

roads are more navigable than during the rainy season. The epidemic strain then spread in the nonimmune population, which had no cohort immune barrier. All age groups had similar attack rates, in contrast to epidemics within the meningitis belt, which essentially affect children; the death rate in the absence of appropriate treatment was 100%. We showed that in 1999 to 2000 in Yaoundé, a large city situated in the tropical rainforest at about 600 km south of the meningitis belt, *N. meningitidis* was isolated in 13.4% of cases of bacterial meningitis, and most of the strains isolated belonged to serogroup A (3). Serogroup A and W135 meningococcal meningitis increased in Yaoundé between 1995 and 2000, possibly attributable to increases in human exchanges between the northern provinces (situated within the meningitis belt) and the central and southern provinces (6).

Other trigger factors frequently considered responsible for epidemics within the African meningitis belt are drought and the "Harmattan" wind because all major epidemics start at the driest period of the dry season and stop with the first rains. The Harmattan wind rarely reaches South Cameroon. Precipitation has been recorded over a number of years at Fontem Missionary Hospital. From 1995 to 1999, yearly rainfall averaged 2,300–2,500 mm, with only 0–50 mm from November to March. In the past 5 years, an average of no more than two consecutive months have been without rain, whereas almost four consecutive months without rain (December to the end of March) occurred just before the epidemic.

Thus, this outbreak appeared to result from several factors: 1) a virulent serogroup A strain belonging to ST-7 that had been responsible for recent epidemics in surrounding countries and was circulating in Cameroon; 2) the expansion of this strain, favored by the absence of an

immune barrier in the population and by commercial exchanges; and 3) an exceptionally dry season. Outbreaks of meningococcal disease are not strictly bound to certain ecologic conditions occurring within the meningitis belt but may break out elsewhere. Since the epidemic reported here, another meningococcus A epidemic (~200 cases) has occurred at a similar equatorial latitude, near Bamenda (approximately 100 km north of Fontem), in 2001 (J. Kamgno, pers. comm.).

Health authorities should be aware of the possibility of such epidemics, be ready to alert medical practitioners and the public about them as they occur, and ensure that patients receive proper treatment and vaccines in these zones.

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West Nile Virus Meningitis in Patient with Common Variable Immunodeficiency

To the Editor: Infection by West Nile virus (WNV) was first recognized in the Western Hemisphere in 1999 in New York (1). Subsequently, this mosquito-borne flavivirus has spread westward and has emerged as an important cause of infectious meningoencephalitis in the United States (2). In September 2002, during a WNV epidemic in Michigan (2), a 38-year-old woman with common variable immunodeficiency (CVID) sought treatment at the University of Michigan Hospital with acute WNV-associated meningitis. Although persons with CVID are at increased risk for enteroviral meningoencephalitis, a greater susceptibility to arthropod-borne flavivirus infections has not been reported.

The patient had a history of recurrent sino-pulmonary infections and gastrointestinal giardiasis and salmonellosis; at 33 years of age, she was diagnosed with CVID that has been subsequently treated with intravenous immunoglobulin (IVIG) every 3 weeks. She was in her usual state of health until 5 days before admission, when she noted the abrupt onset of severe headache, followed by temperatures up to 39.4°C, progressive photophobia, nausea, vomiting, and a

transient papular rash on her trunk and extremities.

On arrival, the patient reported marked photophobia. Physical examination showed a temperature of 40.6°C, heart rate 80 beats per minute, and blood pressure 122/70 mmHg. She had cervical tenderness to palpation and active range of motion with minimal rigidity. Small, diffuse, non-tender lymphadenopathy was noted in the cervical region. No focal or global deficits were found on neurologic exam. Results of the remainder of the physical examination was unremarkable. Initial laboratory values included a peripheral leukocyte count of 3.5 K/mm³ (normal: 4.0–10.0 K/mm³; 42% neutrophils, 52% lymphocytes, and 7% monocytes), which was unchanged from the patient's baseline leukopenia. Her serum IgG level was 1,081 mg/dL (620–1,520 mg/dL).

Cerebrospinal fluid (CSF) sampling indicated the following: erythrocytes 3/mm³, leukocytes 77/mm³ (41% neutrophils, 51% lymphocytes, 7% histiocytes), glucose 50 mg/dL (50–70 mg/dL), and protein 75 mg/dL (15–45 mg/dL). Results of routine Gram stain, bacterial and fungal cultures, polymerase chain reaction testing for herpes simplex virus and *Cryptococcus neoformans* antigen were negative. Assay results of the patient's CSF for WNV by IgM-capture enzyme-linked immunosorbent assay, performed by the Michigan Department of Community Health, were positive.

The patient was initially treated with parenteral ampicillin, cef-tazidime, and acyclovir, which were discontinued within 48 hours. Her symptoms improved with routine medical support, and she was discharged on hospital day 5. We were notified of the positive CSF IgM for WNV approximately 2 weeks after the patient was discharged, at which time her symptoms had completely resolved. At a follow-up visit 3 weeks after her hospitalization, the patient

had no residual symptoms of meningitis.

Patients with agammaglobulinemia, either common variable or X-linked, are known to be susceptible to recurrent infections (3). Bacterial infections are the best described; however, chronic enteroviral meningoencephalitis is also associated with deficiencies in B-cell immunity (4–6). Although the role of immunoglobulins in host defense against WNV infection is not completely understood, evidence suggests that humoral immunity protects against WNV infection and severe disease (7–9).

WNV is a single-stranded RNA virus of the family *Flaviviridae*. Its genome is processed to eight proteins, including the envelope (E) glycoprotein, the matrix protein, the nucleocapsid protein, and five nonstructural proteins (7). E-glycoprotein antibodies develop during human WNV infection (8), and passive immunization of mice with E-glycoprotein antiserum protects against WNV infection and death (7,8). In addition, IVIG therapy was associated with recovery from WNV meningoencephalitis of an immunosuppressed 70-year-old woman (9). Although our patient had normal levels of serum IgG at the time of illness, viral meningitis may occur in agammaglobulinemic patients despite regular IVIG therapy (10).

This case demonstrates the need to consider WNV in patients with CVID. Our patient recovered promptly, without evidence of neurologic sequelae, despite her underlying immunodeficiency. More experience is needed to provide a better understanding of the relationship between CVID and WNV.

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