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SHORT COMMUNICATIONS

HISTOPATHOLOGICAL STUDY OF 60 CASES OF CUTANEOUS VASCULITIS

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Sixty cases of cutaneous vasculitis were selected from department of Skin and STD, Government Medical College/Rajendra Hospital, Patiala. Detailed history, general physical examination, systemic examination and dermatological examination were conducted in each and every case. Routine investigations were done. Clinical diagnosis was confirmed by biopsy. Histopathologic study was conducted by using H & E stain under 100X and 400X by light microscopy.

Key Words : Histopathology, Vasculitis

Introduction

The term cutaneous vasculitis refers to a group of diseases characterized clinically by the spectrum of changes ranging from erythema and urticaria to purpura, ischaemia, necrosis and infarction.[1] Histologically cutaneous vasculitis (CV) is characterized either by destruction of vessel wall due to endothelial cell degeneration or by thickening of vessel wall due to oedema, proliferation of endothelial cells, fibrinoid deposits or infiltration by inflammatory cells. Extravasation of RBCs and occlusion of lumen is also seen.[2,3] There are two main types of vasculitis ie leucocytoclastic vasculitis (leuco vas) and lymphocytic vasculitis (lym vas). The former is characterized by endothelial cell swelling, oedema and fibrinoid degeneration of small cutaneous vessels and perivascular neutrophilic infiltrate (PVNI), at places showing fragmentation of nuclei ie, karyorrhexis or leucocytoclasis.[4] The lymphocytic variety is a reactive process characterized by perivascular lymphocytic infiltrate (PVLI) in the vicinity of capillaries with extravasation of RBCs in early cases and endothelial cell proliferation and dilatation of lumen in late cases.[5] In some cases, features of both types are seen giving a mixed picture.

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Materials and Methods

Sixty cases of cutaneous vasulitis were selected from department of Skin and VD, Rajendra Hospital, Patiala. Detailed history, examination and relevant investigations were done. The histopathology was studied after staining the cut section with H&E stain for typical changes by light microscopy under 100X and 400X and clinicopathological correlation was studied.

Results (Table I)

Out of 60 cases of CV, 17 cases of EM showed PVLI with dilated vessels in 17, endothelial cell proliferation in 10, extravasated RBCs in 4, fibrinoid degeneration in 3 and occlusion of lumen in 2 cases. All the 10 cases of HSP showed nuclear dust, fibrinoid degeneration in 8 cases, extravasated RBCs in 7, endothelial cell proliferation in 5, dilated vessels in 4 and occlusion of lumen in one case with PVNI in 4, mixed ie, PVNI and PVLI in 4 and PVLI with few neutrophils in 2 cases. All the 9 cases of

Table I. Clinicopathological correlation in 60 cases of cutaneous vasculitis

Clinical diagnosis	No. of cases	Histological types of vasculitis		
		Leucocy-toclastic	Mixed	Lymphocytic
Erythema multiforme (EM)	17	0	0	17
Henoch Schoenlein purpura (HSP)	10	4	4	2
Allergic cutaneous vasculitis (ACV)	9	2	4	3
Erythema nodosum (EN)	7	0	1	6
Pyoderma gangrenosum (PG)	3	0	0	3
Schamberg's purpura (SP)	3	0	0	3
Fixed drug eruption (extensive) (FDE)	2	0	0	2
Erythema elevatum diutinum (EED)	2	0	0	2
Urticarial vasculitis (UV)	2	1	0	1
Erythema nodosum leprosum (ENL)	2	0	2	0
Pityriasis lichenoides acuta (PLA)	2	0	0	2
Sweet's syndrome (SS)	1	1	0	0
Total	60	8	11	41

