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Ablation of atypical atrial flutter in a patient after radiotherapy for adenocarcinoma of the right lung using the Coherent CARTOPRIME™ module of the CARTO system

Ablacja atypowego trzepotania przedsionków u pacjenta po radioterapii gruczolakoraka płuca prawego z wykorzystaniem modułu Coherent CARTOPRIME™ systemu CARTO

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Abstract

Atrial flutter (AFl) is the second most frequent persistent supraventricular arrhythmia, after atrial fibrillation (AF). In the most common type of AFl the circuit is localized in the right atrium and it is cavotricuspid isthmus dependent, what is termed typical. In atypical AFl the wave front does not go around the tricuspid annulus. It is often associated with prior cardiac surgery or ablation for AF, including linear lesions or defragmentation, where iatrogenic scar serves as the electrophysiologic substrate for reentry. The number of cases, when the circuit is related to a spontaneous low-voltage zone, in the absence of any previous atrial procedures is limited. The reasons behind it might be a significant heart disease, such as mitral valve dysfunction, impaired diastolic function or hypertension, which lead to fibrosis and functional regions of slow or no conduction (SNO, slow or no conduction zone. However, it is still not well understood how electrically silent areas occur in patients without risk factors mentioned above. We present a case report of a patient who suffered damage to the left atrial wall during radiotherapy treatment, and atypical AFl was induced on the basis of the resulting scar.

Key words: atypical atrial flutter, catheter ablation, radiotherapy, lung cancer

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Introduction

Atrial flutter (AFl) is the second most frequent persistent supraventricular arrhythmia, after atrial fibrillation (AF). In the most common type of flutter, the circuit is localized in the right atrium and it is cavotricuspid isthmus dependent, what is termed typical [1]. In atypical AFl the wave front does not go around the tricuspid annulus. It is often associated with prior cardiac surgery or ablation for AF, including linear lesions or defragmentation, where iatrogenic

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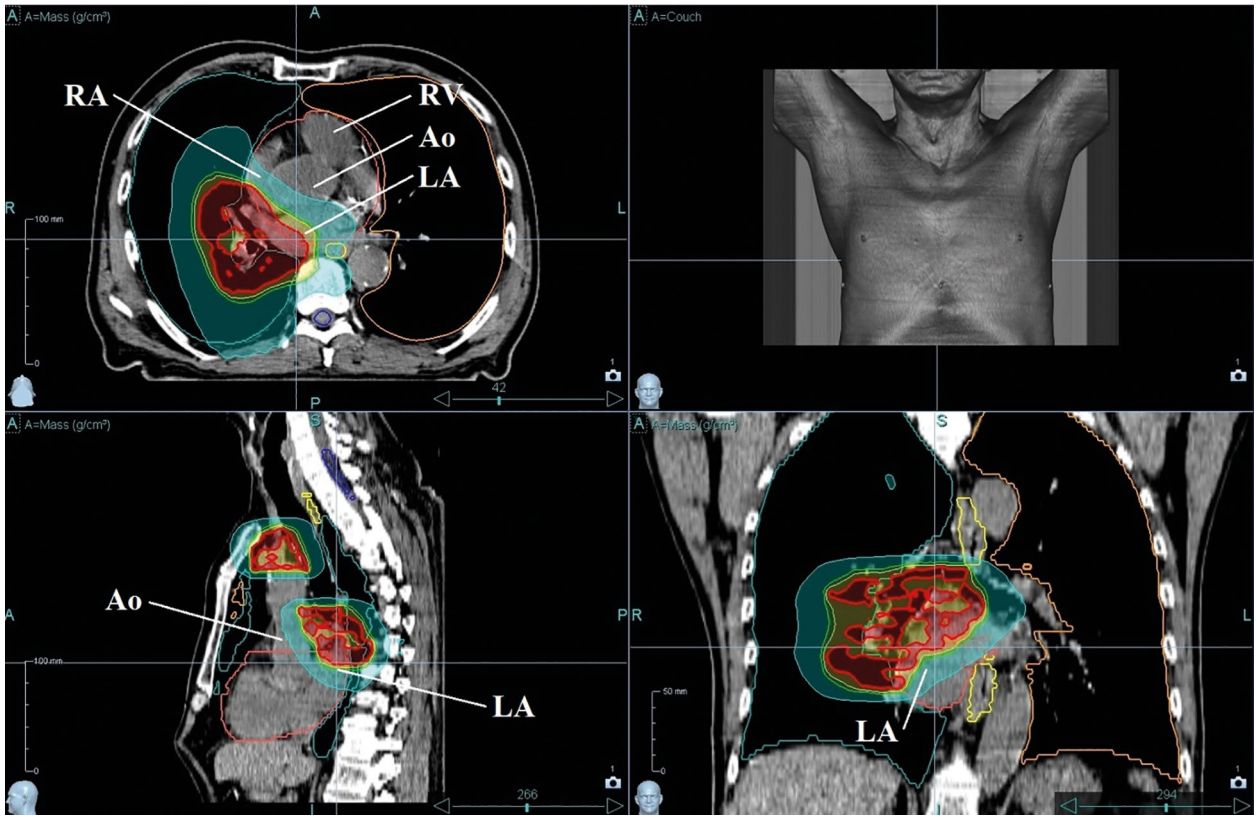

