Bocharov A.V.[1, 2], Popov L.V.[3], Lagkuev M.D.[2]

[1] Kostroma Regional Clinical Hospital named after Korolev E.I., Kostroma, Russia

[2] Republican Clinical Hospital of the Ministry of Health of the Republic of North Ossetia-Alania, Vladikavkaz, Russia

[3] National Medical and Surgical Center named after N.I. Pirogov, Moscow, Russia

The frequency of atrial infarction in patients with supraventricular arrhythmias

Aim	To evaluate the incidence of atrial infarction (AI) based on a retrospective review of 287 case reports of patients with supraventricular arrhythmia and a positive qualitative test for troponin I after pharmacological arrest of arrhythmia; to determine the target localization of lesions and diagnostic signs, that appear in acute ischemic atrial damage, by selective coronary angiography (CA).
Material and methods	A retrospective review was performed of 287 case reports of patients admitted to cardiology departments for atrial fibrillation paroxysm with narrow QRS complexes on electrocardiogram (ECG) from 2018 through 2020. At the prehospital stage, verapamil had been administered intravenously with no effect. In the hospital, the sinus rhythm was successfully restored pharmacologically in all patients. Then ECG, repeated qualitative determination of troponin I, echocardiography, and CA were performed.
Results	77 (27%) patients of the study group had AI signs; 27 (9.5%) of these patients had confirmed AI, and 50 (17.5%) patients had probable AI. The existence of acute ischemic injury was considered absolutely confirmed in the presence of a combination of ECG changes, positive markers of myocardial damage, and reduced blood flow velocity in the left atrial branch of the sinoatrial nodal artery as shown by CA; in the presence of only ECG and biochemical criteria, acute AI was considered probable. According to selective CA, coronary injuries requiring an intervention were absent, and signs of the above-mentioned artery thrombosis were not visualized. However, the blood flow velocity was reduced to the TIMI II level in 9.5% of cases; other atrial branches had an extremely small diameter.
Conclusion	Atrial infarction needs to be excluded for patients with supraventricular arrhythmias, a characteristic clinical picture, and increased levels of myocardial injury enzymes.

Keywords	Atrial infarction; arrhythmia; coronary angiography; diagnostic criteria of atrial infarction
For citations	Bocharov A.V., Popov L.V., Lagkuev M.D. The frequency of atrial infarction in patients with supraventricular arrhythmias. Kardiologiia. 2022;62(3):28–31. [Russian: Бочаров А.В., Попов Л.В., Лагкуев М.Д. Частота возникновения инфаркта предсердий у больных с суправентрикулярными нарушениями ритма сердца. Кардиология. 2022;62(3):28–31]

Corresponding author	Bocharov A.V. E-mail: bocharovav@mail.ru

Introduction

A Vascular atherosclerosis is the main cause of threatening cardiovascular diseases, such as acute myocardial infarction (MI), cerebrovascular accident, critical limb ischemia. Among heart diseases, acute MI is the dominating factor of thanatogenesis [1, 2].

The current (fourth) Universal Definition of MI, Guidelines for the diagnosis and management of acute coronary syndrome, Guidelines on myocardial revascularization automatically imply only ventricular myocardial damage [3]. There are few studies devoted to acute atrial ischemic injury [4–6].

Physicians often miss ischemic changes in the atria in clinical practice because they pay attention to the configuration of the QRS complex and the ST segment of the electrocardiogram (ECG). The cause is also the lack of criteria for atrial infarction (AI) and the blurred clinical picture.

Unfortunately, the incidence of AI is not known. However, the scarce literature suggests, based on necropsy data, that AI occurs in 0.17–42% patients with ventricular infarction [6]. There are single descriptions of clinical cases of isolated AI in the literature [7].

Since atrial walls are thinner than ventricular walls, transmural AI represents the absolute majority of cases. Because of different blood supply of the myocardium of the left and right atria, the latter is injured more often [7].

