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ORIGINAL RESEARCH

Prevalence of Livedoid Vasculopathy Among Patients with Connective Tissue Diseases and Its Association with Thrombophilic Factors: A Hospital-Based Retrospective Cohort (2014–2021)

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Background: Livedoid vasculopathy is a rare, chronic thrombo-occlusive disorder that primarily affects the lower extremities, causing painful ulcerative lesions. Despite its clinical significance, the prevalence and demographic characteristics of LV, particularly among patients with connective tissue diseases, remain underexplored. This study aims to investigate the prevalence of LV among patients diagnosed with CTDs, providing insight into potential epidemiological patterns.

Methods: The study was a retrospective cohort study conducted at Mass General Brigham (MGB), a healthcare system in Greater Boston, Massachusetts, from January 1, 2014, to June 1, 2021. Data extraction was facilitated through the Mass General Brigham Enterprise Data Warehouse utilizing the Research Patient Data Registry (RPDR) system. Data extraction includes age, gender, race and autoimmune diseases associated with LV. SPSS v 29 was used for data analysis.

Results: We reviewed 1730 charts; 108 patients met criteria for LV. Prevalence is reported within the screened cohort (108/1730 = 6.2%). For system context, we also report a hospital-system registered prevalence of 0.0018% (108/6,000,000) based on the RPDR population. The majority of patients were female (74.07%). The mean age of the patients with LV was 44.87 ± 8.24 years. The racial composition was white (84.25%). Among the 17 patients with autoimmune diseases, females were more represented (82.35%). Systemic lupus erythematosus was the predominant condition, affecting 58.82% of these patients. Positive results for phospholipid antibodies were found in 57.40% of patients, with a p-value of 0.065. For anticardiolipin antibodies, p-value 0.095. Hyperhomocystenemia, p-value of 0.952. Activated C resistance, p-value of 0.088.

Conclusion: In this cohort of patients with connective tissue diseases, livedoid vasculopathy was observed more often in women and in middle-aged adults, and it frequently co-occurred with autoimmune conditions, particularly systemic lupus erythematosus. However, none of the between-group comparisons or serological associations reached statistical significance. These results should be interpreted as non-confirmatory trends and require validation in larger, adequately powered studies.

Keywords: livedoid vasculopathy, connective tissue diseases, systemic lupus erythematosus, autoimmune disorders, prevalence, serological markers

Introduction

Livedoid vasculopathy (LV) is an uncommon, persistent inflammatory skin disorder characterised by painful, ulcerative lesions that usually affect the veins supplying the lower limbs.[1] LV has also been referred to as livedo vasculitis, livedoid vasculitis, and livedo reticularis with summer ulceration.[2] The term LV was first used by Bard and Winkelman in 1967.[3] LV is identified as a recurring thrombo-occlusive vascular disorder with an unknown precise etiology. It is believed to be complex and potentially related to autoimmune and microvascular dysfunction. Increased thrombotic activity, decreased fibrinolytic activity, and endothelial injury are thought to contribute to thrombus development in the capillary vasculature, leading to the disease.[4]

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Although inflammation is secondary in LV, hypercoagulability is the primary pathology. Consequently, LV differs from inflammatory vasculitis and is categorized as a vasculopathy, a coagulation disorder that occurs when a thrombus forms in the arterial lumen, impairing blood flow. LV often affects both legs and has a chronic history with sporadic and recurrent exacerbations.[5] Definitive diagnosis of LV is based on histopathological results and clinical symptoms. LV significantly impacts patients' quality of life due to its painful, scarring, and recurring clinical course. The main complaint from LV patients is acute pain, which significantly affects social activities, daily routines, study, work, walking, and overall quality of life.[6] Early disease prognosis and treatment approaches may reduce discomfort and minimize scarring, deformity, and other complications.[2] Prompt recognition of LV especially in patients with systemic autoimmune disease should trigger evaluation for thrombophilia and microvascular dysfunction. Management is often antithrombotic (± immunomodulation) rather than immunosuppression alone, with early, multidisciplinary care improving pain, healing, and scarring.[7]

