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Retrospective Evaluation of Synovial Chondromatosis with Histopathological and Clinical Features

Sinovyal Kondromatozisin Histopatolojik ve Klinik Özellikleriyle Retrospektif Değerlendirilmesi

Özden ÖZ[1], Murat SEZAK[2], Başak DOĞANAVŞARGİL[2], İpek TAMSEL[3], Hüseyin KAYA[4], Elcil KAYA BİÇER[4], Emin BÜYÜKTALANCI[5], Gülçin BAŞDEMİR[6]

[1]İzmir Democracy University Buca Seyfi Demirsoy Training and Research Hospital, Department of Pathology, İzmir, Türkiye
[2]Ege University Faculty of Medicine, Department of Medical Pathology, İzmir, Türkiye
[3]Ege University Faculty of Medicine, Department of Radiodiagnostics, İzmir, Türkiye
[4]Ege University Faculty of Medicine, Department of Orthopedics and Traumatology, İzmir, Türkiye
[5]University of Health Sciences Türkiye, Başakşehir Çam and Sakura City Hospital, Clinic of Medical Pathology, İstanbul, Türkiye
[6]İstanbul Galata University, Health Sciences Basic Field Medical Pathology, İstanbul, Türkiye

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ABSTRACT

Objective: Synovial chondromatosis (SC) is a rare, benign condition characterized by the formation of nodular cartilage within the synovium of joints, bursae, or tendon sheaths. The condition typically presents as loose body-like nodules, which often exhibit calcification and ossification. SC primarily affects adults and most commonly involves large joints. Histologically, it is defined by synovium-lined cartilaginous nodules displaying varying degrees of peripheral cellularity. Due to overlapping features, SC may be misdiagnosed as a chondroid malignancy, particularly in limited biopsy specimens.

Methods: We performed a retrospective review of cases diagnosed as SC in the Department of Pathology at Ege University Faculty of Medicine between 1986 and 2020. Archived materials were analyzed for epidemiological characteristics, clinical presentation, macroscopic and microscopic histopathological features, and imaging findings.

Results: A total of 114 cases were identified, with a nearly equal gender distribution (male-to-female ratio: 0.95). The median age was 54±16.7 years (range: 18-86 years). The knee was the most affected site (56.14%, n=64), followed by the ankle (14.03%), hip (12.28%), wrist (9.64%), elbow (5.26%), and shoulder (2.63%). Histopathological reevaluation was performed on 62 cases. No correlation was found between histological features and patient age, gender, lesion location, or recurrence.

Conclusion: Most cases involved the knee joint, although rarer sites such as the ankle were also observed. Notably, synovial lining over the nodules was not consistently present. Many nodules exhibited increased cellularity, nuclear pleomorphism, and hyperchromatism-features that can mimic low-grade chondrosarcoma. Histological and cytological features did not correlate with recurrence in our cohort.

Keywords: Joint diseases, synovium, chondromatosis, osteochondromatosis

ÖZ

Amaç: Sinovyal kondromatozis (SK), eklemlerin, bursaların veya tendon kılıflarının sinovyumunda nodüler kıkırdak oluşumuyla karakterize nadir, iyi huylu bir eklem hastalığıdır. Tipik olarak, sıklıkla kalsifikasyon ve ossifikasyon gösteren eklem içerisinde serbest nodüller olarak ortaya çıkar. SK öncelikle yetişkinleri etkiler ve çoğunlukla büyük eklemleri tutar. Histolojik olarak, çeşitli derecelerde özellikle periferik hücresellik gösteren sinovyumla kaplı kıkırdak nodülleri olarak tanımlanır. Çakışan özellikleri nedeniyle, SK özellikle sınırlı biyopsi örneklerinde kıkırdak malignitesi olarak yanlış teşhis edilebilir.

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Corresponding Author/ Sorumlu Yazar:
Özden ÖZ, MD,
İzmir Democracy University Buca Seyfi Demirsoy Training and Research Hospital, Department of Pathology, İzmir, Türkiye
ozdenozpat@gmail.com
ORCID: 0000-0001-5601-1567

Yöntem: 1986 ile 2020 yılları arasında Ege Üniversitesi Tıp Fakültesi, Patoloji Bölümü'nde SK tanısı konulan olguların retrospektif bir incelemesini gerçekleştirdik. Arşivlenen materyaller epidemiyolojik, klinik, makroskobik, mikroskobik, histopatolojik özellikleri ve görüntüleme bulguları açısından analiz edildi.

