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French Polynesian experience with maternofetal Zika virus infection

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JMJ, SF, CG, DEG were in charge of the patients

JMJ, SF wrote the manuscript

ILG performed the PCR on amniotic fluids

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The World Health Organization issued an alert on February 2nd^{1,2} on the potential fetal consequences of the Zika virus outbreak after the Brazilian authorities reported an abnormal increase in the number of cases of neonates born with microcephaly³. Although no causal link could be clearly established, circumstantial evidence was considered worrisome enough for several countries to discourage pregnant women to travel to Central and South America⁴.

French Polynesia was affected by an epidemic of Zika between September 2013 and March 2014 **with an estimated 28,000 patients affected, representing around 11.5% of the Polynesian population**⁵. During 2014, local clinicians were struck by an unusual rate of cerebral congenital anomalies. **Within this territory, approximately 4,000 births are recorded each year.** All cases of suspected fetal abnormalities detected during routine prenatal ultrasound examinations are referred to Papeete Hospital. When the referral examination confirms the presence of fetal cerebral anomalies, neuroimaging examinations (ultrasound and/or MRI) are transmitted to the reference center of Trousseau Hospital in Paris for evaluation by experts in fetal neuroimager. In 2014, 13 cases of fetal cerebral anomalies were diagnosed **instead of four and three in the two previous years. In addition, five neonates are currently under investigation for neurological signs of brainstem dysfunction. As part of the prenatal evaluation of the 13 cases of fetal cerebral anomalies**, amniocentesis was offered to all women for fetal karyotype and CMV detection by PCR, and performed in 10 cases. Following the Brazilian alert on possible fetal cerebral damage secondary to maternal Zika infection, we retrospectively tested 6 available stored amniotic fluid samples **using** PCR. Among those, four were found positive for Zika virus. The fetal brain anomalies depicted in

these cases are presented in the table. Microcephaly (Head Circumference<3th centile) was constant but **was observed after 28 weeks of gestation in two cases. Nevertheless, in case 1, ventriculomegaly was present at the second trimester routine scan. In case 3, the first scan was performed at 29⁺² weeks of gestation.** Severe abnormalities of midline structures and the cerebellum as well as abnormal gyration were observed, which may reflect an early embryologic hit of the developing brain. **Thus, a common maternal viral history during the 1st trimester of pregnancy was reported in three out of four cases.**

Only two observations of severe fetal brain damage were recently reported in the Brazilian population⁶. Our population-based experience should raise awareness regarding the fetal risks of Zika materno-fetal infection. It is likely that the true incidence of severe forms of fetal infection has been underestimated in our Polynesian population as we were unable to test all cases retrospectively.

Considering the potential severity of the fetal lesions, congenital Zika virus infection seems available to ultrasound imaging. Fetal neurosonography should be considered from the 2nd trimester in cases of maternal proven Zika virus infection in order to detect subtle cerebral anomalies that may precede the onset of microcephaly. Since we currently do not know the natural history of Zika virus fetal congenital infection, repeated ultrasound examinations should be considered.

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