Journal of Hip Preservation Surgery, 2025, **12**, 11–19
DOI: https://doi.org/10.1093/jhps/hnae040
Advance access publication date: 6 December 2024
Research article

OXFORD

Assessment and management of periacetabular aneurysmal bone cysts—a series of four cases

Reagan S.H. Beyer[1], Quinn Steiner[1], David W. Hennessy[1], Humberto G. Rosas[2], David C. Goodspeed[1], Andrea M. Spiker[1*]

[1]Department of Orthopedic Surgery, University of Wisconsin—Madison, 1685 Highland Ave, Madison, WI 53705, USA and [2]Department of Radiology, University of Wisconsin–Madison, 600 Highland Ave, Madison, WI 53792, USA

*Corresponding author. Department of Orthopedic Surgery, University of Wisconsin—Madison, UW Health at East Madison Hospital, 4602 Eastpark Blvd, Madison, WI 53718, USA. E-mail: spiker@ortho.wisc.edu

ABSTRACT

Aneurysmal bone cysts (ABCs) in the periacetabular region can be challenging to treat because they create unique problems, given their proximity to articular cartilage and a significant weight bearing surface. This case series details the assessment and treatment of four periacetabular ABCs with a review of pertinent current literature. Treatment approaches used include curettage with 6% phenol chemical adjuvant, type III hemipelvectomy, serial image-guided sclerotherapy injections, and in one case, an exostotic lesion was treated with hip arthroscopy.

INTRODUCTION

Aneurysmal bone cysts (ABCs) are benign, highly vascular, locally aggressive, osseous lesions that most commonly occur in adolescents and young adults with 75–90% of cases occurring in patients <20 years [1–6]. ABCs were initially thought to be a reactive process; however, more recent evidence has reclassified this as a benign neoplastic lesion after identification of recurrent chromosomal translocations in 65–70% of cases. *CDH11-USP6* fusion is one of the most frequently identified genetic abnormalities (30% of cases) and additional mutations continue to be described [7–10].

Patients most commonly present with localized pain and/or swelling over the affected region [6, 10]. Given the aggressive nature of ABCs, late diagnosis or lack of treatment may result in bony destruction, pathologic fractures, local recurrences, and the very rare potential for malignant transformation [1, 6, 11–13]. A variety of treatment options are described in the literature with the goal of promoting healing of cystic cavities, controlling pain, decreasing the risk of pathological fracture, and minimizing deformity. Treatment is chosen on an individual basis after considering patient characteristics, surgeon expertise, available equipment, and the extent and location of the lesion.

Most ABCs occur within the long bones of the upper and lower extremities or the spine [14–16] but pelvic lesions have also been described [4, 5, 17–20]. Given that pelvic ABCs, especially lesions about the acetabulum, are less common, guidelines regarding the assessment and management of periacetabular ABCs are not well established. Furthermore, periacetabular ABCs create a unique problem for management given their proximity to articular cartilage and a significant weight bearing surface. Here we describe the assessment and treatment of four periacetabular ABCs to help illustrate the challenges associated with the management of this entity.

CASE PRESENTATIONS

Patient 1

A 16-year-old female presented with a 2-year history of intermittent right pelvic pain following a basketball injury. Palpation of the right pubic ramus elicited pain. Radiographs and subsequent computed tomography (CT) imaging of the pelvis revealed an expansile, multilobulated lytic lesion involving the medial acetabulum with extension into the superior pubic ramus on the right measuring 5.1 × 3.2 × 6.2 cm (anteroposterior × transverse × cranial-caudal respectively; Fig. 1a and b). Magnetic resonance image (MRI) demonstrated a multilobulated cystic lesion with characteristic fluid-fluid levels and septal enhancement consistent with an ABC (Fig. 1c). The patient underwent open surgical resection with intralesional curettage, high-speed burring, and chemical adjuvant (6% phenol) therapy. The patient was supine on the operative table with the plan for an anterior intrapelvic approach (Stoppa window). After initial exposure, the decision was made to add an extended total ilioinguinal approach to use the middle window for proper orientation for curettage of the lesion while staying between the acetabulum

