Meningoencephalitis in adults

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In this issue of the *JRCPE*, Petchiappan et al.[1] present new data on community-acquired meningoencephalitis from a South Indian centre. In their prospective study they included 50 consecutive patients who had acute meningoencephalitis.

Although the sample size was small, in all cases a causative agent was found. This 100% diagnostic certainty is remarkable, it is likely that the cases were preselected and sampled only a targeted population. The infectious burden is very high in South India and *Mycobacterium tuberculosis* ranks as a major contributor to morbidity and mortality in meningoencephalitis. In the present study about 58% of cases were attributed to this organism.[1] Most cases were picked up by simple techniques, such as cerebrospinal fluid analysis, and polymerase chain reaction (PCR) was used only in some instances.

Previously, in a landmark 1998 epidemiological study, the 'California Encephalitis project' an aetiology could not be found in more than 40% of cases. This project was a large study that sought to determine the clinical and epidemiological features of encephalitis in California.[2] It involved testing for around 13 organisms by PCR, such as herpesviruses (herpes simplex virus 1, herpes simplex virus 2, varicella zoster virus, cytomegalovirus, Epstein-Barr virus and human herpesvirus 6) and enteroviruses as well as serological testing for St Louis encephalitis virus, Western equine encephalitis virus, measles virus, adenoviruses, *Chlamydia* species, *Mycoplasma pneumoniae*, influenza viruses, arboviruses and *Bartonella* species, among others. The vast majority of cases (>60%) were idiopathic and infections accounted only for a minority (13%). Moreover a noninfectious aetiology was identified in 10%.[3]

In the present study, a staggering 74% of cases were caused by bacteria and the overall outcome was very good, possibly reflecting the early pick up and targeted treatment.[1] This is not often the case in most centres in India, where the diagnostic accuracy in central nervous system infections is abysmally low. Although it is tempting to extrapolate the results of this study to clinical practice, in reality, even with multiplex PCR panels, it is difficult to achieve diagnostic certainty in >30–40% of cases. Certainly, one must acknowledge the role of noninfectious causes and recognise that we still do not have answers to a substantial proportion of cases afflicted with meningoencephalitis. The list of even infectious organisms causing this syndrome is increasing in leaps and bounds and currently hovers at around 100 different pathogens. The advances in neuroimmunology and the detection of newer antibodies and new syndromes have led to a further increase in the number of potential causative agents.[4] In the past, many chronic meningitis patients where standard testing (Gram stain, acid-fast bacilli stain, automated cultures, fungal cultures, etc.) did not yield any pathogens were started on anti-tuberculous therapy empirically given the high prevalence rate of tuberculosis. However, this is now unacceptable and nowadays more extensive testing is undertaken before any such empirical therapy. Seasonality, epidemiological factors, exposure to zoonoses, mosquito bites and other factors must be taken into consideration. In recent years, numerous epidemics of an acute encephalitic syndrome (AES) of unknown aetiology have swept through various parts of India. Some of the outbreaks included Japanese encephalitis, Dengue and Nipah encephalitis. In a 6-year period (2008–14), more than 44,000 cases of encephalitis and more than 6,000 deaths were reported, particularly in the some of the most populous states of North India (Uttar Pradesh and Bihar). The vast majority have undergone extensive testing without any results.[5] In 2016, an epidemic of encephalitis swept through Uttar Pradesh resulting in over 120 deaths. Again no aetiology was detected despite extensive evaluation involving the Center for Disease Control, USA. A toxin was purported to be the cause of this AES as cerebrospinal fluid testing did not show any inflammatory response.

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Currently the situation in AES epidemics in India remains appalling with a high rate of nondiagnosis and unacceptable mortality rate in contrast to the situation in the West, where the mortality of AES is <5%.[2]

Clearly, more work needs to be carried out on this front to elucidate the mechanisms behind this devastating illness. Petchiappan et al.[1] have fired a salvo across this yawning chasm. This should spur more clinicians to analyse and interpret their data in the Indian context.

References

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