Figure 1. Radiotherapy treatment planning. Red isodose line shows volume receiving dose of 66 Gy or more (Accuray Precision® v2.0.1.1.); Ao — ascending aorta; LA — left atrium; RA — right atrium; RV — right ventricle

scar serves as the electrophysiologic substrate for reentry [1, 2]. The number of cases, when the circuit is related to a spontaneous low-voltage zone (LVZ), in the absence of any previous atrial procedures is limited [3]. The reasons behind it might be a significant heart disease, such as mitral valve dysfunction, impaired diastolic function or hypertension, which lead to fibrosis and functional regions of slow or no conduction (SNO, slow or no conduction zone) [1]. However, it is still not well understood how electrically silent areas occur in patients without risk factors mentioned above [2, 4].

Case report

A 65-year-old patient, who had undergone sequential chemo- and radiotherapy for the adenocarcinoma of the right lung with clinical stage IIIA disease (cT1bN2M0), was admitted to the Cardiology Department due to next episode of atypical AFI. In the patient's medical history, he had undergone successful electrical cardioversion for the same arrhythmia 3 months earlier. The patient had a history of coronary heart disease, having suffered an inferior wall myocardial infarction and undergone right coronary artery angioplasty with the implantation of a drug-eluting stent 12 years before, as well as having heart failure with mildly reduced ejection fraction, an abdominal aortic aneurysm, peripheral arterial disease, nicotinism, hypertension, hyperlipidemia, and hyperthyroidism during treatment. The patient had undergone bladder tumor resection 3 years prior. Patient received oncological treatment, which consisted of 5 cycles of chemotherapy (carboplatin with vinorelbine) and sequentially given radiotherapy. Irradiated volume included right lung tumor, right hilum, subcarinal and right paratracheal lymph nodes. Prescribed dose was 66 Gy in 33 fractions. Patient was treated in helical tomotherapy technique. Mean heart dose was 12.54 Gy and volume receiving more than 30 Gy was 11% (Figure 1). Patient ended radiotherapy 6 months before admission to Cardiology Department without serious adverse events. On admission to Cardiology Department, an electrocardiogram showed atypical AFI with an atrial cycle length of approximately 210ms and a ventricular rate of about 114 per minute (Figure 2). Due to the recurrence and symptoms of the patient's arrhythmia, after ruling out thrombotic material in the left atrium, he was qualified for ablation of the arrhythmia substrate. Using a 3D CARTO electroanatomic system (Biosense Webster, Inc., Diamond Bar, CA, USA) and PentaRay electrode (Biosense Webster,

