nium in the body. Sodium benzoate and sodium phenylacetate are both given orally in the dose of 0.25 g/kg/day.

An absolute requirement for dietary arginine occurs in these patients, because the urea cycle must be intact for the *de novo* biosynthesis of arginine. Hence, arginine must be supplied in the dose of 0.25 g/kg/day) because it imposes a lower nitrogen burden than arginine and is readily converted in the body to arginine.

Most surviving patients have significant mental retardation(2). The severity of neurologic sequelae is proportional to the magnitude of hyperammonemia and duration of coma(5). Hence, early diagnosis and institution of therapy is a must, not only for the survival, but also for improving the quality of life for these patients.

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Transfusion Associated Malaria in Endemic Areas

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Malaria is a common parasitic infection among pediatric patients in endemic areas(1). When transmitted through blood, post transfusion malaria (PTM) may carry special risk to pediatric patients. It usually delays the diagnosis and complicates an already underlying disease(2). Only limited literature is available on PTM in pediatric patients(3,4). The children who received blood transfusion were followed up vigilantly and any patient developing malarial fever was checked alongwith its blood donor for malarial antigen using monoclonal antibody (MAB) to ascertain the diagnosis.

Material and Methods

All pediatric patients who received blood transfusion (excluding thalassemia

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patients) in the Nehru Hospital of PGIMER, Chandigarh were followed for 12 months. They were followed on 3rd, 10th and 30th post transfusion day for development of fever (>1°C skin temperature). When patients developed fever after 3rd day, they were included in the study and their blood donors were followed and investigated, retrospectively. Each subject was screened for parasitemia by Giemsa(5) and Acridine orange fluorescence staining(6), antimalarial antibody detection by micro ELISA(7) and indirect fluorescence antibody (IFA) test(8) and malarial antigen detection by monoclonal antibodies *i.e.,* Monofluo kit *P. falciparum*. Each malaria positive subject was treated with chloroquine base (10 mg/kg).

Results

During the study period 1312 units of whole blood and packed red cells were transfused to 701 patients, of whom 386 patients were followed once (on 3rd post transfusion day), 280 twice (on 10th day) and 35 for three times (on 30th day). Among them, 14 (2.0%) developed post transfusion pyrexia and 3 (0.4%) patients manifested with PTM. In one of these three patients (No. 2) and in one donor (No. 1), the malarial diagnosis was solely based on positive MAB test where both the staining methods failed to detect parasitemia. There was a delay of 3 days in antigen detection after blood sample collection in patient 2. When the patient was followed for treatment, it was found that he was empirically treated with chloroquine base (10 mg/g) by the pediatrician and the fever subsided. Two PTM positive patients (1 and 2) could not show significantly high antibody level and both of them were below the age of 2 years. Two units of blood were transfused to patient 2 where donor 2(a) could not be traced and donor 2(b) did not show any evidence of malarial infection. This child (No. 2) hailed from a malaria non endemic hilly area of Himachal Pradesh and it was presumed that PTM was from donor 3(a) only (*Table I*). Two PTM positive patients (1 and 3) showed *P. vivax* parasitemia on 4th and 5th post transfusion day, respectively, and the third one (No. 2) developed fever on 10th day of transfusion. Pre-transfusion tested of all patients by MAB test was not possible due to shortage of reagents. In other 11 PTM negative patients and their respective blood did not show any evidence of malaria infection.

Discussion

The diagnosis of malaria infection in young children is difficult because symptoms are not typical(1). It is just spiking and recurrent, and the fever lacks the periodicity(9). The diagnosis of PTM is more difficult due to overlapping of symptoms with the existing disease(2). Keeping in view of difficulties in PTM diagnosis, all post transfused pediatric patients were followed for 1 year and 3 (0.4%) of them showed infection in this study. In one observation, 0.099% PTM was recorded among children(4). However, to the best of our knowledge, this is the first report of its kind where in a systematic follow up of pediatric patients for PTM together with retrospective analysis of infection in blood donors was done simultaneously.

During retrospective follow-up of blood donors, two (donor 1 and 3) were directly found to be responsible for PTM. Both the smear techniques failed to detect parasitemia in donor 1 but the presence of antigen was detected by MAB test and it

TABLE I–*PTM Positive Pediatric Patients and Their Respective Blood Donors*

Tests	Patient 1	Donor 1	Patient 2	Donor 2(a)	Donor 2(b)	Patient 3	Donor 3
Giemsa	+ve	Neg	Neg	N/A	Neg	+ve	N/A*
A O	+ve	Neg	Neg	N/A	Neg	+ve	N/A
IFA	N/S	S	N/S	N/A	N/S**	S***	N/A
ELISA	N/S	S	N/S	N/A	N/S	S	N/A
MAB	+ve	+ve	+ve	N/A	Neg	N/A	N/A
Remarks	*P. vivax*		Species unknown	Donor not traceable		*P. vivax*	H/O, malaria 4 days after donation

*N/A = Not available, **N/S Not significant, ***S = Significant, IFA - Indirect fluorescent antibody test, MAB - Monoclonal antibody, ELISA - Enzyme linked immunosorbent assay, AO - Acridine Orange.

was supported by high level of antibody by IFA and ELISA tests. Donor 3 gave a history of malaria infection after 4 days of blood donation was treated by a practitioner before he was contacted. Among various diagnostic techniques, both the smear examination tests showed limited sensitivity (detect 300-500 parasites/μl) as observed by Ambroise Thomas(10). Though, antimalarial antibody detection techniques (IFA and ELISA) were not helpful in the diagnosis of PTM. Inspite of having malaria infection, two patients (1 and 2) did not show any significant rise of antibody by these tests and both of them were below 2 years of age. Similar results were observed in another study where ELISA was negative in three smear positive children which was difficult to explain(11). On the other hand, MAB test showed direct evidence of infection by detecting malaria antigen and it demonstrated better sensitivity (detect 5-30 parasites/μl) as observed by others(10).

It was observed that 0.4% of pediatric patients developed PTM and only 35 patients could be followed upto one month. None of the patients reported back with fever upto 3 months of post transfusion periods as they were adviced during various follow up visits. It was recommended that in malaria endemic areas, chloroquine therapy should be administered to post transfused patients to prevent PTM(12). When post transfusion pyrexia develop in children and a delay in diagnosis is anticipated, they should be treated with chloroquine therapy which might be curative as in patient 2 of this study. Malaria antibody detection tests should not be used to diagnose PTM in pediatric patients due to immature immune system. MAB test should be employed to detect infection in pediatric patients as well as blood donors to establish the diagnosis.

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Epidemic Hysteria Masquerading as Food Poisoning

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Sirosis(1) reviewed 70 distinct outbreaks of epidemic hysteria from western and European literature during the past century. Of these nearly half (34) occurred in schools, whereas the rest appeared in varied settings and very rarely in hospitals. Women (80%), especially in closed settings mostly below 20 years, often adolescents were involved.

From India also such epidemics have been reported(2,3). But unlike those reported by Sirosis, these have occurred outside the school setting. Chandrashekar(4) reported an epidemic of possession in school setting.

Subjects and Methods

The epidemic occurred on independence day, 1988, in Girls School, Ranikhet (U.P.). Few of the girl participants in the

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