used with macrolides, intravenous route should be preferred.

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Camphor Poisoning: Personal Experience

I wish to share my experience regarding camphor poisoning during last 32 years. I happen to see at least 5 to 6 camphor poisoning cases per year. Though I have not kept a record of these cases, my observations are as follows:

1. Camphor poisoning is exclusively seen in Hindus for whom camphor is an important component of puja material.
2. Todlers 1 to 3 years are commonly involved since they have easy access to camphor when they are playing arround grandparents busy in performing puja with camphor arround.
3. Most common presentation is afebrile seizures.
4. Camphor is so rapid acting that child gets seizures within seconds of camphor ingestion.
5. Very small doses can cause seizures. I remember a 1-year-old child brought with seizures who had consumed prasad of coconut piece just coated with camphor because both camphor and coconut were lying in close vicinity in same puja thali.
6. Camphor poisoning is so common in our region that I have made a dictum that any small child if brought with afebrile seizures for the first time in life, always ask history of camphor ingestion. In 50 to 60% cases I could get positive history. Generally parents do not provide history of camphor ingestion unless asked for and I have seen patients getting investigated in detail for that afebrile seizure episode in form of CSF/CT/EEG etc, which is unnecessary if you can extract the correct history.
7. Generally a single dose of IV midazolam was found to be enough and patient became totally normal within one or two hours, with no residual deficit.

All the above observations are based not on literature but purely on personal experience and evidence in pediatric practice over last 32 years.

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Hair Dye Poisoning [Paraphenylenediamine, Super Vasamol 33]

A 14-year-old girl was brought with consumption of around 50 mL of Super vasamol 33 hair dye one hour prior to presentation. She hailed from a village, had lost her father and consumed the dye with suicidal intention. Immediately, gastric lavage was given and she was shifted to PICU. On examination, her vitals were stable and there was no respiratory distress or upper airway obstruction. She developed cervico-facial edema within 4 hours of dye ingestion. Other systems examination was unremarkable. Her blood counts, blood Urea, creatinine, calcium, phosphrous, sodium, potassium, chloride were normal. Urine for albumin, sugar and blood was not detected. Urinary pH was 7.0 and microscopy was normal. Her blood sugar, arterial blood gases, PT and

APTT were normal. Her LDH was raised 513U/L [CK-NAC 97 U/L and CK-MB 21U/L. Patient received supportive care, and injection . hydrocortisone and chlorphenaramine maleate. Her vitals, urine colour and output was normal throughout the hospital stay. After 2 days, cervicofacial edema disappeared and repeat investigations were normal. She recovered and was discharged after psychiatric counselling on 5th day.

Super Vasmol [paraphenylenediamine], a cheap, freely-available, hair dye in rural areas is emerging as a major cause of suicidal poisoning in India [1]. However, it is rare in children. It causes serious multisystem toxicity with significant morbidity and mortality in children and clinical manifestations and outcome are similar to those in adults [2]. The predominant clinical manifestations is early onset (usually within six hours) severe cervico-facial edema and asphyxia often requiring an emergency tracheostomy. Later (within days or weeks); dark urine, oliguria, renal failure and rhabdomyolysis occurs [1]. Out of 150 adults, angioneurotic edema was encountered in all patients and 60% had ARF [3]. In a study from Egypt, cervicofacial and laryngeal edema was the dominating feature in 72% of adults [4]. Out of 1020 adults, typical cervicofacial edema was present in 73% and brown color urine in 52.82%, and mortality was 23.92% [5]. Out of 17 Sudanese children, 76.4% had attempted suicide and clinical manifestations are dominated by cervical and upper respiratory tract edema, rhabdomyolysis and acute renal failure. Out of them, 47% required tracheostomy for severe angioneurotic edema and 71% developed ARF [2]. Death is usually caused by angioneurotic edema or cardiac involvement and is dose-dependent [1,4]. Poor prognostic factors are late presentation, no gastric lavage, requiring intubation, and ventilation or dialysis [1]. The classical presentation that has been described of cervicofacial edema was evident in our child. Our child was brought to emergency in time within one hour and underwent gastric lavage, the most important intervention. Cases usually survive if they present to hospital within 4 hour of dye ingestion [5]. There is no specific diagnostic test or antidote for paraphenylene diamine poisoning.

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ADEM Following Malaria

I read with interest an article on acute demyelinating encephalomyelitis in a child following malaria [1]. Authors have mentioned that they could not find any report of ADEM following malaria in children. I want to highlight that we have reported a case of ADEM following *Plasmodium vivax malaria* in a child [2]. It was an extremely rare case as there were case reports of ADEM after *P. falciparum* malaria but only one case of ADEM after *P.vivax* malaria was reported prior to our case report [3].

Moreover the present case report does not prove that ADEM was solely due to *P. falciparum* malaria without any test for CSF viral antigens or viral serology. Most likely it was due to some viral CNS infections which resolved spontaneously.

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