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Is an intralesional approach a sufficient treatment for solid variant aneurysmal bone cysts in long bones? A case series

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Ethics Committee Approval

The study was approved by the Non-Interventional Research Ethics Committee of Firat University (approval number: 21.03.2025-33231). All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest

No conflict of interest was declared by the authors.

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Abstract

Background/Aim: The solid variant of aneurysmal bone cysts (SVABC) is a rare subtype, representing 3.4–7.5% of all aneurysmal bone cysts, which themselves account for 1.4% of benign skeletal tumors. This study evaluates the outcomes of four patients with SVABC in long bones (three femurs, one humerus), a condition rarely reported in the literature.

Methods: We conducted a retrospective case series analysis of patients who were definitively diagnosed with SVABC through histopathological examination following surgical intervention at our clinic. Inclusion criteria consisted of patients with radiologically identified bone lesions suspected to be SVABC and confirmed by postoperative pathology. Diagnostic workup included detailed imaging with MRI and CT to evaluate lesion morphology, vascularity, and cortical integrity. Surgical treatment involved intralesional curettage, structural allograft bone grafting, and internal osteofixation. Postoperative follow-up included regular clinical examinations and radiographic assessments at 1, 3, 6, and 12 months to monitor healing, detect recurrence, and evaluate functional recovery.

Results: Four patients met the inclusion criteria and underwent surgical management as described. All patients achieved radiological union and demonstrated significant functional improvement, with return to full weight-bearing and daily activity within six months. No postoperative complications, such as infection or hardware failure, were observed. Imaging at one-year follow-up showed no evidence of local recurrence or graft resorption. Functional assessments indicated full range of motion and absence of pain in all cases.

Conclusion: SVABC is a rare benign bone tumor that often mimics other aggressive lesions both radiologically and histologically. Despite its nonaggressive appearance, it requires accurate diagnosis and appropriate surgical management to prevent recurrence. Our findings suggest that thorough preoperative imaging, histopathological confirmation, and a combination of curettage with grafting and fixation can result in excellent clinical and radiological outcomes. SVABC should be considered in the differential diagnosis of solid-appearing bone lesions, particularly those with osteoblastic activity and giant cell—rich histology.

Keywords: aneurysmal bone cyst, solid variant of an aneurysmal bone cyst, long bone, allograft

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Introduction

Aneurysmal bone cysts (ABCs), first described by Jaffe and Lichtenstein in 1942, account for approximately 1.4% of benign skeletal tumors [1]. These rapidly growing, multicystic osteolytic lesions typically affect vertebrae, long bones, or flat bones [1,2]. The solid variant of ABC (SVABC) is even rarer, representing 3.4–7.5% of all ABCs, and often presents in the second decade of life [3,4].

Although benign, ABCs exhibit locally aggressive behavior in 20% of cases and are associated with clonal chromosomal abnormalities, such as the t(16;17) translocation, which activates the TRE17/USP6 oncogene in up to 69% of cases [5]. Their expansive growth can disrupt growth plates and cause pain, swelling, deformities, and pathologic fractures; depending on location, they may also lead to neurologic symptoms [4].

First described by Sanerkin et al. [6] in 1983, SVABC is often misdiagnosed as spindle cell tumors, particularly osteosarcoma, due to overlapping radiographic and histologic features [3,6]. While the pathophysiology of ABCs remains unclear, they are generally considered nonneoplastic reactive lesions linked to intraosseous vascular malformations, trauma, or other bone tumors [1,2,7].

Histologically, ABCs are characterized by blood-filled cystic cavities, osteoclastic giant cells, and fibromyxoid stroma. Radiographically, computed tomography (CT) and magnetic resonance imaging (MRI) reveal expansive osteolytic lesions with fluid-fluid levels, though SVABCs exhibit a more solid pattern with uniform contrast uptake, often resembling giant cell tumors or osteosarcomas [7,8]. Consequently, histopathological analysis remains the gold standard for diagnosis.

SVABCs present a diagnostic challenge due to their variable radiographic and histologic features, including spindle cell proliferation, osteoid formation, and scattered multinucleated giant cells [7]. While symptomatic ABCs are typically treated surgically, asymptomatic cases with minimal bone loss are often monitored [9]. This case series evaluates the outcomes of four patients (three femurs, one humerus) with SVABC in long bones, a condition rarely reported in the literature.

