Primary cutaneous angiosarcoma of the scalp initially misdiagnosed as cellulitis in a 95-year-old Asian man: A case report

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Abstract. Cutaneous angiosarcoma is a rare and aggressive malignant vascular tumor that most commonly affects the scalp and face of elderly individuals. Because its early clinical manifestations are often non-specific, it may be misdiagnosed as a benign inflammatory or infectious disorder, resulting in delayed diagnosis and treatment. The case of a 95-year-old Asian man is reported, who presented with a 5-month history of a progressively enlarging scalp lesion that had initially been diagnosed as cellulitis and treated with antibiotics without improvement. Physical examination revealed a 4x5 cm ill-defined violaceous infiltrative plaque with central ulceration on the scalp. Histopathological examination demonstrated irregular anastomosing vascular channels lined by mildly atypical endothelial cells infiltrating the dermis and subcutis. Immunohistochemical staining was positive for CD31, CD34 and erythroblast transformation-specific related gene, with a Ki-67 index of ~20%; C-MYC staining showed weak focal positivity in ~10% of the lesional cells, whereas cytokeratin and epithelial membrane antigen staining were negative. Additional immunohistochemical staining demonstrated negative human herpesvirus 8 staining and the absence of a continuous smooth muscle actin-positive pericytic layer around the neoplastic vascular channels. Imaging studies showed no evidence of regional or distant metastasis. The patient subsequently underwent wide local excision with a 2 cm gross peripheral margin to the level of the galea aponeurotica. Intraoperative frozen-section and final permanent-section examinations confirmed tumor-free surgical margins. Adjuvant radiotherapy was not administered because of the advanced age of the patient and complete excision with negative margins. At the 8-month postoperative follow-up, clinical examination showed no evidence of local recurrence. The present case highlights that primary cutaneous angiosarcoma of the scalp may closely mimic cellulitis in its early stage, particularly in elderly patients, and that a progressive violaceous scalp lesion unresponsive to conventional antibiotic therapy should prompt early biopsy and histopathological evaluation.

Introduction

Angiosarcoma (AS) is a rare malignant neoplasm of vascular endothelial origin, accounting for ~2% of all soft tissue sarcomas (1). According to anatomical site and etiology, AS may be classified into cutaneous, lymphedema-associated, radiation-induced, breast and deep soft tissue subtypes (1,2). Among these, cutaneous angiosarcoma (cAS) is the most common form and predominantly affects the scalp and face of elderly individuals, with an estimated annual incidence of 0.26-3.0 cases per million population (3,4). Despite its rarity, cAS is clinically important because of its aggressive biological behavior, high local recurrence rate and propensity for early distant metastasis, resulting in a poor overall prognosis, with reported 5-year survival rates ranging from 11-50% (4,5).

Primary cAS is generally considered sporadic, whereas secondary angiosarcoma may develop in association with chronic lymphedema or previous radiotherapy. Although the precise pathogenesis of primary cAS remains incompletely understood, cumulative ultraviolet exposure, advanced age, genetic alterations affecting endothelial cells and dysregulation of angiogenic signaling pathways have been proposed as contributing factors (1-4). Diagnosis requires careful clinicopathological consideration. Histopathological examination typically demonstrates irregular, anastomosing vascular channels lined by atypical endothelial cells, while immunohistochemical markers such as CD31, CD34 and erythroblast transformation-specific related gene (ERG) are useful for confirming endothelial differentiation and excluding histological mimics. Once the diagnosis has been established, appropriate imaging is required to evaluate regional lymph node involvement, local extension and distant metastasis. Prognosis is influenced by the infiltrative and occasionally

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multifocal growth pattern of the tumor, the feasibility of achieving complete surgical excision and the occurrence of local recurrence or metastatic spread (2-5).

One of the major clinical challenges associated with cAS is that its early manifestations are frequently subtle and nonspecific. Initial lesions may appear as bruise-like patches, erythematous plaques or nodules, and may mimic benign inflammatory or infectious conditions, including cellulitis, dermatitis, rosacea and herpes zoster (6-9). This overlap often leads to delayed recognition and delayed biopsy, which may in turn postpone definitive treatment. Early histopathological confirmation is therefore key to avoid diagnostic delay and facilitate timely intervention.

