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To the Editor: The Polio Eradication Campaign was started in
Thailand in 1990, and the last polio case was reported in April 1997.
Although no new cases have been reported, the Polio Eradication
Campaign continues with 4 prevention strategies: high coverage with 3
doses of oral polio in children <1 year of age, acute flaccid paralysis surveill-
ance, acute flaccid paralysis case investigation and response, and
National Immunization Day. Also, the
Ministry of Public Health is prepared
for a national emergency response to polio importation and circulated vac-
cine-derived poliovirus (1).

In April 2003, a case of acute flaccid paralysis was reported from
Phakhao district, Loei province. The patient was an 18-month-old boy with
normal physical development and nutritional status. He had a history of
mild asthma and had received bronchodialator drugs occasionally during
upper respiratory tract infections in the past. The patient had been fully
vaccinated. He had received a total of 5 doses of oral polio vaccine: a dose at
2, 4, and 6 months of age, and 2 doses on National Immunization Day in

On March 27, 2003, while visiting his grandmother in Phoowiang dis-
trict, Khonkaen province (80 km from
his residence), the patient became ill.
Pneumonia was diagnosed; injected
medications were administered into
his left hip once a day for 3 days. The
patient fully recovered.

On April 1, 2003, cellulitis of finger
developed in the patient. The affected
finger was incised and drained, and
oral antimicrobial drugs were adminis-
tered. The inflammation extended to
his elbow but later subsided.

On April 7, fever, cough, and dys-
pnea developed in the patient. Two
days later, the patient’s left leg
became weak. He was admitted to
Phakhao Hospital with a diagnosis of
pneumonia and with weakness in his
left leg. He was later referred to Loei
Provincial Hospital and acute flaccid
paralysis was diagnosed on April 11.
The muscle weakness progressed
until he could not sit.

On April 14, the patient was
referred to Khonkaen Regional
Hospital with weakness in both legs
and arms (grade 0–1). Chest radi-
ograph showed perihilar pneumonia.
Cloxacillin, gentamicin, and
immunoglobulin (Ig) (6 g/day × 4
days, patient weight 12 kg) were
administered intravenously to the
patient. He was discharged on April
30 with a diagnosis of Guillain-Barré
syndrome and bacterial pneumonia.
The muscle tone in his right leg and
both arms was grade 3; however, he
could not move his left leg.

Stool samples were collected on
April 11 and 14 and tested for polio at
the Department of Medical Science
(reference laboratory for polio in
Southeast Asia). Poliovirus type 2 was
isolated in the samples; however, the
results were inconclusive for strain
differentiation. The isolates were sent
to the Centers for Disease Control and
Prevention (CDC), USA, for genetic
sequencing, and the result showed
poliovirus type 2 with 1.6% differ-
ence from Sabin strain poliovirus.
Without evidence of recombination
with other nonpolio enterovirus, the
pattern of genomic change was simi-
lar to the change that occurs in
immunodeficient persons. Immune
system testing of the patient on
August 13 showed IgG = 205.9
mg/dl (normal 800–1,700), IgA <5.5
mg/dl (normal 100–490), IgM <16.8
mg/dl (normal 50–320), and IgE
<18.0 mg/dl (normal 0–100). Anti-
bodies to poliovirus type 1, 2, and 3
were 1:16, 1:32, and 1:8, respectively.
Testing of the follow-up stool samples
showed P1/Sabin on August 10. Test
results were negative on October 13,
and results showed nonpolio enterovirus on November 10 and
December 14.

