

IMPORTANCE OF THE ROLE OF THE BLOOD TRANSFUSIONS IN DELAYED HYPERSENSITIVITY AMONG END-STAGE RENAL DISEASE PATIENTS

F Louis, P Ruedin, D Frei, M Jeannet, H Favre

Hôpital Cantonal Universitaire, Geneva, Switzerland

Summary

Delayed hypersensitivity was evaluated using a skin multi-test and in vitro lymphocyte stimulation in 15 haemodialysed and in 15 continuous ambulatory peritoneal dialysis (CAPD) patients matched for sex, age and duration of treatment. Their nutritional status (triceps skin fold) and the number of blood transfusions were recorded.

In all end-stage renal disease patients, malnutrition and depressed delayed hypersensitivity were present. The degree of impairment of delayed hypersensitivity correlated with the number of blood transfusions. In CAPD patients, the more depressed delayed hypersensitivity, the greater the incidence of peritonitis. Therefore, limitation of blood transfusions in end-stage renal disease patients is recommended.

Introduction

Uraemia is associated with impairment of the immune system, particularly cellular immunity [1-3] which may account for infectious complications in haemodialysis and peritonitis in CAPD patients.

Among the several causes of delayed hypersensitivity, blood transfusion seems to play a specific role [4,5], while the effect of the nutritional status [6], duration and mode of treatment [7] are not well documented.

The present study examines these four parameters and the degree of inhibition of delayed hypersensitivity as well as its clinical consequences.

Method

Fifteen haemodialysis patients, 15 CAPD patients, and 15 healthy subjects (control group) were matched for sex (7 females, 8 males), age (haemodialysis 52 ± 4 years, CAPD 53 ± 3.5 years, control 52.5 ± 4 years) and duration of treatment (haemodialysis 26.6 ± 4.2 months, CAPD 22.1 ± 4.2 months).

Haemodialysis was performed three times a week (total time of dialysis per week 9 hours) using a cuprophane membrane. Patients on CAPD were dialysed seven days a week (four two litre exchanges a day).

Delayed hypersensitivity with a skin multi-test (Merieux) and in vitro lymphocyte stimulation using the same antigens. Skin multi-test results were expressed as a score in millimetres and in vitro lymphocyte stimulation as the amount of incorporation of tritiated ^3H -thymidine. The average cpm obtained in the control group was referred to as 100 per cent.

In all patients, nutritional status was assessed by triceps skin fold (mm) and muscle upper arm fat area (%) [8]. The blood transfusion history and infection episodes were obtained from the patient's records.

Results

Results of delayed hypersensitivity and nutritional status are shown in Table I. Delayed hypersensitivity is inhibited by 72 per cent in haemodialysis and by 50 per cent in CAPD patients compared to controls, when tested with the skin multi-test. Similar patterns were observed when delayed hypersensitivity was estimated by in vitro lymphocyte stimulation. Nutritional status is reduced in both groups of patients compared to normal by 38 per cent in haemodialysis and by 32 per cent in CAPD when assessed by triceps skin fold. Muscle upper arm fat area provides identical results.

TABLE I. Delayed hypersensitivity and nutritional status in controls, haemodialysis and CAPD patients

Groups	Controls (n=15)	Haemodialysis (n=15)	CAPD (n=15)
Skin multi test (score in mm)	16.5±2.1	4.6±1.3	8±1.5
			p<0.002
		p<0.005	
			p<0.01
In vitro lymphocyte stimulation (%)	98±3	54±4	93±2
			p<0.01
		p<0.005	
			p<0.05
Nutritional status triceps skin fold (mm)	18.5±1.5	11.5±2	12.7±2.5
			NS
			p<0.01
			p<0.01
Muscle upper arm fat area (%)	115±3	69.2±10	63.8±16
			NS
			p<0.05
			p<0.05

TABLE II. Influence of blood transfusions on skin multi-test and number of infectious episodes

	CAPD		Haemodialysis	
	<5 (1±0.5)	>5 (15.6±2)	<5 (2.3±1)	>5 (53±12)
Blood transfusions (number)				
Patients (number)	10	5	5	10
Skin multi-test (mm)	10.04±1.6	2.0 ±1.2	7.5±2.4	2.3±1
	←→ p<0.001		←→ p<0.005	
Peritonitis (month/patient)	0.07±0.04	0.16±0.02	None	None
	←→ p<0.05			
Bronchopneumonia (number)	0	1	1	8

Table II shows the influence of the number of blood transfusions on delayed hypersensitivity and infectious complications. In haemodialysis, 10 patients receiving more than five blood transfusions (mean 53±12 blood transfusions) have an 85 per cent decrease in delayed hypersensitivity and bronchopneumonia occurred in 60 per cent of them. The remaining five patients receiving less than five blood transfusions (mean 2.3±1 blood transfusions) have a 63 per cent inhibition of delayed hypersensitivity and only 20 per cent suffered from infectious complications. Among the five CAPD patients receiving more than five blood transfusions (mean 15.4±2 blood transfusions), delayed hypersensitivity is markedly impaired and the incidence of peritonitis was higher than that observed in the 10 patients receiving less than five blood transfusions (mean 1±0.5 blood transfusions).

Discussion

The present work confirms that in all end-stage renal disease patients, the cellular response is impaired.

Conflicting data exist in the literature which suggests that the alteration of the delayed hypersensitivity in haemodialysed patients may be secondary to the duration of the treatment [7], and mediated by the accumulation of middle molecules which exert possible inhibitory effects on T cells [9].

Our data do not support such a hypothesis as we found no relationship between the duration of the dialysis and the decrease in delayed hypersensitivity. Poor nutritional status is also associated with impaired immune responses in several clinical situations [6]. However, in our patients we found no direct correlation between nutritional status and the degree of delayed hypersensitivity.

A striking difference between CAPD and haemodialysis patients is the number of blood transfusions they receive. In the present study, there is a clear relationship between the number of blood transfusions a patient received and cellular immune function independently of the mode of dialysis.

Thus, among all the factors known to alter delayed hypersensitivity, the number of blood transfusions appears to be the more predominant one when one uraemic patient is compared with another.

There are two possible explanations for the mechanism whereby blood transfusions act on delayed hypersensitivity: 1) blood transfusions can exert an inhibitory effect on E rosette formation because of the presence of haemagglutinins [9]; 2) blood transfusions increase suppressor cell production and the formation of anti-idiotypic antibodies [4,5].

The present study shows that the number of infectious complications which occur in a uraemic patient depends upon his delayed hypersensitivity status. Clinically, these data underline the importance of limiting the number of blood transfusions in end-stage renal disease patients in order to minimize the risk of infectious complications resulting from impaired delayed hypersensitivity.

References

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