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Giant pediatric aneurysmal bone cyst of the occipital bone: a case report and review of the literature

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ABSTRACT

Aneurysmal bone cyst (ABC) is a rare tumor-like vascular bone lesion that usually affects the metaphyses of the long bone. Cranial ABCs are uncommon, and occipitoparietal bone involvement is extremely rare. This lesion was first described by Jaffe and Lichtenstein in 1942. The precise etiology and pathophysiology of ABCs remain uncertain.

A 7-year-old boy with a history of occipital swelling, headache, and recurrent bleeding at the swelling site following trivial trauma presented to the clinic. Computed tomography revealed a huge, expansile, multiloculated cystic lesion. The patient had en-bloc resection of the mass and cranioplasty. Histopathology confirmed the diagnosis of ABC. The patient remained stable with no recurrence at the 6-month follow-up.

Differentiation of ABC from other osteolytic lesions of the cranium is difficult. However, a computed tomography scan showed the characteristics of the lesion. A high index of suspicion and imaging are important to diagnose ABC of the cranium as an extremely rare entity.

Key words: Aneurysmal bone cyst, cranial, giant, occipitoparietal bone

INTRODUCTION

Aneurysmal bone cyst (ABC) is an infrequent, vascular, non-neoplastic, benign tumor-like lesion of the bone first described by Jaffe and Lichtenstein in 1942 (1); the term "aneurysmal" simply describes the blow-out appearance on a radiograph. ABC is now regarded as a misnomer because it is a non-neoplastic bony lesion that consists of cystic cavernous cavities with a paucity of endothelial cell lining filled with unclotted blood. The precise etiology and pathophysiology of ABCs remain uncertain, but it is speculated that factors such as trauma, underlying neoplasms, and cytogenetic abnormalities are thought to be the possible pathophysiology of ABCs (2). The lesion can also be secondary to a reactive process following vascular hemodynamic disturbances associated with elevated venous pressure and trauma-induced hemorrhage (3). In some cases, ABCs are associated with underlying lesions such as osteoblastomas, eosinophilic granulomas, fibrous dysplasia, chondroblastomas, osteosarcomas, and chondromyxoid fibromas (4-8). Although, the lesion is classified as benign, malignant transformation has been reported in 3% of patients (9,10). Aneurysmal bone cysts represent only 1%–2% of all primary bone tumors and are usually located in the metaphyses of long bones and in the spine in 50% and 20% of cases, respectively (11). ABCs are occasionally found in the cranium, as described in 3%–6% of cases (12); in extremely rare instances, ABCs are found in the occipital region of the cranium (9,11). Treatment is often achieved by a curative en-bloc resection of the ABC and cranioplasty preferably with a titanium mesh to minimize the risk of recurrence (13). Prognosis is excellent, provided complete resection is achieved, but incomplete removal may affect its cure rate (14).

This case report described a 7-year-old boy with a giant occipital ABC who presented with recurrent traumatic bleeding of the lesion. The study also reviewed the contemporary published English-language literature addressing the clinical presentation, salient imaging features, histopathologic appearances, and treatment strategies.

CASE PRESENTATION

A 7-year-old boy presented with a progressive swelling on the occipital region for 7 months. He had associated headaches that were poorly characterized, occasionally relieved by the ingestion of analgesics. No similar swelling in any part of the body, vomiting, seizures, visual disturbance, or focal weakness and no associated gait disturbance were noted. No trauma to the head occurred prior to the onset of swelling. However, the patient repeatedly bled profusely following trivial trauma to the swelling that warranted recurrent blood transfusion. No other premorbid condition was reported.

On examination, the patient was well preserved with no focal neurological deficits. However, a swelling in the parieto-occipital region was found. It had no differential warmness and was mildly tender and hard to firm in consistency measuring 10×8 cm^2 in its widest dimension, with an ulcer at the summit of the swelling measuring 3×2 cm^2 with contact bleeding. It was not attached to the overlying scalp and was not mobile or pulsatile.

