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Cutaneous Angiosarcoma Post-Mastectomy and Chronic Lymphedema: A Dermatologic-Oncologic Perspective on Diagnosis and Management

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Abstract

Secondary cutaneous angiosarcoma (AS) is a rare but highly aggressive vascular malignancy that poses a critical, long-term challenge for breast cancer (BC) survivors. The tumor typically arises years after treatment, driven by radiation therapy (RT) (post-radiation angiosarcoma, RAAS) or chronic lymphedema (Stewart-Treves syndrome, STS). The cumulative incidence is estimated at 0.16%–0.43% post-mastectomy and RT, with a median presentation age of 67–68 years and a median latency of 6–7 years. Early dermatologic recognition is critical, as AS often presents nonspecifically as violaceous patches, plaques, or hematoma-like lesions, easily mimicking benign conditions and leading to significant diagnostic delays.

Diagnosis requires a deep biopsy, which should be confirmed by immunohistochemistry (IHC) (CD31, ERG, FLI-1) and molecular markers, notably MYC amplification, which is highly characteristic of secondary AS and absent in benign mimics. Management requires a complex multidisciplinary approach, integrating wide surgical excision (aiming for negative margins) with systemic therapy, commonly paclitaxel. Prognosis is poor, with a median overall survival (OS) of approximately 25 months and a high rate of local and distant recurrence. Long-term dermatologic surveillance focusing on early detection of subtle skin changes is essential for all BC survivors, particularly those with chronic lymphedema or a history of extensive RT.

Keywords: Angiosarcoma; Radiation-Associated Sarcoma; Lymphedema; Magnetic Resonance Imaging; Breast Cancer

Abbreviations: AS: Angiosarcoma; BC: Breast Cancer; RT: Radiation Therapy; RAAS: Radiation-Associated Angiosarcoma; STS: Stewart-Treves Syndrome; BCT: Breast-Conserving Therapy; OS: Overall Survival; DSS: Disease-Specific Survival; R0: Complete Resection (Negative Margins); FISH: Fluorescence In Situ Hybridization; IHC: Immunohistochemistry; AVLs: Atypical Vascular Lesions; NGS: Next-Generation Sequencing; MRI: Magnetic Resonance Imaging; PET-CT: Positron Emission Tomography–Computed Tomography

Introduction

Secondary cutaneous angiosarcoma represents a rare but highly aggressive malignancy arising from vascular endothelial cells in the skin, most observed as a late complication following treatment for primary breast cancer [1,2]. These tumors are characterized by infiltrative growth, local recurrence, and a generally poor prognosis [2]. As a significant long-term complication in survivorship, secondary cutaneous angiosarcomas account for approximately 1-2% of all soft tissue sarcomas [3]. The incidence is closely linked to prior iatrogenic factors, particularly mastectomy followed by radiation therapy, with estimated cumulative incidence rates ranging from 0.16% to 0.43% in this high-risk group [1]. The latency period between the initial cancer treatment and the development of the angiosarcoma typically spans 4 to 15 years, although presentation decades after initial therapy is also possible [4]. Risk factors contributing to this complication include higher radiation doses, larger radiation fields, and the overall duration of follow-up [2].

The context of secondary angiosarcoma in breast cancer survivors involves two distinct clinicopathologic entities: Stewart-Treves syndrome (SST) and post-radiation angiosarcoma [3]. SST specifically describes angiosarcoma developing within a chronically lymphedematous limb, typically the upper extremity following axillary lymph node dissection, and often occurs without prior radiation [1]. In contrast, post-radiation angiosarcoma develops directly in the skin of the chest wall or upper extremity within the radiation field, often independently of significant lymphedema [2]. This distinction is clinically relevant, as SST accounts for approximately 0.45% of lymphedema cases and generally presents after a longer latency period, while post-radiation angiosarcoma is increasingly recognized and has a shorter interval to presentation [4].

Though they share features, these two entities may harbor distinct molecular alterations, underscoring the value of recognizing this difference for appropriate risk stratification and patient counseling [3]. Early dermatologic recognition is critical for improving patient outcomes, as a delayed diagnosis severely impacts prognosis and limits therapeutic options [2]. Dermatologists and primary care physicians must maintain a high index of suspicion for suspicious skin lesions in the chest wall or upper extremity of breast cancer survivors [1]. Angiosarcomas often present nonspecifically as rapidly enlarging violaceous or hemorrhagic patches, plaques, or nodules, frequently accompanied by ecchymosis or edema [1-5].

