Patellar Osteochondroma: A Unique Presentation

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The patella is an infrequent location for the onset of benign or malignant bone tumors [1]. Although the tumors are usually benign, they require special attention because the patella plays an irreplaceable biomechanical function in knee extension. Osteochondroma is the most common benign bone tumor. It occurs in 3% of the general population [2].

Osteochondroma appears in the physeal plate of long bones during childhood through endochondral ossification. It is thought to result from displacement of the lateral portion of the growth plate in long bones, which then proliferates in a direction diagonal to the long axis of the bone and away from the nearby joint.

The patella is an accessory bone that forms through membranous ossification.

The presence of osteochondroma in the patella in adults is unique because it differs from its usual appearance in physeal plate of long bones during childhood. This can suggest the diagnosis of malignant transformation to a chondrosarcoma.

Osteochondromas are characterized by a highly structured tissue organization with no cellular atypia, as opposed to enchondromas and conventional chondrosarcomas that exhibit random cellular differentiation patterns, which makes histological examination a problematical diagnostic tool [3,4] [Figure 1A, 1B].

The diagnosis of an osteochondroma requires radiological depiction. A usual finding is that of calcified flakes or linear interruptions inside the cartilaginous component of the osteochondroma.

A computerized tomography (CT) scan can depict the bony lesion in detail, the pathognomonic cortical and marrow continuity of the lesion and parent bone, as well as showing the presence of calcifications [4] [Figure 2A, 2B, 2C].

A review of the existing literature revealed three cases of patellar osteochondroma described in the last 5 decades. All were reported as benign lesions [5].

We describe a 47 year old healthy woman who presented with mild pain and a slowly increased swelling on the mediolateral part of her left knee over a 3 year period. During a physical examination, a non-tender hardened swelling with well-defined margins measuring 40 mm × 20 mm × 20 mm was found [Figure 1A]. A full radiological evaluation revealed a benign tumor formation of bone density in the medial side of the patella that was surgically resected, mainly for cosmetic reasons.

Histological examination confirmed its benignity [Figures 2A, 2B, 2C]. No recurrences transpired.

Figure 1. [A] Macroscopic view after resection showing a peduculated base tumor **[B]** Microscopic view of hyaline cartilage cap overlying medullary bone. Meullary bone contains fatty marrow. The junction cap and bone resembles epiphyseal plate

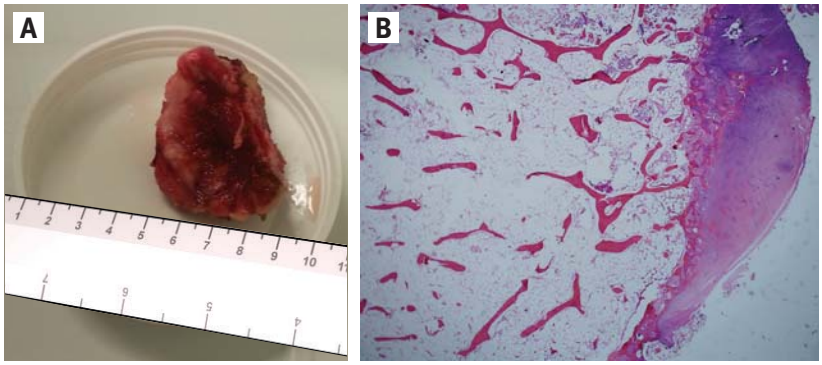
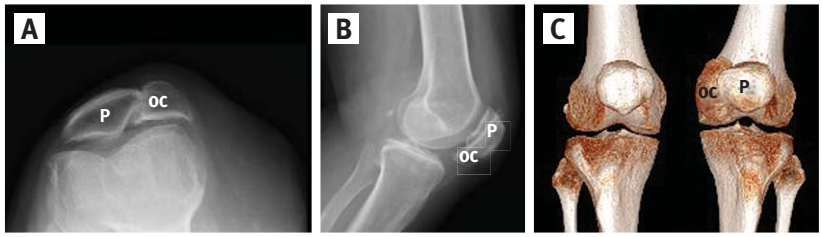


Figure 2. [A] [B] Plain radiographs of the left knee revealing a big tumor of bone density, in the medial side of the patella, **[C]** Three-dimensional computed tomography scan reconstruction of the knee (osteochondroma)



OC = osteochondroma, P = patella

CONCLUSIONS

Patellar osteochondroma is a possible diagnosis for a slow to moderate growing mass on the patella in adults. Its peculiar presentation can be excised en block based on symptomatic or cosmetic basis. We found no recurrences reported in the literature.

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Capsule

Relieving pain with Botox

Chronic pain affects more than 25 million Americans and is associated with reduced life span, anxiety, and depression. Opioid administration is often effective in relieving pain but can cause severe side effects. **Maiarù** and co-authors leveraged the inhibitory effects of botulinum toxin (Botox) on neuronal activity and developed two botulinum-conjugated molecules that silenced pain-related spinal neurons in several mouse models of chronic pain. Intrathecal administration of one dose of either conjugate produced long-term pain relief in the mouse models that was comparable to the effects of opioid treatment. Thus, botulinum-conjugated molecules could potentially provide an opioid-free alternative for treating chronic pain.

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Eitan Israeli

Capsule

Somatic mutations precede acute myeloid leukemia years before diagnosis

The pattern of somatic mutations observed at diagnosis of acute myeloid leukemia (AML) has been well-characterized. However, the premalignant mutational landscape of AML and its impact on risk and time to diagnosis is unknown. **Desai** et al. identified 212 women from the Women's Health Initiative who were healthy at study baseline, but who eventually developed AML during follow-up (median time 9.6 years). Deep sequencing was performed on peripheral blood DNA of these cases and compared to age-matched controls who did not develop AML. The authors discovered that mutations in *IDH1*, *IDH2*, *TP53*, *DNMT3A*, *TET2* and spliceosome genes significantly increased the odds of developing AML. All subjects with *TP53* mutations (n = 21 out of 21 patients) and *IDH1* and *IDH2* (n = 15 out of 15 patients) mutations eventually developed AML in this study. The presence of detectable mutations years before diagnosis suggests that there is a period of latency that precedes AML during which early detection, monitoring and interventional studies should be considered.

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Eitan Israeli

Capsule

A human anti-IL-2 antibody that potentiates regulatory T cells by a structure-based mechanism

Interleukin-2 (IL-2) has been shown to suppress immune pathologies by preferentially expanding regulatory T cells (T_{regs}). However, this therapy has been limited by off-target complications due to pathogenic cell expansion. Recent efforts have been focused on developing a more selective IL-2. It is well documented that certain anti-mouse IL-2 antibodies induce conformational changes that result in selective targeting of T_{regs}. **Trotta** and colleagues reported the generation of a fully human anti-IL-2 antibody, F5111.2, that stabilizes IL-2 in a conformation that results in the preferential STAT5 phosphorylation of T_{regs} in vitro and selective expansion of T_{regs} in vivo. When complexed with human IL-2, F5111.2 induced remission of type 1 diabetes in the NOD mouse model, reduced disease severity in a model of experimental autoimmune encephalomyelitis and protected mice against xenogeneic graft-versus-host disease. These results suggest that IL-2-F5111.2 may provide an immunotherapy to treat autoimmune diseases and graft-versus-host disease.

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