






CASE REPORT

## Radiation-induced breast angiosarcoma: a case report

### *Angiosarcoma mamario radioinducido: reporte de un caso*

Laura Juliana Delgado-Mateus<sup>1</sup>, Néstor Eduardo Llinás-Quintero<sup>2</sup>, Juan Paulo Sandoval-Mesa<sup>3</sup>,  
Laura Herrera-Vásquez<sup>2</sup>, Fernando Alfredo Herazo-Maya<sup>4</sup>

<sup>1</sup> Resident of the Specialization in Mastology, Universidad CES, Medellín, Colombia.

<sup>2</sup> Clinical Oncology, Fundación Colombiana de Cancerología Clínica Vida, Medellín, Colombia.

<sup>3</sup> Clinical Pathology, Fundación Colombiana de Cancerología Clínica Vida, Medellín, Colombia.

<sup>4</sup> Mastology, Fundación Colombiana de Cancerología Clínica Vida, Medellín, Colombia.

**Date of submission:** 27/08/2025  
**Date of acceptance:** 03/12/2025  
**Available online:** 02/03/2026

### Resumen

El angiosarcoma mamario radioinducido (AMRI) es una complicación infrecuente y agresiva del tratamiento para el cáncer de mama. A continuación, se presenta el caso de una mujer de 45 años con antecedente de carcinoma de mama en estadio IIB, tratada con cirugía conservadora y linfadenectomía axilar, seguida de quimioterapia, radioterapia adyuvante y terapia endocrina. Tras cinco años libres de enfermedad después de finalizar el tratamiento, la paciente desarrolló lesiones cutáneas en el sitio irradiado, con histología que confirmó AMRI, por lo que fue manejada con mastectomía con márgenes negativos y quimioterapia adyuvante con paclitaxel semanal. Durante el tratamiento adyuvante, presentó una recurrencia local que requirió resección amplia y reirradiación sobre el lecho quirúrgico. La paciente completó seis meses de supervivencia libre de enfermedad. El objetivo de este reporte de caso fue resaltar los retos en el diagnóstico y tratamiento del AMRI.

**Palabras clave:** angiosarcoma; neoplasias de la mama; neoplasias inducidas por radiación; radioterapia; recurrencia local de neoplasia; inmunohistoquímica; mastectomía; márgenes de escisión; quimioterapia adyuvante.

### Conflict of interests

The authors declare no conflicts of interest.

### Citation

Delgado-Mateus L, Llinás-Quintero N, Sandoval-Mesa J, Herrera-Vásquez L, Herazo-Maya F. Radiation-induced breast angiosarcoma: a case report. Rev Col Cancerol. 2026;30(1):23-30. <https://doi.org/10.35509/01239015.1142>

### Corresponding author

Laura Juliana Delgado Mateus  
Resident of the Specialization in Mastology,  
Universidad CES, Medellín, Colombia.

### Email:

[lauradelgado.md@gmail.com](mailto:lauradelgado.md@gmail.com)

### Abstract

Radiation-induced angiosarcoma of the breast (RIAS) is a rare but aggressive complication of breast cancer treatment. We present the case of a 45-year-old woman with a history of stage IIB breast carcinoma treated with breast-conserving surgery and axillary lymphadenectomy, followed by adjuvant chemotherapy, radiotherapy, and endocrine therapy. After a five-year disease-free period following treatment, the patient developed cutaneous lesions within the previously irradiated field, with histopathology confirming RIAS. She subsequently underwent a mastectomy with negative surgical margins, followed by weekly adjuvant paclitaxel chemotherapy. During adjuvant therapy, she developed a local recurrence that

required wide local excision and re-irradiation of the surgical bed. As of this report, the patient has completed six months of disease-free follow-up. This case highlights the diagnostic and therapeutic challenges of RIAS.

**Keywords:** angiosarcoma; breast neoplasms; neoplasms, radiation-induced; radiotherapy; neoplasm recurrence, local; immunohistochemistry; mastectomy; margins of excision; chemotherapy, adjuvant.