A group of autoimmune diseases known as connective tissue diseases (CTDs) damage and inflame the body's connective tissues, causing inflammation and harm to various organs. A higher risk of vascular problems has been linked to several CTDs, including rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), systemic sclerosis (SSc), Sjögren's syndrome (SS), and dermatomyositis/polymyositis (DM/PM).[8] CTDs often involve microvascular injury —eg, Raynaud phenomenon and digital ischemia in SSc (endothelial dysfunction, luminal narrowing), immune-complex vasculitis in SLE (complement-mediated endothelial damage), and aPL-related hypercoagulability—mechanisms that align with LV's thrombo-occlusive pathophysiology. Prior evidence is largely small case reports/series linking LV with SLE, SSc, SS, RA, and aPL positivity, typically without biopsy confirmation, clear denominators, or comparator groups. This study addresses that gap by quantifying LV within a defined, hospital-screened cohort and testing CTD-linked clinical and serologic associations.

Although LV appears rare in the general population based mainly on small clinic-based series, patients with CTDs who are predisposed to microvascular injury and thrombosis may carry a higher burden. However, the association between LV and CTDs remains incompletely defined, and prevalence data in CTD cohorts are limited. Therefore, this study investigates the hospital-based prevalence of LV among patients with CTDs and explores relevant clinical associations. Clarifying this relationship may illuminate shared pathogenic mechanisms and inform more targeted diagnostic and treatment strategies for LV in CTD populations.

Materials and Methods

This epidemiological study used a retrospective cohort design to estimate the prevalence of livedoid vasculopathy (LV) among patients with connective tissue diseases (CTDs) within Mass General Brigham (MGB), an integrated continuum-of-care system serving ~6 million patients. We queried the MGB Research Patient Data Registry (RPDR; ~6 million patients) for encounters between January 1, 2014, and June 1, 2021, identifying adults with CTDs—including systemic lupus erythematosus (SLE), systemic sclerosis (SSc), rheumatoid arthritis (RA), Sjögren's syndrome (SS), and dermatomyositis/polymyositis (DM/PM)—and ascertaining LV using diagnosis codes and targeted keyword searches (with chart review as needed). All eligible CTD patients in RPDR during the study window were included.

Electronic medical records (EMRs) of eligible patients were reviewed to collect relevant demographic and clinical data, including age, gender, type of CTD, duration of CTD diagnosis, presence of LV, and all pertinent laboratory findings such as antiphospholipid antibodies, cryoglobulins, and antinuclear antibodies. Diagnosis of LV was established based on clinical manifestations consistent with the condition, including painful ulcers with atrophic, porcelain-white centers, surrounded by purpuric borders, on the lower extremities. Histopathological confirmation was not mandatory for inclusion.

This study was conducted in accordance with the principles of the Declaration of Helsinki and approved by the Institutional Review Board (IRB) of Mass General Brigham. Patient confidentiality was strictly maintained throughout the study, and informed consent was waived due to its retrospective nature.

Statistical analysis was performed using SPSS version 26. Chi-square test or Fisher's exact test was used to compare categorical variables. A p-value < 0.05 was considered statistically significant.

Results

A total of 1730 charts were reviewed, resulting in 108 patients diagnosed with LV. Among the reviewed group, LV represented 6.2% (108/1730). Relative to the 6,000,000 patients in the MGB RPDR, this reflects a hospital-system registered prevalence of 0.0018% (108/6,000,000), or 1.8 per 100,000. Most of the patients were women (80/108, 74.1%), while men made up 28/108 (25.9%). The average age was 44.9 ± 8.2 years. Age distribution was: 41–50 years: 32/108 (29.6%); 51–60 years: 23/108 (21.3%). Race/ethnicity data show: White 91/108 (84.3%), Asian 7/108 (6.5%), Hispanic 5/108 (4.6%). Table 1

The results regarding the prevalence of autoimmune disorders in conjunction with LV are summarized in Table 2. A total of 17 patients had autoimmune diseases, with a higher number of females, 14 (82.35%), compared to males, 3 (17.64%). The largest group was those aged 31–40 years, with 5 patients (29.41%). Concerning racial demographics, most individuals were White 11 (64.70%), while a smaller proportion identified as Black 2 (11.76%), Hispanic 2 (11.76%), Asian 1 (5.88%), and Other 1 (5.88%) (Table 2).