Skull radiographs showed a huge, expansile, osteolytic lesion involving the occipitoparietal bones with a sclerotic margin and an overlying soft-tissue mass located on its postero-inferior aspect. The computed tomography (CT) scan of the head (Fig. 1A, 1B, and 1C) showed a markedly expansile, multiloculated, osteolytic lesion, measuring $7.6 \times 6.4 \times 4.5$ cm^3 with cortical thinning noted at the occipitoparietal bone, which contained a cystic area at the center with fluid-fluid levels. A heterogonous, predominantly hypodense, soft-tissue mass on the postero-inferior aspect of the lesion, measuring $5.5 \times 4.7 \times 2.8$ cm^3 was also noted, which represented a scalp swelling with hematoma collection. Multiple cortical destructions of the inner plate adjacent to the lesion were seen. No enhancement of the lesions post IV contrast and evidence of extension into the dura and parenchymal brain tissue were seen.

Surgical excision biopsy of the mass was planned after consent was obtained from his parent; the patient underwent midline parieto-occipital craniotomy. Intraoperatively, the tumor was completely defined and en-bloc excision was done with a giglli saw after making burr holes around the bony mass with about 0.5-cm bony free margin all round (Fig. 2A). The whole bony tumor was encapsulated and extradural. It was composed of multiple cysts filled with unclotted brownish blood in varying sizes. He then had cranioplasty with a titanium mesh to cover the bony defects (Fig. 2B), and the specimen was sent for histopathological evaluation. His postoperative period was uneventful.

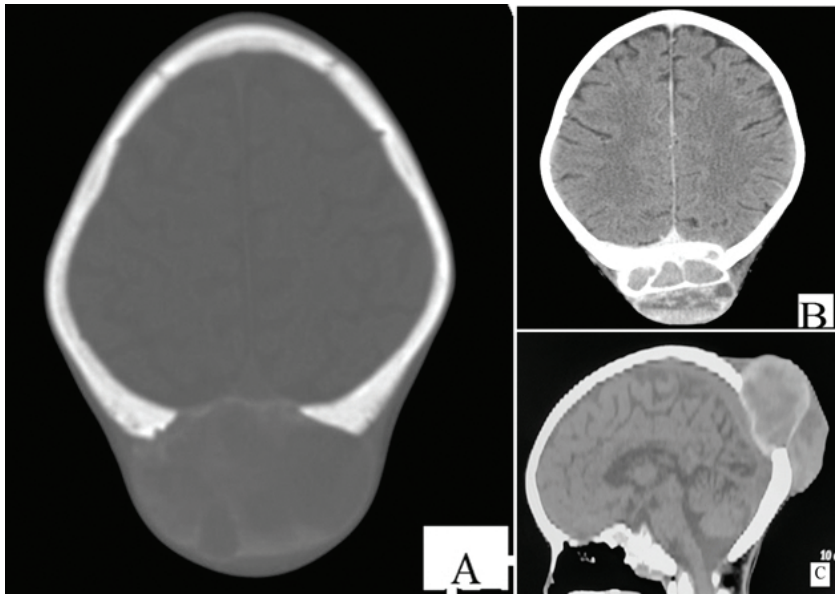

Figure 1 Axial bone window (A), axial contrast-enhanced (B), and sagittal reformatted (C) computed tomographic images of the head showing a huge expansile, multiloculated lytic lesion in the occipitoparietal region with fluid-fluid levels, thinning of the cortex, and overlying soft-tissue swelling.