## Introduction

Breast cancer is the most common neoplasm in women worldwide, with an incidence of 23.8% (1). Although conservative treatment is the standard, adjuvant radiotherapy poses the risk of developing secondary neoplasms. Among these is radiation-induced angiosarcoma of the breast (RIAS), a malignant tumor of mesenchymal origin that develops in previously irradiated skin (2-3). The diagnostic criteria for RIAS include a) location within the radiation zone; b) histology different from that of the primary tumor; c) a minimum latency period of 3-4 years from the initial radiation to the appearance of the angiosarcoma; and d) histological difference from the primary angiosarcoma (PA) (3-5).

In a cohort of 184,000 breast cancer patients treated with radiotherapy in the Netherlands, 209 (0.1%) developed RIAS (6). These results are consistent with studies reporting similar incidence rates of approximately 0.9 per 1,000 patients (2, 7). We present the case of a patient diagnosed with and treated for RIAS at an oncology center in Medellín, Colombia, to highlight the clinical and histopathological findings of this rare and aggressive neoplasm, whose management posed diagnostic and therapeutic challenges.

## Clinical case description

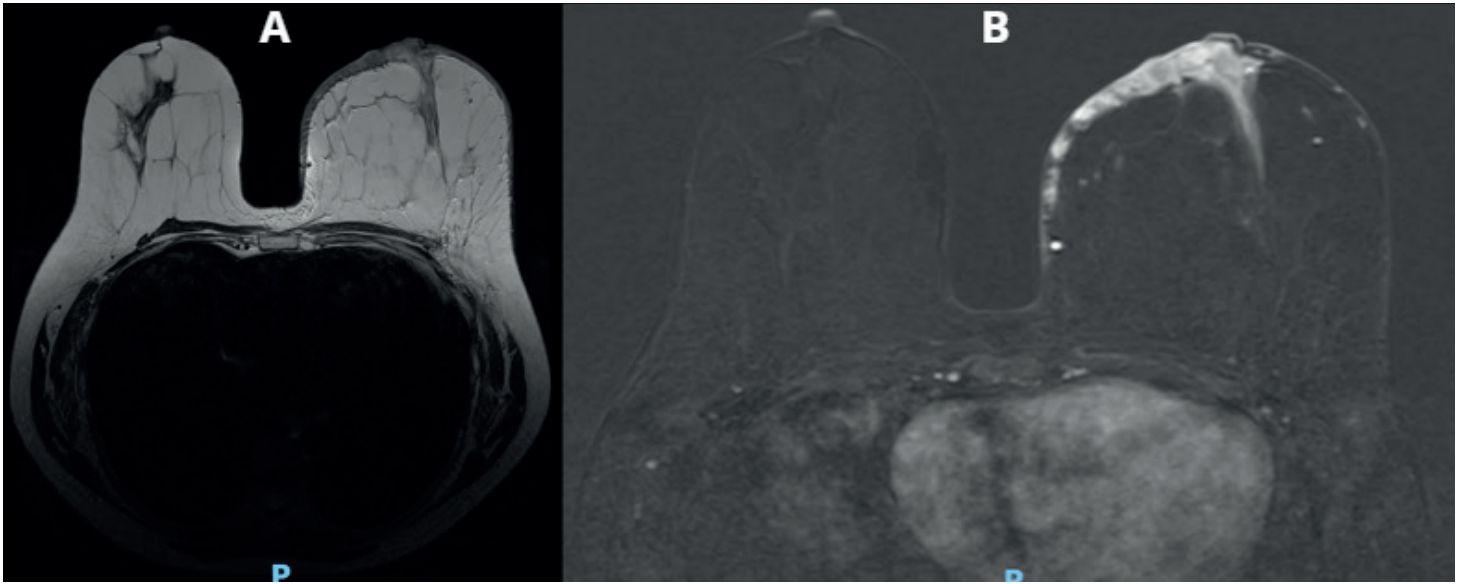
A 45-year-old woman with a history of luminal, stage IIB, infiltrating ductal carcinoma of the left breast. Genetic testing was negative. The patient received multimodal treatment including quadrantectomy, axillary lymphadenectomy with pathology report of pT2N1, followed by

adjuvant chemotherapy with doxorubicin-cyclophosphamide (doxorubicin [Adriamycin] and cyclophosphamide; AC) and paclitaxel, radiotherapy (40 Gy in 15 fractions), and endocrine therapy with tamoxifen and ovarian suppression, planned for 10 and 5 years, respectively.