Because these subtle presentations can easily mimic benign conditions such as dermatitis, cellulitis, or simple hemangiomas, diagnostic delays of several months are common, allowing the tumor to progress to advanced stages [3]. Therefore, comprehensive skin examination should be a routine part of long-term surveillance for all breast cancer survivors, particularly those with a history of extensive radiation or chronic lymphedema [4]. Urgent dermoscopic evaluation and histopathologic confirmation are mandatory for any suspicious vascular lesion, as biopsy remains the diagnostic gold standard [2]. Education for both survivors and healthcare providers regarding these clinical features and risk factors is an essential preventive strategy to enable earlier intervention and potentially enhance therapeutic success in this high-risk population [1].

Clinical Presentation and Dermatologic Features

Angiosarcoma arising in the context of breast cancer treatment can be broadly classified into three distinct clinical categories: primary (spontaneous) breast angiosarcoma, radiation-associated angiosarcoma, and Stewart–Treves syndrome (STS) [6]. Primary breast angiosarcoma is exceedingly rare, representing less than 0.01% of all breast malignancies and typically affecting younger patients, often between 20 and 40 years of age. In contrast, secondary angiosarcomas are those arising after treatment and are diagnosed at a significantly older median age. A review by Abbott and Palmieri reported a median age at presentation of 67 years for secondary angiosarcoma following mastectomy and radiotherapy, with an average latency period of approximately 72 months between radiation and tumor onset [6]. Stewart–Treves syndrome (STS), a distinct form of secondary angiosarcoma, develops in the setting of long-standing lymphedema of the upper limb following mastectomy and axillary lymph node dissection.

The estimated incidence of STS is around 0.07% among patients who undergo axillary dissection, typically appearing approximately ten years after the initial surgery [6]. Due to surgical advancements, the occurrence of STS is thought to be gradually declining [6]. Studies of radiation-associated angiosarcoma (RAAS) show a consistent clinical presentation profile. A review of 21 patients in the Netherlands between 1987 and 1995 determined the median age at diagnosis of RAAS to be 68 years, with the time between breast-conserving therapy (BCT) and the appearance of the angiosarcoma averaging 74 months [7]. The latency period for sarcoma development tends to be shorter in patients who receive BCT combined with radiotherapy compared to more radical procedures [8]. Angiosarcoma exhibits one of the shortest latency periods among radiation-associated sarcomas, usually manifesting between five- and ten-years post-treatment [3,8].

Dermatologic Features and Clinical Correlates

The clinical presentation of cutaneous angiosarcoma is highly variable, often posing a diagnostic challenge due to its rarity and its capacity to mimic benign skin conditions [6]. Skin changes are universally observed in RAAS [7]. The most common features observed include reddish-purple discolorations, hematoma-like lesions, and papular or vesicular changes [7]. Many patients present with a mix of these features in a single area, and multifocal presentation is common in post-radiation cases [7]. Generally, the clinical features can be correlated with the tumor grade:

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low-grade lesions often present as painless, slowly progressive erythematous-violaceous papules or nodules, sometimes with satellite lesions, whereas high-grade lesions typically begin as a red patch that rapidly enlarges, darkens, and becomes prone to bleeding and ulceration [3]. A key clinical clue is the presence of violaceous nodules or purplish discoloration over the irradiated area [8]. The distribution of lesions is crucial for clinical distinction: STS characteristically involves the ipsilateral arm due to chronic lymphedema, while RAAS typically develops within the skin of the breast or chest wall previously exposed to the radiation field [8]. The cutaneous subtype of angiosarcoma, characterized by involvement limited to the skin without extension into the breast parenchyma, is considered a separate clinical entity [9].

Diagnostic Considerations and Differential Diagnosis

Diagnosing cutaneous angiosarcoma is difficult because early lesions commonly resemble benign vascular or inflammatory conditions, which can lead to delayed or incorrect diagnosis [10]. Initial presentations may be subtle, such as patches of bluish discoloration, flat or nodular angioma-like lesions, or atypical telangiectatic patches [10]. As the tumor progresses, lesions may infiltrate deeper tissue, leading to edema, fungation, ulceration, or bleeding [10]. The presence of an enlarging mass, sometimes accompanied by pain, suggests deeper soft tissue or visceral involvement [3]. Metastatic spread occurs primarily via the hematogenous route, with the lungs being the most common site [3].