Bulgular: Toplam 114 olgu tespit edildi ve cinsiyet dağılımı neredeyse eşitti (erkek-kadın oranı: 0,95). Ortanca yaş 54±16,7 (aralığı: 18-86) idi. Diz en çok etkilenen bölgeydi (%56,14, n=64), ardından ayak bileği (%14,03), kalça (%12,28), bilek (%9,64), dirsek (%5,26) ve omuz (%2,63) geldi. Altmış iki olguda histopatolojik yeniden değerlendirme yapıldı. Histolojik özellikler ile hasta yaşı, cinsiyeti, lezyon yeri veya tekrarlama arasında bir korelasyon bulunamadı.

Sonuç: Olguların çoğu diz eklemini içeriyordu, ancak ayak bileği gibi daha nadir bölgeler de gözlendi. Özellikle nodüllerin üzerindeki sinovyal doku daima mevcut değildi. Birçok nodülde artmış hücresellik, nükleer pleomorfizm ve hiperkromazi görüldü; bunlar düşük dereceli kondrosarkomu taklit edebilen özelliklerdi. Histolojik ve sitolojik özellikler kohortumuzda hastalığın tekrarlaması ile ilişkili bulunmadı.

Anahtar Kelimeler: Eklem hastalıkları, sinovyum, kondromatozis, osteokondromatozis

INTRODUCTION

Synovial chondromatosis (SC) is a rare, benign yet locally aggressive neoplastic joint disorder characterized by the formation of multiple lobulated hyaline cartilaginous or osteocartilaginous nodules (chondromas) within the synovial membrane of large joints. Typically originating from synovial connective tissue in adults, SC most often presents intra-articularly, although extra-articular manifestations have been rarely reported. The condition is classified as primary SC when idiopathic, and as secondary SC when associated with pre-existing joint pathology.[1,2] Due to its clinical similarity to other joint disorders, SC is frequently challenging to diagnose accurately.[2-4] Although its etiology remains largely unknown, SC is widely believed to result from metaplasia of synovial connective tissue.[3,5] It most commonly affects individuals in the third to fifth decades of life,[2,3,5,6] with a higher prevalence in males, and predominantly involves large joints, particularly the knee.[2,7]

Clinically, SC poses a diagnostic challenge for orthopedic specialists, as its presentation often mimics other joint pathologies. Patients may experience a prolonged symptomatic period-sometimes extending up to five years-before a definitive diagnosis is established.[4] Common clinical features include joint effusion, tenderness, pain, swelling, crepitus, limited range of motion, and occasionally palpable nodules, resembling osteoarthritis.[2-4,7] Symptoms such as pain at rest, pain that worsens with movement, and joint deformity due to synovial hypertrophy are notable. Even when SC is a primary neoplastic or metaplastic lesion, it may present with secondary osteoarthritic changes resulting from mass effect. In both primary and secondary forms, long-term complications frequently include cartilage erosion and progressive joint degeneration.

Malignant transformation to synovial chondrosarcoma is exceedingly rare and is primarily reported in patients with long-standing disease and multiple recurrences.[5,6,8-11] The estimated incidence of malignant transformation in primary SC ranges from 1-5%,[4,6,9,10,12] although this remains uncertain. The highest reported rate was observed in a large orthopedic oncology database, where 5 out of 78 patients with primary SC developed chondrosarcoma.[4]

Recent molecular investigations have identified recurrent gene rearrangements involving fibronectin 1 (FN1) and activin receptor 2A (ACVR2A) as potential drivers of SC pathogenesis.[5,13] In one cohort, the FN1-ACVR2A gene fusion-detected via fluorescence *in situ* hybridization-was implicated in neoplastic transformation.[5] Mutations in IDH1/2, which are commonly observed in malignant chondroid tumors, have not been identified in SC to date.[14]

Definitive diagnosis of SC requires histopathological examination of biopsied or surgically excised tissue, demonstrating cartilaginous nodule formation within the synovium or as free-floating intra-articular bodies.