In this study, encompassing 17 (15.74%) patients diagnosed with autoimmune diseases, systemic lupus erythematosus (SLE) emerged as the predominant condition, affecting 10 (58.82%) individuals. The majority of SLE cases comprised female patients (70%), with 5 (50%) falling within the age group of 41–50 years. Dermatomyositis was identified in 2 (11.76%) patients, all of whom were female and primarily within the 41–50 age group. Systemic sclerosis was manifested in 2 (11.76%) patients, all female, concentrated within the 50-year age group. Additionally, Sjogren's syndrome was identified in 3(17.64%) patients, all female and in the age group >50 years. Figure 1

In examining serological markers for LV, positive results were observed in 62 (57.40%) for phospholipid antibodies, while 46 (42.59%) tested negative, yielding a p-value of 0.065. For anticardiolipin antibodies, 47 (43.51%) tested positive, while 61 (56.48%) were negative, resulting in a p-value of 0.095. Hyperhomocysteinemia was detected in 56 (51.85%) and absent in 52 (48.14%), showing a non-significant p-value of 0.952. Activated C resistance was found in 13 (12.03%), while 95 (87.96%) showed no evidence, with a p-value of 0.088. Table 3.

Table 1 Demographic Characteristics of the Patients with LV (n=108)

Demographics	Frequency	Percentages
Gender		
Male	28	25.92
Female	80	74.07
Age group		
20-30 years	20	18.51
31-40 years	17	15.74
41-50 years	32	29.62
51-60 years	23	21.29
>60 years	16	14.81
Race		
White	91	84.25
Black	2	1.85
Hispanic	5	4.62
Asian	7	6.48
Other	3	2.77

Table 2 Frequency of Autoimmune Disease with LV (n=17)

Variables	Frequency	Percentages
Gender		
Male	3	17.64
Female	14	82.35
Age group		
20-30 years	2	11.76
31-40 years	5	29.41
41-50 years	3	17.64
51-60 years	3	17.64
>60 years	4	23.52
Race		
White	11	64.70
Black	2	11.76
Hispanic	2	11.76
Asian	1	5.88
Other	1	5.88

Discussion

Livedoid vasculopathy (LV) is an uncommon disorder, with an approximated prevalence rate of 1 in 100,000.[8] It significantly infects females as compared to males, with a ratio of 3:1, and affects primarily young to middle-aged individuals.[9,10] Consistent with these studies, our findings indicate a prevalence rate of 1.8% in individuals with connective tissue disease. Similarly, a significant female predominance was observed, with 80% of patients being female.[11] This increased risk for LV in women was related to sex-specific physiological conditions, such as

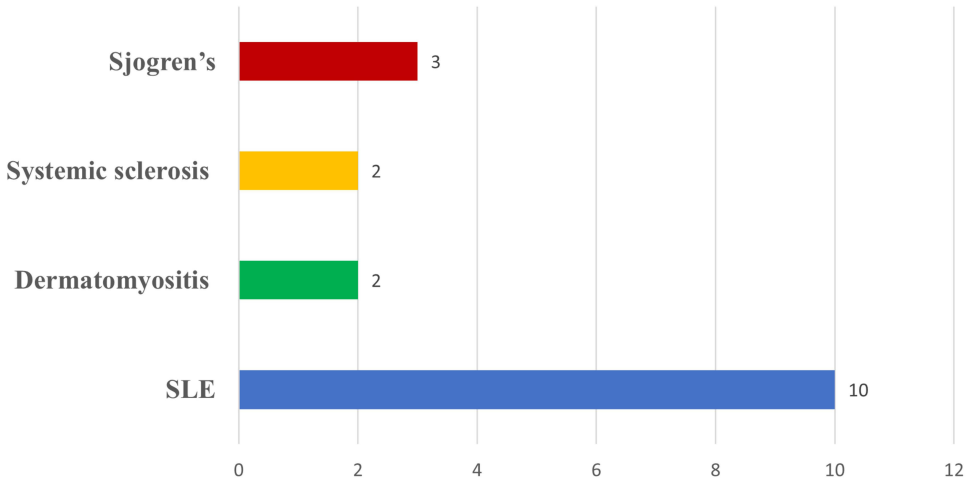

Figure 1 Patients with Autoimmune disease.

