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Symposium 4C: Emerging viral diseases

S4C-1. The story of Nipah virus

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Nipah virus, a novel paramyxovirus, belongs to the newly designated genus, *Henipavirus*, under the family *Paramyxoviridae*. The virus made its first appearance in peninsular Malaysia in September 1998, causing an outbreak of fatal encephalitis in humans and severe respiratory illness with encephalitis in pigs. The outbreak failed to be controlled for a period of 6 months despite the institution of very intensive control measures as the outbreak was initially thought to be due to an endemic Japanese encephalitis virus. The successful isolation and identification of this novel virus from cerebrospinal fluid of patients with acute encephalitis (in early March 1999) as the causative agent of the outbreak was pivotal in changing and determining the correct direction of control measures that subsequently brought the outbreak under control. Epidemiological and laboratory investigations demonstrated that infected pigs were the source of human infections and the spread of the outbreak was associated with the movement of pigs from the initial outbreak farms. The transmission of the virus from infected pigs to humans was through close contact with their infected secretions and excretions. Wildlife surveillance studies serologically indicated flying foxes of Pteropid species as the reservoir hosts. Subsequent isolation of Nipah virus from their urine and swab of their partial eaten fruits corroborated the earlier serological finding. Study supported a complex interplay of multi-factorial events that led to the spillover of this novel virus from its reservoir hosts to domestic pigs and ultimately transmitted from infected pigs to humans, dogs and cats.

S4C-2. Avian Influenza

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Abstract not available at time of printing.

S4C-3. Laboratory medicine of SARS

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Severe acute respiratory syndrome (SARS) was the first epidemic of our 21st Century with significant morbidity and mortality. An international outbreak from February to June 2003 affected 8,098 patients in 29 countries with 774 deaths. The aetiological agent was a new coronavirus (SARS-CoV) spread by droplet transmission. Clinical and general laboratory manifestations included fever, chills, rigor, myalgia, malaise, diarrhoea, cough, dyspnoea, pneumonia, lymphopenia, neutrophilia, thrombocytopenia, and elevated serum LD, ALT and CK activities. Treatment has been empirical with initial potent antibiotics cover, followed by simultaneous ribavirin and corticosteroid, with or without pulse methylprednisolone. Postulated disease progression comprised (1) acute viral infection, (2) hyperactive immune response, and (3) recovery or pulmonary destruction and death. The laboratory response in understanding and controlling this emerging infection has been admirably speedy and successful. SARS-CoV was identified with its genome completely sequenced within weeks of the virus's emergence. Follow-up genomic analysis established molecular epidemiological links between clusters, and traced the history of viral evolution. Early and accurate diagnosis of SARS is important - both false positive and false negative diagnoses can be detrimental to patients, families, healthcare workers and the public. Two months into the epidemic in April 2003, a newly developed RT-PCR assay for SARS-CoV had achieved 80% sensitivity and 100% specificity in blood specimens collected on admission facilitating early triage of patients with quantification of viral load bearing prognostic significance. For efforts in chemical pathology and clinical immunology, we investigated serum LD isoenzymes and blood lymphocyte subsets of SARS patients, and found LD1 activity as the best biochemical prognostic indicator for death, while CD3+, CD4+, CD8+ and natural killer cell counts were promising predictors of ICU admission. Plasma cytokine and chemokine profiles showed markedly elevated T-helper lymphocyte type 1 (Th1) cytokine interferon (IFN)- , inflammatory cytokines interleukin (IL)-1 , IL-6 and IL-12, neutrophil chemokine IL-8, monocyte chemoattractant protein-1 (MCP-1), and Th1 chemokine IFN- -inducible protein-10 (IP-10) for at least two weeks after disease onset, characterising a hyperactive host response that could be attenuated with corticosteroid from 5 – 8 days after treatment. Most immune reactions are a two-edged sword. Communicable diseases such as SARS are contagious, but their morbidity and mortality are always due to the associated complication of a **communication disease** caused by the hyperimmune cytokine and chemokine storm. Basic and clinical studies of intercellular communication and intracellular signaling mechanisms are helpful for understanding the pathophysiology of emerging viral diseases and for developing therapeutic strategies.