An outbreak of extended-spectrum β-lactamase–
producing Escherichia coli in a neonatal care unit began
with transmission from a mother to her newborn twins
during vaginal delivery. Subsequently, infection spread
by healthcare worker contact with other neonates; a healthcare
worker also was infected. Knowledge about transmission
may improve infection control measures.

Gram-negative Enterobacteriaceae expressing extended-spectrum β-lactamase (ESBL) are among the most
multidrug-resistant pathogens in hospitals and are spreading worldwide (1–3). Infections caused by ESBL–producing
organisms have resulted in poor outcomes, reduced rates of clinical and microbiological responses, longer
hospital stays, and greater hospital expenses (4,5). Multiple outbreaks of ESBL-producing Enterobacteriaceae in
intensive care units (ICUs) and increased rates of illness and death, especially in neonatal ICUs, have been reported
(6–10). Physical contact is the most likely mode of transmission. The gastrointestinal tract of colonized or infected
patients is the most frequent reservoir. Several studies indicate that transient carriage of bacteria on the hands of
healthcare workers (HCWs) may lead to transmission to patients (7,11).

We report an outbreak of ESBL-producing Escherichia coli (ESBL E. coli) in a neonatal intermediate care
unit. Initial transmission was from a mother to her newborn twins and subsequently by physical contact of HCWs with
other patients; an HCW also was infected.

The Study

The Department of Obstetrics and Gynecology of
the University Hospital, Basel, Switzerland, has 94 beds;
≈2,000 babies are delivered there each year. The neonatal
unit includes 12 beds for healthy newborns and 9 beds for
infants requiring intermediate care.

A 29-year-old woman with dichorionic twin preg-
nancy was admitted to the antenatal care unit at 32
weeks’ gestation because of spontaneous preterm rupture
of membranes of the first twin. Her medical history was
unremarkable. Screening results for gestational diabetes,
as well as urinary controls and vaginal swabs for group
B Streptococcus, were negative. After confirmation of
preterm rupture of membranes by ultrasound and vaginal
examination, therapy was initiated with amoxicillin/cla-
vulanic acid (3 × 2.2 g/d) for 10 days, tocolysis with bet-
amimetics (hexoprenaline) until 34 weeks’ gestation, and
1 course of steroids for lung maturation (betamethasone
2 × 12 mg with an interval of 24 h).

Five weeks later, the woman spontaneously delivered
2 healthy boys (1,920 g, Apgar scores 9/10/10; and 2,045
g, Apgar scores 8/9/9) under epidural analgesia with place-
ment of a urinary catheter. Two days after delivery, an
asymptomatic urinary tract infection with ESBL E. coli
was detected in the mother; it was treated with trimethop-
rim/sulfamethoxazole for 7 days. Follow-up urinalysis was
negative for ESBL E. coli; however, rectal swab performed
to document colonization was positive for ESBL E. coli.
This pathogen persisted for >7 months after delivery, after
which the patient was lost to follow-up.

Both twins were initially admitted to the neonatal in-
termediate care unit because of their prematurity. Six days
after birth, screening rectal swabs confirmed colonization
with ESBL E. coli in both neonates. The twins did not
show clinical signs of infection and were discharged on
their 20th day.

Screening of the 6 other neonates in the neonatal inter-
termediate care unit during the twins’ stay showed that 3 were
colonized. In addition, rectal screening of 31 HCWs indi-
cated that 2 (7%) were positive for ESBL E. coli. Invasive
infection did not develop in any of the 3 neonates colonized
with ESBL E. coli.

Monthly follow-up screening was performed for the 2
HCWs who were positive for ESBL E. coli. They contin-
ued working after reeducation about general hygiene pre-
cautions. One HCW left her job at the hospital and was lost
to follow-up; the other was negative for ESBL E. coli at
2-month follow-up.

Rectal swab specimens for surveillance of intestinal
 carriage were obtained from all patients in the neonatal in-
termediate care unit during the outbreak and at 2 weeks,
5 months, and 7 months after the outbreak. Screening for
ESBL E. coli carriage among HCWs was performed by ob-
taining rectal swabs.

