arteries and assessment of atherosclerotic lesions in coronary arteries were presented. Some conflicting aspects, as well as advantages and disadvantages of OCT over IVUS were analyzed. In 2012, the second part of the review document was published, which was devoted to clarifying some issues not covered in the first part, as well as describing the method of installing stents under OCT [10]. In 2018, the first part of the approval document of the European Association of Percutaneous Cardiovascular Interventions was published. The document analyzed the advantages and disadvantages of

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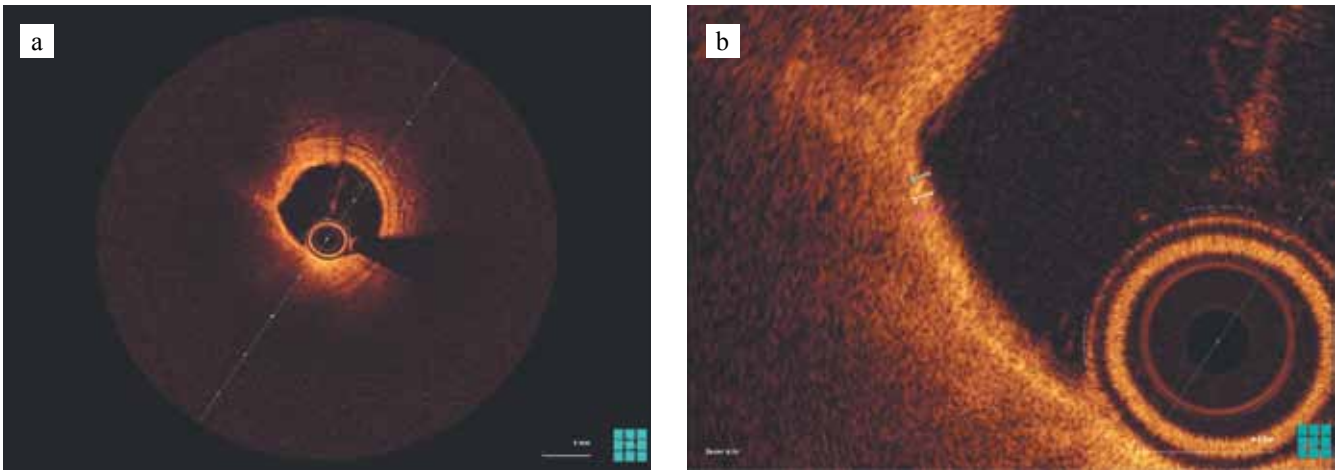

Fig. 1. Thin-cap atherosclerotic plaques (OCT): a – lipid pool at 6–11 hours of conditional dial (lipids occupy more than 1 quadrant, which means that this plaque can be classified as lipid-rich); b – 3-times measured fibrous cap thickness in the thinnest part (60 μm; 60 μm; 70 μm; average: (60 + 60 + 70) / 3 = 63.3 μm)

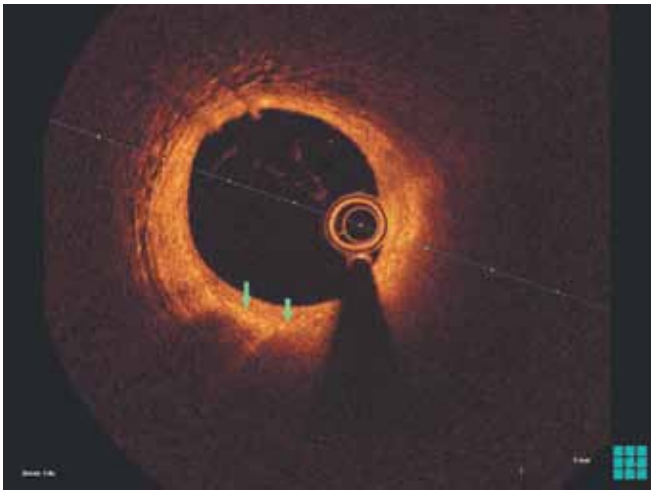

Fig. 2. Cluster of macrophage foam cells (OCT) marked with green arrows: linear areas of high intensity with a "shadow" behind

IVUS and OCT, presented the evidence base for the use of intravascular imaging methods, described in detail the technique of interventions, obtaining and analyzing images, as well as indications and contraindications for IVUS and OCT [11]. The second part of the conciliation document is likely to be published, since OCT is now actively developing and allows one to evaluate many parameters of the morphology of coronary arteries before and after installation of stents and scaffolds. Of all the currently existing intravascular imaging techniques – near-infrared spectroscopy (NIRS), IVUS, IVUS with virtual histology, iMAP-IVUS – OCT has the highest diagnostic value [4].

