3) Misdiagnoses depends on dermatome affected by zoster.

4) Neuritic pain that precedes zoster dermatomes needs to be considered in diagnosis of pathologies with pain syndrome.

doi:10.1016/j.ijid.2010.02.651

84.021

Study on the prevalence of human bocavirus among children with acute respiratory tract infection in Guangdong, China

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Background: To investigate the prevalence of human bocavirus (HBoV) among children with acute respiratory tract infection (ARTI) in Guangdong, China.

Methods: 447 nasopharyngeal aspirates or swabs from children with acute respiratory tract infection in Guangdong were collected from Jun.2007 to May.2008. HBoV capsid protein VP gene fragments were detected by using PCR. Positive PCR products were sequenced. The DNA and the translated amino acid sequences were aligned with known HBoV sequences in GenBank and were done phylogenetic analysis.

Results: 23 (5.1%) specimens were positive for HBoV, among which 43.5% (10/23) were codetected with other respiratory virus. The mainly diagnosis for HBoV positive children were wheezing pneumonia, bronchiolitis and bronchial pneumonia. HBoV positive children ranged from 43days to 6 years old, mainly aged $\leq$1year, among which 43.5% (10/23) were aged 1$\sim$6 months and 39.1% (9/23) were aged 7$\sim$12 months. HBoV were mainly detected in summer, early autumn and late spring. Through sequence alignment and phylogenetic analysis, The DNA and translated amino acid sequences of VP gene fragments of HBoV positive strains showed 97.8$\sim$98.8% and 98.5%$\sim$99.2% identity with ST1, respectively.

Conclusion: HBoV was the important pathogen of ARTI children in Guangdong and was more prevalent in infants $\leq$1year. Although VP gene fragments of HBoV were conservative, there were still some mutant strains leading to amino acid change.

doi:10.1016/j.ijid.2010.02.652

84.022

Deciphering the infectious entry process of human enterovirus 71

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Background: Enterovirus 71 (HEV71) is one of the most clinically significant enteroviruses known to cause severe morbidity and mortality and is most frequently presented as hand, foot and mouth disease (HFMD) in children, although urgent emphasis is therefore being placed on developing anti-viral strategies against this viral pathogen. As yet, little is known of the initial interaction between HEV71 and host cells, which may represent potential anti-viral targeting sites.

Methods: A targeted small-interfering RNA (siRNA) screening platform assay was established and validated to identify and profile key cellular genes involved in processes of endocytosis, cytoskeletal dynamics and endosomal trafficking essential for HEV71 infection. Screen evaluation was conducted via the expression of well-characterised dominant-negative mutants, bioimaging studies (double-labeled immunofluorescence assays, transmission electron microscopy analysis), secondary siRNA-based dosage dependency studies and drug inhibition assays.

Results: The infectious entry of HEV71 into RD cells was shown to be significantly inhibited by siRNAs targeting genes associated with clathrin-mediated endocytosis (CME), such as AP2A1, ARRB1, CLTC, CLTCL1, SYNJ1, ARPC5, PAK1, ROCK1 and WASF1. The functional role of CME was verified by the observation of strong co-localisation between HEV71 particles and clathrin as well as dose-dependent inhibition of HEV71 infection upon siRNA knockdown of CME-associated genes. HEV71 entry by CME was further confirmed via inhibition by dominant-negative EPS15 mutants and treatment of CME drug inhibitors, with more than 80% inhibition observed at 20 μM chlorpromazine. The involvement of other entry pathways, such as caveolae-mediated endocytosis and macropinocytosis, was also found to be minimal, based on the failure of associated drug inhibitors in hampering HEV71 infection. Furthermore, HEV71 infection was shown to be sensitive to the disruption of human genes in regulating early to late endosomal trafficking as well as endosomal acidic pH. The importance and involvement of actin dynamics in mediating the infectious entry of HEV71 was also investigated.

Conclusion: The identification of clathrin-mediated endocytosis as the entry pathway for HEV71 infection of susceptible host cells contributes to a better understanding of HEV71 pathogenesis and enables future development of anti-viral strategies against HEV71 infection.

doi:10.1016/j.ijid.2010.02.653

84.023

The clinical severity of Puumala hantavirus-induced nephropathia epidemica and partial complement protein C4 deficiencies

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Background: Hantaviruses are rodent-and insectivore-borne zoonotic viruses that are found worldwide. Hantaviruses cause two diseases: hemorrhagic fever with renal syndrome (HFRS) in Eurasia, and hantavirus cardiopulmonary syndrome (HCPS) in the Americas. In Finland, Puumala hantavirus causes nephropathia epidemica (NE) that is referred

as a mild form of HFRS with 0.1-0.2% mortality. The pathogenesis of NE is inadequately understood. Previous studies have shown that severe NE is associated with HLA-B8, DR3, DQ2 alleles and this haplotype invariably carries a complement protein C4 null allele. Accordingly, it is speculated that the immune system of the host is involved in the pathogenesis of Puumala virus infection. The role of the complement system in the pathogenesis of NE is poorly studied. The aim of the present study was to evaluate whether the complement protein C4 phenotype associates with the clinical severity of NE. C4 protein, which exists as two isotypes (C4A C4B), is an essential component of the classical complement pathway and deficiencies of C4 are associated with defective processing of immune complexes, impairment of B-cell memory and persistence of bacterial and viral infections.