Received 7 May 2024; revised version accepted 4 November 2024

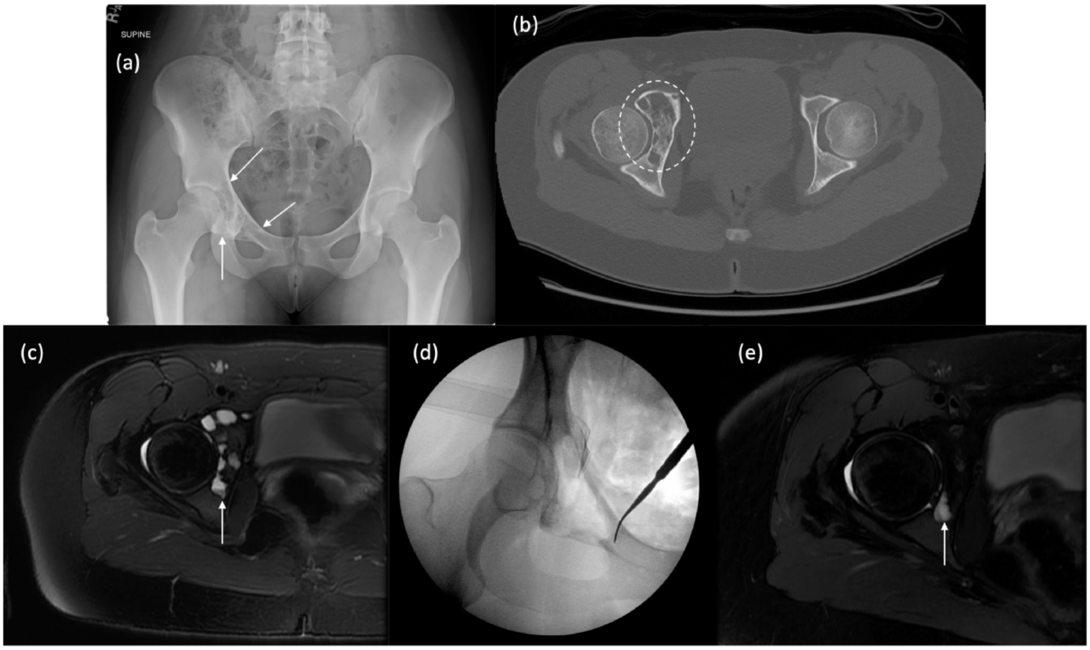

Figure 1. Preoperative and postoperative imaging of the Patient 1's right hip. (a) Preoperative supine AP pelvic radiograph demonstrating an expansile, multilobulated lytic lesion involving the medial acetabulum and extending into the superior pubic ramus (arrows). (b) Preoperative axial CT scan demonstrating a multiseptated lesion involving the medial acetabulum (encircled). (c) Preoperative T2 axial MRI demonstrating a multilobulated cystic lesion with fluid–fluid levels (arrow), favoring appearance of an ABC. (d) Intraoperative fluoroscopic image showing resection of the lesion prior to filling the defect with bone graft. (e) Postoperative T2 axial MRI at the 2-year postoperative interval showing a residual cystic component measuring 1.4 cm in the AP dimension (arrow).

and quadrilateral surface. Incisions for the two approaches were connected. Corona mortis vessels were small and ligated with electrocautery. Obturator nerve and vessels were circumferentially dissected and protected, and the ilioinguinal and lateral femoral cutaneous nerves were identified and protected. The iliopectineal fascia was released, femoral nerve and external iliac vessels were identified at the iliopectineal eminence, and the middle window was established between the nerve and vessels. An oval cortical window was made with a pencil tip Lindemann burr, and the lesion was excised using a combination of bone rongeurs and curettes. Intraoperative fluoroscopic images were compared to preoperative imaging to ensure complete resection of the lesion (Fig. 1d). Phenol was applied as local adjuvant therapy. Before closure, surgeons filled the defect with bone allograft and bone marrow concentrate from the patient's iliac crest. Histological results from intraoperative biopsy of the lesion confirmed the diagnosis of ABC. Postoperative recovery was uncomplicated through 18 months when the patient returned with nocturnal right hip pain and increased pain with activity. MRI showed two nonenhancing cystic areas in the medial acetabular wall. These were thought to represent residual cysts of the original ABC rather than a recurrence requiring repeat intervention. MRI demonstrated no change to the nonenhancing cystic areas of the medial acetabular wall 7 months later (Fig. 1e). The joint space was otherwise preserved with no evidence of articular cartilage damage and the patient elected for nonoperative pain management.

Patient 2

A 19-year-old female presented with a 1-year history of progressive, intermittent right hip and groin pain without known injury or trauma. The physical exam was benign. Radiographs demonstrated a large expansile, lytic lesion with associated pathologic fracture in the right superior ramus that extended into the pubic body (Fig. 2a). MRI revealed a T2 hyperintense expansile osseous lesion measuring 6.1 × 2.0 × 3.5 cm with multiple fluid–fluid levels (Fig. 2b). CT-guided core biopsy confirmed a diagnosis of ABC. The patient underwent a CT-guided sclerosing injection with 80 mg triamcinolone and 200 U of calcitonin. Repeat pelvic radiographs at 3-months demonstrated progressive enlargement with increased lateral extension of the lesion. The patient was then referred for open surgical resection with intralesional curettage and chemical adjuvant (6% phenol) therapy. Partial embolization of the right obturator artery and coil embolization of an anastomotic artery between the obturator and internal pudendal artery were performed the day prior to resection to limit intraoperative hemorrhage. The patient was positioned supine and a Pfannenstiel incision was made right of midline and dissection was carried into the retroperitoneum with caution not to violate the peritoneal space. The rectus was lifted from the periosteum as a musculofascial sheath with the distal attachment intact. Blunt dissection along the superior ramus was carried to the acetabulum and revealed palpable tumor with an adequate window for resection. The bladder was protected, the obturator nerve was identified and protected, the Corona mortis

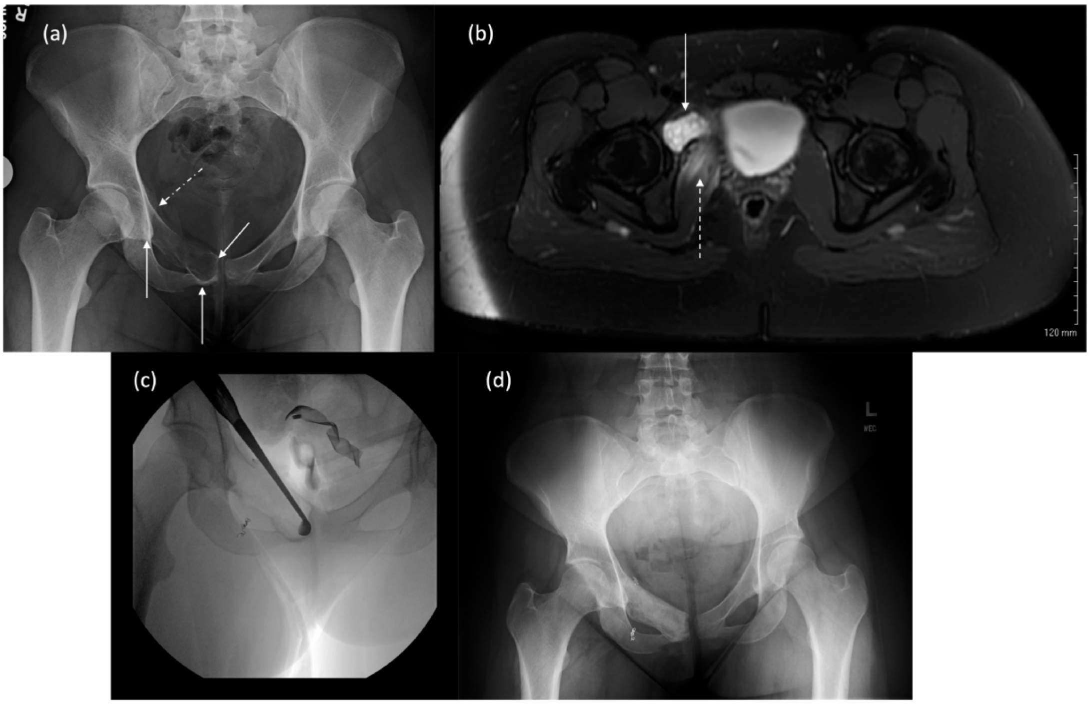

Figure 2. Preoperative and postoperative imaging of Patient 2's right hip. (a) Preoperative standing AP pelvic radiograph demonstrating an expansile, predominantly lytic lesion in the right superior pubic ramus (solid arrows) with an associated pathologic fracture (dashed arrow). (b) Preoperative axial T2-weighted MR image demonstrating a corresponding multiseptated T2 hyperintense lesion (solid arrow) with reactive edema of the adjacent obturator internus and pectineus muscles (dashed arrow) potentially related to either a strain or an occult pathologic fracture. (c) Intraoperative AP fluoroscopic image showing resection of the lesion prior to packing with bone graft. (d) Postoperative AP pelvis radiograph at the 6-month interval showing central bone graft without evidence of recurrence or resorption and preserved joint spaces.