The objective of this study is to analyze a large surgical pathology database of SC cases to describe the clinical and histopathological characteristics of primary SC.

METHODS

Patient Selection of Synovial Chondromatosis Cases

Archival records of patients diagnosed with SC between 1986 and 2020 were obtained from the Department of Pathology at Ege University Faculty of Medicine. The study included only cases involving large joints-specifically the knee, hip, elbow, shoulder, and ankle. Clinical, demographic, histopathological, treatment, and imaging data available in the reporting system were re-evaluated. Of the 124 cases identified from the records, two cases diagnosed under the age of 18 and eight cases with insufficient clinical, demographic, and histopathological data were excluded from the study. The study was therefore conducted with 114 cases.

Histopathological Examination of Synovial Chondromatosis Cases

Since only slides or paraffin blocks of cases after 2000 could be obtained from the archive, hematoxylin and eosin (H&E) stained slides from 62 cases diagnosed between 2000 and 2019 were retrospectively reviewed and confirmed based on current histopathological criteria. Nuclear pleomorphism (focal vs. diffuse),

cellularity (slight, medium, and high), and the presence of calcification and ossification were assessed. Whether these histological features were statistically associated with clinicopathological findings was investigated using the chi-square test. Cases diagnosed prior to 2000 were evaluated solely through the review of archival reports. The study was approved by the Ege University Medical Research Ethics Committee (decision number: 22-3.1T/61, date: 24.03.2022).

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics v21.0. The relationship between demographic features and mucosal pathological characteristics was evaluated with the chi-square test. Fisher's exact test was used for comparisons of categorical variables. A p value of less than 0.05 was considered statistically significant in all analyses.

RESULTS

A total of 114 cases were identified, with a nearly equal gender distribution (male-to-female ratio: 0.95). The median age was 46.96±15.79 years (range: 18-86 years). The most commonly affected joint was the knee (56.14%, n=64), followed by the ankle (14.03%, n=16), hip (12.28%, n=14), wrist (9.64%, n=11), elbow (5.26%, n=6), and shoulder (2.63%, n=3) (Table 1, Supplementary Figure 1). The distribution of cases by decade is also included in the same table.

Radiologically, several cases showed densely radiopaque bodies within both the synovium and joint space (Figure 1a, c), with nodules generally measuring less than 30 mm. Degenerative joint changes, including osteophyte formation, were frequently observed. Plain radiographs often revealed multiple (Figure 1a) and occasionally single (Figure 1c) intra-articular bodies, when calcified. In cases lacking calcification, intra-articular bodies appeared radiolucent and could be missed. Magnetic resonance imaging (MRI) and computed tomography (CT) proved useful for detecting non-calcified nodules. Degenerative changes on the joint surface were also noted (Figure 1c, white arrow). Arthroscopic images of the case in Figure 1a are shown in Figure 1b, and of the case in Figure 1c are shown in Figure 1d.

Gross examination revealed round or oval loose bodies resembling a joint mouse, ranging from 0.1 to 3 cm in diameter, which can sometimes form mass formations (Figure 2a, b). In patients who underwent synovectomy, these nodules were occasionally embedded within the synovium (Figure 3a, black arrow; Figure 3c; Figure 4a). Histologically, the nodules consisted of cartilage covered by synovium with varying degrees of cellularity (Figure 3a, b; Figure 4b) or consisted of degenerated free cartilage nodules (Figure 2c). Most nodules were composed of chondrocyte clusters embedded within a hyaline cartilage matrix and frequently exhibited central calcification and/or ossification. Notably, increased peripheral cellularity was commonly observed, which could potentially be misinterpreted as a chondroid malignancy on biopsy specimens (Figure 4b).

Among the 62 cases with available follow-up data from 2000 to 2019 (excluding 52 patients for whom clinical follow-up could not be obtained), 21 cases (33.87%) were initially diagnosed as tumoral masses, 29 (46.77%) as SC, and 12 (19.35%) as synovitis. Of these, 56 patients (90.33%) underwent total synovectomy, while 6 (9.67%) were treated by removal of joint mice alone. Recurrence occurred in 4 cases (6.45%), whereas 58 cases (93.56%) remained recurrence-free. No statistically significant correlation was found between histological or nuclear features and lesion localization, or between cartilage cellularity and variables such as age, gender, anatomical site, or recurrence (p>0.05, chi-squared test).

