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Stress Urinary Incontinence (SUI) Risk Factor in Pelvic Floor Muscle Strength (PFMS) among Postmenopausal Women: A Case-Control Study

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ABSTRACT

Postmenopausal health problems cause many symptoms that affect the quality of life among women, including the reduction of pelvic floor muscle strength (PFMS). Stress urinary incontinence (SUI) is one of the postmenopausal women's problems due to various etiology. Risk factors analysis and stratification are important for further prevention and treatment. A case-control study that collects postmenopausal women divided into two groups, 52 postmenopausal subjects with SUI and 52 without SUI. Pelvic floor muscle strength is measured by a perineometer (Pertiron 9300 V). There is a correlation between the occurrence of SUI with parity (OR=11.111 95% CI 2.403-51.371) and the delivery mode (OR=8.704 95% CI 2.984-25.387). The cut-off value of PFMS to predict SUI is 27.03 cmH2O with 84.6% of sensitivity and 84.6% specificity. Our study reveals PFMS in combination with parity and delivery was simultaneously significant to the occurrence of SUI. Pelvic Floor Muscle Strength among menopausal women is correlated with SUI. Risk factors of SUI related to the reduction of PFMS are multiparity and vaginal delivery.

1. Introduction

Menopause is a natural process experienced by women associated with the end of ovulation and decreased production of estrogen and progesterone [1]. During menopause, women feel various symptom of somatic and psychological symptoms [2]. Problems related to menopause are increasingly related to the increase in women's life expectancy which currently reaches 75.6 years [3]. Postmenopausal women will experience

several symptoms that will cause social problems [4]. One of the most common symptoms of menopause is urinary incontinence [5]. Stress Urinary Incontinence is the most common type of incontinence with a percentage of 50%-88% of all types of urinary incontinence. This situation occurs characterized by leakage of urine due to increased intra-abdominal pressure, such as sneezing, coughing, and lifting weights [6].

Stress urinary incontinence can occur due to weakening pelvic floor muscles, damage to the cutaneous muscle-ligament-fascia tissue complex, hypermobility, and vesicourethral intrinsic sphincter deficiency [7]. Various risk factors such as delivery mode, baby birthweight, parity, duration of menopause, body mass index, and pelvic organ prolapse [5]. Vaginal delivery is a risk factor for SUI compared with cesarean delivery [8]. This is because the lowering of the head that occurs can cause pressure and strain on the pelvic floor and surrounding nerves [9]. It is also related to the birth weight of the baby and the amount of parity through the mechanism of increased intra-abdominal pressure and intracellular matrix damage [10].

Information and data regarding the interaction of various risk factors for SUI occurrence in postmenopausal women are still very limited. Our study investigates the possible correlation between SUI and various risk factors among postmenopausal women.

Methods

A case-control study was taken on 52 subjects of 3-year minimal postmenopausal women with SUI diagnosed by Questionnaire for Female Urinary Continence Diagnosis (QUID) and 52 subjects without SUI in Teaching Hospitals Faculty of Medicine, Hasanuddin University at Obstetric and Gynecologic Department, Indonesia. This study was approved by the Research Ethics Commission of the Faculty of Medicine, Hasanuddin University (No: 430/UN4.6.4.5.31/PP36/2021). All subjects signed written informed consent. Subjects who have a history of hormone-replacing therapy, diuretic use, cystectomy, oophorectomy, hysterectomy, stroke, cancer, or urinary tract infection at the time of measurement were excluded from the study. Demographic and clinical data were collected regarding age, body mass index (BMI), the highest birthweight, education, menopausal duration, occupation, parity, delivery mode, and pelvic organ prolapse status. All the subject's pelvic muscle strength is measured by a perineometer (Pertiron 9300 V).

Statitical analysis

Baseline data were descriptively summarized, and the differences in each variable between groups were calculated using Mann-Whitney, Chi-square, and Fischer's exact tests. Risk factors associated with SUI were analyzed, and outcomes were reported as odds ratios (OR) with 95% confidence intervals (CI). Significant values were determined at p <0.05. Logistic regression was used to the strongest association between variables with the outcome of SUI. All statistical analyses were performed using the Statistical Program for Social Sciences (IBM SPSS 24, IL, USA).