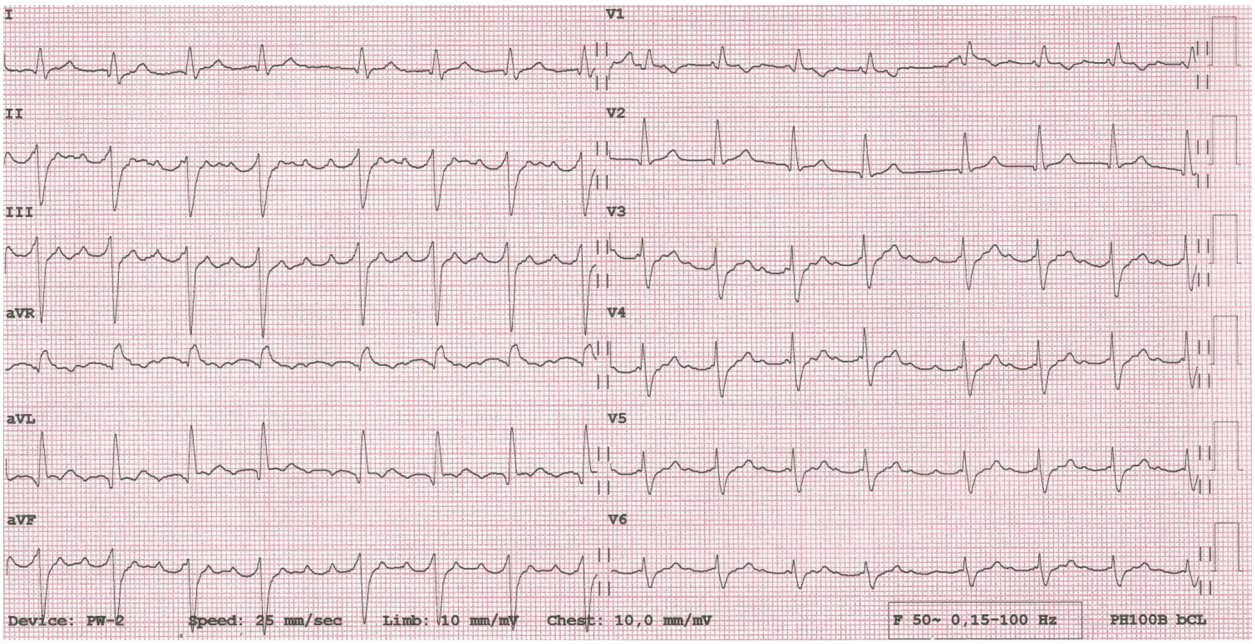

Figure 2. Electrocardiogram with atypical atrial flutter

Inc., Diamond Bar, CA, USA), the left atrium was mapped and SNO was located on the posterior wall of the atrium near the right pulmonary vein ostia. The propagation of AFl activation was then determined using the Coherent CARTOPRIME™ module of the CARTO system, and the critical isthmus of the arrhythmia was located on the roof of the left atrium near the right superior pulmonary vein ostium (Figure 3). Two applications of 40W of radiofrequency energy were then delivered to the site of the critical isthmus of the atypical AFl using a Smarttouch SF electrode (Biosense Webster, Inc., Diamond Bar, CA, USA), resulting in the termination of the arrhythmia and the restoration of sinus rhythm (Figures 4 and 5). Due to the location of the SNO near the pulmonary vein ostia and the risk of recurrence of atypical AFl, additional applications were made around the right pulmonary veins to isolate them and connect the SNO with the isolated right pulmonary veins. An electrophysiological study and aggressive atrial stimulation were then performed, but no arrhythmia was induced, so the left pulmonary veins were not isolated. No recurrence of atrial arrhythmias was observed during a 12-month follow-up observation period.

Discussion

The number of studies on radiotherapy-induced cardiotoxicity for patients with lung cancer is limited. This is mainly due to the fact that side effects of radiotherapy occur many years after treatment [5], while the 5-year survival rate for patients undergoing radio- and chemotherapy for non-small cell lung cancer is low.

The statement of the European Society of Cardiology published in 2016 on the treatment of cancer and cardiovascular toxicity states that arrhythmias occur in 16–36% of patients after radiotherapy. The problem is to determine the direct impact of radiotherapy alone on the occurrence of cardiac events. It is because of the frequent comorbidities, the direct impact of the cancer itself and additional chemotherapeutic treatment [6].

Our case report suggests the possibility of forming a low-voltage zone in the atrium due to radiotherapy. This can be a background for macro reentry arrhythmia, secondary to fibrosis caused by thoracic irradiation. Ischaemia because of artery branch occlusion, inflammation, atrial dysplasia may stand behind regional electrical conduction disturbances [7, 8]. However, AF is a more frequent occurrence in the fibrotic area [9]. In addition, a very few studies have singled out atypical AFl among episodes of arrhythmia after radiotherapy. That might be because the differentiation between atypical AFl and AF is not clear until performing intracardiac electrophysiology study.

The coexistence of coronary artery disease, hypertension, heart failure in our patient is also associated with a higher risk of AFl [1].