After five years of adjuvant endocrine therapy, the patient presented with erythema, edema, and indurated violaceous macules on her left breast, involving the nipple-areola complex and the skin of the inner quadrants (Figure 1). Contrast-enhanced breast magnetic resonance imaging revealed edema and abnormal dermal uptake in the breast, associated with nonfocal mass enhancement in the retroareolar region (Figure 2). The punch biopsy was positive for ERG, D240, CD31, and CD34 markers; negative for CK7, HER2, FXIIIa, CD163, HMB-45, SOX-10, and HHV-8; and showed a Ki-67 tumor proliferation index of 10.0%, findings compatible with angiosarcoma.

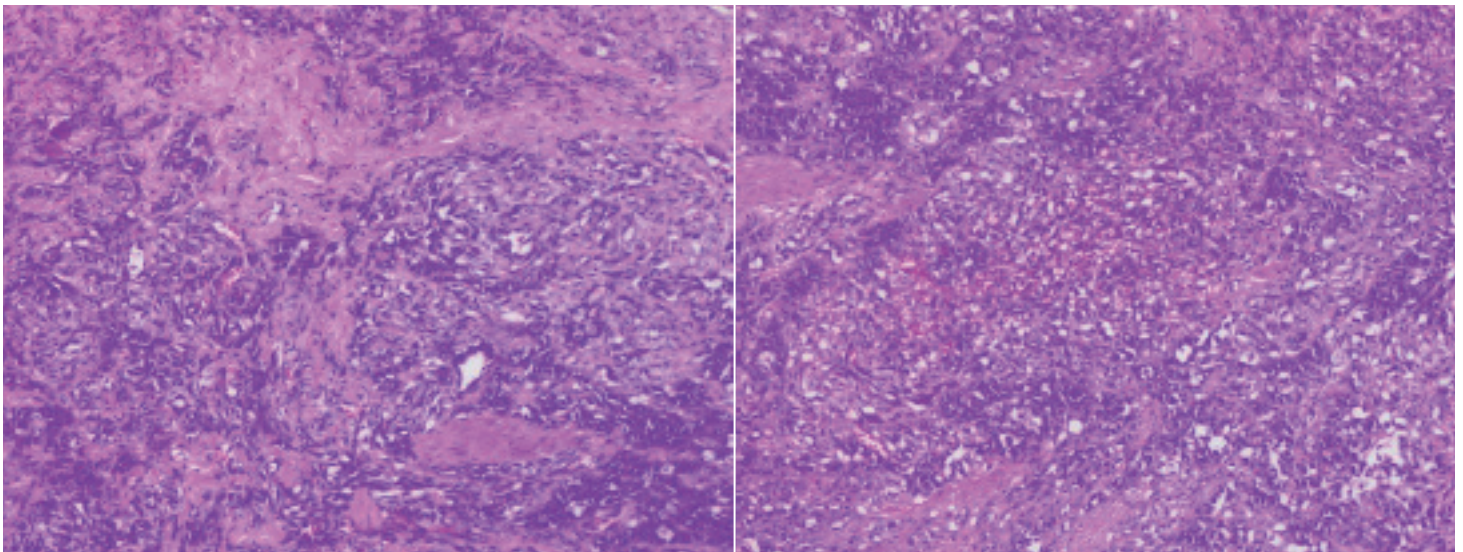


**Figure 1.** Clinical photograph of the left breast. Cutaneous involvement due to RIAS in the left breast, characterized by indurated erythematous macules and plaques, skin edema, and involvement of the nipple-areola complex and the skin of the inner quadrants.