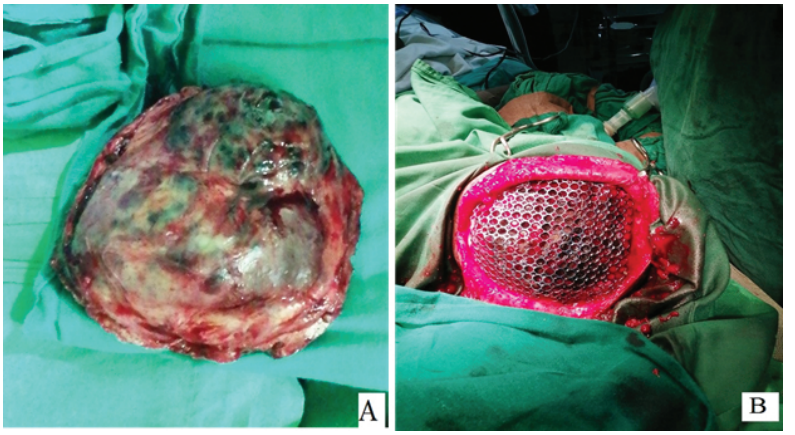

Figure 2 Intraoperative pictures showing craniotomy in the parieto-occipital region (A) and cranioplasty with a titanium mesh (B).

Macroscopy showed a discoid gray white tissue that was partly bony measuring $9 \times 8 \times 2$ cm^3 (Fig. 2A), and the surface of the tissue appeared bosselated. Microscopy showed a tissue composed of dilated and congested vascular channels within the fibroblastic stroma (Fig. 3A). Numerous siderophages were present. Sections from the bony tissue showed bony trabeculae having numerous cystic spaces filled with blood and lined with endothelial cells. Other areas showed marrow cells with no evidence of malignancy (Fig. 3B). The definitive diagnosis of occipital ABC was made.

DISCUSSION

The term "ABC," now regarded as a misnomer, originated from two cases of large blood-containing cystic cavernous cavities without a lining endothelium and was first reported by Jaffe and Lichtenstein in 1942 (1). ABC is a benign, non-neoplastic, expansible lesion of unknown etiology, occurring commonly before the age of 20 years (15). They consisted of blood-filled spaces separated by connective tissue septa composed of bone or osteoid and osteoclast giant cells. The blood-filled fibrous tumor-like cyst expanded the bone to give it a blow-out appearance and could emerge in any bone, including the skull, where it expanded between the inner and the outer tables of the diploe space with or without erosion of the bony plate. In up to 30% of cases, ABCs are associated with other underlying lesions, such as osteoblastomas, chondroblastomas, giant cell tumors, osteosarcomas, or fibrous dysplasia (16). However, the index case had no underlying lesion. Therefore, it is a primary lesion.

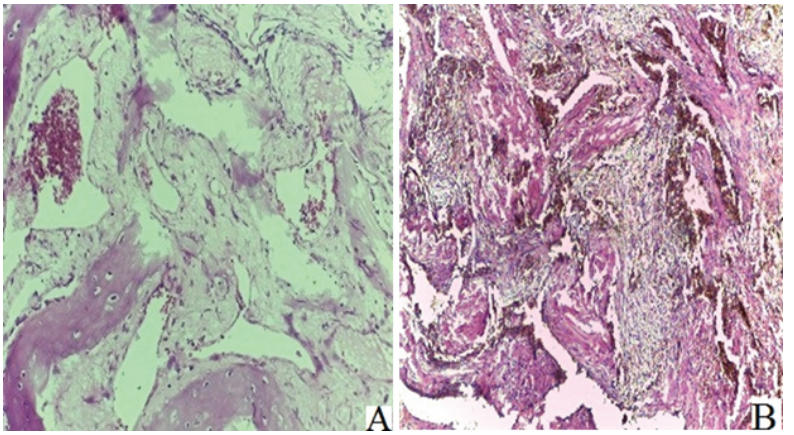

Figure 3 Photomicrograph section (A) shows blood-filled cystic spaces separated by bony trabeculae and cysts lined by endothelial cells. Photomicrograph (B) shows focal areas demonstrating fibroblasts with occasional giant cells surrounding the cystic spaces (hematoxylin and eosin, ×100).

It most frequently occurs in the second decade of life, but it is rare in children younger than 5 years, with the youngest age reported in the literature with ABC of especially the occipital bone at the age of 2 years (5,17). However, this case was a 7-year-old boy.

