Case Report

An Unusual Case of Pigmented Villonodular Synovitis 14 Years After Total Hip Arthroplasty

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Abstract: Pigmented villonodular synovitis (PVNS) is a rare benign proliferation lesion of the synovium of the joint, bursa, and the tendon sheath. We report here a case of PVNS in a 78-year-old woman 14 years after she underwent total arthroplasty of her right hip. Diffuse PVNS was detected in her right hip during surgery to replace her prosthesis, which had loosened. Macroscopically, the surface of the resected tissue was black and composed of papillae and nodules. Histologically, the tissue consisted of proliferative synoviocytes with black pigment in the cytoplasm. Beneath the synoviocytes were foamy cells. Pathologic analysis confirmed the diagnosis of PVNS with black pigment and the presence of hemosiderin. This indicates that implantation of the prosthesis might have caused the lesion or might have caused its proliferation. Keywords: pigmented villonodular synovitis, arthroplasty, hip, etiology.

Nonpigmented villonodular synovitis sometimes occurs after total knee arthroplasty [1,2]. However, there are no reports of pigmented villonodular synovitis (PVNS) occurring after total arthroplasty. We report here a case of PVNS occurring in a patient 14 years after total right hip arthroplasty.

Case Report

A 78-year-old woman underwent total right hip arthroplasty because of femoral head necrosis. Fourteen years later, she presented to our institution, reporting abnormal prosthesis sounds, without joint pain or swelling, which had persisted for 2 weeks. She had no history of trauma, and her right hip joint appeared normal during physical examination. Two years earlier, she had undergone total arthroplasty of her left hip because of femoral head necrosis. Radiographs revealed that the right prosthesis was loosening. During surgery to replace the loosened prosthesis, the orthopedic surgeon unexpectedly found that the synovium was black and had proliferative papillae and nodules that filled the articular cavity. The hyperplastic synovium was resected. The malpositioned prosthesis was in an extreme varus position and had loosened. The liners were severely damaged.

Macroscopically (Fig. 1), the specimen consisted of pieces measuring 10 × 10 × 2 cm. The surface was black, covered with nodules and both long and short papillae. Below the surface, the tissue was pale yellow. Histologically (Fig. 2), the surface of the tissue was composed of...
synoviocytes with black pigment in the cytoplasm. There were numerous foamy cells and multinucleated giant cells below the surface without black pigment or hemosiderin. The diagnosis was PVNS.

Discussion

Pigmented villonodular synovitis is believed to be closely related to giant cell tumors of the tendon sheath. In recent years, reports have shown that PVNS also occurs in children [3]. Pigmented villonodular synovitis of the zygapophyseal joint has also been reported [4]. Those findings may indicate that PVNS is not limited to the large joints of adults.

Although trauma, inflammation, neoplasia, hemorrhagic effusion, and disorders of lipid metabolism have been implicated as causative factors in PVNS [5-8], the etiology of the disease remains unclear. Kanagawa et al [9] reported a case of local PVNS as a loose body after minor trauma in the knee. Recent cytogenetic studies, however, have provided increasing evidence of a neoplastic origin [8].

Bunting et al [10] reported a case of focal PVNS in the knee presenting 12 months after total knee arthroplasty. He suggested that the disease might have been caused by the surgery, which initiated the proliferation.

Local lesion resection is effective for PVNS. If it recurs locally, radiation therapy may be helpful. Prosthesis cinching, fracture, inflammation, and pressure erosion are the usual complications of prosthesis transplantation. Nonpigmented villonodular synovitis is rare and may be caused by the metal in the prosthesis [11].

Ours is the first report of PVNS occurring 14 years after total hip arthroplasty. We believe that our patient’s disease represents a reaction to polyethylene, metal, and cement wear. Perhaps all of these prosthesis components served as inflammatory factors causing the proliferation of synoviocytes into PVNS. However, the prosthesis might have caused trauma during use of the patient’s right leg that in turn initiated the proliferation of synoviocytes. Implantation of prostheses may be the etiopathogenesis of disease or may cause new disease, just as Bunting et al [10] suspected.

References
