dermatoses (n =5). In addition, 1 sample of trichodysplasia spinulosa was included as a positive control.

We performed in situ hybridization by using a custom RNAScope probe (*5*) targeting the complete TSPyV genome on tumor-associated endothelial cells within 25 dermatologic samples representing a range of inflammatory conditions. We used a peptidylprolyl isomerase B RNA probe as a positive control for RNA integrity in all specimens (Figure). We did not detect TSPyV in endothelial cells in any of the 25 inflammatory skin biopsy samples. In contrast, the trichodysplasia spinulosa control had positive staining, and peptidylprolyl isomerase B staining was present in all samples (Table).

We found no evidence of TSPyV infection in endothelial cells in this survey of cutaneous inflammatory conditions, suggesting that TSPyV does not play a role in the cutaneous inflammatory conditions we included. Of note, our cohort is small compared with that of the previous study, which initially led to the identification of TSPyV in 3 cases from 252 specimens (*3*). Therefore, the prevalence of TSPyV infection of endothelial cells might be too low to be detected in a cohort of only 25 cases. Our findings provide evidence that endothelial TSPyV infection is not commonly detected in inflammatory skin biopsy specimens and helps refine the spectrum of tissues and clinical settings in which TSPyV should be considered.

Evaluation and reporting of these cases were performed under research protocols approved by the institutional review board of Stanford University. The patients provided informed consent for the research.

About the Author

Dr. Espinal is a fourth-year anatomic and clinical pathology resident at Stanford University and an incoming gastrointestinal pathology fellow. His research interests include gastrointestinal, hematologic, and microbiologic pathology.

References

1. Lawrence L, Wang A, Charville G, Liu CL, Garofalo A, Alizadeh A, et al. Identification and confirmation via in situ hybridization of Merkel cell polyomavirus in rare cases of posttransplant cutaneous T-cell lymphoma. J Cutan Pathol. 2023;50:835–44. https://doi.org/10.1111/cup.14486
2. Sadeghi M, Aaltonen LM, Hedman L, Chen T, Söderlund-Venermo M, Hedman K. Detection of TS polyomavirus DNA in tonsillar tissues of children and adults: evidence for site of viral latency. J Clin Virol. 2014;59:55–8. https://doi.org/10.1016/j.jcv.2013.11.008
3. van der Meijden E, Horváth B, Nijland M, de Vries K, Rácz EK, Diercks GF, et al. Primary polyomavirus infection, not reactivation, as the cause of trichodysplasia spinulosa in immunocompromised patients. J Infect Dis. 2017;215: 1080–4.4. Borgogna C, Albertini S, Zavattaro E, Veronese F, Peruzzi L, van der Meijden E, et al. Primary trichodysplasia spinulosa polyomavirus infection in a kidney transplant child displaying virus-infected decoy cells in the urine. J Med Virol. 2019;91:1896–900. https://doi.org/10.1002/jmv.25519
5. Lawrence L, Wang A, Charville G, Toland A, Pinsky B, Natkunam Y, et al. Trichodysplasia spinulosa polyomavirus endothelial infection, California, USA. Emerg Infect Dis. 2022;28:1935–7. https://doi.org/10.3201/eid2809.220856

Address for correspondence: Sebastian Fernandez-Pol, Department of Pathology, Stanford University School of Medicine, 300 Pasteur Dr, Rm H2110, Stanford, CA 94305, USA; email: sfernand@stanford.edu

Human Infection with Japanese Encephalitis Virus Genotype V, China, 2025

Wenjing Zou,[1] Yan Peng,[1] Jing Li,[1] Li Cai,[1] Weifeng Tang, Junjing Yang, Xiang Li, Junqiang Xu, Guodong Liang

Author affiliations: Hubei Province Center for Disease Control and Prevention, Wuhan, China (W. Zou, Y. Peng, J. Li, J. Yang, X. Li, J. Xu); Wuhan Center for Disease Control and Prevention, Wuhan (L. Cai, W. Tang); National Key Laboratory of Intelligent Tracking and Forecasting for Infectious Diseases, National Institute for Viral Disease Control and Prevention, Chinese Center for Disease Control and Prevention Chinese Center for Disease Control and Prevention, Beijing, China (G. Liang)

DOI: https://doi.org/10.3201/eid3210.251876

Japanese encephalitis virus genotype V (JEV GV) has caused multiple cases of viral encephalitis in South Korea. We report a human case of JEV GV in China, confirmed by serology, sequencing, and quantitative reverse transcription PCR. We confirm JEV GV infection in China, highlighting the persistent threat of this virus.

