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Case Report

# A rare case of leukocytoclastic vasculitis in autologous breast augmentation post breast cancer treatment

Sean Engelhardt[1], Chloé Marie Victoria Howell[2*], Simon John Nayler[3], Weludo Ngwisanyi[1], Carol-Ann Benn[4]

[1]Faculty of Health Sciences, School of Medicine, University of the Witwatersrand, South Africa
[2]Steve Biko Academic Hospital, South Africa
[3]Drs Gritzman and Thatcher Inc, Wits Donald Gordon Medical Centre and Breast Care Centre of Excellence, Milpark Hospital, Johannesburg, South Africa
[4]Breast Care Centre of Excellence, Milpark Hospital, Johannesburg, South Africa

***Corresponding Author:*** *drhowellrsa@gmail.com*

**ABSTRACT**

Leukocytoclastic vasculitis is a hypersensitivity reaction involving immune complex deposition in dermal capillaries and venules. We report a case of a 34-year-old female patient with left-sided breast carcinoma who developed vasculitis following autologous left breast augmentation, confirmed histologically. Various aspects of the patient's treatment history may be implicated in the development of her leukocytoclastic vasculitis, including chemotherapy and radiation therapy, antibiotics, and the presence of breast carcinoma (leukocytoclastic vasculitis as a paraneoplastic syndrome). Based on the temporal relationship, histological findings, and the site of these pruritic lesions, we postulate that autologous breast augmentation is the most likely aetiology for the leukocytoclastic vasculitis in our patient, an association not previously reported.

## INTRODUCTION

Leukocytoclastic vasculitis (LCV) refers to inflammation of dermal capillaries and venules elicited by immune complex deposition, clinically presenting as haemorrhagic vesicles with palpable purpura.(1) Histologically, LCV involves the infiltration of vessels by an inflammatory cell infiltrate rich in neutrophils coupled with leukocytoclasis, which is the deposition of neutrophil nuclear debris.(2)

The diagnosis of LCV is two-fold. Firstly, confirmation of LCV requires skin biopsies of regions exhibiting cutaneous vasculitis.(1) Optimization of biopsy sensitivity is achieved by performing the biopsy within 48 hours after the appearance of the first vasculitic lesion, ensuring easy visualisation of pathognomonic features.(2) Secondly, serological assessment is essential to exclude vasculitis as a component of systemic vasculitic syndromes, such as granulomatosis with polyangiitis or microscopic angiitis.(1)

Although idiopathic in approximately 50% of cases, LCV is a common secondary manifestation of drug hypersensitivity, including but not limited to beta-lactams, clindamycin, non-steroidal anti-inflammatory drugs, and anticancer agents.(1) Many of these anticancer agents are central to treating solid malignancies of the breast. The development of LCV has been reported in exemestane and everolimus chemotherapy for metastatic breast cancer, anastrozole chemotherapy in oestrogen receptor-positive breast cancer, and letrozole chemotherapy in stage IV oestrogen receptor/HER2 positive breast cancer.(3) Drug-induced vasculitis is commonly associated with the prominence of eosinophils in the inflammatory infiltrate on biopsy.(2)

Vasculitis has also been reported as a paraneoplastic syndrome. Two fundamental criteria for considering LCV as paraneoplastic are (a) the concurrent appearance of the vasculitis and malignancy and (b) the congruent course of the vasculitis and malignancy. Although associated with haematological malignancies, it has previously been suggested that solid organ malignancies should be considered a cause of vasculitis without additional apparent causes. Significantly, it has been illustrated that LCV is the most common histological subtype associated with solid malignancies, including breast malignancies.(5)

## CASE REPORT

In May 2017, a 34-year-old female patient presented with breast cancer characterised by a lump in the upper outer quadrant of her left breast clinically staged to be T2N2M0. Other than the use of contraception and a 15-pack-year smoking history, the patient had no other risk factors. Past medical history was unremarkable, with no previous surgeries, no chronic medication or supplement use, and no known allergies. Significantly, the patient reported a prior history of ovarian cancer in a first-degree relative.