Materials and methods

This retrospective study was approved by the Non-Interventional Research Ethics Committee of Fırat University (approval number: 21.03.2025-33231). The study included patients treated at a tertiary university hospital between January 2020 and January 2024 who were diagnosed with SVABC in the long bones of the extremities.

Case selection

Patients were identified through a comprehensive search of the hospital's pathology database. Inclusion criteria were: (1) histopathologically confirmed diagnosis of SVABC, (2) lesion located in a long bone of the extremities, (3) availability of complete imaging studies including both plain radiography and MRI, and (4) treatment with intralesional curettage, allograft implantation, and osteofixation. Patients with incomplete clinical or imaging data, alternative diagnoses, or less than 11 months of follow-up were excluded. Four patients met these criteria and were included in the study.

Imaging evaluation

All patients underwent preoperative imaging that included standard anteroposterior and lateral radiographs and MRI. Imaging assessments evaluated lesion location (metaphyseal, diaphyseal, or epiphyseal), size (measured in three dimensions), and morphological characteristics such as cystic versus solid content, cortical thinning or breach, and associated soft tissue extension. MRI was also used to detect perilesional edema, fluid-fluid levels, internal hemorrhage, and enhancement patterns. Imaging findings were systematically documented and later correlated with histopathological results to enhance diagnostic accuracy.

Histopathological evaluation

Histological confirmation was essential for inclusion. Formalin-fixed, paraffin-embedded tissue samples were stained with hematoxylin and eosin and re-examined by experienced musculoskeletal pathologists. Diagnostic criteria for SVABC included: proliferation of spindle-shaped stromal cells, scattered multinucleated giant cells, hemorrhagic foci, hemosiderin deposition, fibrocollagenous matrix, and reactive osteoid formation. Absence of significant mitotic activity or cellular atypia helped distinguish SVABC from other giant cell—rich or fibro-osseous lesions. Imaging-pathology correlation was performed to reinforce diagnostic reliability.

Surgical technique

All patients underwent surgery under general anesthesia using a standardized three-step procedure:

- Intralesional Curettage: A cortical window was created to access the lesion. Thorough curettage was performed using curettes and high-speed burrs to remove all gross tumor tissue.
- Cavity Filling with Allograft: The resulting bone cavity was packed with structural cancellous allograft material to restore bone integrity and promote osteoconduction.
- Osteofixation: Internal fixation was performed using titanium elastic nails or plate-screw systems based on the anatomical location and size of the defect. This step ensured mechanical stability and minimized fracture risk.

Postoperative follow-up

Patients were followed clinically and radiographically at regular intervals (1, 3, 6, and 12 months postoperatively). Clinical assessments focused on pain, joint mobility, weight-bearing capacity, and return to function. Radiographs were used to evaluate graft incorporation and bone healing and to detect any signs of recurrence. In selected cases, MRI was repeated at one year to confirm the absence of residual or recurrent lesion. Follow-up duration ranged from 11 to 14 months.

Results

This study evaluated four patients (three males and one female) with an average age of 9 years (range: 7–13 years) diagnosed with SVABC in long bones. Three patients were referred due to pathologic fractures, while one presented with shoulder pain and swelling. None had significant medical histories or systemic symptoms like fever or weight loss. Physical and systemic examinations revealed no additional abnormalities.

All patients underwent radiological evaluations including direct radiographs and MRI (Table 1).

The patient in the first case had a 12-cm lytic lesion in the proximal humerus, showing cystic and solid areas with surrounding edema. Surgical treatment involved curettage, allograft filling, and stabilization with a titanium elastic nail. Histopathology confirmed a mesenchymal tumor with multinuclear giant cells and no mitotic activity. No recurrence was observed at the 11-month follow-up (Figure 1).

The second case had a lytic lesion in the distal femur showing cystic content, hemorrhage, and edema. The patient underwent curettage, allograft filling, and plate screw fixation. Histopathology revealed multinuclear giant cells and stromal cells. No complications or recurrence were noted at the 12-month follow-up (Figure 2).

The third patient had a cystic lesion in the distal femur with hemorrhage and edema. Preoperative biopsy suggested ABCs. Treatment included curettage, allograft filling, and plate screw fixation. Histopathology showed necrotic tissue, osteoclastic giant cells, and new bone formation. No recurrence had occurred by the 14-month follow-up (Figure 3).

