UNIVERSITÁ DEGLI STUDI "G. D'Annunzio" di Chieti - Pescara
DIPARTIMENTO DI NEUROSCIENZE, IMAGING E SCIENZE CLINICHE

AVVISO DI SEMINARIO

Relatore

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Arrhythmic mitral valve prolapse

Giovedì 30 settembre 2021
ore 16:00
Aula Galileo

Modalità residenziale
c/o Palazzina ITAB - I livello
Via Luigi Polacchi, 11
66013 – Chieti Scalo

Modalità webinar
Microsoft Teams - https://bit.ly/3i9rOkg

Abstract: There is an increasing awareness of the association between mitral valve prolapse (MVP) and sudden cardiac death (SCD). Over the last few years, however, we have learnt that MVPs are not all the same, and that the 'arrhythmic MVP' is a peculiar clinical entity, characterised by specific mitral valve apparatus abnormalities, such as myxomatous, redundant and prolapsing leaflets, mitral annular disjunction (MAD), replacement fibrotic changes in papillary muscles and basal myocardium. Furthermore, high electrical instability puts patients, especially young women, at risk of life-threatening VAs and SCD. The pathophysiological link between these features is far from being elucidated, although the Padua group recently proposed a causal relationship between the morpho-functional abnormalities of the mitral annulus (MAD and systolic curling motion), the myxomatous degeneration of the valve, the development of myocardial substrates (left ventricular (LV) hypertrophy and replacement-type fibrosis) and the genesis of malignant VAs. More in detail, the altered geometry and mechanics of the mitral annulus may account for a repetitive mechanical stretch to the valve and LV myocardium (including papillary muscles), progressively leading to myxomatous degeneration and arrhythmogenic myocardial scars, which characterize the arrhythmic (or malignant) MVP phenotype. There are several clinical risk factors associated with an increased risk of MVP-related SCD, most of which can be evaluated with noninvasive diagnostic modalities. For example, characteristic changes on the electrocardiogram (T-wave inversions in the inferior leads), complex ventricular ectopy, spiked configuration of the lateral annular velocities by echocardiography, and evidence of myocardial fibrosis by cardiac magnetic resonance imaging have all been implicated as markers of risk. Epidemiology of SCD in the MVP population, the clinical profile of at-risk patients, and the basic components necessary to initiate and perpetuate ventricular arrhythmias (substrate and trigger), potential interventions to consider for those at highest risk of SCD and novel areas of research will be discussed.