Nikiforova T. V.[1], Charaya K. V.[1], Shchekochikhin D. Yu. [1, 2], Magomedova Z. M. [1, 2], Enokyan M. S.[1], Volovchenko A. N.[1], Khamzatkhanova A. H.[1], Starovoitova T. A.[2], Bogdanova A. A.[1, 2], Karalkin A. V.[2], Pasha S. P.[2], Pershina E. S.[1, 2], Grachev A. E.[3], Zhirov I. V.[4], Andreev D. A.[1]

[1] Sechenov First Moscow State Medical University Moscow, Russia

[2] Pirogov Municipal Clinical Hospital #1, Moscow, Russia

[3] National Medical Research Center of Hematology, Moscow, Russia

[4] Chazov National Medical Research Center of Cardiology, Moscow, Russia

Primary Data on ATTR-Amyloidosis Prevalence Among Elderly Patients With Left Ventricular Hypertrophy in Russia

Aim	To estimate the prevalence of amyloid cardiomyopathy (CM) caused by transthyretin amyloidosis (ATTR) and immunoglobulin light chain (AL) amyloidosis among patients aged >65 years with interventricular septal (IVS) hypertrophy of ≥14 mm.
Material and methods	From January through August 2023, 60 patients (mean age 7.2±7.3 years, 34 (56.67%) men) were enrolled. Patients meeting the inclusion criteria underwent an echocardiographic study with determining the myocardial longitudinal strain, myocardial scintigraphy with ^{99m}Tc-pyrfotech, myocardial single-photon emission computed tomography, measurement of N-terminal fragment of brain natriuretic peptide and troponin I, and the immunochemical study of serum and urine proteins with measurement of free light chains. In the presence of grades 2 and 3 radiopharmaceutical uptake according to scintigraphy, a molecular genetic study was performed for differential diagnosis of wild-type transthyretin amyloidosis (wtATTR) and hereditary/variant (hATTR) ATTR-CM.
Results	According to data of myocardial scintigraphy with 99mTc-pyrfotech, grade 3 uptake in the absence of monoclonal secretion was detected in 5 (8.3%) cases and grade 2 radiotracer uptake in the absence of monoclonal secretion was detected in 6 (10%) patients. Myeloma complicated by AL amyloidosis and primary AL amyloidosis were found in 5 (8.3%) patients.
Conclusion	Among patients aged ≥65 years with IVS hypertrophy ≥14 mm, amyloid CM was detected in 20% of cases (12 patients), including 5 cases (8.3%) of AL amyloidosis and 7 cases (11.7%) of ATTR amyloidosis.

Keywords	Left ventricular hypertrophy; amyloidosis; transthyretin cardiomyopathy
For citations	Nikiforova T. V., Charaya K. V., Shchekochikhin D. Yu., Magomedova Z. M., Enokyan M. S., Volovchenko A. N. et al. Primary Data on ATTR-Amyloidosis Prevalence Among Elderly Patients With Left Ventricular Hypertrophy in Russia. Kardiologiia. 2024;64(4):54–60. [Russian: Никифорова Т. В., Чарая К. В., Щекочихин Д. Ю., Магомедова З. М., Енокян М. С., Воловченко А. Н. и др. Первые данные о распространенности транстиретинового амилоидоза при гипертрофии левого желудочка в России. Кардиология. 2024;64(4):54–60].

Corresponding author	Nikiforova T. V. E-mail: attrcmp@gmail.com

Introduction

Amyloidosis is a heterogeneous group of diseases associated with the deposition of soluble plasma proteins in the extracellular space as abnormal fibrillar glycoproteins [1]. In cardiology, two variants of amyloidosis are of particular interest: immunoglobulin light chain (AL) amyloidosis and transthyretin (ATTR) amyloidosis [2]. Both variants have traditionally been regarded as rare diseases with an extremely poor prognosis, typically affecting elderly patients and often diagnosed posthumously [3].

Improvements in clinical investigations, such as the introduction of myocardial scintigraphy with osteotropic radiopharmaceuticals, have facilitated the noninvasive diagnosis of ATTR-amyloid cardiomyopathy (ATTR-CM) and enabled lifetime diagnosis protocols for the disease. Recent studies have shown that both variants of ATTR-CM (wild-type, formerly referred to as senile, and hereditary) are causes of rapidly progressive heart failure with preserved left ventricular (LV) ejection fraction. New drugs, stabilizers of the ATTR-amyloid tetramer, have demonstrated an ability to improve patient prognosis and functional status when administered early in therapy. Consequently, it is critical to diagnose the disease as early as possible [4]. Currently, tafamidis is the only approved specific ATTR-amyloid tetramer stabilizer for ATTR-CM therapy [5].