Coronary atherosclerosis, extensive ventricular MI involving the atria, chronic pulmonary heart disease, severe pulmonary hypertension, Friedreich's ataxia, etc., are the most common causes of atrial ischemic injury [4].

The clinical manifestations of AI are the following symptoms individually or in combination: heart rhythm disorders, thromboembolism, an increase in the heart failure functional class due to atrial dilation and loss of atrial wall motion, atrial rupture [4].

ISSN 0022-9040. Kardiologiia. 2022;62(3). DOI: 10.18087/cardio.2022.3.n1648

Heart rhythm disorders in AI are present in about 70% of cases. Supraventricular arrhythmias are most common, but other types of rhythm disorders are also possible, such as atrial premature beats, sinus tachycardia or bradycardia, atrial fibrillation (AF) or atrial flutter [6, 8].

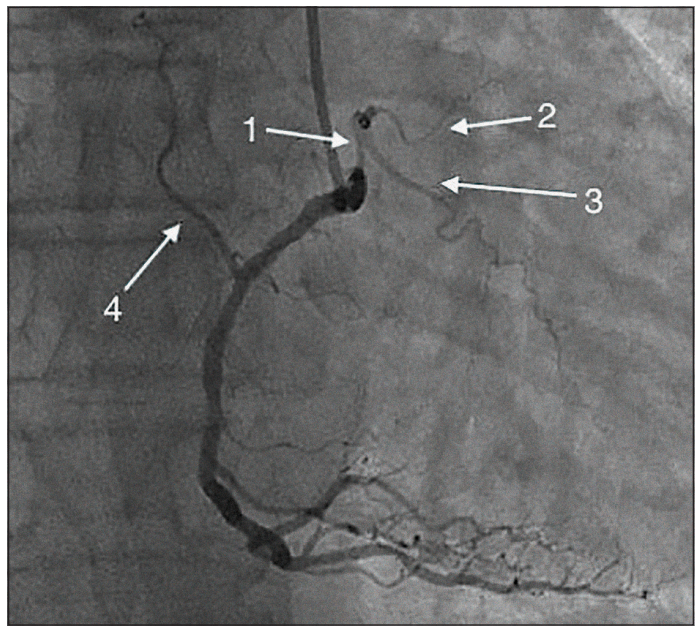
ECG is the main clinical diagnosis method of atrial ischemic injury. The electrical activity of the atria is shown on the ECG by the P wave and the PQ segment [6, 8, 9]. The following electrocardiographic diagnostic criteria are used for AI [8, 10]: PQ segment elevation >0.5 mm in standard lead I with reciprocal PQ segment depression in standard leads II, III; PQ segment elevation >0.5 mm in leads V5, V6 with reciprocal PQ segment depression in leads V1 and V2; PQ segment depression >1.2 mm in standard leads I, II, III, and any supraventricular arrhythmia; PQ segment depression >1.2 mm in leads II, III, and aVF; PQ segment elevation >0.5 mm in leads aVR and V1; PQ segment duration more than 200 ms.

It should be noted that the TP segment is taken as an isoline on the ECG.

Anatomically, atria are supplied from atrial branches of the right and left coronary arteries. The largest is the left atrial branch of the sinoatrial nodal artery. Usually, this branch arises from the right coronary artery near the right conus artery mouth, but it can arise from the circumflex branch of the left coronary artery in almost every third case [11–13].

Selective coronary angiography (CAG) requires tight-bolus contrast-enhancement of the coronary arteries, since selective catheterization and contrast-enhancement of the sinus branch and, thus, the left atrial branch, poses great risk of ventricular fibrillation (Figure 1).

Figure 1. Selective coronary angiography of the right coronary artery



1, 2 – sinoatrial nodal branch; 3 – left atrial branch; 4 – conus branch.

Materials and Methods

The incidence of AI was assessed in the retrospective analysis of 287 case records of patients emergently hospitalized to the cardiology departments of the Kostroma Regional Clinical Hospital named after E.I. Korolev with a diagnosis of paroxysmal AF and narrow QRS complexes from 2018 to 2020. Intravenous verapamil was administered before the hospitalization without any effect.

