portadores del alelo E2 tienen un menor riesgo de CI, mientras que los portadores del alelo E4 tienen un exceso de riesgo [alelo ApoE2 = rs429358(T) + rs7412(T); alelo ApoE3 = rs429358(T) + rs7412(C); alelo ApoE4 = rs429358(C) + rs7412(C)][2].

El papel de los genes en la CI no se limita solo a polimorfismos de base única y a las mutaciones, también se producen cambios epigenéticos como demostraron Guay et al.[12] quienes encontraron que la metilación del ADN del promotor del gen ABCA1 se asocia con CI en pacientes con hipercolesterolemia familiar.

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http://dx.doi.org/10.1016/j.acmx.2013.08.004

Hipertensión arterial pulmonar asociada a virus de la inmunodeficiencia humana. El tópico olvidado

Pulmonary arterial hypertension associated with human immunodeficiency virus. The forgotten topic

Sr. Editor:

La hipertensión arterial pulmonar ha sido motivo de múltiples investigaciones[1].

Durante el quinto congreso mundial de Hipertensión Arterial Pulmonar, realizado en Niza, Francia (http://www,wsph2013.com/pdf/final_program.pdf) se programaron varias conferencias donde se trataba de establecer la nueva clasificación diagnóstica y terapéutica sobre este tópico.

Es de señalar que en ninguna de las mesas redondas ni en los cientos de resúmenes de trabajos libres se consideró la hipertensión arterial pulmonar asociada al virus de la inmunodeficiencia humana como un tema a tratar.

La hipertensión arterial pulmonar asociada al virus de la inmunodeficiencia humana es una de las complicaciones no infecciosas del virus de la inmunodeficiencia humana y causa deterioro y muerte en cientos de pacientes; se ha estimado que un 0.5% de los afectados por este virus pueden padecerla[2] pero en poblaciones con sintomatología cardiovascular llega hasta a un 5%[3].

Aun existe vacío en la fisiopatogenia de la lesión endotelial pulmonar por este virus[4], pero si el congreso con más relevancia en este campo a nivel mundial no lo incluye dentro de su programa, varios grupos no considerarán la relevancia de la investigación de este tópico en el futuro.

Es importante que los especialistas en enfermedades respiratorias, cardiacas y en conjunto con los infectólogos propongan en los congresos venideros la revisión de este tema, para la promoción científica y el bienestar de nuestros pacientes.

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http://dx.doi.org/10.1016/j.acmx.2013.12.001

Substrate-based strategies for paroxysmal refractory VT catheter ablation: Coming of age?

Técnicas basadas en el sustrato para la ablación con catéter de la TV paroxística refractaria: ¿están lo suficientemente maduras?

Dear Editor,

Patients with ischemic and nonischemic cardiomyopathy are at increased risk of sudden death due to ventricular arrhythmias. Implantable cardioverter-defibrillators (ICD) have lowered this mortality rate; however, ICD shocks impact quality of life.[1] Medical treatment with the combination of betablockers with amiodarone reduces ventricular tachycardia (VT) inducibility[2] and ICD shocks,[3] but long-term side effects are unfortunately relatively frequent. In patients with sustained paroxysmal refractory VT, catheter ablation has been proven to reduce VT recurrence by directly addressing the arrhythmogenic substrate, albeit at a procedure-dependent risk; nevertheless, ablation has no effect on mortality.[4]

Catheter mapping of recurrent VT relies on arrhythmia induction and identification of ablation targets (isthmuses and exit sites) by entrainment and activation criteria during ongoing VT. Ideally, it provides an endpoint to the procedure in the case of non-inducibility of VT. However, this approach is somewhat difficult due to some potential limitations: non-inducibility of VT, VT pleomorphism due to complex substrates, hemodynamic compromise. Therefore, these limitations have driven the development of substrate-based mapping and new ablation strategies.

In patients with structural heart disease, ventricular scar represents the anatomical (and electrophysiological) basis for VT initiation and its maintenance. Abnormal local electrograms in sinus rhythm (SR) have been identified in border zones surrounding the scar. Surgical resection of subendocardial and/or epicardial reentry circuits was first attempted as an ablative method.

In the February 2013 issue of this Journal, Vergara et al. published a review of current substrate-based strategies for VT catheter ablation.[5] They comprehensively describe several approaches to substrate ablation. The first two methods reported, conceptually derived from surgical ablation, employ linear ablation lines to cross, encircle or link the scar and anatomical barriers. Arenal et al.[6] elegantly identified channels of delayed conduction through the scar zone through a step-wise reduction in voltage thresholds, targeting these areas with relatively few applications. However, the absence of a clear end point can limit its efficacy. The same technique was used by Berruezo et al.[7] in a series of arrhythmogenic right ventricular dysplasia patients. Alternatively, Vergara et al.[8] focused on complete mapping and ablation of late potentials (LPs), combined with pace-mapping in SR and, when possible, entrainment maneuvers. LPs ablation was associated with low VT recurrence rates and was a good predictor of arrhythmia-free survival. Thus, the authors postulated that ''complete'' LPs elimination is superior to non-inducibility of VT, given its ability to simultaneously address multiple circuits.

In spite of their promising results, several limitations remain. The population of these studies is heterogeneous as to VT induction and hemodynamic stability, thereby providing a possible bias. While the procedural endpoint has mostly been substrate-based, Soejima et al.[9] found that non-identification of an isthmus was associated with recurrence. Also, different bipolar voltage cut-offs were used. Lastly, the ablation strategies have not been directly compared.

Despite these limitations, the current review by Vergara et al. suggests that substrate mapping and ablation strategies could be a valuable addition to the field of chronic recurrent VT catheter ablation, hopefully translating into a better clinical benefit for our patients.

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