The prevalence of ATTR-CM among elderly patients with LV hypertrophy of 12–14 mm is known to range from 18%

ISSN 0022-9040. Kardiologiia. 2024;64(4). DOI: 10.18087/cardio.2024.4.n2611

to 34%, largely determined by the diagnostic capabilities and age structure of study groups [6]. However, there is no available data on the prevalence of ATTR-CM in the Russian Federation. Clinical research on amyloid cardiomyopathy in Russia is limited to case series descriptions.

Objective

To estimate the prevalence of amyloid cardiomyopathy caused by ATTR amyloidosis and AL amyloidosis among patients with ≥ 14 mm interventricular septal (IVS) hypertrophy aged 65 years and older.

Material and Methods

This paper presents interim results from January through August 2023 from a prospective cohort study investigating ATTR-CM prevalence. The study included patients who met two *inclusion criteria:*

1. Age ≥ 65 years
2. IVS hypertrophy ≥ 14 mm

Patients were included in the study after signing the informed consent.

The *exclusion criteria* were:

1) previously diagnosed infiltrative cardiomyopathy (CMP), hypertrophic cardiomyopathy, severe aortic stenosis (aortic valve opening area less than 0.75 cm^2, mean pressure gradient greater than 40 mm Hg, peak velocity greater than 4 m/s), and storage diseases;
2) history of myocardial infarction (MI) less than 4 weeks old;
3) a recent thoracic trauma or surgical interventions, including a history of thoracotomy
4) regular administration of hydroxychloroquine within the past 4 weeks;
5) inability to sign informed consent or refusal to continue participation in the study.

The study was approved by the local ethics committee of the Sechenov First Moscow State Medical University (No. 25–22 dated 08.12.2022).

The study included patients with documented left ventricular hypertrophy (LVH) of 14 mm or more on echocardiography who had been referred from third-party facilities. All patients underwent a repeat echocardiogram on the VIVID E95 system. Upon confirmation of hypertrophy, patients were included in the study after completing the informed consent form.

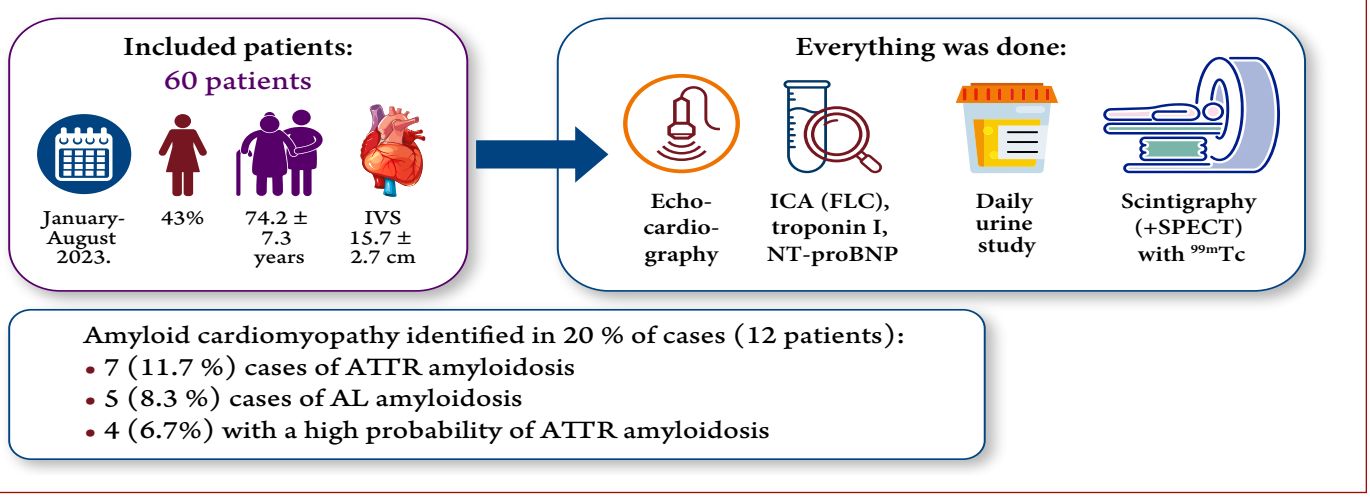
The severity of symptoms of chronic heart failure (CHF) was determined according to the New York Heart Association (NYHA) classification, and followed by a physical examination.

Echocardiographic examinations were conducted according to the established protocol [7], including speckle-tracking imaging to assess LV global longitudinal strain (GLS). A 12-lead electrocardiogram (ECG) was performed.

Venous blood samples were collected to determine levels of N-terminal pro-brain natriuretic peptide (NT-proBNP) and troponin I. Serum creatinine levels were extracted from medical records, no more than three months old, and the glomerular filtration rate (GFR) was calculated using the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) formula. Patient-provided medical documentation was thoroughly examined, including Holter electrocardiogram monitoring (ECG HM) data.

