Proportion of Rosette-forming T Lymphocytes in Peripheral Blood and Cellular Immunity in 15 Children with Malnutrition

Case No.	Weight as % of Reference Standard for Age	T Lymphocytes (%)		Sensitization to DNCB on Admission	Lymphocyte Stimulation Index in Response to PHA (%)	
		On Admission	After Therapy		On Admission	After Therapy
1	73	36	58	Negative	68	113
2	70	40	69	Positive	49	92
3	65	31	70	Negative	50	95
4	65	22	64	Negative	33	110
5	65	25	68	Positive	50	90
6	60	24	59	Negative	23	87
7	60	15	48	Negative	29	97
8	60	22	68	Negative	11	134
9	58	31	59	Negative	44	88
10	56	20	72	Negative	21	113
11	50	18	62	Negative	40	128
12	50	15	56	Negative	47	87
13	46	19	55	Negative	33	93
14	44	11	34	Negative	5	23
15	40	12	61	Negative	17	110
Mean for patients	57	23	60	2 positive	35	97
Mean (and range) for controls		71 (58-79)		All positive	111 (87-228)	

DNCB = 2, 4-Dinitrochlorobenzene. PHA = Phytohaemagglutinin.

ficient (Wybran and Fudenberg, 1973) and in retardation of intrauterine growth, which is a state of fetal malnutrition of diverse aetiology (Chandra, 1974 a). Together with other indicators of immune defence which are adversely affected by malnutrition (Antia, *et al.*, 1968; Sellmeyer, *et al.*, 1972; Chandra, 1972; Seth and Chandra, 1972; Sirisinha, *et al.*, 1973) the reduction in T cells in the peripheral blood, and presumably in the tissues, may account for the increased susceptibility of patients with these disorders to frequent and severe infections. The reversal of the abnormality on feeding for 6 to 16 weeks rules out any basic primary defect of the thymus. It remains to be seen whether the reduction in T cells is the result of any specific nutrient deficiency, since some cellular functions may be dramatically and severely knocked out by the lack of a single nutrient, such as iron (Chandra, 1973 a).

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MEDICAL MEMORANDA

Epidermolysis in a Case of Severe Cytomegalovirus Infection

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During the last decade an increasing number of publications have shown that cytomegalovirus (C.M.V.) is a widespread infectious agent (Stern and Elek, 1965; Hanshaw, 1966; Krech *et al.*, 1971) producing symptoms of amazing variability. The differential diagnosis is often difficult, especially in patients with other underlying or concomitant disease (Müller-Stamou *et al.*, 1973).

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Case Report

A 40-year-old male nurse complained of fatigue, nausea, and a slight cough at the beginning of January 1972. On 5 January he had severe pain in the left side of the chest, and three days later a maculopapular rash appeared at the level of the 8th thoracic segment, which developed within a few days into a vesicular stage. The diagnosis of herpes zoster thoracalis was made.

On admission on 31 January he had an enlarged spleen (3 cm below costal margin) and several firm, mobile, and slightly tender lymph nodes up to the size of a cherry were found, mainly in the axillae, on the right side of the neck, and in both inguinal regions. The liver was clinically normal. There were no other physical signs. Investigations: E.S.R. 25 to 54 mm/hr; eosinophilia 6%. All other haematological and chemical values were normal (see table).

A systemic lymphoreticular disease was first suspected. Lymph node biopsy showed non-specific inflammation however. A bone marrow examination and lymphography showed no signs of a systemic disease. Serological examinations for mononucleosis, toxoplasmosis, syphilis, brucellosis, and viruses such as ornithosis, adenovirus, Q fever, *Mycoplasma pneumoniae*, and influenza A and B as well as the anti-streptolysin titre gave negative results.

Complement fixing antibodies against C.M.V. rose from 1:40 up to more than 1:120 within two weeks. During this time lymph nodes and spleen regressed considerably and the patient felt much better. The pre-existing herpes zoster also had completely disappeared, and on 17 February he was discharged from hospital. On 29 February, 2 March, and 7 March C.M.V. was isolated from the urine.

