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Efficacy and limits of sildenafil citrate in patients with arterial erectile dysfunction: role of peripheral arterial disease and cardiovascular comorbidities

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Aim: To evaluate whether the response to sildenafil in patients with arterial erectile dysfunction (ED) was related to their peak systolic velocity (PSV), peripheral atherosclerosis, cardiovascular risk factors and/or co-morbidities at low cardiovascular risk.

Methods: Ninety-seven patients with one or two arterial risk factors and co-morbidities at low cardiovascular risk, combined with penile arterial ED alone (group A, $n = 27$), ED plus atheromasic plaques and/or increased intima-media thickness of the common carotid arteries (group B, $n = 23$), ED plus lower limb artery abnormalities (group C, $n = 25$), and patients with ED plus carotid and lower limb artery abnormalities (group D, $n = 22$). We also examined the efficacy of sildenafil in selected patients with ≥ 3 risk factors, asymptomatic peripheral atherosclerosis and no cardiovascular co-morbidities (group E, $n = 20$).

Results: The median PSV values were 24.1, 21.0, 19.3, 14.5 and 17.5 cm/sec in groups A, B, C, D and E, respectively. All patients met the criteria for sildenafil prescription (100 mg for 12 weeks). The best response (77.8%) was seen in patients of group A. A lower response was seen in patients of groups B and C (65.2% and 56%, respectively). The worst responses were observed in patients of groups D (45.4%) and E (50%) which were significantly lower than that of the other 3 groups. In addition, the response to sildenafil was negatively influenced by the following factors: presence of ≥ 3 risk factors, asymptomatic peripheral atherosclerosis and no systemic co-morbidity, or presence of at least two risk factors associated with extended atherosclerosis and presence of co-morbidities, at low cardiovascular risk.

Conclusion: This study suggests that low efficacy of sildenafil in patients with ED of arterial origin is associated with extended atherosclerosis.

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Tadalafil and sleep-related erections in normal men: randomized, placebo-controlled, crossover study

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Subjects and Methods: we performed nocturnal penile tumescence and rigidity monitoring (NPTRM) in order to study the effects of Tadalafil on sleep-related erections in 25 adult healthy men (mean age 34.90 ± 8.55 years). The subjects were randomly administered Tadalafil, 10 mg tablet, the 1st (N1) or the 2nd (N2) night (Group A and B respectively) of 3 consecutive nights of erections' monitoring. In Group A the 3rd night monitoring was regarded as control (N-ctr); in Group B the 1st night. Tadalafil was administered 2 h before bedtime. NPTRM parameters analyzed were: number of valid erections, total duration of rigidity ≥60% and ≥70%, maximum rigidity, maximum increase of tumescence and total duration of increase of tumescence ≥30 mm.

Results: Number of valid erections [p1 (p N-ctr vs N1) <0.01; p2 (p N-ctr vs N2) 0.01], total duration of rigidity ≥70% (p1 0.03; p2 0.05) and maximum increase of tumescence (p1 0.03; p2 0.03) were significantly higher in nights 1 and 2 after Tadalafil than in control night; no differences occurred between the 2 nights after Tadalafil. Total duration of rigidity ≥60% (p1 0.03; p2 ns) and total duration of increase of tumescence ≥30 mm (p1 < 0.01; p2 0.06) showed higher values only in night 1 after Tadalafil than in control night.

Conclusion: Our data suggest that Tadalafil is efficacious in improving sleep-related erections. Furthermore in normal men Tadalafil improved NPTRM also in the second night after 24 h from drug assumption.

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Cardiovascular risk engines can help in selecting patients to be evaluated by dynamic penile color doppler ultrasound

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Introduction: The aim of the present study is to evaluate the validity of different risk scores in the identification of patients being screened for arteriogenic erectile dysfunction (ED) at Dynamic-Penile Color Doppler Ultrasound (D-PCDU).

Material and Methods: A consecutive series of 738 (mean age 53.6 ± 9.2 years) patients with ED was studied. All patients underwent D-PCDU. Arteriogenic ED was defined when peak systolic velocity (PSV) was lower than 25 cm/sec. The assessment of cardiovascular risk was evaluated using different risk engines, derived from the Framingham, the PROCAM and the Progetto Cuore studies.

Results: Among the patients studied, 52 (7%) had PSV <25 cm/sec. The area under the ROC curves for pathological PSV in relation to cardiovascular risk estimated with different engines was 0.762 ± 0.03, 0.716 ± 0.03 and 0.667 ± 0.03 for Progetto Cuore, Framingham and PROCAM engines, respectively. Sensitivity and specificity of Progetto Cuore estimated risk were 67%, 71% when a threshold of 15% was chosen. Corresponding figures for Framingham and PROCAM engine were 74%, 57% and 69%, 55%, respectively.

Conclusions: If D-PCDU is performed only on patients with cardiovascular risk >15%, who represent about 1/4 of all patients (26.8%), as estimated by Progetto Cuore, about 70% of cases of arteriogenic ED can be identified. This means that well over two thirds of cases can be diagnosed by performing D-PCDU on one patient out of four. Estimated cardiovascular risk, assessed through risk engines, could be used to identify patients who should undergo D-PCDU evaluation for the diagnosis of arteriogenic ED.

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