Breast ultrasound at presentation revealed a tumour in the upper midline, measuring 21.1 × 18.4 × 14.2 mm, with associated axillary lymphadenopathy. Benign fibroadenomas were observed in the outer and inner midline of the left breast. Following a biopsy, a microscopic analysis of the tumour revealed a grade 3 invasive adenocarcinoma of no special type. Lymphovascular and perineural tumour invasion was apparent. Immunohistochemistry of the biopsy confirmed oestrogen and progesterone receptor positive and HER2, 2+ status.

Subsequently, chemotherapy (4 cycles of doxorubicin and cyclophosphamide and 12 cycles of paclitaxel) was initiated and completed in August 2017. In December 2017, a bilateral mastectomy with Goldilocks reconstruction was performed, followed by external beam orthovoltage radiotherapy from February to March 2018. The left chest wall and draining lymph nodes were subjected to 50 Gy for 25 cycles, followed by 10 Gy for 10 cycles. A follow-up ultrasound was performed at the end of March 2018, with the only significant finding being a large superficial carbuncle in the left axilla, treated to resolution with clindamycin (300 mg, bd, 5 days) and amoxicillin/clavulanic acid (1000 mg, bd, 10 days).

Autologous breast augmentation was performed on the left breast 5 months post-radiotherapy in September 2018. Significantly, the patient reported having developed purpuric skin lesions 3 months following the procedure in early December 2018. Clinically, the patient presented with a firm and tender left breast and purpuric lesions. A punch biopsy was performed in December 2018, showing a normal epidermis with a perivascular inflammatory cell infiltrate (Figure 1). The dermis showed the presence of a perivascular infiltrate comprising a mixed inflammatory cell population (Figure 2), neutrophils, and scattered eosinophils with karyorrhexis and karyorrhectic debris noted (Figure 3). The endothelial cells demonstrated endothelial cell swelling and subtle endotheliitis. No evidence of metastatic breast carcinoma was noted. The features represented leukocytoclastic vasculitis. The patient was given montelukast (10 mg, orally for 3 months) and a zinc mineral supplement, prompting the resolution of the vasculitis.

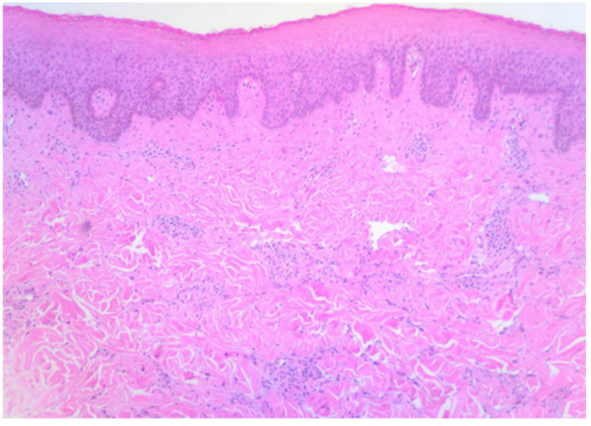

**Figure 1:** Haematoxylin and Eosin-stained section (40× magnification) demonstrating normal epidermis with a mild perivascular inflammatory cell infiltrate.

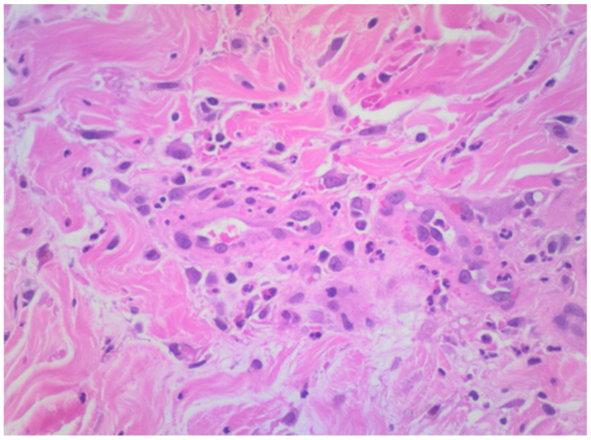

**Figure 2:** Haematoxylin and Eosin-stained section (200× magnification) showing mild perivascular inflammation composed of neutrophils, scattered eosinophils, and karyorrhexis, with endothelial cell swelling.