Radiotherapy treatment plan review revealed that significant part of planning target volume, which received full prescribed dose, was overlapping with right and posterior part of left atrium (Figure 1). High dose regions in this particular volume are potentially responsible for arrhythmia [10]. Mechanism of cardiac toxicity is complex and includes molecular, inflammatory, vascular, immunological and fibrotic mechanism [11].

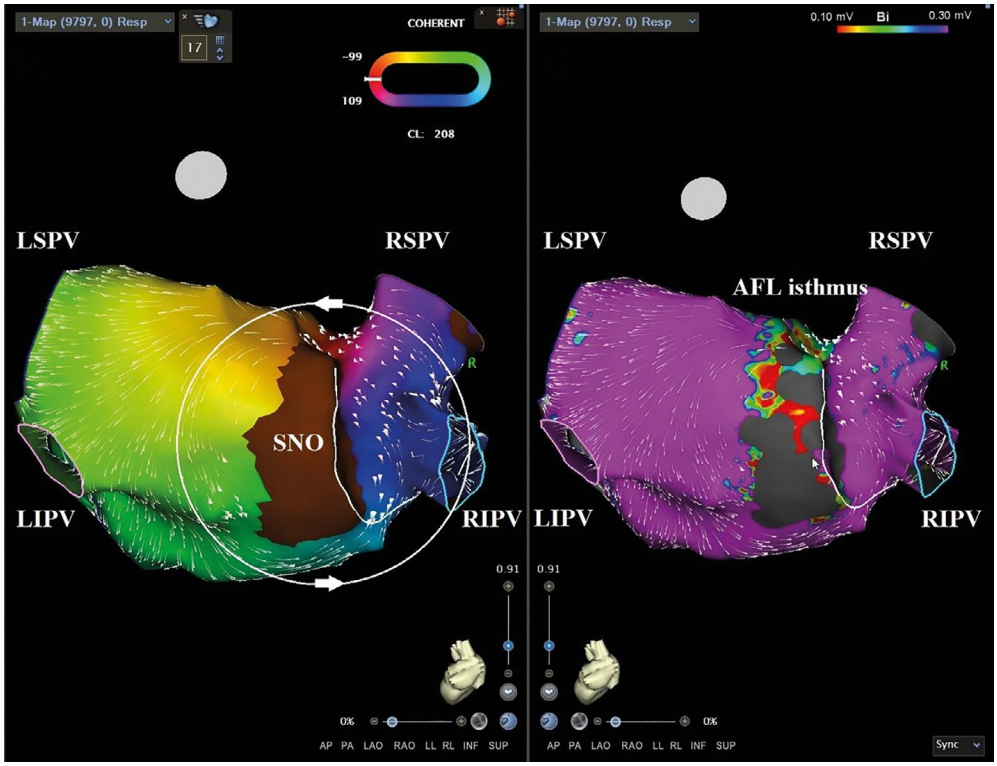

Figure 3. Electroanatomical map of the left atrium of the heart: **A.** Activation map of atypical atrial flutter with the Coherent Mapping module. Macro-reentry loop around the scar zone on the posterior wall of the left atrium near the RSPV and RIPV ostia with the critical isthmus in the roof of the left atrium at the RSPV ostium; **B.** Voltage map of left atrium; LIPV — left inferior pulmonary vein; LSPV — left superior pulmonary vein; RIPV — right inferior pulmonary vein; RSPV — right superior pulmonary vein; SNO — slow or no conduction zone

