NEWS IN BRIEF

The Nipah Virus Outbreak

Patient zero in the Nipah virus outbreak in Kozikhode in Kerala died after a rapidly progressive acute febrile encephalitis. Twelve days later his brother was admitted in Kozikhode's Baby Memorial Hospital with high grade fever, altered sensorium, extreme tachycardia, hypertension, hypotonia and areflexia. The CSF sample was sent to the Manipal Centre for Virus Research where the Nipah virus (Ni V) was isolated. This was reconfirmed by RT PCR and serology in the National Institute of Virology, Pune. As of 2nd June, of the 18 cases which tested positive for Nipah virus, 16 are dead. Superb evaluation, isolation and planning by the local doctors and State and National Health machinery have managed the outbreak in exemplary fashion.

Nipah virus is a zoonotic illness transmitted from fruit bats. The virus appears in the saliva of the bats. Half eaten fruits discarded by bats when consumed by humans may transmit the disease. The sap of the date palm when contaminated by fruit bat urine or saliva may also transmit the virus. The WHO strongly suspects, loss of their natural habitat due to human activity results in nutritional stress culminating in increased viral load in the bats saliva.

In humans the incubation period is around 15 days. It may present either as an ARDS (acute respiratory distress syndrome) or acute encephalitis. Some of the peculiarities of this encephalitis are brainstem involvement presenting as opthalmoplegia, pin point pupils, tachycardia, hypertension, postural tremors and segmental myoclonus. Extreme hypotonia and areflexia are also common.

MRI brain may reveal focal subcortical white matter lesions suggestive of micro infarcts. Diagnosis is confirmed by RT PCR or serology in the CSF, urine, nasal or tracheal secretions. Mortality ranges from 40-75%. There is no specific therapy. However in view of the microvascular angiopathy seen on autopsy, aspirin, pentoxyphylline and ribavarin was empirically used in the Malaysian epidemic in 1999.

(*The Hindu 2 June 2018, N Engl J Med. 2000;342:1229-35*)

Tranplanting Memories

Metanoia or radical rethinking is currently transforming the science behind memories. One of the great dogmas in neuroscience is that our memories live in our synapses. Stronger the synaptic connections, stronger the memory. Or so it was thought. Cutting edge research on the lowly snail (*Aplysia californica*) has shown that when snails receive repeated pulses of electric shocks they withdraw their siphon for longer durations than when they are first stimulated.

In experiments, RNA from the snails (which had received repeated electric pulses) was extracted and injected into naïve snails. Amazingly, these snails now started retracting their siphons for prolonged durations. This meant that the memory of electric shocks had now been transferred. The hypothesis was that RNA affected DNA methylation and this is involved in the formation of memories. When DNA methylation was inhibited, transfer of RNA from the shocked snails failed to transfer the memory. It seems that epigenetic changes mediated by RNA on DNA are the key in the formation of memories. More work has shown that blocking epigenetic changes blocks long term memory formation even when synaptic formations remained intact.

What we once thought were nebulous imaginings may just have a biochemical basis. This ground-breaking work may change the way we treat disorders of memory. The human brain has the unique capability of contemplating itself. And now it is attempting to contemplate the very act of contemplation!
(*Scientific American 14 May 2018*)

Screening for Adolescent Depression

The American Academy of Pediatrics has brought out guidelines to screen, identify and tackle initial management of adolescents with depression. One in five teenagers struggle with depression at any one point in adolescence. Only 50% are identified before adulthood. Pediatricians could make a huge difference if trained to identify and manage initial problems.

All adolescents 12 years and above should be screened every year with a formal self report screening tool. Those with risk factors may need more frequent screening. Risk factors include include a previous history or family history of (1) depression, (2) bipolar disorder, (3) suicide-related behaviors, (4) substance use, and (5) other psychiatric illness; (6) significant psychosocial stressors, such as family crises, physical and sexual abuse, neglect, and other trauma history; (7) frequent somatic complaints; as well as (8) foster care and adoption. The AAP recommends the Columbia Depression Scale for initial screening.

Not all teenagers are able to communicate about depression. They may present with non-specific symptoms like irritability, fatigue, insomnia or sleeping more, weight loss or weight gain, decline in academic functioning or family conflict.

Management should include a written action plan like in asthma. Treatment goals may include the establishment of a regular exercise routine, adequate nutrition, and regular meetings to resolve issues at home. In case of severe depression where suicide risk is possible, adequate support from an adult, removal of lethal substances from the surroundings and close follow up is crucial. (*http://pediatrics.aappublications.org/content/141/3/e20174081, http://lphi.org/CMSuploads/Columbia-Depression-Scale-64716.pdf*)

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