Inclusion criteria: age from 40 to 65 years, sinus rhythm restored with drugs after an episode of paroxysmal AF, positive qualitative tests for troponin I, no hemodynamically significant coronary artery disease according to CAG. Exclusion criteria: left ventricular ejection fraction less than 30%, chronic renal failure, cancer, hematological diseases, and other diseases that reduce life expectancy.

This retrospective study was approved by the local ethics committee.

All patients complained at admission of air hunger, cardiac disorders, and heart discomfort within the previous 12 hours.

A standard examination was performed at admission: ECG (Figure 2), clinical and biochemical blood tests, qualitative troponin I test.

Clinical characteristics of patients are presented in Table 1.

Sinus rhythm was successfully restored with drugs in all hospitalized patients, after which electrocardiography was registered, qualitative troponin I test was repeated, echocardiography and CAG were conducted. CAG was indicated in the case of positive markers of myocardial injury and the presence of atrial ischemic changes in the ECG.

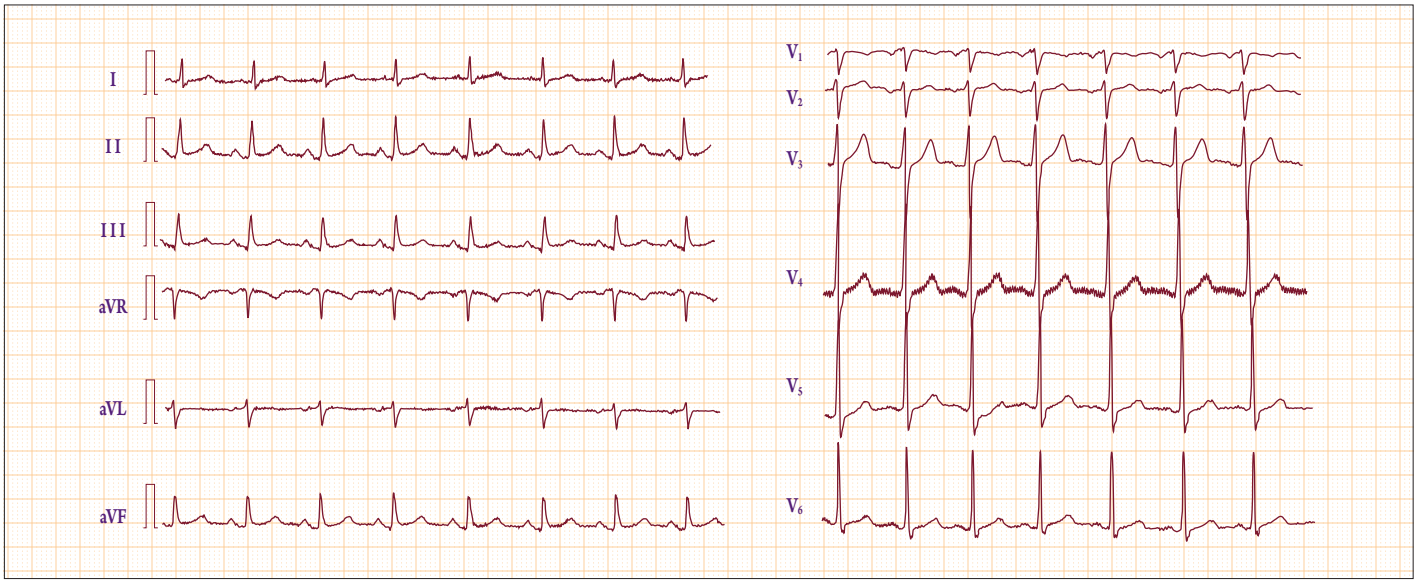
Statistical processing of the data obtained was carried out using Statistica 13.3 (TIBCO Software Inc., 2017, http://statistica.io). The results are described as the median and the interquartile range (25th and 75th percentile) with the asymmetric distribution. The distribution of quantitative variables was estimated using the Kolmogorov-Smirnov/Lilliefors test. Qualitative variables are presented as the absolute values and percentages per group (n (%)).