For localized and technically resectable cAS, complete surgical excision with histologically negative margins remains the principal treatment. However, because the tumor frequently exhibits poorly defined and infiltrative growth, adequate local clearance may be difficult to achieve. Adjuvant radiotherapy may be considered to improve local control, particularly in patients with large, infiltrative or incompletely excised tumors. For unresectable, recurrent or metastatic disease, systemic treatment options include taxane-based chemotherapy and selected anti-angiogenic agents, although treatment responses and long-term outcomes remain variable. Consequently, therapeutic decisions should be individualized according to tumor extent, resectability, patient age and comorbidities, preferably through multidisciplinary assessment (2,4,5).

The present study reports a case of primary cAS of the scalp in a 95-year-old Asian man that was initially misdiagnosed as cellulitis. The clinicopathological features, diagnostic process and therapeutic management are described, and selected previously reported cases are reviewed to highlight the diagnostic pitfalls of this uncommon but aggressive malignancy.

Case report

A 95-year-old Asian man presented to the Department of Dermatology, First People's Hospital of Horqin District (Tongliao, China), in November 2025, with a 5-month history of a progressively evolving scalp lesion and a 1-month history of ulceration and crusting. Five months prior to presentation, a small erythematous nodule had appeared on the vertex of the scalp without pain or pruritus. Over time, the lesion gradually enlarged with peripheral extension and became elevated and firm; 1 month before presentation, minor trauma to the lesion resulted in ulceration and bleeding, followed by crust formation. The surrounding skin progressively developed a violaceous discoloration. The patient had previously been diagnosed with cellulitis at another hospital and treated with systemic antibiotics for 2 weeks; however, no notable clinical improvement was observed. Their medical history included diabetes mellitus and hypertension. There was no history of chronic lymphedema, previous radiotherapy, immunosuppression or similar tumors in the family.

On physical examination, a 4x5 cm ill-defined violaceous infiltrative plaque was observed on the vertex of the scalp, with surrounding purpuric/ecchymotic discoloration, central ulceration and dry crusting (Fig. 1). The lesion was firm on palpation and had poorly demarcated borders. No palpable cervical lymphadenopathy was detected. No additional suspicious cutaneous lesions were identified on routine skin examination.

Routine laboratory investigations, including complete blood count, liver and renal function tests, coagulation profile and urinalysis, were all within normal limits. To evaluate disease extent, computed tomography of the head and neck as well as contrast-enhanced chest-abdomen imaging were performed, and no evidence of regional lymph node involvement or distant metastasis was detected.

Due to the atypical clinical appearance and poor response to antibiotic therapy, a biopsy of the scalp lesion was performed. The biopsy specimen was fixed in 10% neutral-buffered formalin at room temperature for 24 h, routinely processed, embedded in paraffin and sectioned at a thickness of 4 μm. The sections were deparaffinized in xylene and rehydrated through a graded ethanol series. Manual hematoxylin and eosin (H&E) staining was performed at room temperature. Briefly, the sections were stained with Harris hematoxylin for 5 min, rinsed in running tap water, differentiated in 1% acid alcohol, blued in running tap water and counterstained with eosin Y for 2 min. The stained sections were subsequently dehydrated through graded ethanol, cleared in xylene and mounted with neutral resin. Bright-field images were acquired using a NanoZoomer digital slide scanner (Hamamatsu Photonics K.K.), and representative images were reviewed and exported using NDP.view2 software (version 2.9.29; Hamamatsu Photonics K.K.). Histopathological examination of the specimen revealed irregular, anastomosing vascular channels of varying size within the dermis and subcutis. These slit-like and variably dilated vascular spaces were lined by mildly atypical endothelial cells exhibiting focal intraluminal protrusion and infiltrative growth into the surrounding stroma (Fig. 2). The overall morphology was consistent with a well-differentiated vasoformative malignant vascular neoplasm.

