DOI: https://doi.org/10.17816/DD635007

Potential use of cardiac magnetic resonance imaging in differential diagnosis of cardiomyopathies due to light-chain amyloidosis and transthyretin amyloidosis

Zainab M. Magomedova[1,2], Tatyana V. Nikiforova[3], Dmitry Yu. Shchekochikhin[1,2], Ekaterina S. Pershina[1,2], Konstantin V. Kovalev[1], Khadizhar S. Abdulmazhidova[2], Daria S. Rassechkina[2], Alexander E. Grachev[4], Irina G. Rekhtina[4], Alexey N. Volovchenko[2], Susanna D. Sarkisyan[2], Valentin E. Sinitsyn[5], Denis A. Andreev[2]

[1] Pirogov Municipal Clinical Hospital № 1, Moscow, Russia;
[2] Sechenov First Moscow State Medical University, Moscow, Russia;
[3] S.S. Yudin City Clinical Hospital, Moscow, Russia;
[4] National Medical Research Center of Hematology, Moscow, Russia;
[5] Lomonosov Moscow State University, Moscow, Russia

ABSTRACT

BACKGROUND: Cardiac amyloidosis is a serious progressive disease with a high mortality rate. The differential diagnosis of cardiomyopathies due to amyloid light-chain (AL) amyloidosis and transthyretin (ATTR) amyloidosis is important for selecting the optimal treatment strategy.

AIM: The aim of this study was to evaluate the capabilities of cardiac magnetic resonance imaging in the differential diagnosis of cardiomyopathies due to AL and ATTR amyloidosis.

MATERIALS AND METHODS: A retrospective analysis of the medical records of 25 patients with a confirmed diagnosis of amyloid cardiomyopathy was performed. Patients were divided into two groups according to the type of amyloidosis, with group 1 including patients with cardiomyopathy due to AL amyloidosis and group 2 including patients with cardiomyopathy due to ATTR amyloidosis. All patients underwent contrast-enhanced cardiac magnetic resonance imaging. Volumetric and linear cardiac parameters, ventricular function, and late gadolinium enhancement patterns were assessed. Standard statistical methods were used, and differences were considered significant at p <0.05.

RESULTS: Group 2 showed a more significant thickening of the myocardial walls compared to group 1 (interventricular septum: 18 [17; 18] vs. 14.5 mm [12.8; 16.0], p <0.01, posterior wall of the left ventricle: 14 [13; 17] vs. 10.5 mm [10; 12.3], p <0.01). The indexed mass of the left ventricle myocardium was 110 [92; 125] in group 2 and 85 mm [69.3; 91.8] in group 1 (p <0.01). In group 2, late gadolinium enhancement with a transmural left ventricle pattern was more frequently observed in the basal and mid-lower-lateral segments, whereas in group 1, a subendocardial pattern of late gadolinium enhancement was more frequent in the mid-anterior and lower-lateral segments (p <0.05). In addition, frequency of simultaneous contrast enhancement in the subendocardial layers of the interventricular septum on the left ventricle and right ventricle sides was higher in group 2 (100% of cases vs. 50%, p <0.01). Late gadolinium enhancement of the right ventricle was also more common in group 2 (100 vs. 58%, p <0.05), especially in the interventricular septum and inferior wall area (p <0.05). Semi-quantitative assessment of LGE using the Query Amyloid Late Enhancement (QALE) showed greater contrast enhancement in group 2: 13 [12; 14] vs. 10.5 [1.75; 12], p <0.01), and a score greater than 13 differentiated between cardiomyopathy due to AL amyloidosis and ATTR amyloidosis with a sensitivity of 69% and a specificity of 83%.

CONCLUSION: Cardiac MRI identifies typical features of cardiomyopathies due to AL amyloidosis and ATTR amyloidosis for their differential diagnosis. Further research is needed to confirm diagnostic accuracy of the patterns identified.

Keywords: cardiac amyloidosis; systemic amyloidosis; cardiac magnetic resonance imaging; late gadolinium enhancement; QALE; transthyretin amyloidosis; light-chain amyloidosis.

To cite this article:
Magomedova ZM, Nikiforova TV, Shchekochikhin DYu, Pershina ES, Kovalev KV, Abdulmazhidova KhS, Rassechkina DS, Grachev AE, Rekhtina IG, Volovchenko AN, Sarkisyan SD, Sinitsyn VE, Andreev DA. Potential use of cardiac magnetic resonance imaging in differential diagnosis of cardiomyopathies due to light-chain amyloidosis and transthyretin amyloidosis. *Digital Diagnostics.* 2024;5(4):668–681. DOI: https://doi.org/10.17816/DD635007

Received: 08.09.2024 **Accepted:** 24.10.2024 **Published online:** 12.11.2024

ECO•VECTOR

Article can be used under the CC BY-NC-ND 4.0 International License
© Eco-Vector, 2024

DOI: https://doi.org/10.17816/DD635007

Возможности магнитно-резонансной томографии сердца в дифференциальной диагностике кардиомиопатий вследствие амилоидоза лёгких цепей и транстиретинового амилоидоза

З.М. Магомедова[1,2], Т.В. Никифорова[3], Д.Ю. Щекочихин[1,2], Е.С. Першина[1,2], К.В. Ковалёв[1], Х.С. Абдулмажидова[2], Д.С. Рассечкина[2], А.Е. Грачев[4], И.Г. Рехтина[4], А.Н. Воловченко[2], С.Д. Саркисян[2], В.Е Синицын[5], Д.А. Андреев[2]

[1] Городская клиническая больница № 1 имени Н.И. Пирогова, Москва, Россия;
[2] Первый Московский государственный медицинский университет имени И.М. Сеченова, Москва, Россия;
[3] Городская клиническая больница имени С.С. Юдина, Москва, Россия;
[4] Национальный медицинский исследовательский центр гематологии, Москва, Россия;
[5] Московский государственный университет имени М.В. Ломоносова, Москва, Россия

АННОТАЦИЯ

Обоснование. Амилоидоз сердца — серьёзное прогрессирующее заболевание с высокой смертностью. Дифференциальная диагностика кардиомиопатий вследствие амилоидоза лёгких цепей (AL-амилоидоз) и транстиретинового амилоидоза (ATTR-амилоидоз) важна для выбора оптимальной тактики лечения.

Целью исследования является оценка возможностей магнитно-резонансной томографии сердца в дифференциальной диагностике кардиомиопатий вследствие AL- и ATTR-амилоидоза.

Материалы и методы. Проведён анализ медицинских данных 25 пациентов с подтверждённым диагнозом кардиомиопатия, разделённых на две группы в зависимости от типа амилоидоза. 1-я группа — кардиомиопатия вследствие AL-амилоидоза, 2-я группа — вследствие ATTR-амилоидоза. Всем пациентам проведена магнитно-резонансная томография сердца с контрастированием. Оценивали объёмные и линейные показатели сердца, функцию желудочков и паттерны позднего накопления гадолиния. Использовали стандартные статистические методы, различия считали значимыми при p <0,05.

Результаты. У пациентов 2-й группы наблюдали более выраженное утолщение стенок миокарда в сравнении с пациентами 1-й группы (межжелудочковая перегородка 18 [17; 18] против 14,5 мм [12,8; 16], p <0,01, задняя стенка левого желудочка 14 [13; 17] против 10,5 мм [10; 12,3], p <0,01). Индексированная масса миокарда левого желудочка во 2-й группе — 110 г/м^2 [92; 125], тогда как в 1-й группе данный показатель составил 85 г/м^2 [69,3; 91,8], p <0,01). Среди пациентов 2-й группы чаще отмечали позднее накопление гадолиния с трансмуральным паттерном в базальном и среднем нижне-боковых сегментах левого желудочка, в то время как у пациентов 1-й группы — чаще определяли субэндокардиальный паттерн позднего накопления гадолиния в средних передне- и нижне-боковых сегментах (p <0,05). Также у пациентов 2-й группы частота случаев одновременного накопления контрастного препарата в субэндокардиальных слоях межжелудочковой перегородки со стороны левого желудочка и правого желудочка оказалась выше (100% случаев против 50%, p <0,01). Позднее накопление гадолиния в правом желудочке также чаще встречали среди пациентов 2-й группы (100% против 58%, p <0,05), особенно в области межжелудочковой перегородки и нижней стенки (p <0,05). Полуколичественная оценка позднего накопления гадолиния с помощью показателя QALE (The query amyloid late enhancement) показала более обширное накопление контраста у пациентов 2-й группы — 13 [12; 14] против 10,5 баллов [1,75; 12], p <0,01), а количество баллов более 13 предоставило возможность различить кардиомиопатии вследствие AL- и ATTR-амилоидоза с чувствительностью 69% и специфичностью 83%.

