

Brief Report

**SALMONELLA SEPSIS CAUSED
BY A PLATELET TRANSFUSION
FROM A DONOR WITH A PET SNAKE**

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PLATELET transfusions carry a serious risk of bacterial sepsis. Platelets are stored at room temperature for no longer than 120 hours (five days), and a single bacterium of the type that typically contaminates platelets collected for transfusion can generate 10^5 organisms in 27 to 108 hours at 22°C.¹ Estimates of the rate of bacterial contamination of platelet products range from 0.04 to 1.0 percent.² The predominant bacterial contaminants are part of the normal flora of the donor's skin, although some are apparently the result of occult bacteremia in the donor.¹⁻³

The estimated frequency of bacterial sepsis among recipients of platelet transfusions is much lower than the rate of contamination, ranging from 1 in 15,000 units of platelets³ to 1 in 100,000 units,² depending on the stringency of the case definition.² These estimates may be low, because many platelet transfusions are given to patients who have acute hematologic cancers and in whom bacterial sepsis may therefore be attributed to neutropenia and immunosuppression alone.^{1,2}

We report the occurrence of sepsis due to infection with *Salmonella enterica* serotype enteritidis infection in two patients, one of whom died. The infection originated in a single platelet donation that had been divided into two units. The donor had regularly donated platelets obtained by apheresis and was apparently healthy; subsequent studies suggested that he had acquired asymptomatic *S. enterica* bacteremia from handling his pet boa constrictor.

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Only two previous reports have documented salmonella sepsis resulting from platelet transfusions; therefore, a platelet transfusion may not be recognized as the source of a salmonella infection. In one report, seven patients were infected with *S. choleraesuis* from platelets donated by an asymptomatic man who was subsequently discovered to have occult salmonella osteomyelitis.⁴ The other report described *S. enterica* serotype Heidelberg sepsis caused by platelets from a donor who had asymptomatic bacteremia five days after having mild gastroenteritis.⁵ We could find no previous references in the literature to the handling of pet reptiles by platelet donors as a source of transfusion-associated salmonella sepsis, but the increasing frequency of pet reptiles as a source of salmonella infections in general^{6,7} suggests that this may be an unrecognized risk.

CASE REPORTS

Donor

The donor was a 47-year-old man who had donated blood products, including platelets obtained by apheresis, 50 times at the Oklahoma Blood Institute, including 12 times in 2001. He donated platelets obtained by apheresis on March 10, 2001, and again on April 7, 2001. At the time of the latter donation, he had no symptoms of an infectious illness, and his platelet count was 361,000 per cubic millimeter. The platelet yield was high, and therefore 2 units of platelets were prepared. One of the platelet units was transfused into a patient in Tulsa on April 11, 2001, and one was transfused into a patient in Oklahoma City on April 12. Currently, in the case of 60 percent of all donors of platelets collected by apheresis at the Oklahoma Blood Institute, the yield is divided into multiple products, and the institute provides blood products to hospitals throughout the state of Oklahoma.

Patient 1

Patient 1, who received the first unit of platelets from the donor, was a 51-year-old woman who was given a diagnosis of acute promyelocytic leukemia on March 20, 2001, and had a complete remission after treatment with all-*trans*-retinoic acid, cytarabine, and idarubicin. On April 11, the day she was scheduled to be discharged, she was asymptomatic, her white-cell count was 200 per cubic millimeter, and her platelet count was 19,000 per cubic millimeter. She received the unit of platelets immediately before her scheduled discharge. During the platelet transfusion, nausea, vomiting, chills, and fever (temperature of up to 40°C) developed. Blood was drawn for culture, and the patient was immediately treated with ceftazidime and vancomycin. Six hours later, lethargy, hypotension, anuria, and respiratory distress developed. The clinical diagnosis was bacterial sepsis related to her neutropenia. On April 13, she required endotracheal intubation and hemodialysis. Gram-negative bacilli were reported in all blood cultures on April 13, and on April 14, the organisms were identified as salmonella that were sensitive to all antibiotics, including cephalosporins. The suspected sources of the salmonella included cholecystitis (she had a history of cholelithiasis), episodes of diarrhea before hospitalization, and a pet cat.

On April 12, the blood bank at the Tulsa hospital was notified about the case of sepsis at the Oklahoma City hospital in the recipient of the other platelet product from the donor, but the platelet transfusion was not recognized as the source of the sepsis until the final culture results from Oklahoma City were reported on

April 17. Dopamine for blood-pressure support was discontinued on April 19. Patient 1 was extubated on April 27, required hemodialysis until May 2, and was discharged on May 14. As of July 2002, she remains in complete remission from her acute promyelocytic leukemia and is working full time.