Before the large-scale outbreak
response immunization was conduct-
ed, 339 serum samples were collected
from children <5 years of age who
lived in the same district as the patient
or in the same subdistrict as his grandmother. Among 153 children who
brought their vaccination records, the
median dose of oral polio vaccine was
7 (range 2–15). All had antibody
>1:8 to poliovirus types 1, 2, and 3.
Approximately 2,000 stool samples
were collected from children <5 year
of age who lived in the same district
as the case-patient or his grandmother. However, after the immunodeficien-
cies vaccine-derived poliovirus was
identified, isolation of the virus was
attempted only from stool samples
from children who lived in the same
subdistrict as the patient. From 223
stool samples, 4 Sabin strain poliovirus and 32 nonpolio enter-
viruses were isolated. In addition, 2
of 18 stool samples collected in July
from close contacts of the case-patient
were positive for Sabin strain poliovirus and negative for vaccine-
derived poliovirus.

The Loei Provincial Health Office
initially did a small-scale response
immunization in 3 adjacent villages
(128 of 129 children) on the day that
the case of acute flaccid paralysis was
reported. Coverage of third dose of
oral polio vaccine in these villages
was 100%. No response immuniza-
tion was conducted at the village in
Khonkaen. On August 8, genetic
sequencing results showed vaccine–derived poliovirus; the decision was made to launch an outbreak response immunization for 175,000 children <5 years of age living in Loei, Khonkaen, and Nongbualampho provinces (visited by the patient from March to August 2003). Two-round campaigns were conducted in August and September. The estimated vaccine coverage was >95%.

Considering the rate of 1% genomic diversity per year and the immunodeficient status of the patient (2), he should have harbored the vaccine strain virus since he received the first dose of routine oral polio vaccine immunization at 2 months of age, and the virus was replicated in his gut. However, why the virus disappeared in subsequent stool specimens is unknown. Circulating vaccine–derived poliovirus is unlikely in this event, as we found no evidence of recombination with other nonpolio enterovirus, high oral polio vaccine coverage in the community, and no vaccine–derived poliovirus in other children.

Although immunoglobulin levels in this case were low but still detectable, whether the patient’s illness was agammaglobulinemia or hypogammaglobulinemia is uncertain. The detected immunoglobulin levels, as well as the antibody level to poliovirus, may be due to intravenous immunoglobulin (IVIG) the patient received while hospitalized 4 months before testing. Since August 2003, the patient has been on IVIG replacement therapy after prolonged and repeated respiratory tract infections.

In retrospect, problems surrounded this event. First, because of several attempts to confirm the result, identification of strain differentiation was delayed. Second, genetic sequencing was delayed because of a communication gap associated with new bioterrorism regulations in the United States during specimen transfer. Third, knowledge of a possible immune deficiency in the previously healthy child was lacking, testing for the patient’s immune status was delayed.

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**Toscana Virus and Acute Meningitis, France**

To the Editor: Sandfly fever (family Bunyaviridae, genus Phlebovirus) have been recognized as etiologic agents of human illnesses in European countries bordering the Mediterranean Sea. These viruses are responsible for rapidly resolving diseases with nonspecific symptoms such as fever and myalgia. However, infection with Toscana virus may involve the central nervous system; severity may range from aseptic meningitis to meningoencephalitis (1). In most cases, illnesses caused by Toscana virus mimic a flulike syndrome with fever, photophobia, headache, red eyes, and stiff neck. Recently, 2 cases of Toscana virus meningoencephalitis in patients with unusual symptoms and life-threatening complications were described in Italy (2). However, sequelae have never been reported.

Toscana virus infection is now epidemic in Italy and Spain (1,3). Furthermore, sporadic cases have been reported in travelers returning from Italy, Spain, Greece, Portugal, and the South of France (4–6). The epidemiology of Toscana virus in France is still unknown. Although infections with this virus have been diagnosed by serologic tests in French patients and in tourists residing in southeastern France, this pathogen has reportedly never been isolated in France (7,8). Here we describe the clinical and biologic features of autochthonous meningitis due to Toscana virus.

On July 9, 2004, a 57-year-old woman who had never left the southeastern coast of France reported malaise and vomiting. On hospital admission, her body temperature was 38.5°C, and clinical examination showed photophobia and stiff neck.