Table 1. Clinicopathologic findings in the synovial chondromatosis cases

Age	46.96+-15.79 (18-86)	
Gender	Women	42.98%, n=49
	Men	57.02%, n=65
Localization	Right	57.01%, n=65
	Left	42.99%, n=49
Site	Knee	56.14%, n=64
	Ankle	14.03%, n=16
	Hip	12.28%, n=14
	Wrist	9.64%, n=11
	Elbow	5.26%, n=6
	Shoulder	2.63%, n=3
The case distribution of year	1969-1979	14.91%, n=17
	1980-1989	14.03%, n=16
	1990-1999	16.66%, n=19
	2000-2009	26.31%, n=30
	2010-2020	28.07%, n=32
Treatment	Remove of joint mass	5.26%, n=6
	Synovial excision	49.13%, %, n=56
	NA	45.61%, %, n=52
Recurrence	Present	3.51%, n=4
	Absent	50.87%, n=58
	NA	45.61%, n=52

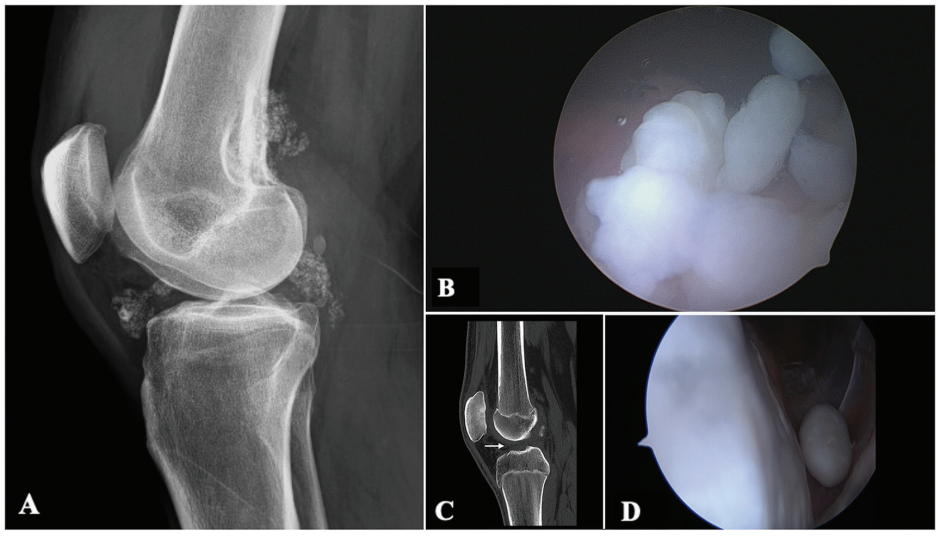

Figure 1. (a) Lateral knee radiograph of a 37-year-old male patient showing multiple well-defined, rounded opacities of similar size confined to the right knee joint space. No bony erosions were present. Concentric calcifications in the form of rings, arcs, and swirls were observed. (b) Arthroscopic view of a case with synovial chondromatosis. (c, d) Arthroscopic and radiological views of the same nodule. The degenerative change of the joint surface is shown in c (white arrow)