Patient 2

Patient 2 was a 50-year-old woman who received the other platelet unit from the donor on April 12, one day after Patient 1. She had been admitted to an Oklahoma City hospital that day for severe upper gastrointestinal bleeding and hypotension caused by esophagitis, gastritis, and multiple gastric ulcers. She had chronic thrombocytopenia as a result of cirrhosis and hypersplenism associated with alcoholism and chronic hepatitis C. On admission, her platelet count was 39,000 per cubic millimeter and her liver function was severely impaired. In the emergency room, she received red cells as well as 2 units of platelets, each of which had been obtained by apheresis from a single donor. Within one hour after the platelet transfusions, chills, fever, tachycardia, and respiratory distress occurred, requiring intubation and mechanical ventilation. The patient was also immediately given ceftazidime. Hematemesis recurred, and there was a further drop in blood pressure. Sepsis related to the platelet transfusion was suspected, and Gram's staining of plasma from the retained segment of tubing used to collect blood revealed many gram-negative bacilli.

The Oklahoma Blood Institute called the Tulsa hospital on April 12 to determine whether the other unit of platelets from the donor had been transfused and was told that the unit had been transfused with no adverse reactions. Patient 2 died of refractory septic shock and hemorrhage on April 12. The infected unit of platelets she had received had been stored for five days, as compared with four in the case of Patient 1, which may have accounted for the greater severity of her bacteremia. On April 17 the bacteria from her blood cultures and from cultures of the infused platelets were both identified as *S. enterica* serotype enteritidis that was sensitive to all antibiotics, including cephalosporins.

METHODS

The serotype of the salmonella isolates was determined with the use of standard methods and commercial antiserum for somatic and flagellar antigens (Difco Laboratories).⁸ Molecular subtyping of the strains was done by pulsed-field gel electrophoresis (PFGE) with the use of standard methods for typing salmonella strains (PulseNet, Centers for Disease Control and Prevention).⁹ The similarity of the strains involved was determined with use of the Gel-Compare program (Applied Maths).⁹

RESULTS

The donor was evaluated on April 19, 2001. He reported that he had been ill with a fever, abdominal pain, and diarrhea characterized by the intermittent appearance of bright red blood in his stool beginning on March 20. He had taken amoxicillin and ciprofloxacin for 5 days until his symptoms resolved, 13 days before his April 7 platelet donation. His illness had been attributed to a recurrence of diverticulitis. His daughter had become ill with fever and vomiting seven days earlier, on March 13; she began treatment with amoxicillin on March 19 and was well on March 20. Physical examination of the donor was entirely normal; there were no signs or symptoms to indicate the need for further diagnostic studies. A blood cul-

ture was sterile, and a stool culture demonstrated no pathogenic organisms. To amplify the potential yield of blood cultures and to attempt to reproduce the infected platelet products, the donor was asked to donate platelets by apheresis four times, on May 8, 9, 10, and 22. All 5 units prepared from these four donations remained sterile for seven days during routine storage conditions. During this evaluation it was learned that the donor had a 9-ft (2.75-m) pet boa constrictor. The donor cared for and handled the snake. A stool sample from the boa grew *S. enterica* serotype enteritidis, the same serotype isolated from the platelet product of April 7 and from both patients. The PFGE patterns for the isolates from both patients, the platelet donor, and the snake were indistinguishable (Fig. 1).

DISCUSSION

This report describes the convergence of two important public health issues: platelet-transfusion-associated bacterial sepsis¹⁻³ and salmonellosis associated with a pet reptile.^{6,7} Although the risk of sepsis may be less with single-donor platelet units than with pooled platelet concentrates,³ more efficient apheresis instruments have increased the likelihood that multiple platelet products can be prepared from a single apheresis donor, creating a risk for sepsis in multiple recipients. Prompt recognition of the platelet transfusion as the source of sepsis is essential to prevent sepsis in subsequent recipients of platelets from the same donations.