The patient in the fourth case had a lytic lesion in the distal femur with cortical thinning, along with fracture. Biopsy confirmed multinuclear giant cells and stromal cells. Treatment involved curettage, allograft filling, and plate screw fixation. No complications or recurrence were reported at the 11-month follow-up (Figure 4).

In all cases, histopathology revealed multinuclear giant cells and stromal cells without mitotic activity. Surgical treatment was successful, with no recurrence or complications during follow-up periods ranging from 11 to 14 months (Table 1).

Figure 1: a: Radiography shows an enlarging lytic lesion with cortical lesions, b: MRI scan shows a tumor with fluid content, c: Intraoperative image shows a solid mass, d: Radiographic imaging in the 1st year of postoperative follow-up



Figure 2: a: Radiograph shows an enlarging lytic lesion with cortical lesions and pathological fracture (white arrow), b, c: MRI scan shows a tumor with fluid content (white arrow), d: Radiographic imaging in the 1st year of postoperative follow-up



Figure 3: a: Radiograph shows an enlarging lytic lesion with cortical lesions and pathological fracture (black arrow), b, c: MRI scan showing a fluid-containing tumor and pathological fracture (black arrow), d: Radiographic imaging in the 1st year of postoperative follow-up



Table 1: Summaries of general data of patients included in the study (age, gender, location, size, treatment, follow-up)

Case	Age/Sex	Location	Presentation	Radiologic Findings	Macroscopic view	Histopathology	Treatment	Follow-Up
1	9/M	Proximal	Pain and	Expansile lytic lesion	$4 \times 4 \times 3$ cm, cream-	Mesenchymal tumor with	Curettage, allograft	No
		humerus	swelling in the	(120 × 25 mm), cystic	brown, hard bone	multinuclear giant cells,	filling, titanium	recurrence at
			shoulder	and solid areas, cortical	consistency, hemorrhagic	no mitotic figures	elastic nail	11 month
				thinning	areas		stabilization	
2	7/F	Distal	Pathologic	Lytic lesion, cystic	$6.6 \times 5.5 \times 2.4$ cm, brown	Multinuclear giant cells,	Curettage, allograft	No
		femur	fracture	content, hemorrhage,	hemorrhagic lesion, off-	stromal cells, no mitotic	filling, plate screw	recurrence at
				edema, cortical loss	white hard areas	figures	fixation	12 month
3	13/M	Distal	Pathologic	Expansile lytic lesion	$6.2 \times 4.9 \times 2$ cm, off-white	Necrotic tissue,	Curettage, allograft	No
		femur	fracture	$(74 \times 40 \text{ mm}),$	to light brown, soft,	osteoclastic giant cells,	filling, plate screw	recurrence at
				hemorrhage, edema,	irregular tissue with	new bone formation	fixation	14 month
				cystic content	bleeding areas			
4	7/M	Distal	Pathologic	Lytic lesion, cortical	$5 \times 4 \times 3$ cm, cream-	Multinuclear giant cells,	Curettage, allograft	No
		femur	fracture	thinning, cystic and	brown, hard bone-like	stromal cells, no mitotic	filling, plate screw	recurrence at
				nodular solid areas	tissue	figures	fixation	11 month

Figure 4: a: Expanding lytic lesion with cortical lesions and pathological fracture on radiography, cortical destruction (white arrow), b, c: Tumor extension with fluid-containing tumor and pathological fracture on MRI scan (white arrow), d: Complete union and no recurrence on radiographic imaging in the 1st year of postoperative follow-up



Discussion

The craniofacial region and the small tubular bones of the hands and feet are the main locations for SVABC [1,6,10-12]. Sanerkin et al. [6] reported four instances of noncystic bone lesions— one cranial bone and three vertebrae—that histologically resembled solid regions of conventional ABCs were documented by.

Referring to these lesions as "solid ABCs," the authors of that study also proposed a tight histologic link between the condition and giant cell reparative granuloma, which typically affects the small tubular bones in the hands and feet as well as the jaw bones. Currently, it is believed that these lesions stem from similar, nonneoplastic reactive processes [10-12]. Because they can be mistaken for giant cell tumors, brown tumors of hyperparathyroidism, and osteosarcoma—all of which are

frequently fibroblastic or low-grade subtypes—these reactive, nonneoplastic lesions have been a challenge for pathologists and treating surgeons [3].

