

genetic study detected a novel frame-shift mutation in the sodium channel alpha subunit gene *SCN5A* (D1816fs), which results in early transcriptional arrest and produces a truncated or shortened protein.

The major mechanism of sudden cardiac death is VF and the most common cause is ischemic heart disease. Ventricular fibrillation is considered to be idiopathic when there is no evidence of structural heart disease, cardiotoxicity, electrolyte disorders, or a predisposing hereditary condition.¹

Although PVC is a benign arrhythmia, there are cases in which PVC originating in the Purkinje system or in the RV outflow tract initiates VF episodes in individuals without heart disease, with structural heart disease, or with channel defects. The absence of heart disease and the early development of PVC fit well with the entity described by Leenhardt et al.³ Haïssaguerre et al reported the efficacy of radiofrequency ablation in PVC in the prevention of new episodes of VF over the short and long terms.²

This case is singular for 2 reasons. On one hand, it highlights the efficacy of ablation in this desperate situation and the difficulty derived from the mechanical suppression of the focus via catheter. On the other hand, it describes a novel mutation in the *SCN5A* gene as a cause of idiopathic VF with a normal electrocardiogram. Thus, it involves an occult channel defect of which VF was the first sign. The incomplete penetration of channel defects explains the fact that individuals with FV who have an apparently normal heart and in whom there is no electrocardiographic evidence of this condition exhibit electrical

instability in relation to a genetically determined occult arrhythmogenic disease.¹

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Available online 17 April 2011

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doi:10.1016/j.rec.2010.11.023

Does the Metabolic Syndrome Need More Descriptive Studies or More Evidence of its Implication in Secondary Prevention?

¿El síndrome metabólico en España necesita más estudios descriptivos o más evidencia de su implicación en prevención secundaria?

To the Editor,

We have read with interest the article "Connection Between the Early Phases of Kidney Disease and the Metabolic Syndrome" recently published in *Revista Española de Cardiología*. The authors describe the association of the metabolic syndrome (MS) and early kidney disease (EKD) with carotid intima-media thickening (CIMT).¹ Interestingly, although they assessed the relationship between a group of risk factors and the presence of subclinical and early vascular lesions, they did not discuss its actual implications regarding prevention or the treatment of these patients. In fact, it remains to be clarified whether these lesions should be considered to be the 2 diagnostic criteria of the MS; this has already been proposed for C-reactive protein, which, besides presenting higher levels in subjects with the MS, improves risk prediction of cardiovascular disease.²

In addition, EKD and CIMT are 2 disorders that can be stabilized but are only resolved with difficulty, especially when the values are close to normal; the mean glomerular filtration rate was 87 mL/min/1.73 m² and CIMT ranged between 0.6 mm and 0.7 mm. Moreover, clinical trials that have investigated CIMT regression or changes in EKD have not found a correlation with improved cardiovascular prognosis^{3,4} suggesting that they behave as markers of vascular injury rather than as risk factors. In contrast, studies based on the MESYAS (Metabolic Syndrome in Active Subjects) registry have shown that the lipid components of the MS,

as measured by the ratio of triglyceride to high-density lipoprotein (HDL), are very specific markers for the presence of other components of the MS⁵ and, more importantly, one of the major risk factors for myocardial infarction.⁶ This association has also been described both for the presence of the MS and for the additive effect of its components.⁷

Finally, the authors considered high blood pressure and diabetes as exclusion criteria in the study,¹ when it has been reported that these are 2 of the main factors associated with the presence of the MS in the Spanish workforce.⁸ Furthermore, they state that they are unaware of the existence of previous data that associate the MS with EKD. Nevertheless, in 2004 and 2005, respectively, data from the NHANES III (National Adult Health Examination Survey) registry⁹ and the MESYAS registry¹⁰ were published regarding this association and coincided regarding the lack of an association between HDL and kidney damage.

We believe that the authors describe an association with few implications for cardiovascular risk stratification, while the actual implication of the MS in patients with established cardiovascular disease remains to be elucidated. During the last decade, the MS has given rise to great interest due to its relevance in the prevention of diabetes mellitus and cardiovascular disease; however, its relevance in patients with established cardiovascular disease remains undefined and unaccepted.

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Available online 11 August 2011

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doi:10.1016/j.rec.2011.05.030

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Does the Metabolic Syndrome Need More Descriptive Studies or More Evidence of Its Involvement in Secondary Prevention? Response

¿El síndrome metabólico en España necesita más estudios descriptivos o más evidencia de su implicación en prevención secundaria? Respuesta

To the Editor,

As Cordero et al. state in the current issue of *Revista Española de Cardiología*, our research group describes the association between the cluster of vascular risk factors known as the metabolic syndrome (MS)¹ and the presence of subclinical and early vascular lesions that, once established, hardly ever return to normal.

The importance of this relationship lies in the fact that we know that MS doubles the risk of cardiovascular (CV) disease and triples the risk of CV death²; that it significantly increases the risk of advanced kidney disease³; and that patients with advanced kidney disease have an increased risk of death that can be almost 6-fold greater than that of patients with normal clearing.⁴ In fact, when signs of kidney disease appear, the structural lesion is already established and, as prevention is considered the best strategy in managing these patients, it is crucial to detect the earliest abnormalities so as to design specific interventions.⁵ Our findings confirm that MS is significantly associated with incipient deterioration of kidney function and increased intima-media thickness, but we cannot conclude these markers should be included in MS diagnostic criteria.

We coincide with other authors⁶ in our belief that management of these patients requires that we perform studies aimed at clarifying this marker's possible role in prognosis and in determining the most effective treatment.

We are fully aware of NHANES registry data for 2004³ and cite the aforementioned study.¹ Its authors evaluate advanced stages of kidney disease and the presence of microalbuminuria independently of the glomerular filtration rate. Therefore, in our opinion,

our results complement and confirm these findings. We did not consider the 2005 MESYAS registry⁷ because it took account of the glomerular filtration rate in isolation, whereas our study looked at this in combination with the presence of microalbuminuria, in accordance with current American Society of Nephrology recommendations.⁸

Finally, we wish to make it clear that given diabetes and high blood pressure are well-known factors contributing to kidney damage, we excluded patients presenting these in order to study the real value of MS in evaluating subclinical CV disease.

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Available online 30 July 2011

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