All patients underwent immunochemical analysis (ICA) of blood serum and daily urine samples (20–30 mL), which included electrophoresis of serum and urine proteins with immunofixation (Sebia, France), and determination of serum immunoglobulin free light chains (FLC) by nephelometry using κλoneus kits (Trimero Diagnostics) on an Immage 800 analyzer (Beckman Coulter). In the event that monoclonal secretion was identified, a consultation was sought with a hematologist, with the objective of determining the indications for bone marrow trepanobiopsy and subcutaneous fat aspiration biopsy.

Central illustration. Primary Data on ATTR-Amyloidosis Prevalence Among Elderly Patients With Left Ventricular Hypertrophy in Russia



All patients underwent cardiac radionuclide scanning, including targeted planar scans and single-photon emission computed tomography (SPECT) using a Discovery NM/CT 670 dual-detector gamma camera with low-energy high-resolution (LEHR) collimators and a CT subsystem. Pirfotech 99 mTc (a Russian analog of pyrophosphate) was used as the intravenous radiopharmaceutical, with an activity of 700–740 MBq (19–20 mCi) per 1 mL. Investigation and interpretation of results followed expert recommendations for multimodality imaging in cardiac amyloidosis (2019) [8, 9].

The myocardial uptake of the radiopharmaceutical of grade 2–3 was interpreted as characteristic of ATTR amyloidosis. A molecular genetic investigation was conducted in this group of patients by direct sequencing of the entire coding sequence and exon-intron regions of the transthyretin (TTR) gene. The objective was to differentiate between wild-type (wtATTR) and hereditary/variant (hATTR) type ATTR-CM.

The final diagnosis of ATTR amyloidosis was confirmed based on the results of scintigraphy with technetium pyrophosphate, in the presence of radiopharmaceutical uptake degree 2–3, and the presence of symptoms and laboratory test/clinical investigation evidence of CHF. The diagnosis of AL amyloidosis was confirmed in patients by the presence of amyloid in biopsy results.

Statistical data processing was conducted using SPSS 22.0 (SPSS Inc., USA). The distribution of quantitative variables was evaluated using the Kolmogorov-Smirnov test. Normally distributed variables were expressed as the mean and standard deviation (M ± SD). Non-normally distributed variables were described by the median and interquartile range between 25th and 75th percentiles and compared using non-parametric tests. The groups were compared by quantitative variables using the analysis of variance (ANOVA). Categorical variables were expressed as the absolute and relative values and were compared using the chi-squared test or Fisher's exact test. The differences were considered statistically significant at $p < 0.05$.

Results

From January to August 2023, 60 patients met the inclusion criteria. The mean age of the patients was 74.2 ± 7.3 years, and 34 (56.67%) patients were male. The study design is shown in Figure 1.

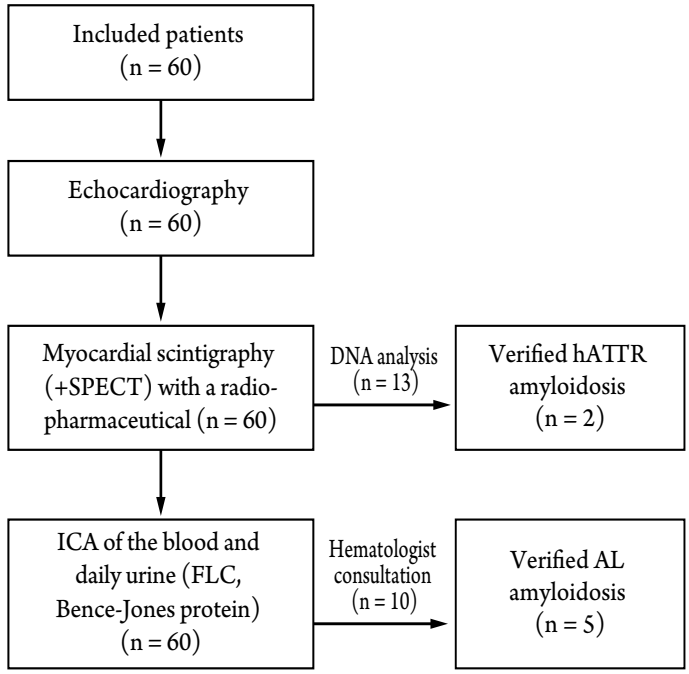
The severity of CHF symptoms was NYHA class I in 3 (13.95%) patients, class II in 30 (50%) patients, and class III–IV in 11 (18.3%) patients.

The baseline clinical and demographic characteristics of patients are shown in Table 1.

Coronary artery disease (CAD), defined as a history of MI, exertional angina (without functional class), or revascularization by percutaneous coronary intervention/coronary artery bypass grafting, was present in 19 (31.6%) patients.

Echocardiography showed a mean IVS thickness of 15.7 ± 2.7 mm, and 5 (8.3%) patients exhibited LVEF below 50%.

Figure 1. Echocardiography study design



SPECT, single-photon emission computed tomography; ICA, immunochemical assay; FLC, free light chain.

