

## Case Series

# Varied presentations of cutaneous vasculitis: a case series

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**Received:** 13 June 2020

**Accepted:** 06 July 2020

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### ABSTRACT

Vasculitis involves a wide spectrum of clinicopathological process with reactive damage to the involved blood vessels. There is loss of vessel integrity instigating haemorrhage & luminal compromise leading to ischemia and necrosis of the tissue supplied by the involved vessels. It may affect varied size and type of blood vessels at different locations. It may be primary or secondary to systemic disease. It may involve a single organ like skin or may involve different organ systems at the same time. This case series include six cases of cutaneous vasculitis affecting different organs with varied presentations. Skin biopsies of six patients with unusual presentations were studied. Their complete history, physical examinations, laboratory investigations including serology were analysed and correlated with histopathological findings. The patients presented with different duration of symptoms varying from as short as 15 days to 1 year. Skin lesions were present in all cases while cardiac manifestation was seen in one. Serology and autoimmune disease markers were negative in all cases except one. However, histopathological features were in concordance with the clinical diagnosis of vasculitis. They were further classified as vasculitis secondary to Churg Strauss syndrome, venous stasis, Henoch Schonlein purpura or leukocytoclastic vasculitis. Vasculitis though a rare disease may manifest as an acute or chronic condition. It needs timely diagnosis by histopathological examination to aid in further management. It is important to assess the clinical severity in primary and secondary vasculitis, as it determines morbidity and mortality.

**Keywords:** Churg strauss syndrome, Clinico-pathological process, Cutaneous vasculitis, Henoch schonlein purpura, Leukocytoclastic vasculitis, Vasculitis

### INTRODUCTION

The vasculitis are characterised by spontaneous damage to mural architecture due to presence of inflammatory leukocytes in vessel walls. It leads to bleeding as a result of insult to vessel integrity and subsequent tissue ischemia and necrosis secondary to compromise of the lumen. It may occur as an initial process or may be due to another underlying disease (autoimmune disease) or can be associated with other precipitants such as drugs, infections or malignancy affecting vessels of different size, type and location. The precise pathogenetic mechanisms triggering these diseases are undetermined.

They are often austere and at times grave requiring prompt cognizance and treatment. The disease manifestation depends on the organ involved which in turn depends on the type of vasculitis.<sup>1</sup>

### CASE SERIES

This case series highlights the varied manifestation of cutaneous vasculitis. Skin biopsies of six patients with unusual presentations were studied. Their complete history, physical examinations, laboratory investigations including serology were analysed and correlated with histopathological findings (Table 1).

### Case 1

58 years old gentleman, farmer from Western India, with a past history of bronchial asthma, presented with a complaint of pain in both lower limbs for 15 days, which got aggravated on walking. He also complained of headache and difficulty in chewing food simultaneously for the last 8 days. He was an average built, fairly nourished person. On evaluation he had tachycardia with blood pressure 160/90mmHg. There was no pallor, icterus, cyanosis, clubbing or pedal edema. There was periorbital oedema and tortuous blood vessels over forehead. On local examination multiple tender erythematous papular, non-purulent lesions were present on the dorsum of foot with blisters (Figure 1 and 2).



**Figure 1: Periorbital oedema, tortuous blood vessel on forehead.**



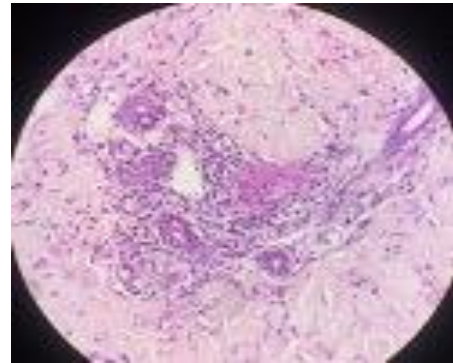
**Figure 2: Bilateral erythematous papular lesions >5 on dorsum of foot.**

Blood investigation showed eosinophilia with raised ESR. Auto-immune workup showed presence of p-ANCA and anti thyroid microsomal antibody.

The patient underwent skin biopsy from lower limb which showed following features (Figure 3).

During the period of hospital stay, while the patient was evaluated for skin lesions, he developed chest pain. On evaluation ECG showed ST elevation with elevated cardiac enzymes (CK-MB and Trop I). He was subsequently diagnosed as suffering from myocardial infarction and managed accordingly.