Заключение. Магнитно-резонансная томография сердца позволяет выявлять характерные особенности кардиомиопатий вследствие AL- и ATTR-амилоидоза, что может помочь в их дифференциальной диагностике. С целью подтверждения диагностической точности, выявленных паттернов, необходимо продолжение исследований.

Ключевые слова: амилоидоз сердца; системный амилоидоз; магнитно-резонансная томография сердца; отсроченное контрастирование гадолинием; QALE; транстиретиновый амилоидоз; амилоидоз лёгких цепей.

Как цитировать:
Магомедова З.М., Никифорова Т.В., Щекочихин Д.Ю., Першина Е.С., Ковалёв К.В., Абдулмажидова Х.С., Рассечкина Д.С., Грачев А.Е., Рехтина И.Г., Воловченко А.Н., Саркисян С.Д., Синицын В.Е., Андреев Д.А. Возможности магнитно-резонансной томографии сердца в диагностике кардиомиопатий вследствие амилоидоза лёгких цепей и транстиретинового амилоидоза // Digital Diagnostics. 2024. Т. 5. № 4. С. 668–681. DOI: https://doi.org/10.17816/DD635007

Рукопись получена: 08.09.2024 **Рукопись одобрена:** 24.10.2024 **Опубликована online:** 12.11.2024

ЭКО•ВЕКТОР

Статья доступна по лицензии CC BY-NC-ND 4.0 International
© Эко-Вектор, 2024

DOI: https://doi.org/10.17816/DD635007

心脏磁共振成像在肺链式淀粉样变性和转甲状腺素淀粉样变性引起的心肌病鉴别诊断中的可能性

Zainab M. Magomedova[1,2], Tatyana V. Nikiforova[3], Dmitry Yu. Shchekochikhin[1,2], Ekaterina S. Pershina[1,2], Konstantin V. Kovalev[1], Khadizhar S. Abdulmazhidova[2], Daria S. Rassechkina[2], Alexander E. Grachev[4], Irina G. Rekhtina[4], Alexey N. Volovchenko[2], Susanna D. Sarkisyan[2], Valentin E. Sinitsyn[5], Denis A. Andreev[2]

[1] Pirogov Municipal Clinical Hospital № 1, Moscow, Russia;
[2] Sechenov First Moscow State Medical University, Moscow, Russia;
[3] S.S. Yudin City Clinical Hospital, Moscow, Russia;
[4] National Medical Research Center of Hematology, Moscow, Russia;
[5] Lomonosov Moscow State University, Moscow, Russia

摘要

论证。心脏淀粉样变性是一种严重的进展性疾病，死亡率很高。肺链淀粉样变性（AL-淀粉样变性）和转甲状腺素淀粉样变性（ATTR-淀粉样变性）引起的心肌病的鉴别诊断，其最佳治疗策略的选择非常重要。

目的。评估心脏磁共振成像鉴别诊断 AL-和ATTR-淀粉样变性引起的心肌病的能力。

材料和方法。对25例确诊为淀粉样变性心肌病的患者的医学数据进行回顾性分析，根据淀粉样变性的类型分为两组。第1组为AL-淀粉样变性引起的心肌病，第2组 为ATTR-淀粉样变性引起的心肌病。所有患者均进行了心脏MRIL造影剂检查。评估了心脏容量和线性指标、心室功能和晚期钆沉积模式。使用标准统计方法，p<0.05时为差异显著。

结果。与第1组患者相比，第2组患者的心肌壁增厚更明显（室间隔18[17;18] vs.14.5mm[12.8;16]，p<0.01，左心室后壁14[13;17]vs.10.5mm[10;12.3]，p<0.01）。第2组的左心室心肌质量指数为110[92;125]，而第1组该指标为85 g/m^2[69.3;91.8]，p<0.01）。在第2组患者中，基底和中下外侧段的晚期钆沉积模式更常见于左心室透壁模式，而在第1组患者中，中前部和下外侧段的晚期钆沉积模式更常见于心内膜下模式（p<0.05）。并且在第2组患者中，造影剂同时在左心室和右心室两侧室间隔心内膜下层聚集的频率较高（100 对 50%，p<0.01）。晚期钆沉积模式在第2组患者中的右心室也更为常见（100 vs.58%，p<0.05），尤其是在室间隔和下壁区域（p<0.05）。使用 QALE（淀粉样蛋白晚期增强）指标对晚期钆沉积模式进行半定量评估显示，第2组患者的对比剂聚集更广泛13[12;14] vs.10.5[1.75;12] 分，p<0.01），评分大于13分可以区分AL-和ATTR-淀粉样变性引起的心肌病，敏感性为69%，特异性为83%。

结论。心脏MRI可以识别AL-和ATTR-淀粉样变性引起的心肌病的特征，这可能有助于它们的鉴别诊断。还需要继续研究来确认所查明模式的诊断准确性。

关键词：心脏淀粉样变性；系统性淀粉样变性；心脏磁共振成像；延迟钆造影剂；QALE；转甲状腺素淀粉样变性；肺链淀粉样变性。

引用本文：
Magomedova ZM, Nikiforova TV, Shchekochikhin DYu, Pershina ES, Kovalev KV, Abdulmazhidova KhS, Rassechkina DS, Grachev AE, Rekhtina IG, Volovchenko AN, Sarkisyan SD, Sinitsyn VE, Andreev DA. 心脏磁共振成像在肺链式淀粉样变性和转甲状腺素淀粉样变性引起的心肌病鉴别诊断中的可能性. *Digital Diagnostics.* 2024;5(4):668–681. DOI: https://doi.org/10.17816/DD635007

收到：08.09.2024 **接受：**24.10.2024 **发布日期：**12.11.2024

ECO•VECTOR
Article can be used under the CC BY-NC-ND 4.0 International License
© Eco-Vector, 2024

BACKGROUND

Cardiac amyloidosis is a serious, progressive disease that causes heart failure and death. It is defined by the extracellular deposition of a specific protein–polysaccharide complex (amyloid) in the myocardium. The most common types of cardiac amyloidosis are amyloid light-chain (AL) amyloidosis and transthyretin (ATTR) amyloidosis, which are caused by immunoglobulin light chain amyloid and transthyretin deposition, respectively. The differential diagnosis of cardiomyopathies due to AL and ATTR amyloidosis is crucial for selecting the optimal treatment strategy [1–3].

Amyloidosis is a rare condition. However, recent findings indicate that amyloid cardiomyopathy is underestimated as a cause of common cardiac disorders. Due to advancements in cardiac imaging and improved diagnosis and treatment strategies, the options for diagnosing and managing cardiac amyloidosis have expanded [4, 5]. Algorithms proposed by the American College of Cardiology [6, 7] and the European Society of Cardiology [8] are currently used for diagnosing this condition.

Cardiac amyloidosis is diagnosed by assessing clonal dyscrasia using an immunochemical analysis of serum and 24-h urine samples. The analysis involved serum and urine protein electrophoresis with immunofixation, as well as a serum-free light chain assay to rule out AL amyloidosis. If the test is positive, a right ventricular (RV) endomyocardial biopsy can be performed to confirm the diagnosis and distinguish between AL and ATTR amyloidosis. In the absence of clonal dyscrasia, ATTR amyloidosis is confirmed using scintigraphy with technetium radiopharmaceuticals (^{99m}Tc-PYP, ^{99m}Tc pyrophosphate; ^{99m}Tc-DPD, ^{99m}Tc-3,3-diphosphono-1,2-propanodicarboxylic acid; ^{99m}Tc-HMDP, ^{99m}Tc-hydroxyl-methylenediphosphonate), with an uptake rate of 2–3; a biopsy is not required. *TTR* genotyping is also possible, particularly for detecting hereditary disease forms, even without family history or signs of polyneuropathy. However, endomyocardial biopsy is the gold standard in cases with inconclusive test results. This method has high specificity and sensitivity for detecting amyloid deposits by Congo red staining [6, 8, 9].

