

Inflammatory Infiltrating Angiolipoma With an Unusual Plasmacytoid Histiocytic Infiltrate

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Abstract

Infiltrating angiolipoma is an uncommon growth pattern of angiolipoma that typically occurs in the trunk and extremities of male patients during the second and third decades of life. Histologically, it is characterized by mature adipocytes admixed with small- to capillary-sized vascular proliferations and infiltrative margins, often making complete surgical excision challenging. To our knowledge, the inflammatory plasmacytoid morphology observed in the present case has not been previously emphasized in published English-language reports of infiltrating angiolipoma. A 25-year-old woman presented with multifocal infiltrating angiolipomas involving both arms and buttocks, with a clinical history spanning more than 17 years. The lesions demonstrated the characteristic features of infiltrating angiolipoma but were notable for a prominent histiocytic infiltrate exhibiting plasmacytoid cellular morphology, an association not previously reported. Microscopic examination revealed mature adipocytic proliferation with infiltrative growth and associated capillary-sized vascular channels. In addition, a distinctive inflammatory plasmacytoid component was identified throughout the lesion, representing a unique morphologic feature that adds to the known histopathologic cellular variant of infiltrating angiolipoma. We report a rare case of multifocal infiltrating angiolipoma in a young woman with an associated unique inflammatory infiltrate exhibiting plasmacytoid histologic morphology. Recognition of this previously undescribed association may help avoid diagnostic confusion and broaden the current understanding of the morphologic diversity of infiltrating angiolipoma.

Categories: Pathology, Oncology

Keywords: case report, histopathology examination, infiltrating angiolipoma, plasmacytoid, rare morphology

Introduction

Angiolipomas are benign subcutaneous neoplasms of unknown etiology [1], most commonly occurring in men in the late second to early third decade of life. Histologically, they consist of mature adipocytes and small clustered vessels, sometimes with intravascular fibrin microthrombi. A cellular variant is described when the vascular component predominates [1,2]. Infiltrating angiolipoma is a benign variant characterized by locally infiltrative growth into adjacent soft tissues, where it may enlarge over time [3]. These lesions may present as solitary tumors or in association with angiolipomatosis, often affecting younger patients [3]. Although non-metastatic, infiltrating angiolipomas have a risk of local recurrence after incomplete excision [3]. They are distinct from deeply infiltrative angiolipomas of parenchymal organs and from intramuscular hemangiomas [4]. Despite long-standing classification efforts since the 1980s, taxonomic debate of two "lipo-vascular" neoplasms based on infiltrative potential persists in the literature [5-7]. Infiltrating angiolipoma was first described in the 1960s as a non-encapsulated variant of angiolipoma [8]. Subsequent reports in the 1970s documented angiolipomatous lesions involving muscle and other deep structures, although some of these cases would now be classified as intramuscular hemangiomas [3]. Over the following decades, the distinction between infiltrating angiolipoma and intramuscular hemangioma remained debated, with authors variably applying both terms to lesions exhibiting overlapping adipocytic and vascular features [4,9-11]. More recent observations have identified tumors with mixed lipomatous and vascular components and variable infiltrative behavior into deep muscles [12,13]. Despite these observations, no recurrent cytogenetic or molecular abnormalities have been established for infiltrating angiolipoma [14]. Consequently, the current World Health Organization (WHO) classification regards infiltrating angiolipoma as a benign angiolipoma with an infiltrative growth pattern rather than a distinct tumor entity [1]. This approach is supported by clinicopathologic and molecular evidence distinguishing angiolipoma, an adipocytic neoplasm with characteristic capillary vascular proliferation, from intramuscular hemangioma, a primary vascular lesion that may contain secondary adipose tissue.

Secondary inflammatory infiltrates in angiolipoma may include lymphocytes, mast cells, and occasional macrophages, typically in association with vascular thrombosis or degenerative changes [3]. In this paper, we present a case of long-standing subcutaneous multiple inflammatory infiltrating angiolipomas with unique plasmacytoid histiocytic infiltrate. To our knowledge, cases with this histopathological morphology have not been previously reported in the English-language literature.