The differential diagnosis for a suspicious vascular lesion in the post-treatment field is broad and includes:

- Benign Vascular Lesions: Capillary or cavernous hemangioma, pyogenic granuloma, epithelioid hemangioma, and post-radiation atypical vascular lesions [3,9].
- Intermediate-Grade Tumors: Kaposi sarcoma and epithelioid hemangioendothelioma [9].
- Other Malignancies: Malignant melanoma and metastatic carcinomas [3,9].

Diagnostic Approach

The diagnostic approach to secondary cutaneous angiosarcoma must prioritize a deep, representative skin biopsy, because superficial sampling may miss the tumor's infiltrative component and lead to false negatives. Incisional biopsies or punch biopsies that include both dermis and subcutis are strongly recommended to ensure accurate diagnosis [11]. Under microscopic examination, the characteristic features include irregular, anastomosing vascular channels, atypical endothelial cells, frequent mitotic figures, and hemorrhagic areas, findings that help distinguish angiosarcoma from benign vascular proliferations [12].

Immunohistochemistry is fundamental; markers such as CD31, ERG, and FLI-1 confirm an endothelial origin, while MYC overexpression or amplification is commonly observed in secondary angiosarcomas, particularly those arising after radiation or chronic lymphedema [13]. Given the morphological complexity and overlap with other spindle-cell or vascular lesions, consultation with a dermatopathologist experienced in soft-tissue tumors is highly advisable [13]. For staging, imaging studies should include MRI to evaluate local extent and soft-tissue involvement, and PET-CT to identify multifocal disease or metastases. In selected patients, sentinel lymph node evaluation may also be considered as part of the regional staging strategy [14].

Oncologic Management

Management of secondary cutaneous angiosarcoma requires a multidisciplinary strategy that integrates surgical, systemic, and radiation-based treatments. Wide surgical excision or radical resection aiming for negative margins is the foundation of therapy, although achieving such margins can be challenging due to the tumor's aggressive and infiltrative nature [15]. Adjuvant systemic therapy usually involves chemotherapy: weekly paclitaxel has demonstrated efficacy in both localized and metastatic settings, and liposomal doxorubicin is an alternative for patients who do not tolerate taxanes or whose disease has progressed [13]. Postoperative radiotherapy may be utilized when surgical margins are close or positive to improve local disease control [15].

Emerging therapies include anti-angiogenic agents such as pazopanib, which target VEGF-driven pathways implicated in tumor angiogenesis [13]. Additionally, immune checkpoint inhibition, particularly with PD-1 inhibitors, has shown promising results in some cases of cutaneous angiosarcoma; biomarkers such as tumor mutational burden and PD-L1 expression may help identify patients most likely to benefit [16]. Because of the disease's rarity, complexity, and high propensity for recurrence, a coordinated care team—comprising dermatology, surgical oncology, plastic surgery, medical oncology, and radiation oncology—is essential to optimize both oncologic outcomes and patient quality of life [13,15].

Prognosis and Follow-Up

The prognosis for secondary cutaneous angiosarcoma (AS), particularly following mastectomy and radiotherapy for breast cancer, remains poor, underscoring the necessity of rigorous, long-term dermatologic surveillance [17-20]. The clinical course is highly aggressive, characterized by a high recurrence rate and early metastatic behavior [20,22]. Studies have consistently shown that more than half of all patients experience local or distant recurrence [20]. A retrospective analysis of secondary angiosarcoma reported a median latency period of approximately

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seven years (range, 3–25 years) between primary breast treatment and AS onset [22]. Despite aggressive initial management, local recurrence developed in 19 of 31 patients, with a short median time to recurrence of six months [22]. Regional and distant metastases occurred in 13 patients after a median of 17 months, often overlapping with local recurrence [22].

Survival Outcomes and Prognostic Factors

Survival outcomes are generally dismal, with the median overall survival (OS) for cutaneous AS post-mastectomy reported to be around 25 months [17]. The median disease-specific survival (DSS) is just over three years [22]. The 5-year overall survival rate remains low. However, several factors are consistently associated with prognosis. Tumor size at diagnosis and the status of resection margins are critical prognostic factors [23-26]. Although two-thirds of patients may develop local recurrence even after an R0 (complete) resection, obtaining clear surgical margins is still considered key to improving overall survival [22-26]. Furthermore, the ability to surgically treat local recurrence is strongly linked to better outcomes; patients whose local recurrence was amenable to surgery had a significantly better survival (median 34 months) compared to those who were not surgical candidates (median 6 months) [22].