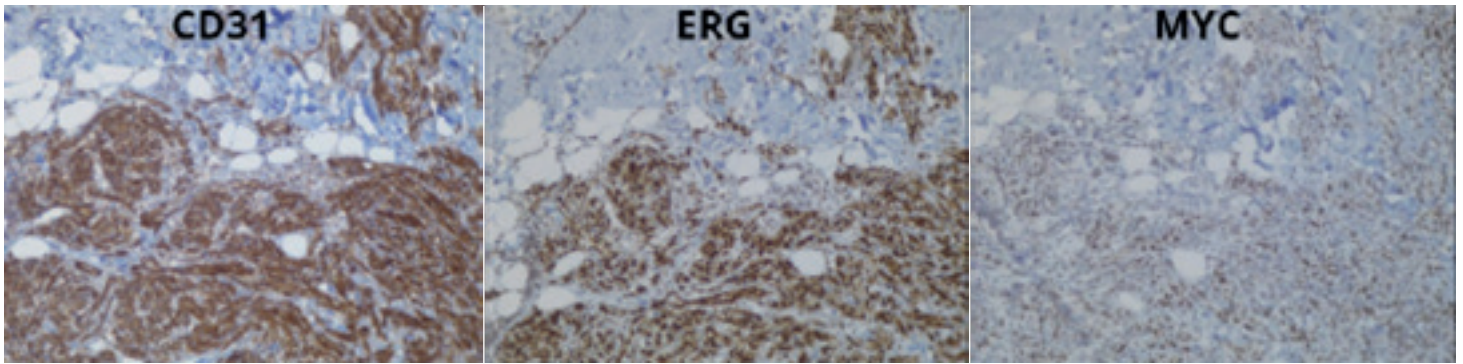


**Figure 2.** Contrast-enhanced breast magnetic resonance imaging. **A.** T1 sequence without fat suppression, showing thickening of the skin and subcutaneous tissue of the left breast. **B.** T1 sequence with fat suppression and contrast phase, showing skin enhancement in the left breast consistent with tumor involvement.

The initial treatment was a modified radical mastectomy. The pathological examination of the surgical specimen reported a malignant mesenchymal tumor proliferation measuring 60 mm × 44 mm, characterized by spindle cells with severe atypia and a high mitotic rate (>20 per 10 high-power fields [HPFs]), arranged in fascicles in a “fishbone” pattern, areas of vascular clefts with erythrocytes, diffusely invading adjacent tissues, and causing skin ulceration. The surgical margins were negative (>14 mm), while the c-MYC marker was positive, thus confirming the diagnosis of RIAS (Figures 3-4).



**Figure 3.** Hematoxylin-eosin staining. 400X. Neoplastic endothelial cells with marked atypia and mitotic activity.



**Figure 4.** Immunohistochemistry. On the left, positive CD31 staining in neoplastic endothelial cells. In the center, positive nuclear staining for ERG in neoplastic cells. On the right, positive nuclear staining for MYC in neoplastic cells.

Subsequently, the patient received weekly paclitaxel chemotherapy. During treatment, she experienced an early relapse, characterized by subcutaneous permeation nodules adjacent to the surgical scar. A wide local resection was performed, with histological findings of atypical vascular proliferation and immunohistochemistry (IHC) markers positive for CD31, ERG, and MYC, confirming recurrent RIAS. The case was reviewed by a multidisciplinary team, which decided to administer adjuvant re-irradiation to the surgical bed, with a total dose of 4,500 cGy. Currently, six months after completing treatment, the patient remains in follow-up with no evidence of relapse.

## Discussion

RIAS is a rare endothelial mesenchymal neoplasm, with an incidence of 0.01% (6). It typically occurs in women who have undergone radiotherapy as treatment for breast carcinoma and is linked to high rates of recurrence and mortality, as noted by Morgan *et al.* (8). Historically, secondary angiosarcoma was associated with lymphedema caused by radical axillary lymphadenectomy (Stewart-Treves syndrome), before the widespread use of radiotherapy in conservative breast cancer treatment (9).

RIAS differs from PA in clinical and histopathological features. PA originates in the breast parenchyma, can affect younger patients, and does not require prior exposure to radiotherapy. In contrast, RIAS originates from the radiation-affected dermis and usually develops at older ages (8).

## Histopathology and immunohistochemistry

RIAS is characterized by aberrant blood vessels invading the dermis. This tumor has complex anastomotic channels, poorly defined infiltrative borders, blood pools caused by extravasation, and endothelial cell proliferation with papillae projecting into the vessel lumen, characterized by enlarged, pleomorphic nuclei and prominent nucleoli (10). In IHC, positivity for the CD31 and ERG markers is the most sensitive and specific finding, while CD34 is sensitive but not exclusive, as it can be positive in other sarcomas. Ki67 is predominantly elevated in the solid component of the tumor, which may also express factor VIII and D240 (podoplanin) in over 60% of cases. Markers such as cytokeratins, HMB45, S100, CD20, CD68, EMA, desmin, estrogen receptor, and progesterone are usually negative and help differentiate from carcinomas (11).