How to cite this article

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Case Presentation

A 25-year-old woman, a known case of dyslipidemia on medication, presented to the plastic surgery clinic complaining of four long-standing masses dangling from her bilateral arms and buttocks for 17 years. The patient denied pain and other symptoms. On physical examination, she has xanthelasma and tendon xanthoma on the elbows, knee, and Achilles tendon. The masses were nontender; two were in the posterior aspect of the arms just above the elbows, and two gluteal masses extended down to the thighs with redundant skin. Results of the complete blood count were all within normal reference range, with an abnormally high lipid profile. Metastatic workup was negative. Radiographically, these masses appeared to be bilateral and symmetrical with direct continuity with the subcutaneous fat tissue of the buttocks. It measured 21 cm in greatest dimension. It appeared as a heavily infiltrated fatty tissue with high T2 signal intensity, intermediate T1 signal intensity, and the persistence of speckles of fat. The clinical diagnosis was lipomatosis. Intraoperatively, the surgeon reported finding abnormal fat that was infiltrative of normal subcutaneous fat above fascial planes, difficult to completely excise. They were cautiously dissected until normal fat was encountered proximal to fascial planes and skeletal muscles, which were devoid of tumor, for reconstructive efforts to follow. Grossly, the mass was covered with unremarkable skin. Sectioning revealed a homogeneous salmon-pink solid rubbery cut surface and scant fibrofatty tissue embedded within (Figure 1). Sectioning of the peripheral fibrofatty tissue at the margin revealed an ill-defined mass margin with trabeculated net-like extensions of fibrous bands encasing unremarkable fibrofatty tissue with focal positive margins.