Only 27 cases of occipital ABCs were so far described in a survey of the contemporary medical English literature. Of these, 15 were male and 12 females, and the median age was 16 years (2, 4-10,13,17-32) (Table 1). Five of these cases (18.5%) exhibited additional pathology to the ABC, including eosinophilic granuloma, fibrous dysplasia, and osteoblastoma, while the remaining cases and the present case were primary lesions. In two of the cases with additional pathology of fibrous dysplasia, one had involvement of the temporal bone and the other had parietal bone involvement in addition to the occipital bone. Of the 27 cases, the presenting symptom in 15 (55.6%) was a headache, with 11 of the 15 cases having additional symptomatology and sign of hyporeflexia, stumbling,

Table 1 Demography, characteristics, and clinical presentation of reported cases of occipital aneurysmal bone cysts

S No.	Author(s), year of publication, and reference no.	Characteristics				
		Age	Sex	Additional pathology to aneurysmal bone cyst	Location*	Clinical presentation
1.	Odeku et al. 1965; (31)	6	M		O	Headache, palpable mass, and hyporeflexia
2.	Luccarelli et al. 1980; (18)	19	F		O	Headache and a palpable mass
3.	Bilge et al. 1983; (19)	18	M		O	Palpable mass
4.	Bilge et al. 1983; (19)	3	F		O	Palpable mass
5.	David et al. 1993; (9)	21	M		O	Focal tenderness
6.	Braun et al. 1987; 20)	4	F		O	Palpable mass
7.	Arthur et al. 1988; (21)	9	F		O	Palpable mass
8.	Chateil et al. 1997; (22)	9	M		O	Headache and vomiting
9.	Kumar et al. 1999; (32)	15	M		O & P	Headache and a palpable mass
10.	Kumar et al. 1999; (32)	44	M		O & P	Headache and focal tenderness
11.	Roncaroli et al. 2001; (5)	2	M	Eosinophilic Granuloma	O	Focal tenderness
12.	Petro & Lancon, 2001; (23)	7	F		O	Headache
13.	Itshayek et al. 2002; (7)	19	M	Fibrous dysplasia	O	Focal tenderness
14.	Iseri et al. 2005; (6)	35	F	Fibrous dysplasia	O & T	Headache
15.	Mattei et al. 2005; (8)	19	F	Fibrous dysplasia	O & P	Headache
16.	Gan & Hockley, 2007; (10)	8	M		O	Exophthalmia
17.	Lin et al. 2007; (24)	54	F		O	Focal tenderness
18.	Han et al. 2008; (4)	20	M	Osteoblastoma	O	Headache
19.	Mehboob et al. 2011; (2)	13	M		O	Palpable mass
20.	Genizi et al. 2011; (17)	2	M		O	Headache and stumbling
21.	Umredkar et al. 2012; (12)	8	M		O	Headache and swelling
22.	Brian et al. 2012; (25)	16	M		O	Headache and hydrocephalus
23.	Sarah et al. 2015; (26)	3	F		O	Rupture following head trauma
24.	Kalina & Wetjen, 2015; (27)	9	M		O	Headache and vomiting
25.	Gurcan et al. 2016; (28)	50	F		O	Palpable mass
26.	Mete et al. 2017; (29)	12	F		O	Headache, palpable mass, and hydrocephalus
27.	Chowdhury et al. 2018; (30)	19	F		O	Headache and gait disturbance
28.	Farouk et al. (present case)	7	M		O & P	Headache, occipital swelling, and bleeding following trivial trauma

* F, Female; M, male; O, occipital; P, parietal; T, temporal.

hydrocephalus, vomiting, focal tenderness, palpable mass, and gait disturbance. In eight cases, a palpable mass, and in five cases, a focal tenderness of the scalp was noted. One each presented with only exophthalmia and bone cyst rupture following trauma. However, the index case presented with headache, swelling, and recurrent bleeding from trivial trauma despite the fact that he had no other signs and symptomatology or any investigative findings suggestive of bleeding disorder. The patient in the present case also had a lesion involving the occipitoparietal bones, which was similar to the two cases reported by Kumar et al. and Mattei et al. (8). However, the present case had no additional pathology, which was in contrast to Mattei's report with additional fibrous dysplasia.