The MYC protein, encoded by the MYC proto-oncogene (8q21), is amplified in 60.0-70.0% of RIAS cases. Detection by IHC or *in situ* hybridization (ISH) confirms the diagnosis with a sensitivity of 77.0% and a specificity of 100.0%, making it the primary differential criterion from PA (11-13). Co-amplification of MYC and FLT4, which encodes vascular endothelial growth factor receptor 3, can also occur and has been observed in approximately 25.0% of RIAS, with potential implications as a predictor of response to targeted therapies; however, further evidence is still needed for its clinical application (11, 13-14).

## Clinical characteristics

RIAS typically occurs in patients aged 60-75 years. Clinically, it presents as rapidly progressing skin lesions—violaceous macules, papules, or nodules—with color changes, ulceration, edema, and an orange-peel texture, usually with multifocal, diffuse distribution (Figure 1). The presence of masses in the breast parenchyma in the absence of associated skin changes is unusual. Distant metastatic involvement occurs in about 30% of cases at diagnosis, with a predilection for organs such as the lung, liver, and lymph nodes (15). In the reported case, systemic staging studies did not show distant metastatic involvement. The latency interval between breast carcinoma and RIAS is usually between 5 and 10 years, although it can be shorter in some cases (16).

## Differential diagnoses

The differential diagnoses for RIAS include atypical vascular lesions, papillary endothelial hyperplasia, hemangioma, pseudoangiomatous stromal hyperplasia, angiolipoma, angiomatosis, and metaplastic spindle cell carcinoma. Atypical vascular lesions typically arise in previously irradiated areas and are characterized by vascular proliferation confined to the dermis, with occasional extension into the subcutaneous tissue. Clinically, they appear as flat, erythematous plaques. Although benign and generally lacking cytological atypia, they can be challenging to distinguish from RIAS. Papillary endothelial hyperplasia corresponds to reactive endothelial proliferation within an organized thrombus. It manifests as a well-defined lesion without infiltration into the surrounding stroma, allowing differentiation from sarcomatous neoplasms. A hemangioma is a benign proliferation of blood vessels lined by flattened endothelium, with a circumscribed architecture. Pseudoangiomatous stromal hyperplasia, on the other hand, involves proliferation of myofibroblasts and formation of stromal clefts that resemble vascular channels or even anastomoses. Angiolipoma combines mature adipose tissue with small, well-defined, and circumscribed capillaries. Generally, the lesions mentioned above are usually small (less than 2 cm) and lack significant atypia (10).

In contrast, metaplastic spindle cell carcinoma is a malignant neoplasm that can exhibit a pseudovascular pattern resembling angiosarcoma, making the use of IHC techniques essential for accurate differentiation. In these cases, both epithelial and endothelial markers should be included because of potential overlap in expression profiles (10).

In the differential diagnosis between carcinoma and angiosarcoma, ERG expression is typically positive in angiosarcoma and negative in carcinoma; however, ERG can also be expressed in atypical vascular lesions, due to which morphological and clinical correlation is essential. Other endothelial markers, such as CD31 and factor VIII, show high expression in angiosarcoma and little or no expression in carcinoma, making them very useful for diagnosis. Conversely, the epithelial markers p63 and cytokeratins are usually positive in carcinoma, very poorly expressed in angiosarcoma, and negative in atypical vascular lesions (11).

Finally, once the diagnosis of angiosarcoma has been established, MYC expression is useful for distinguishing secondary (radiation-induced) angiosarcoma, in which positivity is observed, from PA, in which it is usually negative, thereby supporting its radiation-induced nature (11).

