of hospital admission and at weekly intervals to detect asymptomatic carriage.[10]

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Fatal Outbreak of Polyclonal Candidemia in a Neonatal Intensive Care Unit

To the Editor—Candida species are the third most common cause of late-onset sepsis in neonatal intensive care units (NICUs), accounting for 9%–13% of cases, with an increasing trend compared with previous surveillance studies.[1] The mortality associated with invasive candidiasis (IC) is consistently high, with death rates reaching 40%–50%.[2] The most frequently isolated species is *Candida albicans*, although non-*albicans* species have emerged as important opportunistic pathogens.[3] Early colonization may come from the mother (vertical transmission) or from the environment or hospital staff (horizontal transmission).[4] Risk factors for colonization and posterior infection are prematurity, very low birth weight (VLBW), male sex, vaginal delivery, indwelling catheters, total parenteral nutrition, broad-spectrum antibiotics, ventilatory support, gastrointestinal disorders, previous documented history of bloodstream infection, and abdominal surgery.[2,5,6] Clinical diagnosis is difficult to establish, because IC resembles sepsis syndrome and has a variety of clinical manifestations, such as respiratory insufficiency, feeding intolerance, abdominal distention, temperature instability, and lethargy. Therefore, the definition of IC is a positive culture of a sterile body fluid sample, which can have a sensitivity of only 29%.[3,7]

On January 24, 2013, the State Committee of Hospital Infection Control was notified of the occurrence of 5 newborn deaths of unknown etiology in an NICU of a large public hospital in the Brazilian Amazon within a period of 4 days. On the same day, an investigation was conducted to confirm the outbreak, identify risk factors for infection, identify the microbiological agent involved, and propose recommendations to prevent new infections. Twenty-nine newborns were evaluated in the NICU between December 31, 2012, and March 19, 2013, from admission of the first suspected case patient up to 1 month without the registry of new cases. The NICU consisted of 14 individual nurseries, which, because of the suspected outbreak, had new admissions suspended until laboratory results were available and control measures were in place (a 2-week period). Blood specimens for culture were collected from all newborns and sent to the reference laboratory. Isolates were recovered with a BD Bactec system (Becton Dickinson), and any fungal growth was then cultured on Sabouraud dextrose agar media. Some of the isolates were analyzed by pulsed-field gel electrophoresis (PFGE). We also evaluated all infection control procedures adopted in the unit:

institutional policies for hand hygiene, sterilization's procedures, existence of prophylaxis and therapeutic protocols, and laboratory procedures for collecting and processing of blood cultures. Surveillance cultures were not performed.

During the outbreak, 10 deaths occurred, of which 7 were previous to our arrival at the NICU. Five deaths occurred between January 19 and 22, 2013. Three of these deaths were due to late-onset sepsis (sepsis that developed 72 hours after birth), and the other 2 deaths occurred less than 72 hours after birth. Thirteen blood cultures positive for *Candida* species were identified in 7 infants, 10 of which grew *C. albicans* and 4 of which yielded *Candida tropicalis*. In 2 newborns, both species were detected (Table 1). Four cases were considered to be persistent candidemia (defined as a positive culture after 3 days of therapy). The analysis of 9 *Candida* isolates by PFGE revealed a polyclonal pattern of infection.

An environmental investigation showed an inadequate number of human resources at the NICU. Blood cultures were being sent to a laboratory situated in another state, and between July and December 2012, only 5.1% of cultures were positive, whereas, at the reference laboratory, 50% of the cultures were positive. The hospital had no infection control person or program, and a great lack of materials was observed, particularly with respect to those used in the prevention of bloodstream infection, such as those necessary to the insulation of contact (eg, intravenous catheter, transparent curative, alcohol liquid and gel formulation, apron, mask, and gloves). The results of our intervention were discussed with the hospital staff to improve the infection control measures.

None of the newborns had received antifungal therapy, and only 1 received prophylactic therapy. We started treatment with amphotericin B deoxycholate for patients with cultures positive for *Candida* species and for newborns with a clinical condition suggestive of infection, because studies have shown such treatment to be associated with decreased incidence of disseminated infection and reduced mortality; the duration should be until 21 days after culture clearance.[7,8] Fluconazole prophylaxis was initiated for all newborns with VLBW because of the effect in reducing the incidence for IC infections in this population.[9] Indwelling catheters and other invasive devices were changed and screening examinations, such as total abdomen ultrasonography, echocardiogram, retinoscopy, and liquor analysis, were performed for all newborns, including those with negative blood culture results. Reinforced hand hygiene education and adequate sterilization protocols were also implemented during this period. Despite receiving appropriate treatment, 2 newborns died of IC. After the intervention, only 1 new case of IC was detected among a total of 12 newborns during an 8-week period.

