

A review of the literature yielded no previous reports of Coxsackie B virus infection in children complicated by acute glomerulonephritis. The role of enteroviruses and especially Coxsackie B viruses in nephritis was suspected previously(2). Several experimental and early clinical studies have suggested nephritogenicity of Coxsackie B viruses and in particular B4(2,3). In a murine model, Coxsackie B4 virus found to induce IgA nephropathy(4). Conaldi, *et al.* studying frozen biopsy samples from patients with IgA nephropathy or other glomerulonephritides observed a significant association between Coxsackie B virus and IgA nephropathy(5).

The present report highlights, for the first time an association of Coxsackie B2 and B4 virus infection with acute glomerulonephritis. Further studies including ultrastructural examination for viral inclusions *in situ* hybridization for Coxsackie B virus on renal histology might be necessary to establish a "causal" effect.

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Fatal Rabies Despite Appropriate Post-exposure Prophylaxis

The Indian subcontinent accounts for almost half of the deaths worldwide due to rabies encephalitis. The magnitude of problem is compounded by inappropriate post-exposure rabies prophylaxis(1). We describe a case of fatal rabies despite use of purified chick-embryo vaccine (PCEV) and human rabies immunoglobulin (HRIG).

A 5-year-old girl presented to us with a 7

cm laceration with a flap hanging in front of the left eye following a stray dog bite 6 h back. Wound cleansing with povidone iodine and tetanus toxoid administration had been done at a nearby dispensary immediately after the bite. PCEV (Rabipur, Chiron Behring Vaccines Pvt. Ltd.) was given over deltoid region and 20 IU/kg of HRIG (RABGLOB, Bharat Serums & Vaccines Ltd.) was administered (~ 50% infiltrated locally and rest intramuscularly). Laceration was sutured after 24 h because of the high likelihood of a bad scar in the girl child. She was discharged after the 3rd dose of

Rabipur on day 7 and received the 4th dose on day 14. On day 17 following the bite, she reported with typical features of aerophobia and hydrophobia. Although the diagnosis of rabies was obvious, variants of Guillain-Barre syndrome and acute disseminated encephalomyelitis were also considered in view of the supervised vaccination profile. Investigations revealed polymorphonuclear leucocytosis in blood and lymphocytic pleocytosis in the CSF. The child rapidly deteriorated with dysautonomia, aspiration pneumonia, and seizures and died despite mechanical ventilation over the next 36 hours. Autopsy was positive for Negri bodies and rabies antigen.

Though an estimated 10 million people receive post-exposure treatments each year after being exposed to rabies suspect animals, only sporadic reports of failure of post exposure prophylaxis exist in published literature(2-4). Failure of prophylaxis has often been attributed to non-adherence to the WHO recommendations especially for class III bites(1,4). Given the current dismal status of post-exposure prophylaxis, reported failures in our country are surprisingly uncommon(1).

We encountered a case of failure of rabies prophylaxis despite adherence to WHO recommendations. A short incubation period, failure to infiltrate maximum HRIG locally due to anatomic nonfeasibility and suturing of the wound (even though done after PCEV and HRIG administration) could have been contributory in our case. Immunodepression, chronic disease, surgery under anesthesia,

concurrent use of serum and antimalarials, alcoholism and drugs, inhibition of response of vaccine by antisera/immunoglobulin and inability to maintain cold chain for vaccine or immunoglobulin in developing countries have been postulated for failure of rabies prophylaxis(2-5). Therapeutic failures also indicate an urgent need of reassessment of vaccines and sera in terms of their potency at user level(5).

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