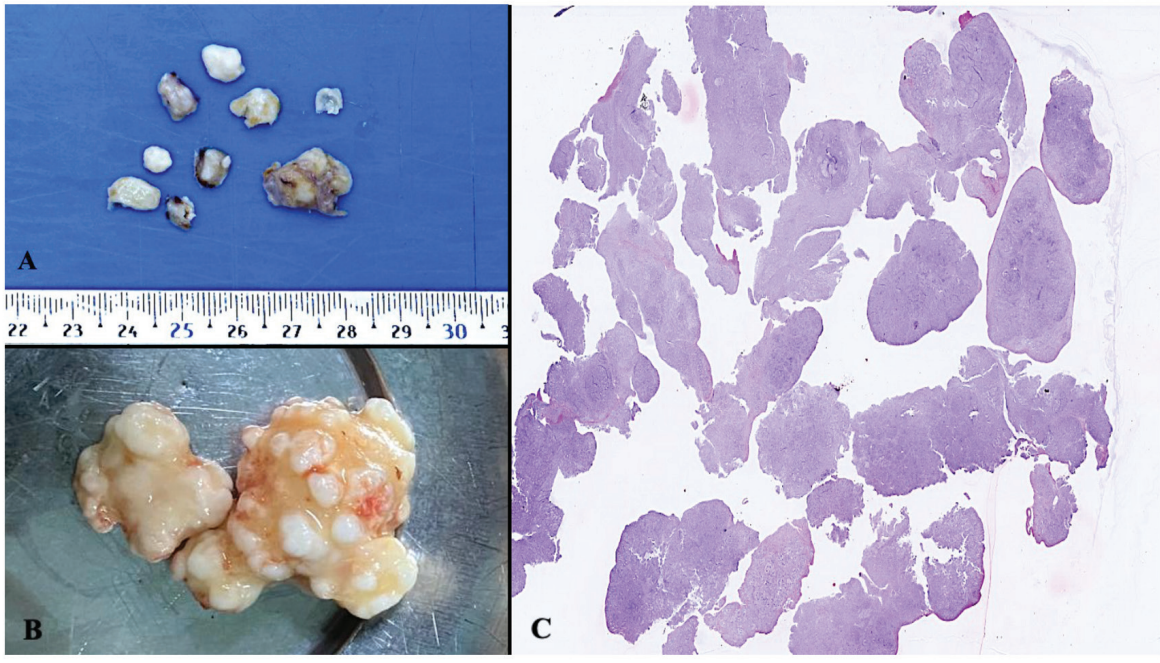

Figure 2. (a, b) Examples of free joint bodies from synovial chondromatosis. Histological confirmation is essential for differentiating these bodies. (c) Histologically confirmed synovial chondromatosis (H&E, ×2)

H&E: Hematoxylin and eosin

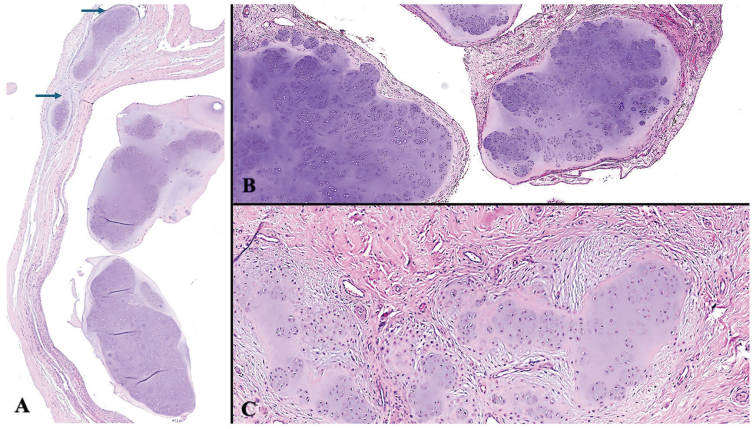

Figure 3. (a, b) Synovial chondromatosis with well-formed nodules in the joint space or just beneath the synovial lining (H&E, ×4 and ×10). (c) Deeper cartilaginous nodules embedded within the synovium (H&E, ×10)

H&E: Hematoxylin and eosin

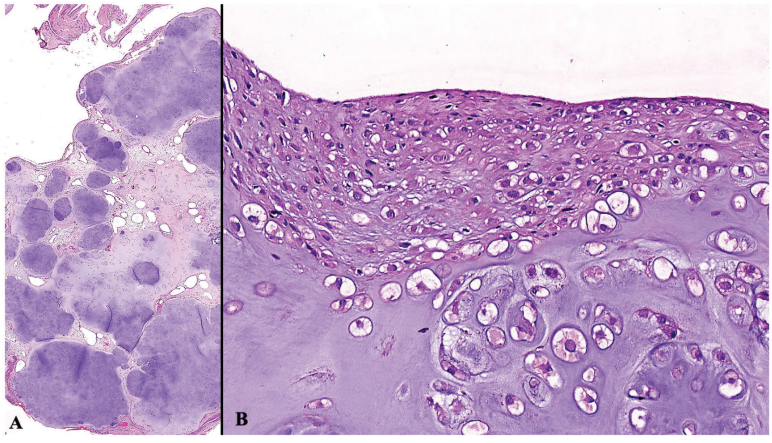

Figure 4. (a) Marked peripheral hypercellularity with lower cellularity in the central regions of the nodules (H&E, ×10). (b) Disorganized peripheral cells with increased cellularity, raising suspicion for chondrosarcoma (H&E, ×20)

