Letters to the Editor

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Association between the metabolic syndrome and parental history of premature cardiovascular disease

The recent study by Dallongeville et al.1 on 390 men and 281 women with the metabolic syndrome (MS) according to the NCEP III criteria clearly showed that parental premature cardiovascular disease was associated with their offspring’s MS, particularly in women. Herewith we present additional data in proof.

In a study of 55 persons with premature coronary heart disease (i.e. documented myocardial infarction before their age of 45; 39.9 ± 4.2 years), all from Split, Croatia,2 similar to the results of Dallongeville et al.,1 we have observed significant differences in terms of MS and other cardiovascular risk factors between these ‘risky’ children and their controls, as shown in the table.

Unfortunately, we did not take their waist circumference or waist-to-hip ratio and we did not measure their triglycerides or HDL cholesterol. However, the observed differences became even more striking when out of these 97 ‘stigmatized’ children were assessed those 50 with at least one additional cardiovascular risk factor.2 For instance, arterial hypertension was present in 46.2% and smoking in 51.3% of such children.

The detected tracking and clustering phenomena1,3 underscore the association of MS and other contributors to familial aggregation of propensity to cardiovascular disease, stressing the importance of early detection for timely intervention, consisting mostly of dietary modifications and other lifestyle measures. It is not yet clear which proportion of the MS components is hereditary and which is environmentally and/or microsocially induced. The just launched, multinational INTER-HEART 2 study3 attempts to answer these questions.

References


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<th>Control children (n = 139)</th>
<th>Premature AMI offspring (n = 97)</th>
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<tr>
<td>Relative weight (%)</td>
<td>99.5 ± 11.2</td>
<td>103.8 ± 15.2**</td>
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<tr>
<td>Plasma cholesterol (mmol/L)</td>
<td>4.4 ± 0.6</td>
<td>5.2 ± 1.1**</td>
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<td>Systolic blood pressure (mmHg)</td>
<td>114.3 ± 8.5</td>
<td>116.6 ± 15.3**</td>
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<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>73.6 ± 7.3</td>
<td>74.3 ± 12.7**</td>
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*Body mass in kilogram as per cent of ideal age/sex weight. **p < 0.05 (two-tailed, paired t-test).

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Incidence of syncope after ICD implantation: low or high?

We read with great interest the article by Abello et al.1 reporting the incidence of syncope in implantable cardioverter recipients with spontaneous syncopal monomorphic ventricular tachycardia.

They make a distinction between patients who presented with syncopal ventricular tachycardia and those with non-syncopal ventricular tachycardia. In the former, eight out of 26 suffered syncope (11 episodes). Syncope presentation, therefore, appears to confer high risk of syncope after ICD implantation.

ICD was developed to protect patients from sudden death, but they also may help to control other symptoms such as syncope. Our previous work assessing the risk of syncope in ICD recipients2 in a population who presented with syncope showed strikingly different results, compared with the results from Abello et al.1 In our series, only three out of 37 patients had syncope recurrence and in none of them was it due to arrhythmias. To date, the largest report on the matter is that from Bansch et al.3 They found recurrence of syncope after ICD implantation in 14.7% of patients. Likewise, an older article by Kou et al.4 reports syncope in 13 of 180 patients.

How can we explain the results of Abello et al. in a particular subset of ICD recipients? First, their population corresponds to a selected group of patients with syncopal tachycardia. Secondly, ICDs can be programmed in a wide variety of ways. In our series, we found a high effectiveness of anti-tachycardia pacing and adjusted energy shocks after a short charging time. We think that this fact would explain the low incidence of syncope when compared with previous reports. The programming features in our patients are similar to those of Abello et al. with one exception. Abello et al. describe the use of “low energy shocks” (LEs) as a therapy in the VT zone aimed at suppressing organized rhythms. As a matter of fact, there is evidence of arrhythmia acceleration after such LES.5 Abello et al. describe the sequence of facts in seven episodes of syncope related to proarrhythmia, and in four out of those seven, LES was present in the sequence of therapies.

In conclusion, the true incidence of syncope in ICD recipients remains controversial. Reports on the incidence of syncope in large populations of ICD recipients are lacking. Previously known proarrhythmic

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therapies such as LESs should probably be avoided.

References

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Incidence of syncope after ICD implantation: low or high?: reply

We agree with them that our results were obtained from a selected implantable cardioverter-defibrillator (ICD) recipient population with syncopal spontaneous ventricular tachycardia (VT) and, therefore, they should not be extrapolated to the general population of patients who presented syncope before ICD implantation.1,4 Syncope can originate from many different mechanisms, which can be related or not to VT. This was discussed in our original manuscript and was also admitted in the report of Garcia Moran et al.,2 who suggested that some patients with non-arrhythmic syncope were enrolled in their study because none of the syncopal recurrences had an arrhythmic mechanism following ICD implantation. Indeed, their population appears substantially different from ours, because none of their 38 patients presented with spontaneous monomorphic VT before ICD implantation and this was induced by programmed electrical stimulation in only 31 patients.2

Garcia Moran and Mont speculated that the high incidence of syncopal recurrence which was found in our patients was related to the use of low-energy shocks (LESs) because very LESs (≤2 J) have been found to be proarrhythmic at ICD implantation.3,7 They also speculated that programmin high energy shocks (HESs) in the VT zone instead may reduce the incidence of VT acceleration and syncope. LES requires less charging time and a shorter time from VT detection to termination which may protect against syncope. On the contrary, LES may also promote syncope by VT degeneration into a faster VT/VF, which, in turn, may increase the final time to arrhythmia termination. However, VT degeneration into VF by HES presented also in three out of seven syncopal recurrences in our patients.1 In addition, despite similar antitachycardia pacing and LES programming, only a single syncopal episode occurred at follow-up in our group of 50 patients presenting with non-syncopal VT before ICD implantation.1 This latter finding matches the low syncope occurrence that Garcia Moran et al.1 found by programming up to 16 antitachycardia pacing sequences which were followed by LES (“low energy cardioversion was attempted by successive shocks of progressively increasing energy”). Therefore, any statement that is not supported by a controlled trial comparing LES and HES should be considered just a speculation.

We disagree with Garcia Moran and Mont about the existence of a controversy about the recurrence of syncope in ICD recipients, since a controversy exists when conflicting data from different sources are available. However, this is not the case for syncope because most reports3,4 establish its recurrence ~15% in the general population of ICD recipients presenting with syncope before implantation and ~30% in the only one report1 studying the subpopulation of patients with syncopal VT before implantation.

In conclusion, syncope at spontaneous VT presentation identifies a subset of patients with high risk of syncope following ICD implantation, despite similar left ventricular ejection fraction, tachycardia cycle length, and device programming that patients presenting with no syncope at VT documentation. This risk cannot be extrapolated to other ICD populations.

References

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