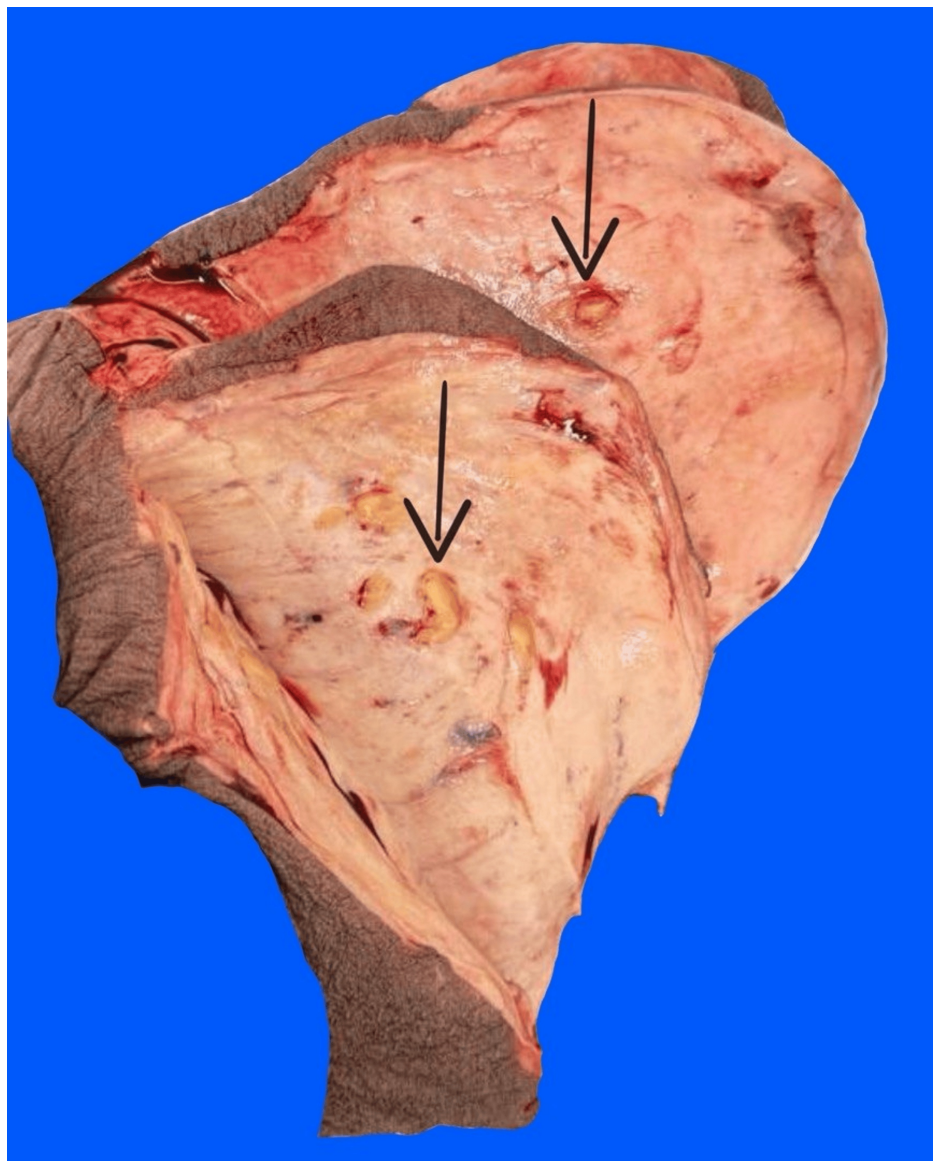


FIGURE 1: Gross appearance of the mass.

Salmon-pink solid rubbery cut surface with entrapped native adipose tissue (arrows).

Microscopic examination showed a non-encapsulated, infiltrative lesion involving the deep dermis and subcutaneous adipose tissue. The mass was composed of islands and lobules of mature adipocytes, admixed with numerous capillary-sized to small thin-walled vascular channels. The intervening stroma contained plump ovoid to prominent plasmacytoid cells diffusely arranged, lacking fascicular or storiform architecture. There was also chronic inflammation consisting of lymphocytes, histiocytes, and occasional multinucleated giant cells, including Touton, Langhans-type giant cells, and osteoclast-like giant cells. The lesional cells of both vascular and adipocytic components lacked atypia, pleomorphism, or hyperchromasia. Extensive sampling and careful search did not reveal any lipoblasts, abnormal mitoses, or necrosis (Figure 2).

