Integrins expression of smooth muscle cells in ureteropelvic junction obstruction

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The ureteropelvic junction obstruction (UPJO) is the most common cause of hydronephrosis. An altered intrinsic malfunction of smooth muscle cells (SMC) is believed to be the underlying mechanism of obstruction. The cytoskeletal structure in SMC in the pathophysiology of UPJO are not intestigated. We have valued the expression of some integrins, talin and β-dystroglycan, that are considered main compound of SMC cytoskeleton. 12 human specimens were obtained during pyeloplasty from children while 6 control specimens were collected during organ explantation. Resected specimens were divided in pelvis, ureteropelvic junction and ureter below the obstruction. An immunofluorescence and a PCR analysis were performed for α7A-, β1A-, α7B- and β1D-integrin, talin and β-dystroglycan. Control reactions showed a strong staining of α7B- and β1D-integrin, talin and β-dystroglycan while a weak positivity of α7A- and β1A-integrin was recorded. Differently, in obstructed UPJ talin and β-dystroglycan were slightly impaired, α7B- and β1D-integrin were severely reduced and α7A- and β1A-integrin were significantly enhanced. Our observations showed a critical alteration of cytoskeleton and these might justify the altered function and the increased apoptosis in SMC of UPJO. A delayed rearrangement of the cytoscheleton of SMC in UPJO might be linked to a post-natal splicing from α7A and β1A to, respectively, α7B- and β1D-integrins. These data perfectly are in accordance with the clinical evidence of a possible post-natal healing of congenital intrinsic UPJO.

Key words
Integrins, smooth muscle, ureteropelvic junction, immunohistochemistry