Table 1. Clinical and demographic characteristics of patients

Parameter	Result
Age, years, M ± SD	74.2 ± 7.3
Male, n (%)	34 (57)
CAD, n (%)	19 (31.6)
History of MI, n (%)	12 (20)
History of revascularization, n (%)	10 (16.6)
Exertional angina, n (%)	3 (5)
Hypertension, n (%)	51 (85)
AF/AFL, n (%)	24 (40)
Diabetes mellitus type 2, n (%)	12 (20)
CHF class III-IV, n (%)	11 (18.3)
Carpal tunnel syndrome, n (%)	4 (6.7)
NT-proBNP > 300 pg/mL, n (%)	45 (75)
AV block grade 1, n (%)	13 (21.6)
LVH on the electrocardiogram (Sokolow-Lyon criteria), n (%)	11 (18.3)
Troponin I > 0.023 ng/mL, n (%)	7 (11.7)
LVEF < 50 %, n (%)	5 (8.3)
Low voltages of the QRS complex on ECG, n (%)*	5 (8.3)
Complete left bundle branch block, n (%)	2 (3.3)

* Low QRS voltages defined as < 5 mm in standard leads or < 10 mm in precordial leads. AF, atrial fibrillation; AFL, atrial flutter; AV, atrioventricular; LVH, left ventricular hypertrophy; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-brain natriuretic peptide.

ISSN 0022-9040. Kardiologiia. 2024;64(4). DOI: 10.18087/cardio.2024.4.n2611

The median NT-proBNP was 694 [317; 2180] pg/mL, and the mean troponin I level was 0.017 ± 0.02 ng/mL. Elevated NT-proBNP (> 300 pg/mL) was identified in 45 (75%) patients, while elevated troponin I (> 0.023 ng/mL) was observed in 7 (11.7%) patients. Elevated levels of both biomarkers were observed in 7 (11.7%) patients.

Monoclonal secretion was detected by ICA in 10 (16.7%) patients. A diagnosis of multiple myeloma complicated by AL amyloidosis or primary AL amyloidosis was confirmed by trepanobiopsy in 5 (8.3%) patients, while the presence of monoclonal gammopathy of undetermined significance (MGUS) was detected in 5 (8.3%) patients.

Myocardial scintigraphy and SPECT imaging revealed that 5 (8.3%) patients exhibited grade 3 myocardial fluorescence (radiopharmaceutical uptake), 8 (13.3%) exhibited grade 2, 21 (35%) exhibited grade 1, and 26 (43.3%) exhibited grade 0. The degree of radiopharmaceutical uptake is provided to characterize patients in Table 2.

The data obtained indicated that patients with Grade 3 exhibited statistically significant elevations in troponin I values, more severe IVS hypertrophy and LV posterior wall (PW) hypertrophy, and a higher NYHA class. In 46 (76.7%) patients, there was an increase in NT-proBNP levels of more than 300 pg/mL. Among the patients with grade 2–3, 5 (38.5%) exhibited clinically significant signs of CHF class III–IV.

A pathogenic nucleotide variant was identified in 2 patients with radiopharmaceutical uptake grade 3 via molecular genetic investigation. In one case, a mutation c.148G>A (p.Val50Met) was identified in exon 2 of the TTR gene in a heterozygous state. In another case, a pathogenic variant c.323A>G (p.His108Arg) was identified in exon 3 of the TTR gene, also in a heterozygous state. These variants have been repeatedly documented in the context of hereditary transthyretin amyloidosis. Two cases of ATTRv amyloidosis were identified among 13 patients with grade 2–3.