vessel was clipped, and the iliopectineal fascia was released. No cortical window was needed to access the ABC due to existing bony destruction from the tumor along the anterior aspect of the superior pubic ramus. Curettage was performed from anterior to posterior and medial to lateral. No burring was performed due to the presence of only thin cortex and fibrous tissue with multiple defects throughout the superior pubic ramus. Intraoperative fluoroscopy was used to confirm complete resection of the lesion (Fig. 2c). Allograft bone chips were used to fill the defect following curettage and phenol adjuvant therapy. Six-month postoperative radiographs demonstrated complete fill in of the lesion without evidence of resorption or lytic expansion to suggest recurrence (Fig. 2d). Joint space was unchanged compared to preoperative radiographs.

Patient 3

A 14-year-old female presented with 1-week history of intermittent right medial thigh pain that progressed distally into the knee joint. There was no history of trauma or injury to the hip. Clinical exam revealed increased pain with adduction and tenderness to the medial hamstring, mid-distal rectus femoris, iliopsoas, and proximal gluteus medius muscles. A right hip radiograph was unremarkable (Fig. 3a). The patient was referred to physical therapy for a potential adductor brevis or longus muscle tear. MRI performed 1-month later due to increased symptom burden demonstrated synovial enhancement of the right hip joint with robust marrow edema and enhancement in the acetabulum and superior pubic ramus and, to a lesser severity, the femoral head. Hyperintense signal on the fluid-sensitive sequences and enhancement of the right pectineus muscle, adductor magnus, and obturator externus were consistent with myositis without intramuscular abscess (Fig. 3b). Based on these findings, the patient was diagnosed with inflammatory right hip arthritis and admitted to our institution's pediatric hospital due to concerns for progressive septic arthritis and osteomyelitis. Inflammatory markers were within normal limits. Synovial fluid aspiration was positive for few Gram-positive cocci pairs, rare pleiomorphic Gram-positive rods, and rare Gram-negative rods. The patient was treated for septic arthritis with ceftriaxone and daptomycin until 16s ribosomal RNA sequencing returned a negative result. CT imaging performed 2 weeks after discharge revealed a bony excrescence about the acetabular fossa in the region of the right femoral fovea with mild sclerosis in the inferior right acetabulum (Fig. 3c). Osteochondroma was the leading differential diagnosis based on the CT images. Right hip arthroscopy for treatment of a previously identified labral tear and debridement of the newly identified exostosis was performed. The lateral portal was first established via the Seldinger technique and hemarthrosis was observed immediately upon introduction of a spinal needle into the joint. A modified mid-anterior portal was created and the interportal capsulotomy performed, both under direct visualization. In the acetabular fossa (Fig. 4a), the ligamentum teres appeared torn and friable. Once the ligamentum teres was debrided, the exostosis was apparent in the anterior and

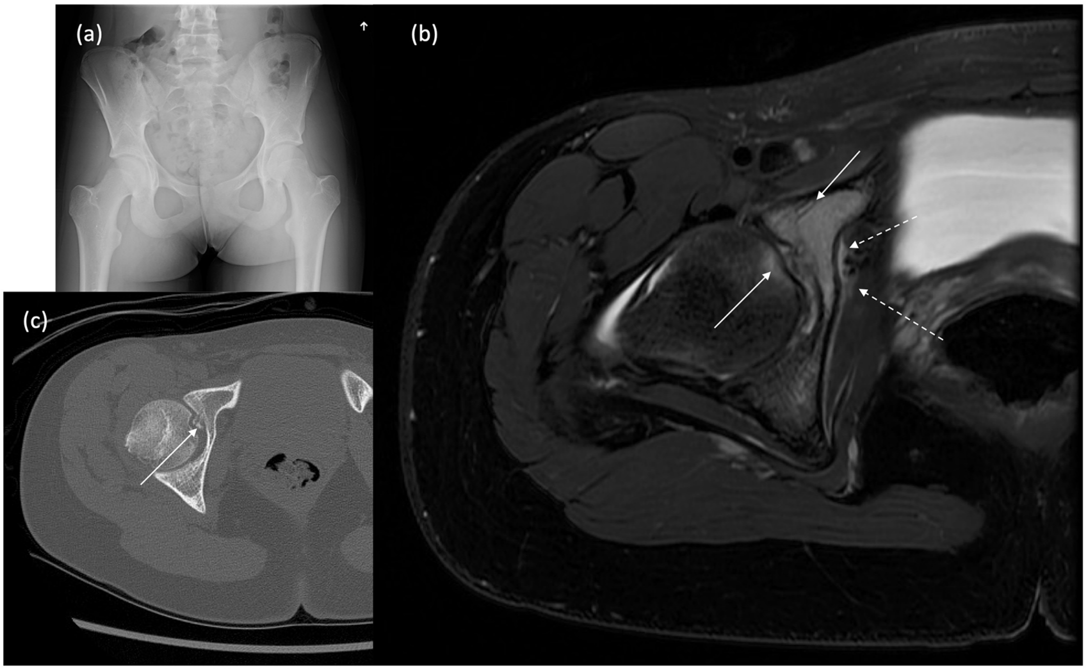

Figure 3. Preoperative imaging of the patient 3's right hip. (a) Unremarkable supine AP radiograph of the pelvis. (b) Post contrast axial T1-weighted MR image demonstrating enhancement of the right acetabulum and pubic root (solid arrows) with reactive changes of the adjacent musculature (dashed arrow), in addition to synovitis and mild subchondral enhancement along the femoral head corresponding to the areas of edema on the fluid-sensitive sequences (data not included). Overall, the findings and distribution were initially concerning for infection. (c) Axial image from a CT scan performed 2 weeks later showing a bony excrescence versus sequela of a subacute/chronic infection versus a cortically based lesion (osteoid osteoma or chondroma) or fracture of the medial right acetabulum (arrow). Subsequent biopsy of this area was shown to represent an ABC.