The clinico-pathologic spectrum was suggestive of Churg Strauss vasculitis which was complicated with myocardial infarction.



**Figure 3: Endothelial cell proliferation obliterating vascular lumen with marked perivascular eosinophil and neutrophilic infiltrate and fibrinoid necrosis of the vessel wall (H&E40X).**

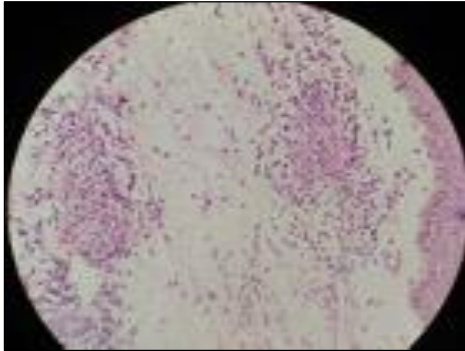
### Case 2

45 years old lady, housewife without any previous comorbidity, presented with a complaint of rashes on both upper and lower limbs along with abdominal pain and vomiting for the last 3 months. She had burning micturition and joint pain involving small joints for the same duration. She was thin built and poorly nourished. On examination, multiple erythematous papules with bilateral lower limb oedema were seen (Figure 4). Her routine blood investigations comprising complete blood count, renal and liver function test were unremarkable. She underwent skin biopsy from the lower limb (Figure 5).



**Figure 4: Multiple erythematous papules with oedema.**

Considering the clinical feature and histopathological features, the patient was further evaluated. Her urine analysis showed hematuria and proteinuria. This clinical correlation led to diagnosis of vasculitis secondary to Henoch Schonlein purpura.



**Figure 5: The dermis perivascular infiltrate consisting of degenerated neutrophils and few eosinophils (H&E 400x).**

### Case 3

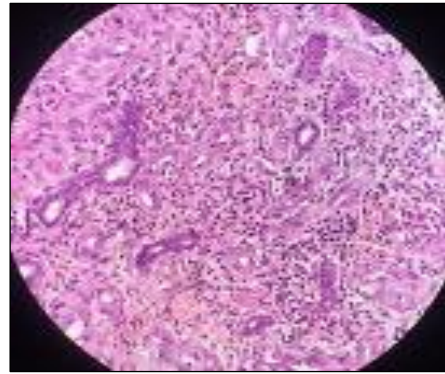
74 years elderly obese male presented with itching over both lower limb for 2 years duration and painful ulcers over leg with discharge for 4 months.



**Figure 6: Single well defined ulcer with discharge.**

He had a past history of trauma and varicose vein. On examination there was a single well defined ulcer with serous discharge present on the middle 1/3<sup>rd</sup> of the right lower limb (Figure 6). The skin biopsy was done from the lower limb (Figure 7).

The clinicopathological spectrum led to the diagnosis of stasis dermatitis with vasculitis. Since it involves varicose veins of the lower limb it was categorized as venous vasculitis.



**Figure 7: Lobular pattern of superficial and deep dermal neovascularization with perivascular lymphocytic infiltrate and fibrosis (H&E-100x).**

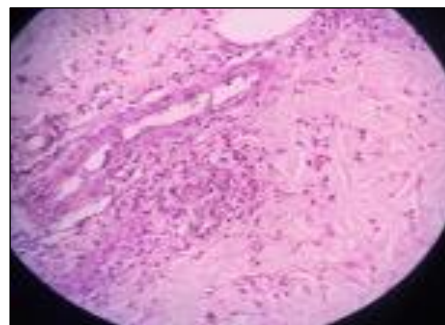
### Case 4

35 years old lady, without any comorbidity presented with a painful ulcer over her leg for 6 weeks duration. Her general condition was fair. On examination a tender indurated erythematous ulcerated papule with scab formation on leg was seen (Figure 8). The patient underwent skin biopsy (Figure 9).

No specific aetiology could be determined. Hence diagnosed as leukocytoclastic vasculitis. The patient was lost to follow up.



