Significance of ST-segment depression during supraventricular tachycardia.

Clues offered by its return to normal at the end of the episode

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The finding of transient ST-segment depression during episodes of supraventricular tachycardia is common but its ischemic significance is usually uncertain. Several authors came to the conclusion that in the absence of positive myocardial scintigraphy these alterations are not associated with a coronary flow-limiting stenosis. Our report tends to confirm this view but we suggest to observe the evolution of ST-segment changes at the very end of the episodes; these mechanisms have not been adequately addressed in previous studies and could provide useful clues to the ischemic or non-ischemic origin of ST-segment abnormalities.

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The finding of transient ST-segment depression during episodes of supraventricular tachycardia is rather common but its ischemic significance is usually uncertain.

Two clinical cases prompted us to address this issue and to review the pertinent literature. Our report suggests that the observation of the evolution of the ST-segment changes at the very end of the episode of supraventricular tachycardia may provide useful clues to their ischemic or non-ischemic origin.

Description of cases

Case 1. A 63-year-old male, with a negative cardiovascular history, no coronary risk factors and practicing frequent and intense recreational activity, presented with complaints of recurrent sudden-onset palpitations, occurring both at rest and during effort.

Cardiovascular evaluation was negative, but Holter monitoring (Fig. 1) and exercise stress testing showed the occurrence

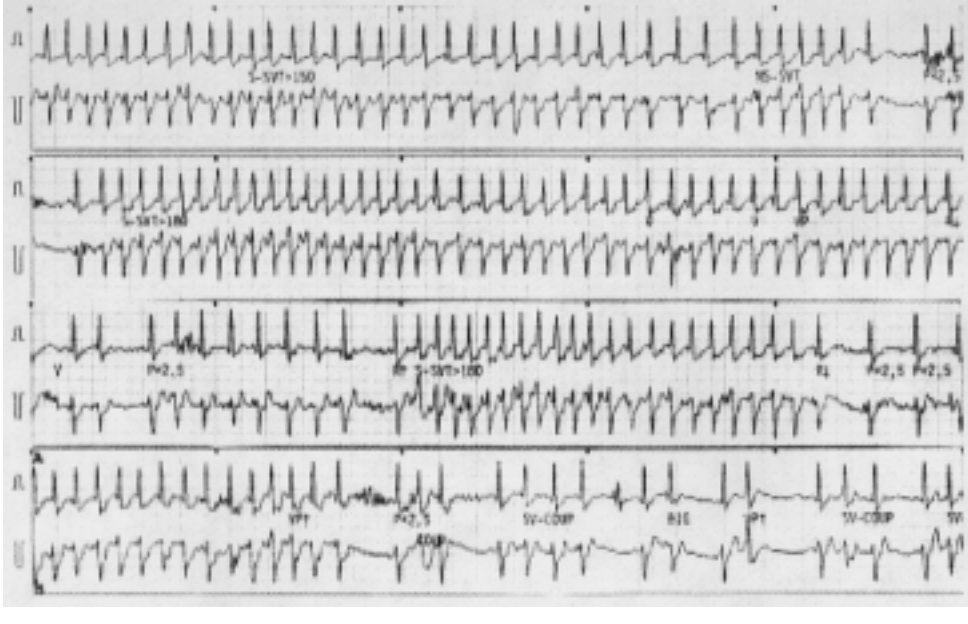

Figure 1. *Holter recording in case 1 showing paroxysmal supraventricular tachycardia with ST-segment depression.*

of supraventricular tachycardia associated with a 4 mm ST-segment depression (Fig. 2) which very promptly disappeared with the return to sinus rhythm (Fig. 3).

In view of a recent case report that ST-segment depression during paroxysmal atrial fibrillation could be a lone manifestation of severe left anterior descending artery stenosis[1] and of the will of the patient who wanted to be completely reassured about the safety of continuing his intense physical activity, a coronary angiography was performed. No stenoses or lumen irregularities in any of the coronary branches were found.