Japanese encephalitis virus genotype V (JEV GV) was initially identified in 1952 from viral encephalitis patients in Malaysia and Singapore (*1*). JEV GV was detected again after an ≈60-year hiatus when it was isolated from field-collected *Culex tritaeniorhynchus* mosquitoes in Tibet (Medog County), China,

[1]These authors contributed equally to this article

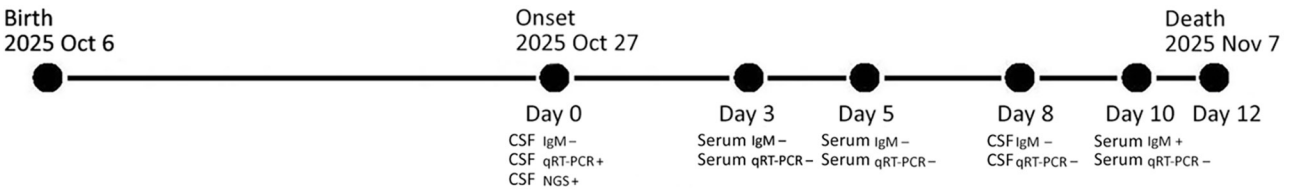

Figure 1. Timeline of a human case of Japanese encephalitis virus genotype V infection in China, 2025. Timeline spans from birth to death, indicating the timing of specimen collection, specimen types, and corresponding laboratory results. CSF, cerebrospinal fluid; IgM, Japanese encephalitis virus IgM detected by capture ELISA; NGS, next-generation sequencing; qRT-PCR, quantitative reverse transcription PCR; +, positive; –, negative.

in 2009 (*2*). Subsequently, JEV GV has been associated with multiple cases of viral encephalitis in South Korea and is now recognized as an emerging pathogen with considerable public health significance (*3*). Despite its reemergence, surveillance data from mainland China after 2009 had detected exclusively JEV genotype I (GI) in both mosquito populations and human clinical samples, suggesting a possible discontinuity in the circulation of JEV GV in this region (*4*). We report a fatal case of JEV GV infection, highlighting the persistent and evolving threat posed by this genotype.

A male neonate was delivered vaginally on October 6, 2025. Both mother and neonate were in good health at discharge. Neither had a recent history of travel to or residence in a JEV-endemic area outside of Wuhan, China. Laboratory testing in the mother indicated no evidence of recent or past JEV infection (IgM- and IgG-negative and JEV quantitative reverse transcription PCR [qRT-PCR]–negative 3 days after illness onset in infant) and she had not been vaccinated against JE. On day 21 after birth, a high fever (39.3°C) developed, and the neonate was admitted to Wuhan Children's Hospital (Wuhan, China). During hospitalization (October 27–November 6, 2025), clinical manifestations were characterized by persistent high fever (37.8–40.0°C), coma, and convulsions. A diagnosis of suspected viral encephalitis was made, and the neonate unfortunately died on November 7, 2025.

We collected 5 specimens for laboratory testing: 2 cerebrospinal fluid (CSF) samples and 3 serum samples. The initial CSF sample, taken at admission (October 27, 2025), tested positive for JEV nucleic acid by qRT-PCR. Laboratory testing detected JEV-specific IgM in the serum sample collected on day 10 of illness. All other specimens were negative for both JEV RNA and IgM (Figure 1) (*5*).