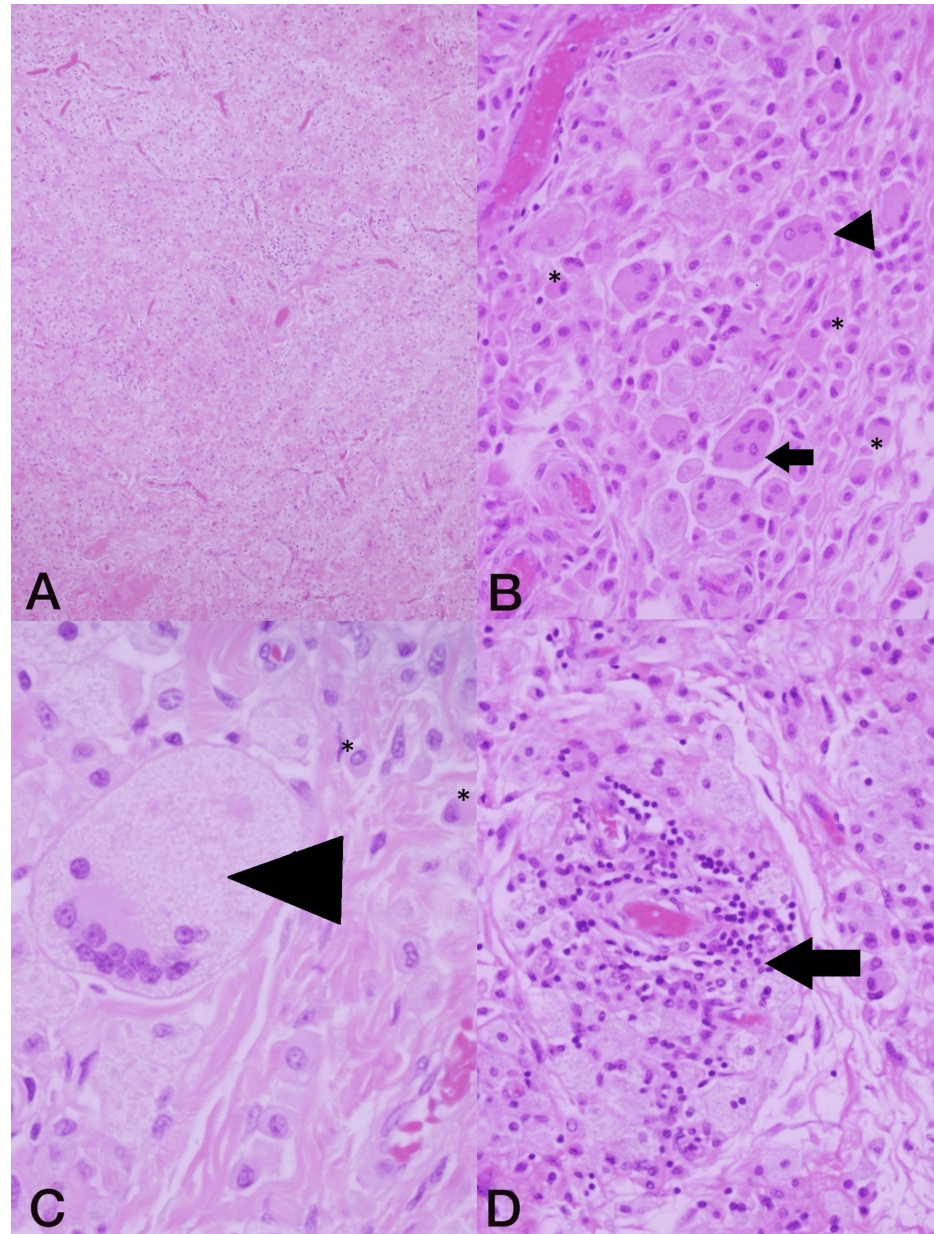


FIGURE 2: Histopathological features of the mass.

(A) $\times 4$ magnification: non-encapsulated, infiltrative lesion involving the deep dermis and subcutaneous tissue, composed of mature adipocytes admixed with numerous small, thin-walled vascular channels. (B-C) $\times 40$ magnification: plump spindle to plasmacytoid stromal cells (asterisk (*)) arranged haphazardly within the collagenous stroma and multinucleated giant cells, including Touton (not shown), Langhans-type (arrowhead), and osteoclast-like giant cells (arrow). (D) Chronic inflammatory infiltrate with lymphocytes (arrow) and histiocytes. All micrographs were stained with hematoxylin and eosin (H&E).

Immunohistochemistry showed that neoplastic adipocytes were diffusely positive for vimentin, which was non-specific. Diffuse CD68 positivity likely reflected the presence of histiocytic cells/macrophages within the lesion and does not indicate adipocytic lineage. S100 showed expression in the mature adipocytic component (Figure 3) and was negative in histiocytes and giant cells. Other markers showed no expression

in neoplastic cells (Table 1). Therefore, immunohistochemistry workup was mainly exclusionary rather than lineage-defining. These included CD1a, H-caldesmon, factor XIII, desmin, pancytokeratin, ALK, HMB45, CD38, CD138, and CD34. Kappa and lambda showed no restriction, and the Ki67 proliferation index was 1% (automated image counter). Although immunohistochemistry aided in characterization, the final diagnosis was determined chiefly by histopathological morphology. At the one-year follow-up examination, the patient remained clinically stable with complete wound healing and no evident local recurrence. Subsequently, she underwent surgical resection of the contralateral gluteal mass 13 months postoperatively. The second mass demonstrated similar histomorphology and immunohistochemical features to the present case. BRAF molecular testing was negative.