Origin of ABCs

Although the precise origin and nature of ABCs remains unclear, several theories exist to explain its origin. One theory hypothesizes that ABCs are a reactive process that follows an inciting trauma inducing a local circulatory disturbance that leads to the development of an ABC; the lesion is thought to occur because of increased venous pressure leading to hemorrhage, with subsequent osteolysis (3). This osteolytic lesion formation promotes additional hemorrhages and, in turn, results in the amplification of the developing cyst (33,34). A second theory is that ABCs are actually neoplasms mostly benign but having unique cytogenetic abnormalities; approximately 50% of patients with these lesions show an abnormality on chromosome 16q or 17p (35). This cytogenetic abnormality is typically a chromosomal translocation that causes the upregulation of ubiquitin-specific proteases, leading to increased cell adhesion (35). A third theory in consideration is that ABCs arise from an underlying neoplasm that induces a venous obstruction or an arteriovenous fistula (36). A current and popular view is that these lesions constitute a distinct entity, classified as primary and secondary depending on the presence or absence of associated pathology, the most common of which are giant cell tumors of bone, chondroblastomas, osteoblastomas, osteogenic sarcomas, chondromyxoid fibromas, and fibrous dysplasia (4-8,16,37-39). The primary form of the lesion, and the predominant form, is thought to be explained by the first proposed theory of traumatic and anomalous venous disruption in the osseous diploe, which causes the lesion to expand (40). A neoplastic basis for the primary ABCs was suggested by demonstrating clonal chromosome band 17p13 translocations and placing the USP6 oncogene under the regulation of the highly active CDH11 promoter (41). The secondary form, however, is thought to be formed by a disruption in the circulation of the involved osseous tissue caused by the associated pathology (40,42). Hormonal influence on the growth of these lesions has also been proposed, and changes in hormones, especially during pregnancy, may play a role in the origin of this tumor.38 Its association with pregnancy and rapid enlargement of the lesion during pregnancy, though rare, have also been reported (43). However, these tumors have displayed negative results on immunohistochemistry investigation for estrogen or progesterone receptors. A rare hereditary case of this lesion has also been reported (44).

Clinical signs and symptoms

Typically, ABCs usually presents in patients during the first or second decade of life as an enlarging mass that progresses rapidly and causes gross physical deformity, with concomitant signs corresponding with the anatomical location of the lesion (2,8,16,19). The commonest presenting symptom is pain at the site of the ABC (36). In the cranium, ABCs can present with exophthalmos (8,45), ptosis (45,46), loss of vision (47), cranial nerve palsies (48-50), symptomatology of raised intracranial tension and hydrocephalus (19,25,29), seizure disorder (50), cerebellar signs (18,30), and in rare circumstances, spontaneous intracerebral hemorrhages (10,51); superimposed bacterial meningitis are also described (52). The mean duration of symptom is typically 4 months (20).

Hemorrhagic lesions of the skull are extremely rare, especially in the pediatric population. Intraosseous hemangiomas and ABCs can both present as benign, enlarging skull masses; however, typically intraosseous hemangiomas are painless, whereas ABCs are painful and tender (26,53). As for malignant skull lesions, osteosarcomas of the skull can also present with hemorrhage (54), although these are more commonly seen in older patients typically in the third to the fourth decade of life, whereas osteosarcoma of the long bones tends to present in adolescents (55).