**Figure 8: Painful indurated erythematous ulcerated papule with scab formation.**



**Figure 9: Endothelial cell proliferation with perivascular degenerated neutrophils and eosinophils along with fibrinoid necrosis (H&E100X).**



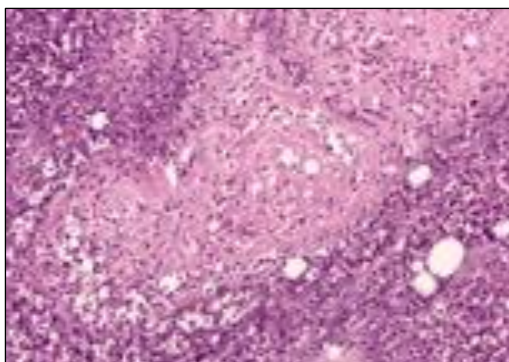
**Table 1: Characteristics of patients.**

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Age	58 years	45 years	74 years	35 years	65 years	70 years
Sex	Male	Female	Male	Female	Male	Male
Symptom	Bilateral lower limb pain Headache Difficulty in chewing Chest pain	Abdominal pain Vomiting Dysuria Multiple joint pain	Itching in bilateral lower limb Single ulcer over leg Varicose vein	Painful Ulcer over leg	Erythema and swelling of Left lower limb	Blackening of Right small toe
Duration	2 weeks	3 months	2 years	6 weeks	2 months	2 months
Sign	Tachycardia Raised BP Periorbital swelling Tortuous vessels over forehead	Tender abdomen	Varicose vein Tender ulcer over leg	Skin ulceration	Cellulitis	Cellulitis
Skin lesions	Multiple tender erythematous, papular, non-purulent lesions with blisters	Multiple erythematous papules.	Ulcer	Tender ulcerated lesion with papular eruptions	Gangrene	Gangrene
Site	Dorsum of foot	Upper and lower limbs	Right lower limb	Lower limb	Lower limb	Right lower toe
Lab inv	Eosinophilia	Hematuria Proteinuria	WNL	WNL	Neutrophilic leukocytosis Thrombocytopenia	Leukocytosis
Autoimmune workup	pANCA+ anti thyroid microsomal Ab+	WNL	WNL	WNL	Not done	Not done
Histopathological Diagnosis	Churg Strauss vasculitis with myocardial infarction.	Henoch Schonlein purpura	Venous vasculitis	Leukocytoclastic vasculitis	Leukocytoclastic vasculitis	Leukocytoclastic vasculitis

WNL- Within Normal Limit.

### Case 5

65 years old gentleman, known case of diabetes mellitus for 10 years and hypertension for 6 years, presented with left lower limb cellulitis followed by gangrene.



**Figure 10: Inflammatory infiltrate in the subcutaneous tissue and fats causing panniculitis and myositis. Neutrophilic infiltrate infiltrating vessel wall and fibrinoid necrosis (H&E 100x).**

He complaint of swelling and erythema of the left lower limb for 2 months followed by discoloration and discharge for the last 8 days. He also developed

intermittent fever, nausea and vomiting. His haemoglobin was 12.7gm/dl and total leucocyte count was 26000/cumm with neutrophil preponderance. His blood sugar was uncontrolled with HbA1c was 7.9 gm/dl.

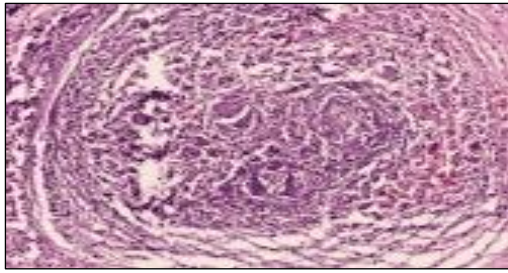
Colour Doppler study of lower limb showed no evidence of deep vein thrombosis. Diffuse subcutaneous soft tissue inflammation and oedema in the left leg was present suggestive of cellulitis. Atherosclerotic changes were seen in artery of both lower limb.

Subsequently he underwent amputation of the left lower limb. On Histopathological examination, he was thus diagnosed as Leukocytoclastic vasculitis (Figure 10).

### Case 6

70 year elderly, diabetic male with history of peripheral vascular disease presented with the complaint of blackening of the right small toe for 2 months with pus discharge.

He has leucocytosis (TLC-14000/cumm) with raised D dimer (526ng/dl) with controlled blood sugar. He underwent amputation of the right small toe. Histopathological examination was done which was suggestive of Leukocytoclastic vasculitis (Figure 11).