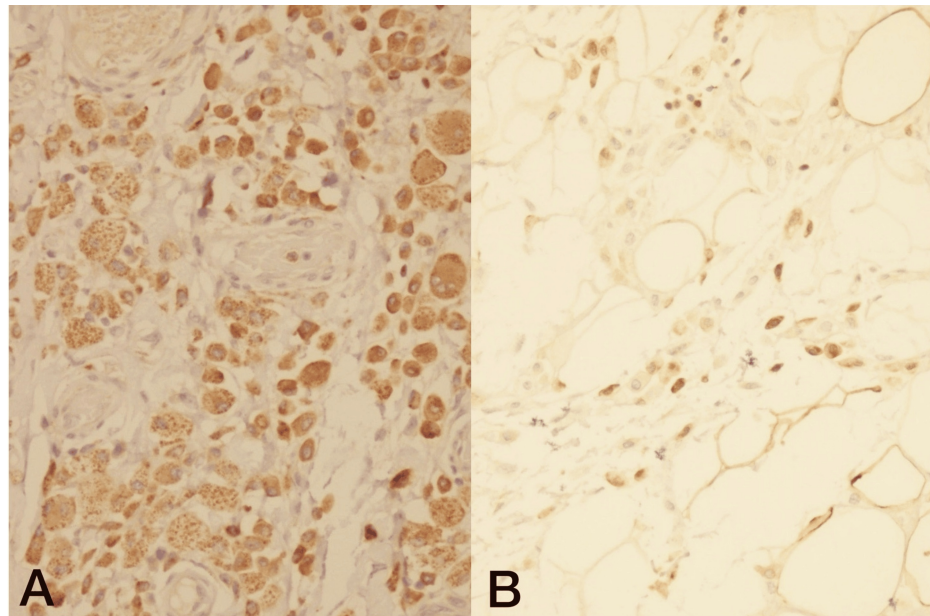


FIGURE 3: Immunohistochemical stains.

(A) $\times 10$ magnification: CD68 showing diffuse expression (granular cytoplasmic) in the plasmacytoid histiocytes.
(B) $\times 10$ magnification: S100 highlighting the mature adipocytes.

Immunohistochemistry marker	Result
CD68	Positive
S100	Positive
CDa1	Negative
H-Caldesmon	Negative
Factor XIII	Negative
Desmin	Negative
Pancytokeratin	Negative
ALK	Negative
HMB45	Negative
CD38	Negative
CD138	Negative
CD34	Negative
Kappa	No restriction
Lambda	No restriction

TABLE 1: Immunohistochemistry findings.
CD68 is diffusely positive in plasmacytoid histiocytes. S100 is expressed in neoplastic adipocytes. All other markers are negative in neoplastic cells.

Discussion

Infiltrating angiolipoma is a benign, unencapsulated histologic variant of angiolipoma composed of mature adipocytes and clustered thin blood vessels, in addition to exhibiting infiltrative growth into adjacent soft tissues [3]. To the best of our knowledge, we did not encounter an inflammatory plasmacytoid histopathological morphology in the English literature. Due to the presence of predominant plasmacytoid cells with granular to vacuolated cytoplasm, this finding widens the differential diagnosis and can be misdiagnosed as any tumor in the plasmacytoid cell category with granular cell change on the first examination. The differential diagnosis in this case included hibernoma (particularly the granular variant), granular cell tumor, xanthogranulomatous/histiocytic lesions, inflammatory myofibroblastic tumor, myofibroma, myopericytic lesions, atypical lipomatous tumor/well-differentiated liposarcoma, and vascular lesions with entrapped adipose tissue.

Although the lesion demonstrated scattered plasmacytoid cells with granular cytoplasm, the overall histomorphologic features, characterized by mature adipocytes and prominent capillary-sized vascular proliferation, supported a diagnosis of angiolipoma rather than hibernoma. Hibernoma typically exhibits brown fat differentiation with multivacuolated granular eosinophilic cytoplasm and characteristic cytologic features of brown adipocytes, which were not identified in this lesion.

Granular cell tumor was also considered, particularly on low-power examination due to the presence of granular cytoplasm and associated vascular proliferation. However, granular cell tumors demonstrate Schwannian differentiation and typically show diffuse S100 and SOX10 positivity, along with infiltrative nests and sheets of polygonal granular cells rather than a mature adipocytic proliferation.