## Treatment

Surgery is the primary treatment for RIAS (15, 17). Li *et al.* retrospectively analyzed 76 patients who underwent surgical resection and found that radical surgery (resection of all or nearly all the irradiated skin) achieved better local control and higher survival rates compared to conservative surgery (resection of only a portion of the irradiated skin). The recurrence rate was 16.0% in the radical surgery group versus 68.0% in the conservative surgery group; the five-year cumulative incidence was 23.0% vs. 76.0% ( $p = 0.01$ ), and the disease-specific survival rate was 86.0% vs. 46.0% ( $p = 0.01$ ), respectively. Furthermore, achieving negative surgical margins was identified as an independent prognostic factor that positively influences survival (18). While radical surgery has a higher risk of complications and often requires flap reconstruction, it has become established as the best strategy. Even so, recurrence and mortality rates remain high, prompting further research

into the potential benefits of adding chemotherapy or radiotherapy as part of a multimodal treatment approach.

The role of neoadjuvant or adjuvant radiotherapy in RIAS remains debated. Without randomized clinical trials, its benefits are uncertain. In the systematic review by Depla *et al.*, which included 74 studies (222 patients), radiotherapy was linked to a significant reduction in local recurrence (hazard ratio [HR] = 0.46; 95% confidence interval [95% CI] = 0.26-0.84;  $p = 0.010$ ), without a benefit in mortality (HR = 0.35; 95% CI = 0.15-0.80;  $p = 0.012$ ). No significant benefit was observed with chemotherapy. However, it is important to note that the average tumor size was smaller in the radiotherapy group. This result should be interpreted cautiously, given the potential for selection bias in observational studies (17). Some small case series suggest a similar effect with neoadjuvant radiotherapy, with up to 88.0% of disease-free cases at 34 months (19).

In a retrospective study from MD Anderson Cancer Center, which included a cohort of 95 patients with RIAS, 52.0% received chemotherapy (neoadjuvant, adjuvant, or both) based on doxorubicin, taxanes, gemcitabine, ifosfamide, interferon, or dacarbazine, as monotherapy or in combination, in addition to surgical treatment. After 10.8 years of follow-up, the addition of chemotherapy was associated with a significant 65.0% reduction in the risk of recurrence (HR = 0.35; 95% CI = 0.15-0.80;  $p = 0.012$ ), with no impact on overall survival (20). These results were similar to those described in a Japanese meta-analysis (21). A retrospective analysis by Palassini *et al.* in 84 patients found that adjuvant radiotherapy significantly reduced the risk of recurrence (HR = 0.25; 95% CI = 0.08-0.83;  $p = 0.02$ ), and gemcitabine-based chemotherapy (neoadjuvant or adjuvant) increased five-year overall survival (69.0% with gemcitabine vs. 52.0% without gemcitabine,  $p = 0.02$ ). These findings support a multimodal management strategy combining surgery with chemotherapy or radiotherapy to optimize oncological outcomes in RIAS (22).

Degnim *et al.* observed that a trimodal therapy consisting of neoadjuvant paclitaxel-based chemotherapy combined with radiotherapy, followed by surgical resection with 5 cm margins from the visible disease, facilitates an optimal oncological margin in patients who do not achieve a complete

pathological response. At the five-year follow-up, no local recurrences were recorded, and only one case of distant recurrence was observed, resulting in a recurrence-free survival rate of 93.0% and an overall survival benefit compared with other management regimens (23). Despite these findings, the limitations of these studies (small sample sizes, heterogeneity of therapeutic regimens, short follow-up periods, and retrospective design) highlight the need for prospective research to clarify the role of neoadjuvant or adjuvant chemotherapy and radiotherapy in RIAS.

## Prognosis

The overall five-year survival rate for patients with RIAS ranges from 40.0% to 50.0% (6, 17). Recurrence rates are high, ranging from 45.0% to 65.0% of cases, and typically occur within the first year of follow-up. The five-year locoregional and distant recurrence-free survival rates are approximately 62.0% and 75.0%, respectively (15, 17). Concerning local recurrence, the prognosis is poor, even with salvage surgery, as only 29.0% of patients remain disease-free in the long term (15).

## Conclusion

RIAS is a highly aggressive neoplasm characterized by a poor prognosis because of its high rate of local recurrence and rapid progression. Its low incidence limits the availability of studies with enough evidence to establish a standardized treatment protocol, leading to controversy in the literature and making its diagnosis and management a clinical challenge.

It is recommended to maintain a high index of suspicion in patients who have undergone radiotherapy and to promote training for medical personnel to facilitate early detection of skin lesions that develop within the irradiated area.

