1. Authors defined the cut-off for 'inadequate iodized salt', and 'insufficient urinary iodine excretion (UIE)'. But further cutoffs for defining the severity as well as toxicity levels are not provided. Their description in methodology will be an ease for readers. Also, UIE <200 µg/L was considered "insufficient" by the authors, whereas WHO as well as NIDCP uses UIE <100 µg/L for defining the same [2-4]. Using a different cut-off will change the prevalence and its public health implications.
2. The median UIE level of the population was 175 µg/L, which signifies 'adequate iodine nutrition' in the population [2,4]. The results of individual patient/subgroup should not be used for drawing the conclusion as the results of spot sample may vary significantly among different specimens from the same individual [4].
3. As per WHO, if the median UIE levels of a population are 'insufficient' the level of iodization of salt, along with factors affecting the utilization of iodized salt (production level quality, packaging, and transport methods, salt intake and cooking habits) should be reassessed [4]. In this study, all households were using packed salt but there is no mention whether it was iodized or not. Also, 432 (80%) out of 540 samples were inadequately iodized at the consumer level. If these levels are despite using iodized salt, it raises serious concern at the level of iodization at production, packing, transport and storage level, and warrants urgent administrative action.
4. A majority (80%) of the population was using inadequately iodized salt, but 36% of children had UIE in toxic level. How can this finding be explained?
5. The authors used only semi-quantitative rapid test kits for iodine estimation of the salt. WHO recommends using quantitative titration method for iodine analysis in sub-sample of salt that has been analyzed by rapid kit [4].

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Editor's note: We did not receive a point-by-point reply to any of these two letters from authors of the study, despite reminders.

Resurgence of Chikungunya: A New Threat to Public Health

We read the recent article by Maria, *et al*. [1] and appreciate them for highlighting this underreported/underdiagnosed condition. Dr Jacob John, in an accompanying editorial [2], has highlighted the public health importance and need for urgent action. We would like to highlight certain points, which might bring more clarity on this issue:.

1. Authors did not mention the gestational age of the study subjects. It is important to ascertain gestation before diagnosing neonatal encephalopathy as the recently proposed definition [3] is applicable for neonates born at or above 35 weeks. Also, the case definition of neonatal encephalopathy is not clearly stated in this paper.
2. The authors did not provide any reference for defining hypoglycorrhachia, increased protein and pleo-cytosis in cerebrospinal fluid. The cut-offs used are quite different from those proposed by National Neonatology Forum [4], and again are dependent upon the gestational age of the neonate. These arbitrary cut-offs may lead to bias in diagnosing meningitis..
3. This study highlights the neurotropism of

Chikungunya infection, which again reminds of the Zika virus. Traditionally Chikungunya virus is thought to be mild neurotropic but current series contradict this hypothesis as all were having neurological features at presentation and two-thirds had persistent radiological findings. As highlighted by Dr. John in his editorial [2], both Chikungunya and Zika virus are transmitted by the same mosquito; and many times co-exists. Also, their coexistence may increase the virulence of each other. A recently published study showed that there is a strong significant correlation between the distribution of infection-related microcephaly and Chikungunya infection rate [5]. Therefore, it may be worthwhile to re-examine the birth/admission head circumference of the enrolled neonates for assessment of microcephaly and to look for an association, if any. Although, the number is small but still it can point towards a new hypothesis and may help in understanding the relation between microcephaly, poverty and co-infection of Zika, Chikungunya and other arboviruses.

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AUTHORS' REPLY

1. All neonates in the case series [1] were of gestation between 36 to 38 weeks. The definition of neonatal encephalopathy (mentioned in Table I of our article) in our series matched that given by American Academy of Pediatrics [2].
2. The cut-offs of biochemical tests were not arbitrary but taken from Avery's textbook of Neonatology 7th edition (also indicated in Table I of our article). National Neonatology Forum (NNF) criteria would have led to underdiagnosis and missing out of seven cases. Also, NNF definition states that "there is no safe cut-off at which one can recommend do not treat. Clinical judgment would have to be used." This highlights the limitations in deciding on any set cut-off values to diagnose hypoglycorrhachia, pleocytosis and increased protein.
3. We thank the authors for pointing out an important observation. We had in fact mentioned a comparison of Chikungunya virus with Zika virus while writing this paper but the details had to be edited out due to word limit. The Centers for Disease Control and Prevention (CDC) case definition of congenital Zika virus disease prominently includes microcephaly as its first clinical criterion apart from others [3]. There is a strong association of microcephaly at birth with Zika virus infection *in utero*, especially if contacted in first trimester of pregnancy [4]. None of the babies in our study had microcephaly at birth. Our cases presented during a Chikungunya outbreak in the city. No transmission of Zika virus was reported during the same period as per government reports [5]. We agree to the proposition that provision for a panel of tests for arboviral infections, including Zika virus and Chikungunya virus, should be in place for neonates suspected to be having co-infection.

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