H&E: Hematoxylin and eosin

DISCUSSION

SC is a benign neoplastic or metaplastic joint disorder characterized by the formation of multiple cartilaginous or osteocartilaginous nodules within the synovium, tendon sheath, bursa, or joint space. First described by Laennec in 1813 as intra-articular loose bodies originating from subsynovial tissues,[3,4,15] SC was later more thoroughly characterized by Fisher in 1920.[16]

SC predominantly affects large joints, with the knee involved in approximately 70% of cases; followed by the hip (20%), then the shoulder, elbow, ankle, and wrist.[2,4,9] SC is typically monoarticular and can affect any synovial joint. In this clinicopathological review, spanning 34 years, of 114 SC cases, the knee was the most commonly involved joint, consistent with previous literature, although the proportion in our series (56.14%) was slightly lower than previously reported.[2-4,9] Notably, we observed a higher-than-expected incidence in the ankle, diverging from existing literature.[17] While shoulder involvement has been infrequently reported,[10,18] ankle localization is even rarer,[17,19] and there is little to no documented evidence of primary or secondary SC in these locations.[1,18,20] No cases of extra-articular SC were identified in our cohort, contrary to earlier reports,[7] possibly due to incomplete radiological records in some patients.

Approximately 70% of SC nodules undergo endochondral ossification; the remainder, particularly those embedded in the synovium, remain unossified (Figure 3c). These nodules can detach into the joint, where they may undergo mineralization or persist in the synovium. Histologically, some nodules displayed densely packed and disorganized cellular architecture (Figure 3b, 4a). Although nuclear features were generally uniform-with fine chromatin and

small nucleoli-occasional atypical or binucleated nuclei were observed (Figure 4b). We found no association between histological features, including nuclear atypia, and clinical parameters such as age, sex, lesion location, or recurrence in our research.

Radiologically, SC is characterized by intra- or extra-articular cartilage nodules, which may become dislodged into the joint space and are often referred to as "joint mice" (Figure 1a-d). Plain radiographs are typically diagnostic, showing multifocal, rounded, mineralized nodules within or around the joint. However, in roughly 20% of cases, mineralization is not evident due to its time-dependent nature.[3] Non-mineralized nodules may mimic joint effusions, leading to misdiagnosis. In such cases, CT and MRI provide valuable information regarding disease extent and involvement of adjacent structures.[2] MRI findings depend on the degree of synovial proliferation and the presence of calcified or uncalcified nodules.

Milgram[20] proposed a widely accepted three-phase progression of SC: (1) active synovitis without loose bodies; (2) a transitional phase with active synovial proliferation and cartilaginous loose bodies; and (3) multiple ossified bodies with inactive synovitis. These loose bodies may cause mechanical erosion of articular surfaces, contributing to joint damage. Milgram's[20] staging is widely accepted today and supports the view that synovial activity varies throughout the disease course.

Treatment primarily involves the removal of loose bodies to relieve symptoms and prevent joint deterioration. Synovectomy is often employed more extensively than other procedures.[2,3] Treatment strategy and prognosis may vary depending on the affected joint.[2] If untreated, SC can lead to progressive joint and periarticular damage. Therefore, early surgical intervention-via arthroscopic or open removal of loose bodies, with or without synovectomy-is recommended. The necessity of synovectomy remains debated, as each approach carries distinct risks for recurrence and complications.[2,3] While local excision is often curative, recurrence is possible. Malignant transformation (i.e., secondary synovial chondrosarcoma) is considered rare and is usually detected through the recurrence of SC.[2-4,6,7,10] Conversely, it has been demonstrated that two lesions can be detected simultaneously in some patients.[10] Due to the small number of cases, the literature lacks an explanation for the underlying mechanism. Recurrence rates are estimated at 15-20%, especially in tenosynovial variants, which account for 15-20% of all cases.[7,11,20] In our cohort, the recurrence rate was lower than previously reported,[7] which we attribute to the routine practice of total synovectomy. No correlation was found between recurrence and cellularity, ossification, or other histological features. This supports the notion that complete synovial excision significantly reduces recurrence and may prevent malignant transformation.