For immunohistochemical analysis, 4 μm thick sections were prepared from formalin-fixed, paraffin-embedded tissue. The tissue was fixed in 10% neutral-buffered formalin at room temperature for 24 h before routine processing and paraffin embedding. Immunohistochemical staining was performed using an Ultra 60 Plus automated immunohistochemistry stainer (OriGene Technologies, Inc.). Heat-induced epitope retrieval for all nine antibodies was performed within the instrument using an EDTA-based antigen retrieval solution (pH 8.0; cat. no. ZLI-9067; OriGene Technologies, Inc.) at 98˚C for 25 min according to the manufacturer's pre-optimized protocol. Endogenous peroxidase activity was blocked with 3% hydrogen peroxide at room temperature for 10 min.

All primary antibodies were supplied by OriGene Technologies, Inc. in a ready-to-use format and required no further dilution. The antibodies used were cytokeratin (CK; clone AE1/AE3; cat. no. ZM-0069), CD31 (clone UMAB30; cat. no. ZM-0044), CD34 (clone QBEnd/10; cat. no. ZM-0046), ERG (clone UMAB78; cat. no. ZM-0103), Ki-67 (clone UMAB107; cat. no. ZM-0166), C-MYC (clone EP121; cat. no. ZA-0555), epithelial membrane antigen (EMA; clone GP1.4; cat. no. ZM-0095), human herpesvirus 8 (HHV-8; clone 13B10; cat. no. ZM-0404) and smooth muscle actin (SMA; clone UMAB237; cat. no. ZM-0003). The sections were incubated with the ready-to-use primary antibodies at 37˚C for 60 min.

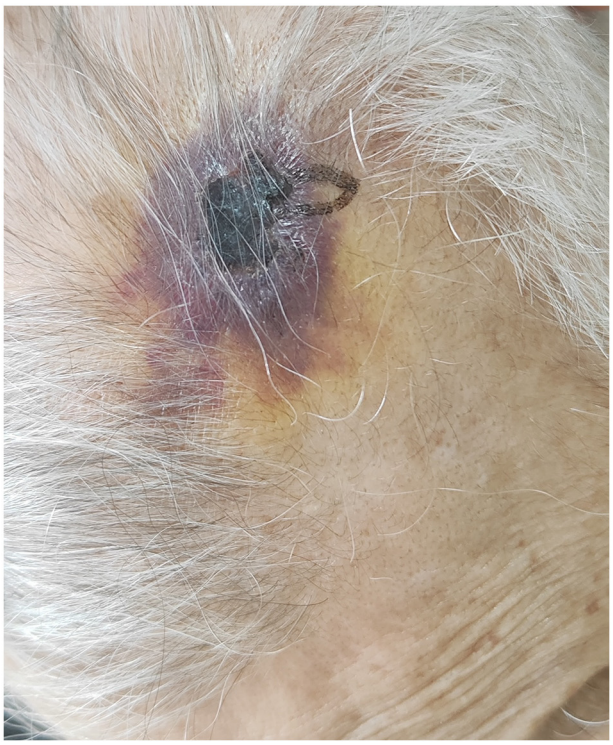

Figure 1. A 4x5 cm ill-defined violaceous infiltrative plaque on the vertex of the scalp with surrounding ecchymotic discoloration and a central dry crust.

A universal two-step mouse/rabbit enhanced polymer detection system (cat. no. PV-9000; OriGene Technologies, Inc.) was used for immunodetection. The polymer detection reagent was incubated at 37˚C for 20 min, and immunoreactivity was visualized using a 3,3'-diaminobenzidine (DAB) chromogenic detection kit (cat. no. ZLI-9017; OriGene Technologies, Inc.). DAB color development was performed at room temperature for 5 min, followed by hematoxylin counterstaining at room temperature for 20 sec. All staining procedures, including deparaffinization, antigen retrieval, endogenous peroxidase blocking, primary antibody incubation, polymer-based detection and chromogenic visualization, were performed automatically according to the manufacturer's standardized protocol. Appropriate positive controls were included for each antibody. The immunohistochemically stained sections were scanned using a NanoZoomer digital slide scanner (Hamamatsu Photonics K.K.), and representative fields were reviewed and exported using NDP.view2 software (version 2.9.29; Hamamatsu Photonics K.K.).