One of the main problems with cardiorenal syndrome is high mortality from cardiovascular diseases in which ischemic heart disease leads [12, 13]. The above facts justify the use of OCT in CRS patients in detecting vulnerable plaques that are susceptible to compromised fibrous cap, leading to thrombosis, and as a result ACS. Such plaques should be detected in a patient's stable condition.

OCT is the only intravascular imaging technique whose axial resolution allows one to estimate the thickness of the plaque cap (Fig. 1, b) [14], which can be affected by statin administration [15]. Statins also have pleiotropic effect, which manifests itself particularly through decreased severity of macrophage inflammation (Fig. 2) [16, 17].

However, estimating the fibrous cap thickness is not enough to classify the plaque as a thin-cap plaque, since there is also the need to evaluate the lipid core volume (Fig. 1, a). Xing et al. demonstrated that the presence of lipid-rich plaques doubles the risk for adverse cardiac events [18].

The presence of vasa vasorum (Fig. 3) and cholesterol crystals (Fig. 4) in the plaque increases the likelihood of plaque disruption. The sensitivity and specificity of OCT to detect plaque neovascularization compared with pathological data are 52% and 68%, respectively [19]. Nakamura et al. showed that lipid-rich plaque is significantly higher in patients with cholesterol crystals [20]. Dai et al. demonstrated that cholesterol crystals are more common in patients with acute ST-elevation myocardial infarction as compared with patients with non-ST elevation acute coronary syndrome (50.8% vs. 34.7%, respectively) [21].

Plaque erosion is the cause of sudden cardiac death in about 30–40% of cases [22, 23]. It has been shown that there is higher prevalence of plaque erosion in younger patients (<50 years old) and it is more often detected in the left coronary artery (LCA). The study revealed a relationship between plaque erosion and CKD. A classification of risk factors for erosion has also been proposed. The classification takes into account both clinical data and data that can only be obtained via OCT, such as presence of a thin cap [24].

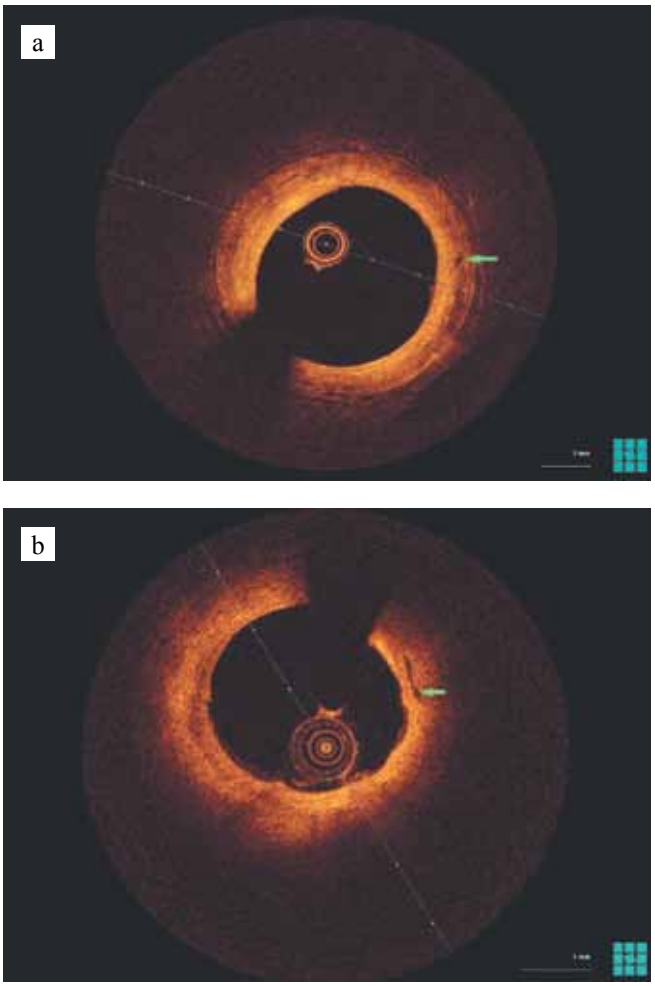

Fig. 3. Microchannels inside the atherosclerotic plaque (marked with a green arrow). OCT