In the cases we describe, it was initially assumed that in Patient 1, sepsis was related to severe neutropenia. Similar assumptions may be common and responsible for the lack of recognition and underreporting of platelet transfusions as a source of sepsis.^{1,2} The apparent rarity of salmonella as a contaminant of platelet-transfusion products may have contributed to the failure to recognize the platelet transfusion as the source of sepsis in Patient 1. We believe that the platelet donor acquired salmonellosis through handling his pet boa constrictor. The infection caused asymptomatic bacteremia at the time of his donation. He had had an episode of fever and diarrhea 18 days before donation, and these symptoms had resolved after antibiotic treatment for 5 days. The antibiotic treatment may have increased the risk of asymptomatic bacteremia.¹⁰

Many reptiles are colonized with salmonella species, and rates of fecal carriage can be as high as 90 percent.^{6,11} With increased ownership of reptiles as pets, the incidence of reptile-associated salmonellosis is increasing in the United States.^{6,7,12} Direct handling of a reptile is not necessary to acquire salmonella infection⁶; indirect contact (such as living in a household with a colonized reptile but not actually handling it) is sufficient to cause most cases of reptile-associated salmonellosis.¹³ Up to 3 percent of U.S.

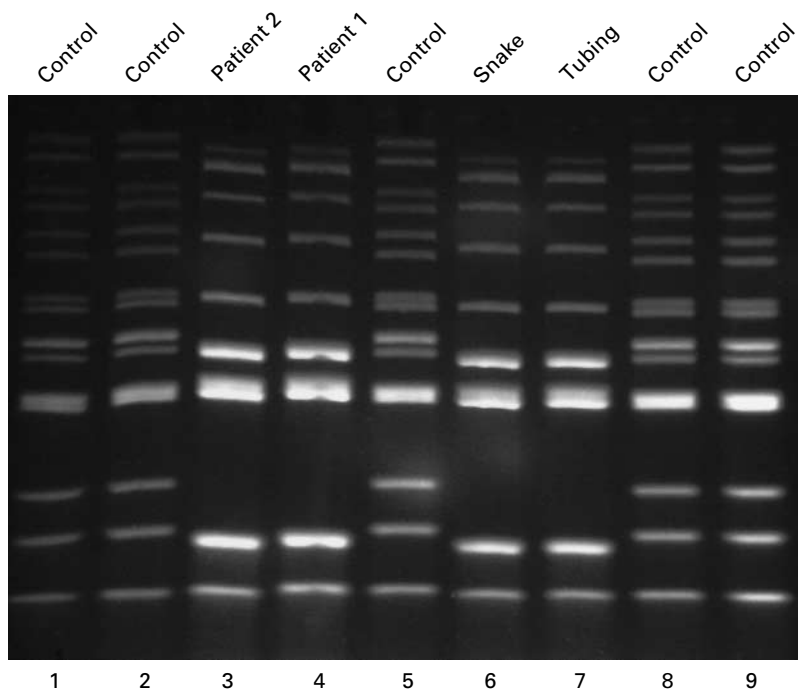


Figure 1. Patterns of Isolates of *Salmonella enterica* Serotype Enteritidis on Pulsed-Field Gel Electrophoresis.

Lanes 1, 2, 5, 8, and 9 show the control salmonella strain am01144. Lane 3 shows the results for the isolate obtained from the culture of blood from Patient 2; lane 4, the isolate obtained from the culture of blood from Patient 1; lane 6, the isolate obtained from the culture of the snake's stool; and lane 7, the isolate obtained from the tubing used to collect blood from the donor. The isolates were digested with *Xba*I endonuclease and subjected to electrophoresis for 18 hours according to the standardized protocol for pulsed-field gel electrophoresis of salmonella isolates (PulseNet, Centers for Disease Control and Prevention).

households have a pet reptile, and these reptiles may account for as many as 3 to 18 percent of the estimated 1.4 million cases of salmonella infections that occur annually in the United States.^{6,7,14} These estimates suggest that reptile-associated salmonellosis could pose an unrecognized risk of contamination of platelet products from apparently healthy donors.

Anticipated changes in transfusion practice will help prevent platelet-transfusion-associated sepsis.² Routine bacterial cultures of platelet products are feasible and could be used to identify contaminated units, since the median storage time for platelet units implicated in cases of sepsis is four days.² Pathogen-inactivation technology is in the advanced stages of development.¹⁵ The value of routine questioning of donors regarding the possession of pet reptiles should be systematically assessed. These cases emphasize that physicians must be aware of the epidemiologic features of salmonellosis, must recognize the potential for asymptomatic salmonella bacteremia among platelet

donors, and must recognize that a contaminated platelet transfusion may be the cause of salmonella sepsis.

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