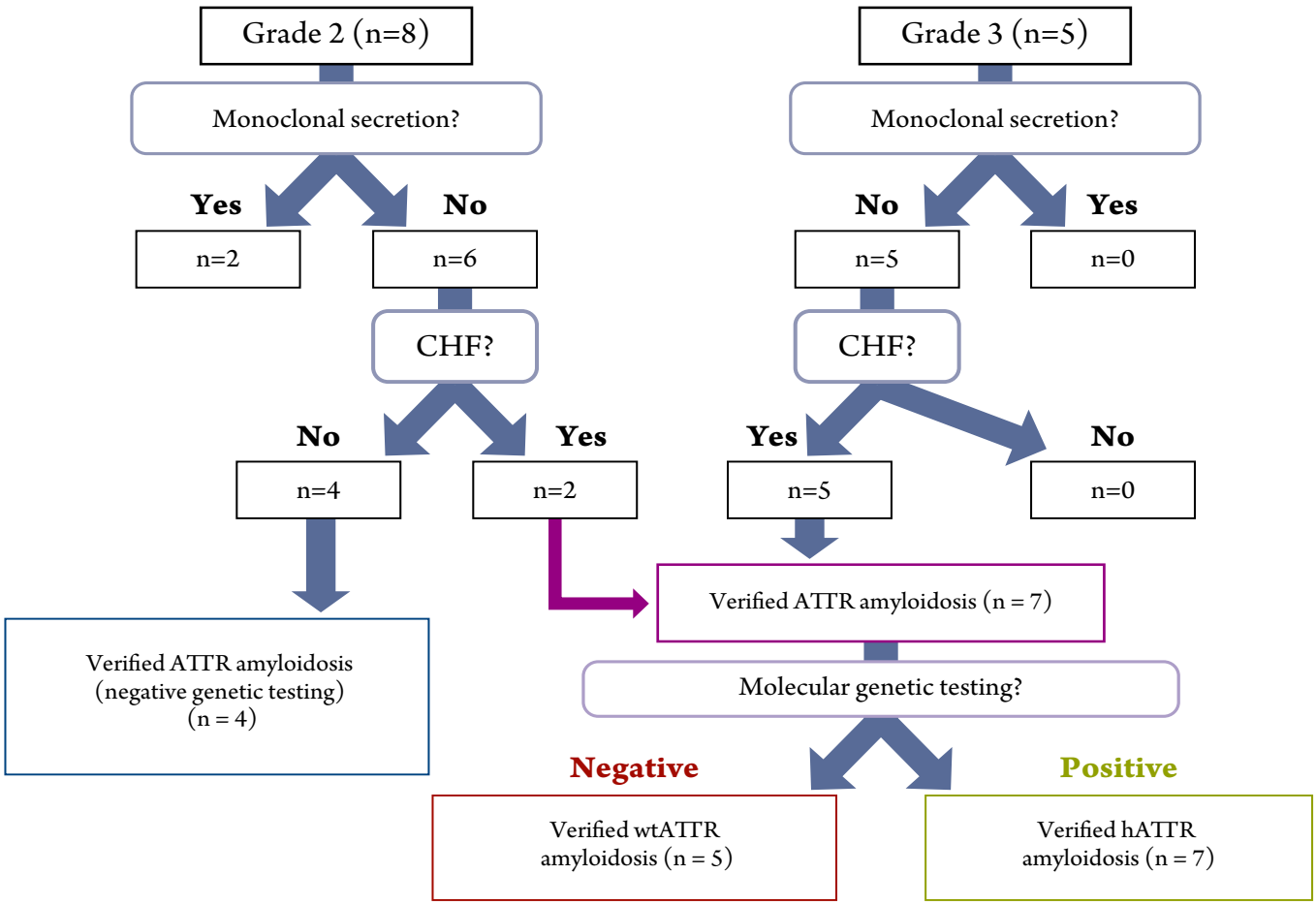
An examination of 60 patients aged 65 years and older with IVS hypertrophy of ≥14 mm revealed 5 (8.3%) cases of AL amyloidosis and 7 (11.7%) cases of transthyretin amyloid cardiomyopathy. In patients with ATTR amyloidosis (2 patients with grade 2 according to scintigraphy using radiopharmaceutical, 5 patients with grade 3), there was a concordant change in the levels of NT-proBNP, GLS, and troponin I. High probability of ATTR amyloidosis was detected in 4 patients with no signs found by clinical investigations and laboratory tests. The patients in this group are currently undergoing dynamic follow-up. A graphical representation

Table 2. Clinical and laboratory characteristics of patients depending on the grade of radiopharmaceutical uptake

Parameter	Grade 0 (n = 26)	Grade 1 (n = 21)	Grade 2 (n = 8)	Grade 3 (n = 5)
Age, years	74.3 ± 6.3	74.1 ± 7.6	70 ± 3.96	80.8 ± 11.48
Male, n (%)	14 (53.8)	13 (61.9)	3 (37.5)	4 (80)
Hypertension, n (%)	22 (84.6)	18 (85.7)	8 (100)	3 (60)
AF/AFL, n (%)*	7 (26.9)	13 (61.9)	2 (25)	2 (40)
Diabetes mellitus, n (%)	6 (23)	5 (23.8)	1 (12.5)	0
CAD, n (%)	5 (19.2)	9 (42.9)	4 (50)	1 (20)
CHF class III-IV, n (%)*#	2 (7.7)	4 (19)	1 (12.5)	4 (80)
Pacemaker implantation, n (%)	1 (3.8)	3 (14.3)	0	0
IVS thickness, mm#	16.2 ± 3	14.7 ± 2.7	15.2 ± 1.57	17.6 ± 0.9
LVPW thickness, mm*#	12.9 ± 1.9	12.45 ± 1.9	11.9 ± 1.5	16 ± 1.2
Troponin I, ng/mL (0–0.023)*#	0.018 ± 0.023	0.011 ± 0.005	0.016 ± 0.017	0.043 ± 0.04
NT-proBNP, pg/mL (0–133)*	610 [296; 1180]	650 [334; 2590]	388 [277; 1338]	9810 [4240; 10000]
Monoclonal secretion according to immunochemical analysis, n (%)*	1 (3.8)	6 (28.6)	2 (25)	1 (20)
GLS, %	-13.4 ± 2.9	-15.4 ± 2.9	-16.3 ± 3.2	-10.7 ± 5.8
LVEF, %	60.1 ± 5.4	56.6 ± 9.4	60.4 ± 6.8	57 ± 4.1
Low voltages on ECG, n (%)	0	2 (9.5)	1 (12.5)	1 (2)
LVH on the electrocardiogram (Sokolow-Lyon criteria), n (%)	7 (26.9)	3 (14.3)	1 (12.5)	0
AV block grade 1, n (%)	4 (15.3)	4 (19)	2 (25)	2 (40)
Complete left bundle branch block, n (%)	0	2 (9.5)	0	0
Final diagnosis				
AL amyloidosis	1 (3.8)	2 (9.5)	2 (25)	0
ATTR amyloidosis	0	0	2 (25)	5 (100)