Surgery is the primary treatment, and better local control and improved overall survival have been demonstrated

when wide margins are achieved, ideally with complete resection of irradiated skin or with disease-free margins of at least 5 cm. Regarding adjuvant treatment, although evidence is limited, some studies suggest that administering taxane- or gemcitabine-based chemotherapy in combination with radiotherapy may improve oncological outcomes; however, there is still no consensus on the optimal therapeutic approach. Therefore, prospective studies are essential to identify standardized diagnostic and treatment strategies that will contribute to improving the prognosis of this rare and challenging condition.

Written informed consent was obtained from the patient for publishing clinical data and images, ensuring her understanding of the report's academic purpose. The patient's identity was protected by excluding personal information and using photographs that do not allow her recognition, ensuring her anonymity and confidentiality.

## References

1. Bray F, Laversanne M, Sung H, Ferlay J, Siegel R, Soerjomataram I, *et al.* Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin.* 2024;74(3):229-63. <https://doi.org/10.3322/caac.21834>
2. Huang J, Mackillop W. Increased risk of soft tissue sarcoma after radiotherapy in women with breast carcinoma. *Cancer.* 2001;92(1):172-80. [https://doi.org/10.1002/1097-0142\(20010701\)92:1%3C172::aid-cncr1306%3E3.0.co;2-k](https://doi.org/10.1002/1097-0142(20010701)92:1%3C172::aid-cncr1306%3E3.0.co;2-k)
3. Mergancová J, Lierová A, Coufal O, Žatecký J, Melichar B, Zedníková I, *et al.* Radiation-associated angiosarcoma of the breast: an international multicenter analysis. *Surg Oncol.* 2022;41:101726. <https://doi.org/10.1016/j.suronc.2022.101726>
4. Arlen M, Higinbotham N, Huvos A, Marcove R, Miller T, Shah I. Radiation-induced sarcoma of bone. *Cancer.* 1971;28(5):1087-99. [https://doi.org/10.1002/1097-0142\(1971\)28:5%3C1087::aid-cncr2820280502%3E3.0.co;2-f](https://doi.org/10.1002/1097-0142(1971)28:5%3C1087::aid-cncr2820280502%3E3.0.co;2-f)
5. Cahan W, Woodard H, Higinbotham N, Stewart F, Coley B. Sarcoma arising in irradiated bone: report of eleven cases. *Cancer.* 1998;82(1):8-34. [https://doi.org/10.1002/\(sici\)1097-0142\(19980101\)82:1%3C8::aid-cncr3%3E3.0.co;2-w](https://doi.org/10.1002/(sici)1097-0142(19980101)82:1%3C8::aid-cncr3%3E3.0.co;2-w)
6. Rombouts A, Huising J, Hugen N, Siesling S, Poortmans P, Nagtegaal I, *et al.* Assessment of radiotherapy-associated angiosarcoma after breast cancer treatment in a dutch population-based study. *JAMA Oncol.* 2019;5(2):267-9. <https://doi.org/10.1001/jamaoncol.2018.6643>
7. Yap J, Chuba P, Thomas R, Aref A, Lucas D, Severson R, *et al.* Sarcoma as a second malignancy after treatment for breast cancer. *Int J Radiat Oncol Biol Phys.* 2002;52(5):1231-7. [https://doi.org/10.1016/s0360-3016\(01\)02799-7](https://doi.org/10.1016/s0360-3016(01)02799-7)
8. Morgan E, Kozono D, Wang Q, Mery C, Butrynski J, Baldini E, *et al.* Cutaneous radiation-associated angiosarcoma of the breast: poor prognosis in a rare secondary malignancy. *Ann Surg Oncol.* 2012;19(12):3801-8. <https://doi.org/10.1245/s10434-012-2563-4>
9. Stewart F, Treves N. Lymphangiosarcoma in postmastectomy lymphedema; a report of six cases in elephantiasis chirurgica. *Cancer.* 1948;1(1):64-81. [https://doi.org/10.1002/1097-0142\(194805\)1:1%3C64::aid-cncr2820010105%3E3.0.co;2-w](https://doi.org/10.1002/1097-0142(194805)1:1%3C64::aid-cncr2820010105%3E3.0.co;2-w)
10. Hicks D, Lester S. Diagnostic pathology breast. 2.<sup>a</sup> ed. Elsevier; 2016.
11. Rosen P, Koerner F, Brogi E, Hoda S. Rosen's Breast Pathology. 4.<sup>a</sup> ed. Vol. 1. Philadelphia: Lippincott Williams & Wilkins; 2014.
12. Cornejo K, Deng A, Wu H, Cosar E, Khan A, St. Cyr M, *et al.* The utility of MYC and FLT4 in the diagnosis and treatment of postradiation atypical vascular lesion and angiosarcoma of the breast. *Hum Pathol.* 2015;46(6):868-75. <https://doi.org/10.1016/j.humpath.2015.02.014>
13. Wei Y, Yang X, Gao L, Xu Y, Yi C. Differences in potential key genes and pathways between primary and radiation-associated angiosarcoma of the breast. *Transl Oncol.* 2022;19:101385. <https://doi.org/10.1016/j.tranon.2022.101385>
14. Guo T, Zhang L, Chang N, Singer S, Maki R, Antonescu C. Consistent MYC and FLT4 gene amplification in radiation-induced angiosarcoma but not in other radiation-associated atypical vascular lesions. *Genes Chromosomes Cancer.* 2011;50(1):25-33. <https://doi.org/10.1002/gcc.20827>
15. Salminen S, Wiklund T, Sampo M, Tarkkanen M, Pulliainen L, Böhling T, *et al.* Treatment and prognosis of radiation-associated breast angiosarcoma in a nationwide population. *Ann Surg Oncol.* 2020;27(4):1002-10. <https://doi.org/10.1245/s10434-019-08085-1>