Case 2. A 62-year-old female presented with a recent history of episodes of fainting, sometimes associated with chest pain compatible with angina. During one of these episodes, the patient was admitted to the emergency department where the ECG showed a supraventricular tachycardia with a right bundle branch block pat-

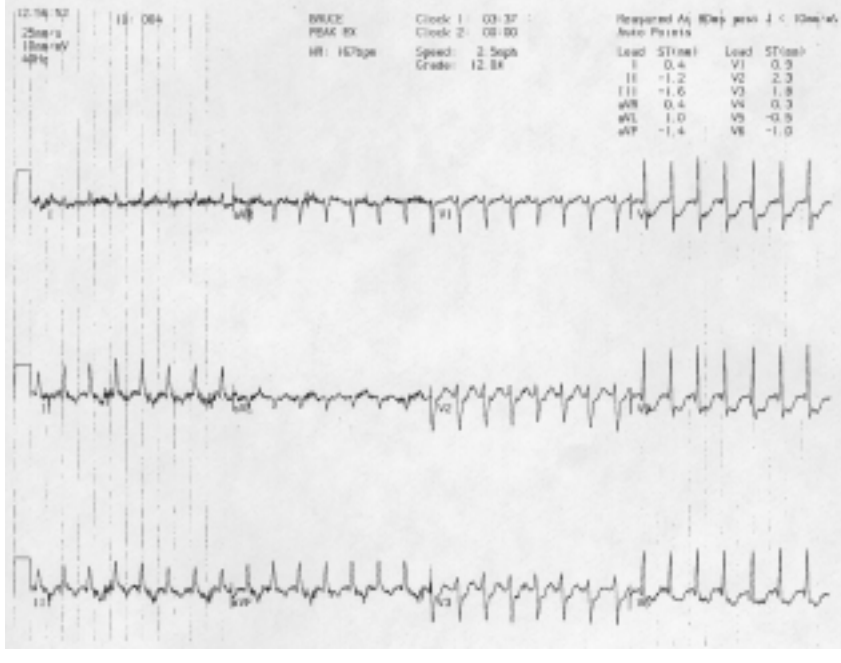

Figure 2. *ECG effort test in the same patient showing a supraventricular tachycardia (167 b/min) with ST-segment depression in leads V_2-V_6.*

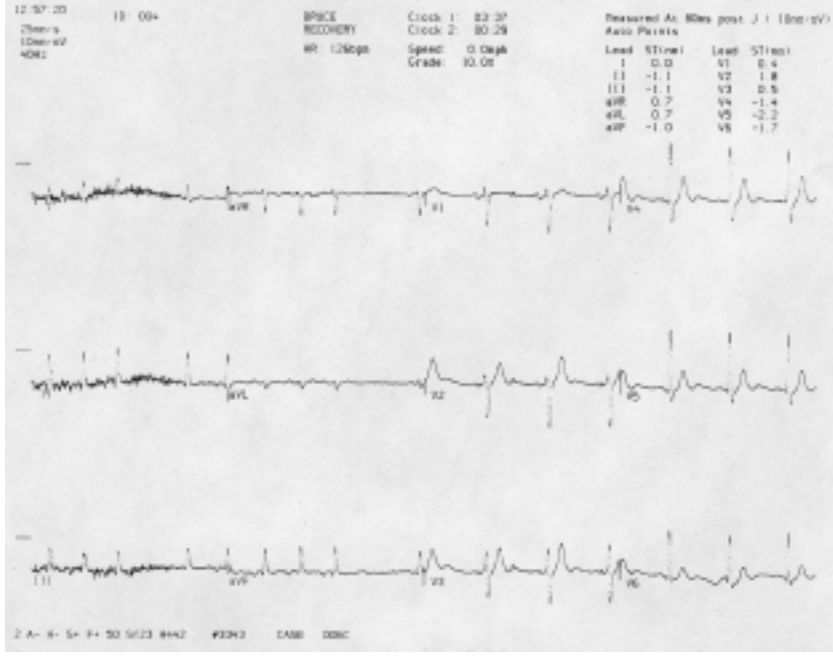

Figure 3. *Interruption of supraventricular tachycardia during the recovery phase of the test with very prompt normalization of the ST segment.*