We conducted next-generation sequencing on the CSF sample collected at admission (*6*). We assembled and designated the complete genome of JEV as the neonate strain (GenBank accession no. PZ393248). Phylogenetic analysis on the basis of the open reading frame sequences of JEV genotypes I–V (GI–GV) revealed that GV segregated into 2 distinct clades. One clade exclusively contained the 1952 prototype strains (Muar from Malaysia and Tengah from Singapore), whereas the other comprised all contemporary isolates, including the neonate strain, the XZ0934 strain (Tibet, China, 2009), and viruses obtained from human cases and mosquitoes in South Korea since 2015 (Figure 2, panel A). The positive qRT-PCR result for JEV in the admission CSF sample, together with the sequencing data, confirmed that the viral encephalitis in this neonate was caused by JEV GV. Further phylogenetic analysis of JEV GV envelope gene sequences revealed that all GV strains fell into 3 distinct evolutionary clades: clade A (the 1952 prototype strains), clade B (including the neonate and XZ0934 strains), and clade C (comprising all South Korea isolates) (Figure 2, panel B). Those results demonstrated a clear phylogeographic pattern of JEV GV, reflecting independent evolution of distinct viral populations in mainland China and South Korea after the reemergence of JEV GV in 2009.

JEV GV has been consistently detected in multiple mosquito species in South Korea, where it has replaced GI as the dominant genotype, and human cases have been reported since 2015 (*2*,*7*). In contrast, since the initial isolation of JEV GV from mosquitoes in Tibet, China, no human cases of JEV GV had been reported in mainland China, nor was it reisolated from vector populations. However, a retrospective serologic analysis of laboratory-confirmed JE cases from 2018–2020 identified 2 patients with possible JEV GV infection in eastern China (Shandong and Zhejiang provinces) (*8*). During previous surveillance in Wuhan in 2009–2010, JEV GI was isolated from mosquitoes, whereas GIII had been detected in earlier mosquito collections (*9*). Together with the identification of the neonate strain in Central China and the earlier detection of the XZ0934 strain in Tibet, our findings confirm the established presence of JEV GV in southwestern, central, and eastern China. Of note, in recent years, JE cases caused by JEV GIV

and GV have been increasing, with geographic expansion into Australia and South Korea (*3*,*10*). The detection of this JEV GV case in mainland China, together with those trends, emphasizes the urgent need to strengthen genotype-based detection and surveillance of JEV for effective Japanese encephalitis prevention and control.

This work was supported by Major Projects of the National Nature Science Foundation of China (project no. 81290342)