On 1 April the patient became ill again with a temperature of 40°C, weakness, poor appetite, nausea, and increasing icterus, with

Clinical and Laboratory Course of Illness

Date:	31/1/72	2/2/72	17/2/72	29/2/72	2/3/72	29/3/72	7/4/72	24/4/72	23/5/72
Lymphadenitis	+++	++	+	—	—	0	0	0	0
Splenomegaly	++	+	(+)	—	—	0	0	0	0
Icterus	0	0	0	—	—	++	++	+	(+)
Epidermolysis	0	0	0	—	—	++	+++	+++	+
Fever	++	(+)	0	—	—	++	+	0	0
E.S.R. (mm/hr)	25-54	—	17-42	—	—	—	77-106	37-68	56/91
Haemoglobin (g/100 ml)	14.2	—	14.2	—	—	11.2	13.3	13.0	12.3
Leucocytes	6,900	—	7,100	(Patient discharged apparently healthy)	—	—	7,200	6,700	7,900
Thrombocytes (× 10 ⁹)	132	—	"Normal"	—	—	—	146	197	126
Bilirubin (mg/100 ml)	0.7	—	0.6	—	—	7.6	7.4	3.2	1.5
Alkaline phosphatase (Bodansky units)	3.1	—	2.0	—	—	—	11.5	9.8	11.8
SGPT (IU)	27	—	18	—	—	—	40	73	64
SGOT (IU)	32	—	15	—	—	—	32	39	33
Complement fixation	—	1/40	1/120	—	—	1/160	1/320	—	—
C.M.V.-specific IgM	—	—	—	0	0	1/20	1/100	—	—
C.M.V.-specific IgG	—	—	—	1/1024	1/512	1/640	1/640	—	—
Virus in urine	—	—	—	3 times positive	—	—	12 times negative once positive	—	—
Virus in lymphocytes	—	—	—	—	—	—	—	—	—

+ = Positive. 0 = Negative. — = Not examined.

dark urine and white stools. On readmission physical examination showed a slightly enlarged liver, spleen normal in size, and no palpable lymph nodes. The most impressive clinical feature was a generalized epidermolysis especially of both hands and feet, which after a month's duration involved also the finger nails and toenails (figs. 1-3).

Investigations this time showed a high sedimentation rate, raised serum levels of bilirubin, alkaline phosphatase, SGPT, and SGOT (see table). Serological examinations for all the above mentioned infections and Australia antigen again gave negative results except for C.M.V. During this second phase C.M.V. was not found in 13 urine samples though it was isolated from the patient's lymphocytes.

On 17 May a liver biopsy was performed. A histological diagnosis of protracted cholestatic hepatitis of the viral type was made. This diagnosis fitted well with the clinical picture and the laboratory findings, which slowly returned to normal after three months.

As there is no established treatment of C.M.V. disease we gave the patient experimental therapy with human interferon during

FIG. 1—Severe epidermolysis of hands.

FIG. 2—Severe epidermolysis of hands.

FIG. 3—Exfoliated pieces of plantar epidermis.

one week of the second phase of the illness (Emödy *et al.*, 1974 a) without convincing benefit, however.

Discussion

Epidermolysis, as far as we know, has not hitherto been described in C.M.V. infection though it is known in other infectious diseases. In our patient there was no clinical or serological findings to suggest the presence of any other infection except the herpes zoster thoracalis, the symptoms of which had completely disappeared before the epidermolysis became manifest. We think our patient had a two-phase course of C.M.V. disease—or rather, a reinfection after apparent clinical recovery with two different clinical patterns: fever, lymphadenopathy, and splenomegaly during the first phase and hepatitis and fever with epidermolysis during the second phase. The fact the C.M.V.-specific IgM was found during the second phase (see table) is an indication that the second phase was caused by a reinfection. The patient excreted C.M.V. in his urine during the first phase of his disease but not during the second, when C.M.V. could be found only in the lymphocytes. An attempt to interpret these two findings will be made later (Emödy *et al.*, 1974 b). We also made similar observations in other patients, including a case of febrile fatal C.M.V. infection (Müller-Stamou *et al.*, 1973).

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