

Partial Glossectomy for Lingual Edema following Injury

A 4-year-old female child was admitted with persistently protruberant swollen tongue following lingual injury sustained by four unprotected epileptic fits in the 12 hours preceding the admission. Attempts to reposition the tongue by the parents/patient had failed. On examination the tongue was edematous, swollen, ulcerated and bleeding. Antiepileptic treatment was started. The child was fed through the nasogastric tube. A part of the tongue became gangrenous and the rest of it was edematous and protruberant even after ten days of supportive treatment. A partial glossectomy removing about onethird of the anterolateral aspect of the tongue was carried out. Postoperatively, the tongue was reducible and the child could take oral feeds and talk normally.

Lingual injury during a generalized

seizure is a frequent occurrence(1). Usually, the injuries are simple and heal very soon. However, rarely severe lingual injuries can be sustained. Severe lingual edema may necessitate nasogastric feeding and occasionally partial glossectomy, as in the present case. In severe cases care must be given to maintain the adequacy of airway and tracheostomy may be needed(2).

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Artemether in Children with Severe Malaria

I read with interest the recent article by Huda, *et al.*(1). Based on an open randomized trial, they found artemether to be a good alternative to quinine for severe *Plasmodium falciparum* malaria. However, I would like to make certain observations.

In previous studies done on male Rhesus monkeys, artemether and other related compounds were found to cause significant brain injury and Petras, *et al.*(2)

found neuropathological lesions in medullary precerebellar nuclei after treatment. A longer duration of follow-up and a larger sample size in this study would have been useful in reassuring that neurological sequelae really did not occur in children after artemether therapy.

Secondly, there is a concern about the bioavailability of intramuscularly administered artemether in children in cerebral malaria. In a previous study(3) done on Kenyan children, 19% were found to have low serum drug concentrations with a significantly longer 50% parasite clearing time.

Finally, there are concerns about increasing prevalence of drug resistance (including artesunate) in India(4). Though the overall cost of artemether is comparable to that of quinine, one should use artemether judiciously in selected cases to prevent an escalation of drug resistance to artemether.

The data presented in this study still does not support the use of artemether as the first choice drug for severe malaria, and quinine is still the drug of first choice in my opinion.

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Reply

The study of Petras *et al.*(1) quoted by

Dr. Kumar was done on male rhesus monkey using high doses (8 mg/kg/day to 24 mg/kg/day) of arteether for 7 to 14 days. Neuropathological lesions were demonstrated only in the 14-day treated monkeys. The recommended doses and duration of therapy in humans is much less. Secondly, it may not be entirely appropriate to extrapolate results in experimental animals to humans. Moreover, a study in mouse model has demonstrated that neuropathological brain-stem damage due to intramuscular artemether is dose dependent and may be reversible(2). We do not perceive how longer duration of follow up in our study could have helped to detect neurological sequelae in the patients when they were clinically normal at discharge. A larger sample size would have definitely helped.

The study quoted on bioavailability of intramuscular artemether was done on just 26 children(3). This observation needs to be validated by larger studies.

We share Dr. Kumar's concern for emerging drug resistance and certainly do not recommend replacing quinine with artemether. We have only suggested that artemether may be a useful alternative to quinine in areas with poor medical facility for reasons already cited in the article.

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