For secondary angiosarcoma arising in the context of chronic lymphedema (Stewart-Treves syndrome), the survival rate of patients who underwent amputation was found to be significantly higher than that of non-amputated patients [17]. Conversely, some studies suggest that treatment modalities such as extended resection, chemotherapy, or radiotherapy may not lead to significant survival improvement in the overall cohort [17,22]. Molecular factors also influence prognosis. MYC amplification has been consistently associated with a shorter survival rate in secondary angiosarcoma, making it a valuable prognostic biomarker [21,22]. Conversely, older age and specific treatment modalities for the angiosarcoma itself may not significantly impact overall survival [20].

Long-Term Dermatologic Surveillance Strategies

Given the aggressive nature and poor prognosis, long-term dermatologic follow-up is essential for breast cancer survivors with a history of radiation or lymphedema. These surveillance strategies must focus on early detection of recurrence, which often presents subtly as new purpuric patches or nodules within the treated field [17]. Regular, comprehensive skin examinations by dermatologists or oncologists are necessary to screen for multifocal recurrence and monitor the potential development of new lesions. Prompt biopsy of any suspicious, rapidly evolving lesion is mandatory to avoid diagnostic delay, which remains a significant factor worsening patient prognosis [21].

Preventive and Future Perspectives

Given the poor prognosis of secondary angiosarcoma (AS), prevention and early detection are essential. Patients with chronic post-mastectomy lymphedema should undergo early and frequent skin surveillance. Timely assessment and management of lymphedema play a central role in reducing the risk of secondary AS, and these measures are practical in routine care [3,18,27]. Patient education on self-monitoring is also essential. Any new red-blue macules or plaque-like lesions warrant immediate evaluation [18]. Early detection also depends on reliable molecular biomarkers. Recent studies have demonstrated MYC overexpression and amplification identified by fluorescence in situ hybridization (FISH) and immunohistochemistry (IHC) in nearly all cases of secondary cutaneous AS [21,28-30].

This alteration is absent in primary AS and atypical vascular lesions (AVLs), making MYC a highly promising diagnostic indicator [21]. In addition, E. Styrring et al. reported upregulation of KIT and RET and downregulation of CDKN2C in secondary AS [29]. CD31 and factor VIII–related antigen may further support the diagnosis in cutaneous AS post-mastectomy [7]. By contrast, imaging has limited value in early detection compared with tissue biopsy and molecular biomarkers. Mammography and ultrasound typically show non-specific findings such as poorly defined lesions, lack of calcification in breast AS [3,18,31,32]. In addition, CT and ultrasound demonstrate non-specific imaging features such as 'subcutaneous oedema' and 'fascial septal thickening' in the edematous upper limbs [3,18].

MRI may aid in diagnosis; Rapidly enhancing dermal lesions with high T2 signal intensity may be seen in high-grade lesions [18]. Because of the tumor's rarity and heterogeneity, standardized diagnostic and therapeutic strategies remain undeveloped. Larger, multicenter investigations, including observational cohorts, randomized controlled trials, and clinical registries, are needed to define biopsy thresholds and validate highly sensitive and specific markers for early diagnosis and effective treatment. Emerging methods such as multi-omics profiling and next-generation sequencing (NGS) may allow for detailed tumor characterization and facilitate individualized management approaches.

Conclusion

Second, cutaneous angiosarcoma represents one of the most devastating late-stage complications of breast cancer treatment. Its high aggression, early metastatic potential, and poor overall survival, combined with a nonspecific and mimicry-prone dermatologic presentation, necessitate a fundamental shift toward proactive, specialized care. The diagnostic pathway must integrate a high index of suspicion, timely deep biopsy, and molecular confirmation using biomarkers like MYC amplification to overcome the challenges of delayed diagnosis.

While aggressive multimodal therapy-anchored by wide surgical excision and taxane-based chemotherapy-remains the standard of care, the persistent high recurrence rates underscore the need for more effective systemic agents. Future strategies

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must focus on personalized medicine via multi-omics profiling, further investigating the role of immune checkpoint inhibitors, and establishing standardized, evidence-based long-term dermatologic surveillance programs to improve outcomes and quality of life for this high-risk population.

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