inferior region of the fossa (zone 1; Fig. 4b), but its consistency was softer than the surrounding bone. This excess tissue was removed with curettage and burring, followed by radiofrequency ablation to the region (Fig. 4b). Chemical adjuvant therapy was not performed given the intra-articular nature of the pathology, and more importantly, because ABC was not part of our pre- or intra-operative differential. Labral pathology was addressed with labral advancement and repair with the assistance of an accessory lateral portal. Intraoperative dynamic range of motion under direct visualization revealed good suction seal of the labrum and appropriate motion without femoroacetabular impingement. Postoperative pathology favored diagnosis of ABC with superimposed fracture callus. Additional genetic testing of lesion cells identified USP6 gene rearrangement and confirmed the ABC diagnosis. Primary versus secondary ABC could not be identified from the pathology samples. Presentation with significant bone and soft tissue edema surrounding a cortically based lesion is more consistent with chondroblastoma and secondary ABC. MRI performed 7 months postoperatively demonstrated no osseous regrowth in the region of the cortical lesion and resected ABC (Fig. 4e) [21].

Patient 4

A 20-year-old male presented with 1-month history of left anterior hip pain that increased with ambulation and weightbearing. The patient had no significant past medical history and symptoms started secondary to gait alteration following a Lisfranc injury of the left foot. Flexion of the left hip elicited pain during clinical exam. Radiographs revealed a predominantly lytic expansile lesion of the left superior pubic ramus with endosteal scalloping and cortical thinning (Fig. 5a). Subsequent MRI arthrogram demonstrated a multiseptated, slightly expansile, predominantly T2 hyperintense lesion ($3.0 \times 5.3 \times 2.8$ cm) of the left superior pubic ramus consistent with an ABC. Repeat radiographs performed ∼2 months later showed significant progression of the lesion (Fig. 5b and c) with increased lucency and size to $6.0 \times 6.5 \times 3.5$ cm. The patient was indicated for open surgical resection given the rapid progression. The index procedure was converted to an open biopsy to rule out malignant etiologies due to significant edema and adhesions, rapid destruction of bone, and a greater than expected amount of bone matrix within the lesion encountered upon exposure. ABC diagnosis was confirmed via histology, and modified type III internal hemipelvectomy (i.e. resection of the superior pubic ramus) was performed the next day. The Pfannenstiel incision from the biopsy was combined with an ilioinguinal incision to gain access to the middle window of the ilioinguinal approach. Vascular surgery performed exposure of the left internal iliac artery and vein common femoral artery and vein due to extensive scarring. The superior ramus was isolated and removed by osteotomes with care to protect the bladder. Residual pubic symphysis and the quadrilateral plate were treated with adjuvant therapy, including argon beam coagulation and high-speed burring under fluoroscopic guidance and attention to the obturator nerve and previously identified neurovascular structures. General surgery performed reconstruction of the abdominal wall

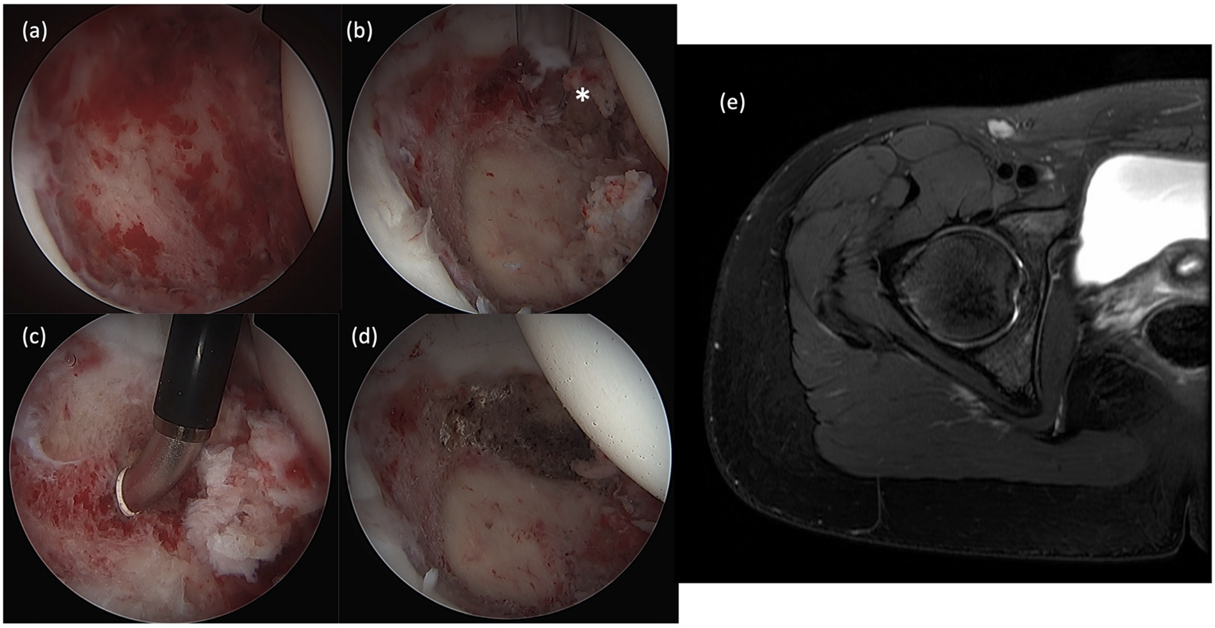

Figure 4. Intraoperative hip arthroscopy and postoperative MRI for Patient 3. In all intraoperative images, femoral head cartilage is visible on the right, with the acetabulum on the left. The anterior acetabulum is oriented at the top of the images. (a) View of the acetabular fossa upon initial inspection of the joint after clearing hemarthrosis. (b) View of acetabular fossa once the residual ligamentum teres was removed with a shaver with the exostosis marked by an asterisk. (c) Radiofrequency ablation of the body exostosis was performed with a curved, hip-length arthroscopic radiofrequency ablation device. (d) Articular surface of the acetabulum following curettage, burring, and radiofrequency ablation of the bony exostosis. (e) Postoperative MRI at the 7-month interval with resolution of the previously seen bone marrow edema and synovitis.