Immunohistochemical staining demonstrated positivity for CD31, CD34 and ERG, supporting endothelial differentiation. The Ki-67 proliferation index was ~20% (Fig. 3), and C-MYC staining showed weak focal positivity in ~10% of the lesional cells (Fig. S1A), whereas CK and EMA staining were negative (Fig. S1B and C). Additional immunohistochemical staining for HHV-8 and SMA was subsequently performed on serial sections obtained from the same representative paraffin block used for the original immunohistochemical evaluation. HHV-8 staining was negative in the lesional cells (Fig. 4A), whereas SMA staining demonstrated the absence of a continuous SMA-positive pericytic layer surrounding the irregular neoplastic vascular channels (Fig. 4B). Taken together, the histopathological and immunophenotypic findings supported the diagnosis of well-differentiated primary cAS and argued against Kaposi sarcoma.

Following clinicopathological confirmation of the diagnosis and imaging exclusion of metastatic disease, the patient underwent wide local excision in the Department of Surgery, First People's Hospital of Horqin District (Tongliao, China), with a gross peripheral margin of 2 cm from the clinically visible tumor border. The lesion was excised en bloc to the level of the galea aponeurotica. Intraoperative frozen-section examination showed no tumor involvement of the circumferential peripheral margins or the deep basal margin. Final permanent-section examination subsequently confirmed that all submitted surgical margins were free of tumor; however, the exact microscopic clearance distances were not reported. The defect was reconstructed using an O-Z flap. The postoperative course was uneventful. Adjuvant radiotherapy was not administered because of the advanced age of the patient and complete excision with negative surgical margins. At the most recent outpatient follow-up in July 2026, ~8 months after surgery, local examination showed no clinical evidence of recurrence.

Discussion

cAS most commonly affects elderly individuals and often develops on the scalp and face (3,4). A major reason for delayed diagnosis is that early lesions frequently present as inconspicuous erythematous or violaceous patches, bruise-like plaques or edematous infiltrative lesions, which can resemble inflammatory or infectious processes rather than a malignant vascular tumor (6-9). In routine clinical practice, especially when scalp erythema, swelling or crusting is present, benign conditions such as cellulitis or dermatitis may be considered first, thereby postponing biopsy.

Several previously published case reports have documented similar diagnostic pitfalls (Table I). Reported initial misdiagnoses include cellulitis (6), diabetic foot ulcer (7), refractory wound (8), herpes zoster (9) and abscess (10). In those reports, the interval between symptom onset and definitive diagnosis ranged from several weeks to months. Such delays are clinically meaningful, as they may allow continued local progression, reduce the likelihood of complete surgical resection and adversely affect prognosis. The cases summarized in Table I suggest a recurring clinical pattern characterized by advanced age, involvement of the scalp or face, violaceous or bruise-like lesions, progressive enlargement and poor response to empirical anti-inflammatory or antibiotic treatment.

The present case closely reflects this pattern. The lesion was initially managed as cellulitis because of erythema, swelling and subsequent ulceration. However, classic cellulitis usually presents with warmth, tenderness and at least some degree of systemic inflammatory response, and most cases improve with appropriate antibiotic treatment. In the present patient, the absence of systemic manifestations and the lack of clinical improvement after antibiotic therapy should have prompted reconsideration of the initial diagnosis. The present case therefore emphasizes an important practical point: In elderly patients, a progressive violaceous scalp lesion that does not respond to conventional antibiotic therapy should raise suspicion for malignancy and prompt early biopsy.

