Case Report

Vasculitis as a Dermatological Presentation of Hepatic Malignancy: A Case Study

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Abstract

Leukocytoclastic vasculitis (LCV) is a histopathologic descriptor for a prevalent type of small-vessel vasculitis (SVV) that affects arterioles, capillaries, and postcapillary venules. Although the association between vasculitis and malignancy only accounts for <5% of vasculitis cases, it has been recognized as a true paraneoplastic syndrome in several studies. A 57-year-old Filipino male presented with erythematous, nonblanching macules on his lower extremities, which rapidly progressed to violaceous lesions on his trunk, buttocks, and lower extremities. He also reported significant weight loss, decreased appetite, and vomiting. A skin biopsy confirmed LCV. Initially treated for meningococcemia, his condition did not improve. Abdominal imaging revealed an enlarged heterogeneous liver with retroperitoneal lymphadenopathy and a parenchymal nodule. He was eventually diagnosed with vasculitis secondary to an underlying hepatic malignancy and expired later from multiorgan failure.

Keywords: Leukocytoclastic vasculitis, malignancy, paraneoplastic syndrome

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Introduction

Leukocytoclastic vasculitis (LCV) is a type of small vessel vasculitis (SVV) affecting arterioles, capillaries, and venules. It is characterized by neutrophilic infiltration, neutrophil nuclei fragmentation, fibrinoid necrosis, and vessel wall damage. Clinically, LCV typically presents as an asymptomatic palpable purpura on the lower legs or other dependent areas.[1] Although most are idiopathic, <5% of vasculitis cases are associated with malignancies as a paraneoplastic phenomenon.

Case Report

A 57-year-old Filipino male presented with a 1-day history of erythematous, nonblanching macules on both lower extremities, which rapidly spread and became violaceous on the trunk, buttocks, and legs. He had no prior medication use or dermatological consultation. Symptoms included decreased appetite, 30% unintentional weight loss over 3 months, and vomiting. He completed treatment for pulmonary tuberculosis and was evaluated for reactivation. His medical history includes type 2 diabetes, for which he was prescribed metformin, a 5-pack-year smoking history, and weekly alcohol consumption (six beers).

On arrival at our institution, the patient was febrile (38°C) and tachypneic (24 bpm), with an oxygen saturation of 95% on room air. Physical examination revealed multiple

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well-defined, irregularly-shaped, violaceous nonlanching macules and patches on the trunk, buttocks, and lower extremities [Figure 1]. The abdomen was nontender with normal bowel sounds and no palpable masses, or organomegaly. The rest of the examination was unremarkable.

The laboratory results indicate severe inflammation, poor glucose control, electrolyte imbalances, and liver and kidney damage. Laboratory results revealed high inflammatory markers, which suggest infection or autoimmune issues, while high troponin levels suggest myocardial injury. He also had impaired liver and renal function,

Given the rapid progression of the lesions, he was referred to the infectious disease service for co-management and initially treated as a case of meningococcemia. He received ceftriaxone (5 days), metronidazole, piperacillin/tazobactam (3 days), and meropenem (2 days), but showed no improvement.

A 4-mm skin punch biopsy from the right lower extremity revealed a basket-woven stratum corneum, flat epidermis, and scattered necrotic keratinocytes. The edematous dermis had perivascular lymphocytes, histiocytes, neutrophils, and extravasated red blood cells. Blood vessels showed endothelial swelling, fibrinoid necrosis, and eosinophilic thrombi. These findings were consistent with LCV [Figure 2].

A whole abdomen ultrasound showed a normal-sized liver with homogeneous echotexture and smooth borders, but also revealed a 3.1 cm × 2.1 cm hyperechogenic focus and a 4.8 cm × 5.8 cm hypoechogenic lesion with rim hyperechogenicity in the right hepatic lobe, with a suggestion of a parenchymal nodule. Furthermore, a plain computed tomography scan of the abdomen showed an enlarged, heterogeneous liver with retroperitoneal lymphadenopathy [Figure 2].

The patient was diagnosed with vasculitis, likely secondary to hepatic malignancy due to the developed symptoms, abnormal masses on imaging studies, and elevated liver function tests. Ten days later, the patient expired due to multiorgan failure.

