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Cell Migration in Patients with Hodgkin's Disease

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Altered function of lymphocytes has been extensively documented in Hodgkin's disease [1, 2], but the significance of neutrophil and monocyte dysfunction is less clear. In 20 patients with Hodgkin's disease (Table I), 7 were found to have markedly reduced neutrophil and/or monocyte migration into Rebuck skin windows. Monocyte or combined defects were most often seen. Six of the 7 defects were in patients with no recent therapy, thus suggesting that the defective migration was related to the under-

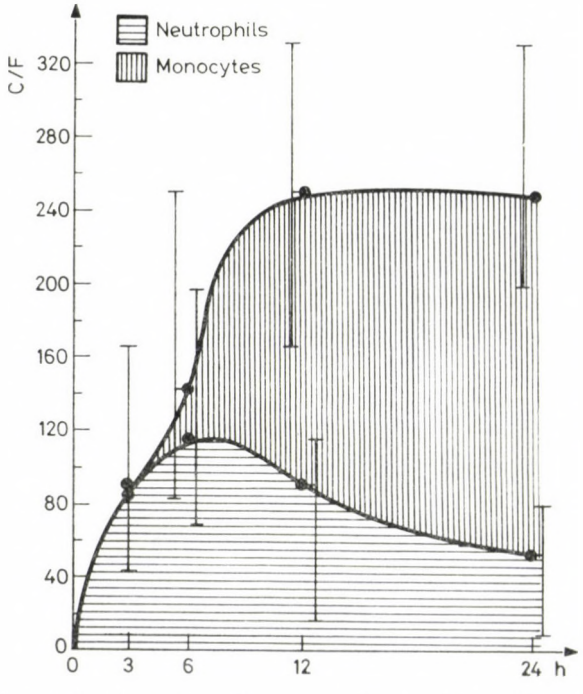

Fig. 1. Rebuck skin window in 6 normals expressed as median cell number and range per 400x field (C/F) as a function of time. 500 cells were counted for each time point

Pat	Subtype	Stage	Sed.rate	Therapy	Skinwindow		Agarose
					neutr	mono	
▲ 1	Mix.	IIA	N	O	↓	↓	O
● 2	NS	IIIB	↑↑↑	I	↓	N	O
3	NS	IIIB	↑↑↑	I	↑	N	O
▲ 4	NS	IIA	↑↑	O	N	N	O
▲ 5	Mix.	IA	N	O	N	↓	O
6	NS	IIIB	↑	O	N	↓	O
● 7	NS	IIIA	↑	I	N	N	O
● 8	NS	IVB	N	Irr.	N	N	O
9	NS	IVB	N	I	N	N	O
▲ 10	Lr	IA	↑	O	N	N	O
● 11	NS	IVA	↑	30	↓	↓	↓
▲ 12	Lr	IIA	↑	O	↑	↑	↑
● 13	NS	IVB	↑↑↑	36	↓	↓	↓
▲ 14	Mix.	IVB	↑↑↑	O	N	N	N
▲ 15	Ld	IIA	↑↑	O	N	N	N
● 16	Mix.	IVA	↑↑↑	48	↑	N	N
▲ 17	NS	IIIA	↑↑	O	N	N	N
▲ 18	Mix.	IA	↑↑	O	↑	N	N
▲ 19	NS	IA	N	O	N	↓	N
▲ 20	Mix.	IIA	↑	O	N	N	N

Hodgkin's disease subtype

Mix. : Mixed
NS : Nodular sclerosis
Lr : Lymphocyte rich
Ld : Lymphocyte depleted
Irr. : Irradiation

▲ : New diagnosis
● : Relapse
O : No previous or present theraphy
x : Last previous theraphy (x month)
I : Therapy interval

TABLE I

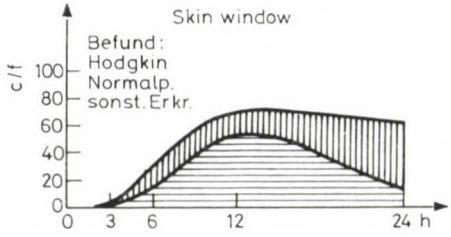

FIG. 2. Rebuck skin window in patient 13. Decreased migration of neutrophils at 3 and 6 hours and of monocytes at 12 and 24 hours

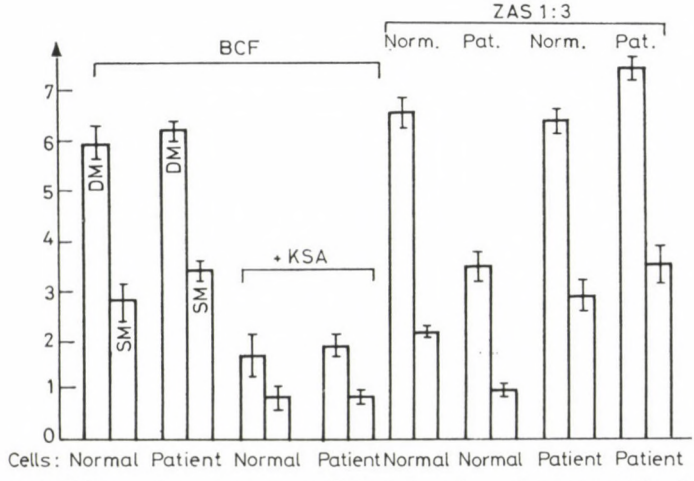

FIG. 3. Directed (DM) and spontaneous (SM) migration of neutrophils from the same patient in vitro (agarose method) Chemotactic factors: BCF — Bacterial chemotactic factor ZAS 1 : 3 — Zymosan activated normal or patient serum diluted 1 : 3 Normal cells showed decreased migration toward zymosan activated patient serum, suggesting the possible presence of an inhibitor. However, patient cells migrated normally. KSA: Control and patient leukocytes were preincubated with Hodgkin's cell line 428 KSA supernatant for 30 minutes, centrifuged and resuspended in RPMI 1640 (control medium). Migration inhibition was observed in both control and patient cells (3)

lying disease. However, decreased monocyte migration was observed in one patient considered to be in remission (patient 6) and normal values were found in several patients with active disease. In 10 of the 20 patients neutrophil migration was tested at the same time.

This study highlights the problems in measuring cell migration in patients (Figs 1–3). Our small control group (n = 6) demonstrated a wide range of cell number in the skin window. Thus, only the most marked defects could possibly be considered as significant. We now perform skin

windows in both controls and patients repeatedly. Cutaneous reactivity to 8 antigens is tested concurrently. The in vitro tests (extended to include monocyte and lymphocyte migration) are performed twice for each corresponding skin window. We hope that these modifications will allow meaningful evaluation of cell migration in patients with lymphoma over the course of their disease.

References

1. Twomey JJ and Rice L: Impact of Hodgkin's Disease Upon the Immune System. Sem Oncol 7: 114, 1980
2. Romagnani S, Ferrini PLR and Ricci M: The Immune Derangement in Hodgkin's Disease. Sem Hematol 22: 41, 1985
3. Schell-Frederick E, Radtke H, Sommer H, Helbing I, Burrichter H, Schaadt M and Diehl V: Inhibition of Human Neutrophil Migration by Supernatants from Hodgkin's Disease Derived Cell Lines. Eur J Clin Invest (in press)