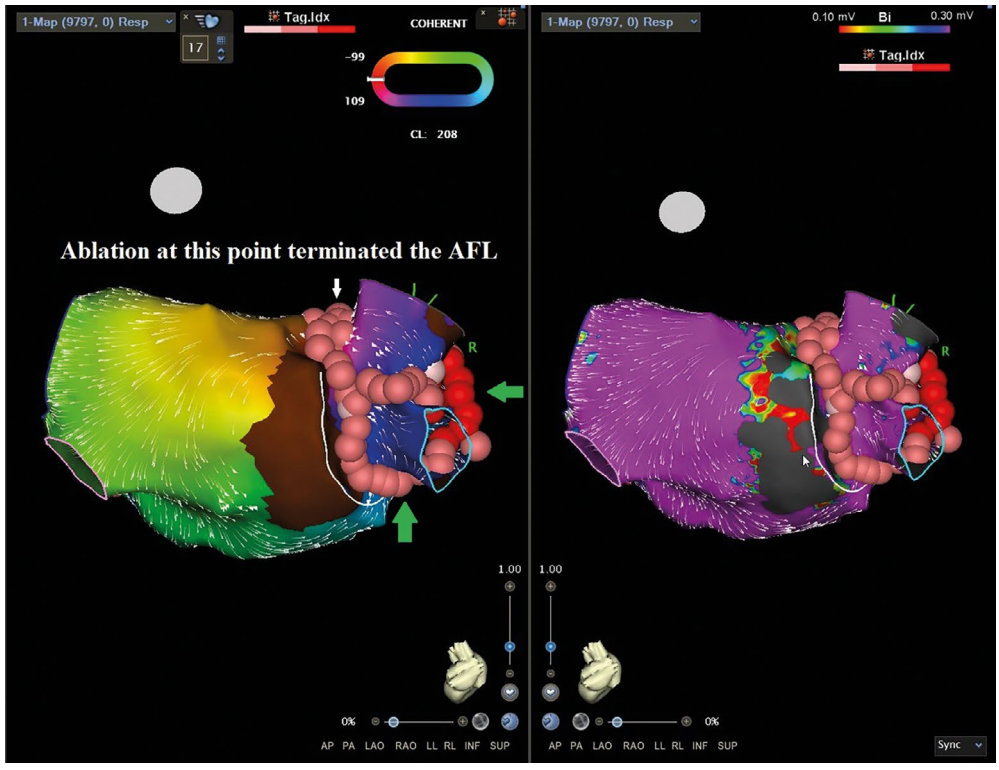

Figure 4. Radiofrequency catheter ablation points around the right pulmonary veins. The first applications performed in the critical isthmus of atypical atrial flutter (AFL) terminated arrhythmia (white arrow). Subsequent applications (green arrows) were then made to isolate the right pulmonary veins to reduce the risk of recurrence of typical AFL