musculature via attachment to the adductor muscle complex and reinforcement with mesh. Recovery was uncomplicated through 4 months when ABC recurrence within the left superior pubic root with extension into the medial acetabulum was identified on MRI (1.9 × 1.8 × 3.3 cm; Fig. 5d). The recurrence was treated with three serial CT-guided cyst aspirations in addition to sclerotherapy with steroid and calcitonin injection (40–80 mg triamcinolone, 200 IU calcitonin) into the remaining cystic cavity performed over a 1-month period. The patient also received monthly injections of denosumab. There is no further evidence of disease recurrence, and the patient remains asymptomatic at last follow-up ∼4 years after the open resection (Fig. 5e and f).

DISCUSSION

Here we have described our assessment and management of periacetabular ABCs and the complexity and variation associated with this pathology. In three cases, we correctly identified the ABC preoperatively and treated the patients with satisfactory postoperative outcomes to date. We include the case (Case 3) of an ABC that was not diagnosed on preoperative imaging and only became clear after arthroscopic biopsy. We also include discussion of disease recurrence management using serial sclerosing injections (Case 4). Currently, the clinical assessment begins with patient history, physical exam, and plain radiographs. ABCs often appear on radiographs as lytic, expansile, lobulated lesions with a distinct sclerotic border. Internal septations are an important characteristic; however, they may be difficult to discern on radiographs alone. Cortical disruption may also be seen with aggressive lesions leading to expansion into the overlying soft tissue [6, 12, 22–25].

Additional imaging is specifically useful within the pelvis and acetabulum, as these three-dimensional areas are difficult to assess on plain films alone. MRI provides contrast resolution allowing for improved discernment of lesion expansion, perilesional edema, and internal composition including septations and fluid-fluid levels, which are highly suggestive of ABCs [22, 23, 25–28]. Mahnken *et al.* demonstrated that MRI or radiographs in isolation are acceptable diagnostic tools with sensitivities of 76.4 and 77.8% and specificities of 55.0 and 66.7%, respectively. However, a combination of radiographs and MRI improved sensitivity and specificity of ABC diagnosis to 82.6 and 70%, respectively [22]. CT scans may also be a useful adjunct to identify areas of cortical defects, articular protrusions, and periosteal reactions [6]. They are especially helpful in the setting of periacetabular lesions to guide possible reconstruction or hip preservation decision-making.

Although imaging is essential to evaluation, findings may be difficult to differentiate from other pathologies (e.g. telangiectatic osteosarcoma, giant cell tumor) [10, 29]. Histologic evaluation remains useful in the diagnosis of ABC. One patient in our series underwent preoperative CT-guided core needle biopsy and the sample was sufficient to diagnose ABC based on histology; classic histologic findings include hemorrhagic cysts surrounded by fibrous septa, mitotic spindle cell proliferation with osteoclast type giant cells, and reactive woven bone [4, 6, 25, 30, 31]. Genetic testing of biopsy samples is an increasingly valuable tool as chromosomal translocations, most often in

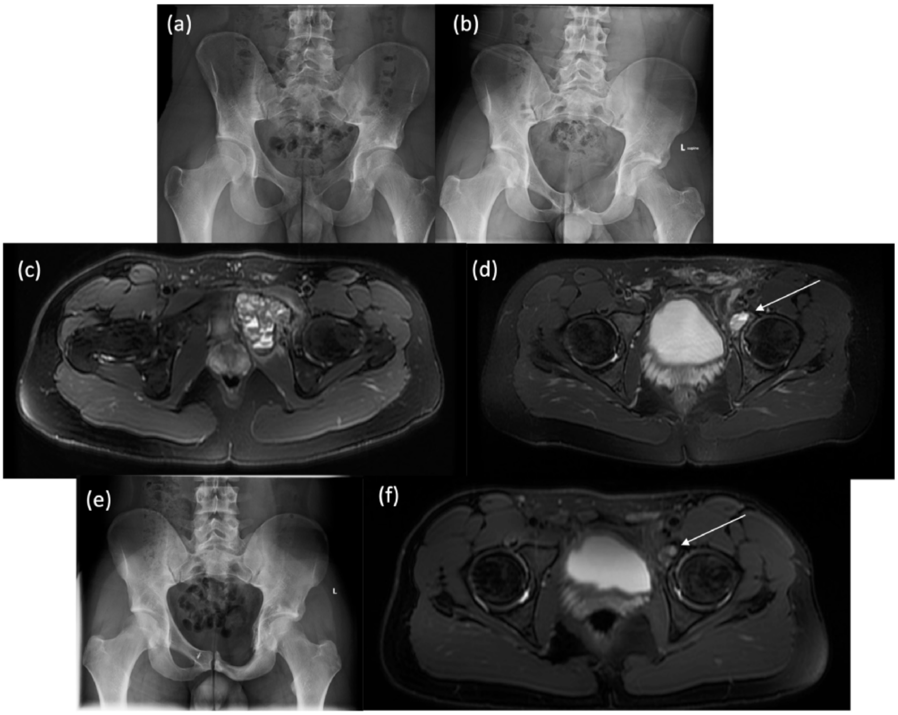

Figure 5. Preoperative and postoperative imaging of Patient 4's left hip. (a) Preoperative standing AP pelvic radiograph demonstrating a lytic lesion in the left superior pubic ramus. (b) Repeat preoperative standing AP radiograph of the pelvis obtained 2 months later demonstrating interval progression with further extension into the pubic body and cortical thinning. (c) Preoperative axial T2-weighted MR image showing an expansile lesion with multiple fluid-fluid levels characteristic of an ABC. (d) Postoperative axial T2-weighted MRI at the 4-month interval demonstrating a small T2 hyperintense lesion with fluid–fluid levels centered at the superior pubic root (arrow) consistent with a recurrence. (e) Postoperative and post sclerotherapy AP pelvis radiograph at the 4-year postoperative interval showing no further evidence of recurrence and preserved joint spaces. (f) Postoperative and post sclerotherapy post contrast axial T1-weighted image showing resolution of recurrence and filling of the lesion (arrow).

the USP6 gene, have been associated with ABCs [8, 9]. These cytogenetic abnormalities are targetable and provide definitive diagnosis [7, 25]. Genetic testing for USP6 rearrangement was essential for diagnosis of our third periacetabular lesion with an atypical presentation and lack of any identifiable radiological findings that raised clinical suspicion for ABC (Case 3).