Time-delayed contrast-enhanced magnetic resonance imaging (MRI) is highly effective in diagnosing cardiac amyloidosis and detects contrast uptake patterns in the myocardium characteristic of amyloid deposits and allows the assessment of cardiac functional disorders [10–12]. Moreover, cardiac MRI allows for the differential diagnosis between AL and ATTR amyloidosis-induced cardiomyopathy, considering the pattern of delayed contrast uptake, signs of severe concentric ventricular hypertrophy, and increased myocardial mass [13].

AIM

To assess the potential of cardiac MRI in the differential diagnosis of cardiomyopathy due to AL or ATTR amyloidosis.

METHODS

Study design

We conducted a cross-sectional, observational, single-arm, single-center study to review the medical records of patients with confirmed cardiomyopathy due to AL or ATTR amyloidosis.

Eligibility criteria

We used contrast-enhanced cardiac MRI findings obtained between January 1, 2021, and May 31, 2024.

Inclusion criteria:

- confirmed cardiomyopathy due to AL or ATTR amyloidosis in accordance with the American College of Cardiology guidelines [14, 15], European Society of Cardiology guidelines [8], and Russian guidelines for the diagnosis and treatment of systemic amyloidosis [9];
- available contrast-enhanced cardiac MRI findings; and
- signed informed consent (the study only included data from patients who signed an informed consent form for the use of their data for research purposes, approved by the City Clinical Hospital No. 1 named after N.I. Pirogov).

Study setting

A contrast-enhanced cardiac MRI was performed in the MRI and CT department of the City Clinical Hospital No. 1 named after N.I. Pirogov. We included the medical records of outpatients and inpatients. Amyloid cardiomyopathy could be the principal diagnosis or a complication of another condition.

Study duration

We reviewed the medical records between June 1, 2024, and July 31, 2024.

Intervention

The analysis of medical records included clinical examination, blood test, electrocardiography (ECG), echocardiography, and cardiac MRI findings.

A cardiac MRI was performed using the *Vantage ExelArt TOSHIBA 1.5T* and *Philips Ingenia 1.5-T Evolution* scanners, according to the optimized protocols for diagnosing cardiac amyloidosis. We used a specific sequence of scanning protocols to assess the heart morphology, ventricular function, and signs of amyloid deposits:

1. A series of scans (localizers) in three planes for examination planning.

2. A cine-MRI in the steady-state free precession mode in two-, three-, and four-chamber views and a series of short-axis scans from the base to the apex of the left ventricle (LV).

3. Fat-suppressed T2-weighted imaging.

4. Black blood T2-weighted imaging.

5. TI-scout (look-locker) imaging 8–10 min after contrast injection to determine the optimal myocardial inversion time (TI) or the phase-sensitive inversion recovery sequence.

6. Post-contrast T1-weighted imaging to assess delayed contrast uptake in the myocardium (late gadolinium enhancement, LGE) 10–15 min after contrast injection.

All examinations were ECG-gated, with breath holding, where necessary. The slice thickness and interslice gap were 6–8 and 2 mm, respectively. The total examination time was ~45–60 min.

Main study outcome

The main study outcome was changes in cardiac MRI in patients with cardiomyopathy due to amyloidosis. We assessed the following parameters: LV volumetric and linear measures; LV and RV LGE patterns.

Additional study outcomes

The additional study outcome was a semiquantitative LGE assessment using the Query Amyloid Late Enhancement (QALE) score.

Subgroup analysis

The study included two groups based on the amyloidosis type:

- Group 1: patients with cardiomyopathy due to AL amyloidosis;
- Group 2: patients with cardiomyopathy due to ATTR amyloidosis.

Outcomes registration

Images were processed and analyzed using the specialist *cvi42 software* (Circle Cardiovascular Imaging Inc., Canada). Two qualified radiologists experienced in cardiac imaging independently assessed the cardiac MRI findings. The interobserver variability was additionally assessed. Changes in LV volumetric and linear measures were recorded. Contrast uptake patterns in the myocardium depending on the type of amyloidosis were identified. We used the QALE score developed by Dungu et al. [13] for a semi-quantitative LGE assessment. The analysis involves three LV levels: basal, middle, and apical. The maximum score for each level is four points, depending on the contrast uptake pattern. If the RV is involved, the maximum score for each level increases to six points. Thus, the total QALE score ranged from 0 (no LGE) to 18 (global transmural LV LGE plus RV involvement).

Ethical review

The Local Ethics Committee of I.M. Sechenov First Moscow State Medical University approved this study (Minutes No. 15–24 of June 6, 2024).

Statistical analysis

Sample size calculation: The sample size was not calculated in advance due to the rarity (orphan) of the disease. Considering the limited number of patients with this condition, all eligible patients were included.

Statistical analysis methods: The qualitative parameters were compared using the χ^2 or Fisher's exact test. The quantitative parameters were compared using the nonparametric Mann–Whitney test. The results were presented as Me [Q25; Q75], where Me is the median and Q25 and Q75 are the 25th and 75th percentiles, respectively. Differences were considered significant at $p < 0.05$.

RESULTS

Participants

The study included 25 patients with confirmed cardiac amyloidosis, with 12 and 13 patients in Groups 1 and 2, respectively. The mean age was 71.7 ± 12 years; 46% of the patients were male.

Table 1 shows the patient characteristics, including demographics and clinical data.

Primary results

MRI: cardiac volumetric and linear measures

Group 2 had more pronounced myocardial wall thickening than Group 1 (interventricular septum, LV involvement, 18 mm [17; 18] vs. 14.5 mm [12.8; 16], $p < 0.01$; LV posterior wall 14 mm [13; 17] vs. 10.5 mm [10.0; 12.3], $p < 0.01$) (Fig. 1). No significant differences were found in LV ejection fraction parameters: 53% [42; 66] vs. 56.5% [51.5; 66.3], $p > 0.05$). However, the LV mass index was higher in Group 2 (110 mm/m^2 [92; 125] vs. 85.0 mm/m^2 [69.3; 91.8], $p < 0.01$). Pleural effusion was detected in 67% and 46% of patients in Groups 1 and 2, respectively, but the difference was not significant ($p = 0.530$).

MRI: delayed contrast uptake in the myocardium

LGE was detected in all patients in Group 2 and 11 (85%) patients in Group 1 (Table 2). RV LGE was detected in all patients in Group 2 and 58% of patients in Group 1 ($p < 0.05$). Group 2 had a significantly higher rate of contrast uptake in the interventricular septum and RV inferior wall (62% vs. 8%, $p < 0.05$) (Fig. 2). A simultaneous subendocardial LGE in the LV and RV in the interventricular septum was found Group 2, resulting in the double-line sign (100% vs. 50%, $p < 0.01$) (Fig. 3). Atrial LGE was observed in 69% and 50% of patients in Groups 2 and 1, respectively ($p > 0.05$).

The analysis of contrast uptake distribution by cardiac segments revealed transmural LGE in Group 2 at the basal and middle levels (inferolateral segments, Fig. 4) ($p < 0.05$). Group 1 showed subendocardial LGE at the middle level (antero- and inferolateral segments, Fig. 5) ($p < 0.05$). The other segments showed no specific contrast uptake patterns ($p > 0.05$) (Table 3). The circular contrast uptake rates were not significantly different.