2. RESULTS

Based on this study, the mean age was 62.21 years. The average parity was 3.41 times giving birth, menopausal duration mean was 8.61 years. The body mass index mean of all subjects was 25.28 kg/m^2. The average birth weight of babies is 3340 grams.

According to statistical analysis, we found a correlation between the occurrence of stress urinary incontinence with menopausal duration (OR=2.825 95% CI 0.99-8.059), parity (OR=11.111 95% CI 2.403-51.371), delivery mode (OR=8.704 95% CI 2.984-25.387), and pelvic organ prolapse (OR=3.726 95% CI 1.463-9.491) (Table 1).

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A pelvic floor muscle strength test was performed on all subjects and we found a correlation between PFMS and the occurrence of SUI. The pressure of the case group (23.22 ± 3.77 cmH_2O) is weaker than the control group (31.58 ± 4.17 cmH_2O). The cut-off value of PFMS to predict SUI is 27.03 cmH_2O with 84.6% of sensitivity and 84.6% specificity strongly correlated with the occurrence of SUI (Table 2 and Table 3).

We performed two logistic regression models, without PFMS (first model) and with PFMS (second model). In the first model, we found that only parity and delivery mode significantly contribute as a predictor of SUI. In the first model, we found type of delivery (OR=4.767 95%CI 1.462-15.549 p=0.01) and parity (OR=5.608 95%CI 1.113-28.257 p=0.037) significant as a predictor of SUI. In the second model, PFMS in combination with parity and delivery mode simultaneously significant to the occurrence of SUI (Table 5).

Table 1. Characteristics of subjects

Variable	SUI		OR 95% CI	p	r	p
	With (N=52)	Without (N=52)				
Age						
>60 years old	32 (52.5%)	29 (47.5%)	1.269 (0.581-2.774)*	0.550	0.059	0.555
≤60 years old	20 (46.5%)	23 (53.5%)				
BMI (kg/m^2)						
≥25	27 (52.9%)	24 (47.1%)	1.260 (0.583-2.722)*	0.556	0.059	0.562
<25	25 (47.2%)	28 (52.8%)				
Highest birthweight						
>4000 gram	3 (100%)	0 (0%)	2.061 (1.686-2.520)**	0.253	0.172	0.080
≤4000 gram	49 (48.5%)	52 (51.5%)				
Menopausal duration						
≥10 years	14 (70.0%)	6 (30%)	2.825 (0.99-8.059)*	0,047#	0.195a	0.047#
<10 years	38 (45.2%)	46 (54.8%)				
Parity						
Multipara	50 (58.1%)	36 (41.9%)	11.111 (2.403-51.371)*	0,000#	0.356b	0.000#
Primipara	2 (11.1%)	16 (88.9%)				
Delivery mode						
Vaginal delivery	47 (63.5%)	27 (36.5%)	8.704 (2.984-25.387)*	0,000#	0.424c	0.000#
Cesarean section	5 (16.7%)	25 (83.3%)				
Pelvic organ prolapses						
With	21 (72,4%)	31 (41,3%)	3.726 (1.463-9.491)*	0.004#	0.279b	0.004#
Without	8 (27,6%)	44 (58,7%)				

*Chi-square test, **Fischer-exact test, #signicant, avery low correlation, blow correlation, cmoderate correlation

Table 2. Pelvic floor muscle strength

Group	N	PFMS (cmH_2O)			p
		Min	Max	Mean ± SD	
Case	52	18.80	35.10	23.22 ± 3.77	0,000#

Control	52	19.17	36.70	31.58 ± 4.17		

Mann-Whitney test, #significant

Table 3. PFMS as a predictor of SUI

Variable	SUI		OR 95% CI	p	r	p
	With (N=52)	Without (N=52)				
PFMS (cmH_2O)						
≤27.03	44 (84.6%)	8 (15.4%)	30.250 (10.424-87.781)*	0.000#	0.692d	0.000
>27.03	8 (15.4%)	44 (84.6%)				