Results

All patients had normal clinical and biochemical blood test and urinalysis results. There were positive qualitative troponin I test results, and there were no new zones of impaired left ventricular wall motion.

Selective CAG showed neither coronary lesions requiring interventions, nor signs of coronary artery thrombosis. However, slow flow (TIMI II) was detected in many patients (Table 2), the other atrial branches had very small diameters.

Figure 2. Electrocardiogram of a patient with atrial infarction (25 mm/s, 10 mm/mV)



Diagnostically significant PQ segment depression in leads II, III, aVF. Diagnostically significant PT segment elevation in leads aVR and V1.

Acute atrial ischemic injury was confirmed if a patient had a combination of changes in the ECG according to the above criteria, positive markers of myocardial damage, and slow flow in the left atrial branch of the sinoatrial nodal artery shown by CAG; acute AI was considered potential in the presence of only ECG and biochemical criteria (see Table 2).

Thus, 77 (27%) patients of the study group (n=287) had signs of AI, of whom 27 (9.5%) patients had confirmed AI and 50 (17.5%) patients had potential AI.

Table 1. Characteristics of patients examined (n=287)

Parameter	Value
Male, n (%)	192 (67)
Age, years	57.2 [52; 61]
Body mass index, kg/m2	27.6 [24; 36]
Smoking, n (%)	174 (61)
Alcohol misuse, n (%)	14 (5)
Arterial hypertension, n (%)	264 (92)
Chronic bronchitis, n (%)	34 (12)
History of cerebrovascular accident, n (%)	6 (2)
History of acute myocardial infarction, n (%)	11 (4)
Diabetes mellitus, n (%)	10 (3.5)
Continuous antihypertensive therapy (angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, diuretics), n (%)	130 (45)
History of documented heart rhythm disorders, n (%)	66 (23)
History of coronary artery disease, n (%)	234 (82)
Left ventricular ejection fraction (Simpson) after rythm normalization, %	46 [42; 48]

Discussion

AI is a very understudied cardiac pathology. There are very few works on this issue. For example, there are no data on the incidence of acute atrial ischemic injury, and the available data on the incidence of AI in patients with left ventricular MI vary greatly from 0.17% to 42% [6]. ECG changes characteristic of this pathology are often neglected by cardiologists despite the existing criteria [8, 10]. Transthoracic cardiac ultrasound provides almost no information in acute atrial ischemic injury, although there is the characteristic transesophageal echocardiographic picture [14]. We did not find any papers on the atrial blood supply and the relevant changes in AI in the available literature. Based on results of the analysis of selective CAG data in patients with ECG signs of AI and biochemical signs of myocardial damage, we concluded that only slow flow in the left atrial branch of the sinoatrial nodal artery is the main criterion for angiographic diagnosis of this pathology. The specificity and sensitivity of this feature are to be clarified.

Table 2. Incidence of acute atrial ischemic injury in the study group (n=287)

Parameter	Value
Electrocardiographic criteria for atrial infarction, n (%)	77 (27)
Biochemical markers of myocardial damage after rhythm recovery, n (%)	287 (100)
Slow flow to TIMI II in the left atrial branch of the sinoatrial nodal artery according to coronary angiography, n (%)	38 (13)
Atrial infarction, n (%)	77 (27)
• verified	27 (9.5)
• probable	50 (17.5)

ISSN 0022-9040. Kardiologiia. 2022;62(3). DOI: 10.18087/cardio.2022.3.n1648

Attention is drawn to the relatively high incidence of potential or confirmed acute atrial ischemic damage (17.5% and 9.5%, respectively; the total of 27% of the sample) in patients with supraventricular heart rhythm disorders and positive test for the markers of myocardial damage (troponin I). That issue needs to be studied further.

Conclusion

Atrial infarction should be ruled out in patients with supraventricular arrhythmias, typical clinical picture, and elevated levels of myocardial injury enzymes.