Table 1. Comparison of patient characteristics

Characteristics	Group 1, *n* = 12	Group 2, *n* = 13	*p*-value
Demographics			
Age, years	64.5 [59.3; 71.8]	79 [74; 84]	<0.01
Males, *n* (%)	5 (42)	11 (85)	0.07
Clinical findings			
Chronic heart failure, NYHA class II, *n* (%)	7 (58)	6 (46)	0.83
Chronic heart failure, NYHA class III, *n* (%)	5 (42)	7 (54)	0.83
Hypertension, *n* (%)	4 (33)	4 (31)	1.0
Coronary artery disease, *n* (%)	2 (17)	5 (38)	0.44
Polyneuropathy, *n* (%)	2 (15)	3 (23)	1.0
Spinal stenosis, *n* (%)	0 (0)	1 (8)	1.0
Carpal tunnel syndrome, *n* (%)	0 (0)	1 (8)	1.0
Anamnestic findings			
Pacemaker implantation, *n* (%)	2 (15)	1 (8)	0.94
History of revascularization, *n* (%)	1 (8)	3 (23)	0.65
History of myocardial infarction, *n* (%)	2 (17)	3 (23)	1.00
Electrocardiography			
Low QRS voltage on ECG, *n* (%)	5 (42)	5 (38)	1.00
Pseudo-infarction changes, *n* (%)	6 (50)	2 (15)	0.15
Complete right bundle branch block, *n* (%)	1 (8)	2 (15)	1.00
Grade 1 atrioventricular block, *n* (%)	1 (8)	4 (31)	0.37
Atrial fibrillation, *n* (%)	6 (50)	7 (54)	1.00
Laboratory findings			
NT-proBNP > 300 pg/mL, *n* (%)	12 (100)	13 (100)	0.07
Troponin I > 0.023 ng/mL, *n* (%)	5 (42)	7 (54)	0.83
Proteinuria > 1.0 g/day, *n* (%)	11 (92)	1 (8)	<0.01
Echocardiography			
Left ventricular ejection fraction, %	57 [48; 63]	54 [54; 58]	0.51
Interventricular septum, mm	15 [14; 17]	17 [16; 19]	0.01
Left ventricular posterior wall, mm	13 [12; 15]	16 [14; 17]	0.09

Note. Low voltage on ECG was defined as all QRS amplitudes < 5 mm (standard leads) or < 10 mm (precordial leads). NYHA, New York Heart Association classification; NT-proBNP, N-terminal prohormone of brain natriuretic peptide.

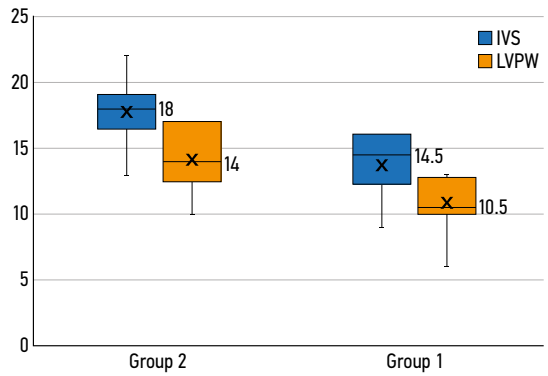

Fig. 1. Box plot of the linear measures of cardiac magnetic resonance imaging in Groups 1 and 2. AL, light-chain amyloidosis (Group 1); ATTR, transthyretin amyloidosis (Group 2); IVS, interventricular septum; LVPW, left ventricular posterior wall.

Secondary results

Semi-quantitative LGE assessment of cardiac amyloidosis

We performed a semiquantitative LGE assessment using the QALE score during the analysis of postcontrast T1-weighted images of the ventricles. Group 2 had larger LGE areas than Group 1: 13 points [12; 14] vs. 10.5 points [1.75; 12], $p < 0.01$ (Fig. 6). We performed a receiver operating characteristic (ROC) analysis to determine a predictive model for the QALE score in patients with cardiomyopathy, depending on the amyloidosis type. The analysis confirmed the QALE score's usefulness in determining the amyloidosis type: the area under the ROC curve (AUC) was 0.83 (sensitivity 69%, specificity 83%, QALE threshold ⩾ 13 points).

Table 2. Comparison of late gadolinium enhancement cases depending on the segment on MRI.

	Group 1, *n* = 12				Group 2, *n* = 13			
	0	**1**	**2**	**3**	**0**	**1**	**2**	**3**
Basal level								
Segment 1 (anterior), *n*	4	2	6	0	3	0	8	2
Segment 2 (anteroseptal), *n*	7	1	3	1	4	3	5	1
Segment 3 (inferoseptal), *n*	6	2	4	0	4	3	5	1
Segment 4 (inferior), *n*	2	2	5	3	1	2	7	3
Segment 5 (inferolateral), *n*	2	1	5	4*	0	1	1	11*
Segment 6 (anterolateral), *n*	3	2	5	2	1	2	5	5
Middle level								
Segment 7 (anterior), *n*	5	1	6	0	4	3	5	1
Segment 8 (anteroseptal), *n*	5	3	4	0	6	1	5	1
Segment 9 (inferoseptal), *n*	6	1	5	0	5	2	5	1
Segment 10 (inferior), *n*	3	1	7	1	6	1	4	2
Segment 11 (inferolateral), *n**	3	0	9*	0*	3	1	1*	8*
Segment 12 (anterolateral), *n**	4	0	8*	0	5	4	2*	2
Apical level								
Segment 13 (anterior), *n*	6	0	6	0	7	0	5	1
Segment 14 (septal), *n*	6	0	6	0	5	1	5	2
Segment 15 (inferior), *n*	6	0	6	0	9	1	3	0
Segment 16 (lateral), *n*	6	0	6	0	8	1	4	0
Total*, *n*	9	5	10	4*	12	9	12	12*
Combination of the segments								
Basal level of the interventricular septum (segments 2 and 3), *n*	6	1	3	0	4	3	5	1
Middle level of the interventricular septum (segments 8 and 9), *n*	5	1	4	0	5	1	5	1
Interventricular septum (segments 2, 3, 8, 9, and 14), *n*	4	0	2	0	2	1	2	0
Basal level, lateral wall (segments 5 and 6), *n*	2	1	3	2	0	1	1	5
Middle level, lateral wall (segments 11 and 12), *n*	3	0	8*	1	3	1	1*	1
Lateral wall (segments 5, 6, 11, 12, and 16), *n*	2	0	3	0	1	0	0	0
Circular distribution with a subendocardial pattern								
Basal level, *n*			2				0	
Middle level, *n*			3				1	
Apical level, *n*			6				2	
Circular distribution with any contrast uptake pattern								
Basal level, *n*			4				7	
Middle level, *n*			6				5	
Apical level, *n*			5				4	

Note. 0, no late gadolinium enhancement areas; 1, intramyocardial late gadolinium enhancement pattern; 2, subendocardial late gadolinium enhancement pattern; 3, transmural late gadolinium enhancement pattern; *, significant intergroup difference (cases of late gadolinium enhancement with the same pattern in the groups).

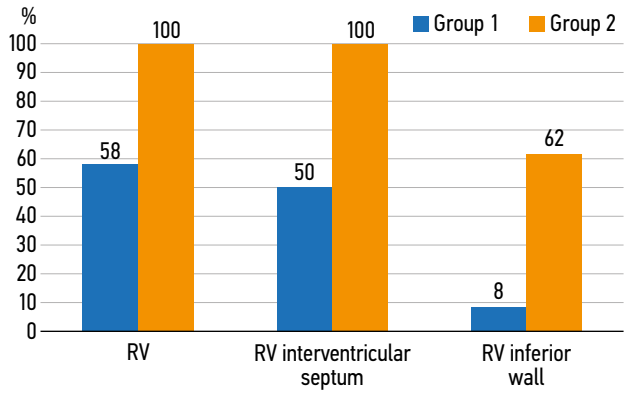

Fig. 2. Distribution of late gadolinium enhancement cases in the right ventricle in the groups. RV, right ventricle; AL, light-chain amyloidosis (Group 1); ATTR, transthyretin amyloidosis (Group 2); LGE, late gadolinium enhancement.

DISCUSSION

Summary of primary results

This retrospective study revealed that cardiac MRI plays a significant role in the differential diagnosis of AL amyloidosis- and ATTR amyloidosis-induced cardiomyopathy. This conclusion is clinically significant because these two conditions require fundamentally different therapeutic approaches [16].