Table 3 Comparison of Serological Marker Prevalence Among Patients with Livedoid Vasculopathy (n=108)

Serological Marker	Positive	Negative	P Value
Positive phospholipid antibodies	62(57.40%)	46(42.59%)	0.065
Anticardiolipin antibodies	47(43.51%)	61(56.48)	0.095
Hyperhomocysteinemia	56(51.85%)	52(48.14%)	0.952
Activated C resistance	13(12.03%)	95(87.96)	0.088

pregnancy[12] or a higher incidence of LV-associated conditions, such as connective tissue diseases, hypercoagulable states, and venous stasis, in women.

The high prevalence rate was observed among the 41–50-year age group screened for connective tissue diseases (CTDs). This indicates that LV primarily affects middle-aged adults, which aligns with previous reports showing the age range from 32 to 53 years in other study populations.[13–15] This finding offers valuable data on the prevalence of LV within this specific patient population. The racial distribution of the sample showed that the majority of patients infected with LV were white,[16–19] followed by Asian,[20–23] and the Hispanic population.[24] While the study offers a snapshot of the racial composition within our specific patient population, the racial distribution (majority white) may not accurately represent the broader population. Further research involving more diverse populations is necessary to ascertain whether racial disparities exist in LV prevalence among CTD patients.

LV disorders are most common in individuals with connective tissue issues compared to the general population. A retrospective study on 45 patients with LV, Hairston et al found that six individuals had associated connective tissue diseases.[25] However, these abrasions were initially linked to vasculitis, but now it has been investigated that inflammation injury of vessels has been associated with fewer than 20% of lesions in CTD populations.[26] A significant association was observed between LV and the presence of autoimmune disorders. Our study showed that up to 15.74% of the LV-infected individuals were positive for CTD, including systemic lupus erythematosus (SLE), which emerged as the predominant condition co-occurring with LV, with the majority of SLE cases comprising female patients. Subsequently, Sjogren's syndrome was identified as a moderate condition in LV patients, all of whom were females. Remarkably, all individuals with Sjogren's syndrome identified were females over 50 years old, suggesting a potential age-related link. In contrast, previous studies show that LV is uncommon in systemic lupus erythematosus (SLE); however, when it persists, it is mostly associated with antiphospholipid antibodies. Sopeña et al reported that individuals with a previous history of LV and a background of SLE reveal negative antiphospholipid antibodies,[27] Indicating that alternative pathways, such as the presence of circulating immune complexes, may play a crucial role in the progression of lupus vasculitis (LV) within the systemic lupus erythematosus (SLE) population. However, other studies have reported that rheumatoid arthritis (RA) is the most common connective tissue disease (CTD) associated with LV. Additionally, undifferentiated CTDs, mixed CTDs, and scleroderma were also observed within the CTD population in similar proportions.[25,28] A subsequent study conducted by Cardoso et al examined LV within a population afflicted by Sjögren's syndrome, indicating that the condition has been associated with mutations in the enzyme methylenetetrahydrofolate reductase, which contribute to a hypercoagulable state. This finding implies a potential correlation between LV and Sjögren's disease.[19] However, Further exploration with larger sample sizes is required to validate these associations and investigate underlying pathways.

Regarding serological markers, our findings indicate the presence of phospholipid antibodies with moderate positivity following anticardiolipin antibodies. Hyperhomocysteinemia and Activated C resistance did not demonstrate significant evidence respectively. Individuals with antiphospholipid antibodies are particularly exposed to LV. In a retrospective study conducted by Hairston et al,[25] shows that anticoagulant was detected in 17.9% of individuals exhibiting LV, whilst anticardiolipin antibodies were identified in 28.6% of the subjects. Additionally, a separate study suggested that LV might represent a manifestation of antiphospholipid syndrome, which is associated with elevated

levels of anticardiolipin antibodies.[29] However, the serological marker in our study shows no significant importance. Therefore, larger studies with accurate control groups are essential to definitively determine the association of these markers with LV in CTD individuals. Finally, this investigation provides valuable insights into the characteristics of LV patients within the CTD population. The observed co-occurrence of LV with autoimmune disorders, particularly SLE, suggests a potential link. However, limitations such as selection bias and non-significant serological marker analysis require further investigation. Replicating the study with a more diverse population, including control groups without LV to strengthen comparisons, investigating the biological mechanisms connecting LV and specific autoimmune disorders, and exploring the potential influence of CTD medications on LV development are necessary. Conducting additional research could lead to a more comprehensive understanding of LV in CTD patients, ultimately improving diagnostic tools and treatments approaches.