**Figure 11: Lobular neovascularizations, blood vessels showing transmigrating lymphocytes, endothelial proliferation and fibrinoid necrosis (H&E 100x).**

## DISCUSSION

Though vasculitis is rare, the potential for severe organ damage or death from these diseases, makes it imperative for the physician and pathologist to have a high degree of suspicion. It should be evaluated in patients who present with systemic or constitutional symptoms along with single and/or multiorgan dysfunction, and especially with some other key manifestations (skin, renal etc).

Traditionally vasculitis was classified on the basis of dimension of vessels involved. But the recent The Chapel Hill Consensus Conference (CHCC) concede that some types of vasculitis do not comprise of a single predominant size of vessel (variable-vessel vasculitis) (Table 2). Apart from large, medium and small vessel vasculitis, it also incorporated varying vessels, solo organ and vasculitis accompanying systemic disease or particular etiology.<sup>2</sup>

We will be discussing cases of cutaneous vasculitis with varied atypical manifestation. The initial feature of vasculitis in vessels which were smaller than arteries to be comprehensively studied was purpura. In 1808, Willan differentiated purpura instigated by systemic febrile infections from those of non-infectious cause. He observed that non-infectious purpura had a predisposition for the lower limbs, was manifested by recurrent groups of lesions, and might be associated with systemic disease. A wide spectrum of signs and symptoms that were related with purpura, and thus with small-vessel vasculitis, including nephritis, pulmonary haemorrhage, arthritis, peripheral neuropathy, abdominal pain, epistaxis and iritis were exemplified by Schönlein, Henoch, Osler, and others in the twentieth century. Osler identified that these clinical pictures were due to necrotizing inflammation in small vessels.<sup>3</sup>

The first case depicts a very rare scenario of Churg Strauss Syndrome with myocardial infarction. The patient here presented with a history of asthma along with headache, lower limb pain, periorbital edema and on evaluation was detected to have tortuous blood vessels, palpable purpura, eosinophilia, pANCA and AMA positivity and subsequently he developed myocardial infarction.

**Table 2: The 2012 international chapel hill consensus conference on the nomenclature of vasculitides adopted the following names for vasculitides.**

<b>Small-vessel vasculitis</b>
Immune complex small-vessel vasculitis
IgA vasculitis (Henoch-Schönlein Purpura)
Hypocomplementemic urticarial vasculitis (anti-C1q vasculitis)
Anti-glomerular basement membrane disease
Cryoglobulinemic vasculitis
ANCA-associated vasculitis
Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
Granulomatosis with polyangiitis (Wegener's granulomatosis)
Microscopic polyangiitis
<b>Medium-vessel vasculitis</b>
Kawasaki disease
Polyarteritis nodosa (PAN)
<b>Large-vessel vasculitis</b>
Giant cell arteritis
Takayasu arteritis
<b>Single-organ vasculitis</b>
Isolated aortitis
Primary central nervous system vasculitis
Cutaneous arteritis
Cutaneous leukocytoclastic angiitis
Others
<b>Variable-vessel vasculitis</b>
Cogan's syndrome
Behçet's syndrome
<b>Vasculitis associated with systemic disease</b>
Sarcoid vasculitis
Lupus vasculitis
Rheumatoid vasculitis
Others
<b>Vasculitis associated with probable etiology</b>
Drug-associated immune complex vasculitis
Drug-associated ANCA-associated vasculitis
Hepatitis B virus-associated vasculitis
Hepatitis C virus-associated cryoglobulinemic vasculitis
Syphilis-associated aortitis
Cancer-associated vasculitis
Others

Coronary artery involvement is seen in vasculitic disorders like Wegener's granulomatosis, Churg Strauss syndrome, polyarteritis nodosa, infectious angiitis, Takayasu's arteritis, granulomatous giant cell arteritis and thromboangiitis obliterans.<sup>4</sup>

Churg-Strauss syndrome (now known as Eosinophilic granulomatosis with polyangiitis, EGPA) is described by the American College of Rheumatology (ACR) as necrotising eosinophilic vasculitis of small and medium

sized vessels which is associated with presence of one or more of the following: asthma, more than 10% eosinophils in differential leukocyte count, sinusitis, migratory or transient pulmonary infiltrates, and mononeuropathy, or polyneuropathy.<sup>5</sup> ANCA is present in approximately 40 percent of patients, usually anti-MPO ANCA. It is a multisystem disorder; the most commonly affected organ is lung followed by skin, however any organ can be involved.<sup>5</sup>