ACV had endothelial cell proliferation, 8 showed extravasated RBCs, 6 had nuclear dust and fibrinoid degeneration, 4 had dilated vessels and one case showed occlusion of lumen with PVNI in 2 (Fig. 1), mixed in 4 and largely PVLI in 3 cases. Out of 7 cases of EN, fibrinoid degeneration and dilated vessles were seen in 5 cases, endothelial cell proliferation and extravasated RBCs in 4 cases, occluded vessels in 2 and nuclear dust in one case with perivascular mixed infiltrate in one case and PVLI in 6 cases. All the 3 cases of PG showed PVLI, endothelial cell proliferation and dilated vessels. All the 3 cases of SP had PVLI, endothelial cell proliferation, occluded vessels, haemosidirin deposition (Fig. 2) and extravasated RBCs in one case. Two cases of FDE had PVLI with endothelial cell profliferation and dilated vessels in 2, occluded vessel and extravasated RBCs in one case only. Both the cases of UV showed fibrinoid degeneration and nuclear dust, endothelial cell

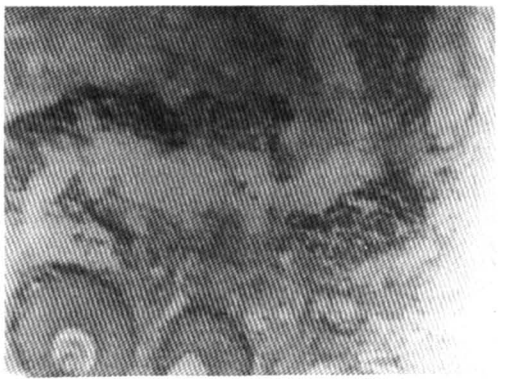

Fig. 1. Leucocytoclastic vasculitis as vessel walls are thickened due to fibrinoid deposits leading to occlusion of lumen associated with perivascular neutrophili infiltrate and nuclear dust.

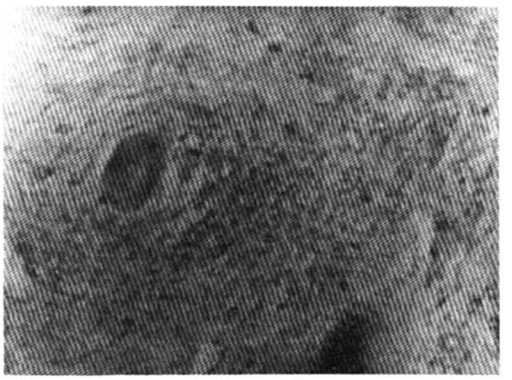

Fig. 2. Occluded dermal vessel due to fibrinoid deposits and multilayering of endothelial cells associated with perivascular lymphohistiocytic infiltrate and haemosidirin deposits seen in lymphocytic vasculitis.

proliferation in 1 case, dilated vessels, occluded vessels and extravasated RBSs in one case wiht PVNI and PVLI in each case. Both the cases of ENL had mixed type of perivascular infiltrate, nuclear dust, fibrinoid degeneration, dilated vessels, extravasated RBCs, and endothelial cell proliferation with occluded vessels in one case. Both the cases of PLA had PVLI with dilated and occluded vessels in 2, fibrinoid degeneration, endothelial proliferation and extravasated RBCs in one case. Solitary case of SS showed nuclear dust, fibrinoid degeneration, endothelial cell proliferation, dilated and occluded vessels and extravasated RBCs with dense PVNI.

Discussion

All cases of EM, HSP, EN, FDE, PLA and SS showed types of vasculitis consistent with the literature. Out of 9 cases of ACV, two showed typical leucocytoclastic vasculitis, 4 had mixed vasculitis with additional infiltration by mononuclears and two late cases had lymphocytic vasculitis. This variation from literature could be as late cases of leuco vas at times change into lym vas when neutrophils decrease and mononuclears infiltrate it or it could be due to overalp seen in vasculitis. This could also be the reason of variance seen in one case of UV. Both cases of EED had lym vas as they were late cases and larger number of mononuclears in comparison to neutrophils could be due to granulation and fibrosis. Some authors do not consider PG in spectrum of vasculitis but our findings are consistent with Su et al[6] as both our cases showed typical lym vas. Both the cases of ENL were induced by antileprosy drugs and showed mixed vasculitis rather than pure leuco vas. As 47/60 (78.3%) cases of CV showed clinicohistopathological correlation, so in the rest, variation could either be due to overlap or due to variation in duration of lesions.

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