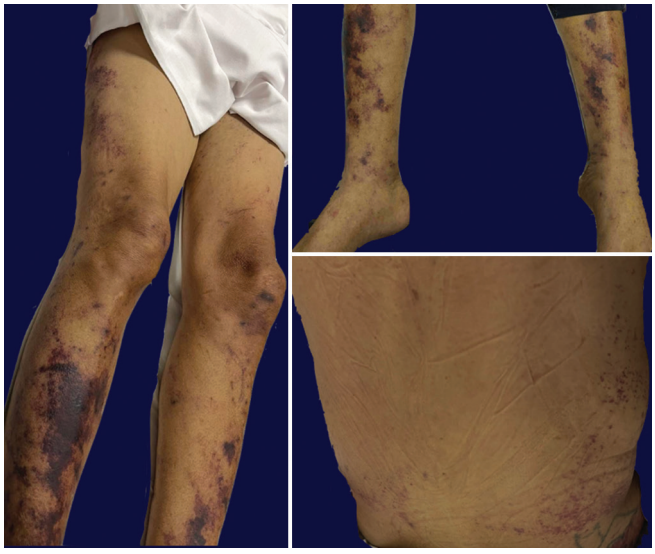

Figure 1: Physical examination of a 57-year-old Filipino male revealed multiple erythematous well-defined, irregularly shaped, hyperpigmented to violaceous nonblanching macules and patches on trunk, buttocks, and lower extremities

Discussion

LCV is a histopathologic descriptor for a prevalent type of SVV that affects arterioles, capillaries, and postcapillary venules.[1] The typical clinical presentation is asymptomatic palpable purpura, predominantly affecting the lower legs or other dependent areas.[1,2] The lesions coalesce into confluent patterns but usually disappear after 2–3 weeks, leaving hyperpigmentation.[1]

Data indicates that approximately 40% of cutaneous vasculitis cases are idiopathic. Other causes include adverse drug reactions (20%), infections, mainly upper respiratory (22%), connective tissue diseases such as lupus and rheumatoid arthritis (12%), Henoch–Schönlein

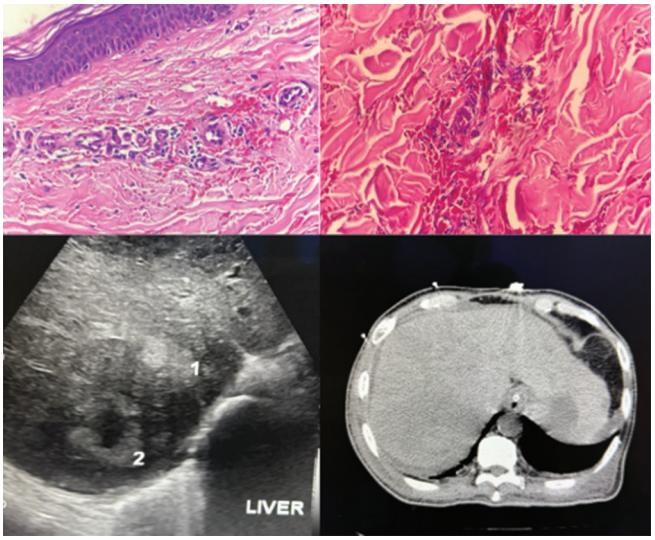

Figure 2: Microsection showed basket-woven stratum corneum, flat epidermis with scattered necrotic keratinocytes, blood vessels exhibiting endothelial cell swelling, fibrinoid necrosis of vessel walls, and eosinophilic thrombi within the lumen. The whole abdomen ultrasound showed a hyperechogenic focus and a hypoechogenic lesion with rim hyperechogenicity in the right hepatic lobe, while a plain computed tomography scan revealed an enlarged heterogeneous liver with retroperitoneal lymphadenopathy

purpura (10%), and <5% due to malignancy, giant cell arteritis, or other systemic inflammatory conditions.[3]