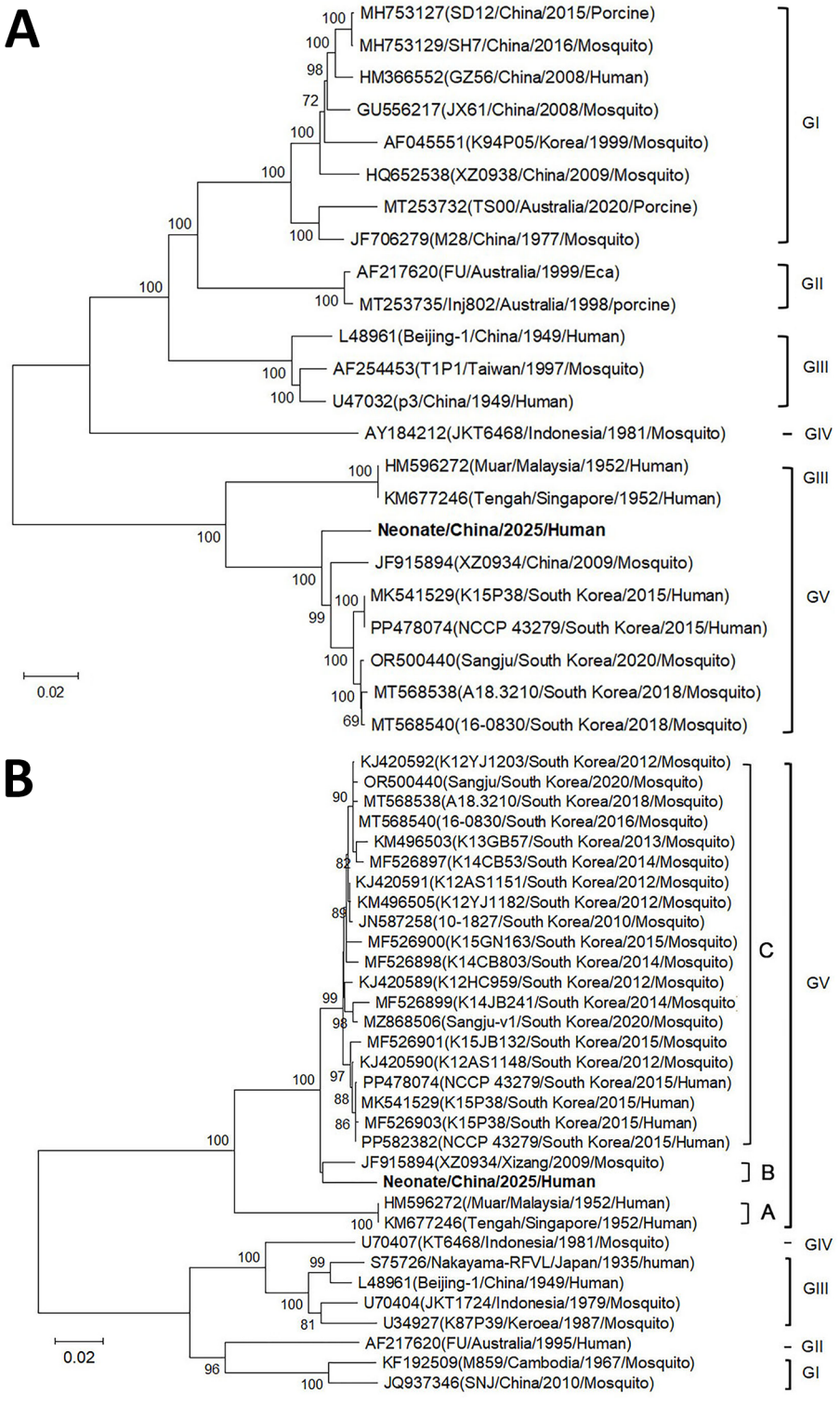

Figure 2. Phylogenetic trees of Japanese encephalitis virus (JEV) genes recovered from a human case in China, 2025. A) Phylogenetic tree built on the basis of the nucleotide sequences of the open reading frame of JEV. B) Phylogenetic tree built on the basis of the nucleotide sequences of the envelope gene of JEV. We constructed the phylogenetic trees by using the neighbor-joining method in MEGA 12.0 software (https://www.megasoftware.net). Neonate sequence from this study is in boldface. Branch numbers indicate percent bootstrap support values. Scale bars indicate nucleotide substitutions per site.

About the Author

Dr. Zou is a professor at the Hubei Provincial Center for Disease Control and Prevention, Wuhan, China, specializing in medical microbiology. Her current research focuses on the detection and diagnosis of mosquitoborne infectious diseases.