Discussion of primary results

Distinctive signs of amyloid cardiomyopathy include more pronounced myocardial wall thickening (interventricular septum, LV involvement, and LV posterior wall) and an increased LV mass index in ATTR amyloidosis compared with AL amyloidosis, which was consistent with previous findings, indicating that ATTR amyloidosis is associated with more severe myocardial hypertrophy. Based on Dungu et al. [13], AL amyloidosis was characterized by a minimal increase in the LV mass index compared with ATTR amyloidosis. They reported a significant LV wall thickening in ATTR amyloidosis compared with AL amyloidosis: 18 ± 2 vs. 14 ± 3 mm. Kriste et al. reported similar findings: the myocardial mass in ATTR amyloidosis compared with AL amyloidosis was 164 ± 57 vs. 159 ± 61 mg. The maximum LV wall thickness in ATTR amyloidosis was significantly higher than that in AL amyloidosis. These changes are due to an increased amyloid load in ATTR amyloidosis [14, 15].

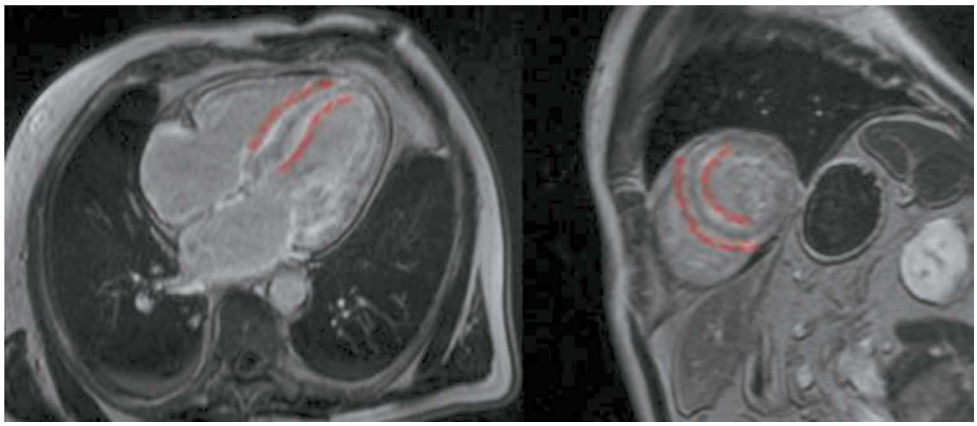

Fig. 3. Time-delayed contrast-enhanced cardiac magnetic resonance imaging scans in transthyretin amyloidosis. Subendocardial contrast uptake in the interventricular septum (right and left ventricular involvement) (red dashed lines).

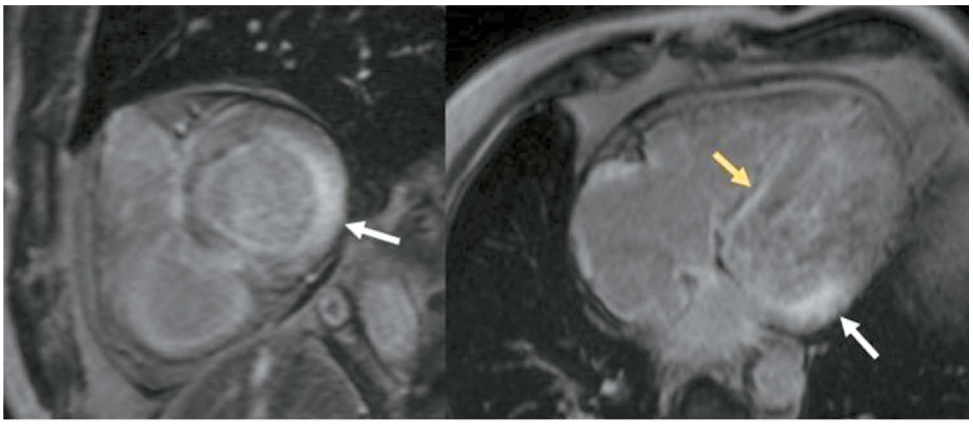

Fig. 4. Time-delayed contrast-enhanced cardiac magnetic resonance imaging scans in transthyretin amyloidosis. Transmural contrast uptake at the basal and middle levels (inferolateral segments), subendocardial contrast uptake at the basal level (anterior, anterolateral, and inferior segments) of the left ventricular myocardium (white arrows), and subendocardial contrast uptake in the interventricular septum (right ventricular involvement) (yellow arrow).

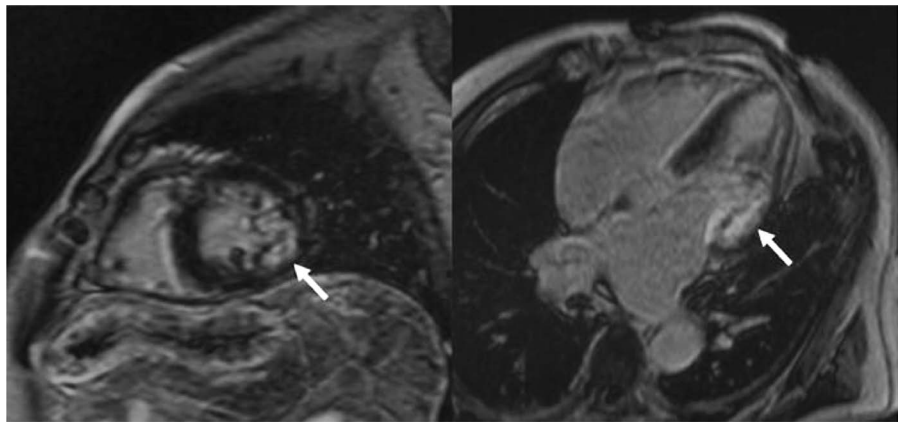

Fig. 5. Time-delayed contrast-enhanced cardiac magnetic resonance imaging scans in light-chain amyloidosis. Subendocardial contrast uptake at the basal and middle levels (inferolateral segments) of the left ventricular myocardium (white arrows).

Table 3. Comparative analysis of late gadolinium enhancement cases in various cardiac structures in the groups.

Characteristics	Group 1, *n* = 12	Group 2, *n* = 13	*p*-value
Late gadolinium enhancement in the right ventricle, *n* (%)	7 (58)	13 (100)	<0.05
Late gadolinium enhancement in the right ventricular inferior wall, *n* (%)	1 (8)	8 (62)	<0.05
Late gadolinium enhancement in the interventricular septum (right ventricular involvement), *n* (%)	6 (50)	13 (100)	<0.05
Late gadolinium enhancement in the right ventricular wall, *n* (%)	6 (50)	9 (69)	>0.05
Late gadolinium enhancement in the atria, *n* (%)	6 (50)	9 (69)	>0.05
Late gadolinium enhancement in the interventricular septum (right and left ventricular involvement), *n* (%)	6 (50)	13 (100)	<0.05

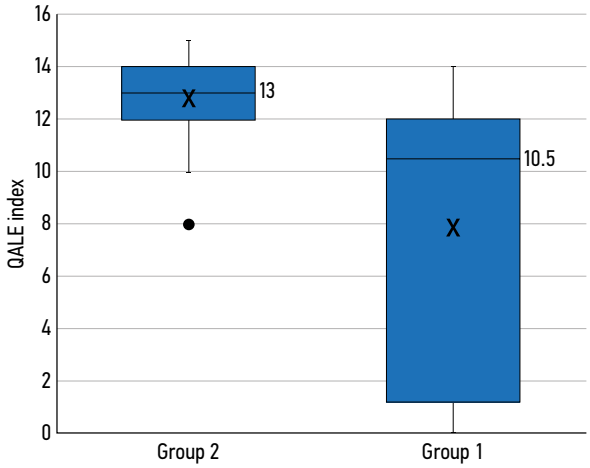

Fig. 6. Box plot of the Query Amyloid Late Enhancement (QALE) score in Groups 1 and 2. AL, light-chain amyloidosis (Group 1); ATTR, transthyretin amyloidosis (Group 2).