*Chi-square test, #significant, dstrong correlation

Table 4. Logistic regression without PFMS (first model)

Variable	SUI		OR 95% CI	p
	With (N=52)	Without (N=52)		
Menopausal duration				
≥10 years	14 (70.0%)	6 (30%)	1.686 (0.529-5.374)	0,377
<10 years	38 (45.2%)	46 (54.8%)		
Parity				
Multipara	50 (58.1%)	36 (41.9%)	5.608 (1.113-28.257)	0,037#
Primipara	2 (11.1%)	16 (88.9%)		
Delivery mode				
Vaginal delivery	47 (63.5%)	27 (36.5%)	4.767 (1.462-15.549)	0,010#
Cesarean section	5 (16.7%)	25 (83.3%)		
POP				
With	21 (72,4%)	31 (41,3%)	1.685 (0.597-4.756)	0.324
Without	8 (27,6%)	44 (58,7%)		

#significant

Table 5. Logistic regression with PFMS (second model)

Variable	OR CI 95%	p
PFMS **PFMS 27.03 cmH_2O + Parity**	25.667(8.084-81.851)	0.000#
PFMS 27.03 cmH_2O + Delivery Mode	27.682(7.585-101.035)	0.000#
PFMS 27.03 cmH_2O + Parity + Delivery Mode	24.465(6.374-93.908)	0.000#

#significant

3. DISCUSSION

Pelvic floor muscle strength is measured to define of SUI among postmenopausal women [11], [12]. Menopause is related to the reduction of estrogen levels which causes sensitive estrogen-related tissue in the support system of the pelvic floor. The Pelvic Floor Support System (PFSS) has dense estrogen, progesterone, and androgen receptor [13]. The hormonal action affects the detrusor due to muscarinic modification and inhibits calcium ion influx. This mechanism explains hormonal changes affect PFMS [14]. The majority of the subject's age is more than 60 years old. There was no significant difference in age

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between the two groups. A study by [15] shows that age over 50 is not a risk factor for SUI. Another study also stated that there was no relationship between urinary function and old age in the incidence of SUI [16]. Age was not a significant factor for patients with many other risk factors such as multiparity and vaginal delivery [17].

Contrary to the subject's age, postmenopausal duration is a significant risk factor for SUI. Pathophysiological changes were only can be explained due to hormonal changes. The decreased amount of circulating estrogen reduces the support system of the pelvic floor [5].

The pathophysiology of SUI is related to age through decreased extracellular matrix synthesis, hormones, and exposure to oxidative stress [18]. Decreased extracellular matrix synthesis relates to the resistance to supporting pelvic floor structures. Decreased synthesis causes a reduction of elasticity to support the pelvic floor structure and sphincter muscle strength. This decrease in elasticity is related to the mechanism of SUI [19]. Hormonal changes, especially estrogen, cause SUI through atrophy of the pelvic organs and PFSS. Oxidative stress affects the occurrence of SUI in old age related to the mechanism of cellular damage. Oxidative stress also impairs collagen metabolism [20].

Body mass index (BMI) is a factor in the occurrence of SUI. The pathophysiology associated with BMI in the incidence of SUI includes increased intra-abdominal pressure and biomechanical impacts [21]. Increased intra-abdominal pressure suppresses all intra-abdominal organs. This repeated pressure for a long time causes a decrease in the elasticity and strength of the supporting structure of the pelvic floor, including ligaments and muscles. Decreased elasticity reduces the ability of the pelvic floor structures to provide urinary stability. The biomechanical impact that occurs due to increased intra-abdominal adipose tissue mass causes excessive pressure on the pelvic organs. Biomechanical load shows a direct role and predisposes to increased intra-abdominal pressure [22]. Biomechanical loads are also influenced by the method of delivery and parity [23].

A study by [24] revealed there was no relationship between the occurrence of SUI and BMI. Other studies have stated that increased BMI is not the sole factor in multipathological conditions and only exacerbates existing conditions. The incidence of SUI due to higher BMI is not a single risk factor but affects women with multiparity and vaginal delivery in postmenopausal women [25].