Once dislodged into the joint, these nodules-"joint mice"-can absorb nutrients from synovial fluid and continue to grow, potentially causing secondary joint surface degeneration. In line with previous studies, nearly all cases in our series featured ossified or calcified nodules.

Histopathologically, SC is characterized by multiple cartilaginous nodules or loose bodies within the synovium. The nodules may be composed of hyaline cartilage with low (Figure 3a, c) or typically high cellular content, particularly at the periphery, and often exhibit cytological atypia (Figure 4b). Features such as multinucleated cells, myxoid matrix generation, nuclear crowding, and pleomorphism were observed. In some cases, the synovial lining over the nodules was not consistently present, and areas of increased cellularity were observed (Figure 3b). If the nuclear pleomorphism and hyperchromasia are evaluated without knowledge of lesion location or radiologic context, they may be diagnosed as low-grade chondrosarcomas.

Primary SC is thought to be more likely neoplastic in origin and typically occurs in the third or fourth decade of life, without identifiable underlying joint pathology (clinically, radiologically, or histopathologically).[3,4,21] In contrast, secondary SC is more common, usually appearing in the fifth or sixth decade and associated with conditions such as synovitis, trauma, osteoarthritis, or neuropathic arthropathy.[2,3,21] Case reports of SC in athletes are also available in the literature and support the repetitive trauma origin.[22] In suspected primary cases, a neoplastic mechanism may be more plausible, while secondary cases are generally attributed to synovial metaplasia due to existing joint pathology. Primary SC tends to feature more numerous and uniformly sized nodules, whereas secondary SC typically involves fewer, larger, and irregularly shaped bodies.[8,23] Some reports estimate that 70-95% of SC cases are primary, while others suggest a higher prevalence of secondary SC.[3] Distinguishing between the two remains a subject of ongoing debate and is often clinically challenging. Regardless of type, SC leads to the formation of multiple chondroid nodules and osteochondral loose bodies, and results in degeneration of the joint space.[2]

Villacin et al.[1] proposed differentiation between primary and secondary SC based on their histopathological characteristics. In our classification, which takes into account the distinction based on histopathological nuclear atypia, we found no association between nuclear atypia and clinical outcomes such as recurrence during the follow-up period. We evaluated the cases histopathologically with

archive record information, and because we could not access the radiological records of all cases pre-2000, it was not possible to determine whether there were underlying degenerative joint findings in our series. For this reason, our inability to distinguish primary from secondary in our cases represents a limitation of our series.

Furthermore, even in presumed primary SC, degenerative changes may arise over time, thereby diminishing the diagnostic value of radiological evidence alone. No significant correlation was observed between histological/cytological features and clinical parameters in our cohort. In conclusion, our findings suggest that the commonly proposed histological and clinical parameters for distinguishing primary from secondary SC-such as the presence of joint degeneration, metaplastic cartilage, cellular atypia, calcification patterns in nodules, ossification type 1, nodule size uniformity, and number or age-may lack diagnostic reliability.[6,8]

Other joint pathologies may also present with loose bodies and synovial proliferation, including crystal deposition diseases, osteochondral fractures, osteochondritis dissecans, neurotrophic arthritis, rheumatoid arthritis, degenerative arthritis, and tuberculous arthritis. Additionally, synovial soft tissue tumors-such as synovial hemangioma, arborizing lipoma, and diffuse-type tenosynovial giant cell tumor-should be considered in the differential diagnosis. While these entities may overlap clinically, they are generally distinguishable by histopathological examination. Although rare, malignant lesions such as intraosseous low-grade chondrosarcoma extending into the joint or synovial sarcoma involving adjacent bone, should also be included in the differential diagnosis. SC is typically non-invasive, with repeated recurrences being the primary concern regarding potential malignant transformation. H&E staining alone is insufficient to distinguish SC from malignancy; clinical history and imaging are essential for accurate diagnosis.[3]