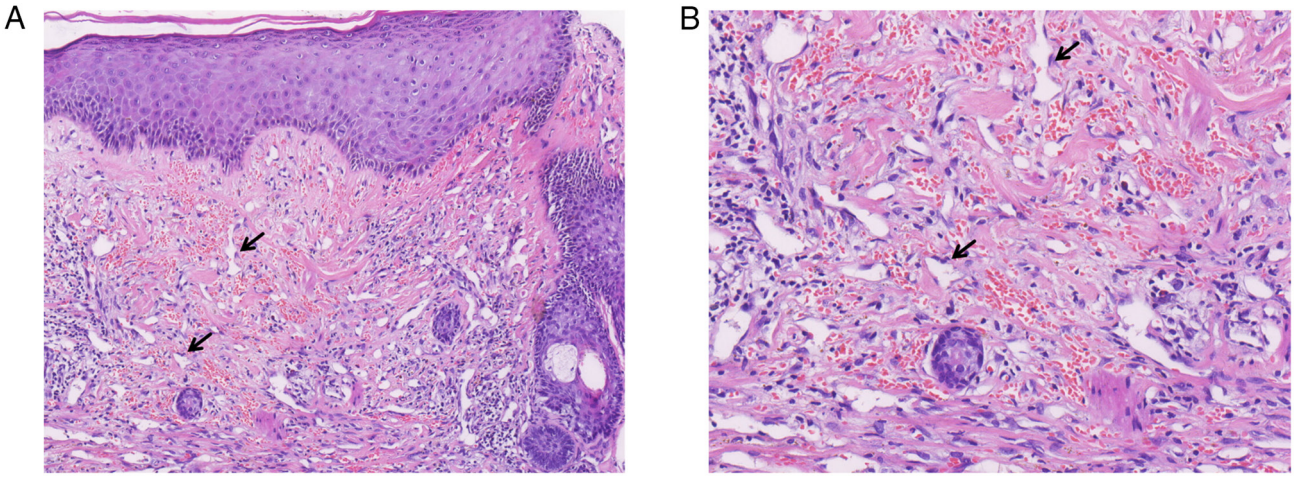

Figure 2. Histopathological examination of the scalp lesion. (A) Medium-power view showing irregular, anastomosing vascular channels (arrows) infiltrating the dermis and subcutis (H&E staining; magnification, x100). (B) Higher-power view showing slit-like and variably dilated vascular spaces lined by mildly atypical endothelial cells and exhibiting focal intraluminal protrusions (arrows), consistent with an infiltrative growth pattern (H&E staining; magnification, x200).

