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Title (type in CAPITAL LETTERS, leave one blank line before the text):

BIOCHEMICAL CHANGES ASSOCIATED WITH STRESS URINARY INCONTINENCE AND THE EFFECT OF MENOPAUSE AND HOROMONE REPLACEMENT THEREAPY: A CONTROLLED STUDY

Aims of study

Genuine stress incontinence (GSI), a common gynaecological condition, is frequently due to bladder neck hypermobility caused by a weakness in the supporting structures of the pelvic floor. Its aetiology is almost certainly multifactorial. Collagen, a fibrous protein, forms the major structural component of vaginal epithelium and imparts tensile strength to the tissue. A significant reduction in total collagen, of vaginal tissue, has been demonstrated in nulliparous premenopausal women when compared to controls. There was an associated reduction in intermolecular collagen cross-linking, suggesting that the underlying defect within this population may be congenital rather than acquired (1). A similar reduction in collagen has not been clearly demonstrated in post menopausal women with GSI when compared to controls however, oestrogen therapy has been shown to produce a reduction in collagen content (2). The increased incidence of GSI around the menopause would suggest an alteration in collagen metabolism occurs at this time. We set out to further clarify the pathophysiological changes seen in women with bladder neck hypermobility: investigating the effect of menopause and additional hormone replacement therapy (HRT) on the supporting tissue on the pelvic floor.

Methods

Women recruited into this controlled study were placed in three groups: pre menopausal, postmenopausal without HRT and postmenopausal with at least 1 year of standard HRT. These were matched with continent controls in similar groups. All those with stress urinary incontinence symptoms had the diagnosis of GSI confirmed by conventional cystometric testing. The validated Bristol Female Lower urinary tract symptom questionnaire was used to exclude urinary incontinence in the control groups. The International Continence Society's female pelvic organ prolapse grading system was used to assess genitourinary prolapse and women were withdrawn with a score greater than 1. Tissue samples ware taken peri-urethrally from the anterior vaginal wall using Eppendorfer punch biopsy forceps. The tissue was stored at –80 °C before undergoing biochemical analysis. Total collagen content was determined by hydroxyproline analysis and sulphated proteoglycan assay using dimethylmethylene blue. The protein content was assayed by microkjeldahl analysis. The data underwent analysis of variance using. Further analysis of glycation end products and proteinase activity is taking place.

Results

There were 116 women recruited into this study, 58 women in the incontinence group and 58 in the control group. In the incontinence group: 28 were premenopausal with a mean age 43 years (range 26-53), 14 were post menopausal without HRT with mean age 59 years (range 48-77) and the 14 in the postmenopausal group with HRT had a mean age of 56 years (range 46-63). In the control group: 28 were premenopausal with a mean age 41 years (range 28-56), 14 were post menopausal without HRT with mean age 61 years (range 52-73) and the 14 in the postmenopausal group with HRT had a mean age of 60 years (range 53-68).

	Control		**GSI**	
	Collagen (%)	Proteoglycans (μg/g)	Collagen (%)	Proteoglycans (μg/g)
Premenopausal	51.2 (± 14)	8.6 (± 0.3)	38.4 (± 11) *	9.6 (± 0.6) †
Postmenopausal No HRT	68.8 (± 18)	8.3 (± 0.5)	60.2 (± 13) *	10.4 (± 0.6) †
Postmenopausal with HRT	61.38 (± 23)	8.7 (± 0.3)	48.1 (± 13) *	9.6 (± 0.4) †

* Collagen in the GSI groups was lower than controls ($p<0.001$), postmenopausal women had higher collagen concentration ($p<0.001$) and HRT caused a reduction in collagen content ($p=0.017$).
† Poteoglycan levels were higher in the GSI group throughout ($p<0.001$). Both groups reacted differently to the menopause ($p=0.004$) and to additional HRT ($p=0.007$).
The protein content of the tissue was lower in the GSI group except in the postmenopausal group when the GSI group fell and control group incresed however, this difference was significant ($p=0.02$).

Conclusion
We have confirmed previous findings that: GSI is associated with a reduction in total collagen, of premenopausal vaginal skin when compared to controls. In addition we have shown this reduction in collagen is also present between postmenopausal women and controls and verified that oestrogen therapy produces a fall in collagen in postmenopausal women. Our findings, although surprising, suggest that the reduction in total collagen associated with HRT may actually indicate an attempt, by the tissue, to return to its premenopausal collagen state. The collagen and elastin fibres of the ECM are embedded in an amorphous ground substance, of which proteoglycans are a constituent, which is essential for tissue organisation. The increase in proteoglycans, and associated reduction of collagen, in the GSI group may indicate a dilutional effect within the tissue. Interestingly, the ground substance in both groups appears to react differently to HRT possibly demonstrating an alteration in metabolism between the two groups. We intend to present additional findings, using indicators of collagen metabolism, to explain the changes seen between these groups.
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Title (type in CAPITAL LETTERS, leave one blank line before the text):
ELECTROMYOGRAPHIC STUDY OF THE STRIATED URETHRAL SPHINCTER IN TYPE 3 STRESS INCONTINENCE: EVIDENCE OF MYOGENIC-DOMINANT DAMAGES

Aims of Study: Recently, several studies have reported evidences of partial denervation in the pelvic floor muscles in Stress incontinence(SI) patients, using electrophysiological and histomorphological methods. These results suggested that the denervation in the pelvic floor muscles possibly caused by childbirth vaginally is one of the etiological factors of type 2 SI accompanied with urethral hypermobility. Whereas, an etiology of another type of SI, that is type 3 SI considered due to intrinsic sphincteric deficiency remains unclear. We determined electromyographic features of the striated urethral sphincter in type 3 SI patients and evaluated the correlation of electromyographic changes with the clinical findings.
Methods: We performed electromyography(EMG) of the striated urethral sphincter muscle and urodynamic studies including Valsalva leak point pressure measurement in a total of 51 women, 41 female patients with type 3 SI due to intrinsic sphincteric deficiency(ISD) and 10 women with normal urinary control(NUC). EMG parameters evaluated were duration, amplitude, and number of phases in individual motor unit potentials(MUP) of the striated urethral sphincter at rest, and also an interference pattern at maximal voluntary contraction.
Results: In patients with SI, mean values of MUP parametersü}SD were duration 4.2ü}0.9 msec., amplitude 299.9ü}112.0 mV. and number of phases 4.1ü}0.7. The values in women with NUC were 5.2ü}0.3 msec., 428.2ü}25.6 mV., and 3.4ü}0.4, respectively. The MUP of SI patients showed significantly shorter duration (p=0.0014) , lower amplitude (p=0.0008) and larger number of phases (p=0.0022). 30 (73%) of the SI patients showed an obvious low amplitude (<350 mV)/short duration(<4.5 msec.)/polyphasic pattern and early recruitment of interference activity with low amplitude at voluntary contraction of the striated sphincter, both indicating existence of myogenic damages. These patients showing myogenic damages had significantly lower Valsalva leakpoint pressure (p<0.0001) and more leakage in pad weigh test (p=0.0101), compared with the SI patients without myogenic damage findings.