RESTORING THE UROTHELIAL BARRIER IN BLADDER PAIN SYNDROME.

Hypothesis / aims of study
An important feature and a possible cause of interstitial cystitis/bladder pain syndrome (IC/BPS) is a decreased urothelial barrier. This makes it possible for urine constituents to leak into the bladder wall which can cause severe chronic inflammation leading to symptoms as bladder pain, dysuria and voiding frequency as seen with BPS. A substantial part of the urothelial barrier is the glycosaminoglycan (GAG) layer. Of these GAG’s, chondroitin sulphate (CS) seems to be most contributing to the barrier and is absent on the urothelium in patients with BPS.
It is generally believed that exogenous CS instilled intravesically could improve the urothelial barrier. This method is currently applied in the clinic although the scientific evidence for its efficacy is scarce. In this study, CS was evaluated using an in-vitro model.

Study design, materials and methods
Primary porcine urothelial cells were cultured on 6 Transwell inserts over which the Trans Epithelial Electrical Resistance (TEER) was measured. This is considered a measure for the barrier function. The cells were driven into differentiation to form a tight urothelium with a high resistance. The inserts were divided in 3 groups to evaluate different instillations. The groups were as follows with n=2 in each group: negative control, protamin and protamin & CS. The cells were treated with protamin to damage the top layer and mimic the situation in BPS with a deficient barrier. In the protamine & CS group this was followed by addition of 0,2% CS to the medium. After this the TEER was measured.

Results
After instillation of protamine for one day the TEER drops. After addition of CS the TEER rises most in the protamine+CS group after another day. The graph shows the percentual change in TEER on day 2 after CS was added to the last group. The difference in percentual TEER change between the protamine and protamine+CS group was not significant (P=0,338).

Interpretation of results
The negative control showed hardly any change in TEER as expected. The difference was not statistically different but we used only 2 inserts per group which could explain this.

Concluding message
We need to perform this experiment on a larger scale to see if we can provide additional evidence for the hypothesis tested in this study. Possibly, CS has a beneficiary effect on the recovery of barrier function in damaged urothelium.

Disclosures
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