* $p < 0.05$ between groups; # $p < 0.05$ between grade 3 and the rest of the patient groups.
IVS, interventricular septum; LVPW, left ventricular posterior wall; GLS, global longitudinal strain; LVEF, left ventricular ejection fraction; LV, left ventricular; AF, atrial fibrillation; AFL, atrial flutter; AV, atrioventricular; NT-proBNP, N-terminal pro-brain natriuretic peptide; AL, immunoglobulin amyloid light-chain; ATTR, transthyretin amyloidosis.

Figure 2. Schematic image of the results of the verification of amyloidosis in patients with grade 2-3 radiopharmaceutical uptake



CHF, chronic heart failure; wtATTR, wild-type transthyretin amyloidosis; hATTR, hereditary ATTR amyloidosis

of the results obtained in the verification of transthyretin cardiomyopathy is presented in Figure 2.

Table 3 presents the comparative characteristics of clinical manifestations, laboratory test results, and clinical investigation findings, as well as red flags of the disease among patients with verified AL amyloidosis and ATTR amyloidosis.

It is noteworthy that 4 patients exhibited radiopharmaceutical uptake corresponding to grade 2 yet had neither clinical signs of CHF nor elevated levels of troponin I and NT-proBNP. The definitive diagnosis in this group of patients remains unclear.

Discussion

This paper presents preliminary data on the prevalence of ATTR-CM among Russian patients aged ≥ 65 years with LVH ≥ 14 mm. The epidemiology of amyloidosis in the Russian Federation remains an open question. It is known that the detection rate of cardiac amyloidosis at a Moscow hospital between 2008 and 2019 was 12.6 per 100,000 people [10].

The issue of ATTR-CM prevalence is currently a subject of great interest to researchers abroad. This is due to the potential for non-invasive diagnosis of the disease and the development of specific therapy that can influence the course of the disease [3, 4].

The modern diagnostic algorithm developed by Bokhari et al. [2], based on studies with 99mTc pyrophosphate, has greatly simplified the detection of ATTR-CM. Since its introduction into clinical practice, the above algorithm has been employed in a number of clinical trials, including this study. The algorithm enables the differential diagnosis of AL–CMP and ATTR-CM, thus facilitating the clarification of data on the prevalence of the latter. The final step in the diagnosis of ATTR-CM is a molecular genetic study that allows wtATTR- and hATTR-type CMP to be differentiated.

Foreign data indicate a high prevalence of ATTR-CM. A study conducted by Italian authors among patients ≥55 years of age with LVH ≥12 mm in female patients and ≥13 mm in female patients revealed a prevalence of cardiac amyloidosis of 28%. Of this, 23.5% was attributable to ATTR-CM [3]. In the Finnish registry, autopsy revealed the presence of wtATTR-CM in 25% of patients aged ≥ 85 years [11]. Spanish data indicates that wtATTR-CM is present in 13% of patients with HFpEF, LVH ≥ 12 mm at the age of 60 years and older [12]. American study results indicate that among patients with CHF, LVEF >

ISSN 0022-9040. Kardiologiia. 2024;64(4). DOI: 10.18087/cardio.2024.4.n2611

40% and LVH > 12 mm, the prevalence of ATTR-CM is 6% or higher, with an increasing prevalence with age. In patients aged 60 to 69 years, the prevalence is 0%, while in patients older than 90 years, it is 21% [13]. In a study conducted by Lindmark et al. [14] in Sweden, the prevalence of wtATTR-CM among patients with CHF and LVH ≥ 14 mm was 20%.

Our data indicates that the prevalence of ATTR-CM among elderly patients with LVH is 11.7%, with wtATTR-CM representing 71.4% of this group. However, when high-probability ATTR-CM is taken into account, the prevalence reaches 18.3%. Additionally, in 5 (8.3%) cases, AL amyloidosis was identified for the first time in the absence of other clinically significant organ manifestations at the time of examination. A cardiologist was the first specialist to diagnose a hematologic disease.