The mainstay of ABC management is curettage of the cyst cavity and reconstruction of the defect with bone graft as originally described by Jaffe and Lichtenstein [32]. In a review of 11 studies of ABCs (790 patients), curettage with or without bone grafting (or polymethylmethacrylate, PMMA) was the most common treatment strategy (65.6%). Other treatment approaches included en bloc resection and intralesional curettage (16.2%), curettage and radiotherapy (7.7%), radiotherapy alone (2.5%), and embolization or steroid injection (2.4%) [33]. Curettage necessitates complete resection to reduce recurrence, but incomplete curettage is more likely with lesions that are difficult to access surgically, including periacetabular ABCs. Despite attempted confirmation of complete resection via fluoroscopy in this case series, Patient 1 likely had residual cysts that contributed to postoperative pain and represents a limitation of curettage as a treatment strategy.

Although curettage is the primary treatment, recurrence rates vary widely and have been reported in up to 59% of primary ABC cases [33, 34]. A wide range of adjuvants such as use of cement, high-speed burrs, argon beams, phenol, and cryotherapy have been employed in an attempt to reduce recurrence rates [10, 35]. Comparative study of these adjuvants is limited. In our patients who were treated with curettage, chemical adjuvant with 6% phenol was applied after identification of normal-appearing cortical bone. Phenol creates a more significant rim of necrosis than burring alone; however, direct studies found that recurrence did not depend on the use of phenol adjuvant [36, 37]. Notably, high-speed burring could not be used in one patient given the presence of only thin and fibrous tissue with multiple defects throughout the superior pubic ramus. Furthermore, Patient 4 underwent type III internal hemipelvectomy as significant cortical thinning limited the possibility of extensive adjuvant treatment and there was high concern for recurrence with curettage and grafting given the aggressive nature of the lesion. No chemical adjuvant was applied for the intra articular ABC to preserve articular cartilage. Notably, hip arthroscopy is not considered standard treatment for ABC; if ABC were the most likely diagnosis prior to surgical intervention, then surgical hip dislocation would have likely been performed.

In cases of aggressive and recurring lesions, en bloc resection is the preferred treatment progression. Recurrence following en bloc resection is less likely (5.4%); however, the procedure is more technically challenging and carries significant morbidity. Flont *et al.* reported higher rates of pain, limb-length discrepancy, reduced range of motion, and muscle strength impairment in pediatric patients treated with en bloc resection when compared to a similar cohort treated with curettage [38]. In the pelvis, especially periacetabular lesions, en bloc resection is increasingly challenging and may necessitate joint replacement if there is significant involvement of the acetabulum. Patient 4 in our case series was advised that hip replacement would be indicated for definitive treatment if sclerosing injections failed to prevent progression of the ABC within the acetabulum.

More recent advances in the treatment of ABC focus on minimally invasive options such as the use of sclerosing agents or selective arterial embolization to prevent intraoperative hemorrhage associated with open curettage and resection of involved pelvic lesions. At present, no single sclerotherapy treatment is proven superior; therefore, protocols for percutaneous sclerosis are institution-specific [39]. Studies of repetitive sclerotherapy using polidocanol suggest lower recurrence rates (range, 3.2–14%) but include risks of necrosis at the injection site, osteomyelitis, and severe hypersensitivity reactions [13, 17, 40, 41]. Similar results are seen in percutaneous embolization with alcoholic zein (Ethibloc; no longer available for use) and *N*-2-butyl cyanoacrylate, but significant complications are reported including inflammatory reactions, aseptic osteitis, and pulmonary embolism [10, 42–45]. At our institution, calcitonin with steroid is used due to the low complication rate and efficacy of combining the inhibitory angiostatic and fibroblastic effects of the steroid with the trabecular bone-stimulating properties of calcitonin. Despite a technically successful injection in Patient 2, the ABC continued expanding and the patient was indicated for open curettage and chemical adjuvant just 3-months later. Contrastingly, serial CT-guided injection of triamcinolone and calcitonin successfully treated recurrence in Patient 4. These results are consistent with those reported by Chang *et al.* in which 50% of patients ($n = 9$) showed substantial resolution in radiographs following a similar injection protocol without any complications [46].

Furthermore, selective arterial embolization can be used unless the ABC lacks identifiable feeding vessels or share supply with vital tissues and organs [10, 46]. Finally, medical therapy including percutaneous placement of intralesional doxycycline or systemic use of bisphosphonates or denosumab are being studied but remain inconclusive as primary ABC therapies and further clinical studies are warranted [10, 47–51]. Patient 4 underwent systemic denosumab therapy, but we cannot conclude whether this contributed to halting disease progression as the patient also underwent concomitant CT-guided sclerosing injections.

Overall, the current treatment approach to ABC considers surgical management as preferred therapy when the location of the lesion is amenable to resection and reconstruction without significant morbidity. In the cases of periacetabular lesions presented in this series, surgical resection was significantly more complicated and morbid. As such, our institution is now more likely to attempt an imaging-guided procedure (e.g. CT-guided triamcinolone and calcitonin injection) prior to surgical resection if the ABC demonstrates no significant progression over time. For cases where the ABC is highly aggressive (Patient 4) or CT-guided injection fails to limit disease progression and patients require surgery (Patient 2), preoperative embolization is preferred to limit excess bleeding [52, 53]. Patient 4 in this case series did not undergo preoperative embolization and surgeons encountered significant bleeding leading to multiple blood transfusions postoperatively.