No conflict of interest is reported.

The article was received on 06/04/2021

REFERENCES

1. Yeh RW, Sidney S, Chandra M, Sorel M, Selby JV, Go AS. Population Trends in the Incidence and Outcomes of Acute Myocardial Infarction. New England Journal of Medicine. 2010;362(23):2155–65. DOI: 10.1056/NEJMoa0908610
2. Chang M, Lee CW, Ahn J-M, Cavalcante R, Sotomi Y, Onuma Y et al. Predictors of long-term outcomes after bypass grafting versus drug-eluting stent implantation for left main or multivessel coronary artery disease. Catheterization and Cardiovascular Interventions. 2017;90(2):177–85. DOI: 10.1002/ccd.26927
3. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA et al. Fourth universal definition of myocardial infarction (2018). European Heart Journal. 2019;40(3):237–69. DOI: 10.1093/eurheartj/ehy462
4. Gardin JM, Singer DH. Atrial infarction. Importance, diagnosis, and localization. Archives of Internal Medicine. 1981;141(10):1345–8. DOI: 10.1001/archinte.141.10.1345
5. Lazar EJ, Goldberger J, Peled H, Sherman M, Frishman WH. Atrial infarction: Diagnosis and management. American Heart Journal. 1988;116(4):1058–63. DOI: 10.1016/0002-8703(88)90160-3
6. Lu MLR, De Venecia T, Patnaik S, Figueredo VM. Atrial myocardial infarction: A tale of the forgotten chamber. International Journal of Cardiology. 2016;202:904–9. DOI: 10.1016/j.ijcard.2015.10.070
7. Cunningham KS, Chan KL, Veinot JP. Pathology of isolated atrial infarction: case report and review of the literature. Cardiovascular Pathology. 2008;17(3):183–5. DOI: 10.1016/j.carpath.2007.05.002
8. Liu CK, Greenspan G, Piccirillo RT. Atrial Infarction of the Heart. Circulation. 1961;23(3):331–8. DOI: 10.1161/01.CIR.23.3.331
9. Van Diepen S, Siha H, Fu Y, Westerhout CM, Lopes RD, Granger CB et al. Do baseline atrial electrocardiographic and infarction patterns predict new-onset atrial fibrillation after ST-elevation myocardial infarction? Insights from the Assessment of Pexelizumab in Acute Myocardial Infarction Trial. Journal of Electrocardiology. 2010;43(4):351–8. DOI: 10.1016/j.jelectrocard.2010.04.001
10. Sivertssen E, Hoel B, Bay G, Jörgensen L. Electrocardiographic atrial complex and acute atrial myocardial infarction. The American Journal of Cardiology. 1973;31(4):450–6. DOI: 10.1016/0002-9149(73)90293-2
11. Anderson RH, Becker AE, Allwork SP, Meun WP, Verhoeven RE. Cardiac anatomy: an integrated text and colour atlas. -London: Edinburgh; New York: Gower Medical Pub.; Churchill Livingston;1980. - 248 p. ISBN 978-0-906923-00-9
12. Heart disease: a textbook of cardiovascular medicine. (Gensini GG. Coronary arteriography. P.374-400). Braunwald E, editor -Philadelphia: Saunders;1984. - 1844 p. ISBN 978-0-7216-1938-5
13. Kyriakidis MK, Kourouklis CB, Papaioannou JT, Christakos SG, Spanos GP, Avgoustakis DG. Sinus node coronary arteries studied with angiography. The American Journal of Cardiology. 1983;51(5):749–50. DOI: 10.1016/S0002-9149(83)80126-X
14. Vargas-Barrón J, López-Meneses M, Roldán F-J, Romero-Cardenas Án, Keirns C, Espinola-Zavaleta N et al. The impact of right atrial ischemia on inferior myocardial infarction with extension to right ventricle: Transesophageal echocardiographic examination. Clinical Cardiology. 2002;25(4):181–6. DOI: 10.1002/clc.4960250409