A noteworthy aspect of the presented results is the broad inclusion criteria, which does not depend on clinical signs or the presence or absence of CHF. A group of patients presenting with no clinical signs or laboratory test and clinical investigation evidence of CHF but with grade 2 radiopharmaceutical uptake according to myocardial scintigraphy was identified. It is hypothesized that the preclinical stages of CMP have been identified in this group. These patients are subjected to active monitoring, with repeated laboratory tests and clinical investigations conducted every three months. These include echocardiography and the study of biomarkers (NT-proBNP and troponin I). The necessity of cardiac magnetic resonance imaging and/or endomyocardial biopsy is discussed.

Thus, of the studied group, 11 (18.3%) patients met the criteria for the diagnosis of ATTR-CM, while 4 (6.7%) patients exhibited no clinical signs and lacked laboratory test and clinical investigation evidence of CHF. The findings underscore the significance of understanding the prevalence of amyloidosis across diverse age groups and its correlation with heart failure. Moreover, they highlight the necessity for a heightened level of vigilance among specialists, prompt routing of patients, and the administration of specific treatment [15]. Further large-scale studies are required to confirm these findings and provide greater insight into amyloidosis epidemiology in Russia.

Table 3. Comparative characterization of patients with amyloidosis

Characteristics	AL amyloidosis (n = 5)	ATTR amyloidosis (n = 7)
Clinical signs		
Reduced exercise tolerance, n (%)	5 (100)	5 (71)
Polyneuropathy, n (%)	3 (60)	5 (71)
Laboratory evidence		
GFR, mL/min/1.73m2	47 [35.8; 60.8]	53.5 [51.5; 54.8]
Proteinuria > 1.0 g/day, n (%)	2 (40)	0
Troponin I, ng/mL (0–0.023)	0.03 ± 0.02	0.02 ± 0.01
NT-proBNP, pg/mL (0–133)	9440 [8610; 11700]	4240 [1925; 9905]
Monoclonal component, n (%)	5 (100)	1 (14.3)
ECG		
AV block, n (%)	1 (20)	3 (43)
Atrial fibrillation, n (%)	3 (60)	3 (43)
SVT, n (%)	0	2 (29)
LBBB/RBBB, n (%)	1 (20)	1 (14)
Low voltages QRS in the standard leads, n (%)	1 (20)	1 (14)
Echocardiography		
IVS, mm	14.8 ± 2.1	17.1 ± 1.2
GLS, %	-11.4 ± 5.8	-10.9 ± 5.3

AL, imunoglobulin amyloid light-chain; ATTR, transthyretin amyloidosis; GFR, glomerular filtration rate; NT-proBNP, N-terminal pro-brain natriuretic peptide; AV, atrioventricular; SVT, supraventricular tachycardia (unstable paroxysms on Holter ECG monitoring); LBBB, left bundle branch block; RBBB, right bundle branch block; IVS, interventricular septum; GLS, global longitudinal strain.

Limitations

The primary limitation of this study is the relatively small sample size and the inclusion of patients regardless of the presence or absence of symptoms. Additionally, the study was designed as a single-center study, which may limit the generalizability of the findings. This paper presents the interim results of the study.

Conclusion

Among patients aged 65 years and older with interventricular septal hypertrophy of ≥ 14 mm, amyloid cardiomyopathy was identified in 20% of cases (12 patients). Specifically, 5 (8.3%) cases of AL amyloidosis and 7 (11.7%) cases of transthyretin amyloid cardiomyopathy were identified.

Acknowledgments

The authors thank Saliya Akhsanovna Marjina, Alla Mikhailovna Kovrigina and other employees of the National Medical Research Center of Hematology.

Funding

The project was funded by an independent grant from Pfizer. The authors received no financial or editorial support from Pfizer related with the preparation of this publication.

No conflict of interest is reported.