Xanthogranulomatous reaction is included in the differential diagnosis due to the presence of giant inflammatory cells. However, the absence of a typical xanthogranulomatous pattern with haphazard arrangement of foamy histiocytes localized strictly to zones of degenerating fat or hemorrhage, and accompanying fibrosis, argued against this diagnosis. Also, the uniform plasmacytoid histiocyte component is intimately integrated throughout the vascular-adipose architecture without massive necrosis. Most strikingly is the uniformity of plasmacytoid morphology of the histiocytes which suggests a shift in plasticity driven by the tumor microenvironment associated with this specific case of infiltrating angiolipoma, rather than a standard, garden-variety reactive fat necrosis [15]. The tumor microenvironment and the role of tumor-associated macrophages in tumor progression are well established [15]. Although their role has mainly been described in malignant neoplasms, in our case, the homogeneous cytomorphology of these histiocytes in both masses favors tumor-directed recruitment. They may also have a role in the infiltrative

nature of these angiolipomas [15]. Or more plausibly, the cells transitioned from a scavenging xanthogranulomatous reaction driven by lipid-rich stroma toward immunosuppressive M2 polarization that promotes tumor progression [15].

Another consideration in the differential diagnosis is histiocytic disorders, such as Erdheim-Chester disease, which may exhibit variable cytoplasmic appearances and perivascular clustering of foamy histiocytes. However, they generally present with systemic involvement and characteristic skeletal or organ manifestations, which were not observed in this case. As noted previously, angiolipomas occur independently of systemic hyperlipidemia, implying that local tissue handling of lipids is a unique tumor microenvironmental tissue response rather than a reflection of a systemic metabolic storage disorder.

The lesion showed limited overlap with myofibroblastic tumors due to focal plasmacytoid cells and associated inflammation. Nevertheless, the absence of a spindle cell proliferation, fascicular architecture, or supportive myofibroblastic immunophenotype excluded inflammatory myofibroblastic tumor and myofibroma and myopericytic lesions.

Malignant adipocytic neoplasms were also considered. However, the lack of cytologic atypia in adipocytes and endothelial cells, the absence of lipoblasts, fibrosis, or atypical stromal cells, and the overall benign morphology argued against atypical lipomatous tumor/well-differentiated liposarcoma and other low-grade sarcomas. Vascular lesions with entrapped adipose tissue were excluded based on the organized proliferation of adipose tissue with characteristic capillary vascularity rather than a primary vascular neoplasm.

The granular and vacuolated cytoplasmic changes observed in some adipocytes were interpreted as degenerative or metabolic alterations within adipocytic elements rather than evidence of skeletal muscle differentiation, as seen in adult rhabdomyoma. This interpretation was supported by the absence of rhabdomyomatous architecture, cross-striations, and myogenic marker expression.

Taken together, the morphologic features, immunohistochemical findings, and clinical-radiologic correlation supported the diagnosis of inflammatory infiltrating angiolipoma, with the granular and vacuolated cytoplasmic changes representing a distinctive degenerative/metaplastic alteration within the adipocytic component. The previously excised mass from the gluteal region was verbally reported by the patient to be benign and similar in appearance to the current masses. Unfortunately, we could not retrieve a histopathological report to document the diagnosis and tumor recurrence. The patient would be followed up for a long period to establish further recurrence. No genetic testing was offered at the current time.

Several cases of infiltrating and non-infiltrating angiolipomas in the head and neck have been reported in the literature [16–18]. Only two cases that match the WHO definition of angiolipoma with infiltrative growth are summarized here. The first described an infiltrative angiolipoma of the cheek in an infant, an uncommon age and location for this entity. Radiologic studies revealed an ill-defined deep soft tissue mass. Histopathology demonstrated mature adipocytes with a conspicuous capillary-type vascular component, increased cellularity, and infiltration into surrounding tissues, including muscle. No cytologic atypia or malignant features were identified [16]. Although molecular studies were not performed, the morphologic features supported classification as infiltrating angiolipoma, underscoring that infiltrative growth may occur in pediatric cases without implying aggressive behavior [2].

The second, although not a typical angiolipoma, is a large infiltrative angiolipoma of the back presenting as an extensive soft tissue mass. Imaging demonstrated a poorly marginated lesion with mixed fatty and vascular components, suggestive of a complex benign process. Histologic evaluation showed mature adipose tissue admixed with prominent vascular channels and infiltrative growth into adjacent soft tissues. The lesion was associated with a venous malformation, and molecular analysis identified somatic alterations within the PI3K/AKT pathway, consistent with changes reported in vascular malformations [19]. These findings highlight the diagnostic overlap between infiltrative angiolipoma and vascular anomalies while supporting an adipocytic primary component [12]. Table 2 summarizes these studies along with our case.

