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DOES SMOKING CIGARETTE MODIFY URODYNAMICS IN WOMEN WITH STRESS URINARY INCONTINENCE (SUI)?

Hypothesis / aims of study

Some authors have reported that cigarette smoking is related to urinary incontinence in women [1]. The aim of this study was 1) to analyze the consequences of cigarette smoking on the results of urodynamic testing in women with stress urinary incontinence, and 2) to search for differences with non-smokers.

Study design, materials and methods

Fifty hundred and sixty women neurological disease, diabetes mellitus, pelvic prolapse of grade > II or previous surgery for incontinence were investigated for urinary incontinence between September 2002 and December 2004. Seventy eight patients were smokers; of them, 16 were heavy current smokers (more than 20 cigarettes per day and average lifetime cigarette consumption 11 pack-years) and complained of SUI. Mean age was 42.5 years (range [30-55 years]). All patients underwent a complete urogynaecological examination and a standardized urodynamic evaluation in semi-recumbent position. The last included uroflowmetry (post void residual urine measurement), urethrocystometry, urethral pressure profilometry and a second uroflowmetry. In addition, a bladder diary was recorded.

Modelized analysis of the free uroflows was performed using the VBN® micturition model [2]; in absence of pressure recording it allowed only to test the hypothesis of a urethral obstruction (constrictive or compressive).

Comparison was done with the urodynamic data of a similar non-smoker female population (40 patients) with complaint of SUI previously studied [3].

Results

Parity was the same in the 2 groups (mean: 2). Pelvic floor muscle testing was slightly better for non-smokers (> 3 for 75 % vs 62%). Grade I cystocele was found in 56% of non smokers (12% of smokers). The maximum urethral closure pressure was increased in 31% of smokers while only in 19% of non smokers. Urethral instability was more frequent in smokers (56%) than in non smokers (19%).

The flow curves were bell shaped in the same proportion (60%). The voided volume (372 $\pm$ 236 mL) was similar in both groups but the maximum flow rate was significantly increased for smokers (32.5 $\pm$ 12.8 mL/s vs 26.3 $\pm$ 9.6 mL/s).

The value of TQmax/T100 was .39 in the smoker group (.36 in the non-smoker group), near the value assumed as normal (.33).

The bladder diary showed an increased fluid intake (> 2.5 L per day) for smokers.

Analysis of the free uroflows for smokers was consistent with a urethral compression in 31%, a constriction in 12%, a gaping in 18% and an absence of obstruction in 39%. These results were similar to those of the non smokers.

Interpretation of results

Development of SUI seemed not clearly related with smoking in absence of other risk factors.

Increased fluid intake could be related with an increased 24 hours urine production and so, a higher risk of incontinence episodes.

In the studied smoker population, the urethral pressure was more frequently increased as previously found [1] but without any significant effect on the urethral function compared to a non smoker group (i.e. delayed opening of the sphincter or urethral obstruction). Neither usual voiding parameters nor VBN® parameters differed from similar non smoker population but significant differences were found: urethral instability was more frequent, pelvic floor muscles were less efficient.

Concluding message

Despite the small size of our smoker population, our findings are consistent with the earlier hypotheses [1] which assumed that violent coughing by smokers promotes the development of an anatomic defect allowing incontinence. Increased fluid intake could be a determinant for occurrence of stress incontinence. To study a more large group would be of great interest in order to verify the absence of change in the voiding parameters.

[1} Am J Obstet Gynecol (1992) 167: 1213-18; (1994) 170: 579-82
[2] Neurourol Urodyn (2000) 19: 153-76
[3] Ann Réadap Méd Phys (2002) 45: 26-32