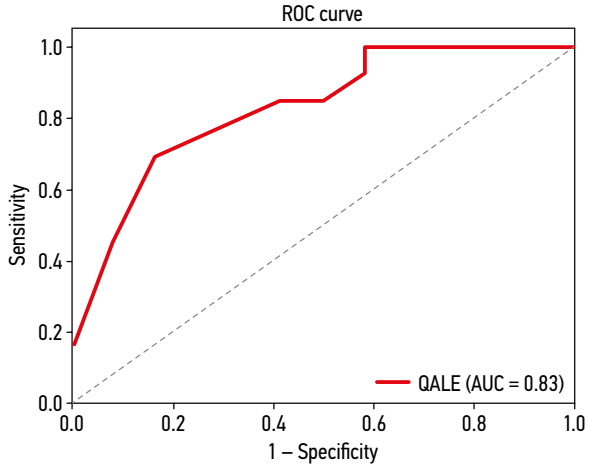

Fig. 7. ROC curve for the Query Amyloid Late Enhancement (QALE) score. Solid line: QALE score, area under the curve (AUC) 0.83 (95% confidence interval: 0.64–0.97); sensitivity 69%; specificity 83%.

LGE patterns in these types of cardiac amyloidosis differ, which can be useful in the differential diagnosis. AL amyloidosis is more commonly associated with a global subendocardial contrast uptake, whereas ATTR amyloidosis is characterized by a transmural or focal contrast uptake [13, 17]. Despite these differences, the differential diagnosis between AL amyloidosis- and ATTR amyloidosis-induced cardiomyopathy based on cardiac MRI findings can be challenging due to similar visual patterns. Semiquantitative LGE assessment and additional imaging techniques (scintigraphy with ^{99m}Tc-DPD) have been proposed to improve the diagnostic accuracy [18].

The analysis of LGE characteristics revealed specific patterns for each amyloidosis type, which can be used for the differential diagnosis. The most valuable findings are as follows:

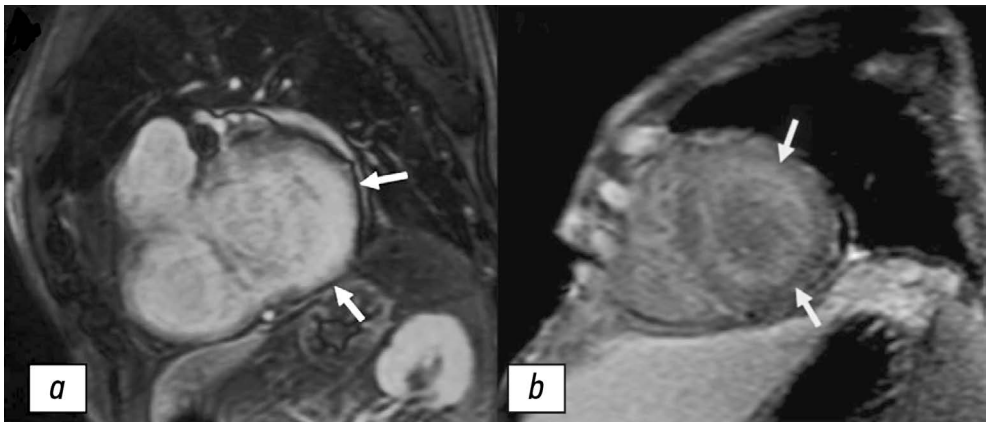

Fig. 8. Time-delayed contrast-enhanced cardiac magnetic resonance imaging scans in transthyretin amyloidosis. a, transmural contrast uptake at the basal level (lateral segments) and intramural contrast uptake at the basal level (inferior segment) of the left ventricular myocardium (white arrows), QALE score: 15 points. b, circular subendocardial contrast uptake at the middle level (all segments) of the left ventricular myocardium (yellow arrows), QALE score: 10 points.

- *ATTR amyloidosis:* more commonly associated with a pronounced transmural LGE at the basal and middle levels, in the inferolateral segments of the LV, with RV involvement, particularly in the interventricular septum (LV involvement) and the RV inferior wall (Fig. 8, *a*).
- *AL amyloidosis:* more commonly associated with a subendocardial LGE, mostly at the middle levels, in the antero- and inferolateral segments (Fig. 8, *b*).

These findings are consistent with those of previous studies [13, 17, 19], which also found specific LGE patterns in AL and ATTR amyloidosis, including a more pronounced contrast uptake in ATTR amyloidosis ($p < 0.001$) [17].

Based on the available data, ATTR amyloidosis can be distinguished from AL amyloidosis with a sensitivity and specificity of 82% and 76%, respectively, based on the QALE score of ⩾ 13 points [13]. We found that ATTR amyloidosis was similarly characterized by a more pronounced LGE compared with AL amyloidosis.

The significant amyloid buildup in the myocardium in ATTR amyloidosis is most likely due to a longer disease duration than in AL amyloidosis. In AL amyloidosis, myocardial damage is caused by both amyloid buildup and the direct toxic effect of the immunoglobulin light chains, resulting in lower amyloid levels in the myocardium.

Our study revealed that patients with AL amyloidosis have a higher risk of *pleural effusion* than patients with ATTR amyloidosis. This is a significant finding because it can be associated with more severe systemic involvement in AL amyloidosis, as confirmed by Binder et al. [20].

The double-line sign in the interventricular septum in ATTR amyloidosis is an interesting phenomenon, indicating simultaneous subendocardial LGE in the LV and RV (See Fig. 3). This sign was observed in all patients with ATTR amyloidosis, possibly making it an important diagnostic marker. However, available publications do not describe this contrast uptake pattern, necessitating further studies.

Notably, no significant intergroup differences were observed in the LV ejection fraction and volumetric measures. In this context, myocardial deformation assessment will likely be more useful for assessing the functional aspects of the heart in amyloidosis [21].

Study limitations

This study has the following limitations:

- the sample size was relatively small.
- the study did not assess the effect of concomitant cardiovascular diseases and interventions.

However, we found no significant differences when comparing the clinical and anamnestic signs in the groups (See Table 1). Notably, a significant age difference of 11 years was observed between the groups ($p < 0.01$), which likely affected the result interpretation. However, despite the observed differences, many characteristics of cardiac MRI findings in amyloid cardiomyopathy are nonspecific and can be seen in both amyloidosis types, highlighting the importance of a comprehensive diagnosis, including clinical, laboratory, and imaging examinations.

CONCLUSION

The study findings show that contrast-enhanced cardiac MRI is a highly effective tool for the differential diagnosis of AL amyloidosis- and ATTR amyloidosis-induced cardiomyopathy. Using typical contrast uptake patterns and additional imaging techniques can significantly improve the diagnostic accuracy. Further studies and new diagnostic criteria and tools are required to improve the diagnosis and treatment of this complex condition.

ADDITIONAL INFORMATION

Funding source. This study was not supported by any external sources of funding.

Competing interest. The authors declare that they have no competing interest.

Authors' contribution. All authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation

of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work. Z.M. Magomedova — collection and analysis of patient's data, literature review, statistical analysis, preparation, writing and editing of the article; T.V. Nikiforova, Kh.S. Abdulmazhidova, S.D. Sarkisyan — collection and analysis of patient's data; D.Yu. Shchekochikhin, V.E. Sinitsyn, D.A. Andreev — editing the text of the article; E.S. Pershina — literature review, collection and analysis of patient's data; K.V. Kovalev — collection and analysis of patient's data; A.E. Grachev, I.G. Rekhtina, A.N. Volovchenko — literature review, collection and analysis of patient's data.