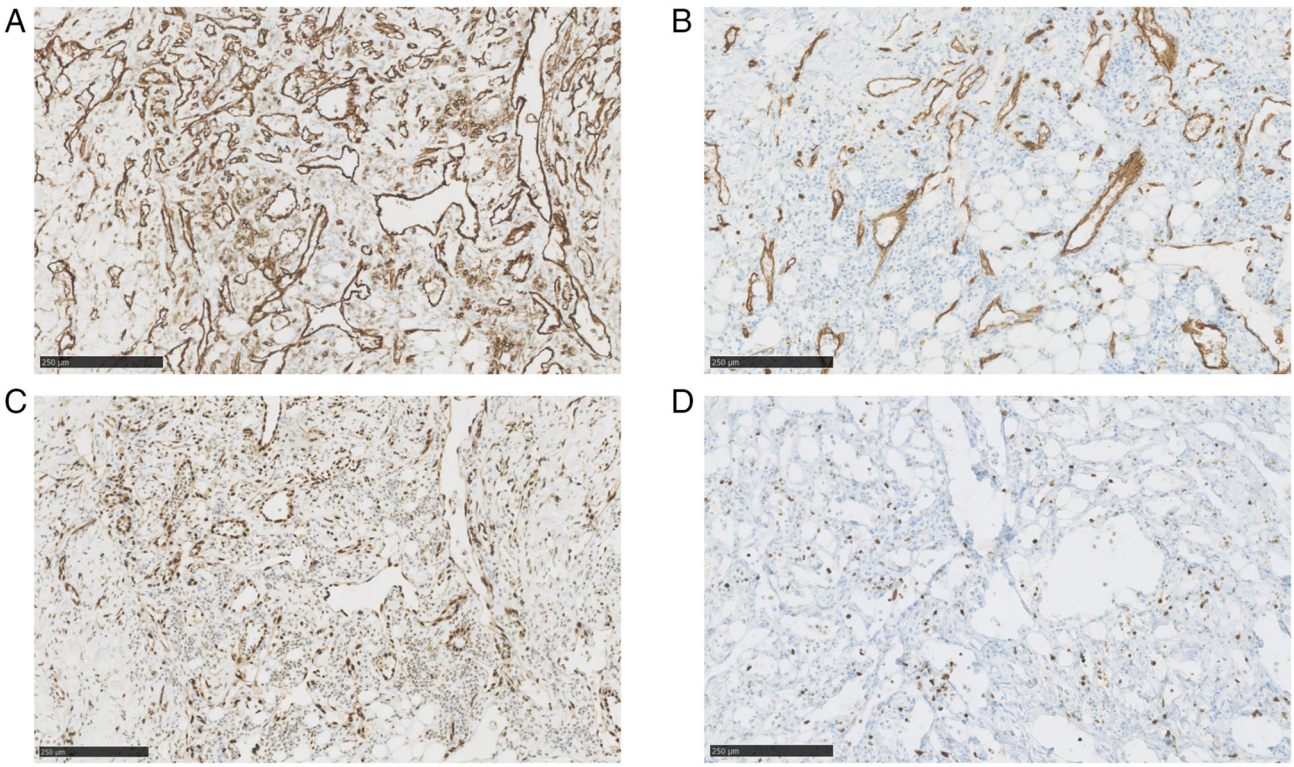

Figure 3. Immunohistochemical staining of the tumor showing positivity for (A) CD31, (B) CD34 and (C) erythroblast transformation-specific related gene, with (D) a Ki-67 proliferation index of ~20% (magnification, x100).

Histopathological examination remains the cornerstone of diagnosis. Well-differentiated cAS typically demonstrates irregular anastomosing vascular channels lined by atypical endothelial cells infiltrating the dermis and subcutis, whereas poorly differentiated lesions may show reduced vasoformation and may mimic poorly differentiated carcinoma or melanoma (2,4). Although the endothelial atypia in the present case was relatively mild, the diagnosis of well-differentiated cAS was supported by the irregular and anastomosing vascular channels, infiltrative dissection of the surrounding stroma, extension into the subcutis and focal intraluminal endothelial protrusion. For this reason, immunohistochemistry is of considerable diagnostic value. Endothelial markers such as CD31, CD34, ERG and friend leukemia integration 1 are commonly expressed, with CD31 and ERG considered particularly useful markers of endothelial differentiation (2,4). In the present case, the diagnosis was supported by the characteristic vasoformative morphology together with positivity for CD31, CD34 and ERG, and negativity for CK and EMA, which helped exclude an epithelial neoplasm.

Table I. Selected misdiagnosed cases of cutaneous angiosarcoma in the literature.

First author, year	Age/sex	Location	Initial misdiagnosis	Diagnostic delay	Outcome	(Refs.)
AlNodali *et al*, 2024	87/F	Neck, face and scalp	Cellulitis and dermatitis	2 months	Unresectable	(6)
Almheirat *et al*, 2024	48/M	Foot	Diabetic foot ulcer	3 months	Deceased at 6 months after amputation	(7)
Yan *et al*, 2023	78/M	Scalp	Refractory wound	4 months	Surgical resection	(8)
Yıldırım *et al*, 2021	50/M	Scalp	Herpes zoster	2 months	Deceased at 6 months	(9)
Lara-Martinez *et al*, 2021	53/M	Face	Abscess	4-6 months	Metastatic progression	(10)

F, female; M, male.

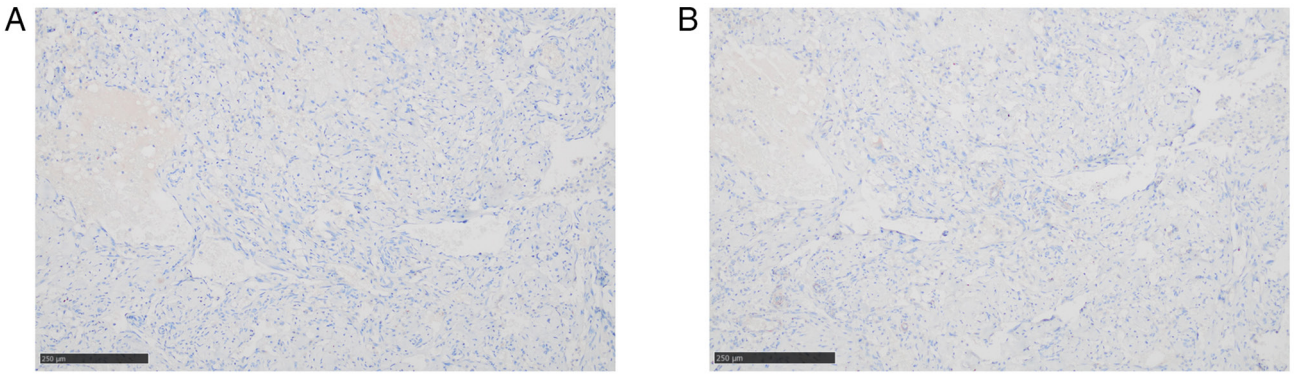

Figure 4. Additional immunohistochemical findings. (A) Human herpesvirus 8 staining was negative in the lesional cells (magnification, x100). (B) Smooth muscle actin staining demonstrated the absence of a continuous smooth muscle actin-positive pericytic layer surrounding the irregular neoplastic vascular channels (magnification, x100).