Skin lesions were seen in more than half of patients with EGPA, which usually presents as tender subcutaneous nodules on the extensor surfaces of the arm, particularly the elbows, and also on hands and legs.<sup>6</sup> These can also manifest as an erythematous rash which may be macular or papular or varied hemorrhagic lesions, like petechiae, palpable purpura, or extensive ecchymosis.<sup>7</sup> Skin biopsy classically reveals a leukocytoclastic vasculitis with eosinophilic infiltration. Palisading granulomas and/or eosinophilic infiltration of dermal nerve fibres may also be noted.<sup>7</sup>

Cardiac involvement which may be seen in up to 60% of patients, presents as myocarditis, congestive heart failure, pericardial effusions and intracardiac thrombosis. Rarely Myocardial infarction (MI) too can occur.<sup>8-11</sup> The morbidity and mortality associated with EGPA is mainly due to vasculitis of extrapulmonary organs. The mean age at diagnosis of EGPA is 40 years.<sup>12</sup> There is no gender predisposition. Etiopathogenesis is not clear but is due to deranged immune function and some genetic factors.<sup>13,14</sup> Several drugs like leukotriene modifying agents, inhaled glucocorticoids,omalizumab and cocaine was associated with the disease.<sup>13</sup>

The next case is of a middle aged female who presented with erythematous papular rash over upper and lower limb along with pain abdomen, vomiting, dysuria, haematuria and arthralgia. She is diagnosed as Henoch Schonlein purpura (HSP).

Immunoglobulin A (IgA) vasculitis (or HSP) is a vasculitis with systemic manifestation distinguished by the tissue deposition of IgA1-dominant immune complexes involving mostly small vessels (primarily capillaries, venules, or arterioles). Skin and gastrointestinal tract are predominantly affected, often leads to arthritis also. Children account for 90% of cases. The disease is usually preceded by an upper respiratory tract infection, especially streptococcus. Rarely drugs or vaccination has been suspected as causative agent.<sup>15</sup>

The disease manifestations and epidemiology significantly differs between adults and children. The incidence in adults with available data is around showing 13- 14 cases per million adults per year. The mean age of adults with IgAV was around 50 years in various large series. Adult men and women are equally affected as compared to children where male preponderance is documented. The role of seasonal variability, and

antecedent infections are less common in adults as opposed to children. The disease is usually more severe in adults. Nephritis manifests in 50-80% of adults, but only 20-40% of children. End stage renal disease develops in 10-20% of adults with nephritis as compared to 1% of children.<sup>15-17</sup>

The mandatory diagnostic criterion involves purpura (often palpable and in clusters) or petechiae, with lower limb predominance and absence of thrombocytopenia or coagulopathy; with one or more of the following:<sup>18</sup>

- Abdominal pain (usually diffuse, with acute onset)
- Arthritis or arthralgia (acute onset)
- Renal involvement (proteinuria, hematuria)
- Leukocytoclastic vasculitis or proliferative glomerulonephritis, with predominant IgA deposition.

IgAV (HSP) is an immune-mediated vasculitis related to IgA deposition. Pathogenesis involves immunologic, genetic, and environmental factors.<sup>19</sup>

IgAV (HSP) is distinguished by leukocytoclastic vasculitis with presence of IgA immune complexes within involved organs. The histopathological examination of purpuric lesions shows the involvement of small vessels (primarily postcapillary venules) within the papillary dermis. The inflammatory infiltrate typically consist of neutrophils and monocytes.<sup>20</sup>

The next case is of an elderly male with varicose vein who developed stasis dermatitis and ulceration. Stasis dermatitis, or stasis eczema, is a fairly common inflammatory dermatosis of the lower limbs presenting in patients with chronic venous insufficiency, often in relation with varicose veins, dependent chronic edema, hyperpigmentation, ulceration and lipodermatosclerosis.<sup>21</sup>

The most common risk factors are increasing age, female sex, obesity, prolonged standing, pregnancy and a history of deep venous thrombosis.<sup>22</sup>

The pathogenesis involves development of venous hypertension which further leads to chronic venous insufficiency.<sup>23</sup> The gaiter area of the leg, which extends from just above the malleolus to below the knee, is most common site of venous insufficiency ulcers; especially the medial aspect.