REFERENCES

1. Wechalekar AD, Gillmore JD, Hawkins PN. Systemic amyloidosis. *Lancet.* 2016;387(10038):2641–2654. doi: 10.1016/S0140-6736(15)01274-X
2. Rapezzi C, Lorenzini M, Longhi S, et al. Cardiac amyloidosis: the great pretender. *Heart Fail Rev.* 2015;20(2):117–124. doi: 10.1007/s10741-015-9480-0
3. Myasnikov RP, Andreyenko EYu, Kushunina DV, et al. Cardiac amyloidosis: modern aspects of diagnosis and treatment (clinical observation). *Clinical and experimental surgery.* 2014;(4):72–82. EDN: TRLZYN
4. Maurer MS, Elliott P, Comenzo R, et al. Addressing common questions encountered in the diagnosis and management of cardiac amyloidosis. *Circulation.* 2017;135(14):1357–1377. doi: 10.1161/CIRCULATIONAHA.116.024438
5. Ruberg FL, Grogan M, Hanna M, et al. Transthyretin amyloid cardiomyopathy: JACC state-of-the-art review. *J Am Coll Cardiol.* 2019;73(22):2872–2891. doi: 10.1016/j.jacc.2019.04.003
6. Kittleson MM, Maurer MS, Ambardekar AV, et al.; American Heart Association Heart Failure and Transplantation Committee of the Council on Clinical Cardiology. Cardiac amyloidosis: evolving diagnosis and management: a scientific statement from the American Heart Association. *Circulation.* 2020;142(1):e7–e22. doi: 10.1161/CIR.0000000000000792
7. Maurer MS, Bokhari S, Damy T, et al. Expert consensus recommendations for the suspicion and diagnosis of transthyretin cardiac amyloidosis. *Circ Heart Fail.* 2019;12(9):e006075. doi: 10.1161/CIRCHEARTFAILURE.119.006075
8. Garcia-Pavia P, Rapezzi C, Adler Y, et al. Diagnosis and treatment of cardiac amyloidosis: a position statement of the ESC Working Group on Myocardial and Pericardial Diseases. *Eur Heart J.* 2021;42(16):1554–1568. doi: 10.1093/eurheartj/ehab072
9. Lysenko LV, Rameev VV, Moiseev SV, et al. Clinical guidelines for diagnosis and treatment of systemic amyloidosis. *Clinical pharmacology and therapy.* 2020;29(1):13–24. EDN UCEZAB doi: 10.32756/ 0869-5490-2020-1-13-24
10. Syed IS, Glockner JF, Feng D, et al. Role of cardiac magnetic resonance imaging in the detection of cardiac amyloidosis. *JACC Cardiovasc Imaging.* 2010;3(2):155–164. doi: 10.1016/j.jcmg.2009.09.023
11. Butorova EA, Stukalova OV. Role of cardiac MRI in the diagnosis of cardiac amyloidosis. Clinical cases. *Clinical review for general practice.* 2021;(2):16–20. EDN MUWTXO doi: 10.47407/kr2021.2.2.00037
12. Dorbala S, Ando Y, Bokhari S, et al. ASNC/AHA/ASE/EANM/HFSA/ISA/SCMR/SNMMI expert consensus recommendations for multimodality imaging in cardiac amyloidosis: Part 1 of 2-evidence base and standardized methods of imaging. *J Nucl Cardiol.* 2019;26(6):2065–2123. doi: 10.1007/s12350-019-01760-6
13. Dungu JN, Valencia O, Pinney JH, et al. CMR-based differentiation of AL and ATTR cardiac amyloidosis. *JACC Cardiovasc Imaging.* 2014;7(2):133–142. doi: 10.1016/j.jcmg.2013.08.015
14. Itzhaki Ben Zadok O, Vaturi M, Vaxman I, et al. Differences in the characteristics and contemporary cardiac outcomes of patients with light-chain versus transthyretin cardiac amyloidosis. *PLoS One.* 2021;16(8):e0255487. doi: 10.1371/journal.pone.0255487
15. Quarta CC, Solomon SD, Uraizee I, et al. Left ventricular structure and function in transthyretin-related versus light-chain cardiac amyloidosis. *Circulation.* 2014;129(18):1840–1849. doi: 10.1161/CIRCULATIONAHA.113.006242
16. Stern LK, Patel J. Cardiac Amyloidosis Treatment. *Methodist Debakey Cardiovasc J.* 2022;18(2):59–72. doi: 10.14797/mdcvj.1050
17. Kristen AV., aus dem Siepen F, Scherer K, et al. Comparison of different types of cardiac amyloidosis by cardiac magnetic resonance imaging. *Amyloid.* 2015;22(2):132–141. doi: 10.3109/13506129.2015.1020153
18. Gillmore JD, Maurer MS, Falk RH, et al. Nonbiopsy diagnosis of cardiac transthyretin amyloidosis. *Circulation.* 2016;133(24):2404–2412. doi: 10.1161/CIRCULATIONAHA.116.021612
19. Martinez-Naharro A, Treibel TA, Abdel-Gadir A, et al. Magnetic resonance in transthyretin cardiac amyloidosis. *J Am Coll Cardiol.* 2017;70(4):466–477. doi: 10.1016/j.jacc.2017.05.053
20. Binder C, Duca F, Binder T, et al. Prognostic implications of pericardial and pleural effusion in patients with cardiac amyloidosis. *Clinical Research in Cardiology.* 2021;110(4):532–543. doi: 10.1007/s00392-020-01698-7
21. Reddy A, Singh V, Karthikeyan B, et al. Biventricular strain imaging with cardiac MRI in genotyped and histology validated amyloid cardiomyopathy. *Cardiogenetics.* 2021;11(3):98–110. doi: 10.3390/cardiogenetics11030011

СПИСОК ЛИТЕРАТУРЫ

1. Wechalekar A.D., Gillmore J.D., Hawkins P.N. Systemic amyloidosis // Lancet. 2016. Vol. 387, N 10038. P. 2641–2654. doi: 10.1016/S0140-6736(15)01274-X
2. Rapezzi C., Lorenzini M., Longhi S., et al. Cardiac amyloidosis: the great pretender // Heart Fail Rev. 2015. Vol. 20, N 2. P. 117–124. doi: 10.1007/s10741-015-9480-0
3. Мясников Р.П., Андреенко Е.Ю., Кушунина Д.В., и др. Амилоидоз сердца: современные аспекты диагностики и лечения (клиническое наблюдение) // Клиническая и экспериментальная хирургия. 2014. № 4. С. 72–82. EDN: TRLZYN
4. Maurer M.S., Elliott P., Comenzo R., et al. Addressing common questions encountered in the diagnosis and management of cardiac amyloidosis // Circulation. 2017. Vol. 135, N 14. P. 1357–1377. doi: 10.1161/CIRCULATIONAHA.116.024438
5. Ruberg F.L., Grogan M., Hanna M., et al. Transthyretin amyloid cardiomyopathy: JACC state-of-the-art review // J Am Coll Cardiol. 2019. Vol. 73, N 22. P. 2872–2891. doi: 10.1016/j.jacc.2019.04.003

6. Kittleson M.M., Maurer M.S., Ambardekar A.V., et al.; American Heart Association Heart Failure and Transplantation Committee of the Council on Clinical Cardiology. Cardiac amyloidosis: evolving diagnosis and management: a scientific statement from the American Heart Association // Circulation. 2020. Vol. 142, N 1. P. e7–e22. doi: 10.1161/CIR.0000000000000792

7. Maurer M.S., Bokhari S., Damy T., et al. Expert consensus recommendations for the suspicion and diagnosis of transthyretin cardiac amyloidosis // Circ Heart Fail. 2019. Vol. 12, N 9. P. e006075. doi: 10.1161/CIRCHEARTFAILURE.119.006075

8. Garcia-Pavia P., Rapezzi C., Adler Y., et al. Diagnosis and treatment of cardiac amyloidosis: a position statement of the ESC Working Group on Myocardial and Pericardial Diseases // Eur Heart J. 2021. Vol. 42, N 16, P. 1554–1568. doi: 10.1093/eurheartj/ehab072

9. Лысенко Л.В., Рамеев В.В., Моисеев С.В., и др. Клинические рекомендации по диагностике и лечению системного амилоидоза // Клиническая фармакология и терапия. 2020. Т. 29, № 1. С. 13–24. EDN UCEZAB doi: 10.32756/ 0869-5490-2020-1-13-24

10. Syed I.S., Glockner J.F., Feng D., et al. Role of cardiac magnetic resonance imaging in the detection of cardiac amyloidosis // JACC Cardiovasc Imaging. 2010. Vol. 3, N 2, P. 155–164. doi: 10.1016/j.jcmg.2009.09.023

11. Буторова Е.А., Стукалова О.В. Возможности магнитно-резонансной томографии сердца в диагностике амилоидоза сердца. Клинические примеры // Клинический разбор в общей медицине. 2021. № 2. С. 16–20. EDN MUWTXO doi: 10.47407/kr2021.2.2.00037