The article was received on 20/11/2023

REFERENCES

1. Lachmann H, Hawkins P. Systemic amyloidosis. Current Opinion in Pharmacology. 2006;6(2):214–20. DOI: 10.1016/j.coph.2005.10.005
2. Bokhari S, Castaño A, Pozniakoff T, Deslisle S, Latif F, Maurer MS. (99m) Tc-Pyrophosphate Scintigraphy for Differentiating Light-Chain Cardiac Amyloidosis From the Transthyretin-Related Familial and Senile Cardiac Amyloidoses. Circulation: Cardiovascular Imaging. 2013;6(2):195–201. DOI: 10.1161/CIRCIMAGING.112.000132
3. Porcari A, Bussani R, Merlo M, Varrà GG, Pagura L, Rozze D et al. Incidence and Characterization of Concealed Cardiac Amyloidosis Among Unselected Elderly Patients Undergoing Post-mortem Examination. Frontiers in Cardiovascular Medicine. 2021;8:749523. DOI: 10.3389/fcvm.2021.749523
4. Kittleson MM, Maurer MS, Ambardekar AV, Bullock-Palmer RP, Chang PP, Eisen HJ et al. Cardiac Amyloidosis: Evolving Diagnosis and Management: A Scientific Statement From the American Heart Association. Circulation. 2020;142(1):7–22. DOI: 10.1161/CIR.0000000000000792
5. Maurer MS, Schwartz JH, Gundapaneni B, Elliott PM, Merlini G, Waddington-Cruz M et al. Tafamidis Treatment for Patients with Transthyretin Amyloid Cardiomyopathy. New England Journal of Medicine. 2018;379(11):1007–16. DOI: 10.1056/NEJMoa1805689
6. Antonopoulos AS, Panagiotopoulos I, Kouroutzoglou A, Koutsis G, Toskas P, Lazaros G et al. Prevalence and clinical outcomes of transthyretin amyloidosis: a systematic review and meta-analysis. European Journal of Heart Failure. 2022;24(9):1677–96. DOI: 10.1002/ejhf.2589
7. Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, Foster MC et al. Guidelines for Performing a Comprehensive Transthoracic Echocardiographic Examination in Adults: Recommendations from the American Society of Echocardiography. Journal of the American Society of Echocardiography. 2019;32(1):1–64. DOI: 10.1016/j.echo.2018.06.004
8. Dorbala S, Ando Y, Bokhari S, Dispenzieri A, Falk RH, Ferrari VA 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. Circulation: Cardiovascular Imaging. 2021;14(7):e000029. DOI: 10.1161/HCI.0000000000000029
9. Schockling EJ, Farrell MB, Embry-Dierson M, Warren J, Jerome S. Cardiac Amyloidosis Imaging, Part 2: Quantification and Technical Considerations. Journal of Nuclear Medicine Technology. 2023;51(2):90–8. DOI: 10.2967/jnmt.123.265416
10. Reznik E.V., Stepanova E.A., Nguyen T.L., Sirenova I.O., Salikov A.V., Nikitin I.G. Retrospective analysis of cardiovascular involvement in patients with systemic amyloidosis. Cardiovascular Therapy and Prevention. 2021;20(1):35–46. [Russian: Резник Е.В., Степанова Е.А., Нгуен Т.Л., Сиренова И.О., Саликов А.В., Никитин И.Г. Ретроспективный анализ поражения сердечно-сосудистой системы у больных системным амилоидозом. Кардиоваскулярная терапия и профилактика. 2021;20(1):35-46]. DOI: 10.15829/1728-8800-2021-2496
11. Tanskanen M, Peuralinna T, Polvikoski T, Notkola I, Sulkava R, Hardy J et al. Senile systemic amyloidosis affects 25% of the very aged and associates with genetic variation in alpha2-macroglobulin and tau: a population-based autopsy study. Annals of Medicine. 2008;40(3):232–9. DOI: 10.1080/07853890701842988
12. González-López E, Gallego-Delgado M, Guzzo-Merello G, de Haro-del Moral FJ, Cobo-Marcos M, Robles C et al. Wild-type transthyretin amyloidosis as a cause of heart failure with preserved ejection fraction. European Heart Journal. 2015;36(38):2585–94. DOI: 10.1093/eurheartj/ehv338
13. AbouEzzeddine OF, Davies DR, Scott CG, Fayyaz AU, Askew JW, McKie PM et al. Prevalence of Transthyretin Amyloid Cardiomyopathy in Heart Failure With Preserved Ejection Fraction. JAMA Cardiology. 2021;6(11):1267–74. DOI: 10.1001/jamacardio.2021.3070
14. Lindmark K, Pilebro B, Sundström T, Lindqvist P. Prevalence of wild type transtyrethin cardiac amyloidosis in a heart failure clinic. ESC Heart Failure. 2021;8(1):745–9. DOI: 10.1002/ehf2.13110
15. Tereshchenko S.N., Zhirov I.V., Moiseeva O.M., Adasheva T.V., Ansheles A.A., Barbarash O.L. et al. Practical guidelines for the diagnosis and treatment of transthyretin amyloid cardiomyopathy (ATTR-CM or transthyretin cardiac amyloidosis). Therapeutic Archive. 2022;94(4):584–95. [Russian: Терещенко С.Н., Жиров И.В., Моисеева О.М., Адашева Т.В., Аншелес А.А., Барбараш О.Л. и др. Практические рекомендации по диагностике транстиретиновой амилоидной кардиомиопатии (ATTR-КМП или транстиретинового амилоидоза сердца). Терапевтический архив. 2022;94(4):584–59]. DOI: 10.26442/00403660.2022.04.201465

ISSN 0022-9040. Kardiologiia. 2024;64(4). DOI: 10.18087/cardio.2024.4.n2611