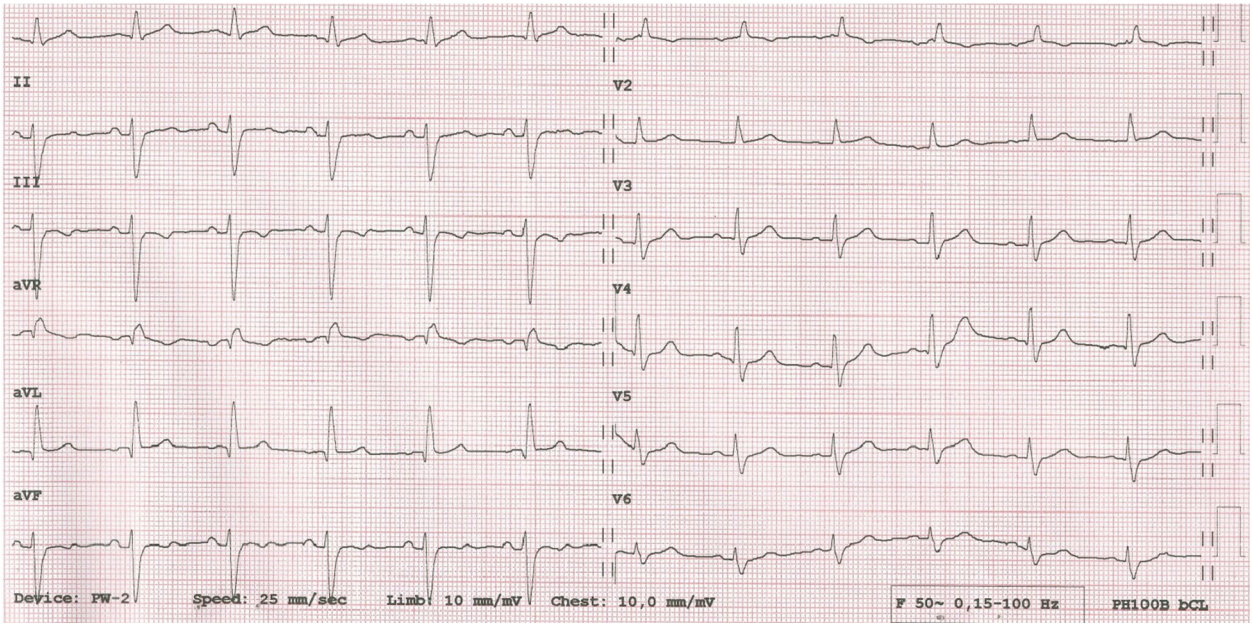

Figure 5. Sinus rhythm 72 per minute after ablation

In research of Vaidya et al. [12] it is reported that among patients with an incident of AF previously treated with radiotherapy for cancer, the frequency of concomitant AFl was higher than in the general AF population. What is more, patients treated with radiotherapy were less likely to have a history of heart failure, but had a similar rate of coronary artery disease.

Another study describing the occurrence of AFl flutter in a group of 112 patients who underwent radiotherapy for stage III non-small cell carcinoma was study by Wang et al. [13]. In this study, 26 (23.2%) patients experience a cardiac event, including 12 (8.9%) who had an arrhythmia. The study also focused on the effect of radiation dose on the rate of cardiac events. It has been shown that a mean dose above 20 Gy significantly increases the risk of cardiotoxic effects.

In a retrospective study conducted between 2010 and 2015 by Borkenhagen et al. [14] in a group of 76 patients, an association between radiation doses to individual heart structures and the appearance of cardiotoxicity was found. In this study atrial arrhythmias were found in 5 (6.6%) patients.

In a study by Dess et al. [15] in a group of 125 patients, 37 (29.6%) had cardiac events, including 11 (8.8%) arrhythmias. However, in the study group, 84% were undergoing chemotherapy and 27% had pre-existing heart disease, which may have influenced the development of myocardial conduction disturbances.

Errahmani et al. [16] conducted a study in which they assessed the occurrence of cardiac arrhythmias in breast cancer patients treated with radiotherapy with a radiation dose to the whole heart, left and right atrium, and right and left ventricle. In this study, despite the insignificant results, the authors emphasized that patients with right-sided breast cancer and irradiated right atrium may require special attention due to the risk of arrhythmias and conduction disturbances.

Hashiguchi et al. [17] demonstrated that in patients with breast cancer treated with radiotherapy, there is a tendency to a greater LVZ in the left atrium. However, the existence of LVZ did not differ significantly between the breast cancer cohort and the control group.

Article information

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None.

Author contributions
PW — 70%; JW — 10%; MD — 10%; MM — 1%; PD — 1%; RS — 3%; BWK — 5%.

Conflict of interest
The authors declare no conflict of interest.

Ethics statement
The case report did not require approval from the Bioethics Committee. Data in the manuscript are anonymised.

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Supplementary material
None.

Streszczenie

Trzepotanie przedsionków (AFl) jest drugą po migotaniu przedsionków (AF) pod względem częstości występowania przetrwałą arytmią nadkomorową. W najczęstszym typie AFl obwód arytmii zlokalizowany jest w prawym przedsionku, jest zależny od cieśni trójdzielno-żylnej (CTI), a określa się go mianem typowego. W atypowym AFl obwód arytmii nie jest zależny od CTI. Atypowe AFl często związane z wcześniejszą operacją kardiochirurgiczną lub ablacją AF, w tym linijnymi zmianami lub defragmentacją, gdzie jatrogenna blizna służy jako elektrofizjologiczne podłoże do stworzenia obwodu arytmii. W praktyce klinicznej rzadko dochodzi samoistnego tworzenia stref nieskonapięciowych w ścianie przedsionków. W większości przypadków przyczyną może być choroba serca, taka jak dysfunkcja zastawki mitralnej, upośledzona funkcja rozkurczowa lub nadciśnienie tętnicze, które prowadzą do zwłóknienia i tworzenia obszarów czynnościowych o wolnym lub zerowym przewodzeniu potencjału elektrycznego. Jednakże nie jest jasne, w jaki sposób powstają strefy niskonapięciowe w ścianie przedsionków u pacjentów bez czynników ryzyka przedstawionych powyżej. W niniejszej pracy przedstawiono przypadek pacjenta, u którego doszło do uszkodzenia ściany lewego przedsionka podczas leczenia radioterapią, a na podłożu powstałej blizny doszło do indukcji atypowego AFl.

Słowa kluczowe: atypowe trzepotanie przedsionków, ablacja, radioterapia, rak płuca

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