Regarding follow-up after primary ABC treatment, there is no standardized procedure. More than 90% of recurrences occur within 6–12 months after treatment, thus most studies recommended frequent surveillance during this interval through 2 years after treatment. MRI is the preferred imaging modality given the lack of radiation exposure and sensitivity for ABC lesions. Disease recurrence for Patient 4 in our series occurred at 4 months postoperatively and was identified on MRI though the patient was asymptomatic. Based on their literature review of prior ABC studies, Hauschild *et al.* recommend clinical survey and MRI at 3, 6, 12, 18, and 24-months with yearly studies until 5 years after initial treatment [33]. Our follow-up with patients involved less frequent monitoring using radiographs or MRI at ∼6 month intervals. If the patient was not experiencing symptoms 9–12 months post-operatively, the surgeon recommended reducing to yearly surveillance.

There are significant limitations to this study given the small sample size at a single institution. We aimed to present different management options for periacetabular ABC but caution that this is not a standardized, comparative study of the different treatments used.

CONCLUSION

Here, we presented four cases of periacetabular ABC diagnosis and management. There are no well-established guidelines regarding the assessment and management of ABCs within the periacetabular region. However, awareness of the available options with an understanding of the complexity of this anatomy as well as maintenance of the load-bearing portion of the acetabulum and articular cartilage is vital to providing quality care and improving outcomes in this patient population.

CONFLICT OF INTEREST

A.M.S. is a paid consultant for Stryker, an Associate Editor for the American Journal of Sports Medicine, on the editorial board of Arthroscopy Journal, Video Journal of Sports Medicine, and the Journal of Hip Preservation Surgery, and is a Board Member of ISHA.

FUNDING

None declared.

DATA AVAILABILITY

The data underlying this article cannot be shared publicly due to HIPAA privacy laws which do not allow the sharing of patient identities or personal information. For further inquiries, please send a reasonable request to the corresponding author.

REFERENCES

1. Leithner A, Windhager R, Lang S *et al.* Aneurysmal bone cyst. A population based epidemiologic study and literature review. *Clin Orthop Relat Res* 1999;**363**:176–79.
2. Cottalorda J, Kohler R, Sales de Gauzy J *et al.* Epidemiology of aneurysmal bone cyst in children: a multicenter study and literature review. *J Pediat Orthopaed B* 2004;**13**:389–94.

