diagnostic criteria, additional investigation techniques and determine target therapies for each patient, in order to improve their clinical outcome.
**Key words:** MINOCA, coronary disorders, cardiovascular disease

## 237. THYROTOXIC CARDIOMYOPATHY: A CASE REPORT

Author: **Svetlana Diuvenji**
Co-authors: Ina Cazacliu, Elena Samohvalov, Irina Benesco
Scientific adviser: Alexandra Grejdieru, MD, PhD, Associate professor, Department of Internal Medicine, Cardiology, *Nicolae Testemitanu* State University of Medicine and Pharmacy, Chisinau, Republic of Moldova; Romeo Grajdieru, MD, PhD, Associate professor, Department of Internal Medicine, Cardiology, *Nicolae Testemitanu* State University of Medicine and Pharmacy, Chisinau, Republic of Moldova.

**Background.** Heart failure (HF) is the final common pathway of many cardiovascular diseases. It imposes significant socio-economic and health care burden to both patients and healthcare systems. Although the most common cause of HF is ischemic heart diseases, other less common causes such as hyperthyroidism (thyrotoxicosis), severe anemia, arrhythmia should also be considered during diagnosis to improve overall clinical management of HF.
**Case report.** The 42-year-old man was admitted to cardiology department with mixed (inspiratory and expiratory) dyspnea at moderate effort, palpitations, fatigue, the loss in weight of about 15 kg during 9-10 months. Anamnesis: general condition worsened the last 2 months when appeared generalized edema and mixed dyspnea. During this time did not address to doctor, any treatment has not received. Physical examination revealed swelling in the legs, ankles, ascites, an irregular pulse, at a rate of 130 beats/min, BP- 110/70mmHg. On ECG - atrial fibrillation with rate - 120-57 b/min, electric axis of heart is normal. Signs of left ventricular hypertrophy. The chest X-ray -pulmonary congestion, bilateral pleural effusion. The abdominal X-ray – fluid levels with air on the left. On TTE- thickening of the walls of the aorta and valve apparatus. Dilatation of all heart chambers, significant dilatation of the right atrium and right ventricle, and moderate dilatation of the left atrium and the left ventricle. Contractile function of the left ventricular myocardium is moderately reduced. Ejection fraction = 42%. The second degree mitral regurgitation and third-fourth -degree tricuspid regurgitation. Moderate pulmonary arterial hypertension (PASP= 52mmHg). Sheets of the pericardium are thickened. Fluid in the pleural cavity up to 11 millimeters in the region of the right atrium. Bilateral pleurisy - inhomogeneous fluid with floating elements on the left - about 1,000 milliliters, to the right - about 800 milliliters. Cytological analysis of fluid from pleural cavity pointed to the inflammatory etiology of the effusion. On the ultrasound examination of the thyroid gland – fourth –degree hyperplasia, multiple diffuse changes.On the ultrasound examination of abdominal cavity - ascites, bilateral pleuritic, diffuse changes in the parenchyma of the liver. The glycemic profile -7-00: 4.7 mmol/l, 13-00: 6.3 mmol/l, 17-00: 10.6 mmol/l, glycated hemoglobin - 5,6%. Analysis of thyroid hormones- free Triiodothyronine – 17,22 Pmol/l, free Thyroxine – 79,52 Pmol/l. TSH – ‹ 0, 05 uIU/ml; anti TPO- 144 IU/ml. Tumor marker CA 19-9 - <3.0 U/ml. During hospitalization was consulted by endocrinologist, surgeon. After 11 days of complex treatment with diuretics, anticoagulants, beta-adrenoblockers, antithyroid drugs, cardiac glycosides, corticosteroids, histamine-2-receptor blockers - the general condition improved: dyspnea and general swelling disappeared, general weakness was reduced.

**Conclusions.** The incidence and prevalence of thyrotoxic heart failure (THF) provide a wide variation from 12% to 68% in hyperthyroid patients. Up to 90% of patients with thyrotoxicosis may develop Atrial Fibrillation, 47% Left Ventricle systolic dysfunction and 1% dilated THF and a third of these cases are reversible. Mortality in THF patients is 1.2 higher than in patients with hypertension, valvular heart disease or coronary artery disease, and 1.4 higher than in the general population. Hyperthyroidism is a potentially reversible and curable cause of THF, so it should be excluded in every new patient with HF, especially in young patients and in the absence of coronary artery disease and other structural heart diseases.
**Key words:** thyrotoxic cardiomyopathy, heart failure.

## 238. ATRIAL FIBRILATION IN BRUGADA SYNDROM

Author: **Ina Cazacliu**
Co-authors: Diuvenji Svetlana, Elena Samohvalov, Irina Benesco,
Scientific adviser: Alexandra Grejdieru, MD, PhD, Associate professor, Department of Internal Medicine, Cardiology, *Nicolae Testemitanu* State University of Medicine and Pharmacy, Chisinau, Republic of Moldova; Romeo Grajdieru, MD, PhD, Associate professor, Department of Internal Medicine, Cardiology, *Nicolae Testemitanu* State University of Medicine and Pharmacy, Chisinau, Republic of Moldova.

**Introduction.** Atrial fibrilation is the most common cardiac arrhythmia with the worldwide prevalence of more than 33.5 million people and is a subject with increased interest in clinical trials. The reason is the awareness of the high risk of embolic events that in 75 % are complicated by cerebrovascular accidents. It is estimated that the number of patients with AF in 2030 in Europe will be 14–17 million and the number of new cases of AF per year at 120,000–215,000. In approximately 80% of patients, atrial fibrillation is associated with organic heart disease including valvular heart disease (mostly mitral valve disease), coronary artery disease, hypertension, hypertrophic or dilated cardiomyopathy. In 20% of cases, atrial fibrillation occurs in the absence of organic heart disease. Besides the danger of embolic events, atrial fibrillation is the most common atrial arrhythmia found in Brugada syndrome which is associated with malignant ventricular arrhythmias and sudden cardiac death.
**Aim of the study.** The purpose of this study was to review data about characteristics and management of atrial fibrillation in Brugade syndrome.
**Materials and methods.** The source of information was represented by articles published in the online databases: PubMed, HINARI, SCOPUS, EMBASE
**Results.** Current evidence revealed that the prevalence of AF in patients in BrS vastly differs among publish studies, ranged from 6% to 39%. The only genetic mechanism of arrhythmias is related to the mutation of the SCN5A gene that encodes cardiac sodium channels. However, as this sodium channel is found not only in the ventricular tissue, but also in the atria, this could lead to reentrant tachyarrhythmias in the atrium. Nevertheless, management of BrS with AF remains a difficult task, as medication for AF, such as sodium channel blockers, confers their risk owing to their proarrhythmic effects in patients with BrS. In addition, other than quinidine and disopyramide cannot be used because they block sodium channels and cause ventricular arrhythmias. Recent evidence suggested that catheter ablation could be utilized as a first-line therapy for paroxysmal AF in BrS patients. For the last 2 decades, ICD therapy has been considered as the cornerstone therapy of patients with documented ventricular tachyarrhythmia, but recent studies has been associated ICD therapy with a significant rate of