References

1. Lee AR, Song JM, Seo SU. Emerging Japanese encephalitis virus genotype V in Republic of Korea. J Microbiol Biotechnol. 2022;32:955–9. https://doi.org/10.4014/jmb.2207.07002
2. Li MH, Fu SH, Chen WX, Wang HY, Guo YH, Liu QY, et al. Genotype V Japanese encephalitis virus is emerging. PLoS Negl Trop Dis. 2011;5:e1231. https://doi.org/10.1371/journal.pntd.0001231
3. Zhang W, Yin Q, Wang H, Liang G. The reemerging and outbreak of genotypes 4 and 5 of Japanese encephalitis virus. Front Cell Infect Microbiol. 2023;13:1292693. https://doi.org/10.3389/fcimb.2023.1292693
4. Liu W, Fu S, Ma X, Chen X, Wu D, Zhou L, et al. An outbreak of Japanese encephalitis caused by genotype Ib Japanese encephalitis virus in China, 2018: a laboratory and field investigation. PLoS Negl Trop Dis. 2020;14:e0008312. https://doi.org/10.1371/journal.pntd.0008312
5. Li W, Feng Y, Zhong H, Jiang M, Zhang J, Lin S, et al. Incongruence between confirmed and suspected clinical cases of Japanese encephalitis virus infection. Front Cell Infect Microbiol. 2024;14:1302314. https://doi.org/10.3389/fcimb.2024.1302314
6. Wang R, Yin Q, Nie K, Li F, Fu S, Cui Q, et al. Is the pathogen spectrum of encephalitis/meningitis changing in China? J Infect. 2025;90:106424. https://doi.org/10.1016/j.jinf.2025.106424
7. Woo JH, Jeong YE, Jo JE, Shim SM, Ryou J, Kim KC, et al. Genetic characterization of Japanese encephalitis virus genotype 5 isolated from patient, South Korea, 2015. Emerg Infect Dis. 2020;26:1002–6. https://doi.org/10.3201/eid2605.190977
8. Zhang WJ, Zhao JR, Yin QK, Liu SH, Wang RC, Fu SH, et al. Serological investigation into the infected genotypes of patients with Japanese encephalitis in the coastal provinces of China. Biomed Environ Sci. 2024;37:716–25.
9. Hu Q, Chen B, Zhu Z, Tian J, Zhou Y, Zhang X, et al. Recurrence of Japanese encephalitis epidemic in Wuhan, China, 2009–2010. PLoS One. 2013;8:e52687. https://doi.org/10.1371/journal.pone.0052687
10. Wang Z, Zhen L, Wei K, Cui B, Wang Z, Farid A, et al. Non-dominant genotypes (GII, GIV and GV) of Japanese encephalitis virus exhibit an elevated evolutionary rate in nature. Microorganisms. 2025;13:2792. https://doi.org/10.3390/microorganisms13122792

Address for correspondence: Junqiang Xu, Hubei Province Center for Disease Control and Prevention, 35 North Zhuodaoquan Rd, Hongshan District, Wuhan 430070, China; email: xjqhbcdc@163.com; or Guodong Liang, National Key Laboratory of Intelligent Tracking and Forecasting for Infectious Diseases, National Institute for Viral Disease Control and Prevention, Chinese Center for Disease Control and Prevention, 155 Chinghai St, Changping District, Beijing 102206, China; email: gdliang@hotmail.com or lianggd@chinacdc.cn

Human Papillomavirus Condylomas of Extragenital Site in Immunocompetent Man, New York, USA, 2025

Sabrina Fong, John G. Zampella

Authors affiliation: New York University Langone Health, Preston Robert Tisch Center for Men's Health, New York, New York, USA

DOI: http://doi.org/10.3201/eid3210.260872

Human papillomavirus types 6 and 11 are associated with mucosal condylomata acuminata, with rare reports of extragenital manifestations. We report a case of condyloma localized to the neck of an immunocompetent heterosexual man, without concurrent anogenital lesions. Our findings challenge theories of the site-specific tropism of these human papillomavirus types.

Human papillomavirus (HPV) types 6 and 11 have long been associated with condylomata acuminata, accounting for 90% of warts affecting the anogenital region (*1*). History of immunosuppression, both primary or secondary, is associated with HPV-related diseases, making affected patients susceptible to development of cutaneous and mucosal warts (2). Reports of extragenital manifestations of condylomas are rare, with previous cases being associated with pediatric populations, men who have sex with men, and persons with a history of HIV. We report a case of HPV types 6 and 11–positive condylomas localized to the lateral neck of an immunocompetent heterosexual man without concurrent anogenital involvement.

In 2025, a heterosexual man in his 40s with an unremarkable medical history sought treatment for a growth on the right side of his neck present for 1 year prior, with reported increase in size. On examination, we noted 6 clustered verrucous-to-mamillated brown papules distributed along the right lateral neck (Figure). Our initial differential diagnosis included inflamed seborrheic keratoses and verruca vulgaris. We treated the lesions with cryotherapy, which provided temporary resolution. The patient returned to our clinic 11 months later for recurrence. We performed a shave biopsy, which showed papillated epidermal hyperplasia with hypergranulosis, compact orthokeratosis, and dilated blood vessels in the papillary dermis. HPV testing by in situ hybridization was positive for HPV types 6 and 11. Screening tests for sexually transmitted infections revealed negative results for gonorrhea, chlamydia, syphilis, and HIV. Complete blood count testing and a comprehensive