3. Vergel De Dios AM, Bond JR, Shives TC *et al.* Aneurysmal bone cyst. A clinicopathologic study of 238 cases. *Cancer* 1992;**69**:2921–31.
4. Papagelopoulos PJ, Choudhury SN, Frassica FJ *et al.* Treatment of aneurysmal bone cysts of the pelvis and sacrum. *J Bone Joint Surg Am Vol* 2001;**83**:1674–81.
5. Capanna R, Bertoni F, Bettelli G *et al.* Aneurysmal bone cysts of the pelvis. *Arch Orthop Trauma Surg* 1986;**105**:279–84.
6. Restrepo R, Zahrah D, Pelaez L *et al.* Update on aneurysmal bone cyst: pathophysiology, histology, imaging and treatment. *Pediatr Radiol* 2022;**52**:1601–14.
7. Pižem J, Šekoranja D, Matjašič A *et al.* The role of molecular diagnostics in aneurysmal and simple bone cysts – a prospective analysis of 19 lesions. *Virchows Archiv* 2021;**479**:795–802.
8. Hiemcke-Jiwa LS, van Gorp JM, Fisher C *et al.* *USP6* -Associated neoplasms: a rapidly expanding family of lesions. *Int J Surg Pathol* 2020;**28**:816–25.
9. Papke DJ, Bredella MA, Lozano-Calderon S *et al.* Aneurysmal bone cyst with an unusual clinical presentation and a novel*VDR* - *USP6* fusion. *Genes Chromosomes Cancer* 2021;**60**:833–36.
10. Park HY, Yang SK, Sheppard WL *et al.* Current management of aneurysmal bone cysts. *Curr Rev Musculoskelet Med* 2016;**9**:435–44.
11. Nasri E, Reith JD. Aneurysmal bone cyst: a review. *J Pathol Transl Med* 2023;**57**:81–87.
12. Mascard E, Gomez-Brouchet A, Lambot K. Bone cysts: unicameral and aneurysmal bone cyst. *Orthop Traumatol Surg Res* 2015;**101**:S119–27.
13. Deventer N, Deventer N, Gosheger G *et al.* Current strategies for the treatment of solitary and aneurysmal bone cysts: A review of the literature. *J Bone Oncol* 2021;**30**:100384.
14. Tillman B, Dahlin D, Lipscomb P *et al.* Aneurysmal bone cyst: an analysis of ninety-five cases. *Mayo Clin Proc* 1968;**43**:478–95.
15. Papagelopoulos PJ, Currier BL, Shaughnessy WJ *et al.* Aneurysmal bone cyst of the spine. *Spine* 1998;**23**:621–28.
16. Lichtenstein L. Aneurysmal bone cyst; observations on fifty cases. *J Bone Joint Surg Am* 1957;**39-A**:873–82.
17. Deventer N, Budny T, Gosheger G *et al.* Aneurysmal bone cyst of the pelvis and sacrum: a single-center study of 17 cases. *BMC Musculoskelet Disord* 2022;**23**:405.
18. Wong KPL, Mahadev A. Rare right acetabulum aneurysmal bone cyst treated with curettage and local adjuvants in a 4-year-old girl. *BMJ Case Rep* 2021;**14**:e245586.
19. Rossi G, Angelini A, Mavrogenis AF *et al.* Successful treatment of aneurysmal bone cyst of the hip in a child by selective transcatheter arterial embolization. *J Vasc Interv Radiol* 2010;**21**: 1591–95.
20. Yildirim E, Ersözlü S, Kirbaş I *et al.* Treatment of pelvic aneurysmal bone cysts in two children: selective arterial embolization as an adjunct to curettage and bone grafting. *Diagn Interv Radiol* 2007;**13**:49–52.
21. Suneja R, Grimer RJ, Belthur M *et al.* Chondroblastoma of bone. *J Bone Joint Surg Br* 2005;**87-B**:974–78.
22. Mahnken A, Nolte-Ernsting C, Wildberger J *et al.* Aneurysmal bone cyst: value of MR imaging and conventional radiography. *Eur Radiol* 2003;**13**:1118–24.
23. Rapp TB, Ward JP, Alaia MJ. Aneurysmal bone cyst. *J Am Acad Orthop Surg* 2012;**20**:233–41.
24. Bonakdarpour A, Levy WM, Aegerter E. Primary and secondary aneurysmal bone cyst: A radiological study of 75 cases. *Radiology* 1978;**126**:75–83.
25. Noordin S, Ahmad T, Umer M *et al.* Aneurysmal bone cyst of the pelvis and extremities: Contemporary management. *Int J Surg Oncol (N Y)* 2019;**4**:e71–e71.
26. Sullivan RJ, Meyer JS, Dormans JP *et al.* Diagnosing aneurysmal and unicameral bone cysts with magnetic resonance imaging. *Clin Orthop Relat Res* 1999;**366**:186–90.
27. Rajeswaran G, Malik Q, Saifuddin A. The role of needle biopsy for focal bone lesions with complete fluid-fluid levels on magnetic resonance imaging. *Skeletal Radiol* 2013;**42**:765–69.
28. Zishan US, Pressney I, Khoo M *et al.* The differentiation between aneurysmal bone cyst and telangiectatic osteosarcoma: a clinical, radiographic and MRI study. *Skeletal Radiol* 2020;**49**:1375–86.
29. Josip E, Kremser C, Haider B *et al.* MRI analysis of simple and aneurysmal bone cysts in the proximal humerus: what actually matters in clinical routine. *Skeletal Radiol* 2022;**51**:819–27.
30. Ayala AG, Zornosa J. Primary bone tumors: percutaneous needle biopsy. Radiologic-pathologic study of 222 biopsies. *Radiology* 1983;**149**:675–79.
31. Docquier P-L, Delloye C, Galant C. Histology can be predictive of the clinical course of a primary aneurysmal bone cyst. *Arch Orthop Trauma Surg* 2010;**130**:481–87.
32. Jaffe HL. Solitary unicameral bone cyst. *Arch Surg* 1942;**44**:1004.
33. Hauschild O, Lüdemann M, Engelhardt M *et al.* Aneurysmal bone cyst (ABC) : treatment options and proposal of a follow-up regime. *Acta Orthop Belg* 2016;**82**:474–83.
34. Dormans JP, Hanna BG, Johnston DR *et al.* Surgical treatment and recurrence rate of aneurysmal bone cysts in children. *Clin Orthop Relat Res* 2004;**421**:205–11.
35. Wang EHM, Marfori ML, Serrano MVT *et al.* Is curettage and high-speed burring sufficient treatment for aneurysmal bone cysts? *Clin Orthop Relat Res* 2014;**472**:3483–88.
36. Bombardier B, Haase D, Sweeney K *et al.* A comparison of depth of necrosis among adjuvant therapies used for the treatment of benign bone tumors. *J Surg Oncol* 2021;**123**:1299–303.
37. Grahneis F, Klein A, Baur-Melnyk A *et al.* Aneurysmal bone cyst: a review of 65 patients. *J Bone Oncol* 2019;**18**:100255.
38. Flont P, Kolacinska-Flont M, Niedzielski K. A comparison of cyst wall curettage and en bloc excision in the treatment of aneurysmal bone cysts. *World J Surg Oncol* 2013;**11**:109.
39. Cruz GS, Cuevas-Suárez CE, Saavedra JPA *et al.* Percutaneous treatments of primary aneurysmal bone cysts: systematic review and meta-analysis. *Eur J Orthop Surg Traumatol* 2021;**31**:1287–95.
40. Varshney MK, Rastogi S, Khan SA *et al.* Is sclerotherapy better than intralesional excision for treating aneurysmal bone cysts? *Clin Orthop Relat Res* 2010;**468**:1649–59.
41. Gupta G, Pandit RS, Jerath N *et al.* Severe life-threatening hypersensitivity reaction to polidocanol in a case of recurrent aneurysmal bone cyst. *J Clin Orthop Trauma* 2019;**10**:414–17.
42. De Cristofaro R, Biagini R, Boriani S *et al.* Selective arterial embolization in the treatment of aneurysmal bone cyst and angioma of bone. *Skeletal Radiol* 1992;**21**:523–27.
43. George HL, Unnikrishnan PN, Garg NK *et al.* Long-term follow-up of Ethibloc injection in aneurysmal bone cysts. *J Pediat Orthopaed B* 2009;**18**:375–80.
44. Guibaud L, Herbreteau D, Dubois J *et al.* Aneurysmal bone cysts: percutaneous embolization with an alcoholic solution of zein—series of 18 cases. *Radiology* 1998;**208**:369–73.
45. Rossi G, Rimondi E, Bartalena T *et al.* Selective arterial embolization of 36 aneurysmal bone cysts of the skeleton with N-2-butyl cyanoacrylate. *Skeletal Radiol* 2010;**39**:161–67.
46. Chang CY, Kattapuram SV, Huang AJ *et al.* Treatment of aneurysmal bone cysts by percutaneous CT-guided injection of calcitonin and steroid. *Skeletal Radiol* 2017;**46**:35–40.
47. Pelle DW, Ringler JW, Peacock JD *et al.* Targeting receptor-activator of nuclear kappaB ligand in aneurysmal bone cysts: verification of target and therapeutic response. *Transl Res* 2014;**164**:139–48.
48. Skubitz KM, Peltola JC, Santos ER *et al.* Response of aneurysmal bone cyst to denosumab. *Spine* 2015;**40**:E1201–4.
49. Lange T, Stehling C, Fröhlich B *et al.* Denosumab: a potential new and innovative treatment option for aneurysmal bone cysts. *Eur Spine J* 2013;**22**:1417–22.

50. Cornelis F, Truchetet ME, Amoretti N *et al*. Bisphosphonate therapy for unresectable symptomatic benign bone tumors: A long-term prospective study of tolerance and efficacy. *Bone* 2014;**58**: 11–16.
51. Wong MN, Braswell LE, Murakami JW. Doxycycline sclerotherapy of cervical spine aneurysmal bone cysts: single-institution 13-year experience. *Pediatr Radiol* 2022;**52**: 1528–38.
52. Owen R. Embolization of musculoskeletal bone tumors. *Semin Intervent Radiol* 2010;**27**:111–23.
53. Kónya A, Szendröi M. Aneurysmal bone cysts treated by superselective embolization. *Skeletal Radiol* 1992;**21**:167–72.