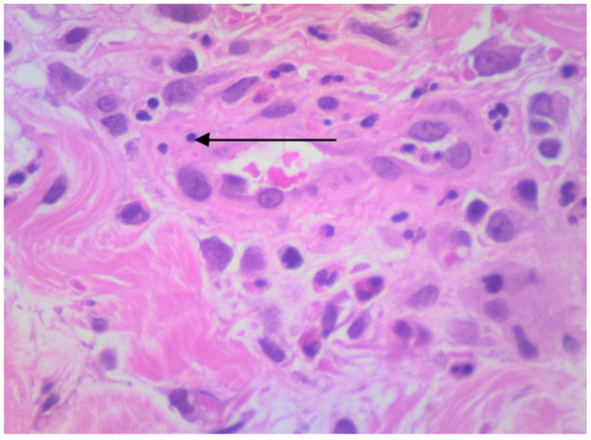

**Figure 3:** Haematoxylin and Eosin-stained sections (400×) showing the neutrophils and karyorrhexis debris (arrow).

## DISCUSSION

In this patient, the aetiological factors leading to the development of LCV may have been due to various origins. Aspects of the patient's treatment history, including chemotherapy, antibiotic administration, radiation therapy, and the history of a solid organ malignancy (potential for paraneoplastic vasculitis), all represent plausible aetiologies.

Leukocytoclastic vasculitis is a common hypersensitivity adverse effect to an array of commonly used anticancer drugs. Interestingly, in drug-induced LCV, the purpuric

lesions are most commonly noted on the extremities (due to the relatively decreased blood flow in these regions) with a prominence of eosinophils.(2). In August 2017, the patient completed a chemotherapy regimen comprising doxorubicin, cyclophosphamide, and paclitaxel. The development of LCV occurred in September 2018. It involved the skin of the patient's left breast, while minimal eosinophils were noted. Thus, chemotherapy as the aetiology of LCV is temporally, histologically, and locationally unlikely. Additionally, cyclophosphamide is often used to manage LCV, rendering it an unlikely drug to elicit an LCV hypersensitivity reaction.(1)

Our patient anticancer regimen was consolidated with radiotherapy in February 2018. LCV occurring in previously irradiated tissue has been reported in a few cases; however, in these cases the LCV developed in the irradiated tissue whilst receiving chemotherapy, particularly with mTOR inhibitors, and resolution occurred after the discontinuation of these agents. Kuhar et al. reviewed these various cases and compared them to their case, where an exemestane and everolimus chemotherapy regimen was used to treat breast cancer in previously irradiated breast tissue. It was concluded that LCV in previously irradiated tissue could be attributed to radiation recall and LCV resolution occurred with discontinuation of the response-inducing agents.(4) The above suggests that the consolidative radiotherapy post chemotherapy is unlikely to have contributed to our patient's LCV as our patient only started developing purpuric lesions in December 2018, 10 months after consolidative radiotherapy and 16 months after chemotherapy.

The patient reported here is also unlikely to have developed LCV due to antibiotic administration (clindamycin and amoxicillin/clavulanic acid) as a treatment approach for her axillary carbuncle reported in March 2018. Antibiotic-associated cutaneous vasculitis has previously been reported. An investigation assessing 773 cutaneous vasculitis cases attributed the most significant proportion of drug-associated cutaneous vasculitis to antibiotics (mainly β-lactams) with additional hypersensitivity features presenting as nephropathic, joint, and gastrointestinal manifestations.(6) Significantly, antibiotic-mediated LCV ensues within days of drug administration and resolves soon after drug cessation. In our patient, LCV occurred approximately five months after antibiotic administration. Thus, antibiotics are an unlikely aetiology of LCV in our patients.

The development of LCV in this case is also unlikely to represent a paraneoplastic syndrome secondary to solid organ malignancy. Previous investigations have suggested that LCV may ensue before, concomitantly, or after developing a solid organ malignancy. In one study, the development of LCV and solid organ malignancy diagnosis were separated by no more than 12 months.(5) Our patient presented with LCV 18 months after the initial breast carcinoma diagnosis.

## CONCLUSION

To our knowledge, this is the first reported case of a patient who developed LCV after autologous fat-fill breast augmentation, supported in our patient by temporal evidence, location of the vasculitic lesions, and exclusion of other plausible aetiological factors.

**Consent:** Written informed consent was obtained from the patient.

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