Conclusion

This study reveals a notable prevalence of livedoid vasculopathy (LV) among patients with connective tissue diseases (CTDs), with a significant female predominance and a higher occurrence in middle-aged adults. The link between LV and autoimmune conditions, particularly systemic lupus erythematosus (SLE), underscores the need for increased clinical vigilance. Although serological markers such as phospholipid antibodies, anticardiolipin antibodies, hyperhomocysteinemia, and activated C resistance showed trends toward association with LV, they were not statistically significant.

Disclosure

The author reports no conflicts of interest in this work.

References

1. Bilgic A, Ozcobanoglu S, Bozca BC, Alpsoy E. Livedoid vasculopathy: a multidisciplinary clinical approach to diagnosis and management. *Int J Womens Dermatol*. 2021;7(5Part A):588–599. doi:10.1016/j.ijwd.2021.08.013
2. Eswaran H, Googe P, Vedak P, Marston WA, Moll S. Livedoid vasculopathy: a review with focus on terminology and pathogenesis. *Vasc Med*. 2022;27(6):593–603. doi:10.1177/1358863x221130380
3. Bard JW, Winkelmann R. Livedo vasculitis: segmental hyalinizing vasculitis of the dermis. *Arch Dermatol*. 1967;96(5):489–499. doi:10.1001/archderm.1967.01610050011002
4. Burg MR, Mitschang C, Goerge T, Schneider SW. Livedoid vasculopathy - A diagnostic and therapeutic challenge. *Front Med*. 2022;9:1012178. doi:10.3389/fmed.2022.1012178
5. Criado PR, Rivitti EA, Sotto MN, et al. Livedoid vasculopathy: an intringuing cutaneous disease. *An Bras Dermatol*. 2011;86(5):961–977. doi:10.1590/s0365-05962011000500015
6. Polo Gascón MR, de Carvalho JF, de Souza Espinel DP, Barros AM, Alavi A, Criado PR. Quality-of-life impairment in patients with livedoid vasculopathy. *J Am Acad Dermatol*. 2014;71(5):1024–1026. doi:10.1016/j.jaad.2014.06.030
7. Seguí M, Llamas-Velasco M. A comprehensive review on pathogenesis, associations, clinical findings, and treatment of livedoid vasculopathy. *Front Med*. 2022;9:993515. doi:10.3389/fmed.2022.993515
8. Alavi A, Hafner J, Dutz JP, et al. Livedoid vasculopathy: an in-depth analysis using a modified Delphi approach. *J Am Acad Dermatol*. 2013;69(6):1033–1042.e1031. doi:10.1016/j.jaad.2013.07.019
9. Weishaupt C, Strölin A, Kahle B, et al. Anticoagulation with rivaroxaban for livedoid vasculopathy (RILIVA): a multicentre, single-arm, open-label, phase 2a, proof-of-concept trial. *Lancet Haematol*. 2016;3(2):e72–79. doi:10.1016/s2352-3026(15)00251-3
10. Di Giacomo TB, Hussein TP, Souza DG, Criado PR. Frequency of thrombophilia determinant factors in patients with livedoid vasculopathy and treatment with anticoagulant drugs--a prospective study. *J Eur Acad Dermatol Venereol*. 2010;24(11):1340–1346. doi:10.1111/j.1468-3083.2010.03646.x
11. Gardette E, Moguelet P, Bouaziz JD, et al. Livedoid vasculopathy: a french observational study including therapeutic options. *Acta Derm Venereol*. 2018;98(9):842–847. doi:10.2340/00015555-2965
12. Sankar A, Hinshaw K. Livedoid vasculopathy and pregnancy. *Int J Gynaecol Obstet*. 2009;107(3):248–249. doi:10.1016/j.ijgo.2009.06.020
13. Weishaupt C, Strölin A, Kahle B, et al. Characteristics, risk factors and treatment reality in livedoid vasculopathy - a multicentre analysis. *J Eur Acad Dermatol Venereol*. 2019;33(9):1784–1791. doi:10.1111/jdv.15639
14. Renner R, Dissemond J, Goerge T, Hoff N, Kröger K, Erfurt-Berge C. Analysis of the German DRG data for livedoid vasculopathy and calciphylaxis. *J Eur Acad Dermatol Venereol*. 2017;31(11):1884–1889. doi:10.1111/jdv.14190
15. Criado PR, Rivitti EA, Sotto MN, de Carvalho JF. Livedoid vasculopathy as a coagulation disorder. *Autoimmun Rev*. 2011;10(6):353–360. doi:10.1016/j.autrev.2010.11.008
16. Khenifer S, Thomas L, Balme B, Dalle S. Livedoid vasculiltis associated with a double heterozygous Factor V Leiden and prothrombin G20210A gene mutations. *Clin Exp Dermatol*. 2009;34(8):e811–813. doi:10.1111/j.1365-2230.2009.03541.x
17. Marsch WC, Komatsuzaki S, Mueller A, et al. Livedoid vasculopathy: does hyperhomocysteinaemia play an aetiological role? *Eur J Dermatol*. 2019;29(3):287–293. doi:10.1684/ejd.2019.3554