The principal histopathological differential diagnoses included radiation-associated atypical vascular lesions, benign vasoformative lesions and Kaposi sarcoma. A radiation-associated atypical vascular lesion was considered unlikely because the patient had no history of radiotherapy and because the lesion demonstrated infiltrative involvement of both the dermis and subcutis. The absence of chronic lymphedema also supported classification of the tumor as primary rather than lymphedema-associated cAS. Furthermore, SMA staining showed that the irregular neoplastic vascular channels lacked a continuous SMA-positive pericytic layer, supporting a malignant vasoformative proliferation rather than a benign vascular lesion. HHV-8 staining was negative in the lesional cells, arguing against Kaposi sarcoma. C-MYC staining showed only weak focal positivity in ~10% of the lesional cells, rather than diffuse strong expression; MYC gene amplification was not evaluated. Taken together, these morphological and immunophenotypic findings supported the diagnosis of well-differentiated primary cAS.

Complete surgical excision with histologically negative margins remains the cornerstone of treatment for localized cAS (4,11). However, because cAS often exhibits infiltrative and occasionally multifocal growth, achieving adequate local control may be challenging. Adjuvant radiotherapy may improve local control in selected cases, whereas systemic treatments, including taxanes and anti-angiogenic agents, may be considered in unresectable or metastatic disease (2,11-13). Despite this, prognosis remains guarded, and multidisciplinary management is generally recommended (5,14). In the present patient, no distant metastasis was detected on preoperative imaging, and wide local excision was performed with a gross peripheral margin of 2 cm from the clinically visible tumor border, extending to the level of the galea aponeurotica. Both intraoperative frozen-section examination and final permanent-section examination confirmed that the submitted peripheral and deep margins were free of tumor, although the exact microscopic clearance distances were not reported. Adjuvant radiotherapy was not pursued because of the advanced age of the patient and complete excision with negative surgical margins. At the most recent outpatient follow-up in July 2026, ~8 months after surgery, local examination showed no clinical evidence of recurrence. Nevertheless, recurrence is common; in a retrospective cohort of 143 patients with cAS of the face and scalp, 61 patients (43%) developed local recurrence and 83 patients (58%) experienced disease recurrence overall (15). Therefore, long-term surveillance remains necessary.

The present report has certain limitations. First, as a single case report, the conclusions that may be drawn are limited. Second, the postoperative follow-up period was limited to 8 months and was based on clinical examination without repeat staging imaging; therefore, longer follow-up is required to assess long-term local control and metastatic risk. Third,

although all submitted surgical margins were tumor-free on permanent-section examination, the exact microscopic clearance distances were not documented in the original pathology report. In addition, C-MYC expression was assessed using immunohistochemistry, but MYC gene amplification was not evaluated. Despite these limitations, the present case adds to the existing literature on the varied presentation of cAS and reinforces the importance of early tissue diagnosis in atypical scalp lesions. Reports of other rare sarcomas have similarly emphasized the value of integrating detailed morphological and immunophenotypic characterization with a review of previously reported cases to improve diagnostic recognition (16).

In summary, primary cAS of the scalp may clinically mimic cellulitis, particularly in elderly patients. Failure to respond to antibiotic therapy, especially in the setting of a progressive violaceous scalp lesion, should prompt early biopsy and histopathological evaluation. Early diagnosis and timely definitive treatment are important for reducing diagnostic delay and facilitating appropriate management.

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Availability of data and materials

The data generated in the present study are included in the figures and/or tables of this article.

