

Clinical and epidemiological investigation of a fatal transfusional malaria case, Olancho, Honduras, April 2006



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INTRODUCTION AND PURPOSE

On April 2006, a 17-month-old girl hospitalized at University Hospital (HE) developed *Plasmodium falciparum* malaria and died. She had been referred from Hospital San Francisco (HSF), Olancho, and hospitalized on March 17 with lower GI tract bleeding and secondary anemia (Hb 4.8 gr/dL). The patient received three blood transfusions (March 16, 17, and April 4). She developed a fever on April 6 and afterwards, *P. falciparum* was identified in the blood smear. An investigation was initiated to characterize the clinical case, identify the transmission source, and conduct prevention and control measures.

METHODS

Clinical case characterization. We use clinical (sign and symptoms) and parasitological parameters (parasite density) to describe the case.

Identification of transmission source. The infection could have originated in the endemic area where the patient resided or could have been acquired through blood transfusions she received at HE or HSF. **Endemic area.** The patient resided in the district of Gualaco (see map). **Active case detection (ACD)** was conducted among family and neighbours. The mother lived in a neighbouring district, San Esteban. **Transfusional malaria.** Blood donors were identified at each hospital. They were interviewed and tested with thick smears and rapid diagnostic test (RDT). Plasma units in both hospitals were tested. ACD was conducted among family and neighbours of the implicated donor and a second blood recipient.



Laboratory methods. Thick smears were stained with 5% Giemsa solution. We used a *P. falciparum* RDT (Paracheck Pf). DNA was extracted from whole blood and plasma units' sediment. Polymerase chain reaction (PCR) using variable molecular markers based on *P. falciparum* Block II *MSP1* gene was performed to compare parasites from the implicated donor and patient.

Prevention and control measures. All patients with asymptomatic and symptomatic infections were treated with chloroquine (25 mg/Kg in 48 hours) and primaquine (0.9 mg/Kg) according to Ministry of Health guidelines.

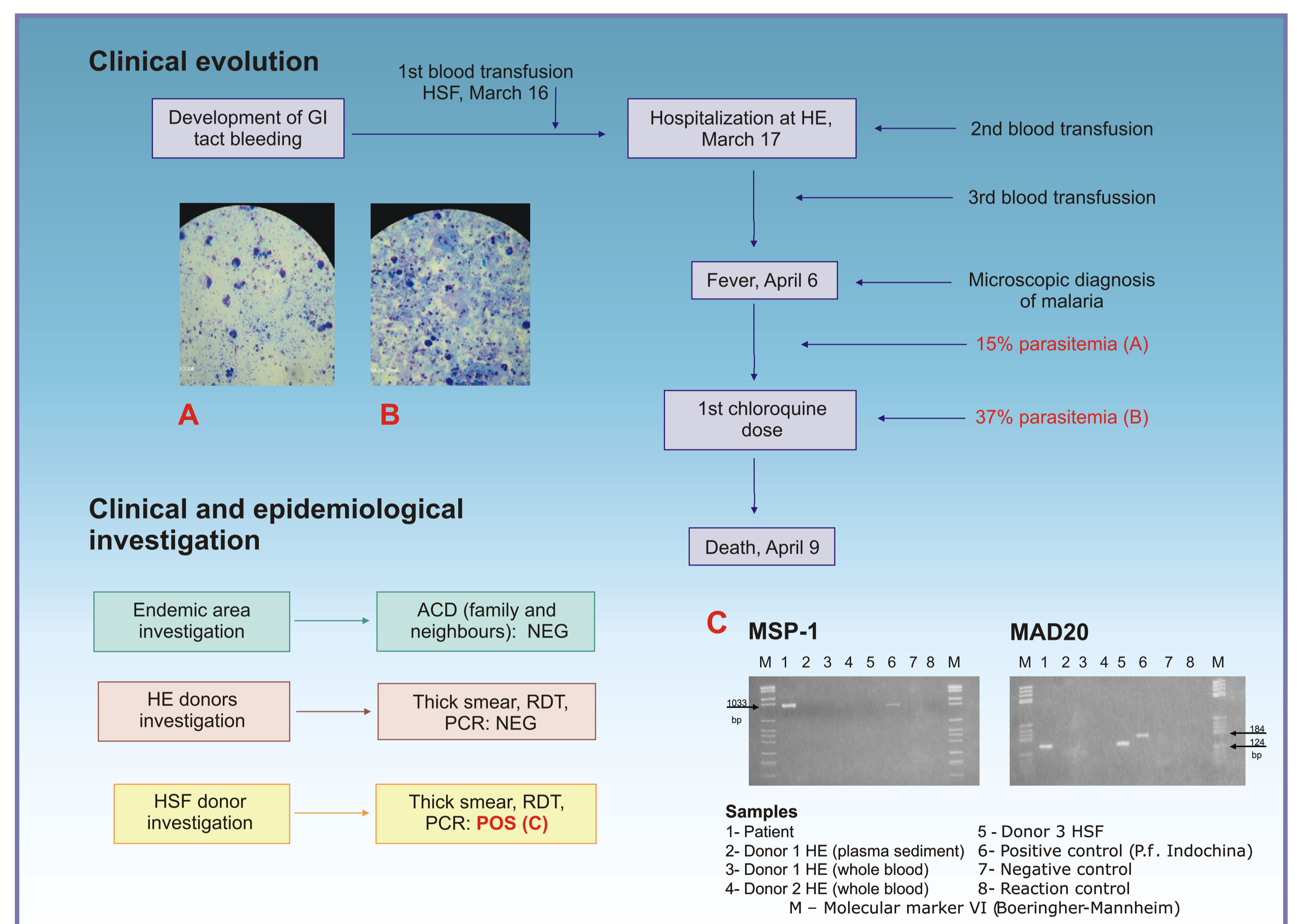
RESULTS

Clinical characterization. This was a case of severe, complicated malaria with hyperparasitemia (see diagram). The patient died on April 9.

Transmission source. Blood smears of family members (n= 7) and neighbours (n= 14) were negative. Two blood donors from HE were negative. An aliquot of plasma from the HSF donor was positive with RDT Paracheck Pf. The donor, a 22 year old man, was asymptomatic but had a positive thick smear with 8 *P. falciparum* gametocytes / 100 immersion oil fields. Among 35 family members and neighbours of the implicated donor, one additional asymptomatic case was detected with 30 *P. falciparum* gametocytes / 100 fields. A second blood recipient was identified, a 3 month old boy who received a blood transfusion on March 14. He was pale and feverish, and was diagnosed with *P. falciparum* ++ plus 5 gametocytes/100 fields. No additional case was found among family and neighbours.

PCR molecular markers. PCR products using MAD20 marker (*P. falciparum* Block II *MSP1* gene) showed a similar pattern between the patient and donor (see diagram).

Clinical and epidemiological investigation of the case



CONCLUSIONS

1. We confirmed two cases of transfusional *P. falciparum* malaria, one of which was severe and fatal. The epidemiological and laboratory evidence demonstrated that the infection was not acquired in the endemic area; it was acquired through a blood transfusion from an asymptomatic donor.
2. We recommend that a thick smear be performed on all blood donors and that parenteral antimalarials be incorporated into the basic drug scheme of the Ministry of Health.



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