Ulcers are classically shallow, tender with irregular borders and yellow, fibrinous exudate overlying the wound bed. In the absence of arterial insufficiency, arterial pulses are normal.

Erythema and scaling may be seen around ulcers suggestive of venous stasis dermatitis. Lipodermatosclerosis, also known as sclerosing panniculitis, may develop in the setting of prolonged venous hypertension and insufficiency. It manifests as

induration and fibrosis of the lower medial leg, which may be erythematous and tender and thus mistaken for cellulitis.<sup>24</sup>

In uncomplicated stasis dermatitis the epidermis shows changes of parakeratosis, hyperkeratosis, acanthosis, and mild spongiosis. Spongiosis may be easily seen in cases with a superimposed contact dermatitis. Dermal involvement includes small blood vessels proliferation in the papillary dermis, perivascular lymphocytic infiltration, extravasated erythrocytes, hemosiderin-laden macrophages and variable dermal fibrosis.<sup>22</sup> A positive iron stain and the evaluation of the iron deposition pattern may be helpful to confirm the diagnosis.<sup>23</sup>

Ultrastructural morphometric findings of dermis include increased mast cell number, thickened capillaries basal lamina, and presence of transforming growth factor-beta 1 (TGF-beta 1).<sup>25</sup> Lipodermatosclerosis is characterised by septal involvement of the subcutaneous fat, fat necrosis, lipomembranous changes, microcyst formation, elastosis, and calcification of adipocytes.<sup>24</sup>

The next three cases were leukocytoclastic vasculitis (LCV). One patient was a middle aged female without any comorbidity or predisposition while other two were elderly with comorbidities and peripheral vascular disease.

Cutaneous leukocytoclastic vasculitis is the term coined by unanimity by the Chapel Hill Conference in place of hypersensitivity vasculitis as an isolated cutaneous leukocytoclastic angiitis without systemic vasculitis or glomerulonephritis.<sup>2</sup> The most common manifestation of LCV is that of (non-blanching) palpable purpuric lesions that occur mainly on dependent areas, mostly the feet and lower limbs. Smaller lesions less than 3 mm are known as petechiae. Lesions may be seen on the forearms and hands, rarely on the upper part of the trunk. Other cutaneous features of LCV consist of erythematous plaques, urticarial wheals, bullous hemorrhagic lesions or ulcers. Rarely one may get livedo reticularis, deep skin ulcers and nodules.<sup>26</sup>

Two deep punch biopsy including the deep dermis and subcutis should be carried out for histopathological examination within 1-2 days of the onset of the lesion; one for hematoxylin eosin staining and other for direct immunofluorescence (DIF) staining.<sup>27</sup>

LCV involves arterioles, capillaries and postcapillary venules. The characteristic features of LCV are: infiltration of neutrophil within and around the vessel wall with signs of activation, degranulation and destruction of neutrophils, manifested by leukocytoclasia (nuclear dust), fibrinoid necrosis (intramural or perivascular fibrin deposition), and signs of damage (which involve red blood cells extravasation, endothelial cells damage) of the vessel wall and surrounding tissue.<sup>28</sup> Luminal thrombus can be seen in

patients with severe lesions. Immunoreactants (immunoglobulins) deposition in vessels by DIF is noted in up to 92% of the vasculitic histopathological specimens in the initial hours of the onset of lesion.<sup>28</sup>

The annual incidence varies from 2.3-50.4 cases per million population in different studies. Male are affected slightly more often than females, while in others no difference in gender is seen. There is no seasonal predilection.<sup>28</sup>

It may be either a primary disease, limited to the skin or affecting other organs in the body, or secondary to drugs, infections, autoimmune diseases or malignancies.<sup>28</sup>

## CONCLUSION

After reviewing all the six cases, with skin being the most common target organ for vasculitis, it is fair enough to conclude that diagnosis is best determined by histopathological finding. However for exact categorization of vasculitis correlation with clinical features and immunological investigation is essential. Categorizing vasculitis into leukocytoclastic, eosinophilic or lymphocytic vasculitis on histopathology can help to provide a window to the underlying systemic disorder and early management for the same.

*Funding: No funding sources*

*Conflict of interest: None declared*

*Ethical approval: Not required*

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**Cite this article as:** Gupta P, Ganorkar S, Bhalekar S, Rao R. Varied presentations of cutaneous vasculitis: a case series. *Int J Res Med Sci* 2020;8:3066-73.