Study	Age/gender	Anatomic location	Radiographic description (CT or MRI)	Histopathologic description	Treatment and prognosis	Comments
Shahi et al. [2]	9 months/female	Cheek: buccal mucosa	Contrast-enhanced CT showed a 1.7 cm heterogeneously enhancing, predominantly hypodense lesion without underlying bony erosion or destruction	Well-delineated adipocyte lobules separated by loose fibrous anastomosing septa with numerous endothelial-lined spaces of varying sizes	Surgical excision No recurrence in 1.5 years	This location involves the mucosal surface in an infant which may account for a congenital cause or an emerging different entity
Zhao et al. [19]	35 years/male	Trunk	CT and MRI showed the tumor invaded the deep fascia with diffuse thickening of the soft tissues, and vascular malformations were distributed in the adipose tissue	Infiltrative lesion composed of mature adipocytes admixed with abundant thin-walled vascular channels, extending into surrounding soft tissue and associated with venous malformation; no cytologic atypia or malignancy	Surgical excision	Not a typical infiltrating angioliipoma. Started at age 3 years
					No recurrence in 6 months	A genetic mutation is identified in FCGR2A, BMP5, MUC2, and KRTAP4-9
Present case	25 years/female (onset at 7 years of age)	Arms and buttocks	Large mass; heavily infiltrated fatty tissue with high T2 signal intensity and intermediate T1 signal intensity with the persistence of speckles of fat	Lobules of mature adipocytes with numerous small capillary-type vessels; haphazard spindle to plasmacytoid stromal cells with chronic lymphohistiocytic inflammation and occasional multinucleated giant cells; no cytologic atypia	Possibly recurred after 17 years	Diagnosed according to the WHO distinction between intramuscular hemangioma and angioliipoma with infiltrating growth pattern

TABLE 2: Summary of infiltrating angioliipoma cases diagnosed according to the current definition of WHO

CT: computed tomography; MRI: magnetic resonance imaging; WHO: World Health Organization

Infiltrating angioliipoma is a rare entity, and involvement of the bilateral gluteal region appears to be exceedingly uncommon. Interestingly, our patient was seven years old when the first mass appeared, which is uncommon at this age [1]. But, given the slow-growing nature and the reassurance of a “benign growth” that our patient received after excision of the first mass at another institution, she opted out of surgical removal until the age of 25 years for cosmetic reasons. Management of angioliipoma is based primarily on complete surgical removal for both infiltrating and non-infiltrating variants [3]. Excision of infiltrating lesions may be technically challenging because of their locally invasive growth, resulting in an increased risk of local recurrence [3]. When surgical margins are inadequate, adjuvant radiotherapy has been employed [3]. Our patient did not have a family history of solitary or multiple angioliipomas. However, multiple angioliipomas raise the concern of a familial or genetic cause [12,20]. Reliable recurrence rates are difficult to ascertain due to heterogeneity and inconsistency in nomenclature and diagnostic classification across published reports.

Conclusions

Herein, we present a case of infiltrating angioliipoma notable for distinctive plasmacytoid histiocytic and giant cell-rich inflammatory infiltrates. This underrecognized morphology needs to be distinguished from a

variety of neoplasms, ranging from hematologic to mesenchymal, through careful and intentional clinicopathological correlation. Emphasizing histopathological morphology is particularly important to distinguish unique variants and prevent misdiagnosis for accurate prognostication and optimal clinical management.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Afaf Shaabi, Fahd Refai, Sarah Alghamdi

Acquisition, analysis, or interpretation of data: Afaf Shaabi, Fahd Refai, Sarah Alghamdi

Drafting of the manuscript: Afaf Shaabi

Critical review of the manuscript for important intellectual content: Afaf Shaabi, Fahd Refai, Sarah Alghamdi

Supervision: Fahd Refai, Sarah Alghamdi

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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