LETTERS TO THE EDITOR

N-acetylcysteine for the prevention of postoperative atrial fibrillation: a prospective, randomized, placebo-controlled pilot study

I read with great interest the article by Ozaydin et al.1 recently published in European Heart Journal. They briefly compared N-acetylcysteine (NAC) therapy for prevention of postoperative atrial fibrillation (AF) by a randomized, placebo-controlled study and showed that NAC significantly decreased the incidence of postoperative AF. Only three patients in the NAC group, as opposed to 12 patients in the control group, developed AF (P = 0.01). Table 3 shows the follow-up findings and the median duration as well as the range of AF episodes of both groups. The range of AF duration in the NAC group was between 10 and 96 min, whereas it was between 1 and 240 min for control patients. Although the authors accepted an AF episode lasting longer than 5 min as endpoint, it seems that patients with AF episodes <5 min duration were also included in the control group, according to Table 3. If it is so, we may then conclude that the number of control patients with AF episodes was overestimated, indicating a selection bias because some of them had AF episodes <5 min duration. However, this is not the case in NAC patients because all of them had AF episodes of >5 min duration (range, 10–96 min). This means that some of the patients with AF duration <5 min were included in the control group to increase the number of patients with AF and cause statistical significance. Additionally, the technique by which the patients were observed or monitored in intensive care unit was not clearly described. Whether the rhythm was continuously recorded by telemetry and Holter as well as rhythm strips were retrospectively evaluated at a later date by blinded investigators should be clearly described within the text, because short episodes of AF with 1 or 2 min even >5 min duration might certainly be overlooked just by watching the monitor and taking ECGs.

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N-acetylcysteine for the prevention of postoperative atrial fibrillation: a prospective, randomized, placebo-controlled pilot study: reply

We would like to thank Dr Erdogan for his careful reading and his kind interest in our manuscript.1 As he stated in his letter, although an AF duration of >5 min was accepted as endpoint in the study, it seems that patients with AF episodes <5 min duration were also included in the control group according to Table 3.

We excluded the patients with an AF duration of <5 min, and none of the patients in either group had an AF episode of <5 min. We would like to thank Dr Erdogan for helping us to correct a mistake: although it seems that the range of duration in control group is 1–240 min (Table 3), however, it is actually 10–240 min. We realized that we made a typing error while preparing the manuscript. We apologize for the mistake.

The rhythm was continuously recorded by telemetry during the first 2 postoperative days in the intensive care unit. It was continuously monitored and alarm-triggered abnormal rhythms were printed out by cardiac surgery team, which was not included in the study. The printouts of abnormal rhythms were consulted with blinded cardiologists.

Rhythm strips were also retrospectively evaluated and reviewed on a daily basis by blinded cardiologists.

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The new European definition of cardiomyopathies: which space for muscle dystrophies?

I read with interest the new classification of the cardiomyopathies of the European Society of Cardiology1 and the Editorial of Thiene et al.2 In relation with new knowledge of genetic alterations and with terrific progress in imaging techniques of the recent years, this issue is very controversial and the European classification differs deeply from similar classification formulated from the American Heart Association in 2006.3 The effort of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases shall be acknowledged with enthusiasm since cardiomyopathy subtypes are characterized and the concept of new, unclassified cardiomyopathies is introduced. The European Working Group defines cardiomyopathy as ‘a myocardial disorder in which heart muscle is structurally and functionally

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abnormal in the absence of coronary artery disease, hypertension, valvular disease, and congenital heart disease. In this light, a space should be created for an appropriate identification of myocardial alterations occurring in muscle dystrophies, such as X-linked Duchenne’s and Becker’s dystrophy, Steinert’s myotonic dystrophy, and less-frequent Emery-Dreifus dystrophy and associated disorders, limb-girdle muscular dystrophy, and facial–scapular–humeral muscular dystrophy. Diagnostic imaging techniques, mainly transthoracic Doppler echocardiography, have allowed the acquisition of important information on myocardial involvement of these diseases. In the Duchenne’s muscular dystrophy, the most frequent inherited neuromuscular disorder (30 per 100 000 live male births), cardiac involvement is common (80% of patients) and death is due to cardiac dysfunction in about 10% of the cases. Children with Duchenne’s dystrophy develop a significant decrease of ejection fraction as well as an enlarged left ventricle and left atrium and abnormalities of left ventricular (LV) diastolic function; even acquired regions of LV hypertrabeculation/non-compaction localized to LV apex and lateral wall have been reported in rare cases. Becker muscular dystrophy is less common (3 per 100 000 live male births), and the heart is affected in about 30% of patients in the early stages but in up to 90% in the advanced stages. Cardiac involvement leads to myocardial thickening, regional wall motion abnormalities, dilated cardiac chambers, secondary valve regurgitation and systolic and/or diastolic dysfunction. Up to one-third of the patients develop dilated cardiomyopathy and LV hypertrabeculation/non-compaction is more frequent than in Duchenne’s dystrophy. Also patients with Steinert’s myotonic dystrophy, a disease occurring in 13 100 000 births may develop a specific cardiomyopathy and symptoms of heart failure in the advanced stages. Subclinical biventricular involvement may be found in myotonic dystrophy and the greater predisposition of the right ventricle to early decompensation is consistent with the finding of the well-known altered electro-anatomic pattern in the right chambers of these patients. In view of these findings, it is my opinion that cardiomyopathies related to muscular dystrophies should find a specific space in the classification of cardiomyopathies. The early identification of myocardial abnormalities may be crucial in the therapeutic management of these pathologies.

References

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The new European definition of cardiomyopathies: which space for muscle dystrophies?: reply

We thank Dr Galderisi for his response to the European Society of Cardiology classification of cardiomyopathy. His comments on neuromuscular disorders such as Duchenne’s and Becker’s muscular dystrophies illustrate the spectrum of diseases that can cause cardiomyopathies. The aim of the working group for myocardial and pericardial diseases was to ensure that the classification system was sufficiently flexible to allow incorporation of a whole range of disorders under the umbrella of diagnosis of cardiomyopathy. This is achieved by basing the classification on the cardiac phenotype. As heart disease caused by neuromuscular disorders usually falls into one of the major morphological subtypes, it is not necessary to create a separate sub-classification for muscular dystrophies.

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