Fig. 4. Cholesterol crystals inside the plaque (marked with a green arrow). OCT

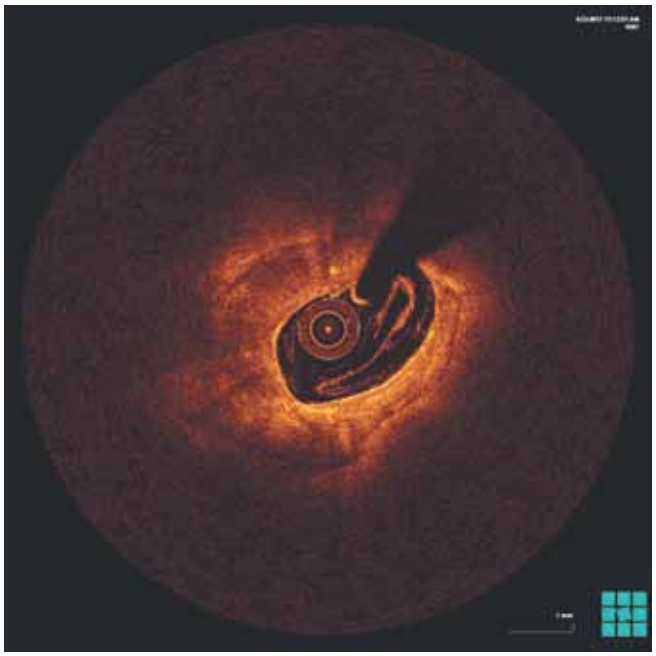

Fig. 5. Calcified nodule inside the atherosclerotic plaque (OCT). Calcifications are determined along the entire circumference of the vessel lumen (4 quadrants)

Calcification is one of the ACS development mechanisms (Fig. 5). In patients with even initial signs of CKD, calcifications are more likely to be detected as compared to the general population. Calcification is becoming more pronounced as renal dysfunction progresses. It is independently associated with cardiovascular mortality. Calcification is most pronounced in patients with end-stage renal disease (ESRD) [25]. It has been demonstrated that the number of calcifications distorting the vessel lumen is higher in patients with acute ST-elevation myocardial infarction [26].

There are few studies on coronary artery atherosclerosis in CKD patients. These studies are heterogeneous. Reports published to date significantly differ in methodology and criteria for inclusion of patients. Therefore, the results of these studies also differ [11, 27, 28].

Kato et al. conducted a study of the morphological characteristics of coronary atherosclerotic plaques using OCT among CKD and non-CKD patients. CKD was defined as eGFR <60 mL/min per 1.73 m^2 calculated using the Modification of Diet in Renal Disease (MDRD) equation. When lipid was present in ≥90° in any of the cross-sectional images within the plaque, it was considered to be a lipid-rich plaque. In lipid-rich plaques, the lipid arc was measured on the cross-sectional view at 1-mm intervals over the entire length, and the values were all averaged. In addition, the lipid length, lipid index (mean lipid arc multiplied by lipid length, mm°), and the fibrous cap thickness were measured. The authors also studied the presence of thin-cap fibroatheroma (a lipid-rich plaque with fibrous cap thickness ≤65 μm at the thinnest part), calcifications, macrophage accumulations, cholesterol crystals, microchannels, plaque disruption,

and intracoronary thrombus (divided into "white" and "red"). It was shown that compared with non-CKD patients, plaques in the CKD patients had a wider lipid arc, longer lipid length and a larger lipid index. In addition, calcifications, cholesterol crystals, and plaque disruption were more prevalent in CKD patients. Prevalence of lipid-rich plaques, thin-cap fibroatheroma, macrophage accumulations, microchannels, and thrombus was similar between the groups. Lower GFR and the presence of diabetes were independently associated with a larger lipid index [29].

In a study by Dai et al. decreased renal function was determined by changes in GFR levels. Creatinine level, cystatin C level and both indicators were used in calculating the eGFR. Patients were divided into 3 groups according to the eGFR calculated: the 1st group consisted of patients with GFR ≥90 mL/min/1.73 m^2, second – GFR 60–89 mL/min/1.73 m^2, third – GFR <60 mL/min/1.73 m^2. The fibrous cap thickness varied in all patient groups. The average lipid arc, lipid length and lipid index were different only when comparing groups 1 and 2, and groups 1 and 3. The number of plaques with cholesterol crystals significantly differed only when comparing groups 1 and 2. The number of plaques with calcification significantly differed only when comparing groups 1 and 3 [30].

In a study by Chin et al., hemodialysis patients who underwent OCT imaging were compared 1:1 with non-CKD patients. CKD absence was defined as eGFR >60 mL/min per 1.73 m^2, as well as absence of any signs of kidney injury. Analysis of the culprit plaques showed that the main and control groups of patients differed in the mean calcium arc and maximum calcium arc. Analysis of non-culprit plaques showed that the main and control groups of patients also had different mean calcium arc and maximum calcium arc. The authors conducted additional analysis of the main group of patients, dividing the latter into tertiles according to hemodialysis duration. Analysis of the culprit plaques demonstrated that the patient subgroups differed in mean calcium arc and maximum calcium arc, as well as number of thin intimal calcium, which was defined as an arc of calcium >30° within intima <0.5 mm thick. In the analysis of non-culprit plaques, the patient subgroups differed only in the mean calcium arcs [27].