Malignancy-associated vasculitis is believed to result from immunogenic factors released by neoplastic cells, forming immune complexes that trigger inflammation in vessel walls.[4] While it accounts for <5% of cases, paraneoplastic vasculitis has been reported in several studies.[3] Solans-Laqué *et al.* examined cases of concurrent vasculitis and solid tumors at their institution over 15 years, where both conditions were diagnosed within 12 months of each other.[5] The malignancies linked to vasculitis included urinary bladder, prostate, lung, renal, and breast carcinomas.[5] Meanwhile, a study by Hutson *et al.* showed an equal distribution between patients with hematologic malignancies, including lymphoma and leukemia, and those with solid organ cancers, such as breast, pancreatic, and lung cancer.[6] LCV was the most common phenotype observed in these patients in both studies.[5,6] Paraneoplastic vasculitis linked to hepatocellular carcinoma is rare, with reports mostly limited to case studies, highlighting how uncommon hepatobiliary cancers are as underlying causes.[7]

A skin punch biopsy is an essential initial tool to diagnose LCV.[3] Key features include neutrophilic infiltration around and within the vessel walls, neutrophil nuclear fragmentation, fibrinoid necrosis with fibrin deposition, and evidence of vessel wall or tissue damage such as red blood cell extravasation.[1] A skin punch biopsy performed 24–48 h after lesion onset is sufficient for diagnosing cutaneous vasculitis.[8] In cases of inconclusive biopsy results, a second biopsy with direct immunofluorescence may be warranted.[8]

Routine screening for underlying cancer in vasculitis is not recommended due to the rarity of malignancy-associated vasculitis.[6] However, a high index of suspicion for malignancy should be maintained in persistent vasculitis despite appropriate treatment, particularly in individuals older than 50 years old.[4] According to Carlson's algorithm, paraneoplastic vasculitis should be considered in patients with purpura, hematuria, and hematologic abnormalities. Imaging studies are recommended to detect any abnormal masses suggestive of malignancy.[3] In addition, reviewing the patient's recent history of drug use, infections, or environmental exposures is crucial to determining the likelihood of a paraneoplastic diagnosis.

Paraneoplastic vasculitis often follows the course of the underlying malignancy, emerging before, at diagnosis, or during relapse.[9] Effective cancer treatment often leads to improvement of vasculitis, with remission commonly aligning with therapy success. Meanwhile, recurrence or worsening of vasculitis may indicate cancer relapse or progression.[6,7] The type of cancer primarily drives the overall prognosis for patients with malignancy-associated vasculitis. In most cases, it is the malignancy that leads to death.[7]

In resistant vasculitis, a thorough etiological investigation is crucial. Histopathology with laboratory and imaging studies aids diagnosis and guides targeted therapy, potentially improving outcomes, even in rare malignancy-related cases.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

References

1. Fraticelli P, Benfaremo D, Gabrielli A. Diagnosis and management of leukocytoclastic vasculitis. Intern Emerg Med 2021;16:831-41.
2. Bezerra AS, Polimanti AC, de Oliveira RA, Fürst RV, Criado PR, Corrêa JA. Early diagnosis and treatment of leukocytoclastic vasculitis: Case report. J Vasc Bras 2020;19:e20180072.
3. Carlson JA, Cavaliere LF, Grant-Kels JM. Cutaneous vasculitis: Diagnosis and management. Clin Dermatol 2006;24:414-29.
4. Rabin SS, Dudelzak J, Lee JR, Sheehan DJ. Leukocytoclastic vasculitis: A marker of underlying malignancy. Consultant 2006;46:1196-8.
5. Solans-Laqué R, Bosch-Gil JA, Pérez-Bocanegra C, Selva-O'Callaghan A, Simeón-Aznar CP, Vilardell-Tarres M. Paraneoplastic vasculitis in patients with solid tumors: Report of 15 cases. J Rheumatol 2008;35:294-304.
6. Hutson TE, Hoffman GS. Temporal concurrence of vasculitis and cancer: A report of 12 cases. Arthritis Care Res 2000;13:417-23.
7. Loricera J, Calvo-Río V, Ortiz-Sanjuán F, González-López MA, Fernández-Llaca H, Rueda-Gotor J, *et al.* The spectrum of paraneoplastic cutaneous vasculitis in a defined population: Incidence and clinical features. Medicine (Baltimore) 2013;92:331-43.
8. Chango Azanza JJ, Calle Sarmiento PM, Lopetegui Lia N, Alexander SA, Modi V. Leukocytoclastic vasculitis: An early skin biopsy makes a difference. Cureus 2020;12:e7912.
9. Kurzrock R, Cohen PR, Markowitz A. Clinical manifestations of vasculitis in patients with solid tumors. A case report and review of the literature. Arch Intern Med 1994;154:334-40.