

Etiology of POP is multifactorial. Most of them are well known. Pelvic floor traumas as provoked by pregnancy and vaginal delivery are very important. They are responsible for tissue elongation, nerve and vessel damage, elastic tissue breaks. Postmenopausal atrophy of the pelvic floor tissues is another well-demonstrated factor, frequently destabilizing a pre-existing injury. Obesity and chronic bronchial obstructive disease increase the risk of prolapse.

One of the main risk factors for POP is the quality of the connective tissue in the pelvis and the perineum. Many series are now available, assessing samples of uterosacral ligaments, vaginal tissue from the apex, from the anterior wall, from the Paraurethral position. Significant modifications are pointed out. For the apex, smooth muscle cells and collagen III as well as active matrix metalloproteinase 9 (MMP 9) concentrations are raised in POP [1, 2]. For the uterosacral ligaments, collagen density, collagen III and Tenascin concentrations are raised in POP with a decrease in Elastin [3–5]. Only one series [6] finds no significant difference in uterosacral ligaments as in vaginal tissue. But the exact site of vaginal tissue sampling is not described in the paper. There are different variations in apex tissue and uterosacral ligaments compared to anterior vaginal wall or paraurethral tissue, where collagen III, I and VI concentrations, Vitronectin expression and extra cellular matrix density are reduced in POP [7–10]. Another publication reveals reduced amount of smooth muscle cells in the round ligaments of patients with POP [11]. All authors insist on the alterations of the extra cellular matrix of the pelvic floor connective tissue associated with decrease of smooth muscle cells. The tissue of the fascias and the ligaments is less elastic and more breakable.

The real question is: are these changes aetiology or consequence of the POP? Three publications tend to underline the primary weakness of the collagen in POP. A recent study [12] has shown positive correlation between low bone densitometry and pelvic organ prolapse (POP). As we know that osteoporosis is first a disease of the collagen matrix of the bone, especially of its turnover, we can imagine a similar mechanism for POP. Furthermore, a review upon SERM in 2006 [13] shows very diverse effects of SERM on POP: Some, like Raloxifen, are protective while some

others have been retrieved from trials because of their POP inducing effects. Knowing that SERM are modifying the expression of several genes involved in collagen turnover and extra cellular matrix integrity, we can argue that a dysfunction of these genes leads to connective tissue breakdown and POP. Another paper has established a strong association (OR3.12, $p < 0.05$) between two connective tissue disorders: striae and POP [14].

It appears that genetically induced bad connective tissue or premature ageing of this tissue may be the “primum movens” of POP. But we all know that small prolapses are likely to regress in time. Handa in 2004 has confirmed these data [15]: regression rate for grade 1 prolapses after 2–8 years is 23.5% for cystocele, 22% for rectocele and 48% for uterine prolapse. Progression rates are only 9.5%, 13.5% and 1.9% respectively. This means that many women undergo vaginal distension and distortion at the time of the delivery, but most of them are able to repair properly their tissues. Furthermore, undergoing the same mechanical stress, different patients may have various degrees of pelvic floor tissue injuries. The degree is correlated to the elasticity and the resistance of their connective tissue.

To summarize, we can say that POP is due to distension and/or disruption of weak, fibrous and inelastic connective tissue possibly associated to decreased ability to repair it. Thus, a correct repair should fix the anatomic disruptions or distensions and improve the quality of the supportive tissue.

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