tern and with a heart rate of 210 b/min. Furthermore, a widespread ST-segment depression (Fig. 4) which persisted for a few minutes after interruption of the tachyarrhythmia (Fig. 5) but which was no longer detectable 6 hours later was observed (Fig. 6). No chest pain was reported by the patient during any of her supraventricular tachycardia episodes. Comparison of the ECG tracings reported in figures 5 and 6 showed subtle differences in lead DIII, which, in figure 5, showed a tendency towards ST-segment elevation, suggesting a possible dorsal transmural ischemia with reciprocal antero-lateral ST-segment depression. However, there were no symptoms indicating vasospastic angina in the clinical history of the patient. Unfortunately, leads V_7-V_9 which would have permitted the detection of isolated dorsal transmural ischemia were not included in the ECG. Echocardiography, performed a few days later, failed to show any evidence of regional left ventricular dysfunction. Coronary angiography was suggestive of normal coronary arteries, without any lumen irregularities. An electrophysiological study showed a slow conduction pathway within the atrioventricular node, which was successfully ablated. At 1 year of follow-up the patient was symptom free.

Discussion

A review of the literature showed that the issue of the clinical meaning of ST-segment depression occurring during supraventricular tachycardia, was addressed by several authors, who usually came to the conclusion that, in the absence of a positive myocardial scintigraphy, supraventricular tachycardia-related ST-segment depression is not associated with flow-limiting coronary stenosis (Table I)[2-7].

The clinical assessment of our 2 cases tends to confirm this view. Nevertheless, some doubts remain regarding our second patient, in whom a persistence for some minutes after supraventricular tachycardia interruption of relevant ST-segment depression in the anterior leads was associated with a tendency to ST-segment elevation in lead DIII, suggesting a possible isolated dorsal transmural ischemia, caused by coronary artery spasm. Unfortunately, the posterior leads were not recorded at the time of the episode and the patient was not submitted to echocardiographic evaluation. Therefore, a conclusive diagnosis cannot be drawn from our findings.

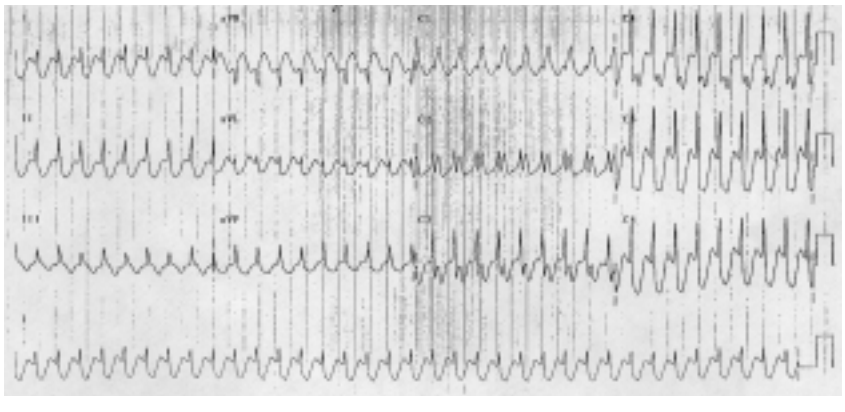

Figure 4. *ECG at the time of admission of case 2 showing a supraventricular tachycardia (210 b/min) with extensive anterior ST-segment depression and a right bundle branch block pattern.*

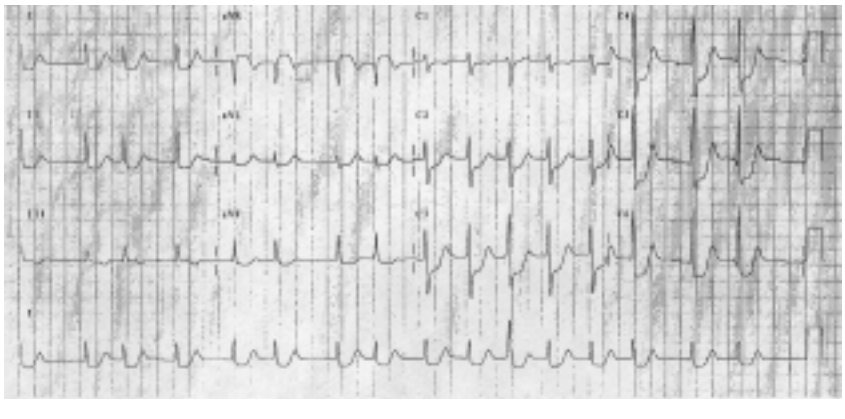

Figure 5. *ECG of the same patient at the time of interruption of the supraventricular tachycardia showing persistent ST-segment depression.*