12. Dorbala S., Ando Y., Bokhari S., et al. ASNC/AHA/ASE/EANM/HFSA/ISA/SCMR/SNMMI expert consensus recommendations for multimodality imaging in cardiac amyloidosis: Part 1 of 2-evidence base and standardized methods of imaging // J Nucl Cardiol. 2019. Vol 26, N 6. P. 2065–2123. doi: 10.1007/s12350-019-01760-6

13. Dungu J.N., Valencia O., Pinney J.H., et al. CMR-based differentiation of AL and ATTR cardiac amyloidosis // JACC Cardiovasc Imaging. 2014. Vol. 7, N 2. P. 133–142. doi: 10.1016/j.jcmg.2013.08.015

14. Itzhaki Ben Zadok O., Vaturi M., Vaxman I., et al. Differences in the characteristics and contemporary cardiac outcomes of patients with light-chain versus transthyretin cardiac amyloidosis // PLoS One. 2021. Vol. 16, N 8. P. e0255487. doi: 10.1371/journal.pone.0255487

15. Quarta C.C., Solomon S.D., Uraizee I., et al. Left ventricular structure and function in transthyretin-related versus light-chain cardiac amyloidosis // Circulation. 2014. Vol. 129, N 18. P. 1840–1849. doi: 10.1161/CIRCULATIONAHA.113.006242

16. Stern L.K., Patel J. Cardiac amyloidosis treatment // Methodist Debakey Cardiovasc J. 2022. Vol. 18, N 2. P. 59–72. doi: 10.14797/mdcvj.1050

17. Kristen A.V., aus dem Siepen F., Scherer K., et al. Comparison of different types of cardiac amyloidosis by cardiac magnetic resonance imaging // Amyloid. 2015. Vol. 22, N 2. P. 132–141. doi: 10.3109/13506129.2015.1020153

18. Gillmore J.D., Maurer M.S., Falk R.H., et al. Nonbiopsy diagnosis of cardiac transthyretin amyloidosis // Circulation. 2016. Vol. 133, N 24. P. 2404–2412. doi: 10.1161/CIRCULATIONAHA.116.021612

19. Martinez-Naharro A., Treibel T.A., Abdel-Gadir A., et al. Magnetic resonance in transthyretin cardiac amyloidosis // J Am Coll Cardiol. 2017. Vol. 70, N 4. P. 466–477. doi: 10.1016/j.jacc.2017.05.053

20. Binder C., Duca F., Binder T., et al. Prognostic implications of pericardial and pleural effusion in patients with cardiac amyloidosis // Clinical Research in Cardiology. 2021. Vol. 110, N 4. P. 532–543. doi: 10.1007/s00392-020-01698-7

21. Reddy A., Singh V., Karthikeyan B., et al. Biventricular strain imaging with cardiac MRI in genotyped and histology validated amyloid cardiomyopathy // Cardiogenetics. 2021. Vol 11, N 3, P. 98–110. doi: 10.3390/cardiogenetics11030011

AUTHORS' INFO

*** Zainab M. Magomedova,** MD;
address: 4B Verkhnyaya str., village Rumyantsevo, 108811, Moscow, Russia;
ORCID: 0000-0001-6753-1525;
eLibrary SPIN: 5271-4915;
e-mail: magomedova.zainab.97@mail.ru

Tatyana V. Nikiforova, MD;
ORCID: 0000-0003-3072-8951;
eLibrary SPIN: 4997-0330;
e-mail: attrcmp@gmail.com

Dmitry Yu. Shchekochikhin, MD, Cand. Sci. (Medicine), Assistant Professor;
ORCID: 0000-0002-8209-2791;
eLibrary SPIN: 3753-6915;
e-mail: agishm@list.ru

Ekaterina S. Pershina, MD, Cand. Sci. (Medicine);
ORCID: 0000-0002-3952-6865;
eLibrary SPIN: 7311-9276;
e-mail: pershina86@mail.ru

ОБ АВТОРАХ

*** Магомедова Зайнаб Магомедовна;**
адрес: Россия, 108811, Москва, деревня Румянцево, ул. Верхняя, д. 4Б;
ORCID: 0000-0001-6753-1525;
eLibrary SPIN: 5271-4915;
e-mail: magomedova.zainab.97@mail.ru

Никифорова Татьяна Вячеславовна;
ORCID: 0000-0003-3072-8951;
eLibrary SPIN: 4997-0330;
e-mail: attrcmp@gmail.com

Щекочихин Дмитрий Юрьевич, канд. мед. наук, доцент;
ORCID: 0000-0002-8209-2791;
eLibrary SPIN: 3753-6915;
e-mail: agishm@list.ru

Першина Екатерина Сергеевна, канд. мед. наук;
ORCID: 0000-0002-3952-6865;
eLibrary SPIN: 7311-9276;
e-mail: pershina86@mail.ru

Konstantin V. Kovalev, MD;
ORCID: 0009-0004-4841-041X;
e-mail: radix606@yandex.ru
Khadizhat S. Abdulmazhidova;
ORCID: 0009-0008-5064-7802;
e-mail: abdulmazhidova.kh@mail.ru
Daria S. Rassechkina, MD;
ORCID: 0009-0007-8825-8485;
e-mail: rassechkina@yandex.ru
Alexander E. Grachev, MD, Cand. Sci. (Medicine);
ORCID: 0000-0001-7221-9392;
eLibrary SPIN: 4281-3923;
e-mail: gra4al@yandex.ru
Irina G. Rekhtina, MD, Dr. Sci. (Medicine);
ORCID: 0000-0001-5440-4340;
eLibrary SPIN: 4920-7144;
e-mail: rekhtina.i@blood.ru
Alexey N. Volovchenko, MD, Cand. Sci. (Medicine);
ORCID: 0000-0002-0923-735X;
eLibrary SPIN: 4120-8740;
e-mail: dr.volovchenko@mail.ru
Susanna D. Sarkisyan;
ORCID: 0000-0002-6454-1370;
e-mail: sysanna.sarkisyan.2001@mail.ru
Valentin E. Sinitsyn, MD, Dr. Sci. (Medicine), Professor;
ORCID: 0000-0002-5649-2193;
eLibrary SPIN: 8449-6590;
e-mail: vsini@mail.ru
Denis A. Andreev, MD, Dr. Sci. (Medicine);
ORCID: 0000-0002-0276-7374;
eLibrary SPIN: 8790-8834;
e-mail: dennan@mail.ru

Ковалёв Константин Витальевич;
ORCID: 0009-0004-4841-041X;
e-mail: radix606@yandex.ru
Абдулмажидова Хадижат Салахудиновна;
ORCID: 0009-0008-5064-7802;
e-mail: abdulmazhidova.kh@mail.ru
Рассечкина Дарья Сергеевна;
ORCID: 0009-0007-8825-8485;
e-mail: rassechkina@yandex.ru
Грачев Александр Евгеньевич, канд. мед. наук;
ORCID: 0000-0001-7221-9392;
eLibrary SPIN: 4281-3923;
e-mail: gra4al@yandex.ru
Рехтина Ирина Германовна, д-р мед. наук;
ORCID: 0000-0001-5440-4340;
eLibrary SPIN: 4920-7144;
e-mail: rekhtina.i@blood.ru
Воловченко Алексей Николаевич, канд. мед. наук;
ORCID: 0000-0002-0923-735X;
eLibrary SPIN: 4120-8740;
e-mail: dr.volovchenko@mail.ru
Саркисян Сусанна Давитовна;
ORCID: 0000-0002-6454-1370;
e-mail: sysanna.sarkisyan.2001@mail.ru
Синицын Валентин Евгеньевич, д-р мед. наук, профессор;
ORCID: 0000-0002-5649-2193;
eLibrary SPIN: 8449-6590;
e-mail: vsini@mail.ru
Андреев Денис Анатольевич, д-р мед. наук;
ORCID: 0000-0002-0276-7374;
eLibrary SPIN: 8790-8834;
e-mail: dennan@mail.ru

* Corresponding author / Автор, ответственный за переписку