Methods: Complement protein C4 phenotype was determined in 61 hospitalized patients with NE. Phenotyping was performed by immunofixation electrophoresis. To study the clinical relevance of C4 deficiencies, a number of laboratory parameters and clinical findings reflecting the clinical severity of NE were evaluated with regard to C4 phenotype.

Results: Thirty-eight of 61 (62%) patients had either C4A or C4B null allele. Partial deficiency of C4B was found in 19 patients (31%) and of C4A in 17 patients (28%). No patient had homozygous deficiency of C4A but two patients (3%) had total deficiency of C4B.

Conclusion: The C4 deficiency was not statistically significantly associated with any of the clinical variables measured during acute NE. However, all the 6 patients who had abnormalities in chest X ray, indicating a more severe form of the disease, had C4 null allele (p=0.068, Fisherĭs exact test). This observation indicates a trend that C4 deficiency may be associated with more severe forms of NE although statistical significance is not obtained possibly due to too small sample size.

doi:10.1016/j.ijid.2010.02.654

84.024

Diagnosis of congenital cytomegalovirus infection by detection of viral DNA in urine of neonates in Honduras-Advances

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Background: Human cytomegalovirus (HCMV) is one of the most commonly found agents of congenital infections, with an incidence of 0.5—3% of live births worldwide. Most of the infections are asymptomatic but may be detected from urine positivity for human cytomegalovirus deoxyribonucleic acid in the newborn period. Epidemiology and clinical outcomes are known to vary with socio-economic background, but few data are available from developing countries, where the overall burden of infectious diseases is frequently high. Due to non existing data on congenital HCMV infection in the country, we determined the prevalence of congenital HCMV infection in a defined population in Tegucigalpa, Honduras by urine polymerase chain reaction (PCR) in the babies, and assessed the seroprevalence and activity of the virus in the mothers.

Methods: Urine samples were collected at birth and tested by PCR for the presence of HCMV DNA in newborns; at the same time, mothers' blood samples were tested for the virus by PCR and by ELISA for IgG/IgM antibodies; the IgG avidity test was performed to determined IgG maturation and evolution of infection. Furthermore, antigenemia for CMV pp65 was assessed in the mothers.

Results: Two of the 20 newborns (10%) were found to have HCMV DNA in their urine, consistent with congenital CMV infection. In the mothers, 40% were positive for HCMV DNA. Cytomegalovirus IgG antibodies were detected in 95% of the 20 mothers' samples investigated; 14 (70%) women had both IgG and IgM antibodies at delivery. Two (10%) women presented low avidity IgG antibodies with positive IgM, suggesting primary infection. Among the 20 pregnant women, 50% presented IgM antibodies with a high avidity IgG index, implying recurrent infections. Five percent of the women presented positive pp65 antigenemia.

Conclusion: Although we have not established the prevalence of HCMV congenital infection, these preliminary results permit evaluate the frequency of congenital CMV among the population studied.

By using the urine PCR screening method, we were able to evaluate the magnitude of the problem in a defined population. Therefore, an accurate diagnostic method such as PCR is highly advisable for the management of congenital infection in newborns.

doi:10.1016/j.ijid.2010.02.655

84.025

Epidemiology of human rhinovirus C (HRV-C) in Hong Kong reveals a potential HRV-C subgroup

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Background: A novel human rhinovirus species, HRV-C, was recently identified, but its epidemiology, compared with HRV-A and HRV-B, remains poorly understood, especially in adults.

Methods: One thousand two hundred nasopharyngeal aspirates (NPAs) from hospitalized children and adults during a 12-month period were collected and subjected to HRV detection by reverse-transcriptase polymerase chain reaction. The epidemiology of HRV-A, HRV-B and HRV-C was analyzed.

Results: Of 600 NPAs from children, 178 (29.7%) were positive for HRVs, while of 600 NPAs from adults, 42 (7%) were positive for HRVs. HRV-A showed the highest prevalence (n = 111), followed by HRV-C (n = 91) and HRV-B (n = 18). Although upper respiratory tract infection was the most common presentation in children, 8 (62%) of the 13 adults with HRV-C infection had pneumonia, compared with 6 (27%) of the 22 adults with HRV-A infection ($P < .05$). Wheezing episodes were also more common among individuals with HRV-C (37%) and HRV-A (20%) infections than among those with HRV-B (0%) infection ($P < .05$). Clinical and molecular data analysis indicated HRV-C as a frequent cause of community and institutionalized outbreaks. A possible distinct