It is uncommon for SVABC to occur in long bones. An uncommon instance of the disease manifesting as a giant cell reparative granuloma in the tibia's subperiosteal region was documented by Kenan et al. [13]. In 1994, Karabela-Bouropoulou et al. [14] reported a rare instance of solid ABC that included both femurs. In a 40-year retrospective review, Ilaslan et al. [7] documented 30 cases of SVABC in long bones. The most common location in these studies was the femur, which occurred in 10 cases; the ulna, tibia, humerus, fibula, and radius were implicated in 7, 7, 2, 2 and 2 cases, respectively. Consistent with the literature, in the present study, three of the four patients had lesions in the femur and one in the humerus.

The metaphysis and metadiaphysis are frequently affected in SVABC. Ilaslan et al. [7] reported juxta-articular involvement in only five cases. Men and women are almost equally affected, and the lesion is most common in the second and fifth decades of life. Yamamoto et al. [15] reported the oldest case, which affected the humerus of a 69-year-old woman, while Ilaslan et al. [7] reported the youngest, occurring in a 2-year-old patient.

Although three of the four patients in the present study presented with pathologic fractures, pathologic fractures from SVABC are not common in the literature [7]. The reason for this may be that this pathology is more common in skull bones and vertebrae than in long bones. While most of the SVABCs described in the literature are red to brown in color, fragile, soft, and have largely blood-filled cavities [16], Yamamoto et al. [15] reported a bronze to white color and hard tissues without cystic cavities. In the present study, the macroscopic images of the cases showed cream—to brown-colored hard tissue with foci of hemorrhage.

In the microscopic images of cases in the literature, tissues have been found to consist of fibroblasts, histiocytes, and numerous osteoclastic multinucleated giant cells on a collagenous ground with lymphocyte and plasma cell infiltration. Mitotic figures have been observed in places, but most studies have reported stromal cells devoid of nuclear atypia, absent aneurysmal sinuses, irregularly shaped woven bone, and osteoid deposition in the extracellular matrix in large areas, as well as some fibromyxoid areas [3,13,14,17-20].

In the present study, sections of biopsy specimens showed fibroadipose and muscular tissues as well as almost necrotic tissue fragments with numerous osteoclastic giant cells and hemosiderin accumulation scattered in a loose swirling stroma composed of spindle cells. In between, areas of new bone

formation surrounded by activated osteoblasts were found. The sections showed a mesenchymal tumor with well-circumscribed, fibrocollagenized stroma. The tumor was composed of homogeneously distributed multinuclear giant cells and stromal cells without mitotic figures, similar in appearance to giant cell nuclei. Also, two of the four patients had osteosarcoma in differential diagnoses during histopathological examinations.

SVABC of long bones has an excellent prognosis. Following surgery, our patients' pathologic fractures healed. Following bone unions, marginal sclerosis occurred. We did not find any recurrence in the follow-up data. However, SVABC in the short tubular bones of the hands and feet has been found to have local recurrence rates of 33–50% [21,22].

Although radiological imaging and histopathological diagnosis of SVABC cases present various difficulties, long bone involvement is especially rare. As a treatment option for long bone involvement, aggressive curettage followed by allograft application and internal fixation were found to yield good results in terms of union and absence of recurrence. We therefore recommend the use of curettage followed by allograft application and internal fixation as a treatment option, especially for long bone SVABC presenting with pathological fractures.

The most important limitation of this study is its retrospective nature. The other limitations of our study include the limited number of cases and the lack of a homogeneous age distribution. While SVABC cases are rare, especially in the long bones, comprehensive studies of the management of SVABC should be conducted. In this way, a consensus on treatment can be reached and supported by long-term results.

Conclusion

SVABC is an uncommon form of benign ABC that often has a nonaggressive radiographic appearance. However, diagnostic challenges are exacerbated by perplexing imaging findings, such as intact internal architecture and significant contrast enhancement in MRI. SVABC also shares several histologic characteristics with other solid bone cancers. When a solid-appearing tumor with osteoblastic activity, fibroblastic proliferation, and the presence of giant cell tumor-like areas is observed on a bone tumor, it should be considered a differential diagnosis in histopathology because SVABC is frequently missed radiologically.

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