Cultures were performed by using CHROMagar ori-
entation medium (Becton Dickinson BBL Diagnostics,
Sparks, MD, USA). ESBL production was identified ac-

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According to the guidelines of the Clinical Laboratory Standards Institute (12). Routine susceptibility testing was performed by microbroth dilution (Micronaut-S; Merlin, Bornheim-Hersel, Germany). Four cephalosporins (cephpodoxime, ceftriaxone, ceftazidime, and aztreonam) were used for screening. If $\geq 1$ of the cephalosporins showed increased MICs, ESBL E. coli was confirmed with Etest strips (AB Biodisk, Solna, Sweden) containing cefotaxime or ceftazidime, each with and without clavulanic acid.

Molecular typing was performed by pulsed-field gel electrophoresis (PFGE). ESBL was molecularly confirmed by PCR amplifying genes for TEM, SHV, and CTX-M $\beta$-lactamases. Amplicons were sequenced by using an ABI 3130 Genetic Analyzer (Applied Biosystems, Foster City, CA, USA).

Genotyping by PFGE showed 1 dominant ESBL E. coli strain; 2 different genotypes were found in 1 HCW and in 1 of the screened neonates staying in the same unit as the twins (Figure 1). The outbreak strain was found in the index patient, her twins, 2 neonates staying in the neonatal intermediate care unit at the same time, and 1 HCW (Figure 2). Sequencing of the ESBL gene showed TEM-29 type. Surveillance cultures performed on all patients in the neonatal intermediate care unit indicated no further ESBL E. coli was present at 2 weeks, 5 months, and 7 months after the outbreak.

Before the outbreak, a quaternary ammonium–based disinfectant was used daily to clean the neonatal unit. HCWs routinely cared for healthy babies without using gloves but did use an alcohol-based hand sanitizer. Products for patient care were shared among neonates; in particular, no protective covering was used for clinical thermometers.

After screening showed ESBL E. coli, reinforced infection control strategies were established. A schedule of training sessions emphasizing proper hand hygiene, routine use of protective covering for clinical thermometers, environmental cleaning using an aldehyde-based disinfectant, and routine use of gloves and gowns for any patient contact (particularly changing diapers) was instituted. Furthermore, separate care products were used for each neonate.

**Conclusions**

We report an outbreak caused by transmission of ESBL E. coli from a mother to her newborn twins and subsequent spread to other neonates and 1 HCW. The mother was most likely colonized before hospitalization, and a urinary tract infection developed peripartum. Transmission by contact during vaginal delivery of the twins and transmission by physical contact to 1 of the HCWs and the other neonates was the most likely mode of transmission. We interpret the detection of ESBL E. coli infection in 1 of the neonates and the other HCW as a coincidence because both had a different genotype (TEM-12) and PFGE pattern type of ESBL E. coli.

Because we screened only for ESBL E. coli, we might have underestimated the true extent of the outbreak. However, the ESBL-encoding gene, which is on a plasmid, could have been transferred to other Enterobacteriaceae and would have been missed. Risk factors for colonization in newborns include low birthweight, duration of hospitalization, total parenteral nutrition, previous use of antimicrobial drugs, and mechanical ventilation in a neonatal ICU (13). In the intermediate care setting, breastfeeding was associated with a lower risk for ESBL-producing E. coli in Neonatal Care Unit

![Dendrogram](http://example.com/dendrogram.png)

Figure 1. Molecular typing of extended-spectrum $\beta$-lactamase–producing Escherichia coli isolates by pulsed-field gel electrophoresis. Dendrogram shows a cluster of 6 isolates with identical banding pattern and 2 isolates with 2 distinct patterns.

![Spread diagram](http://example.com/spread-diagram.png)

Figure 2. Spread of extended-spectrum $\beta$-lactamase–producing Escherichia coli outbreak. NICU, neonatal intensive care unit.
Enterobacteriaceae (14) because breastfed neonates have more contact with their mothers and therefore are possibly less frequently handled by HCWs. Our patients had only 1 identified risk factor: the twins from the colonized mother had low birthweight; the other neonates had no risk factors. Improved infection control strategies may be necessary to limit spread of ESBL E. coli in maternity wards because transmission to neonates during delivery is possible. A feasible approach could be to screen mothers whose neonates need to be transferred to ICUs; an outbreak in this setting would be particularly harmful.

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References

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