The etiology of SC remains poorly understood and is believed to involve activation of the synovial membrane.[4,6]

It is still unclear whether SC represents a reactive metaplastic process or a true neoplastic transformation.[3,6] Although synovial metaplasia is the more widely accepted mechanism, recent genetic studies have provided evidence suggesting a neoplastic origin.[5,13] If primary SC is considered a neoplastic entity, the identification of genetic alterations, particularly involving the FN1 and ACVR2A genes,[5] in excised SC tissue may help distinguish it from secondary SC, which is currently viewed as a more metaplastic process. ACVR2A encodes a receptor for Activin A, BMP-4, and BMP-6, all of which are involved in skeletal development.[24] Testing for IDH1 and IDH2 mutations may further assist in differentiating SC from chondrosarcoma, a malignant process, either through immunohistochemistry or through identifying the mutated gene. In addition to histopathological and molecular assessments, radiologic evaluation plays a critical role in making this distinction. Together, these tools can guide more aggressive treatment strategies and follow-up protocols for cases that exhibit genetic alterations.

Study Limitations

The inability to apply molecular and genetic tests is a limitation of our study.

CONCLUSION

SC is a rare, benign condition that can lead to significant joint dysfunction. It is widely recognized to occur in both primary and secondary forms, most commonly affecting the knee. Differentiating between these forms remains challenging due to overlapping clinical features and limitations in radiological and histopathological criteria. Early diagnosis and appropriate surgical intervention are critical to preventing disease progression and joint damage.

Although treatment outcomes are relevant, our study did not yield clinical or histological predictors of recurrence. Further research is warranted to elucidate the pathogenesis of SC, particularly the molecular mechanisms underlying both primary and secondary forms. While primary SC may involve neoplastic processes, secondary SC is more often associated with mechanical joint pathology; however, these pathways may intersect. A comprehensive understanding of the clinical, pathological, molecular, and therapeutic dimensions of SC is essential for the effective management of this uncommon but impactful disease.

Ethics

Ethics Committee Approval: The study was approved by the Ege University Medical Research Ethics Committee (decision number: 22-3.1T/61, date: 24.03.2022).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: H.K., E.K.B., Concept: Ö.Ö., M.S., B.D., Design: Ö.Ö., B.D., Data Collection or Processing: Ö.Ö., İ.T., E.K.B., E.B., Analysis or Interpretation: Ö.Ö., M.S., B.D., G.B., Literature Search: Ö.Ö., Writing: Ö.Ö., B.D., G.B.

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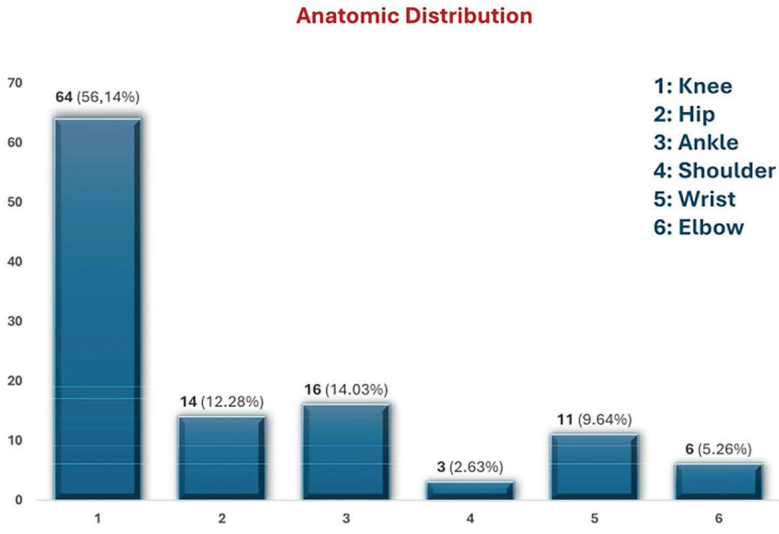

Supplementary Figure 1. The distribution of synovial chondromatosis cases