Arthrocentesis was performed and sterile synovial fluid was found. Magnetic resonance images displayed a bone fragment detachment from the humeral condyle of the right elbow with synovium thickening and persistent effusion: the diagnosis of OCD was pointed out.

The patient showed minor dysmorphismsw (i.e. dolicocephaly, hypotelorism, arched palate and brachydactyly of the IV finger of both hands) and parents reported a previous episode of OCD when he was 12: at that time, symptoms resolved with non-weightbearing and non-steroidal anti-inflammatory therapy after few days. Furthermore, the patient went under regular endocrinologist follow-up for short stature since he was 8. At the age of 10, his height was 123 cm. SDS 2.4, and growth hormone (GH) stimulation tests showed partial response to insulin tolerance test (GH peak 6.27 ng/mL). Bone age at the X-Ray of right hand and wrist was delayed of 12 months. Human recombinant GH replacement therapy was administered without significant growth-velocity improvement. Although the patient came to observation because of suspected elbow septic arthritis, we re-considered the diagnosis: namely, i. recurrent episodes of OCD; ii. short stature that was poorly responsive to the human recombinant GH treatment, iii. mild skeletal and facial dysmorphisms, led us to hypothesize a form of acrogeneopathy.

Molecular analysis of the ACAN gene revealed the novel missense variant c.6970T>C, p.Thr2324Arg in the G3 domain of the protein. Notably, another mutation of the G3 domain (c.7249G>A) has been previously related to aggreca

**REFERENCES**

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**AB0094**

SYNDECAN-4 IS INCREASED IN OSTEOARTHRITIC KNEE, BUT NOT HIP OR SHOULDER, ARTICULAR HYPERPLASTIC CHONDROCYTES

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**Background:** Syndecan-4 plays a critical role in cartilage degradation during osteoarthritis (OA).

**Objectives:** To investigate the expression and localization of syndecan-4 in different OA joint tissues.

**Methods:** Syndecan-4 mRNA levels were quantified by RT-PCR in human OA primary cells to compare non-hypertrophic vs hypertrophic articular chondrocytes, non-sclerotic vs sclerotic subchondral osteoblasts and normal/reactive vs inflamed fibroblasts-like synoviocytes. Syndecan-4 was localized by immunohistochemistry in knee, hip or shoulder OA bone/cartilage biopsies. Syndecan-4 was quantified by immunomassay in chondrocytes culture supernatant and cell fraction.

**Results:** By immunohistochemistry, syndecan-4 was observed in chondrocytes clusters in the superficial zone of OA knee, but not in OA hip or shoulder cartilage. No staining was observed in the deep zone of cartilage and in subchondral bone.

No difference between syndecan-4 expression level in sclerotic and non-sclerotic osteoblasts was observed. Syndecan-4 tended to be increased in inflamed synoviocytes compared to normal/reactive ones but difference was not significant. Differentiated hypertrophic chondrocytes from knee, but not from hip cartilage, expressed more syndecan-4 than non-hypertrophic cells. Using an immunomassay for the extracellular domain of syndecan-4, we found 68% of the syndecan-4 in the culture supernatant of OA chondrocytes culture, suggesting that a large majority of the syndecan-4 is shed and released in the extracellular medium. The shedding rate was not affected by hypertrophic differentiation state of the chondrocytes or their joint origin.

**Conclusion:** Syndecan-4 could be related to the hypertrophic differentiation of the OA chondrocytes, but the pathway seems to be knee specific. Even if chondrocytes clusters are seen in OA knee, hip and shoulder cartilage and hypertrophic differentiation appears in knee and hip OA articular chondrocytes, syndecan-4 synthesis only increased in knee. These findings suggest the presence of biochemical difference between articular cartilage according to their location and that syndecan-4 could be a biochemical marker specific for knee OA.

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