18. Kavala M, Kocaturk E, Zindanci I, Turkoglu Z, Altintas S. A case of livedoid vasculopathy associated with factor V Leiden mutation: successful treatment with oral warfarin. *J DermatolTreat*. 2008;19(2):121–123. doi:10.1080/09546630701670305
19. Cardoso R, Gonçalo M, Tellechea O, et al. Livedoid vasculopathy and hypercoagulability in a patient with primary Sjögren's syndrome. *Int J Dermatol*. 2007;46(4):431–434. doi:10.1111/j.1365-4632.2007.03229.x
20. Nakamura S, Kishibe M, Nishi K, et al. Livedoid vasculopathy; favorable clinical response with low dose warfarin. *Eur J Dermatol*. 2011;21 (6):1011–1012. doi:10.1684/ejd.2011.1529
21. Tsai TF, Yang CH, Chu CY, et al. Polymorphisms of MTHFR gene associated with livedoid vasculopathy in Taiwanese population. *J Dermatol Sci*. 2009;54(3):214–216. doi:10.1016/j.jdermsci.2008.12.010
22. Martorell-Calatayud A, Requena C, Nagore-Enguídanos E, Guillén-Barona C. Multiple, painful, treatment-resistant leg ulcers associated with dermatomyositis-like lesions over the interphalangeal joints induced by hydroxyurea. *Actas Dermosifiliogr*. 2009;100(9):804–807. doi:10.1016/S0001-7310(09)72554-2
23. Shankar S, Vasudevan B, Deb P, Langer V, Verma R. Clinical Images: livedoid vasculopathy—a vasculitic mimic. *Arch Dermatol*. 2006;142:1413–1418. doi:10.1001/archderm.142.11.1413
24. Ray R, Sharma A, Vasudevan B, Sridhar J, Deo R, Mohanty CS. Livedoid vasculopathy with hyperhomocysteinemia responding to hyperbaric oxygen therapy. *Indian J Dermatol*. 2015;60(5):524. doi:10.4103/0019-5154.159657
25. Hairston BR, Davis MD, Pittelkow MR, Ahmed I. Livedoid vasculopathy: further evidence for procoagulant pathogenesis. *Arch Dermatol*. 2006;142(11):1413–1418. doi:10.1001/archderm.142.11.1413
26. Shanmugam VK, Steen VD, Cupps TR. Lower extremity ulcers in connective tissue disease. *Isr Med Assoc J*. 2008;10(7):534–536.
27. Sopeña B, Pérez-Rodríguez MT, Rivera A, Ortiz-Rey JA, Lamas J, Freire-Dapena MC. Livedoid vasculopathy and recurrent thrombosis in a patient with lupus: seronegative antiphospholipid syndrome? *Lupus*. 2010;19(11):1340–1343. doi:10.1177/0961203310373783
28. Cojocaru M, Cojocaru IM, Silosi I, Vrabie CD, Tanasescu R. Extra-articular Manifestations in Rheumatoid Arthritis. *Maedica*. 2010;5(4):286–291.
29. He H, Wu N. A 75-year-old woman with primary antiphospholipid syndrome presenting with livedoid vasculopathy. *Dermatol Ther*. 2020;33(4): e13480. doi:10.1111/dth.13480

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