narrow lanes in Rohtak unlike Delhi. The sunlight penetrates to almost all the houses. Therefore, children living in Rohtak with abundant sunshine throughout the year are free from rickets even with low vitamin D intake in diet(11).

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Platelet Function Tests in Protein Energy Malnutrition

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Although hemorrhagic manifestations are well described in protein energy malnutrition (PEM), its pathogenesis is not very clear(1). Vascular lesions have been implicated as a causal factor by Bhattacharya *et al.*(2) while coagulation factor abnormalities and presence of disseminated intravascullar coagulation are described by others(3,4). Abnormalities in platelet function and thrombocytopenia are also described(1,5).

In these studies, children with acute infections, mostly severe, have been included. Since the presence of infection can cause hemostatic abnormalities(6), it is difficult to attribute the observed defects to PEM. Therefore, we studied platelet functions in children with PEM who did not have active infection.

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Material and Methods

Thirty malnourished children, between one to five year old, attending Kalawati Saran Children's Hospital, New Delhi were the subjects of the study. Twenty age matched children with normal weight for age formed the control group. Malnourished children were classified into various grades of PEM (IAP classification)(7). A detailed history was obtained and complete general and systemic physical examination was performed. Hemorrhagic manifestations were particularly looked for and Hess tourniquet test was done in all children. Children with acute infections were excluded from the study.

A complete hemogram including estimation of hemoglobin (cynmethomoglobin method), total and differential leucocyte count, erythrocyte sedimentation rate, packed cell volume and examination of peripheral blood smear were done by standard methods. Investigations done in both groups of children to assess platelet functions included platelet count, platelet adhesiveness *in vivo*, platelet aggregation to collagen, platelet aggregation to ADP and platelet factor-3 availability(8-10).

For statistical analysis, Grades I and II PEM cases were grouped together and Grades III and IV PEM cases were included in other group.

Data obtained was tabulated and analyzed using Student's 't' test and Chi-square test.

Results

The age and sex distribution were comparable in study and control group children (mean age 35.6 and 27.3 months, respectively). Six children (20%) had Grade I, 11 (36.7%) had Grade II, 10 (33.3%) had Grade III and 3 (10%) had Grade IV PEM. Five children (16.6%) had edema (Gr. I-1, Gr. II-2, Gr. III-2).

None of the children had any clinical evidence of bleeding and Hess test was negative in all.

As shown in *Table I*, mean platelet count was lower in malnourished children, especially with Grades III and IV PEM, but this difference was not significant ($p > 0.05$). However, thrombocytopenia (defined as platelet count less than 1,50,000/cumm) occurred more frequently in the study group (in severe PEM). In comparison to the control group, 6 cases (20%) in the study group had low platelet count ($p < 0.05$).

Table II shows that the mean value of platelet adhesiveness was significantly

TABLE I—*Platelet Count in Study and Control Group*

Platelet count (thousand/ cu mm)	Control group (n = 20)*	Study group (All cases) (n = 30)**	Grades I & II PEM (n = 17)***	Grade III & IV PEM (n = 13)****	p value
> 150	20 (100)	24 (80)	15 (88)	9 (70)	
< 150	0	6 (20)	2 (12)	4 (30)	*/** <0.05
					*/*** >0.05
					*/**** <0.01
Mean ± S.D.	176.6 ± 23.67	168.1 ± 48.64	179.23 ± 47.37	153.6 ± 47.18	*/*** >0.5
					*/**** >0.5

Figures in parentheses indicate percentages.

TABLE II–*Platelet Function Tests in Malnourished Children*

Platelet functions	Control group (n = 20)*	Grades I & II PEM (n = 17)**	Grades III & IV (n = 130)***	p value	
Mean platelet adhesiveness	40.85	37.18	36.15	*/**	<0.01
(% ± Standard deviation)	(±4.06)	(±2.98)	(±4.82)	*/***	<0.001
Mean platele aggregation to collagen	58.97	57.8	56.3	*/**	>0.5
(% ± Standard deviation)	(±8.77)	(±6.96)	(±12.50)	*/***	>0.5
Abnormal platelet adhesiveness	Nil	Nil	2	*/**	>0.5
				*/***	>0.5
Abnormal platelet aggregation to collagen	Nil	Nil	2	*/**	>0.5
				*/***	>0.5
Abnormal platelet aggregation to ADP	Nil	Nil	2	*/**	>0.5
				*/***	>0.5
Abnormal platelet factor-3 availability	Nil	Nil	3	*/**	>0.5
				*/***	<0.025

lower in malnourished children. No significant difference was observed in mean value of platelet aggregation to collagen in malnourished and control group. Abnormal platelet adhesiveness and abnormal platelet aggregation to collagen and ADP were observed in two children with Grade III PEM. One of these also had abnormal platelet factor-3. In two other children with Grade IV, PEM, abnormal platelet factor-3 availability was observed as an isolated finding. None of the children with abnormal platelet function tests were thrombocytopenic.

Discussion

Of 30 children with PEM, 10 (33.3%) had one or more abnormalities of platelet function or thrombocytopenia. Six (20%) children had thrombocytopenia. Thrombocytopenia has also been observed earlier and in some studies the incidence was higher because only those PEM cases were studied who had clinical evidence of bleeding(1,3,4).

Platelet functions were deranged in four malnourished children with Grades III and IV PEM. Abnormal platelet aggregation to ADP and collagen were observed in 6.6% of malnourished children. A similar incidence (5.5%) of abnormal viscous metamorphosis (aggregation) had been observed earlier in malnourished children with no bleeding. A higher incidence of abnormal viscous metamorphosis was observed in children with purpura(1,5).

Why thrombocytopenia and platelet function abnormalities occur in PEM is not well understood. Bello *et al.* found lactate concentration abnormalities in platelet rich plasma of PEM cases and pointed out that there might be an intrinsic metabolic alteration in platelets in PEM cases or an abnormal microenvironment exists(5).

Bhattacharya and Basu studied the bone marrow aspirates of thrombocytopenic PEM cases and found active

megakaryocytes, thus ruling out megakaryocytic hypoplasia(11). Iron deficiency and megaloblastic anemia have been implicated to cause thrombocytopenia and/or defective platelet functions(12,13). Presence of disseminated intravascular coagulation might explain occurrence of thrombocytopenia in some of the cases(4).

The results of this study show that in PEM even in absence of acute infection, mild thrombocytopenia and some abnormalities of platelet functions exist. Though no clinical evidence of bleeding was noticed, these abnormalities render the PEM cases more prone to bleeding with some other additional insult to hemostatic mechanisms.

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Gastric Teratoma in Children

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Gastric teratoma is a rare benign tumor, found most frequently in boys. World literature yields only 55 reports of gastric

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