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International Journal of Cardiology 109 (2006) 344 – 350

International Journal of Cardiology

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Prevalence of inducible paroxysmal supraventricular tachycardia during esophageal electrophysiologic study in patients with unexplained stroke

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Received 23 March 2005; received in revised form 6 June 2005; accepted 11 June 2005
Available online 21 July 2005

Abstract

Background: The relationships between stroke and atrial tachycardia or atrial fibrillation were previously reported. Electrophysiological study is one of the means, used to detect and evaluate these atrial tachyarrhythmias. But, some other arrhythmias as paroxysmal supraventricular tachycardia, can be induced during electrophysiologic study and their significance in stroke is unknown. The aim of the study was to assess the significance of inducible paroxysmal supraventricular tachycardia (PSVT) in stroke.
Methods: One hundred thirty seven patients, aged 61 ± 12 years had unexplained stroke (group I) and were compared to 60 subjects aged 45+/− 18.5 years without stroke and history of tachycardia (group II); Holter monitoring (HM), echocardiogram and esophageal electrophysiologic study (EPS) in basal state and after isoproterenol were performed.
Results: Heart disease was noted in 19 group I patients (14%) and 10 group II patients (17%). In group I, atrial fibrillation or tachycardia (AF − AT) was induced in 20 patients (15%) and PSVT was induced in 19 patients (14%) aged 66 ± 12 years. In group II, AF/AT was induced in 3 patients (5%); no group II patient had induced PSVT. After 3 ± 1 years, in group I, one of 98 patients without induced arrhythmias had new strokes and 2 had AF; 5 patients with induced AT/AF developed AF; 5 patients with induced PSVT had PSVT's, requiring ablation in 4 of them; 1 died from a new stroke; one had a second non-fatal stroke and 3 patients developed AF (16%). In group II, there were no events.
Conclusion: In 14% of patients with unexplained stroke, PSVT was inducible during esophageal electrophysiologic study. Further studies are warranted to assess the significance of this finding in patients with unexplained stroke.

Keywords: Stroke; Electrophysiologic study; Paroxysmal supraventricular tachycardia

The relationships between strokes atrial tachycardia, atrial fibrillation [1–3] or atrial flutter [4] were previously reported. Electrophysiological study is one of the means, used to detect and evaluate these atrial tachyarrhythmias [5–8]. But, some other arrhythmias as paroxysmal supraventricular tachycardia, can be induced during electrophysiologic study and their significance in stroke is unknown.

The purpose of the study was to assess the results of electrophysiological study and in the significance of inducible paroxysmal supraventricular tachycardia (PSVT) in patients with unexplained stroke.

1. Methods

1.1. Population of study

One hundred thirty seven patients (group I), 77 men and 60 women, aged 32 to 82 years (mean 61 ± 12) were prospectively recruited because they have been admitted in our hospital for an unexplained stroke: these patients were initially admitted in neurology and a complete evaluation was performed to rule out a vascular lesion; the stroke was unrelated to a cerebral vascular lesion; there was no evident

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0167-5273/$ - see front matter © 2005 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.ijcard.2005.06.031

cause for an embolic event, nor uncontrolled hypertensive disease.

The neurological symptoms were totally or partially regressive. These patients had no history of supraventricular tachycardias. The surface ECG at the time of stroke and at the time of investigations was in sinus rhythm; there was no preexcitation syndrome.

The evaluation was performed from one week to one month after acute stroke. They were compared to a group II of 60 patients, consecutively recruited and selected on a prospective way if they had the following inclusion criteria: they had no history of stroke and no history of suspected or documented supraventricular tachyarrhythmias; they were 39 men and 21 women, aged 18 to 80 years (mean 45±18.5) studied for sinus bradycardia ($n=27$), unexplained dizziness ($n=20$) or a for a suspicion of preexcitation syndrome, which was excluded by the electrophysiologic study.

1.2. Diagnostic and electrophysiologic studies

After clinical evaluation, the patients underwent the following procedures, which were approved by ethical committee, after giving written informed consent:

After clinical evaluation including extensive neurological examination (generally in the Neurology department), all group I and group II patients underwent a 12-leads ECG, chest X-ray, extracranial carotid echography Doppler and transthoracic Doppler echocardiography. In 111 group I patients, a transesophageal echocardiogram and in all patients a 24-h electrocardiographic Holter were also performed.