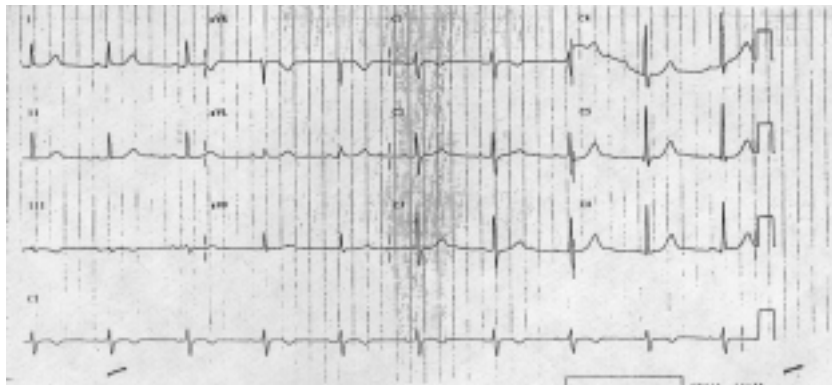

Figure 6. *ECG of the same patient 6 hours later. Note that ST-segment depression has resolved and that the very small QRS is associated with an isoelectric ST-segment in lead DIII. This suggests a possible isolated dorsal transmural ischemia as a cause of the ST-segment changes observed in figure 5, which shows elevation of the R wave and slight ST-segment elevation in lead DIII associated with anterior ST-segment depression. Unfortunately, no recording was obtained of leads V_7-V_9 which could have verified the presence of dorsal ischemia by showing ST-segment elevation.*

The mechanism and clinical significance of persistent ST-segment abnormalities after supraventricular tachycardia have not adequately been addressed in previous studies. Our observation, however, suggests that they merit more careful future investigation, in order to establish whether, in contrast to ST-segment depression appearing only during supraventricular tachycardia, that persisting for a few minutes following the termination of the episode of supraventricular tachycardia may actually be related to myocardial ischemia, possibly consequent to coronary spasm[8]. An echocardiographic examination performed during ST-segment changes might be particularly helpful in this situation, as it would permit confirmation or exclusion of myocardial ischemia by the assessment of regional myocardial contractility.

Table I. Review of the previous reports on the ischemic origin of ST-segment depression during supraventricular tachycardia (SVT).

Authors	No.	Evidence of CAD and methods to assess ischemia	Results
Nelson et al.[2], 1988	Study group 25, control group 7 with known CAD	Measurement of lactate in the coronary sinus during SVT associated with ST↓	Appearance of chest pain in 64% of the study group patients with no significant increase in lactate production during SVT (HR 180 ± 25 b/min). In the control group with CAD, SVT (HR 168 ± 25 b/min) was associated with an increase in lactate production despite a lower ST↓
Petsas et al.[3], 1990	16	n=16 no CAD/exercise testing	ST↓ during SVT was not reproducible with exercise test. SVT with ST↓ during exercise test occurred in only 3 cases
Kim et al.[4], 1991	100	n=80 no CAD, n=15 CAD, n=5 other cardiac diseases	ST↓ does not give diagnostic information about neither the SVT mechanism nor the presence of CAD
Takayanagi et al.[5], 1993	54	n=49 no CAD, n=4 CAD, n=1 other cardiac disease/ exercise test (n=21) and coronary angiography (n=16)	Greater ST↓ with a high HR during SVT in young patients Conclusions: do not perform coronary angiography in young patients with ST↓ during SVT
Imrie et al.[6], 1990	100	n=97 no CAD, n=3 CAD/ stress test, scintigraphy and coronary angiography	ST↓ during SVT is common but not specific and poorly predictive of CAD (predictive value 6%, specificity 51%)
Gulec et al.[7], 1999	39 (group 1, patients with ST↓ during SVT; group 2, no ST↓ during SVT)	n=39 no CAD/exercise test (n=37), thallium scintigraphy (n=38), coronary angiography (all group 1 patients)	In group 1: 7 patients with myocardial scintigraphy and positive exercise test showed CAD Conclusions: perform exercise test or myocardial scintigraphy in patients with ST↓ during SVT, even in the absence of symptoms

CAD = coronary artery disease; HR = heart rate.

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