Pathological features

ABCs usually exhibit an expansion of both the inner and outer tables of the skull in a symmetric fashion, and therefore almost always have an intracranial extension (17). Grossly, ABCs consist of a friable, hemorrhagic material that is often gritty, and are divided

into classic (containing cysts; 95%) and solid (5%) (56). In en-bloc resections, cysts and cortical destruction can be appreciated as we did in the index case. Microscopically, ABCs show a stroma composed of fibroblasts, as well as cystic spaces often filled with unclotted blood, and an increased number of giant cells lining the cavernous spaces with the paucity of endothelial cells. The cysts are separated by septa composed of spindle-celled fibrous tissue that contains multinucleated giant cells and osteoid tissue (57).

Radiological characteristics

The classical radiological appearance of an ABC on plain radiographs is that of an eccentric, ballooned, cystic expansion surrounded by a sclerotic rim (58). The periosteum may be raised by new bone formation growing between the margin of the ABC and the adjacent, unaffected bony cortices. Plain films demonstrate either single or multiple blown-out lesions in the bone, hence the term aneurysmal.59 Although ABCs are typically radiolucent in 87% of cases, 2% may be radiopaque and mixed in 11% of cases.56,60 A "soap-bubble" or "blow-out" appearance of single or multiple cysts is also commonly seen in ABCs (56).

CT imaging of an ABC shows usually multiloculated or uniloculated lesions and demonstrates a soap-bubble appearance.60 The multiloculated lesion typically consists of several cavities with different densities, as in the index case. The vascular stroma and septa may enhance peripherally after administration of the contrast material (18,50). Fluid-fluid levels can be demonstrated on a CT scan, as in the present case, which presents as varying densities using a narrow window setting.59 Ultrasonography may demonstrate multiple fluid-fluid levels, which are characteristic of an ABC and can be used to complement other imaging modalities in indeterminate cases and resource-poor setting like ours (61).

Magnetic resonance imaging is, however, the preferred imaging modality and typically demonstrates the cystic components of the lesion and multiple fluid levels within multiloculated cysts resulting from unclotted blood, separated from the soft tissue and medullary bone. Fluid-fluid levels are an important cross-sectional imaging finding, but they are nonspecific and are more readily seen on an MRI than on a CT scan.59 The cyst contents consist of blood degradation products in different stages of evolution and, as such, the signal intensity of the degradation products varies, a finding considered to be attributable to methemoglobin (59). On angiography, the features of cysts elsewhere in the body are usually characterized by a pathological circulation with a patchy distribution of contrast with persistent venous circulation and occasional arteriovenous shunting. This finding may be absent when the lesion is located in the cranium, indicating that it is an avascular mass lesion (18,62). Usually, the imaging appearance of ABC reflects the underlying pathological changes.

Radiologically, ABCs may be staged according to the Enneking classification, as follows: Stage I (latent), ABC remains static or heals spontaneously; Stage II (active), ABC grows progressively but without cortical destruction; and Stage III (aggressive), ABC has progressive growth with cortical destruction (63). The index case was a stage 111 according to this classification because of progressive growth with cortical destructions.

Treatment

Surgical excision with a wide margin of the lesion is the most accepted treatment option and is usually curative, if the lesion can be easily accessed as in the present case. However, the difficult ones to access surgically can be treated effectively with external-beam radiation therapy or arterial embolization (25). Embolization and sclerotherapy are less invasive than surgical procedures and have shown good results in treating ABCs (25). Preoperative embolization also reduces bleeding during surgery (24). A few cases can be treated by partial resection and intra-lesional curettage, with adjuvant therapy, including preoperative embolization, postoperative radiotherapy, or cryotherapy (30).

CONCLUSIONS

Preoperative differentiation of ABC from other causes of osteolytic lesions of the cranial vault was difficult. However, the CT scan showed the characteristics of the lesion, which included an expansile multiloculated bony lesion with fluid-fluid levels. A clinically high index of suspicion and appropriate imaging technique are important to diagnose ABC of the cranial vault because it is an extremely rare entity.

Ethics:

Informed Consent: Informed consent was obtained.

Conflicts of Interest: The authors declare no conflicts of interest.

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