An esophageal electrophysiologic study was performed in all group I and II patients, according to a previously reported protocol [9,10]. The patients were in a fasting non-sedated state. All drugs, having an effect on cardiac electrophysiologic properties were stopped since at least 5 half-lives. The possible discomfort induced by esophageal pacing was explained to patients before the test. A bipolar silicone esophageal lead (Prothia 8-Osypka—Germany and then Esosoft 2-Fiab—Italy, Ela) with electrodes spaced at 29 mm, was used. The bipolar electrode was passed through the mouth into the distal esophagus and its position adjusted until the proximal electrode showed the greatest amplitude. Pacing threshold was determined. The stimulus amplitude slightly in excess of that resulting in consistent atrial capture was selected. For the stimulation we used an electrophysiologic stimulator (Explorer 2000, Ela), connected to an Ela pulse amplifier that can deliver pulses of 20 ms width and 29 mA output. Standard intracardiac filtering (30/500 Hz) was used for the esophageal electrocardiogram recording.

Esophageal electrogram was recorded simultaneously with standard ECG leads I, II, VL and V1, V5, V6. The electrograms were displayed on a multichannel oscilloscope (Hellige, VR12 and then Midas of Marquette-Hellige, USA) and recorded on 12-lead ECG at 25 and 100 mm/s. Arterial blood pressure was continuously monitored by an external sphygmomanometer (Baxter).

The electrophysiological protocol was as follows:

Sinus node recovery time was measured [11]. Incremental atrial pacing up to second degree atrioventricular block, and programmed atrial stimulation at a basic cycle length of 600 and 400 ms with the introduction of 1 and 2 extrastimuli were performed. Premature stimuli (S2) were initiated after every 8 paced complexes beginning in late diastole, and at progressively closer coupling intervals until atrial refractoriness occurred. Then, the shortest coupling interval (S1–S2) resulting in consistent atrial capture was chosen, and a second premature stimulus (S3) was introduced beginning with S2–S3 interval of 200 ms. The S2–S3 interval was shortened by 10 ms decrements until S3 no longer resulted in atrial depolarization. If atrial fibrillation or atrial tachyarrhythmia was not initiated under basal conditions, isoproterenol (0.01 to 0.1 μ/kg/min) was infused to increase the sinus rate to at least 130 bpm and the pacing protocol was repeated [5]. If PSVT, the protocol was continued to verify the reproducibility of the induction of PSVT and to look for the induction of another tachycardia.

1.3. Measurements and definitions

Atrial refractory periods were measured for each driven atrial rhythm. Sinus node function was considered abnormal, when the corrected sinus node recovery time exceeded 525 ms [11].

Sustained supraventricular tachyarrhythmia was defined as atrial tachycardia flutter or fibrillation (AT/AF) or other supraventricular tachycardia that lasted 1 min or longer.

As the tachycardia was induced, the atrial electrocardiogram was recorded to define the exact nature of the induced tachycardia. A regular rapid atrial activity (>200 bpm) was in favour of an atrial tachycardia and an irregular and rapid atrial activity was in favour of atrial fibrillation. In the case of induced PSVT, which was reproducibly induced and stopped by atrial stimulation, we have used the criteria that we have previously reported, including the measurement of ventriculoatrial interval and the morphology of atrial activity in leads D1, D3 and V1 [9]: atrial activity was inside the ventriculogram and not visible in these leads but only on the esophageal electrogram when AV nodal reentrant tachycardia was induced; when atrial activity followed the ventriculogram, the negativity of P wave in lead D3 indicated a junctional tachycardia; the negativity of P wave in D1 and/or a P wave occurring after the atrial esophageal electrogram were in favour of a reentrant tachycardia via a concealed left accessory pathway [9]. When P wave was positive in D1 and P wave in V1 occurred before esophageal atrial activity, the mechanism could be an atypical AV node reentrant tachycardia or a reentry through a concealed septal or right accessory