

## Diagnosis of cerebral toxoplasmosis

Sir,

We appreciate that the readers have made some interesting observations regarding our study.<sup>[1]</sup> We would like to offer the following in response.

- The negative controls were from cases of road traffic accident (RTA) victims who were tested for toxoplasmosis.<sup>[2]</sup> These cases on testing were found to be negative for toxoplasmosis—it was not a deliberate selection. The cerebrospinal fluid samples from RTA cases are not collected at autopsy as the sample is usually contaminated with blood and will not be an ideal sample, to test. Similarly, in the voluntary blood donors where we reported 20.3% positivity<sup>[3]</sup> (and not 11% as indicated by Wadia *et al.*)<sup>[1]</sup>: it just reflects seroprevalence of the infection in the general population and not disease manifestation. This also can be considered a limitation. Given these constraints, in the samples we have tested, we have reported our observations.

The readers had two issues:

- One is regarding the high sensitivity of the tests that we have reported. However, we wish to emphasize that not mere positivity of serum IgG but the high serum IgG titers is of significance in the clinical setting. The serum IgG titers in the patients of group I (cases of confirmed central nervous system [CNS] toxoplasmosis) ranged from 196 to 1016 IU/mL [Table 2].<sup>[2]</sup> We do agree that the mean serum IgG titers in our study were very high (646 IU/mL) since they were all terminally ill cases; however, a serum IgG titer of >200 IU/mL is considered positive in recrudescence cases indicating active disease and is useful in the antemortem diagnosis of CNS toxoplasmosis.<sup>[4]</sup> Our findings are in agreement with this observation indicated in literature.<sup>[4]</sup> Also, as is evident from our IgM and IgG avidity results in group I, except for one case of primary toxoplasmosis (Table 2: ID 00/TB/C130, which had both IgM and low avidity IgG antibodies), all the other cases were confirmed to be cases of recrudescence (with high avidity anti-*Toxoplasma gondii*-specific IgG), although this has not been reiterated in our article.<sup>[2]</sup> A study from Mumbai in HIV-positive patients with a clinical diagnosis of cerebral toxoplasmosis also showed a mean serum IgG titer of 250 IU/mL.<sup>[5]</sup>

Lumbar puncture is often contraindicated in the clinical setting of cerebral toxoplasmosis with multiple

intracranial lesions with perilesional edema. In this context, a *T. gondii*-specific serum IgG titer of >200 IU/mL could be considered a useful marker to clinch the diagnosis of toxoplasmosis in patients and could be considered as strong supportive evidence to initiate the treatment since it indicates active disease and helps in differentiating from mycobacterial pathology. Considering all these points we suggested the usefulness of serology to identify the cases of CNS toxoplasmosis.

- Secondly, with regard to the high specificity: We chose a known negative group (RTA) for assessing the specificity of a test as is the norm. To further establish specificity, we have selectively chosen samples from autopsy confirmed cases of tuberculous meningitis without histological evidence of toxoplasmosis to serve as disease controls. This aspect has been discussed in the manuscript.<sup>[2]</sup>
- Early cases of cerebral toxoplasmosis are often not recognized as they remain subclinical. We used the term cerebral toxoplasmosis to denote patients with clinical manifestations of an encephalitic process of variable degree. The duration of clinical illness in our sample varied from 3 days to 16 weeks.

Seropositivity for toxoplasmosis of >200 IU/mL in serum is of great help in cases where lumbar puncture is contraindicated and where polymerase chain reaction is not freely available as in resource restricted settings. Wadia *et al* have referred to their study with 67% positivity in “normal” people (presumably HIV negative but with no neurologic disorder) attending Ruby Hall clinic/neurological services.<sup>[1,6]</sup> The very high seroprevalence rates quoted by Wadia *et al* may not reflect seroprevalence in general population who constitute voluntary blood donors. It therefore becomes essential to acquire seroprevalence data in healthy general population from different geographic locales in India.

**Sreenivas Adurthi, Anita Mahadevan<sup>1</sup>, Radhika Bantwal, Parthasarthy Satishchandra<sup>2</sup>, Sujay Ramprasad<sup>3</sup>, Hema Sridhar, S.K. Shankar<sup>1</sup>, Avindra Nath<sup>4</sup>, R.S. Jayshree**

Departments of Microbiology, Kidwai Memorial Institute of Oncology, <sup>1</sup>Neuropathology and <sup>2</sup>Neurology, National Institute of Mental Health and Neurosciences, <sup>3</sup>Anand

Institute of Laboratory Medicine, Bangalore, India,  
 4Neurology, Johns Hopkins University, Baltimore, Maryland,  
 USA

**For correspondence:**

**Dr. Jayshree R.S.**, Department of Microbiology, Kidwai  
 Memorial Institute of Oncology, Bangalore - 560 029, India.  
 E-mail: microjayshree@gmail.com

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