

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

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Introduction. Atrial fibrillation 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

complications, and should be avoided in asymptomatic patients. The most common of these complications are inappropriate shocks, which cause pain, and can produce psychological trauma. Pulmonary vein isolation (PVI) is an effective method for controlling paroxysmal AF. The literature indicates that the success rate of PVI is 79.8% in the long term in patients with brugada syndrome.

Conclusions. According to studies, PVI has been shown to have minimal risk of complications and is considered one of the most effective long-term methods in the control of atrial fibrillation and brugada syndrome. This treatment method could be considered the first line of treatment for atrial fibrillation and in brugada syndrome.

Key words: atrial fibrillation, brugada syndrome, sudden cardiac death, implantable cardioverter-defibrillators, catheter ablation.

239. WELLENS` SYNDROME IN AN ELDERLY PATIENT

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Background. Wellens' syndrome consists of particular T-wave changes in the precordial leads on ECG accompanied by severe proximal left anterior descending artery stenosis, and is often associated with sudden cardiac death and acute myocardial infarction. It is a pre-infarction state. However, this syndrome is not always an acute process. There are two ECG patterns of Wellens syndrome. Type-A: up sloping ST waves, no or mild ST elevation at the J point and biphasic T waves, with initial positivity and terminal negativity. These T wave findings are present in about 25% of cases. Type-B: symmetrical deeply inverted T waves, in approximately 75% of cases. Both types, R waves preserved in the precordial leads

Case report. A 65-year-old male patient, was admitted in the Intensive Care Unit of MCH "Holy Trinity" with Non-STE ACS. Complaining on angina: burning chest pain felt as well in the neck and lower jaw, occurring at mild exertion lasting for ≥ 40 min and relieved by i/v nitrates. Other complains: shortness of breath at mild exertion and fatigue. History: his condition worsened for about 5 days ago while being on a ski resort in Ukraine and felt for the first time angina chest pain lasting about 1h. He was admitted in the ICU of the Regional non-PCI hospital and acute MI diagnose was established, based on a troponin I test – 3,14ng/ml. Because of high costs of the medical care he left the hospital and came back to Moldova by car. During the long trip (5h) he felt several angina episodes, the longest lasting about 40min. ECG at admission: sinus rhythm, normal axis, HR = 76 bpm, up slopping ST segment in V2-V4, ST elevation at the J point max 0,5 mm in V3, biphasic T waves in V2-V4 initially positive than negative. Echography: no wall motion abnormality revealed, EF 58%. Serum troponin T – 0.21 ng/ml (0,3ng ml reference limit), CK-MB - 17 U/l (reference limit 24 U/l). Coronary angiography: two-vessel disease, sub occlusive stenosis of proximal LAD (99%), severe on RCA (75-90%). PCI of the culprit lesion with one DES of new generation was performed successfully and the second PCI on RCA scheduled in two weeks (aiming complete revascularization). ECG on the second day following PCI showed no biphasic T-waves in the precordial leads. At 1 month after the complete revascularization, the patient has no symptoms even at intense exertion.