Authors' contributions

BQ conceived and designed the case report, participated in the clinical diagnosis and management of the patient, acquired and interpreted the clinical data, and drafted the manuscript. QL and YG contributed to the acquisition, analysis and interpretation of the clinical data, conducted the literature review and critically revised the manuscript for important intellectual content. YT collected the clinical data. YY contributed to the conception and design of the case report, participated in the clinicopathological diagnosis and therapeutic decision-making, interpreted the clinical and pathological findings, and critically revised the manuscript for important intellectual content. BQ and YY confirm the authenticity of all the raw data. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

The present study was approved by the Ethics Committee of the First People's Hospital of Horqin District (Tongliao, China; approval no. 2026001). Written informed consent was obtained from the patient.

Patient consent for publication

Written informed consent was obtained from the patient for publication of the present case report and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

References

1. Young RJ, Brown NJ, Reed MW, Hughes D and Woll PJ: Angiosarcoma. Lancet Oncol 11: 983-991, 2010.
2. Cao J, Wang J, He C and Fang M: Angiosarcoma: A review of diagnosis and current treatment. Am J Cancer Res 9: 2303-2313, 2019.
3. Shustef E, Kazlouskaya V, Prieto VG, Ivan D and Aung PP: Cutaneous angiosarcoma: A current update. J Clin Pathol 70: 917-925, 2017.
4. Yamakawa K, Ogata D, Namikawa K, Nakano E, Yamaguchi Y and Yamazaki N: A review of cutaneous angiosarcoma: Epidemiology, diagnosis, prognosis, and treatment options. Jpn J Clin Oncol 55: 707-719, 2025.
5. Houpe JE, Seger EW, Neill BC, Kang BY, Hocker TLH, Alam M and Tolkachjov SN: Treatment of angiosarcoma of the head and neck: A systematic review. Cutis 111: 247-251, 2023.
6. AlNodali N, Alqahtani N, Sharafuddin A, Atef R, AlNodali H, Dhulaimi M, Alshomrani B and Alwagdani Y: Cutaneous angiosarcoma masquerading as photodermatitis: A case report. Acta Dermatovenerol Alp Pannonica Adriat 33: 213-215, 2024.
7. Almheirat Y, Elyamani L, Hormi O, Zerrouki N, Zizi N and Dikhaye S: Misdiagnosis of cutaneous epithelioid angiosarcoma as diabetic foot ulcer: A case study. Wounds 36: 43-46, 2024.
8. Yan ZH, Li ZL, Chen XW, Lian YW, Liu LX and Duan HY: Misdiagnosis of scalp angiosarcoma: A case report. World J Clin Cases 11: 3099-3104, 2023.
9. Yıldırım S, Erdemoğlu Y, Durmaz EO, Erfan G, Duman D and Dikicioğlu Çetin E: Primary cutaneous angiosarcoma of the scalp with cranial invasion in a patient with metastatic breast cancer. J Clin Aesthet Dermatol 14: 22-24, 2021.
10. Lara-Martinez H, Weinberg M, Baratam P, Horn J, Ward K and Styler M: Angiosarcoma of the face: A case study and literature review of local and metastatic angiosarcoma. Case Rep Oncol Med 2021: 8823585, 2021.
11. Florou V and Wilky BA: Current and future directions for angiosarcoma therapy. Curr Treat Options Oncol 19: 14, 2018.
12. Richards E, Schimmel J, Renzi M and Lawrence N: An update on cutaneous angiosarcoma diagnosis and treatment. Cutis 113: 218-223, 2024.
13. Klein JC and Dominguez AR: Cutaneous angiosarcoma. JAMA Dermatol 159: 332, 2023.
14. Sturm EC, Marasco IS and Katz SC: Multidisciplinary management of angiosarcoma: A review. J Surg Res 257: 213-220, 2021.
15. Yoder AK, Farooqi AS, Wernz C, Subramaniam A, Ravi V, Goepfert R, Sturgis EM, Mitra D, Bishop AJ and Guadagnolo BA: Outcomes after definitive treatment for cutaneous angiosarcomas of the face and scalp: Reevaluating the role of surgery and radiation therapy. Head Neck 45: 1943-1951, 2023.
16. Bai Q, Lin Y, Fu W, Ning Y, Niu Q, Su W, Wang X, Lan Y, Wang Y, Zhang Y, *et al*: Characteristics of intracranial sclerosing epithelioid fibrosarcoma and review of related literature. Oncol Transl Med 11: 187-194, 2025.