Many outbreaks of candidemia have been documented for which a common source was not identified, but in most cases, outbreaks were controlled after optimization of infection control policies. Although we also did not found a common source of infection, our finding that infections were due to different *Candida* species, from different clones, contributes to the hypothesis of contamination from the environment through horizontal transmission; this hypothesis is also supported by the fact that there were multiple flaws in several aspects of the NICU's infection control measures and by the reduction in infection that occurred after the implementation of new policies. We emphasize that surveillance for IC should be continued to avoid such outbreaks and that environmental infection control measures should be instituted; such measures are especially difficult to implement in resource-limited settings, where cost-oriented policies can lead to improvised hygiene of surfaces with inadequate materials and a reduced number of human resources, which can lead, in turn, to an increased risk of horizontal transmission.

TABLE 1. Neonates with Blood Cultures Positive for *Candida* Species during an Outbreak of Polyclonal Candidemia in a Neonatal Intensive Care Unit

			Blood culture			
Patient	Gestational age, weeks	Risk factors	First	Second	Third	Outcome
1	39	BSA, TPN, SR	*Escherichia coli*	*Candida albicans*	Negative	Survival
2	28	PRE, LBW, BSA, TPN, CVC, SR, VS	Negative	*Candida tropicalis*[a]	*C. tropicalis*	Death
3	32	PRE, BSA, TPN, CVC	*C. albicans*[a]	*C. albicans*[a]	*C. albicans*[a]	Survival
4	28	PRE, VLBW, BSA, TPN, CVC, SR, VS	Negative	*C. albicans*	Not done	Survival
5	33	PRE, LBW, BSA, CVC	CoNS	*C. albcans*	Not done	Death
6	32	PRE, VLBW, BSA, CVC, SR	*C. albicans*[a]	*C. albicans*[a]	*C. tropicalis*,[a] *C. albicans*[a]	Survival
7	32	PRE, VLBW, TPN, CVC	*C. albicans*[a]	*C. tropicalis*[a]	Not done	Survival

NOTE. BSA, broad-spectrum antibiotics; CoNS, coagulase-negative staphylococcus; CVC, central venous catheter; LBW, low birth weight (<2,500 g); PRE, premature birth (<37 weeks gestation); SR, abdominal surgery; TPN, total parenteral nutrition; VLBW, very low birth weight (<1,500 g); VS, ventilatory support.

[a] Analyzed by pulsed-field gel electrophoresis.

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Oral Wounds and Hepatitis B Virus Transmission

To the Editor—Sharing toothbrushes is commonly considered a risk factor for hepatitis B virus (HBV) infection.[1] However, sharing eating utensils and drinking glasses and kissing are dismissed as risks.[1] Does the act of sharing drinking glasses or kissing not carry a viral transmission risk if the persons involved have open oral wounds?

Open wounds are the primary route of HBV infection. Thus far, however, none of the experimental studies that have examined the oral infectivity of HBV in primates have included the creation of oral wounds in their experimental designs. Without the inclusion of oral wounds, the reported animal studies on the oral infectivity of HBV may be compared with tests of HBV infectivity through the skin in the absence of skin wounds.

Similarly, clinical observation studies on oral infectivity have examined the normal situation but not the uncommon situation of having open oral wounds. Investigations of infection sources and contact tracing have been based on commonly recognized infection routes but have not paid attention to whether there were oral wounds present during the suspected infectious stage. Furthermore, there has been no discussion found in the scientific literature on the role of oral wounds in HBV transmission.

Many aspects of epidemiology regarding HBV remain unknown. For example, the identifiable risk factors are unclear for over one-third of patients with acute HBV infection.[2] Although HBV may attach to and infect a few types of human cells (hepatocytes, fibroblasts, peripheral blood mononuclear cells, and the plasma membranes derived from the 2 cell types),[3,4] the human oral mucosa does not contain targets for HBV attachment and infection. We propose that open oral wounds can be a route to mediate HBV transfer while kissing or sharing drinking glasses, eating utensils, or food. This hypothesis explains many of the long-standing questions regarding HBV epidemiology.

Why is there a steep increase in HBV prevalence among children who have not been exposed to any known routes of infection?[5] We explain that children often share drinking glasses, dinnerware, or food with family members, thus increasing the risk of HBV transmission via oral routes.

Why does HBV infection show intrafamilial clustering?[6] We propose that one possible mechanism is that it is more common for family members to share drinking glasses, dinnerware, or foods with each other.

A study involving patients with various types of oral diseases who were admitted to hospitals for surgery showed that hepatitis B surface antigen levels were much higher in patients with benign oral tumors than in patients with 6 other oral diseases (teeth impactions, jaw deformities, oral cancers, oral