Most of the subject's highest birthweight is less than 4000 grams. There was no significant difference between the two groups. The birthweight also affects the occurrence of SUI due to biomechanical loads during pregnancy and stretching during labor if delivered vaginally. The biomechanical load induced by fetal mass is related to the tensile strength of the supporting structures of the pelvic floor. This biomechanical load can cause irreversible damage to the elasticity and strength of the supporting structures of the pelvic floor. Vaginal delivery with a large baby causes damage to the birth canal and temporary ischemia to the pelvic floor structures. The condition of decreased elasticity of the supporting structures of the pelvic floor due to biomechanical loads during labor is partially reversible to irreversible [26]. Research conducted by [27] shows no relationship between birthweight and the incidence of SUI [27]. In another study, with birthweight over 4000 grams, augmentation and instrumentation during vaginal delivery influenced the occurrence of SUI [28].

This study revealed that parity affects the occurrence of SUI. Parity can affect the occurrence of SUI through the mechanism of repetitive biomechanical loading on the supporting structures of the pelvic floor, repeated stretching, ischemia and changes in the extracellular matrix of the supporting structures of the

pelvic floor [29]. This complex mechanism occurs repeatedly causing the higher the parity level, the higher the damage to the supporting structures of the pelvic floor so that the incidence of SUI is higher in groups with high parity with the same other risk factors [30].

Parity is supported by various other studies as a causative factor for SUI. Parity can be an independent factor in the occurrence of SUI. This risk factor can even occur in groups that are not yet menopausal. This factor can be a factor with potentiation for other factors. The correlation between grande-multipara and non-grande multipara in experiencing SUI is very strong [31].

Parity is a highly modifiable factor from a young age. Data collection and education in SUI risk groups with parity can be carried out [32]. Family planning programs can reduce parity rates, reducing the risk of SUI. Contraceptive education has a significant effect on lowering SUI rates [33].

The delivery mode is related to SUI. The vaginal delivery method triggers SUI through a mechanism similar to parity, namely biomechanical loads, especially during the second stage, and tissue ischemia and delivery tools [34]. These conditions simultaneously affect the quality of support to the pelvic floor structures. The perceived biomechanical load causes ischemia and irreversible damage to the extracellular matrix [35].

The delivery mode can be a single factor in the process of SUI. This condition can also potentiate SUI, especially in childbirth using assistive devices. This occurs because the damage to the supporting structures of the pelvic floor is more significant [36]. Delivery by cesarean section is a protective factor against SUI [37].

Pelvic organ prolapse (POP) is one of the conditions that influence the incidence of SUI. This happens because the mechanism for the occurrence of SUI based on risk factors is also a risk factor for POP. Severe POP symptoms can manifest as SUI [38]. The risk factors for POP for SUI have been supported by various studies. This condition is an implication of the same mechanisms and mutual potentiation between risk factors [39].

We found the cut-off value of PFMS to predict SUI is 27.03 cmH_2O with 84.6% of sensitivity and 84.6% specificity with strong correlation. Pelvic floor muscle strength related specifically to SUI. The supporting structure around PFMS weakened multipathologically [40]. All the combination with PFMS with parity and the delivery mode are significant for screening and diagnostic tools of SUI

4. CONCLUSION

Pelvic floor muscle strength PFMS among menopausal women is correlated with SUI. Risk factors of SUI related to the reduction of PFMS are multiparity and vaginal delivery. All of the risk factors combined among postmenopausal women related to SUI due to the reduction of PFMS. This study found an association between PFMS and SUI among postmenopausal women. Although it has not been directly proven, this finding should be a matter of caution in terms prediction of SUI by analyzing the risk factors. However, SUI could be affected by various risk factors. Further studies need to be conducted to exclude other risk factors. We suggest a model of screening using a history of the delivery mode with parity and diagnostic tools among post-menopausal women using a perineometer.

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All authors have contributed to all processes in this research, including preparation, data gathering, analysis,

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drafting, and approval for publication of this manuscript.

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