16. Cozzi S, Finocchi S, Tava F, Bardoscia L, Najafi M, Ruggieri M, *et al.* Radiation-associated angiosarcoma of the breast: the state of the art of a rare and aggressive disease. *J Pers Med.* 2024;14(8):859. <https://doi.org/10.3390/jpm14080859>
17. Depla A, Scharloo-Karels C, de Jong M, Oldenburg S, Kolff M, Oei S, *et al.* Treatment and prognostic factors of radiation-associated angiosarcoma (RAAS) after primary breast cancer: a systematic review. *Eur J Cancer.* 2014;50(10):1779-88. <https://doi.org/10.1016/j.ejca.2014.03.002>
18. Li G, Fairweather M, Wang J, Orgill D, Bertagnolli M, Raut C. Cutaneous radiation-associated breast angiosarcoma. *Ann Surg.* 2017;265(4):814-20. <https://doi.org/10.1097/sla.0000000000001753>
19. Asha W, Al-Hilli Z, Djohan R, Budd G, Obi E, Fleming-Hall E, *et al.* Neoadjuvant reirradiation for radiation therapy-associated angiosarcoma of the breast. *Int J Radiat Oncol Biol Phys.* 2024;119(4):1082-5. <https://doi.org/10.1016/j.ijrobp.2023.12.050>
20. Torres K, Ravi V, Kin K, Yi M, Guadagnolo B, May C, *et al.* Long-term outcomes in patients with radiation-associated angiosarcomas of the breast following surgery and radiotherapy for breast cancer. *Ann Surg Oncol.* 2013;20(4):1267-74. <https://doi.org/10.1245/s10434-012-2755-y>
21. Takehara Y, Matsuda N, Kobayashi D, Yoshida A, Takei J, Kanomata N, *et al.* Radiation-induced angiosarcoma of the breast: individual participant meta-analysis of Japanese population. *Breast Cancer.* 2023;30(5):739-47. <https://doi.org/10.1007/s12282-023-01466-1>
22. Palassini E, Baldi G, Ciniselli C, Gennaro M, Gronchi A, Sangalli C, *et al.* Outcome improvement with chemotherapy and radiotherapy in primary, localized, radiation-associated angiosarcoma of the breast region: a retrospective case series analysis. *ESMO Open.* 2024;9(6):103474. <https://doi.org/10.1016/j.esmooop.2024.103474>
23. Degnim A, Siontis B, Ahmed S, Hoskin T, Hieken T, Jakub J, *et al.* Trimodality therapy improves disease control in radiation-associated angiosarcoma of the breast. *Clin Cancer Res.* 2023;29(15):2885-93. <https://doi.org/10.1158/1078-0432.ccr-23-0443>