362

Makedonsky I
Children's Hospital, Dnipropetrovsk, Ukraine

DETRUSOR NEURONAL MATURATIONAL DELAY IS A POSSIBLE COURSE OF INCONTINENCE IN THE BLADDER EXSTROPHY PATIENTS

Aims of Study

Unfortunately, not all exstrophy closures results continence. The factors responsible for the morphology and autonomic innervation changes in bladder exstrophy patients are not determined.

Some patients have a small noncontractile bladder, others an adequate capacity bladder may not contract normally. Innervation of the detrusor determines its ability to contract. The availability of specific immunostains allows a better differentiation of the neural elements in tissue specimens. The formation of synapses requires a series of steps including the generation of neurons and their target cells, the guidance of axons to their targets, and the induction of a specialized presynaptic terminal and postsynaptic membrane. Our study focused on investigation of the formation of neuromuscular synapses in exstrophic detrusor.

Methods

Biopsies were obtained from the anterior wall of 12 exstrophic bladders at the time of initial closure and compared with 12 bladder tissue samples obtained from patients with vesico-uretheral reflux were used as a control. Patient age at bladder closure ranged from 1 to 90 days (mean 19). Tissue samples were investigated immunocytochemically and histochemically. Microscopic fields were sequentially examined with a morphometric system comprising a microscope, video camera and personal computer with a video frame grabber. Specimens were formalin fixed and paraffin embedded. The average number of immunostain material per field was compared between the exstrophic bladders and normal controls. The proliferation cell nuclear antigen (PCNA), neurospecific enolase activity (NSE), S-100 protein, epidermal growth factor (Her 2, neu), tissue matrix ratio were examined.

Results

Compared to control the exstrophy bladders had a significant increase in collagen 44,2% versus smooth muscle 31,4% ($p<0,01$). The differences in tissue matrix ratio were 1,4 in exstrophy group and 0,76 in control group. PSNA was detected in epithelial cells 6% (control -1%), stromal cells 3% (0,2%), smooth muscle cells 0,1% (0,1%). Average number per field NSE and S-100 protein positive material in the exstrophic bladders (12) was significantly reduced compared to normal controls (28, $p< 0,001$). The average number of myelinated nerves per field in the exstrophic bladders (0,21 per field) was significantly lower compared to controls (1,07 per field) and statistically significant ($p<0,001$). Her 2, neu positive material was detected in smooth muscle cells and was significantly reduced compared to controls (21,8%, control-39, 7%, $p< 0,001$).

Conclusions

The present results showed bladder neuronal maturational delay in the exstrophy closure patients which is mostly evident on the level of neuromuscular synapses. There is an indication for histological evaluation of all cases of bladder exstrophy in which reconstructive surgery is to be attempted.