Editorial

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Editorial: The Relationship between Obesity and Urinary Incontinence and its Causes

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Introduction

Up to 50% of adult women report urinary incontinence (UI) [1]. It has a major impact on quality of life, equivalent to diabetes or rheumatoid arthritis [2,3]. Similarly, obesity is also an increasing worldwide epidemic with pervasive associations with arthritis, hypertension, diabetes, cancer and cardiovascular disease [4]. The prevalence of obesity now exceeds 25% of the adult population in Europe, Australia and the United States [5]. Aging, childbirth and overweight and obesity are identified as the three major risk factors for incontinence [6,7]. Age and parity are not modifiable, but being overweight or obesity is potentially highly modifiable.

Many epidemiological studies have shown a positive association between incontinence and obesity in women. There is a clear dose-response effect of weight on UI with each 5-unit increase in BMI being associated with 20% to 70% increase in the risk of daily incontinence [8]. However, the literature regarding the relationship between obesity and the different subtypes of incontinence is mixed. In EPINCONT, the largest population based study of urinary symptoms; a high BMI was associated with each type of UI, with the strongest association in severe mixed incontinence [9]. A subsequent large systematic review suggested a stronger association between weight and stress incontinence, than for urgency incontinence and overactive bladder syndrome [10]. However, in another study of obese women with a BMI greater than 40kg/m^2 who were planning weight reduction surgery the prevalence of incontinence was 60% to 70% with a prevalence of 28% for pure stress type, 4% for pure urge type and 32% for mixed type [11-13]. In the population- based FINNO study, obesity doubled the risk of stress incontinence and tripled the risk of urgency incontinence [14]. Longitudinal cohort studies have demonstrated an association of overweight and obesity with incident UI, providing evidence for a temporal relationship between a possible cause and an outcome. In 5-10 years follow up the odds of incident UI increased by around 7%-12% for each 1kg/m^2 unit increase in BMI [15]. This association was again strongest for stress and mixed incontinence, and weaker for urge incontinence and overactive bladder syndrome.

The association of overweight and obesity with stress incontinence has been shown in numerous studies [15-18]. However in the Nurses' Health study increased waist circumference, but not BMI predicted incident stress UI among middle aged women [17]. Therefore, cross sectional studies show that in addition to BMI, waist-hip ratio and thus abdominal obesity may be an independent risk factor for incontinence in women. Other studies have bolstered evidence that waist circumference may represent a reasonable metric for denoting voiding dysfunction. In the results of a cluster analysis of the Boston Area Community Health Survey where 1899 men were studied and classified into five clusters depending on the severity and pattern of urinary symptoms, more symptomatic men tended to have larger WC as well as higher rates of hypertension, diabetes and cardiovascular symptoms [19].

Although a direct cause-effect relationship between obesity and incontinence has not yet been established, there is evidence that weight reduction might be beneficial to obese incontinent women [12,20-23]. Only a few interventional studies have been carried out to assess the effect of weight reduction on incontinence, but they all show that incontinence is reduced by weight loss. In a large trial of 338 obese women with urinary incontinence, a 6-month structured weight loss programme was more effective than education alone for weight loss 8% body weight average weight loss for treatment versus 1.6 % for the control group] and reducing the frequency of urinary incontinence (47.4 vs 28.1%) [8]. This benefit was more pronounced for stress than urge incontinence. Moderate weight loss seems to be an adequate first line therapy for urinary incontinence in women. The interventional and experimental evidence is also seen as a strong support for the causation hypothesis. In women with type two diabetes moderate weight loss has been shown to reduce the incidence but not improve resolution rates of urinary incontinence at 1 year [24]. Each Kg of weight lost was associated with a 3% reduction in the odds of urinary incontinence developing and weight losses of 5%-10% reduced these odds by 47%. As in other studies, larger weight loss did not appear to result in greater benefit [16]. This is encouraging as such moderate weight loss can be largely achieved and maintained through lifestyle interventions.

It is widely proposed that obesity may contribute to stress incontinence through increased intra-abdominal pressure from

central adiposity, which in turn increases bladder pressure and urethral mobility, thus exacerbating UI [20,22]. The same mechanisms are thought to exacerbate detrusor instability and over active bladder [20,22]. It is also suggested that this pressure may damage the pelvic floor in obese women, causing chronic strain, stretching and weakening of the muscles, nerves and other structures in the pelvic floor. Weight loss may reduce forces on the bladder and pelvic floor, thus reducing stress incontinence preferentially. With moderate weight loss of 13% of baseline weight significant correlations between weigh change and decreased initial intravesical pressure have been noted [22].

Apart from a mechanical effect, a metabolic effect on incontinence has been postulated. Several metabolic disorders observed in the metabolic syndrome (Enlarged waist, high triglyceride level, low high-density lipoprotein cholesterol, arterial hypertension and hyperglycaemia) are associated with urinary disorders in women [25,26]. A recent systematic review has clearly demonstrated an association between obesity, MetS and OAB and LUTS [27]. Some evidence points to inflammation as the mediator between voiding dysfunction and the metabolic syndrome and obesity. Rohrmann et al, for example reported on an analysis of 2337 men aged 60 years or older from the Third National Health and Examination Survey (NHANES III); the presence of lower urinary tract symptoms was positively associated with serum levels of C- reactive protein, a non-specific marker of inflammation, even after adjustment for the presence of the metabolic syndrome [28]. This association has been confirmed more recently in both men and women [29]. Interestingly significantly higher levels of CRP have been shown in women with OAB wet, rather than OAB dry. This suggests that serum levels of CRP may correlate with severity of symptoms [30]. In other studies the occurrence of chronic low-grade inflammation in the bladder of obese women was indicated by increased urinary chemokines [31]. Animal studies have also been used to investigate the effect of metabolic perturbations on the bladder. Insulin resistance caused by obesity is a significant component of MetS and it is argued that the increasing prevalence of metabolic syndrome in the last 10 years has been linked to the increasing consumption of a fructose containing diet [32]. Thus, rats fed fructose rich diets have been adopted as experimental models in which to stimulate environmentally induced metabolic syndrome. In the early phase of metabolic syndrome the over expression of muscarinic receptors and dysfunctional mitochondria in the detrusor were associated with detrusor over-activity in the fructose fed rats [33,34]. In animal models and human patients, the metabolic effects of fructose play important roles in the activation of cellular stress pathways [31]. Oxidative stress, mitochondrial dysfunction and increased cell apoptosis, which may be induced by hyperglycaemia and metabolic perturbation, contribute to diabetic neuropathy and myopathy [35-37].

Another potential aetiology could lie in pelvic atherosclerotic disease caused by presence of metabolic syndrome. A close correlation between pelvic ischemia and bladder over-activity has already been documented [38-41]. A pro-inflammatory state increased free fatty acids, hypercoagulability, and cellular oxidative stress can lead to premature vascular disease ischemia and pelvic atherosclerosis, which may result in primary bladder dysfunction [42]. Azadzoi has reported increased levels of TGF-B1 expression leading to fibrosis, smooth muscle atrophy and lowered compliance in the bladders subjected to atherosclerotic injury and high cholesterol diets [43].

The evidence clearly shows a strong relationship between obesity and incontinence. We are yet to ascertain the exact mechanisms involved but a link with metabolic syndrome seems likely. Weight loss has the potential to alter the natural history of voiding dysfunction, and as so should be considered as a valuable first line treatment option in incontinence.

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