Sugiyama et al. studied the morphological characteristics of native plaques in patients that were divided into 3 groups according to the values of eGFR calculated by a formula adapted for a Japanese population: the non-CKD group (eGFR ≥60 mL/min/1.73 m^2), CKD group (15 ≤ GFR < 60 mL/min/1.73 m^2) and ESRD group (GFR <15 mL/min/1.73 m^2 and/or hemodialysis). To count some parameters, the CKD and non-CKD groups were combined into a non-ESRD group. The CKD group had a larger lipid arc, longer lipid length and higher prevalence of lipid-rich plaque than the non-CKD group. The ESRD group had a thinner fibrous cap, higher prevalence of plaque rupture, and larger calcification arc than the non-ESRD group. Age, diabetes, and hemodialysis, but not GFR, were independently associated with the presence of calcified plaques [31].

Minami et al. investigated 140 non-culprit plaques in 84 patients with coronary artery disease who were treated with a statin and had two optical coherence tomography imaging: at first admission and 6 months after. Response of thin-cap area (fibrous cap thickness <200 μm) to statin therapy was the criterion. CKD was defined as eGFR <60 mL/min per 1.73 m^2 calculated using the MDRD equation. Compared with the initial OCT, a follow-up OCT revealed that there was a decrease in thin-cap area, average lipid arc, maximum lipid arc, lipid length, lipid index, size of stenosis by area and size of macrophage accumulations. Fibrous cap thickness at the thinnest part increased. Patients with larger thin-cap area at baseline OCT had a more significant reduction in thin-cap area at a follow-up OCT. Compared with those who initially took statins, patients who had not previously taken statins had a more significant reduction in thin-cap area at a follow-up OCT. CKD was a predictor for unfavorable response to statin therapy, while ACS at first hospitalization was a predictor for favorable vascular response to statin therapy [32].

ESRD patients are more likely to be detected with multivessel coronary artery disease and plaques with increased media thickness, activation of macrophages and marked calcification [33]. Most of these factors can be detected via OCT imaging, but such patients have a particularly high risk of developing contrast-induced nephropathy. In order to solve this problem, crystalloid or colloidal solutions (or their mixture) are used in place of an X-ray contrast agents, which avoids deterioration of kidney function. Karimi Galougahi et al. published a clinical case where a patient with advanced CKD (creatinine = 4.5 mg/dL [397.8 μmol/L], eGFR = 13 mL/min/1.73 m^2) not requiring haemodialysis had his LCA and diagonal artery successfully imaged with OCT. An optically clear medium was created by a mixture of saline and colloid infusate. Post-OCT renal function remained stable [34]. Unfortunately, the above study contains limited information on the features of atherosclerotic coronary lesions in this patient and the solution used. Azzalini et al. published a detailed description of a clinical case where a stage IV SD and CKD patient (GFR 16 ml/min/1.73 m^2) had his LCA and diagonal artery successfully imaged with OCT. An optically clear medium was created with a Dextran 40 colloidal solution at an infusion rate of 4.0 mL/min, the total amount of solution introduced being 14.0 mL. Kidney function remained stable during the patient's hospitalization [35]. Koga et al. published a clinical case where a patient receiving hemodialysis was visualized *in vivo* with calcified thrombus by OCT, IVUS and angioscopy. The authors did not indicate which solution was used to create the optically clear medium [36]. Ozaki et al. enrolled 22 pati-

ents with 25 coronary stented lesions in their study. Each patient was subjected to OCT using a contrast medium and OCT using a mixture of dextran 40 and lactated Ringer's solution. It was demonstrated that the number of segments available for analysis did not differ between OCT using contrast medium and OCT using a mixture of dextran 40 and lactated Ringer's solution (97.9% vs. 96.5%, respectively) [37].

CONCLUSION

In patients with impaired renal function, OCT permits more accurate assessment of the morphology of plaques, inflammation severity and "vulnerability" of plaques in general. OCT allows to clearly visualize calcification and evaluate its severity with high accuracy. Only OCT can enable one to see plaque erosion and distinguish it from plaque rupture. This may be important for determining further patient management tactics, such as revascularization feasibility. If revascularization is the choice, the decision then shifts to optimal choice of a stent or scaffold. There are major limitations on the use of OCT in CKD patients due to the risk of developing contrast-induced nephropathy. Therefore, further improvement on the technique for replacing blood with an optically clear medium is needed. One of the crucial tasks at present is to carry out large-scale multicenter studies to clarify the possibility of detecting vulnerable atherosclerotic plaques via routine use of OCT and identifying the possibility of reducing ACS risk. Also critical is to identify the impact on patient survival without adverse events as one of the underlying goals of any intervention.

The authors declare no conflict of interest.

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